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Archives of Endocrinology and Metabolism

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Prezados colegas, pesquisadores e congressistas,

É com enorme honra e entusiasmo que desejamos a todos as boas-vindas ao **CBEM 2026 – Congresso Brasileiro de Endocrinologia e Metabologia.**

Nesta edição, cruzamos a tradição e o prestígio da nossa especialidade com o que há de mais inovador na medicina contemporânea.

Sob uma perspectiva que une solidez científica, tecnologia de ponta e um olhar humanizado, estruturamos uma programação focada na vanguarda da Endocrinologia e da Metabologia. Nosso objetivo é debater desde os avanços genômicos e as terapias de precisão até o manejo clínico prático que transforma a vida dos nossos pacientes diariamente.

O CBEM 2026 foi desenhado para ser uma experiência imersiva e acolhedora. Este é o espaço ideal para reencontrar amigos, firmar novas colaborações e compartilhar o conhecimento que move a nossa comunidade médica para a frente.

Expresso meu profundo agradecimento à Comissão Organizadora, aos palestrantes nacionais e internacionais, e a cada um de vocês, que fazem deste congresso o maior ponto de encontro da Endocrinologia e Metabologia brasileira.

Aproveitem ao máximo cada conferência, painel, simpósio e momento de troca.

Sejam muito bem-vindos!
Cordialmente,



Dr. Rodrigo Moreira
Presidente CBEM 2026

MESSAGE FROM THE PRESIDENT/MENSAGEM DO PRESIDENTE

Endocrinology and Metabolism is experiencing an extraordinary moment. New concepts, pathophysiological mechanisms, technologies, medications, and therapeutic strategies are emerging at an impressive pace, rapidly transforming the way we understand and treat endocrine and metabolic diseases.

It is in this scenario that the 37th Brazilian Congress of Endocrinology and Metabolism (CBEM) will take place, with the mission of bringing Brazilian endocrinologists the most current and relevant advances in our field. Four intense days of knowledge, discussion, encounters, exchange of experiences, collaboration, and celebration await us, an opportunity not only to update our knowledge, but above all to connect with what is happening in Endocrinology in Brazil and around the world.

We are witnessing a true revolution: diseases that once had few or no effective treatment options are now being treated with therapies capable of changing their natural history; new disease mechanisms are being unveiled; and precision medicine is becoming a reality in clinical practice. At the same time, artificial intelligence is arriving to transform Medicine, opening new possibilities for diagnosis, clinical decision-making, and patient care. We need to embrace it, understand it, and use it in the service of science and, above all, our patients.

Ultimately, this will be much more than a scientific update. It will be an opportunity to witness this remarkable transformation as it unfolds.

The CBEM will challenge the laws of physics, bringing the present and the future together, at the same time and in the same place.

We warmly welcome you all to the 37th Brazilian Congress of Endocrinology and Metabolism.

A Endocrinologia e Metabologia vive um momento extraordinário. Novos conceitos, mecanismos fisiopatológicos, tecnologias, medicamentos e estratégias terapêuticas surgem em ritmo impressionante, transformando rapidamente a maneira como compreendemos e tratamos as doenças endócrinas e metabólicas.

É nesse cenário que se realiza o 37º Congresso Brasileiro de Endocrinologia e Metabologia (CBEM), com a missão de trazer para os endocrinologistas brasileiros o que há de mais atual e relevante na nossa especialidade. Serão quatro dias intensos de conhecimento, discussões, encontros, troca de experiências, colaboração e confraternização. Uma oportunidade de atualização e, sobretudo, de conexão com o que acontece na Endocrinologia no Brasil e no mundo.

Vivemos uma verdadeira revolução: doenças antes sem tratamentos eficazes passam a contar com terapias capazes de mudar sua história natural; novos mecanismos são desvendados; e a medicina de precisão começa a se tornar realidade na prática clínica. Ao mesmo tempo, a inteligência artificial chega para transformar a Medicina, trazendo novas possibilidades para o diagnóstico, a tomada de decisões e o acompanhamento de nossos pacientes. Precisamos aceitá-la, compreendê-la e utilizá-la em favor da ciência e de nossos pacientes.

Enfim, este será muito mais do que um congresso de atualização científica. Será uma oportunidade de testemunhar essa grande transformação.

O CBEM desafiará a física, mostrando-nos o presente e futuro ao mesmo tempo e no mesmo lugar.

Sejam todos muito bem-vindos ao 37º Congresso Brasileiro de Endocrinologia e Metabologia.



Dra. Marise Lazaretti Castro

Chair, CBEM Scientific Committee
Scientific Director, SBEM

Presidente da Comissão Científica do CBEM
Diretora científica da SBEM

MENSAGEM DO PRESIDENTE

CBEM 2026. Na Cidade Maravilhosa, um evento maravilhoso. Foi assim que a diretoria da SBEM e as comissões científica e organizadora idealizaram e planejaram o maior congresso da especialidade de endocrinologia e metabologia do Brasil e da América Latina. Que tenha sido essa também a percepção dos mais de 3.200 congressistas durante todo o período de intenso intercâmbio científico e cultural.

A cidade onde a SBEM nasceu, expandiu-se e decidiu firmar sua sede própria acolhe de braços abertos, por meio do simbolismo do Cristo Redentor, todos os participantes – palestrantes, congressistas, colaboradores, imprensa, expositores e patrocinadores. Uma Sociedade diversa e inclusiva, construtora e defensora da ciência e firmada no cerne da ética, organizará sempre grandes eventos... e não foi diferente dessa vez.

Por meio dos Prêmios SBEM, a Sociedade reconhece e homenageia quatro de seus associados pelo brilhantismo científico e pela contribuição para a pesquisa, o ensino e a própria construção da sociedade: Claudio Elias Kater, Ana Claudia Latrônico Xavier, Margaret Cristina da Silva Boguszewski e Henrique de Lacerda Suplicy, que, respectivamente, foram agraciados por seus pares com os prêmios Luiz César Póvoa, José Dantas de Souza Leite, José Schermann e Antônio Barros de Ulhôa Cintra.

Também como reconhecimento ao estudo e às publicações científicas, foram entregues os prêmios ao primeiro colocado da prova do concurso para a obtenção do Título de Especialista em Endocrinologia e Metabologia (TEEM) (prêmio intitulado Bernardo Leo Wajchenberg) ao Dr. Carlos Henrique Bizinoto Martins, e aos melhores trabalhos publicados em 2025 nos AE&M: o da área clínica (Prêmio Waldemar Berardinelli), ao Dr. José Gustavo Olijnyk, e o da área Básica (Prêmio Thales Martins) ao Dr. Leonardo Maeda.

A prática de um bom estilo de vida, como mecanismo de prevenção e controle das principais doenças da endocrinologia e metabologia, é uma das premissas da especialidade. A SBEM, por meio do Departamento de Endocrinologia do Esporte e do Exercício, decidiu reconhecer, com o Prêmio Endo Atleta, médicos que dão muito mais que uma receita desse princípio – exercem, praticam e comprovam. Os cinco agraciados foram: Paulo Gustavo Sampaio Lacativa, Ronaldo José Pineda Wieselberg, Ana Claudia Ramalho Lacerda, Dhianah Santini de Oliveira Chachamovitz e Vivian Carole Moema Ellinger.

Em mais uma edição do “Projeto Preceptoria: Endocrinologista do Futuro”, 137 jovens residentes tiveram acesso a uma programação variada e relevante para a construção da carreira na especialidade.

Esta edição dos AE&M terá um sentido duplamente especial: coincidirá com os 75 anos da revista científica oficial da SBEM e trará os 516 trabalhos selecionados e aprovados para apresentação no CBEM 2026, a fim de que possam ser lidos e estudados, de modo a propiciar atualização também no pós-congresso. Passado o excelente evento, tenham uma ótima leitura desse suplemento.



Dr. Neuton Dornelas Gomes

Presidente da SBEM (2025-2026)

Divulgados os Laureados dos Prêmios SBEM em Cerimônias Surpresa



A Sociedade Brasileira de Endocrinologia e Metabologia (SBEM) anunciou os laureados dos Prêmios SBEM 2026, uma das mais importantes distinções da especialidade no país.

Em uma iniciativa marcada por emoção e proximidade, os quatro especialistas foram comunicados por meio de cerimônias surpresa realizadas online e presenciais, reunindo diretoria, colegas, alunos e familiares.

A entrega oficial da premiação acontecerá durante o Congresso Brasileiro de Endocrinologia e Metabologia (CBEM 2026), no Rio de Janeiro, de 26 a 29 de agosto, quando os homenageados também participarão de conferências especiais.

Nesta edição, foram reconhecidos:

- Dra. Margaret Boguszewski – Prêmio José Schermann
- Dra. Ana Claudia Latrônico – Prêmio José Dantas de Souza Leite
- Dr. Claudio Kater – Prêmio Luiz César Póvoa
- Dr. Henrique Suplicy – Prêmio Antonio Barros de Ulhôa Cintra

A premiação reforça o compromisso da SBEM em valorizar trajetórias que contribuíram de forma decisiva para o avanço da Endocrinologia e Metabologia, tanto na assistência quanto na pesquisa e na formação de especialistas.

RECONHECIMENTO COM EMOÇÃO

A Dra. Margaret Boguszewski foi surpreendida durante o encerramento da turma de residentes no Serviço de Endocrinologia e Metabologia do Hospital de Clínicas da UFPR, em Curitiba. A homenagem contou com a participação do presidente da SBEM, Dr. Neuton Dornelas, da diretoria da SBEM Paraná, além de equipes da Sociedade. Também estiveram presentes, de forma online, os Drs. Luiz Claudio Castro e Sonir Antonini, representando a endocrinologia pediátrica, e o Dr. Rodrigo Moreira, presidente do CBEM 2026.

Indicada pela Dra. Adriane Cardoso-Demartini e pelo Dr. Luiz de Lacerda Filho, com apoio de 30 associados da SBEM-PR, a especialista recebeu o reconhecimento com emoção. “A SBEM faz parte da minha formação e da minha vida”, afirmou. O Prêmio José Schermann destaca profissionais com atuação relevante no ensino e na prática clínica.

A Dra. Ana Claudia Latrônico foi comunicada de sua premiação em uma sessão coordenada pelo Dr. Madson Almeida. Escolhida para receber o Prêmio José Dantas de Souza Leite, teve sua indicação apoiada por diversos colegas da especialidade.

Durante a homenagem, o presidente da SBEM ressaltou a relevância histórica do prêmio e a contribuição da Dra. Ana Claudia para a medicina, especialmente na pesquisa e na formação acadêmica. Já a Dra. Marise Lazaretti Castro, diretora Científica da SBEM, destacou sua liderança e excelência científica. Emocionada, a Dra. Ana Claudia reforçou o caráter coletivo da conquista, reconhecendo o papel de mentores, colaboradores e alunos em sua trajetória.

Já o Dr. Claudio Kater recebeu o Prêmio Luiz César Póvoa durante uma sessão na Unifesp, após indicação da Dra. Flavia Amanda e do Dr. Magnus Silva. Surpreendido durante o que imaginava ser uma discussão clínica, celebrou o reconhecimento ao lado de colegas da Universidade e da diretoria da SBEM.

Durante a homenagem, o Dr. Neuton Dornelas ressaltou a trajetória do endocrinologista, marcada pela atuação em hospitais e pela formação de diversas gerações de especialistas. A Dra. Marise destacou que a escolha do nome do Dr. Claudio Kater foi praticamente unânime entre cerca de 40 colegas consultados.

O Dr. Henrique Suplicy foi surpreendido durante um evento no Paraná, com o anúncio do Prêmio Antonio Barros de Ulhôa Cintra. Na ocasião, o Dr. Neuton Dornelas e a diretoria da SBEM que estavam reunidos em um evento em Brasília, destacou que sua trajetória se confunde com a própria consolidação da endocrinologia no Paraná e no Brasil.

Emocionado, Dr. Suplicy agradeceu muito às lembranças de sua longa dedicação à especialidade, incluindo a missão recebida do Dr. Luiz César Póvoa de contribuir para a preservação da história da Endocrinologia e Metabologia brasileira. A homenagem foi marcada por momentos de celebração entre colegas.

VALORIZAÇÃO DA CIÊNCIA E INSPIRAÇÃO PARA NOVAS GERAÇÕES

Os quatro laureados representam diferentes áreas de atuação dentro da Endocrinologia e Metabologia, mas compartilham em comum uma sólida produção científica, forte atuação institucional e dedicação à formação médica.

Ao reconhecer essas trajetórias, a SBEM reafirma seu compromisso com a valorização da ciência, da ética e do ensino como pilares fundamentais para o avanço da especialidade no Brasil.

Mais do que homenagear carreiras consolidadas, o Prêmio SBEM também inspira novas gerações de endocrinologistas a seguir contribuindo para o desenvolvimento da medicina com ética e valores.

CBE 2026 RIO DE JANEIRO

37° CONGRESSO BRASILEIRO DE ENDOCRINOLOGIA E METABOLOGIA

SBEM

SCIENTIFIC PROGRAM

26/08/2026 – QUARTA-FEIRA

WINDSOR OCEÂNICO HOTEL: SALA ARPOADOR (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 MESA-REDONDA
ENGRAVIDEI E AGORA? MANEJO DAS DOENÇAS ADRENAIS NA GESTAÇÃO

08:30-10:00
Moderador: DANIELA ESPÍNDOLA ANTUNES (GO) – Unifesp

08:30-10:00
Moderador: MARGARET DE CASTRO (SP) – FMRP-USP

08:30-08:40 Tema livre: Total RNA Sequencing Reveals an Expanded Landscape of Gene Fusion Complexity in Pheochromocytomas and Paragangliomas
Apresentador(a): EDUARDO CUNHA LOBATO

08:40-08:55 Insuficiência Adrenal
Palestrante: SÉRGIO LERIAS DE ALMEIDA (RS)

08:55-09:10 Feocromocitoma
Palestrante: GUSTAVO F. C. FAGUNDES

09:10-09:25 Hiperplasia Adrenal Congênita
Palestrante: MARIVÂNIA DA COSTA SANTOS (PB) – UFPB

09:25-09:40 Hiperaldosteronismo primário
Palestrante: FLAVIA AMANDA COSTA BARBOSA (SP) – EPM/Unifesp

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 PAINEL
CONVERSA COM PROFESSOR

10:30-11:00 Hiperprolactinemias e Prolactinomas – Discussão de casos clínicos
Palestrante: CESAR LUIZ BOGUSZEWSKI (PR) – UFPR

11:00-11:30 Hiperprolactinemias e Prolactinomas – Discussão de casos clínicos
Palestrante: ANDREA GLEZER (SP) – HC-FMUSP

11:40-12:50 PROJETO PRECEPTORIA
"ENDOCRINOLOGISTA DO FUTURO" – PARTE 1

11:40-12:50
Moderador: RAQUEL BEATRIZ GONÇALVES MUNIZ (RJ)

11:40-12:50
Moderador: MARISE LAZARETTI CASTRO (SP)

11:40-11:50 Abertura e boas-vindas aos residentes e especializandos
Palestrante: NEUTON DORNELAS GOMES (DF)

11:40-11:50 Abertura e boas-vindas aos residentes e especializandos
Palestrante: RODRIGO DE OLIVEIRA MOREIRA (MG)

11:50-12:05 Oportunidades na carreira da Endocrinologia: na medicina diagnóstica
Palestrante: MARIA IZABEL CHIAMOLERA (SP)

12:05-12:20 Oportunidades na carreira da Endocrinologia: na pesquisa clínica
Palestrante: PAULO GUSTAVO SAMPAIO LACATIVA (RJ)

12:20-12:35 Oportunidades na carreira da Endocrinologia: Desafios e oportunidades para uma carreira no exterior
Palestrante: ANA PAULA ABREU METZGER (ESTADOS UNIDOS DA AMÉRICA)

12:35-12:50 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PROJETO PRECEPTORIA
"ENDOCRINOLOGISTA DO FUTURO" – PARTE 2

13:30-15:00
Moderador: RAQUEL BEATRIZ GONÇALVES MUNIZ (RJ)

13:30-15:00
Moderador: ANTONIO BENTES DE FIGUEIREDO JUNIOR (SP)

13:30-13:45 Como construir um consultório sólido nos primeiros anos de carreira
Palestrante: ANDRESSA MARTINS DE OLIVEIRA (RJ)

13:45-14:00 Publicidade médica – o que é ético?
Palestrante: MATEUS DORNELLES SEVERO (RS) – HUSM/UFSM

14:00-14:15 Inteligência artificial na endocrinologia: aplicações reais no consultório
Palestrante: JOSE LUCIANO DE FRANÇA ALBUQUERQUE (PE)

14:15-14:30 Empatia na prática: como melhores resultados clínicos através da comunicação
Palestrante: PRISCILLA GIL (RJ)

14:30-14:50 Perguntas e Respostas
14:50-15:00 Sorteios, Encerramento e Confraternização (durante o Coffee)

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 PAINEL
COM VISTAS PARA O FUTURO DA MEDICINA E DA PESQUISA: QUAL É O CAMINHO PARA NOS TORNARMOS PROTAGONISTAS?

15:30-16:30
Moderador: MARISE LAZARETTI CASTRO (SP)

15:30-16:30
Moderador: MARIA TEREZA NUNES (SP)

15:30-15:40 Panorama da medicina e da pesquisa no Brasil: avanços e gargalos
Palestrante: DENISE PIRES DE CARVALHO (RJ)

15:40-15:50 Como transformar pesquisa em inovação?
Palestrante: MARCO ANTONIO ZAGO (SP)

15:50-16:10 Como as sociedades de especialidades podem auxiliar neste processo?
Palestrante: DENISE PIRES DE CARVALHO (RJ)

15:50-16:10 Como as sociedades de especialidades podem auxiliar neste processo?
Palestrante: MARCO ANTONIO ZAGO (SP)

16:10-16:30 Perguntas e Respostas

16:30-18:00 PAINEL
DA BANCADA À CLÍNICA: DESTAQUES NO ANO TRANSLACIONAL EM ...

16:30-18:00
Moderador: RENAN MAGALHÃES MONTENEGRO JUNIOR (CE) – Faculdade de Medicina da UFC

16:30-18:00
Moderador: FRANCISCO JOSÉ ALBUQUERQUE DE PAULA (CE) – FMRP-USP

16:30-16:45 Diabetes
Palestrante: CRISTIANE BAUERMANN LEITÃO (RS) – UFRGS e HCPA

16:45-17:00 Neuroendocrinologia
Palestrante: SÍLVIA REGINA CÔRREA DA SILVA (SP) – Unifesp

17:00-17:15 Obesidade
Palestrante: MARIO JOSÉ ABDALA SAAD (SP)

17:15-17:30 Onco-endocrinologia
Palestrante: LAURA STERIAN WARD (SP)

17:30-18:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA BARRA DA TIJUCA (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
TRT NO HIPOGONADISMO MASCULINO: AQUI PODE?

08:30-10:00
Moderador: RENATO GALVÃO REDORAT (RJ)

08:30-10:00
Moderador: LUCAS BANDEIRA MARCHESAN (RS) – Hospital Nossa Senhora da Conceição

08:30-08:40 Tema livre: Switching from short-acting to long-acting testosterone in transgender men: higher serum testosterone levels without increased erythrocytosis
Apresentador(a): FELIPE BEHRENDS RODRIGUES

08:40-08:55 Hipogonadismo funcional: SERM vs TRT
Palestrante: MARCELO FERNANDO RONSONI (SC) – UFSC

08:55-09:10 Câncer de próstata
Palestrante: FRANCISCO RICARDO NOGUEIRA AZEREDO COUTINHO (RJ)

09:10-09:25 Fraturas
Palestrante: TAYANE MUNIZ FIGHERA (RS)

09:25-09:40 Risco cardiovascular
Palestrante: FÁBIO FERREIRA DE MOURA (PE) – Universidade de Pernambuco

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 PONTO DE VISTA
VIROSES, IMUNIZAÇÕES E REPERCUSSÕES NO DIABETES

10:30-11:30
Moderador: ADRIANA COSTA E FORTI (CE) – UFC

10:30-11:30
Moderador: JOÃO EDUARDO NUNES SALLES (SP) – Faculdade de Ciências Médicas da Santa Casa de São Paulo

10:30-11:00 Apresentação e discussão de casos clínicos
Palestrante: REINE MARIE CHAVES FONSECA (BA)

11:00-11:30 Apresentação e discussão de casos clínicos
Palestrante: SAULO CAVALCANTI DA SILVA (MG)

11:40-12:40 – SIMPÓSIO PATROCINADO
SIMPÓSIO: BESINS

11:40-12:40 Conversando com os especialistas – Quando a menopausa, o hipogonadismo masculino e a obesidade chegam juntos na vida do casal
Palestrante: ALEXANDRE HOHL (SC) – UFSC

11:40-12:40 Conversando com os especialistas – Quando a menopausa, o hipogonadismo masculino e a obesidade chegam juntos na vida do casal
Palestrante: TAYANE MUNIZ FIGHERA (RS)

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 SIMPÓSIO
SBEM X LATS – DESTAQUES DO GUIDELINE DA ATA PARA CÂNCER DE TIREOIDE

13:30-15:00
Moderador: CÉLIA REGINA NOGUEIRA (SP) – Unesp

13:30-15:00
Moderador: DANIEL ALVES BULZICO (RJ)

13:30-13:40 Tema livre: Relationship Between BRAF V600E Allele Frequency and Aggressive Clinicopathological Features in Papillary Thyroid Carcinoma
Apresentador(a): MARCOS TADEU DOS SANTOS (SP) – Onkos/mir-THYpe

13:40-14:00 Estratificação de risco
Palestrante: JOSE MIGUEL DOMINGUEZ (CHILE)

14:00-14:20 Tratamento inicial
Palestrante: CAROLINA FERRAZ DA SILVA (PE) – FCMSCSP

14:20-14:40 Uso racional de RAI
Palestrante: DÉBORA LÚCIA SEGURO DANILOVIC (SP) – ICESP/FMUSP

14:40-15:00 Perguntas e Respostas
15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 – SIMPÓSIO PATROCINADO
SIMPÓSIO: BRACEPHARMA

15:30-16:00 Da Dopamina ao GLP-1: Estratégias Combinadas no Tratamento da Compulsão e da Obesidade
Palestrante: ALEXANDRE HOHL (SC) – UFSC

16:00-16:30 Da Dopamina ao GLP-1: Estratégias Combinadas no Tratamento da Compulsão e da Obesidade
Palestrante: DANIEL MARTINEZ (SP)

16:30-17:00 PAINEL
SBEM - INTERNATIONAL DIABETES FEDERATION – IDF

16:30-17:00
Moderador: HERMELINDA CORDEIRO PEDROSA (DF) – FEPECS – International Diabetes Federation (IDF)

16:30-17:00 Conferência: Diabetes tipo 5: acumulando evidências para esta diferente forma de diabetes
Palestrante: MEREDITH A. HAWKINS (ESTADOS UNIDOS DA AMÉRICA)

17:00-18:00 SIMPÓSIO
SBEM – INTERNATIONAL DIABETES FEDERATION – SBD: MESA-REDONDA: REVISITANDO DIABETES RELACIONADO À DESNUTRIÇÃO

17:00-18:00
Moderador: JOÃO EDUARDO NUNES SALLES (SP) – Faculdade de Ciências Médicas da Santa Casa de São Paulo

17:00-18:00
Moderador: FÁBIO FERREIRA DE MOURA (PE) – Universidade de Pernambuco

17:00-17:15 Chamando a atenção global para uma forma importante, mas ainda negligenciada, de diabetes (“Bringing Global Attention to a Neglected yet Important Form of Diabetes”)
Palestrante: MEREDITH A. HAWKINS (ESTADOS UNIDOS DA AMÉRICA)

17:15-17:30 O papel da desnutrição precoce no desenvolvimento do diabetes tipo 5 ("The role of early-life malnutrition in the development of type 5 diabetes")
Palestrante: BIANCA DE ALMEIDA PITITTO (RJ)

17:30-17:45 Superando os desafios diagnósticos: implementando a plataforma de rastreamento da IDF no Brasil ("Overcoming Identification Challenges: Implementing the IDF Screening Platform in Brazil")
Palestrante: HERMELINDA CORDEIRO PEDROSA (DF) – FEPECS – International Diabetes Federation (IDF)

17:45-18:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA COPACABANA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 OFICINA
OFICINA ENDOCRINOLOGIA DO EXERCÍCIO: 1 – SÍNDROME DA DEFICIÊNCIA ENERGÉTICA RELATIVA AO EXERCÍCIO – REDs

08:30-10:00
Moderador: DOLORES PEROVANO PARDINI (SP)

08:30-10:00
Moderador: HELOISA THEODORO (RS)

08:30-08:40 Tema livre: Beyond Binary Cutoffs: A Percentile-Based Tool for Handgrip Strength Interpretation
Apresentador(a): EDUARDO MEDEIROS FERREIRA DA GAMA (RJ)

08:40-08:55 Diagnóstico e conduta da REDs
Palestrante: CRISTIANO ROBERTO GRIMALDI BARCELLOS (SP)

08:55-09:10 Avaliação e conduta da disponibilidade energética
Palestrante: FERNANDA BRUNACCI (SP)

09:10-09:25 Quando e como iniciar reposição hormonal em mulheres atletas?
Palestrante: ANDREA MESSIAS BRITTO FIORETTI (SP) – Unifesp

09:25-09:40 Avaliação da massa óssea e composição corporal em atletas
Palestrante: SERGIO SETSUO MAEDA (SP) – Unifesp

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-12:00 OFICINA
OFICINA ENDOCRINOLOGIA DO EXERCÍCIO: 2 – DESAFIOS DA ENDOCRINOLOGIA DO EXERCÍCIO

10:30-11:50
Moderador: JOÃO CARLOS RAMOS DIAS (SP)

10:30-11:50
Moderador: ADRIANA COGNOLATO (SP)

10:30-10:40 Tema livre: Physical activity independently modulates metabolic adaptation during testosterone replacement in men with congenital hypogonadotropic hypogonadism: a randomized longitudinal study
Apresentador(a): MAICON CESAR CANUTO (PR)

10:40-10:55 Meu filho pode praticar musculação?
Palestrante: ROGÉRIO FRIEDMAN (RS)

10:55-11:10 Vencendo a inércia – novas estratégias para estimular a prática do exercício (snack exercise, contagem de passos etc.)
Palestrante: CRISTINA DA SILVA SCHREIBER DE OLIVEIRA (SC)

11:10-11:25 Abuso de esteroides anabolizantes
Palestrante: ALEXIS DOURADO GUEDES (BA) – Centro de Diabetes e Endocrinologia do Estado da Bahia

11:25-11:40 Exercício: o grande medicamento de combate à dependência funcional do idoso
Palestrante: LUCIANA DE OLIVEIRA LOTTENBERG (MG)

11:40-12:00 Perguntas e Respostas

12:00-12:40 OFICINA
OFICINA ENDOCRINOLOGIA DO EXERCÍCIO: 3 – DICAS DE MÉDICOS ATLETAS

12:00-12:40
Moderador: FULVIO CLEMO SANTOS TOMASELLI (RS) – Universidade Regional de Blumenau

12:00-12:40
Moderador: CRISTINA DA SILVA SCHREIBER DE OLIVEIRA (SC)

12:00-12:10 Natação
Palestrante: LÍVIA MARCELA DOS SANTOS (SP) – Centro Universitário São Camilo

12:10-12:20 Maratona
Palestrante: JOSIVAN GOMES DE LIMA (RN) – UFRN

12:20-12:30 Corrida
Palestrante: ANA CLÁUDIA REBOUÇAS RAMALHO (BA)

12:30-12:40 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 MESA-REDONDA
MESA-REDONDA: QUAL O IMPACTO DO EXERCÍCIO

13:30-15:00
Moderador: VICTÓRIA ZEGHBI COCHENSKI BORBA (PR) – UFPR

13:30-15:00
Moderador: JOSIVAN GOMES DE LIMA (RN) – UFRN

13:30-13:40 Tema Livre: ASSOCIATION BETWEEN TYPES OF PHYSICAL ACTIVITY AND THE CARDIOMETABOLIC PROFILE OF WOMEN IN PERIMENOPAUSE: A CROSS-SECTIONAL STUDY IN PERNAMBUCO, BRAZIL
Apresentador(a): ISABELA CAROLINA LOPES COELHO

13:40-13:55 No Lipedema?
Palestrante: CRISTINA DA SILVA SCHREIBER DE OLIVEIRA (SC)

13:55-14:10 Na Obesidade Sarcopênica?
Palestrante: FULVIO CLEMO SANTOS TOMASELLI (RS) – Universidade Regional de Blumenau

14:10-14:25 Na Osteosarcopenia?
Palestrante: MONICA LONGO DE OLIVEIRA (SP)

14:25-14:40 No Controle Glicêmico?
Palestrante: DENISE REIS FRANCO (SP)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 PAINEL
O QUE AS NOVAS DIRETRIZES DE OBESIDADE FALAM SOBRE

15:30-16:30
Moderador: CAROLINA CASTRO PORTO SILVA JANOVSKY (SP)

15:30-16:30
Moderador: AMÉLIO FERNANDO GODOY MATOS (RJ)

15:30-15:50 O tratamento Farmacológico
Palestrante: FERNANDO GERCHMAN (RS) – UFRGS

15:50-16:10 A Doença Cardiovascular
Palestrante: MARCELLO CASACCIA BERTOLUCI (RS) – UFRGS

16:10-16:30 Perguntas e Respostas

16:30-18:00 PAINEL
ENFRENTANDO OS MEDOS DO USO DAS TERAPIAS BASEADAS EM INCRETINAS

16:30-18:00
Moderador: ROSANE KUPFER (RJ) – IEDE

16:30-18:00
Moderador: MARCIO CORRÊA MANCINI (SP) – HCFMUSP

16:30-16:40 Tema Livre: Dietician Support Substantially Enhances Weight Loss Outcomes in a Digital GLP-1 Receptor Agonist Program: A Real-World Brazilian Cohort Study
Apresentador(a): ÂNGELA CRISTINE BERSCH FERREIRA (SP)

16:40-16:55 Desfechos Oculares: o que o endocrinologista precisa saber?
Palestrante: FERNANDO KORN MALERBI (SP)

16:55-17:10 Efeitos adversos gastrointestinais: como manejar adequadamente
Palestrante: CAROLINA CASTRO PORTO SILVA JANOVSKY (SP)

17:10-17:25 Nódulos e câncer de tireoide
Palestrante: GLAUCIA MARIA FERREIRA DA SILVA MAZETO (SP) – Universidade Estadual Paulista "Júlio de Mesquita Filho" – UNESP

17:25-17:40 Segurança do uso em adolescentes
Palestrante: LÍVIA LUGARINHO CÔRREA DE MELLO (RJ) – IEDE

17:40-18:00 Perguntas e Respostas

18:00-19:30 SESSÃO
SESSÃO DE ABERTURA

18:00-19:30

Sala: SALA COPACABANA (WINDSOR OCEÂNICO HOTEL)

Eixo: CBEM 2026

WINDSOR OCEÂNICO HOTEL: SALA IPANEMA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 MESA-REDONDA
OBESIDADE – SÓ PENSO EM COMIDA: FOOD NOISE OU O QUÊ?

08:30-10:00
Moderador: CINTIA CERCATO (SP) – Faculdade de Medicina da Universidade de São Paulo

08:30-10:00
Moderador: MARIO JOSÉ ABDALA SAAD (SP)

08:30-08:40 Tema Livre: USE OF THE BRAZILIAN PORTUGUESE VERSION OF THE EBA-O-BR QUESTIONNAIRE (EATING BEHAVIORS ASSESSMENT FOR OBESITY) IN PEOPLE WITH TYPE 2 DIABETES MELLITUS AND OBESITY
Apresentador(a): DANIELLE LUMI KAGUE

08:40-08:55 Food noise: neurobiologia e manejo na prática clínica
Palestrante: SIMONE VAN DE SANDE LEE (SC)

08:55-09:10 Transtorno da compulsão alimentar
Palestrante: MARIA FERNANDA FREITAS FERREIRA MOREIRA

09:10-09:25 Uso de escala de comportamento alimentar no tratamento da obesidade
Palestrante: ANDRESSA HEIMBECHER SOARES (SP)

09:25-09:40 Obesidade, cérebro e recompensa: novas fronteiras terapêuticas
Palestrante: LICIO AUGUSTO VELLOSO (SP)

09:00-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:40 PRÊMIO
PRÊMIO LUIZ CESAR PÓVOA

10:30-10:55 Apresentação (do significado do prêmio e anúncio do laureado)
Palestrante: NEUTON DORNELAS GOMES (DF)

10:55-11:10 Apresentação: (da biografia do laureado)
Palestrante: MAGNUS REGIOS DIAS DA SILVA (SP) – EPM/Unifesp

10:55-11:10 Apresentação: (da biografia do laureado)
Palestrante: FLAVIA AMANDA COSTA BARBOSA (SP) – EPM/Unifesp

11:10-11:40 Conferência: "60 anos trabalhando e se divertindo ao mesmo tempo"
Palestrante: CLAUDIO ELIAS KATER (SP) – Unifesp

11:40-12:40 DEBATE
CONDUTAS NO PACIENTE COM HIPERALDOSTERONISMO PRIMÁRIO SOB A ÓTICA DO CONSENSO DA ENDOCRINE SOCIETY 2025

11:40-12:40
Moderador: AYRTON CUSTODIO MOREIRA (SP) – Faculdade de Medicina de Ribeirão Preto da Universidade de São Paulo FMUSP-RP

11:40-12:10 Indicações de uso de antagonistas do receptor mineralocorticoide *versus* indicação de adrenalectomia
Palestrante: LÍVIA MARA MERMEJO (SP)

12:10-12:40 Indicações de uso de antagonistas do receptor mineralocorticoide *versus* indicação de adrenalectomia
Palestrante: MADSON QUEIROZ DE ALMEIDA (SP)

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
COMPLICAÇÕES CRÔNICAS NO DIABETES

13:30-15:00
Moderador: ALBERTO DA SILVA DIAS FILHO (GO)

13:30-15:00
Moderador: ANTONIO ROBERTO CHACRA

13:30-13:40 Tema Livre: Relation between diet components and the progression of chronic kidney disease in patients with type 2 diabetes: a prospective cohort study
Apresentador(a): CAROLINA BANDAZ DE MENEZES

13:40-13:55 Desfechos oculares e uso de análogos de GLP1 e duplos
Palestrante: SOLANGE TRAVASSOS DE FIGUEIREDO ALVES (RJ) – Departamento de Saúde Ocular da SBD

13:55-14:10 Cognição e diabetes: estamos esquecendo de alguma coisa?
Palestrante: MARIA LUCIA CARDILLO CORRÊA GIANNELLA (SP)

14:10-14:25 DM1 e doença renal crônica
Palestrante: JOÃO ROBERTO DE SÁ (SP) – Centro Universitário Faculdade de Medicina do ABC

14:25-14:40 Obesidade no DM1
Palestrante: WELLINGTON SANTANA DA SILVA JÚNIOR (MA) – UFMA

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 PAINEL
CASOS DESAFIADORES PELO EXCESSO DE FRATURAS

15:30-16:30
Moderador: MIGUEL MADEIRA (RJ) – UFRJ

15:30-16:30
Moderador: ANGELA CRISTINA GOMES BORGES LEAL (SE) – UFS

15:30-15:40 Tema Livre: The unexplored role of vitamin D pathway polymorphisms on phenotypic heterogeneity of sporadic primary hyperparathyroidism in a Brazilian population
Apresentador(a): GUSTAVO KENDY CAMARGO KOGA (PR)

15:40-16:00 Fragilidade óssea na pré-menopausa
Palestrante: MANUELA GIULIANI MARCONDES ROCHA BRAZ (SP) – Universidade de São Paulo (USP)

16:00-16:20 Fragilidade óssea na gestação, puerpério e na lactante
Palestrante: SERGIO SETSUO MAEDA (SP) – Unifesp

16:20-16:30 Perguntas e Respostas

16:30-18:00 PAINEL
AVANÇOS NAS DISLIPIDEMIAS ATEROGÊNICAS

16:30-18:00
Moderador: JOANA RODRIGUES DANTAS VEZZANI (RJ) – Hospital Universitário Clementino Fraga Filho –UFRJ

16:30-18:00
Moderador: JORGE EDUARDO DA SILVA SOARES PINTO (RJ)

16:30-16:40 Tema Livre: Coronary artery calcium score in cardiovascular risk stratification for type 1 diabetes mellitus: a systematic review and meta-analysis of prevalence, prognostic value, and incremental value
Apresentador(a): DANIEL MARTINS DOS SANTOS

16:40-16:55 Novas Evidências com iPCSK-9
Palestrante: JOAQUIM CUSTÓDIO DA SILVA JUNIOR (SP) – UFBA

16:55-17:10 Lp(a) elevada: rastreio, estratificação ... e aí?
Palestrante: FLÁVIA COIMBRA PONTES MAIA (MG)

17:10-17:25 Papel da terapia anti-inflamatória no tratamento do risco residual
Palestrante: MÁRCIO WEISSHEIMER LAURIA (MG) – UFMG

17:25-17:40 Novas perspectivas no tratamento das dislipidemias: o que está por vir?
Palestrante: MARIA HELANE COSTA GURGEL (CE) – Universidade Federal do Ceará UFC

17:40-18:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA LEBLON (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
ATUALIZAÇÃO NO MANEJO DA DOENÇA DE GRAVES E DOENÇA OCULAR DA TIREOIDE

08:30-10:00
Moderador: MÁRIO VAISMAN (RJ)

08:30-10:00
Moderador: JOÃO ROBERTO MACIEL MARTINS (MG)

08:30-08:40 Tema Livre: Iodide Availability Controls Thyroid Cell Identity, ERK Signaling, and Redox Balance
Apresentador(a): ANTONIO WESKLEY SOARES

08:40-08:55 Discussão de casos clínicos
Palestrante: MÁRIO VAISMAN (RJ)

08:40-08:55 Discussão de casos clínicos
Palestrante: JOÃO ROBERTO MACIEL MARTINS (MG)

08:55-09:10 Peculiaridades do controle da função tireoidiana em pacientes com DOT
Palestrante: GUSTAVO CANCELA E PENNA (MG)

09:10-09:25 Tratamento definitivo na DG: quem e quando?
Palestrante: ADRIANO NAMO CURY (SP) – Faculdade de Medicina da Santa Casa de São Paulo

09:25-09:40 Tratamento medicamentoso da DOT em 2025
Palestrante: DANILO GLAUCO PEREIRA VILLAGELIN NETO (SP) – Hospital PUC Campinas

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 PAINEL
DA BANCADA À CLÍNICA: O ANO EM TIROIDE

10:30-11:40
Moderador: VALÉRIA CUNHA CAMPOS GUIMARÃES (DF)

10:30-10:50 Básico/translacional
Palestrante: MARIA TEREZA NUNES (SP)

10:50-11:10 Clínico
Palestrante: RUI MONTEIRO DE BARROS MACIEL (SP) – Unifesp

11:10-11:40 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
QUANDO O HIPOGONADISMO MASCULINO ACONTECE FORA DO ÓBVIO

13:30-15:00
Moderador: CICILIANA MAILA ZILIO RECH (RS) – Universidade Federal da Fronteira Sul

13:30-15:00
Moderador: LETICIA FERREIRA GONTIJO SILVEIRA (MG) – UFMG

13:30-13:40 Tema Livre: Evaluation of Insulin Resistance Prevalence Using the TyG Index and associated risk factors in a Cohort of Brazilians Elderly
Apresentador(a): LARA IMBROISI ERRERA

13:40-13:55 Síndrome pós-finasterida
Palestrante: MARCELO FERNANDO RONSONI (SC) – UFSC

13:55-14:10 Canabidiol
Palestrante: DANIEL BARRETTO KENDLER (RJ) – Universidade do Estado do Rio de Janeiro UERJ

14:10-14:25 Usuários de esteroides anabolizantes androgênicos
Palestrante: RICARDO DE ANDRADE OLIVEIRA (RJ)

14:25-14:40 Associado a REDs
Palestrante: FULVIO CLEMO SANTOS TOMASELLI (RS) – Universidade Regional de Blumenau

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 – SIMPÓSIO PATROCINADO
SIMPÓSIO: BOEHRINGER

15:30-16:30 Glucagon na medida certa: de renegado a aliado na perda de peso qualificada e na saúde metabólica
Palestrante: CYNTHIA MELISSA VALERIO (RJ) – UFRJ

15:30-16:30 Glucagon na medida certa: de renegado a aliado na perda de peso qualificada e na saúde metabólica
Palestrante: BRUNO GELONEZE NETO (SP) – Unifesp

16:30-18:00 PAINEL
CORPO, IDENTIDADE E HORMÔNIOS

16:30-18:00
Moderador: AMANDA VALÉRIA LUNA DE ATHAYDE (RJ)

16:30-18:00
Moderador: AMANDA DE ARAUJO LAUDIER (RJ)

16:30-16:40 Tema Livre: TESTOSTERONE USE IN TRANSGENDER MALE ATHLETES: THERAPEUTIC LIMITS, SAFETY MONITORING, AND INTERFACE WITH ANTI-DOPING.
Apresentador(a): RODRIGO EDUARDO LOPES COSTA

16:40-16:55 Bases biológicas para a identidade de gênero
Palestrante: LUCIANA MATTOS BARROS OLIVEIRA (BA)

16:55-17:10 Iniciando a terapia hormonal de afirmação de gênero em homens e mulheres trans
Palestrante: ELAINE MARIA FRADE COSTA (SP)

17:10-17:25 Cuidados com o indivíduo em transição
Palestrante: ERIK TROVAO DINIZ (PE)

17:25-17:40 Evidências em envelhecimento de pessoas trans: onde estamos?
Palestrante: KAREN FAGGIONI DE MARCA (RJ)

17:40-18:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA LEME (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
PARATIREOIDE EM FOCO: DIAGNÓSTICOS COMPLEXOS E NOVAS ABORDAGENS

08:30-10:00
Moderador: ANA AMÉLIA FIALHO DE OLIVEIRA HOFF (SP)

08:30-10:00
Moderador: MONIQUE NAKAYAMA OHE (SP) – Unifesp-EPM

08:30-08:40 Tema Livre: PTH Measurement as a Predictor of Post-Parathyroidectomy Hypocalcemia in Primary Hyperparathyroidism
Apresentador(a): ISABELLA GIMENES MORON (SP)

08:40-08:55 Tratamento clínico do Hiperparatireoidismo primário
Palestrante: FRANCISCO ALFREDO BANDEIRA E FARIAS (PE)

08:55-09:10 Quando a hiper/normocalcemia parece hiperparatireoidismo, será que é primário?
Palestrante: ANGELA CRISTINA GOMES BORGES LEAL (SE) – UFS

09:10-09:25 Hipercalcemia independente do PTH
Palestrante: REGINA MATSUNAGA MARTIN

09:25-09:40 Conduta cirúrgica é diferente no Hiperparatiroidismo de causa genética?
Palestrante: MARIA CRISTINA ARAUJO MAYA (RJ)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-12:40 PAINEL
SIMPÓSIO CIATEM – INTELIGÊNCIA ARTIFICIAL EM ENDOCRINOLOGIA E METABOLOGIA

10:30-12:40
Moderador: MARCIO KRAKAUER (SP) – Faculdade de Medicina do ABC

10:30-12:40
Moderador: MILENA GURGEL TELES BEZERRA (CE)

10:30-10:40 Tema Livre: Diagnosis of Acromegaly Through Voice Analysis: Cross-Validation of Four Machine Learning Models
Apresentador(a): FELLIPE SOUSA DA SILVA

10:40-10:50 Boas-vindas e contextualização
Palestrante: MARCIO KRAKAUER (SP) – Faculdade de Medicina do ABC

10:50-11:00 IA na educação médica
Palestrante: WALMIR FERREIRA COUTINHO (RJ) – PUC-Rio; IEDE

11:00-11:10 IA na rotina médica: produtividade e presença digital
Palestrante: MILENA GURGEL TELES BEZERRA (CE)

11:10-11:20 IA, mídias sociais, apps no consultório e teleatendimento
Palestrante: MATEUS DORNELLES SEVERO (RS) – HUSM/UFSM

11:20-11:30 Evidências científicas após 10 anos do InsulinAPP no controle glicêmico hospitalar
Palestrante: MARCOS TADASHI KAKITANI TOYOSHIMA (SP)

11:30-12:00 Perguntas e Respostas

12:00-12:40 Game interativo com a plateia (Mentimeter) – Quiz sobre IA na prática médica, com participação em tempo real e discussão com os membros da mesa

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 MESA-REDONDA
DESAFIOS NO MANEJO DA INSUFICIÊNCIA ADRENAL

13:30-15:00
Moderador: ALINE BARBOSA MORAES (RJ) – Universidade Federal Fluminense (UFF)

13:30-15:00
Moderador: MILENA COELHO FERNANDES CALDATO (PA)

13:30-13:40 Tema livre: Patient Education on Glucocorticoid Dose Adjustment in Stressful Situations: A Survey of Brazilian Patients with Adrenal Insufficiency
Apresentador(a): ALINE BARBOSA MORAES (RJ) – Universidade Federal Fluminense (UFF)

13:40-13:55 Interpretação do cortisol e afins no diagnóstico da insuficiência adrenal
Palestrante: ANDREA FARIA DUTRA FRAGOSO PEROZO (RJ)

13:55-14:10 Manejo da insuficiência adrenal induzida por glicocorticoides: desafios e conduta
Palestrante: GISELLE FERNANDES TABOADA (RJ)

14:10-14:25 Avaliação óssea no paciente com as diversas causas de insuficiência adrenal
Palestrante: LEONARDO COSTA BANDEIRA E FARIAS (PE) – Grupo Fleury/Unifesp

14:25-14:40 Desafios da avaliação da Insuficiência adrenal induzida por imunoterápicos
Palestrante: LEONARDO VIEIRA NETO (RJ) – UFRJ

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 PAINEL
PREMATURIDADE: COMO O INÍCIO DA VIDA MOLDA O SISTEMA ENDÓCRINO

15:30-16:30
Moderador: LATIFE SALOMÃO TYSZLER (RJ) – IEDE

15:30-16:30
Moderador: JOSÉ ANTONIO DINIZ FARIA JUNIOR (BA)

15:30-15:45 Prematuridade e crescimento: trajetórias e limites de recuperação
Palestrante: MARGARET CRISTINA DA SILVA BOGUSZEWSKI (PR) – UFPR

15:45-16:00 Prematuridade: repercussões na puberdade e na fertilidade
Palestrante: LUIZ CLAUDIO GONÇALVES DE CASTRO (DF) – UNB

16:00-16:15 Prematuridade e risco cardiometabólico: o que ainda estamos subestimando?
Palestrante: PAULO FERREZ COLLETT SOLBERG – UERJ

16:15-16:30 Perguntas e Respostas

16:30 – 18:00 PAINEL
DESCOMPASSO MODERNO: ENDOCRINOLOGIA SOB RISCO

16:30-18:00
Moderador: DIANA VIEGAS MARTINS (MG)

16:30-18:00
Moderador: ANA KARINA DE MELO BEZERRA SODRÉ (CE) – Universidade de Fortaleza

16:30-16:50 O papel das instituições como guardiãs da ética na medicina
Palestrante: NEUTON DORNELAS GOMES (DF)

16:50-17:10 Critérios da ANVISA para regulação dos agonistas de GLP-1 sintéticos: por que podemos confiar
Palestrante: DANIELA MARRECO CERQUEIRA (ANVISA) (DF)

17:10-17:30 Conselho Federal de Medicina: colocando limites para publicidade médica
Palestrante: BRUNO LEANDRO DE SOUZA (PB)

17:30-18:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA SÃO CONRADO (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
PRESENTE E FUTURO DO TRATAMENTO DA ACROMEGALIA

08:30-10:00
Moderador: MÔNICA GADELHA (RJ)

08:30-10:00
Moderador: JOSÉ ÍTALO SOARES MOTA (CE)

08:30-08:40 Tema Livre: A RETROELEMENT–IMMUNE AXIS DEFINES A DISTINCT IMMUNOBIOLOGICAL PHENOTYPE IN SOMATOTROPH TUMORS
Apresentador(a): RAFAEL LOCH BATISTA (SP)

08:40-08:55 O que diz o novo consenso de acromegalia sobre tratamento?
Palestrante: RAQUEL SOARES JALLAD (PB) – Hospital das Clínicas da Faculdade de Medicina da USP

08:55-09:10 Tratamento da acromegalia na vida real
Palestrante: LUCIANA ANSANELI NAVES (GO) – Universidade de Brasília

09:10-09:25 Rastreio de comorbidades
Palestrante: MONIKE LOURENÇO DIAS RODRIGUES (GO)

09:25-09:40 Perspectivas terapêuticas
Palestrante: LEANDRO KASUKI JOMORI DE PINHO (RJ) – IECPN

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:40 PAINEL
DOS GENES AOS ALGORITMOS: O FUTURO DO TRATAMENTO DA OBESIDADE

10:30-11:30
Moderador: CYNTHIA MELISSA VALERIO (RJ) – UFRJ

10:30-11:30
Moderador: KAREN FAGGIONI DE MARCA (RJ)

10:30-10:50 Genética/fenótipo ou inteligência artificial: qual é o futuro da seleção de pacientes na obesidade?
Palestrante: PAULA ISABEL MARQUES SIMOES DE FREITAS (PORTUGAL)

10:50-11:10 Da abordagem única à farmacoterapia baseada em fenótipos: onde estamos no manejo da obesidade?
Palestrante: LUCA BUSETTO (ITÁLIA)

11:10-11:30 Perguntas e Respostas

12:40-13:30 - INTERVALO
ALMOÇO

13:30-15:00 PAINEL
PASSANDO O BASTÃO: DESAFIOS E DECISÕES NA FASE DE TRANSIÇÃO

13:30-15:00
Moderador: ANA PAULA NEVES BORDALLO (RJ)

13:30-15:00
Moderador: MAGNUS REGIOS DIAS DA SILVA (SP) – EPM/Unifesp

13:30-13:40 Tema Livre: Impact of growth hormone deficiency and GH replacement in adult patients with childhood-onset GH deficiency: changes in body composition without concomitant changes in insulin sensitivity
Apresentador(a): KETTYUSCIA COELHO E OLIVEIRA (PA)

13:40-14:00 Deficiência de GH na transição: manter, reavaliar ou suspender?
Palestrante: KETTYUSCIA COELHO E OLIVEIRA (PA)

14:00-14:20 Síndrome de Klinefelter na adolescência: estamos diagnosticando e intervindo a tempo?
Palestrante: GIL GUERRA JUNIOR (SP) – FCM UNICAMP

14:20-14:40 Síndrome de Turner na adolescência: desafios e particularidades na continuidade do cuidado
Palestrante: NATHÁLIA LISBOA ROSA ALMEIDA GOMES (MG)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 – SIMPÓSIO PATROCINADO
SIMPÓSIO: NOVO NORDISK

15:30-16:00 O futuro é semanal: você está pronto para Awiqli (Icodec)?
Palestrante: ANDRÉ GUSTAVO DAHER VIANNA (PR) – Centro Diabetes Curitiba

16:00-16:30 O futuro é semanal: você está pronto para Awiqli (Icodec)?
Palestrante: WELLINGTON SANTANA DA SILVA JÚNIOR (MA) – UFMA

16:30-18:00 PAINEL
INDUÇÃO DE FERTILIDADE EM PACIENTES COM HIPOGONADISMO HIPOGONADOTRÓFICO

16:30-18:00
Moderador: BERENICE BILHARINHO DE MENDONÇA (MG)

16:30-18:00
Moderador: FERNANDA DE AZEVEDO CORRÊA (SUÍÇA)

16:30-16:40 Tema Livre: Once Daily Atumelnant (CRN04894) Enables Lowering of Glucocorticoid Doses With Sustained Androgen Reduction in Adults With Congenital Adrenal Hyperplasia
Apresentador(a): TANIA APARECIDA SANCHEZ BACHEGA (SP) – USP

16:40-17:00 Uso de clomifeno na infertilidade masculina
Palestrante: JÚLIO ZAKI ABUCHAM FILHO (SP) – Universidade Federal de São Paulo (SP) Unifesp

17:00-17:20 GnRH em bomba de infusão
Palestrante: NELLY PITTELOUD (SUÍÇA)

17:20-17:40 Técnicas de reprodução assistida
Palestrante: ROBERTO DE AZEVEDO ANTUNES (RJ)

17:40-18:00 Perguntas e Respostas

27/08/2026 – QUINTA-FEIRA

WINDSOR OCEÂNICO HOTEL: LOUNGE DA SBEM (ÁREA DE EXPOSIÇÃO – SUBSOLO)

15:00-15:30 SESSÃO
CAFÉ COM DIVERSIDADE

WINDSOR OCEÂNICO HOTEL: SALA ARPOADOR (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 OFICINA DE RARAS
DO GENE À CLÍNICA ... DESAFIOS DIAGNÓSTICOS NO CICLO DE VIDA - PARTE I

08:30-10:00
Moderador: NATHÁLIA LISBOA ROSA ALMEIDA GOMES (MG)

08:30-10:00
Moderador: FERNANDA DE AZEVEDO CORRÊA (SUÍÇA)

08:30-08:50 Como vive o indivíduo com DDS 46, XY na vida adulta?
Palestrante: BERENICE BILHARINHO DE MENDONÇA (MG)

08:50-09:10 Acondroplasia: a vigilância endócrina que vai além da estatura
Palestrante: VANESSA RADONSKY (SP)

09:10-09:30 McCune-Albright: O mosaico da suspeita e o desafio da hiperfunção múltipla
Palestrante: JULIENNE ÂNGELA RAMIRES DE CARVALHO (PR) – UFPR

09:30-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-12:40 OFICINA DE RARAS
DO GENE À CLÍNICA ... DESAFIOS DIAGNÓSTICOS NO CICLO DE VIDA – PARTE II

10:30-12:40
Moderador: BERENICE BILHARINHO DE MENDONÇA (MG)

10:30-12:40
Moderador: JOSÉ VIANA LIMA JÚNIOR (SP) – Unifesp/EPM

10:30-10:40 Tema Livre: Diagnostic Yield of Exome Sequencing in indeterminate 46,XY differences of Sex Development (DSD): Insights from 80 Patients
Apresentador(a): MARIA JIMENA CHAFLOQUE MESIA (SP)

10:40-11:00 Hipopituitarismo: da genética aos fenótipos extra-hipofisários
Palestrante: FERNANDA DE AZEVEDO CORRÊA (SUÍÇA)

11:00-11:20 Dislipidemias graves: fenótipos genéticos de alto risco
Palestrante: JOSIVAN GOMES DE LIMA (RN) – UFRN

11:20-11:40 Neoplasias endócrinas múltiplas do tipo 1: Implicações da genética no tratamento do paciente e rastreio nas famílias
Palestrante: DELMAR MUNIZ LOURENÇO JUNIOR (SP) – HCFMUSP

11:40-12:10 Perguntas e Respostas

12:10-12:40 Palavras finais: Onde estamos no cuidado com as doenças raras da endocrinologia?

12:40-13:30 – INTERVALO
ALMOÇO

15:00-15:30 – INTERVALO
INTERVALO

WINDSOR BARRA HOTEL: SALA BARRA DA TIJUCA (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
PERIMENOPAUSA: QUE FASE É ESSA?

08:30-10:00
Moderador: RUTH CLAPAUCH (RJ)

08:30-10:00
Moderador: GRAZIELLA NOGUEIRA MENDONÇA MONTEIRO DE BARROS (RJ)

08:30-08:40 Tema Livre: Development of an Online Extraction Liquid Chromatography Tandem Mass Spectrometry Method for Simultaneous Quantification of Seven C19 Steroids in Serum
Apresentador(a): HELENA PANTELIOU LIMA VALASSI (GRÉCIA)

08:40-08:55 Diagnóstico clínico e exames complementares: o que fazer nesta fase?
Palestrante: THYCIARA FONTENELE MARQUES (CE) – Faculdade de Medicina de Juazeiro do Norte

08:55-09:10 Doses e via de administração: como escolher o estrogênio?
Palestrante: LARISSA GARCIA GOMES (SP) – HCFMUSP

09:10-09:25 Endométrio em foco: avaliação e proteção durante a THM
Palestrante: RICARDO VASCONCELLOS BRUNO (RJ)

09:25-09:40 Perimenopausa e contracepção
Palestrante: RUTH CLAPAUCH (RJ)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 SESSÃO
SESSÃO CONJUNTA SBEM/EUROPEAN SOCIETY OF ENDOCRINOLOGY

10:30-11:30
Moderador: LUCIANA ANSANELI NAVES (GO) – Universidade de Brasília

10:30-11:30
Moderador: MÔNICA GADELHA (RJ)

10:30-11:10 Diretrizes clínicas para o manejo dos tumores agressivos de hipófise
Palestrante: ANA PAULA ABREU METZGER (ESTADOS UNIDOS DA AMÉRICA)

11:10-11:30 Discussão

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
ENDOCRINOLOGIA DO ADOLESCENTE: DILEMAS DO CONSULTÓRIO

13:30-15:00
Moderador: VANESSA RADONSKY (SP)

13:30-15:00
Moderador: FABIANO SANDRINI (SC)

13:30-13:40 Tema Livre: CORTISOL COSECRETION IN PRIMARY ALDOSTERONISM: A PHENOMENON INDEPENDENT OF ADRENAL NODULAR DISEASE?
Apresentador(a): ITHALLO RICARDO DANTAS ENNSER

13:40-14:00 Um adolescente com baixa estatura chega ao consultório: ainda se pode fazer algo
Palestrante: ALEXANDER AUGUSTO DE LIMA JORGE (SP) – FMUSP

14:00-14:20 Fraturas na adolescência: quando investigar fragilidade óssea?
Palestrante: CYNTHIA MARIA ALVARES BRANDÃO (SP)

14:20-14:40 O adolescente com Diabetes: quando pensar além dos tipos 1 e 2?
Palestrante: REGINA CELIA M. SANTIAGO MOISES (SP)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 – SIMPÓSIO PATROCINADO
SIMPÓSIO: NOVO NORDISK – SOGROYA: PRESCREVENDO O FUTURO EM DISTÚRBIOS DO HORMÔNIO DO CRESCIMENTO

15:30-16:30 Abertura institucional e Q&A
Moderador: MARIANA WERNECK (RJ)

15:30-16:30 Sogroya: Prescrevendo o Futuro em Distúrbios do Hormônio do Crescimento
Palestrante: MÔNICA GADELHA (RJ)

15:30-16:30 Sogroya: Prescrevendo o Futuro em Distúrbios do Hormônio do Crescimento
Palestrante: RENATA SZUNDY BERARDO (RJ) – HFSE

16:30-18:00 SIMPÓSIO
SIMPÓSIO SBEM – ALEG (ASOCIACIÓN LATINOAMERICANA DE ENDOCRINOLOGIA GINECOLÓGICA) – TEMAS ESPECIAIS SOBRE A MENOPAUSA

16:30-18:00
Moderador: POLI MARA SPRITZER (RS) – UFRGS

16:30-18:00
Moderador: KAREN FAGGIONI DE MARCA (RJ)

16:30-16:40 Tema Livre: Effect of exposure to BPA from childhood to postpuberty on uterine and ovarian function and morphology
Apresentador(a): SAMANTHA DOS SANTOS TOMÉ

16:40-17:00 Como abordar os distúrbios do sono
Palestrante: SUSANA PILNIK (ARGENTINA)

17:00-17:20 Terapia hormonal na paciente com histórico de endometriose
Palestrante: FERNANDO MARCOS DOS REIS (MG)

17:20-17:40 Como manejar as mudanças na composição corporal
Palestrante: DOLORES PEROVANO PARDINI (SP)

17:40-18:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA COPACABANA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
PERDENDO PESO, PRESERVANDO E MELHORANDO O MÚSCULO

08:30-10:00
Moderador: MATEUS DORNELLES SEVERO (RS) – HUSM/UFSM

08:30-10:00
Moderador: FELIPE HENNING GAIA DUARTE (SP)

08:30-08:40 Tema Livre: Incretin-Based Therapies Attenuate Muscle Function Decline in MASLD: A Real-World Longitudinal Cohort
Apresentador(a): FERNANDO DE QUADROS IORRA (RS)

08:40-08:55 Disfunção muscular associada à obesidade
Palestrante: CLAYTON LUIZ DORNELLES MACEDO (RS)

08:55-09:10 Impacto das novas medicações antiobesidade na composição corporal
Palestrante: CYNTHIA MELISSA VALERIO (RJ) – UFRJ

09:10-09:25 Abordagem diagnóstica da perda de massa/função muscular
Palestrante: FÁBIO FERREIRA DE MOURA (PE) – Universidade de Pernambuco

09:25-09:40 Manejo terapêutico: dieta, suplementos, exercícios.
Palestrante: ANA CARLA MONTENEGRO (PE)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:10 BATE-PAPO TEMÁTICO
DIABETES E OSTEOPOROSE

10:30-11:10 Painel
Palestrante: BÁRBARA CAMPOLINA CARVALHO SILVA (MG) – Santa Casa de Belo Horizonte

10:30-11:10 Painel
Palestrante: CATARINA BRASIL D´ALVA (CE) – Universidade Federal do Ceará UFC

10:30-11:10 Painel
Palestrante: FRANCISCO JOSÉ ALBUQUERQUE DE PAULA (CE) – FMRP-USP

11:10-12:40 MESA-REDONDA
DILEMAS DO DENOSUMABE NO CONSULTÓRIO. COMO MANTER O TRATAMENTO SOB CONTROLE

11:10-12:40
Moderador: NARRIANE CHAVES PEREIRA DE HOLANDA (PB)

11:10-12:40
Moderador: MANUELA GIULIANI MARCONDES ROCHA BRAZ (SP) – Universidade de São Paulo (USP)

11:10-11:20 Tema Livre: Parathyroidectomy reduces circulating endothelial cells and progenitor cells without improving vascular reactivity in classical primary hyperparathyroidism: a 12-month prospective study
Apresentador(a): THIAGO SIQUEIRA CHAGAS

11:20-11:35 Mecanismos de ação e de descontinuação do denosumabe
Palestrante: CATARINA BRASIL D´ALVA (CE) – Universidade Federal do Ceará UFC

11:35-11:50 Na criança e no adulto jovem: riscos da prescrição e descontinuação
Palestrante: GUIDO DE PAULA COLARES NETO (CE)

11:50-12:05 Pode ser para sempre? Evidências e decisões clínicas
Palestrante: DALISBOR MARCELO WEBER SILVA (SC)

12:05-12:20 Transição terapêutica: do e para o denosumabe
Palestrante: LUIZ HENRIQUE MACIEL GRIZ (PE) – Universidade de Pernambuco

12:20-12:40 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
DIAGNÓSTICO DIFERENCIAL NO DIABETES

13:30-15:00
Moderador: SAULO CAVALCANTI DA SILVA (MG)

13:30-15:00
Moderador: ROSANE KUPFER (RJ) – IEDE

13:30-13:40 Tema Livre: Excess body weight is frequent but shows different metabolic phenotypes in adults with GCK-MODY and type 1 diabetes: data from the Brazilian Monogenic Diabetes Study (BRASMOD) and Brazilian Type 1 Diabetes Study Group (BrazDiab1SG)
Apresentador(a): ANITA ANDRADE REIS (SP)

13:40-13:55 Peculiaridades do diabetes tipo 2 no adolescente
Palestrante: BALDUÍNO TSCHIEDEL (RS) – Instituto da Criança com Diabetes – Grupo Hospitalar Conceição – Porto Alegre-RS

13:55-14:10 Diabetes Monogênico: tem visto por aí?
Palestrante: FERNANDO DE MELLO ALMADA GIUFFRIDA (BA) – Universidade do Estado da Bahia (UNEB)

14:10-14:25 Peculiaridades do DM1 no idoso
Palestrante: RODRIGO NUNES LAMOUNIER (DF) – Rede Mater Dei Hub BH-MG

14:25-14:40 Diabetes Secundário ao câncer de pâncreas – busca de marcadores
Palestrante: MARIA LUCIA CARDILLO CORRÊA GIANNELLA (SP)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 RODA DE CONVERSA
MANEJO DO CONTROLE GLICÊMICO NO ATLETA COM DM1: O ANTES, O DURANTE E O DEPOIS – APRESENTAÇÃO DE CASOS CLÍNICOS

15:30-16:30 DM1 usuário de insulina – DM1 usuário de bomba de insulina
Palestrante: MAURO SCHARF PINTO (PR)

15:30-16:30 DM1 usuário de insulina – DM1 usuário de bomba de insulina
Palestrante: ANA CLÁUDIA REBOUÇAS RAMALHO (BA)

15:30-16:30 DM1 usuário de insulina – DM1 usuário de bomba de insulina
Palestrante: ROBERTO LUIS ZAGURY (RJ)

16:30-18:00 PAINEL
DIABETES E GRAVIDEZ

16:30-18:00
Moderador: RUY LYRA DA SILVA FILHO (PE) – UFPE

16:30-18:00
Moderador: BIANCA DE ALMEIDA PITITTO (RJ)

16:30-16:40 Tema Livre: Delayed Recovery After Inpatient Hypoglycemia: Retesting Gaps and Recurrent Risk Across 307,634 Point-of-Care Glucose Records
Apresentador(a): ALINA COUTINHO RODRIGUES FEITOSA (BA)

16:40-16:55 Planejamento da gestação em mulheres em uso de Análogo de GLP-1
Palestrante: CRISTINA FIGUEIREDO SAMPAIO FAÇANHA (CE) – Universidade de Fortaleza-Unifor

16:55-17:10 Análise Crítica do uso de CGM no Diabetes Melito Gestacional
Palestrante: LENITA ZAJDENVERG (RJ) – Universidade Federal do Rio de Janeiro UFRJ

17:10-17:25 Manejo do DM1 da concepção ao parto
Palestrante: PATRÍCIA MEDICI DUALIB (SP)

17:25-17:40 A gestante com complicações crônicas: retinopatia, doença renal e cardiovascular
Palestrante: CARLOS ANTONIO NEGRATO (SP) – Faculdade de Medicina de Bauru- USP

17:40-18:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA IPANEMA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
AVALIAÇÃO E CONDUTA EM NÓDULOS DE TIREOIDE

08:30-10:00
Moderador: ANA CAROLINA NADER VASCONCELOS MESSIAS (RJ)

08:30-10:00
Moderador: ROSSANA CORBO RAMALHO DE MELLO (RJ)

08:30-08:40 Tema Livre: Disrupted Membrane Trafficking of the Sodium/Iodide Symporter as a Potential Therapeutic Target in Thyroid Cancer
Apresentador(a): VINICIUS GONÇALVES RODRIGUES

08:40-08:55 A nova classificação ecográfica da ATA: prós e contras
Palestrante: RAQUEL ANDRADE DE SIQUEIRA (GO)

08:55-09:10 Como marcadores moleculares podem auxiliar na conduta?
Palestrante: JANETE MARIA CERUTTI (SP) – Unifesp

09:10-09:25 Citologia indeterminada: decisões práticas na vida real
Palestrante: JOSÉ MIGUEL SILVA DORA (RS) – HCPA/UFRGS

09:25-09:40 Radioablação: em quem indicar?
Palestrante: LEONARDO GUIMARÃES RANGEL (RJ)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 PRÊMIO
PRÊMIO JOSÉ DANTAS DE SOUZA

10:30-10:45 Apresentação (do significado do prêmio e anúncio da laureada)
Palestrante: MARISE LAZARETTI CASTRO (SP)

10:45-11:00 Apresentação: (da biografia da laureada)
Palestrante: MADSON QUEIROZ DE ALMEIDA (SP)

11:00-11:30 Conferência: Arquitetura genética e epigenética dos distúrbios da puberdade
Palestrante: ANA CLAUDIA LATRONICO XAVIER (SP) – FMUSP

11:40-12:40 DEBATE
A MUSCULAÇÃO PODE SER INDICADA A CRIANÇAS E ADOLESCENTES?

11:40-12:40
Moderador: NATHÁLIA LISBOA ROSA ALMEIDA GOMES (MG)

11:40-12:10 A visão do endocrinologista
Palestrante: FABIANO SANDRINI (SC)

12:10-12:40 A visão do educador físico
Palestrante: HUGO TOURINHO FILHO (SP)

12:40-13:30 – INTERVALO
ALMOÇO

12:40-13:30

13:30-15:00 PAINEL
DA SOP À SOMP: APENAS UMA MUDANÇA DE NOME OU UMA MUDANÇA DE PARADIGMA?

13:30-15:00
Moderador: MÔNICA DE OLIVEIRA (PE) – IMIP/PE

13:30-15:00
Moderador: ANDRESSA MARTINS DE OLIVEIRA (RJ)

13:30-13:40 Tema Livre: Combined Interventions In The Mental Health Of Women With Polycystic Ovary Syndrome: A Systematic Review and Meta-Analysis
Apresentador(a): LARA ALVES TAVARES

13:40-14:00 Síndrome ovariana metabólica poliendócrina (SOMP): como a troca de nome pode influenciar no diagnóstico, na pesquisa e na vida das pacientes
Palestrante: POLI MARA SPRITZER (RS) – UFRGS

14:00-14:20 O Fenótipo Cerebral na SOMP: Andrógenos, Anovulação e Gênero
Palestrante: ADRIANA LAGES (PORTUGAL)

14:20-14:40 Risco cardiometabólico em familiares
Palestrante: LUCAS BANDEIRA MARCHESAN (RS) – Hospital Nossa Senhora da Conceição

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 PAINEL
DISCUSSÃO – PRINCIPAIS RECOMENDAÇÕES DO NOVO CONSENSO DE PUBERDADE CENTRAL

15:30-16:30
Moderador: ANA CLAUDIA LATRONICO XAVIER (SP) – FMUSP

15:30-16:30
Moderador: JACQUELINE ROSÂNGELA DE ARAÚJO (PE) – HC-UFPE

15:30-16:00
Palestrante: ANA PINHEIRO MACHADO CANTON (SP)

15:30-16:00
Palestrante: VINÍCIUS NAHIME DE BRITO (SP) – FMUSP

15:30-16:00
Palestrante: SONIR ROBERTO RAUBER ANTONINI (SP) – FMRP-USP

16:30-18:00 PAINEL
NOVIDADES DO TRATAMENTO FARMACOLÓGICO ORAL PARA OBESIDADE

16:30-18:00
Moderador: CAROLINA GARCIA SOARES LEÃES RECH (RS)

16:30-18:00
Moderador: SIMONE VAN DE SANDE LEE (SC)

16:30-16:40 Tema Livre: Efficacy and Safety of Sibutramine–Topiramate Combination versus Sibutramine Monotherapy for Weight Loss in Adults with Obesity: A 48-Week Randomized, Double-Blind, Placebo-Controlled Trial
Apresentador(a): HELOISA HARUMI YAMAMOTO (SP)

16:40-16:55 Associação Sibutramina e Topiramato
Palestrante: CINTIA CERCATO (SP) – Faculdade de Medicina da Universidade de São Paulo USP

16:55-17:10 Naltrexona e Bupropiona
Palestrante: THAISA DOURADO GUEDES TRUJILHO (BA)

17:10-17:25 GLP-1 Orais
Palestrante: BRUNO HALPERN (SP)

17:25-17:40 Simpaticomiméticos
Palestrante: FÁBIO ROGÉRIO TRUJILHO (BA) – CEDEBA-BA

17:40-18:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA LEBLON (WINDSOR BARRA HOTEL)

08:30-10:00 MESA-REDONDA
UPDATE NA INVESTIGAÇÃO LABORATORIAL E DE IMAGEM DAS DOENÇAS ADRENAIS

08:30-10:00
Moderador: MARGARET DE CASTRO (SP) – FMRP-USP

08:30-10:00
Moderador: CLAUDIO ELIAS KATER (SP) – Unifesp

08:30-08:40 Tema Livre: Clinicopathological and Molecular Profile of Brazilian Patients with Cushing Syndrome Due to Primary Pigmented Micronodular Adrenal Hyperplasia
Apresentador(a): ISABELA NARDO

08:40-08:55 Armadilhas na interpretação da aldosterona e renina no diagnóstico do HAP
Palestrante: THAIS CASTANHEIRA DE FREITAS RESENDE (GO)

08:55-09:10 Como manejar o HAP sem dispor de cateterismo de veias adrenais
Palestrante: ALINE BARBOSA MORAES (RJ) – Universidade Federal Fluminense (UFF)

09:10-09:25 Haja Adrenalina! Pitfalls na investigação laboratorial do Feocromocitoma/paraganglioma
Palestrante: JOSÉ VIANA LIMA JÚNIOR (SP) - Unifesp/EPM

09:25-09:40 Papel da medicina nuclear nas hiperfunções adrenais
Palestrante: FRANCISCO DE ASSIS ROMEIRO (RJ)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 DEBATE
TERAPIA ADJUVANTE NO DM1

10:30-11:30
Moderador: MONICA ANDRADE LIMA GABBAY (SP) – Unifesp

10:30-11:00 Prós
Palestrante: RODRIGO NUNES LAMOUNIER (DF) – Rede Mater Dei Hub BH-MG

11:00-11:30 Contras
Palestrante: MAURO SCHARF PINTO (PR)

11:40-12:40 – SIMPÓSIO PATROCINADO
SIMPÓSIO: NOVO NORDISK

11:40-12:40 Two routes, one goal: O algoritmo metabólico para perda de peso e benefício cardiometabólico.
Palestrante: CRISTIANO ROBERTO GRIMALDI BARCELLOS (SP)

11:40-12:40 Two routes, one goal: O algoritmo metabólico para perda de peso e benefício cardiometabólico.
Palestrante: BRUNO HALPERN (SP)

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
DIRETRIZES CLÍNICAS BASEADAS EM EVIDÊNCIAS DO TRATAMENTO DA DOENÇA DE CUSHING NO BRASIL

13:30-15:00
Moderador: MAURO ANTÔNIO CZEPIELEWSKI (RS) – UFRGS

13:30-15:00
Moderador: ANNE ELISE ALEXANDRE (SP)

13:30-13:40 Tema Livre: ACTH as a Cost-Effective Marker of Dexamethasone Ingestion in the Dexamethasone Suppression Test: Preliminary Evidence Challenging Current Practice with Implications for Resource-Limited Settings
Apresentador(a): MATEUS ANDRADE BOMFIM MACHADO

13:40-13:55 Qual papel do tratamento medicamentoso?
Palestrante: VANIA DOS SANTOS NUNES NOGUEIRA (SP)

13:55-14:10 Cushing grave: como manejar
Palestrante: MÁRCIO CARLOS MACHADO (SP)

14:10-14:25 Profilaxia para trombose: sempre?
Palestrante: MARGARET DE CASTRO (SP) – FMRP-USP

14:25-14:40 Revisitando critérios de controle e recorrência do hipercortisolismo
Palestrante: GUILHERME ALCIDES FLÔRES SOARES ROLLIN (RS)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 – SIMPÓSIO PATROCINADO
SIMPÓSIO: ASTRAZENECA

15:30-16:30 A Osteoporose do Meu Paciente Não Melhora: O Que Pode Estar Passando Despercebido?
Palestrante: MARIA LUIZA BRANDI (ITÁLIA)

15:30-16:30 A Osteoporose do Meu Paciente Não Melhora: O Que Pode Estar Passando Despercebido?
Palestrante: SERGIO SETSUO MAEDA (SP) – Unifesp

16:30-18:00 PAINEL
AMBIENTE, EXPOSIÇÕES E TIREOIDE: A NOVA FRONTEIRA DAS DOENÇAS ENDÓCRINAS

16:30-18:00
Moderador: LAURA STERIAN WARD (SP)

16:30-18:00
Moderador: ILEANA GABRIELA SANCHEZ DE RUBIO (SP) – Unifesp

16:30-16:40 Tema Livre: Irgarol as a potential thyroid endocrine disruptor: evidence from human tumoral and non-tumoral cell lines
Apresentador(a): REINALDO ROPKE

16:40-16:55 O risco invisível: contaminantes ambientais e disfunções da tireoide
Palestrante: CAROLINE SERRANO DO NASCIMENTO (SP)

16:55-17:10 Quando a nutrição encontra a tireoide: o papel dos micronutrientes
Palestrante: ROSALIA DO PRADO PADOVANI (SP) – Santa Casa de São Paulo

17:10-17:25 Fármacos e Tireoide: interfaces entre terapêutica, fisiologia e efeitos adversos
Palestrante: FERNANDA VAISMAN (RJ) – INCA

17:25-17:40 Obesidade e câncer de tireoide: evidências, mecanismos e controvérsias
Palestrante: IURI MARTIN GOEMANN (RS) – UFRGS

17:40-18:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA LEME (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
DISLIPIDEMIAS EM SITUAÇÕES ESPECIAIS

08:30-10:00
Moderador: LÚCIA HELENA OLIVEIRA CORDEIRO (PE) – Universidade Federal do Rio Grande do Norte

08:30-10:00
Moderador: RICARDO MENDES MARTINS (RJ)

08:30-08:40 Tema Livre: Ixekizumab With Tirzepatide in Psoriasis or Psoriatic Arthritis: A Pooled Analysis of TOGETHER-PsA and TOGETHER-PsO
Apresentador(a): LARA GUEDES SILVA

08:40-08:55 Mulheres: gravidez, contracepção e menopausa
Palestrante: JOANA RODRIGUES DANTAS VEZZANI (RJ) – Hospital Universitário Clementino Fraga Filho –UFRJ

08:55-09:10 A pessoa com HIV
Palestrante: LEONARDO FONTENELE EKSTERMAN (RJ)

09:10-09:25 Idosos 80 +
Palestrante: JOAQUIM CUSTÓDIO DA SILVA JUNIOR (SP) – UFBA

09:25-09:40 Doença renal grave
Palestrante: HUMBERTO BATISTA FERREIRA (MG)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:40-12:00 HOT TOPICS
ENDOCRINOLOGIA DO EXERCÍCIO

10:40-12:00
Moderador: ROGÉRIO FRIEDMAN (RS)

10:40-12:00
Moderador: ANDREA MESSIAS BRITTO FIORETTI (SP) – Unifesp

10:40-10:55 Biomecânica Do Movimento
Palestrante: LEONARDO METSAVAHT (RJ)

10:55-11:10 Impacto Da Musculação No Crescimento De Crianças E Adolescentes
Palestrante: JOSÉ ANTONIO DINIZ FARIA JUNIOR (BA)

11:10-11:25 IA e a Endocrinologia Do Exercício
Palestrante: MARCOS MORAES DE OLIVEIRA

11:25-11:40 Novos Conceitos Do Treinamento Resistido
Palestrante: ADRIANA COGNOLATO (SP)

11:40-12:00 Perguntas e Respostas

12:10-12:40 CONFERÊNCIA
NEM TODO SUOR VALE A PENA: REPENSANDO O EQUILÍBRIO ENTRE PERFORMANCE E EXAUSTÃO NO EXERCÍCIO

12:10-12:40
Moderador: ROGÉRIO FRIEDMAN (RS)

12:10-12:30
Palestrante: ANDREA MESSIAS BRITTO FIORETTI (SP) – Unifesp

12:30-12:40 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
DISFUNÇÕES TIREOIDIANAS NAS DIFERENTES FASES DA VIDA DA MULHER

13:30-15:00
Moderador: CLEO OTAVIANO MESA JÚNIOR (PR) – PUC PR

13:30-15:00
Moderador: ROSALIA DO PRADO PADOVANI (SP) – Santa Casa de São Paulo

13:30-13:40 Tema Livre: Prepubertal exposure to a glyphosate-based herbicide alters the thyroid proteomic profile
Apresentador(a): REGINA MAURINA BRANCALIONE

13:40-13:55 Novas perspectivas de avaliação de hipo congênito
Palestrante: ILEANA GABRIELA SANCHEZ DE RUBIO (SP) – Unifesp

13:55-14:10 Hipo subclínico e fertilidade
Palestrante: SUEMI MARUI (SP)

14:10-14:25 Hipotireodismo subclínico e doença cardiovascular
Palestrante: JOSÉ AUGUSTO SGARBI (SP) – FAC. MEDICINA DE MARILIA

14:25-14:40 Disfunção tireoidiana no envelhecimento
Palestrante: GISAH MUSIALOWSKI DO AMARAL (PR) – UFPR

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 CONFERÊNCIA
O ANO EM DISLIPIDEMIA E ATEROSCLEROSE

15:30-16:00
Moderador: MARIA HELANE COSTA GURGEL (CE) – Universidade Federal do Ceará UFC

15:30-16:00
Palestrante: ANDREI CARVALHO SPOSITO (SP)

15:30-16:30
Palestrante: MARCELLO CASACCIA BERTOLUCI (RS) – UFRGS

16:30-18:00 PAINEL
DISLIPIDEMIAS GENÉTICAS E DIAGNÓSTICO DE PRECISÃO

16:30-18:00
Moderador: MARIA HELANE COSTA GURGEL (CE) – Universidade Federal do Ceará UFC

16:30-18:00
Moderador: JOANA RODRIGUES DANTAS VEZZANI (RJ) – Hospital Universitário Clementino Fraga Filho – UFRJ

16:30-16:40 Tema Livre: Latent Autoimmune Diabetes in Adults (LADA): Clinical and Phenotypic Characterization in Patients from a Tertiary Hospital in São Paulo
Apresentador(a): GIOVANNA LEME VENDRAMINI

16:40-16:55 Hipercolesterolemia Familiar: do Fenótipo ao Painel Genético
Palestrante: LÚCIA HELENA OLIVEIRA CORDEIRO (PE) – Universidade Federal do Rio Grande do Norte

16:55-17:10 Desafios diagnósticos na dislipidemia combinada
Palestrante: FLÁVIA COIMBRA PONTES MAIA (MG)

17:10-17:25 Diagnóstico Diferencial da Hipertrigliceridemia grave
Palestrante: VIRGINIA OLIVEIRA FERNANDES CORTEZ (CE)

17:25-17:40 Fenótipos Clínicos e Diagnóstico Diferencial das Lipodistrofias
Palestrante: RENAN MAGALHÃES MONTENEGRO JUNIOR (CE) – Faculdade de Medicina da UFC

17:40-18:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA SÃO CONRADO (WINDSOR BARRA HOTEL)

08:30-10:00 MESA-REDONDA
ANOREXIA, BULIMIA E VIGOREXIA NA CRIANÇA E NO ADOLESCENTE

08:30-10:00
Moderador: MARÍLIA MARTINS GUIMARÃES (RJ) – UFRJ

08:30-10:00
Moderador: LUIZ CLAUDIO GONÇALVES DE CASTRO (DF) – UNB

08:30-08:40 Tema Livre: Worse sleep patterns associated with binge eating in patients living with obesity: an overlooked combination that deserves more attention
Apresentador(a): LARISSA DE CASTRO CARIOCA FEITOZA (AM)

08:40-09:00 O cérebro nos transtornos alimentares e de imagem corporal: neurobiologia e psicopatologia
Palestrante: WAGNER DE SOUSA GURGEL (SP)

09:00-09:20 Impacto no crescimento, puberdade e ciclos menstruais
Palestrante: RENATA SZUNDY BERARDO (RJ) – HFSE

09:20-09:40 Entre restrição e excesso: transtornos alimentares, vigorexia e saúde óssea
Palestrante: GUIDO DE PAULA COLARES NETO (CE)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:00 CONFERÊNCIA
MUDANÇAS DE PARADIGMA NO TRATAMENTO DA OBESIDADE: ANÁLISE CRÍTICA DAS DIRETRIZES RECENTES E DOS AVANÇOS TERAPÊUTICOS

10:30-11:00
Moderador: FÁBIO ROGÉRIO TRUJILHO (BA) – CEDEBA-BA

10:30-11:00
Palestrante: LUCA BUSETTO (ITÁLIA)

11:00-11:30 CONFERÊNCIA
ALIMENTOS ULTRAPROCESSADOS E SAÚDE HUMANA: A TESE E AS EVIDÊNCIAS

11:00-11:30
Moderador: MARIA EDNA DE MELO (SP) – Hospital das Clínicas da Faculdade de Medicina da USP

11:00-11:30
Palestrante: MARIA LAURA DA COSTA LOUZADA (SP)

11:40-12:40 – SIMPÓSIO PATROCINADO
SIMPÓSIO: LILLY

11:40-12:40 O peso é só o começo: Mounjaro transformando a saúde metabólica
Palestrante: LUCA BUSETTO (ITÁLIA)

11:40-12:40 O peso é só o começo: Mounjaro transformando a saúde metabólica
Palestrante: JOÃO EDUARDO NUNES SALLES (SP) – Faculdade de Ciências Médicas da Santa Casa de São Paulo

11:40-12:40 O peso é só o começo: Mounjaro transformando a saúde metabólica
Palestrante: SIMONE VAN DE SANDE LEE (SC)

12:40-13:30 – INTERVALO
ALMOÇO

12:40-13:30

13:30-15:00 PAINEL
OS DESAFIOS DA TERAPIA OSTEOANABÓLICA

13:30-15:00
Moderador: BÁRBARA CAMPOLINA CARVALHO SILVA (MG) – Santa Casa de Belo Horizonte

13:30-15:00
Moderador: LUIZ HENRIQUE MACIEL GRIZ (PE) – Universidade de Pernambuco

13:30-13:40 Tema Livre: As alterações na densidade e na microarquitetura ósseas estão associadas ao controle glicêmico em homens jovens com diabetes mellitus tipo 1
Apresentador(a): FILIPE BARBOSA LINHARES

13:40-13:55 Quem é o paciente bom respondedor para terapia anabólica?
Palestrante: DALISBOR MARCELO WEBER SILVA (SC)

13:55-14:10 Estratégia para transição para romosozumabe: como prever os resultados?
Palestrante: LEONARDO COSTA BANDEIRA E FARIAS (PE) – Grupo Fleury/Unifesp

14:10-14:25 Teriparatida: quando usar e o que esperar na prática?
Palestrante: JOÃO LINDOLFO CUNHA BORGES (DF) – SBEMDF

14:25-14:40 Quando a terapia anabólica estaria contraindicada?
Palestrante: VICTÓRIA ZEGHBI COCHENSKI BORBA (PR) – UFPR

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 – SIMPÓSIO PATROCINADO
SIMPÓSIO: ABBOTT

15:30-16:30 Precisão e qualidade que importam: o papel do único iCGM disponível no Brasil no cuidado do DM2
Moderador: JELLIN CHIAOTING CHUANG ZAMBON

15:30-16:30 Precisão e qualidade que importam: o papel do único iCGM disponível no Brasil no cuidado do DM2
Palestrante: JOÃO EDUARDO NUNES SALLES (SP) – Faculdade de Ciências Médicas da Santa Casa de São Paulo

15:30-16:30 Precisão e qualidade que importam: o papel do único iCGM disponível no Brasil no cuidado do DM2
Palestrante: MAURO SCHARF PINTO (PR)

16:30-17:00 CONFERÊNCIA
NEW HORIZONS: GNRH REPLACEMENT AND NEUROCOGNITION

16:30-17:00
Moderador: ANA CLAUDIA LATRONICO XAVIER (SP) – FMUSP

16:30-17:00
Palestrante: NELLY PITTELOUD (SUÍÇA)

17:00-18:00 DEBATE
EM TUMORES DE HIPÓFISE CLINICAMENTE NÃO FUNCIONANTES, O USO DA CABERGOLINA

17:00-18:00
Moderador: MANOEL RICARDO ALVES MARTINS (PE) – Universidade Federal do Ceará UFC

17:00-17:30
Palestrante: RAFAEL LOCH BATISTA (SP)

17:30-18:00
Palestrante: PAULO AUGUSTO CARVALHO MIRANDA (MG)

28/08/2026 – SEXTA-FEIRA

WINDSOR OCEÂNICO HOTEL: LOUNGE DA SBEM (ÁREA DE EXPOSIÇÃO – SUBSOLO)

15:00-15:30 SESSÃO
CAFÉ COM DIVERSIDADE

WINDSOR OCEÂNICO HOTEL: SALA ARPOADOR (WINDSOR OCEÂNICO HOTEL)

08:30-09:45 MESA-REDONDA
MEDICINA DE PRECISÃO NAS DOENÇAS ADRENAIS. COMO OS TESTES MOLECULARES PODEM AUXILIAR NA PRÁTICA CLÍNICA?

08:30-09:45
Moderador: LEONARDO VIEIRA NETO (RJ) – UFRJ

08:30-09:45
Moderador: FLAVIA AMANDA COSTA BARBOSA (SP) – EPM/Unifesp

08:30-08:45 Hiperplasia adrenal congênita
Palestrante: TANIA APARECIDA SANCHEZ BACHEGA (SP) – USP

08:45-09:00 Feocromocitoma/Paraganglioma
Palestrante: JOSÉ VIANA LIMA JÚNIOR (SP) – Unifesp/EPM

09:00-09:15 Carcinoma de córtex adrenal
Palestrante: MARIA CANDIDA BARISSON VILLARES FRAGOSO (SP)

09:15-09:30 Hiperaldosteronismo primário
Palestrante: MADSON QUEIROZ DE ALMEIDA (SP)

09:30-09:45 Perguntas e Respostas

09:45-10:00 PRÊMIO
PRÊMIO ARCHIVES OF ENDOCRINOLOGY AND METABOLISM (AE&M) TALES MARTINS

09:45-09:48 Entrega do prêmio
Palestrante: CESAR LUIZ BOGUSZEWSKI (PR) – UFPR

09:48-09:55 Molecular investigation of primary aldosteronism: exploring genetic heterogeneity in understudied populations
Palestrante: LEONARDO KENZO MAEDA (SP)

09:55-10:00 Perguntas e Respostas
10:00-10:30 – INTERVALO
INTERVALO

10:30-12:30 OFICINA
DESAFIOS NA AVALIAÇÃO LABORATORIAL HORMONAL: O QUE O ENDOCRINOLOGISTA PRECISA SABER

10:30-12:30
Moderador: MARIA IZABEL CHIAMOLERA (SP)

10:30-12:30
Moderador: MARIA TEREZA NUNES (SP)

10:30-10:50 Para além da glicemia: os desafios de interpretar peptídeo C, insulina e hemoglobina glicada
Palestrante: MILENA GURGEL TELES BEZERRA (CE)

10:50-11:10 Novas aplicações da tiroglobulina no seguimento do CDT de acordo com ATA 2025
Palestrante: ROSA PAULA MELLO BISCOLLA (SP)

11:10-11:30 Princípios e aplicações da espectrometria de massa nas dosagens hormonais
Palestrante: JOSÉ GILBERTO HENRIQUES VIEIRA (SP)

11:30-11:50 Testes Dinâmicos em Endocrinologia – Quando e por quê?
Palestrante: LUCIANI RENATA SILVEIRA DE CARVALHO (SP) – HCFMUSP

11:50-12:10 Pitfalls na avaliação hormonal laboratorial
Palestrante: LUCIANA ANSANELI NAVES (GO) – Universidade de Brasília

12:10-12:30 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

15:00-15:30 – INTERVALO
INTERVALO

16:30-18:00 PAINEL
ONCO-ENDOCRINOLOGIA TRANSLACIONAL: DA BIOLOGIA TUMORAL À MEDICINA DE PRECISÃO

16:30-18:00
Moderador: CAROLINA FERRAZ DA SILVA (PE) – FCMSCSP

16:30-18:00
Moderador: LUCAS LEITE CUNHA (SP)

16:30-16:40 Tema Livre: Genetic investigation of TERT promoter and ATRX variants in pheochromocytomas and paragangliomas
Apresentador(a): LUCAS BATINI ARAUJO

16:40-16:55 Marcadores Moleculares dos Tumores Hipofisários Agressivos
Palestrante: ERICKA BARBOSA TRARBACH

16:55-17:10 Imunidade e hormônios: o microambiente tumoral nas neoplasias endócrinas
Palestrante: LAURA STERIAN WARD (SP)

17:10-17:25 Biópsia Líquida e Oncologia de Precisão
Palestrante: CLEBER PINTO CAMACHO (SP) – Universidade Nove de Julho (UNINOVE)

17:25-17:40 O papel dos microRNAs e vesículas extracelulares na tumorigênese endócrina
Palestrante: CESAR SEIGI FUZIWARA (SP)

17:40-18:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA BARRA DA TIJUCA (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
DO CRESCIMENTO À MATURIDADE: DOENÇAS ÓSSEAS RARAS NO MUNDO REAL

08:30-10:00
Moderador: BÁRBARA CAMPOLINA CARVALHO SILVA (MG) – Santa Casa de Belo Horizonte

08:30-10:00
Moderador: VICTÓRIA ZEGHBI COCHENSKI BORBA (PR) – UFPR

08:30-08:40 Tema Livre: Estimated prevalence and epidemiological mapping of hypophosphatasia in the State of São Paulo: DATASUS data mining and development of the Hypophosphatasia Risk Score (HPP-RS)
Apresentador(a): IGOR CANDIDO VIANA GONÇALVES

08:40-09:00 Raquitismo hipofosfatêmico
Palestrante: ÉRICO HIGINO CARVALHO (PI)

09:00-09:20 Hipofosfatasia
Palestrante: MARIA LUIZA BRANDI (ITÁLIA)

09:20-09:40 Osteomalácia Oncogênica
Palestrante: MAYRA MACENA GOMES (SP)

09:40-10:00 Discussão

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:00 CONFERÊNCIA
HYPOPARATHYROIDISM: FROM CONVENTIONAL THERAPY TO NOVEL APPROACHES

10:30-11:00
Moderador: MARISE LAZARETTI CASTRO (SP)

10:30-11:00
Palestrante: MARIA LUIZA BRANDI (ITÁLIA)

11:00-11:30 CONFERÊNCIA
MANEJO DA SAÚDE ÓSSEA DURANTE OS TRATAMENTOS INTENSIVOS PARA PERDA DE PESO

11:00-11:30
Moderador: PAULO GUSTAVO SAMPAIO LACATIVA (RJ)

11:00-11:30
Palestrante: MARIA LUCIA FLEIUSS DE FARIAS (RJ)

11:40-12:40 – SIMPÓSIO PATROCINADO
SIMPÓSIO: LIBBS

11:40-12:40 Além do fogacho: Estamos subutilizando o potencial metabólico da TH?
Palestrante: CRISTIANO ROBERTO GRIMALDI BARCELLOS (SP)

11:40-12:40 Além do fogacho: Estamos subutilizando o potencial metabólico da TH?
Palestrante: MARCELO LUIS STEINER

11:40-12:40 Além do fogacho: Estamos subutilizando o potencial metabólico da TH?
Palestrante: THALITA RUSSO DOMENICH

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
DOS ANTICORPOS À ORQUESTRA DIGITAL: AS NOVAS FRONTEIRAS DO DIABETES TIPO 1 AO LONGO DA VIDA

13:30-15:00
Moderador: FERNANDO DE MELLO ALMADA GIUFFRIDA (BA) – Universidade do Estado da Bahia (UNEB)

13:30-15:00
Moderador: PATRÍCIA MEDICI DUALIB (SP)

13:30-13:40 Tema Livre: Glycemic Metrics and Albuminuria in Adults With Type 1 Diabetes Initiating Automated Insulin Delivery: A Subanalysis of a Prospective Observational Study
Apresentador(a): ANA RITA LEITE (PORTUGAL)

13:40-13:55 Rastreamento e detecção precoce: onde estamos e para onde vamos?
Palestrante: MELANIE RODACKI (RJ) – UFRJ

13:55-14:10 Adiar o Inadiável: imunoterapia e esperança no diabetes tipo 1
Palestrante: EMERSON CESTARI MARINO (SP)

14:10-14:25 Entre sensores e sonhos: a revolução das insulinas e dos sistemas automatizados no DM1
Palestrante: TALITA LETÍCIA TREVISAN (SC)

14:25-14:40 Além da glicemia: monitorização e prevenção de complicações crônicas em crianças e adolescentes
Palestrante: MAURO SCHARF PINTO (PR)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 – ENCONTRO COM O PROFESSOR
LIPODISTROFIAS E HIPERTRIGLICERIDEMIAS GRAVES

15:30-16:30
Palestrante: JOSIVAN GOMES DE LIMA (RN) – UFRN

15:30-16:30
Palestrante: HUMBERTO BATISTA FERREIRA (MG)

15:30–16:30
Palestrante: NATALIA ROSSIN GUIDORIZZI (SP)

16:30-18:00 PAINEL
O QUE HÁ DE NOVO NA ENDOCRINOLOGIA DO EXERCÍCIO?

16:30-16:30
Moderador: ALEXIS DOURADO GUEDES (BA) – Centro de Diabetes e Endocrinologia do Estado da Bahia

16:30-16:30
Moderador: ANDREA MESSIAS BRITTO FIORETTI (SP) – Unifesp

16:30-16:40 Tema Livre: Muscle Beats Liver: Quality of Life in MASLD Is a Matter of Strength, Not Fibrosis
Apresentador(a): FERNANDO DE QUADROS IORRA (RS)

16:40-16:55 Perilipinas e incretinas
Palestrante: CLAYTON LUIZ DORNELLES MACEDO (RS)

16:55-17:10 Miocinas
Palestrante: MARIA TEREZA NUNES (SP)

17:10-17:25 Autorização para uso terapêutico
Palestrante: ROGÉRIO FRIEDMAN (RS)

17:25-17:40 Impacto da terapia de reposição hormonal na performance de pacientes hipogonádicos
Palestrante: JOÃO CARLOS RAMOS DIAS (SP)

17:40-18:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA COPACABANA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
DOENÇA CARDIOVASCULAR NO DIABETES

08:30-10:00
Moderador: REINE MARIE CHAVES FONSECA (BA)

08:30-10:00
Moderador: SÉRGIO ATALA DIB (SP) – UNIVERSIDADE FEDERAL DE SÃO PAULO – UNIFESP

08:30-08:40 Tema Livre: Circulating microRNA 146-a and Cardiovascular Risk In Type 1 Diabetes: A Systematic Review
Apresentador(a): MARIA ANTÔNIA CARDOSO BORJA DE BRITO

08:40-08:55 Diabetes e DCV: uma visão panorâmica
Palestrante: ANDREI CARVALHO SPOSITO (SP)

08:55-09:10 Diabetes e DCV: quais os biomarcadores e quando os utilizar
Palestrante: FERNANDO VALENTE (SP) – Faculdade de Medicina do ABC

09:10-09:25 Diabetes e IC: desafios no diagnóstico e tratamento
Palestrante: MARIA ELIZABETH ROSSI DA SILVA (SP) – Hospital das Clínicas da Faculdade de Medicina da USP

09:25-09:40 Risco cardiovascular em pessoas com DM1: como avaliar e minimizar?
Palestrante: ISABELLA CRISTINA PALIARES (SP) – Unifesp

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-12:00 PAINEL
TRATAMENTO DA OBESIDADE: O QUE HÁ DE NOVO NO HORIZONTE?

10:30-12:00
Moderador: MARISA HELENA CÉSAR CORAL (SC)

10:30-12:00
Moderador: FÁBIO ROGÉRIO TRUJILHO (BA) – CEDEBA-BA

10:30-10:40 Tema Livre: Improvements in Cardiometabolic Risk Factors by Body Weight Reduction with Tirzepatide in Adults with Obesity and Prediabetes: Post Hoc Analysis of SURMOUNT-1
Apresentador(a): BIANCA MARIANI GONÇALVES

10:40-10:55 Agonista duplo GLP1/Glucagon
Palestrante: JOÃO EDUARDO NUNES SALLES (SP) – Faculdade de Ciências Médicas da Santa Casa de São Paulo

10:55-11:10 Agonista duplo GLP1 antagonista GIP
Palestrante: FÁBIO ROGÉRIO TRUJILHO (BA) – CEDEBA-BA

11:10-11:25 Análogo de amilina
Palestrante: MARCIO CORRÊA MANCINI (SP) – HCFMUSP

11:25-11:40 Análogos triplos GLP1/GIP/Glucagon
Palestrante: CAROLINA GARCIA SOARES LEÃES RECH (RS)

11:40-12:00 Perguntas e Respostas

12:10-12:40 MINICONFERÊNCIA
CÂNCER MEDULAR DE TIREOIDE: DESAFIOS, AVANÇOS E PERSPECTIVAS TERAPÊUTICAS.

12:10-12:40
Moderador: ANA LUIZA SILVA MAIA (RS)

12:10-12:40
Palestrante: ANA AMÉLIA FIALHO DE OLIVEIRA HOFF (SP)

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
DILEMAS NO TRATAMENTO DA OBESIDADE

13:30-15:00
Moderador: HENRIQUE DE LACERDA SUPLICY (PR) – SEMPR – UFPR

13:30-15:00
Moderador: BRUNO HALPERN (SP)

13:30-13:50 Estratégias para promoção da alimentação saudável na Atenção Primária à Saúde: experiências de sucesso
Palestrante: MARIA LAURA DA COSTA LOUZADA (SP)

13:50-14:10 Reganho de peso na obesidade: mecanismos subjacentes e implicações clínicas
Palestrante: JOÃO SÉRGIO NEVES (PORTUGAL)

14:10-14:30 Tratamento da obesidade no idoso
Palestrante: JULIANA SANTOS SIMONETTI (ESTADOS UNIDOS DA AMÉRICA)

14:30-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 HOT TOPICS
HOT TOPICS EM OSTEOMETABOLISMO: COISAS QUE VOCÊ QUER SABER MAS NUNCA PERGUNTA

15:30-16:30
Moderador: FRANCISCO ALFREDO BANDEIRA E FARIAS (PE)

15:30-16:30
Moderador: FRANCISCO DE ASSIS PEREIRA (SE)

15:30-15:45 Como eu investigo e trato a hipercalciúria
Palestrante: CAROLINA AGUIAR MOREIRA (PR) – UFPR

15:45-16:00 Manejo da Osteossarcopenia
Palestrante: LÍVIA MARCELA DOS SANTOS (SP) – Centro Universitário São Camilo

16:00-16:15 K2 e Mg necessidade real ou moda
Palestrante: IANA MIZUMUKAI DE ARAÚJO (SP)

16:15-16:30 Perguntas e Respostas

16:30-17:00 CONFERÊNCIA
GLICOCENTRISMO VS. ADIPOCENTRISMO: REPENSANDO O PARADIGMA DA ABORDAGEM DO DM

16:30-17:00
Moderador: AMÉLIO FERNANDO GODOY MATOS (RJ)

16:30-17:00
Palestrante: RUY LYRA DA SILVA FILHO (PE) – UFPE

17:00-18:00 PAINEL
TECIDO ADIPOSO ECTÓPICO NA DIABESIDADE

17:00-18:00
Moderador: RUY LYRA DA SILVA FILHO (PE) – UFPE

17:00-18:00
Moderador: JOÃO ROBERTO DE SÁ (SP) – Centro Universitário Faculdade de Medicina do ABC

17:00-17:15 Fígado esteatótico: resistência insulínica ou ao glucagon?
Palestrante: AMÉLIO FERNANDO GODOY MATOS (RJ)

17:15-17:30 Esteatose renal na gênese da doença renal crônica
Palestrante: GIOVANA DE GOBBI AZEVEDO (SP)

17:30-17:45 Mioesteatose: quando a qualidade importa mais que quantidade
Palestrante: ROSANE KUPFER (RJ) – IEDE

17:45-18:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA IPANEMA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
QUANDO O CICLO PAUSA: ATUALIZAÇÕES NO MANEJO DA PACIENTE COM AMENORREIA

08:30-10:00
Moderador: KAREN FAGGIONI DE MARCA (RJ)

08:30-10:00
Moderador: ELAINE MARIA FRADE COSTA (SP)

08:30-08:40 Tema Livre: Mental health screening with MINI RTM in trans women and travestis attended at reference centers in Bahia: psychiatric burden, childhood adversity, and quality of life
Apresentador(a): DEBORA JAMILE PEREIRA DA SILVA

08:40-08:55 Amenorreia Hipotalâmica Funcional
Palestrante: DOLORES PEROVANO PARDINI (SP)

08:55-09:10 Insuficiência Ovariana Prematura
Palestrante: MÔNICA DE OLIVEIRA (PE) – IMIP/PE

09:10-09:25 Síndrome dos ovários policísticos (SOP)
Palestrante: LUCAS BANDEIRA MARCHESAN (RS) – Hospital Nossa Senhora da Conceição

09:25-09:40 Doenças crônicas sistêmicas
Palestrante: THYCIARA FONTENELE MARQUES (CE) – Faculdade de Medicina de Juazeiro do Norte

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:00 PRÊMIO
PRÊMIO ANTÔNIO BARROS DE ULHOA CINTRA

10:30-10:40 Apresentação (em nome da Comissão de História da Endocrinologia, uma apresentação sobre o significado do prêmio e anúncio do nome do laureado)
Palestrante: JOÃO MODESTO FILHO

10:40-10:50 Apresentação: (da biografia do laureado)
Palestrante: CESAR LUIZ BOGUSZEWSKI (PR) – UFPR

10:50-11:00 Agradecimento
Palestrante: HENRIQUE DE LACERDA SUPLICY (PR) – SEMPR – UFPR

11:00-11:30 CONFERÊNCIA
DIABETES ATÍPICO: DIAGNÓSTICO E ABORDAGEM ATUAL

11:00-11:30
Moderador: HENRIQUE DE LACERDA SUPLICY (PR) – SEMPR – UFPR

11:00-11:30
Palestrante: SÉRGIO ATALA DIB (SP) – UNIVERSIDADE FEDERAL DE SÃO PAULO – UNIFESP

11:30-12:00 PRÊMIO
PRÊMIO JOSE SCHERMANN

11:30-11:40 Apresentação (sobre o significado do prêmio e anúncio do nome do laureado)
Palestrante: RODRIGO DE OLIVEIRA MOREIRA (MG)

11:40-11:50 Apresentação: (da biografia do laureado)
Palestrante: ADRIANE DE ANDRE CARDOSO DEMARTINI (PR) – UFPR

11:50-12:00 Agradecimento
Palestrante: MARGARET CRISTINA DA SILVA BOGUSZEWSKI (PR) – UFPR

12:10-12:40 CONFERÊNCIA
O ENIGMA BRASILEIRO DOS TUMORES ADRENAIS PEDIÁTRICOS: POR TRÁS DA MAIOR PREVALÊNCIA MUNDIAL

12:10-12:40
Moderador: LUIZ CLAUDIO GONÇALVES DE CASTRO (DF) – UNB

12:10-12:40
Palestrante: SONIR ROBERTO RAUBER ANTONINI (SP) – FMRP-USP

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
A QUÍMICA INVISÍVEL QUE REESCREVE O CICLO DA VIDA: DESREGULADORES ENDÓCRINOS EM FOCO

13:30-15:00
Moderador: MARIA IZABEL CHIAMOLERA (SP)

13:30-15:00
Moderador: CAROLINE SERRANO DO NASCIMENTO (SP)

13:30-13:40 Tema Livre: Subchronic di(2-ethylhexyl) phthalate exposure alters neurobehavior and disrupts thyroid axis signaling in adult zebrafish
Apresentador(a): ANDRESSA PEREIRA CABRAL (RJ)

13:40-14:00 Tireoide
Palestrante: LEANDRO MIRANDA-ALVES (RJ)

14:00-14:20 Obesidade
Palestrante: PATRÍCIA CRISTINA LISBOA (RJ) – UERJ

14:20-14:40 Fertilidade
Palestrante: RENATA MARINO ROMANO (PR)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:00 CONFERÊNCIA
O ANO EM NEUROENDOCRINOLOGIA

15:30-16:00
Moderador: CESAR LUIZ BOGUSZEWSKI (PR) – UFPR

15:30-16:00
Palestrante: MÔNICA GADELHA (RJ)

16:00-16:30 CONFERÊNCIA
MEDICINA DE PRECISÃO: O PAPEL DOS TESTES GENÉTICOS NO TRATAMENTO DA OBESIDADE

16:00-16:30
Moderador: CLAYTON LUIZ DORNELLES MACEDO (RS)

16:00-16:30
Palestrante: JULIANA SANTOS SIMONETTI (ESTADOS UNIDOS DA AMÉRICA)

16:30-18:00 HOT TOPICS
HOT TOPICS EM OBESIDADE E METABOLISMO

16:30-18:00
Moderador: RODRIGO DE OLIVEIRA MOREIRA (MG)

16:30-18:00
Moderador: JACQUELINE RIZZOLLI (RS) – Hospital São Lucas da PUCRS

16:30-16:40 Tema Livre: Is there a metabolic dysfunction-associated fatty kidney disease (MAFKD) in individuals with grade 1 obesity?
Apresentador(a): GIOVANA DE GOBBI AZEVEDO (SP)

16:40-16:55 Lipedema: limites no diagnóstico e tratamento
Palestrante: ANDRÉ MURAD FARIA (SP)

16:55-17:10 O desafio da obesidade na infância
Palestrante: ADRIANE DE ANDRE CARDOSO DEMARTINI (PR) – UFPR

17:10-17:25 Sono, cronobiologia e ganho de peso: o que fazer no consultório?
Palestrante: RICARDO DE ANDRADE OLIVEIRA (RJ)

17:25-17:40 Desprescrição: o que muda depois que o paciente atinge o peso-alvo?
Palestrante: RODRIGO DE OLIVEIRA MOREIRA (MG)

17:40-18:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA LEBLON (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
DESAFIOS METABÓLICOS NA ENDOCRINOLOGIA PEDIÁTRICA

08:30-10:00
Moderador: MICHELINE ABREU RAYOL DE SOUZA (RJ) – UFRJ

08:30-10:00
Moderador: BRUNO LEANDRO DE SOUZA (PB)

08:30-08:40 Tema Livre: Rising burden and racial inequity in diabetic ketoacidosis hospitalizations among Brazilian children and adolescents: a 15-year nationwide trend analysis (2010–2024)
Apresentador(a): SÁVIO CARVALHO GOMES

08:40-09:00 Adrenarca prematura: marcador precoce de risco metabólico?
Palestrante: THAIS MILIONI LUCIANO (SP)

09:00-09:20 Endocrinologia do paciente oncológico: estratégias no cuidado
Palestrante: PAULO ALONSO GARCIA ALVES JUNIOR (RJ) – INCA/MS

09:20-09:40 Osteoporose induzida por glicocorticoides: por que na criança é diferente?
Palestrante: LUIZ CLAUDIO GONÇALVES DE CASTRO (DF) – UNB

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 DEBATE
DEBATE COM CASO CLÍNICO – HIPOTIREOIDISMO SUBCLÍNICO EM CRIANÇAS E ADOLESCENTES: TRATAR OU NÃO TRATAR?

10:30-11:30
Moderador: PAULO ALONSO GARCIA ALVES JUNIOR (RJ) – INCA/MS

10:30-11:30
Moderador: JOSÉ AUGUSTO SGARBI (SP) – FAC. MEDICINA DE MARILIA

10:30-10:50 A favor
Palestrante: CRISTIANE KOPACEK (RS) – UFCSPA

10:50-11:10 Contra
Palestrante: ANA PAULA NEVES BORDALLO (RJ)

11:10-11:30 Discussão

11:40-12:40 – SIMPÓSIO PATROCINADO
SIMPÓSIO: ADIUM

11:40-12:40 Mudança de Paradigma no Hipoparatireoidismo Crônico: Estamos prontos para olhar além do cálcio?
Palestrante: VICTÓRIA ZEGHBI COCHENSKI BORBA (PR) – UFPR

11:40-12:40 Mudança de Paradigma no Hipoparatireoidismo Crônico: Estamos prontos para olhar além do cálcio?
Palestrante: ALUÍZIO BARBOSA DE CARVALHO (SP)

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
THM NA VIDA REAL: ESTRATÉGIAS PARA CENÁRIOS COMPLEXOS

13:30-15:00
Moderador: MÔNICA DE OLIVEIRA (PE) – IMIP/PE

13:30-15:00
Moderador: RUTH CLAPAUCH (RJ)

13:30-13:40 Tema Livre: Trans-Cardio Score: a translational precision cardiometabolic framework for transgender individuals integrating biological determinants and machine learning
Apresentador(a): MAICON CESAR CANUTO (PR)

13:40-13:55 Impacto das Lesões Prévias de Mama
Palestrante: MARIA JULIA GREGÓRIO CALAS (RJ)

13:55-14:10 História familiar de câncer de mama
Palestrante: RODRIGO GUINDALIN (RJ)

14:10-14:25 Trombofilias
Palestrante: FLÁVIA LÚCIA CONCEIÇÃO (RJ)

14:25-14:40 Câncer de ovário
Palestrante: GRAZIELLA NOGUEIRA MENDONÇA MONTEIRO DE BARROS (RJ)

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-16:30 DEBATE
A TERAPIA HORMONAL DA MENOPAUSA REDUZ O RISCO CARDIOVASCULAR?

15:30-16:30
Moderador: MARCELO FERNANDO RONSONI (SC) – UFSC

15:30-16:30
Palestrante: TAYANE MUNIZ FIGHERA (RS)

15:30-16:30
Palestrante: CICILIANA MAILA ZILIO RECH (RS) – Universidade Federal da Fronteira Sul

16:30-18:00 PAINEL
ENDOCRINOLOGIA E ARTE

16:30-18:00
COORDENAÇÃO: ERIK TROVAO DINIZ (PE)

16:30-16:43 "O assassino da lua das flores" – Doçura Amarga: Como mudanças alimentares geram diabetes
Palestrante: SERGIO SETSUO MAEDA (SP) – Unifesp

16:43-17:03 O Nanismo entre a Arte, a Ciência e as Políticas Públicas
Palestrante: AYRTON CUSTODIO MOREIRA (SP) – Faculdade de Medicina de Ribeirão Preto da Universidade de São Paulo FMUSP-RP

17:03-17:16 "Moulin Rouge – o amor em vermelho" – Luz, Cena e Fragilidade Óssea: qual a doença de Toulouse-Lautrec?
Palestrante: SERGIO SETSUO MAEDA (SP) – Unifesp

17:16-17:36 Eunucoidismo: Biologia e/ou Construção Cultural?
Palestrante: AYRTON CUSTODIO MOREIRA (SP) – Faculdade de Medicina de Ribeirão Preto da Universidade de São Paulo FMUSP-RP

17:36-17:49 "Lições de 'O Agente Secreto': o que a dona Sebastiana tem? 10 minutos: Interação com plateia"
Palestrante: SERGIO SETSUO MAEDA (SP) – Unifesp

17:49-18:00 Interação com a plateia

WINDSOR OCEÂNICO HOTEL: SALA LEME (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
TEMAS QUENTES EM NEUROENDOCRINOLOGIA!

08:30-10:00
Moderador: SÍLVIA REGINA CÔRREA DA SILVA (SP) – Unifesp

08:30-10:00
Moderador: LUCIO VILAR RABELO FILHO (PB) – Universidade Federal de Pernambuco (UFPE)

08:30-08:40 Tema Livre: Exon 3 Deletion Growth Hormone Receptor Polymorphism Modulates Fasting Glycemia in Childhood Onset GH Deficiency Adult Patients on Continuous Growth Hormone Replacement
Apresentador(a): RYANN LUCCAS DE MIRANDA SOUZA

08:40-08:55 Craniofaringioma: perspectivas terapêuticas
Palestrante: MALEBRANCHE BERARDO CARNEIRO DA CUNHA NETO (SP) – HCFMUSP

08:55-09:10 Novo Consenso sobre tumores hipofisários agressivos: o que mudou?
Palestrante: BEATRIZ CARVALHO LOPES (MG)

09:10-09:25 Distúrbios hipofisários em Oncologia
Palestrante: FELIPE HENNING GAIA DUARTE (SP)

09:25-09:40 Deficiência de AVP: nova nomenclatura, novos testes?
Palestrante: JULIANA BEAUDETTE DRUMMOND (MG)

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 OFICINA
OFICINA DE IMAGEM EM ADRENAL: O QUE O ENDOCRINOLOGISTA PRECISA SABER (E NEM SEMPRE PERGUNTA)

10:30-11:30
COORDENAÇÃO: ANDREA GLEZER (SP) – HC-FMUSP

10:30-11:30
COORDENAÇÃO: FLAVIA AMANDA COSTA BARBOSA (SP) – EPM/Unifesp

10:30-11:30
Moderador: MAURO ANTÔNIO CZEPIELEWSKI (RS) – UFRGS

10:30-11:30 Endocrinologista
Palestrante: RAFAEL BUCK GIORGI (SP) – Unifesp

10:30-11:30 Radiologista
Palestrante: DANIELLA BRAZ PARENTE (RJ)

10:30-11:30 Médico Nuclear
Palestrante: FRANCISCO DE ASSIS ROMEIRO (RJ)

10:30-11:30 Radiointervencionista
Palestrante: ALINE CRISTINE BARBOSA SANTOS CAVALCANTE (SP)

11:30-12:30 OFICINA
OFICINA DE IMAGEM EM NEUROENDÓCRINO: O QUE O ENDOCRINOLOGISTA PRECISA

11:30-12:30
COORDENAÇÃO: ANDREA GLEZER (SP) – HC-FMUSP

11:30-12:30
COORDENAÇÃO: FLAVIA AMANDA COSTA BARBOSA (SP) – EPM/Unifesp

11:30-12:30
Moderador: JÚLIO ZAKI ABUCHAM FILHO (SP) – Universidade Federal de São Paulo (SP) Unifesp

11:30-12:30 Endocrinologista
Palestrante: MALEBRANCHE BERARDO CARNEIRO DA CUNHA NETO (SP) – HCFMUSP

11:30-12:30 Radiologista
Palestrante: NINA VENTURA (RJ)

11:30-12:30 Neurocirurgião
Palestrante: PAULO NIEMEYER (RJ)

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
OBESIDADE E GÊNERO: UMA DISCUSSÃO SOBRE DIVERSIDADE EQUIDADE E INCLUSÃO

13:30-15:00
Moderador: ERIK TROVAO DINIZ (PE)

13:30-15:00
Moderador: LUCIANA MATTOS BARROS OLIVEIRA (BA)

13:30-13:40 Tema Livre: Association between body image and sexual function in women: the role of obesity
Apresentador(a): LAURA RABAIOLI

13:40-14:00 Desigualdades no Diagnóstico e Tratamento em Homens, Mulheres e Pessoas Trans – Discussão sobre como a obesidade é abordada de forma distinta para diferentes identidades de gênero e as implicações disso no manejo da condição.
Palestrante: MAGNUS REGIOS DIAS DA SILVA (SP) – EPM/Unifesp

14:00-14:20 Estigmatização da Obesidade: Impactos no Diagnóstico, Tratamento e Bem-estar de Pacientes – Análise do estigma associado à obesidade, como ele afeta a adesão ao tratamento e quais estratégias podem ser usadas para promover um cuidado livre de preconceitos.
Palestrante: CAROLINA GARCIA SOARES LEÃES RECH (RS)

14:20-14:40 Como a obesidade se manifesta de maneira diferente em diferentes etnias, idades, gêneros e classes sociais, e como podemos personalizar abordagens terapêuticas para cada grupo
Palestrante: MARIA EDNA DE MELO (SP) – Hospital das Clínicas da Faculdade de Medicina da USP

14:40-15:00 Perguntas e Respostas

15:00-15:30 – INTERVALO
INTERVALO

15:30-17:00 SIMPÓSIO
CIRURGIA ENDÓCRINA – MÓDULO: PARATIREOIDE

15:30-15:35 ABERTURA
Moderador: MANUEL DOMINGOS DA CRUZ GONÇALVES (RJ – PORTUGAL)

15:35-15:50 O hiperparatireodismo primário clássico: quando indicar cirurgia
Palestrante: MARIA LUCIA FLEIUSS DE FARIAS (RJ)

15:50-16:05 Quando indicar cirurgia no HPT primário com secreção inapropriada de PTH
Palestrante: MONIQUE NAKAYAMA OHE (SP) – Unifesp-EPM

16:05-16:20 A cirurgia minimamente invasiva do hiperparatireodismo
Palestrante: ALBERTO TREIGUER (RS)

16:20-16:35 Decisões cirúrgicas na abordagem em HPT sem localização prévia
Palestrante: EVANDRO VASCONCELOS (PR)

16:35-17:00 Discussão com os painelistas

17:30-19:00 SIMPÓSIO
CIRURGIA ENDÓCRINA – MÓDULO: ADRENAL

17:30-17:35 ABERTURA
Moderador: SILVIO HENRIQUES DA CUNHA NETO (RJ)

17:35-17:50 A prevalência do hiperaldosteronismo em hipertensos
Palestrante: LEONARDO VIEIRA NETO (RJ) – UFRJ

17:50-18:05 O diagnóstico diferencial nas lesões de adrenais pelo cateterismo: o benefício clínico justifica a rotina?
Palestrante: MADSON QUEIROZ DE ALMEIDA (SP)

18:05-18:20 Qual é o melhor acesso para adrenalectomia?
Palestrante: JOSÉ GUSTAVO OLIJNYK (RS)

18:20-18:35 O preparo pré-operatório na cirurgia do feocromocitoma
Palestrante: RAFAEL NAGATO (RJ)

18:35-19:00 Discussão com os painelistas

WINDSOR BARRA HOTEL: SALA SÃO CONRADO (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
DISFUNÇÃO TIREOIDIANA NA GESTAÇÃO: O MANEJO QUE EVITA DANOS IRREVERSÍVEIS

08:30-10:00
Moderador: GIOVANNA APARECIDA BALARINI LIMA (SP)

08:30-10:00
Moderador: GISAH MUSIALOWSKI DO AMARAL (PR) – UFPR

08:30-08:40 Tema Livre: IODINE STATUS AND URINARY CONCENTRATIONS OF ENDOCRINE DISRUPTING CHEMICALS IN PREGNANT WOMEN WITH THYROID DISORDERS
Apresentador(a): MARIA IZABEL CHIAMOLERA (SP)

08:40-08:55 Micronutrientes associados a função tiroidiana na gestação – suplementar ou não?
Palestrante: SUEMI MARUI (SP)

08:55-09:10 "Diagnóstico de disfunção tireoidiana na gestação"
Palestrante: MARIA IZABEL CHIAMOLERA (SP)

09:10-09:25 Manejo do hipotireodismo na gestação
Palestrante: ROSALIA DO PRADO PADOVANI (SP) – Santa Casa de São Paulo

09:25-09:40 Manejo do hipertireoidismo na gestação
Palestrante: CLEO OTAVIANO MESA JÚNIOR (PR) – PUC PR

09:40-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 DEBATE
"A POLARIZAÇÃO NA ENDOCRINOLOGIA: DEBATE DISLIPIDÊMICO" – O NOVO SCORE PREVENT DE RISCO CV É MESMO SUPERIOR?

10:30-11:30
Moderador: MÁRCIO WEISSHEIMER LAURIA (MG) – UFMG

10:30-11:30
Moderador: AIRTON GOLBERT (RS)

10:30-11:00
Palestrante: MARIA HELANE COSTA GURGEL (CE) – Universidade Federal do Ceará UFC

11:00-11:30
Palestrante: MARCELLO CASACCIA BERTOLUCI (RS) – UFRGS

11:40-12:40 – SIMPÓSIO PATROCINADO
SIMPÓSIO: LILLY

11:40-12:40 O Futuro da Endocrinologia: Ciência e Inovação em Obesidade
Palestrante: CINTIA CERCATO (SP) – Faculdade de Medicina da Universidade de São Paulo USP

11:40-12:40 O Futuro da Endocrinologia: Ciência e Inovação em Obesidade
Palestrante: LUCA BUSETTO (ITÁLIA)

11:40-12:40 O Futuro da Endocrinologia: Ciência e Inovação em Obesidade
Palestrante: FERNANDO VALENTE (SP) – Faculdade de Medicina do ABC

12:40-13:30 – INTERVALO
ALMOÇO

13:30-14:00 CONFERÊNCIA
HIDDEN HYPERCORTISOLISM IN TYPE 2 DIABETES: INSIGHTS FROM ADRENAL–PANCREATIC CROSSTALK

13:30-14:00
Moderador: MARIVÂNIA DA COSTA SANTOS (PB) – UFPB

13:30-14:00
Palestrante: RICHARD J. AUCHUS (ESTADOS UNIDOS DA AMÉRICA)

14:00-15:00 HOT TOPICS
REPOSIÇÃO DE GH EM ADULTOS: QUANDO E COMO?

14:00-15:00
Moderador: ANA BEATRIZ WINTER TAVARES (RJ)

14:00-15:00
Moderador: PAULO AUGUSTO CARVALHO MIRANDA (MG)

14:00-14:10 Como diagnosticar a deficiência de GH?
Palestrante: MANUEL HERMÍNIO DE AGUIAR OLIVEIRA (SP) – Faculdade de Medicina de Ribeirão Preto

14:10-14:20 Quando tratar?
Palestrante: SÍLVIA REGINA CÔRREA DA SILVA (SP) – Unifesp

14:20-14:30 GH semanal
Palestrante: MANOEL RICARDO ALVES MARTINS (PE) – Universidade Federal do Ceará UFC

14:30-14:40 Efeitos metabólicos da reposição
Palestrante: ANNE ELISE ALEXANDRE (SP)

14:40-15:00 Discussão

15:00-15:30 – INTERVALO
INTERVALO

16:30-18:00 PAINEL
HIPERPLASIA ADRENAL CONGÊNITA: EXPECTATIVAS E REALIDADE

16:30-18:00
Moderador: CLAUDIO ELIAS KATER (SP) – Unifesp

16:30-18:00
Moderador: MARIVÂNIA DA COSTA SANTOS (PB) – UFPB

16:30-16:40 Tema Livre: Somatic Mutations as Predictors of Recurrence in TP53 p.R337H-Associated Adrenocortical Tumors: A Pediatric–Adult Comparative Study.
Apresentador(a): MATEUS ANDRADE BOMFIM MACHADO

16:40-16:55 Deficiência de 21-hidroxilase: O que há de novo no tratamento
Palestrante: FLAVIA AMANDA COSTA BARBOSA (SP) – EPM/Unifesp

16:55-17:10 Diagnóstico de HAC 21 Hidroxilase na infância: o teste de estímulo com ACTH é mesmo necessário?
Palestrante: NATHÁLIA BORDEIRA CHAGAS (SP)

17:10-17:25 Manejo do adulto com formas não clássicas
Palestrante: TANIA APARECIDA SANCHEZ BACHEGA (SP) – USP

17:25-17:40 Oxyandrogens in the Management of 21-Hydroxylase Deficiency Congenital Adrenal Hyperplasia
Palestrante: RICHARD J. AUCHUS (ESTADOS UNIDOS DA AMÉRICA)

17:40-18:00 Perguntas e Respostas

29/08/2026 – SÁBADO

WINDSOR OCEÂNICO HOTEL: SALA ARPOADOR (WINDSOR OCEÂNICO HOTEL)

08:30-09:30 PREMIAÇÃO
COMISSÃO JULGADORA DOS PRÊMIOS – BÁSICO TRANSLACIONAL

08:30-09:30
COORDENAÇÃO: MARISE LAZARETTI CASTRO (SP)

08:30-09:30
COORDENAÇÃO: LAURA STERIAN WARD (SP)

08:30-09:30
COORDENAÇÃO: PATRÍCIA CRISTINA LISBOA (RJ) – UERJ

08:30-08:45 Total RNA Sequencing Reveals an Expanded Landscape of Gene Fusion Complexity in Pheochromocytomas and Paragangliomas
Apresentador(a): EDUARDO CUNHA LOBATO

08:45-09:00 Irgarol as a potential thyroid endocrine disruptor: evidence from human tumoral and non-tumoral cell lines
Apresentador(a): REINALDO ROPKE

09:00-09:15 Retroelement–Immune Axis Defines A Distinct Immunobiological Phenotype In Somatotroph Tumors
Apresentador(a): RAFAEL LOCH BATISTA (SP)

09:15-09:30 Somatic Mutations as Predictors of Recurrence in TP53 p.R337H-Associated Adrenocortical Tumors: A Pediatric–Adult Comparative Study
Apresentador(a): MATEUS ANDRADE BOMFIM MACHADO

09:30-10:30 PREMIAÇÃO
COMISSÃO JULGADORA DOS PRÊMIOS – CLÍNICO

09:30-10:30
COORDENAÇÃO: MARIA LUCIA FLEIUSS DE FARIAS (RJ)

09:30-10:30
COORDENAÇÃO: FLÁVIA COIMBRA PONTES MAIA (MG)

09:30-10:30
COORDENAÇÃO: VICTÓRIA ZEGHBI COCHENSKI BORBA (PR) – UFPR

09:30-09:45 Is there a metabolic dysfunction-associated fatty kidney disease (MAFKD) in individuals with grade 1 obesity?
Apresentador(a): GIOVANA DE GOBBI AZEVEDO (SP)

09:45-10:00 Clinicopathological and Molecular Profile of Brazilian Patients with Cushing Syndrome Due to Primary Pigmented Micronodular Adrenal Hyperplasia
Apresentador(a): ISABELA NARDO

10:00-10:15 Exon 3 Deletion Growth Hormone Receptor Polymorphism Modulates Fasting Glycemia in Childhood Onset GH Deficiency Adult Patients on Continuous Growth Hormone Replacement
Apresentador(a): RYANN LUCCAS DE MIRANDA SOUZA

10:15-10:30 ACTH as a Cost-Effective Marker of Dexamethasone Ingestion in the Dexamethasone Suppression Test: Preliminary Evidence Challenging Current Practice with Implications for Resource-Limited Settings
Apresentador(a): MATEUS ANDRADE BOMFIM MACHADO

10:00-10:30 – INTERVALO
INTERVALO

10:30-16:00 PAINEL
ENDOQUIZ – DESAFIO SBEM (VENHA TESTAR SEU CONHECIMENTO E APRENDER CONOSCO)

10:30-16:00
COORDENAÇÃO: ALESSANDRA SALDANHA MATHEUS FERNANDES DA COSTA (RJ)

10:30-16:00
COORDENAÇÃO: LUCIANNE RIGHETI MONTEIRO TANNUS (RJ)

10:30-16:00
COORDENAÇÃO: ANDRÉ GONÇALVES DA SILVA (PI)

10:30-16:00
COORDENAÇÃO: RAFAEL VAZ MACHRY (RS)

10:30-12:40 Jogo das Glândulas: Apresentação das regras, Equipes avançarão sobre um tabuleiro gigante com casas temáticas e receberão desafios surpresas! A equipe que conseguir avançar mais casas será a vencedora

12:40-13:30 Almoço

13:30-15:00 Batalha Endo na Meta: Apresentação das regras, Divisão das equipes por cores, para responderem a um quiz sobre endocrinologia e diabetes, 3 equipes vencedoras ganharão medalhas e certificados

15:00-16:00 Encerramento

12:40-13:30 - INTERVALO
ALMOÇO

WINDSOR BARRA HOTEL: SALA BARRA DA TIJUCA (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
TEMAS QUENTES EM ENDOPEDIATRIA

08:30-10:00
Moderador: SONIR ROBERTO RAUBER ANTONINI (SP) – FMRP-USP

08:30-10:00
Moderador: ANA PINHEIRO MACHADO CANTON (SP)

08:30-08:45 Sexagem fetal: quando o chá revelação pode ser um problema.
Palestrante: GIL GUERRA JUNIOR (SP) – FCM UNICAMP

08:45-09:00 Desreguladores endócrinos e distúrbios da puberdade: evidência científica ou excesso de alarme?
Palestrante: CAROLINE SERRANO DO NASCIMENTO (SP)

09:00-09:15 Puberdade precoce central em meninos: avanços e desafios na prática clínica
Palestrante: JACQUELINE ROSÂNGELA DE ARAÚJO (PE) – HC-UFPE

09:15-09:30 Análogos do GLP-1 para a criança com obesidade grave?
Palestrante: CRISTIANE KOCHI (SP)

09:30-09:45 Hiperfagia em crianças e adolescentes: do reconhecimento ao manejo clínico
Palestrante: LATIFE SALOMÃO TYSZLER (RJ) – IEDE

09:45-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-12:40 OFICINA
MÉTODOS DIAGNÓSTICOS NA AVALIAÇÃO DA PESSOA COM OBESIDADE

10:30-12:40
Moderador: LUCIANA LOPES DE SOUZA

10:30-10:45 Métodos de Imagem na avaliação do Lipedema
Palestrante: JOANA RODRIGUES DANTAS VEZZANI (RJ) – Hospital Universitário Clementino Fraga Filho –UFRJ

10:45-11:00 Ultrassonografia para avaliação do Lipe (Demonstração Prática)
Palestrante: ADRIANA SOUSA E SILVA (RJ)

11:00-11:15 Métodos de imagem na avaliação da distribuição da gordura corporal
Palestrante: RAQUEL BEATRIZ GONÇALVES MUNIZ (RJ)

11:15-11:30 Ultrassonografia para avaliação da gordura visceral (Demonstração Prática)
Palestrante: JOÃO MARCELLO DE ARAUJO NETO (RJ)

11:15-11:30 Ultrassonografia para avaliação da gordura visceral (Demonstração Prática)
Palestrante: PRISCILA CALVET (RJ)

11:30-11:50 Elastografia com Fibroscan na avaliação da esteatose e rigidez hepática (Demonstração Prática)
Palestrante: JOÃO MARCELLO DE ARAUJO NETO (RJ)

11:50-12:10 Métodos de Imagem para avaliação de massa muscular
Palestrante: JOÃO MARCELLO DE ARAUJO NETO (RJ)

11:50-12:10 Métodos de Imagem para avaliação de massa muscular
Palestrante: PRISCILA CALVET (RJ)

12:10-12:40 Discussão

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
MUDANÇAS DE RUMO NO TRATAMENTO DO PACIENTE COM DM AO LONGO DA VIDA

13:30-15:00
Moderador: NELSON RASSI (GO)

13:30-15:00
Moderador: DOMINGOS AUGUSTO MALERBI (SP)

13:30-13:45 Quando o paciente se torna idoso?
Palestrante: BEATRIZ DAGORD SCHAAN (RS)

13:45-14:00 E quando chega a doença renal crônica?
Palestrante: FERNANDO VALENTE (SP) – Faculdade de Medicina do ABC

14:00-14:20 E quando surge a neuropatia autonômica cardíaca?
Palestrante: LUIZ CLEMENTE DE SOUZA PEREIRA ROLIM (SP)

14:20-14:40 E quando o paciente necessita de cirurgia de grande porte?
Palestrante: SILMARA APARECIDA DE OLIVEIRA LEITE (PR)

14:40-15:00 Perguntas e respostas

WINDSOR OCEÂNICO HOTEL: SALA COPACABANA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 PAINEL
CIRURGIA BARIÁTRICA E METABÓLICA

08:30-10:00
Moderador: FERNANDO GERCHMAN (RS) – UFRGS

08:30-10:00
Moderador: CYNTHIA MELISSA VALERIO (RJ) – UFRJ

08:30-08:45 Prevendo a perda de peso pós-bariátrica: calculadora SOPHIA
Palestrante: JOÃO SÉRGIO NEVES (PORTUGAL)

08:45-09:00 Tratamento farmacológico antes e após a cirurgia bariátrica
Palestrante: JACQUELINE RIZZOLLI (RS) – Hospital São Lucas da PUCRS

09:00-09:15 Técnicas endoscópicas no tratamento da obesidade-TDB
Palestrante: EDUARDO MADEIRA (RJ)

09:15-09:30 Novas Técnicas cirúrgicas aprovadas
Palestrante: DENIS PAJECKI (SP)

09:30-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 DEBATE
HÁ ESPAÇO PARA A CIRURGIA METABÓLICA NOS PACIENTES COM IMC 35, NA DISPONIBILIDADE DAS NOVAS MEDICAÇÕES?

10:30-11:30
Moderador: JACQUELINE RIZZOLLI (RS) – Hospital São Lucas da PUCRS

10:30-10:50
Palestrante: DENIS PAJECKI (SP)

10:50-11:10
Palestrante: MARCIO CORRÊA MANCINI (SP) – HCFMUSP

11:10-11:30 Discussão

11:40-12:40 PAINEL
CROSSTALK PÂNCREAS-ADRENAL-PARATIROIDE: NOVOS HORIZONTES

11:40-12:40
Moderador: FLAVIA AMANDA COSTA BARBOSA (SP) – EPM/Unifesp

11:40-12:40
Moderador: FRANCISCO JOSÉ ALBUQUERQUE DE PAULA (CE) – FMRP-USP

11:40-11:55 Impacto metabólico do hipercortisolismo leve
Palestrante: DANIELA ESPÍNDOLA ANTUNES (GO) – Unifesp

11:55-12:10 Hiperaldosteronismo e hiperparatiroidismo: desafios diários
Palestrante: MONIQUE NAKAYAMA OHE (SP) – Unifesp -EPM

12:10-12:25 Quando pensar nas doenças monogênicas (MENs e Cia)
Palestrante: DELMAR MUNIZ LOURENÇO JUNIOR (SP) – HCFMUSP

12:25-12:40 Perguntas e Respostas
12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
DESAFIOS EM NEUROENDOCRINOLOGIA

13:30-15:00
Moderador: MONIKE LOURENÇO DIAS RODRIGUES (GO)

13:30-15:00
Moderador: LUIZ EDUARDO ARMONDI WILDEMBERG (MG)

13:30-13:45 Manejo perioperatório das lesões hipofisárias
Palestrante: PAULA CONDÉ LAMPARELLI ELIAS (SP)

13:45-14:00 Armadilhas no diagnóstico da doença de Cushing
Palestrante: TOBIAS SKREBSKY DE ALMEIDA (RS)

14:00-14:15 Armadilhas e desafios na investigação de hiperprolactinemia
Palestrante: LUCIO VILAR RABELO FILHO (PB) – Universidade Federal de Pernambuco (UFPE)

14:15-14:30 Manejo da apoplexia hipofisária
Palestrante: MANOEL RICARDO ALVES MARTINS (PE) – Universidade Federal do Ceará UFC

14:30-15:00 Perguntas e Respostas
15:00 – 16:00 ENCERRAMENTO
ENTREGA DE PRÊMIOS E ENCERRAMENTO

WINDSOR OCEÂNICO HOTEL: SALA IPANEMA (WINDSOR OCEÂNICO HOTEL)

08:30-10:00 OFICINA
OFICINA DE SEXUALIDADE SBEM/ABEMSS: SEXUALIDADE NA PRÁTICA CLÍNICA DO ENDOCRINOLOGISTA

08:30-10:00
Moderador: MÔNICA DE OLIVEIRA (PE) – IMIP/PE

08:30-10:00
Moderador: ALEXANDRE HOHL (SC) – UFSC

08:30-08:45 Como abordar a sexualidade na consulta: comunicação e linguagem
Palestrante: CARMITA ABDO (SP)

08:45-09:00 Padrões alimentares e sexualidade
Palestrante: PRISCILLA GIL (RJ)

09:00-09:15 Como estresse e burnout impactam a libido e a função sexual?
Palestrante: NATHALIE RAIBOLT (RJ)

09:15-09:30 Uso de contraceptivos hormonais e sexualidade
Palestrante: LARISSA GARCIA GOMES (SP) – HCFMUSP

09:30-09:45 Acompanhamento endocrinológico de pessoas trans e não binárias
Palestrante: TAYANE MUNIZ FIGHERA (RS)

09:45-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:00 CONFERÊNCIA
OFICINA DE SEXUALIDADE SBEM/ABEMSS

10:30-11:00 O futuro da intimidade – papel das redes sociais e da IA na sexualidade
Palestrante: MICHELLE SAMPAIO (SP)

11:00-12:40 OFICINA
OFICINA DE SEXUALIDADE SBEM/ABEMSS: DISFUNÇÕES SEXUAIS – DIAGNÓSTICO E MANEJO

11:00-11:15 Abordagem da baixa libido do casal
Palestrante: MICHELLE SAMPAIO (SP)

11:15-11:30 Além da dosagem da testosterona: sensibilidade androgênica e genética do receptor de androgênio
Palestrante: RAFAEL LOCH BATISTA (SP)

11:30-11:45 Disfunção erétil na prática endocrinológica: muito além do PDE5
Palestrante: MARCELO FERNANDO RONSONI (SC) – UFSC

11:45-12:00 Desejo hipoativo em mulheres na pós-menopausa: abordagem baseada em evidências
Palestrante: ALEXANDRE HOHL (SC) – UFSC

12:00-12:15 Como tratar a baixa libido feminina com técnicas não farmacológicas
Palestrante: MÔNICA SANTOS LOPES (RJ)

12:15-12:30 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 FÓRUM
MULHERES QUE CUIDAM, MULHERES QUE LIDERAM: DA PROTEÇÃO À TRANSFORMAÇÃO

13:30-15:00
Moderador: KAREN FAGGIONI DE MARCA (RJ)

13:30-15:00
Moderador: TAYANE MUNIZ FIGHERA (RS)

13:30-15:00
Moderador: MARISE LAZARETTI CASTRO (SP)

13:30-13:45 Sexualidade e saúde mental da mulher
Palestrante: CARMITA ABDO (SP)

13:45-14:00 Câncer de mama: como conviver com essa ameaça constante?
Palestrante: MARIA JULIA GREGÓRIO CALAS (RJ)

14:00-14:15 Repercussões da violência doméstica sobre a saúde: o que o médico precisa saber
Palestrante: ANE CRISTINE SCHEELE SANTOS (RJ)

14:15-14:30 Desafios ainda subestimados: risco cardiovascular da mulher após a menopausa
Palestrante: CYNTHIA MELISSA VALERIO (RJ) – UFRJ

14:30-15:00 Debate com a plateia

WINDSOR BARRA HOTEL: SALA LEBLON (WINDSOR BARRA HOTEL)

08:30-09:45 MESA-REDONDA
INCIDENTALOMAS ADRENAIS NA PRÁTICA CLÍNICA

08:30-10:00
Moderador: MARIA CANDIDA BARISSON VILLARES FRAGOSO (SP)

08:30-10:00
Moderador: ALINE BARBOSA MORAES (RJ) – Universidade Federal Fluminense (UFF)

08:30-08:45 Armadilhas no diagnóstico de secreção autônoma leve de cortisol (MACS)
Palestrante: DANIELA ESPÍNDOLA ANTUNES (GO) – Unifesp

08:45-09:00 Seguimento e possíveis complicações de pacientes não submetidos a adrenalectomia
Palestrante: RAFAEL BUCK GIORGI (SP) – Unifesp

09:00-09:15 Indicação cirúrgica da secreção autônoma leve de cortisol
Palestrante: LEONARDO VIEIRA NETO (RJ) – UFRJ

09:15-09:30 Diagnóstico diferencial das massas adrenais bilaterais
Palestrante: GUILHERME ASMAR ALENCAR (MG) – HU-UFSC

09:30-09:45 Perguntas e Respostas

09:45-10:00 PRÊMIO
PRÊMIO ARCHIVES OF ENDOCRINOLOGY AND METABOLISM (AE&M) WALDEMAR BERARDINELLI

09:45-09:48 Entrega do prêmio
Palestrante: CESAR LUIZ BOGUSZEWSKI (PR) – UFPR

09:48-09:55 Epidemiological trends of adrenalectomies in Brazil: A cohort-based study of the Brazilian public health system
Palestrante: JOSÉ GUSTAVO OLIJNYK (RS)

09:55-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:30-11:30 DEBATE
TRATAMENTO DA OBESIDADE COM INCRETÍNICOS É PARA TODA A VIDA?

10:30-11:30
Moderador: ALEXANDER KOGLIN BENCHIMOL (RJ) – IEDE/PUC/RJ

10:30-10:50
Palestrante: BRUNO HALPERN (SP)

10:50-11:10
Palestrante: MARIA TERESA ZANELLA (SP)

11:10-11:30 Discussão

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
TRATAMENTO DA OBESIDADE EM SITUAÇÕES ESPECIAIS

13:30-15:00
Moderador: MARCIO CORRÊA MANCINI (SP) – HCFMUSP

13:30-15:00
Moderador: MARIA TERESA ZANELLA (SP)

13:30-13:45 No paciente hiperfágico
Palestrante: MARIA EDNA DE MELO (SP) – Hospital das Clínicas da Faculdade de Medicina da USP

13:45-14:00 Na gestante e lactante
Palestrante: ERIKA PANIAGO GUEDES (GO)

14:00-14:15 No idoso
Palestrante: JOÃO EDUARDO NUNES SALLES (SP) – Faculdade de Ciências Médicas da Santa Casa de São Paulo

14:15-14:30 Na doença renal grave
Palestrante: ALEXANDER KOGLIN BENCHIMOL (RJ) – IEDE/PUC/RJ

14:30-15:00 Perguntas e Respostas

WINDSOR OCEÂNICO HOTEL: SALA LEME (WINDSOR OCEÂNICO HOTEL)

08:30-11:00 SIMPÓSIO
SIMPÓSIO CIRURGIA ENDÓCRINA – MÓDULO: TIREOIDE

08:30-10:00
Moderador: ALBERTO SALGUEIRO MOLINARI (RS)

08:30-08:50 Indicação cirúrgica do carcinoma bem diferenciado de tireoide
Palestrante: FERNANDA VAISMAN (RJ) – INCA

08:50-09:10 Indicações cirúrgicas do bócio multinodular
Palestrante: GLAUCIA MARIA FERREIRA DA SILVA MAZETO (SP) – Universidade Estadual Paulista "Júlio de Mesquita Filho" – UNESP

09:10-09:30 Indicações cirúrgicas do hipertireoidismo da Doença de Graves
Palestrante: JOÃO ROBERTO MACIEL MARTINS (MG)

09:30-09:45 Coffee break

09:45-10:05 A tireoidectomia para carcinoma bem diferenciado de tireoide
Palestrante: EVANDRO VASCONCELOS (PR)

10:05-10:25 A cirurgia nas doenças benignas de tireoide
Palestrante: PEDRO HENRIQUE GONÇALVES (RJ)

10:25-11:00 Discussão com os painelistas

10:00-10:30 – INTERVALO
INTERVALO

11:00-12:00 SIMPÓSIO
SIMPÓSIO CIRURGIA ENDÓCRINA – PAINEL – NOVA ERA DA CIRURGIA ENDÓCRINA: TOETVA E RADIOABLAÇÃO NAS PATOLOGIAS CIRÚRGICAS DA TIREOIDE

11:00-12:30
Moderador: MÁRIO VAISMAN (RJ)

11:00-11:20 Outras abordagens nas cirurgias de tireoide (TOETVA/TORTVA)
Palestrante: LEONARDO GUIMARÃES RANGEL (RJ)

11:20-11:40 As indicações de radioablação nas patologias da tireoide
Palestrante: ERIVELTO MARTINHO VOLPI (SP)

11:40-12:00 Discussão com os painelistas

12:40-13:30 – INTERVALO
ALMOÇO

13:30-15:00 PAINEL
MANEJO DO CDT AVANÇADO: EVIDÊNCIAS, DESAFIOS E CAMINHOS TERAPÊUTICOS

13:30-15:00
Moderador: FERNANDA VAISMAN (RJ) – INCA

13:30-15:00
Moderador: GLAUCIA MARIA FERREIRA DA SILVA MAZETO (SP) – Universidade Estadual Paulista “Júlio de Mesquita Filho” – UNESP

13:30-13:45 Desafios do diagnóstico molecular
Palestrante: SUSAN CHOW LINDSEY (SP) – EPM – Unifesp

13:45-14:00 Quando operar (e quando não operar)
Palestrante: MICHELLE AZEVEDO GOMES (RJ)

14:00-14:15 Tratamento neoadjuvante: quando e para quem
Palestrante: HELTON ESTRELA RAMOS (BA) – UFBA

14:15-14:30 Terapia sistêmica: o que já conhecemos e o que há de novo
Palestrante: ANA LUIZA SILVA MAIA (RS)

14:30-15:00 Perguntas e Respostas

WINDSOR BARRA HOTEL: SALA SÃO CONRADO (WINDSOR BARRA HOTEL)

08:30-10:00 PAINEL
E COMO FICA O OSSO?

08:30-10:00
Moderador: MIGUEL MADEIRA (RJ) – UFRJ

08:30-10:00
Moderador: CATARINA BRASIL D´ALVA (CE) – Universidade Federal do Ceará UFC

08:30-08:45 Com os agonistas do GLP-1
Palestrante: MARIA CAROLINE ALVES COELHO AMARAL (RJ)

08:45-09:00 Após a cirurgia bariátrica
Palestrante: NARRIANE CHAVES PEREIRA DE HOLANDA (PB)

09:00-09:15 Com as dietas veganas e dietas hiperproteicas
Palestrante: MAYRA MACENA GOMES (SP)

09:15-09:30 No bloqueio puberal
Palestrante: JULIENNE ÂNGELA RAMIRES DE CARVALHO (PR) – UFPR

09:30-10:00 Perguntas e Respostas

10:00-10:30 – INTERVALO
INTERVALO

10:00-12:10 PAINEL
O ABC DA DENSITOMETRIA PARA O ENDOCRINOLOGISTA

10:00-12:10
Moderador: CAROLINA AGUIAR MOREIRA (PR) – UFPR

10:00-12:10
Moderador: JOÃO LINDOLFO CUNHA BORGES (DF) – SBEMDF

10:00-10:20 Densitometria óssea para o endocrinologista: do uso clínico à área de atuação
Palestrante: MIGUEL MADEIRA (RJ) – UFRJ

10:20-10:40 Interpretação do exame de composição corporal em tempos incretinomiméticos
Palestrante: HENRIQUE PIEROTTI ARANTES (SP)

10:40-11:00 Onde está o erro nessa densitometria? Perguntas e Respostas
Palestrante: CYNTHIA MARIA ALVARES BRANDÃO (SP)

11:00-11:20 Como avaliar a densitometria óssea durante a perda de peso pós-cirurgia bariátrica?
Palestrante: FRANCISCO DE ASSIS PEREIRA (SE)

11:20-11:40 Explorando outras aplicações da densitometria óssea: TBS, VAT, VFA etc.
Palestrante: MARIA CAROLINE ALVES COELHO AMARAL (RJ)

11:40-12:10 Perguntas e Respostas

12:40-13:30 – INTERVALO
ALMOÇO

12:40-13:30

13:30-15:00 PAINEL
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13:30-15:00
Moderador: ANDREA MESSIAS BRITTO FIORETTI (SP) – Unifesp

13:30-15:00
Moderador: MARCOS MORAES DE OLIVEIRA

13:30-13:45 Whey/aminoácidos essenciais/BCAA
Palestrante: ROBERTO LUIS ZAGURY (RJ)

13:45-14:00 Bicarbonato, beta-alanina e nitratos
Palestrante: FERNANDO REIS GONÇALVES (RJ)

14:00-14:15 Termogênicos
Palestrante: FERNANDA BRUNACCI (SP)

14:15-14:30 HMB e creatina
Palestrante: HELOISA THEODORO (RS)

14:30-15:00 Perguntas e Respostas


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CBE 2026 RIO DE JANEIRO

37° CONGRESSO BRASILEIRO DE ENDOCRINOLOGIA E METABOLOGIA

SBEM

AWARD-WINNING ORAL PRESENTATIONS



BASIC/TRANSLATIONAL

ADRENAL AND HYPERTENSION

01 (2886) TOTAL RNA SEQUENCING REVEALS AN EXPANDED LANDSCAPE OF GENE FUSION COMPLEXITY IN PHEOCHROMOCYTOMAS AND PARAGANGLIOMAS

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Introduction: Pheochromocytomas and paragangliomas (PPGLs) are neuroendocrine neoplasms from the adrenal medulla or sympathetic/parasympathetic ganglia, respectively. Although some genotypes are associated with higher metastatic risk (SDHB gene and MAML3 fusions), there are few predictors of metastatic progression. **Objective:** To characterize a cohort of PPGLs through transcriptomic analysis, with particular focus on fusion events. **Methods:** Tumor RNAs from frozen samples of 44 PPGLs from 43 patients (33 pheochromocytoma and 10 paraganglioma; 15 metastatic) were subjected to RNA sequencing (Illumina). Genotypes included: SDHB (n = 6), NF1 (n = 6), RET (n = 5); HRAS, EPAS1, VHL and H3F3A (n = 3 each); and SDHD, MAX, and FGFR1 (n = 1 each). Twelve PPGLs had unknown genetic status. Our dataset was integrated with two independent PPGL datasets for comparative cluster analysis (n = 242). Fusion genes were confirmed by RT-PCR. **Results:** RNA sequencing identified 5 gene fusions (11.6%), including a novel DLG5::RET in-frame fusion gene in one patient with unknown genotype; this sample clustered within the kinase signaling group, and with a RET::GRB2 fusion sample, supporting its pathogenic role. MAML3 fusions were identified in four patients (TCF4::MAML3 and UBTF::MAML3 (n = 2 each)). Unsupervised clustering allowed a good discrimination of samples based on genotype, including metastatic PPGLs with previously known drivers (5 SDHB, 3 NF1, 2 MAML3, SDHD, HRAS, H3F3A and RET), that clustered by genotype rather than by metastatic behavior. TCF4 tumors were part of a distinct cluster, separate from UBTF tumors, and co-clustered with MAX-and H3F3A-related tumors. This pattern was reproducible when integrating data from external cohorts: our TCF4::MAML3 samples co-clustered with other available TCF4 tumors (n = 2), whereas UBTF::MAML3 samples co-clustered with other UBTF::MAML3 PPGLs. Both PPGLs with TCF4::MAML3 fusions were metastatic, whereas those with UBTF::MAML3 have remained non-metastatic to date. All MAML3 fusion PPGLs were noradrenergic (n = 4). **Conclusion:** We identified 2 cases of the rare TCF4::MAML3 fusion and a novel DLG5::RET fusion. TCF4::MAML3 tumors displayed a distinct profile from UBTF::MAML3, and were associated with metastatic disease, supporting the existence of biologically distinct MAML3-rearranged subgroups and suggesting that TCF4::MAML3 defines a higher-risk subtype within PPGLs. Funding: Fapesp (2019/15873-6, 25/24129-0, 2025/00196-0); CNPq(304091/2021-9); NIH-NCI CA264248.
Keywords: pheochromocytoma; fusion gene; metastasis.

THYROID

02 (3009) IRGAROL AS A POTENTIAL THYROID ENDOCRINE DISRUPTOR: EVIDENCE FROM HUMAN TUMORAL AND NON-TUMORAL CELL LINES

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Introduction: Irgarol is an herbicide widely used as a booster biocide in antifouling paints for ship hulls and submerged structures. It was introduced as a substitute for organotin compounds, such as tributyltin (TBT), following their global ban due to high endocrine toxicity and environmental persistence. Despite its intended use, Irgarol has significant environmental persistence, with an estimated half-life of 100 to 350 days in seawater, leading to its widespread detection in coastal and port regions worldwide. Emerging evidence indicates that Irgarol may act as an endocrine-disrupting compound (EDC) for non-target organisms. Toxicological studies have shown that exposure disrupts homeostasis in fish, inducing metabolic disorders and oxidative stress. However, direct effects on mammals, particularly on thyroid cells, remain poorly characterized. **Objective:** This study aimed to investigate the effects of Irgarol on human thyroid normal and tumoral cells, assessing viability, migratory behavior, and metabolic profile. **Methods:** The human thyroid cell lines TPC-1 (papillary carcinoma, RET/PTC1 rearrangement), BCPAP (BRAF-mutated papillary carcinoma), and Nthy-ori 3-1 (immortalized follicular epithelium) were exposed to environmentally relevant concentrations of Irgarol (0.1, 1, and 10 µg/L) for 72 hours. Cell viability was assessed using the PrestoBlue assay, followed by total nuclei quantification via Hoechst staining. Migratory behavior was evaluated through a wound healing assay. Metabolic profile and ATP production rates were determined via Seahorse XF analysis, measuring oxygen consumption rate (OCR) and extracellular acidification rate (ECAR). **Results:** Exposure to Irgarol for 72 hours altered viability, proliferation, and migration across all tested cell lines, with effects varying by lineage and concentration and following non-monotonic dose-response curves. Irgarol reduced cell viability in all models, while wound healing assays revealed delayed migration. Seahorse XF analysis demonstrated a shift in ATP production toward glycolysis, suggesting mitochondrial impairment. **Conclusion:** These findings suggest that Irgarol disrupts key cellular processes in thyroid cells, with differential effects across tumoral and non-tumoral lineages. At environmentally relevant concentrations, these results support the potential classification of Irgarol as a thyroid EDC and highlight the need for further mechanistic investigation.
Keywords: endocrine disruptor; thyroid; irgarol.

NEUROENDOCRINOLOGY

03 (3046) A RETROELEMENT–IMMUNE AXIS DEFINES A DISTINCT IMMUNOBIOLOGICAL PHENOTYPE IN SOMATOTROPH TUMORS

Rafael Loch Batista[1], Flora Ladeira Craveiro[1], Christiane Gruetzmacher[1], Raquel Soares Jallad[1], Nathália da Roz Alessandre[2], Gabriela D. A. Guardia[2], Juliana Moreira Marques[1], Iberê Cauduro Soares[1], Ericka Barbosa Trarbach[1], Pedro Alexandre Favoretto Galante[2]

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Introduction: The mechanisms shaping the immune microenvironment of somatotroph tumors remain poorly understood. LINE-1 retroelements have emerged as endogenous sources of immune activation in several cancers, but their relevance to pituitary tumor biology has not been established. **Objective:** To investigate whether LINE-1 expression is associated with immune infiltration and defines a distinct immunobiological phenotype in somatotroph tumors. **Methods:** We profiled 140 pituitary neuroendocrine tumors using tissue microarray immunohistochemistry for LINE-1, CD8+ tumor-infiltrating lymphocytes, and immune-regulatory markers (PD-L1, HHLA2, and B7-H3). Associations were analyzed according to lineage, hormonal status, and clinicopathological features. Key findings were independently assessed in 43 clinically annotated functioning somatotroph tumors. RNA-seq analysis in a subset of tumors and interrogation of public pituitary tumor transcriptomic datasets were used to explore whether this phenotype was reflected at the transcriptomic level. **Results:** Somatotroph tumors showed a distinctive immunobiological profile compared with other pituitary tumor subtypes. CD8+ T-cell infiltration was higher in PIT1-lineage tumors than in SF1- or TPIT-lineage tumors (median 3.6 vs. 0.6 vs. 0.8 cells/HPF; $p < 0.0001$) and in somatotroph than in corticotroph tumors (4.0 vs. 0.8 cells/HPF; $p = 0.0004$). LINE-1 expression followed a similar pattern, with higher expression in PIT1-lineage tumors ($p = 0.04$), functioning PIT1-lineage tumors ($p = 0.01$), and somatotroph compared with corticotroph tumors ($p = 0.008$). Across the cohort, LINE-1 expression correlated positively with CD8+ T-cell density and with B7-H3 and HHLA2 expression. Integrated profiling identified a CD8-high/LINE1-high phenotype enriched in acromegaly, whereas SF1-lineage tumors were predominantly CD8-low/LINE1-low. This LINE-1/CD8 co-enriched phenotype was recapitulated in the independent somatotroph cohort. In-house RNA-seq and public transcriptomic datasets further supported higher LINE-1 transcriptional activity in somatotroph tumors. **Conclusion:** LINE-1 expression and CD8+ T-cell infiltration converge to define a reproducible immune-associated phenotype enriched in somatotroph tumors. These findings reveal a previously unexplored link between retroelement biology and the pituitary tumor immune microenvironment, supporting a biologically grounded axis for lineage-informed stratification in acromegaly.
Keywords: neuroendocrinology; retroelement; tumor microenviroment.

ADRENAL AND HYPERTENSION

04 (3105) SOMATIC MUTATIONS AS PREDICTORS OF RECURRENCE IN TP53 P.R337H-ASSOCIATED ADRENOCORTICAL TUMORS: A PEDIATRIC-ADULT COMPARATIVE STUDY

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Introduction: Adrenocortical tumors associated with the TP53 p.R337H germline pathogenic variant (GPV) are highly prevalent in Brazil and show variable outcomes between children and adults. The biological basis for these age-related differences remains unclear. **Objective:** We hypothesized that the gradual accumulation of somatic pathogenic variants (SPVs), particularly in TP53, could contribute to differences in tumor aggressiveness. **Methods:** We conducted an analytical cohort study including 70 patients stratified into four groups: Adult p.R337H (n=17), Adult wild-type (WT) (n = 26), Pediatric p.R337H (n = 21), and Pediatric WT (n = 6). Tumor DNA from fresh or formalin-fixed, paraffin-embedded samples was analyzed by PCR and Sanger sequencing of TP53 (exons 2–11) and CTNNB1 (exon 3). Overall survival (OS) and recurrence-free survival (RFS) were analyzed using Kaplan-Meier curves and multivariable Cox models including age and disease stage. **Results:** Age at diagnosis appeared to influence prognosis, with each additional year linked to a 5% increase in risk of death (HR = 1.05, $p < 0.005$) and 6% increase in risk of recurrence (HR = 1.06, $p < 0.005$). Interestingly, pediatric patients tended to experience better OS and RFS than adults ($p < 0.001$). Adults mostly presented with advanced disease (60% ENSAT III; 40% ENSAT IV), with a mean RFS of 5.2 months. TP53 SPVs were observed only in adults, occurring in 35.3% of p.R337H carriers and 26.9% of WT patients, and their presence correlated with higher recurrence risk (HR = 2.68, $p = 0.02$). Variants included missense (p.Cys135Ser, p.Leu348Pro, p.Val147Leu), nonsense (p.Val172Ile), and splice-site (p.Thr125) mutations. They were located in DNA-binding and tetramerization domains and, when coupled with LOH, were often found in more aggressive tumors. CTNNB1 mutations (p.133T>C, p.110C>G) were mainly observed in pediatric p.R337H carriers. No germline or somatic mutations were detected in pediatric WT patients, although the small sample size (n=6) limits firm conclusions. **Conclusion:** Clinical behavior of p.R337H-associated adrenocortical tumors appears not to depend solely on germline status but may be influenced by somatic alterations. TP53 SPVs identified a subgroup with higher recurrence risk and could serve as potential biomarkers for risk stratification. Incorporating molecular profiling into clinical practice may help refine prognosis and guide individualized therapy.
Keywords: adrenocortical carcinoma; TP53; CTNNB1.

CLINICAL

OBESITY

01 (2896) IS THERE A METABOLIC DYSFUNCTION-ASSOCIATED FATTY KIDNEY DISEASE (MAFKD) IN INDIVIDUALS WITH GRADE 1 OBESITY?

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Introduction: Obesity leads to ectopic fat deposition in non-adipose tissues, including the kidneys. Renal steatosis may trigger local inflammation, oxidative stress, and renin-angiotensin system activation, potentially contributing to renal impairment even in early stages of the disease. **Objective:** To assess intra-renal lipid (IRL) content by proton magnetic resonance spectroscopy (1H-MRS) in non-diabetic adults with grade I obesity and to investigate its association with estimated glomerular filtration rate (eGFR) and urinary alb-umin-to-creatinine ratio (ACR). **Methods:** Cross-sectional observational study with two groups matched by sex and age: eutrophic controls (C, n = 34) and grade I obesity (O, n = 34). Anthropometric parameters, biochemical tests, and body composition by dual-energy X-ray absorptiometry were evaluated. IRL was measured by 1H-MRS (single-voxel, left renal cortex). Renal function was assessed by eGFR (CKD-EPI) and ACR. Spearman correlation was used; significance set at $p < 0.05$. **Results:** The O group showed significantly higher body mass index (31.94 [30.61–33.53] vs. 22.24 [21.11–22.77] kg/m², $p < 0.001$), visceral adipose tissue (1072.0 [733.5–1390.0] vs. 296.0 [124.75–491.5] g, $p < 0.001$), fasting glucose (94.5 ± 6.22 vs. 89.76 ± 4.91 mg/dL, $p = 0.001$), and HOMA-IR (3.60 [2.55–4.80] vs. 1.65 [1.48–2.32], $p = 0.0001$). IRL did not differ significantly between groups (0.439 [0.319–0.972] vs. 0.298 [0.204–0.561], $p = 0.228$). However, in the total sample, IRL correlated negatively with eGFR ($r = -0.27$, $p = 0.034$) and positively with ACR ($r = 0.35$, $p = 0.031$). In the obesity group, the correlation between IRL and ACR was stronger ($r = 0.56$, $p = 0.024$). **Conclusion:** Despite no significant difference in IRL between groups, intra-renal lipid content negatively correlates with eGFR and positively correlates with ACR, with a stronger association observed in the obesity group. These findings suggest that IRL may be an early marker of renal dysfunction in obesity, encouraging further longitudinal *studies.*

Keywords: renal steatosis; obesity; magnetic resonance spectroscopy.

ADRENAL AND HYPERTENSION

02 (3134) CLINICOPATHOLOGICAL AND MOLECULAR PROFILE OF BRAZILIAN PATIENTS WITH CUSHING SYNDROME DUE TO PRIMARY PIGMENTED MICRONODULAR ADRENAL HYPERPLASIA

Isabela Nardo[1], Luís Eduardo Cruvinel Pinto[1], Mariana Peduti Halah[1], Renata Danielle Sicchieri Pugliesi[1], Wendy Turatti[1], Marcelo Pinho Bittar[1], Lívia Mara Mermejo[1], Ayrton Custodio Moreira[1], Margaret de Castro[1], Sonir Roberto Rauber Antonini[1]

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Objective: To describe the clinicopathological and molecular characteristics of Brazilian patients with pigmented nodular adrenocortical disease (PPNAD) with or without Carney complex (CNC), a population yet not reported in the literature. **Methods:** Single-center cohort of patients with Cushing syndrome caused by histopathologically confirmed PPNAD. Molecular strategy: PRKAR1A gene sequencing by Sanger followed by whole-exome sequencing (WES). Clinicopathological features were compared using Welch's test and proportions by Fisher's test. **Results:** 85.7% of the 14 patients had CNC, and 28.6% had a family history of PPNAD or CNC. Most were women (64.3%), with a median symptom-onset age of 9.7 years (range: 2–29.8). The median age at diagnosis was 14.9 years (range: 3.3–67.1), and the median symptom duration was 2.8 years (range: 0.3–37.3). Weight gain (50%), lentiginosis (21.4%), and short stature (14.2%) were the most frequent complaints. Following the diagnosis, in CNC patients, lentiginosis was found in 50%, breast nodules in 43%, and cardiac myxomas in 29%. On adrenal imaging (n= 12), 66.7% had hyperplasia or nodules, 50% had both, and 2 patients had normal imaging. Median baseline cortisol was 23.4 µg/dL (7.7–26, n = 10). ACTH was suppressed (<5 pg/mL) in 66,7% and between 5-20 pg/mL in 33,3% (n = 12). Median 23 h salivary cortisol level was 840 ng/dL (220–2480, n = 13) and median morning blood cortisol after 1 mg overnight dexamethasone was 21 µg/dL (2.1–31.4, n = 11). Most patients were treated by bilateral adrenalectomy (78.6%). Of the three patients submitted to unilateral adrenalectomy, one had a later contralateral adrenalectomy, and one developed cyclical Cushing's. Pathogenic PRKAR1A variants were identified in 9 patients (64.3%): in eight by Sanger and in one only by WES. After detailed WES analysis, no variants in the PDE11A or PDE8B genes were found in the remaining six patients. Comparing patients with or without PRKAR1A variants there were no differences in age of onset (MD –8.9 [95% CI: –18.3; 0.4], $p = 0.06$), baseline cortisol (MD 2.7 µg/dL [95% CI: –6.4; 11.9], $p = 0.5$), post 1 mg dexamethasone cortisol (MD 5.3 µg/dL [95% CI: –13.3; 23.9], $p = 0.51$) or ACTH (MD 1.1 pg/mL [95% CI: –8.1; 10.4], $p = 0.79$). **Conclusion:** PRKAR1A variants, but not PDE11A or PDE8B variants, were present in 64.3% of Brazilian PPNAD patients, and no clinicopathological features distinguished mutated from wild-type PRKAR1A. Only bilateral adrenalectomy eliminates the hypercortisolism.

Keywords: PPNAD; Cushing syndrome; molecular analysis.

NEUROENDOCRINOLOGY

03 (3200) EXON 3 DELETION GROWTH HORMONE RECEPTOR POLYMORPHISM MODULATES FASTING GLYCEMIA IN CHILDHOOD ONSET GH DEFICIENCY ADULT PATIENTS ON CONTINUOUS GROWTH HORMONE REPLACEMENT

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Introduction: The polymorphism involving the growth hormone receptor exon 3 deletion (d3-GHR) has been associated with increased longevity, lean body composition, and greater linear growth. **Objective:** To evaluate d3-GHR impact on glycemic homeostasis in Childhood Onset GH Deficiency Adult Patients (CO-GHD), comparing those who maintained GH replacement therapy (GHRT-C) during the transition period into adult life with those who discontinued treatment after puberty (GHRT-I) and with paired healthy controls. **Materials and methods:** We evaluated 60 individuals, including 30 with CO-GHD (two groups: 15 GHRT-C and 15 GHRT-I). Each group was paired with 15 control individuals matched for age, sex, and BMI. GHR was genotyped by multiplex PCR, and allele frequencies were tested for Hardy-Weinberg equilibrium. Fasting blood glucose and glycated hemoglobin were collected, and the euglycemic-hyperinsulinemic clamp was performed to evaluate insulin sensitivity by calculating the metabolic value (M-value). A two-way ANOVA was used to test the interaction between genotype and groups. Patients and controls were grouped according to the presence or absence of the d3 allele (dominant model). Data normality was assessed using the Shapiro-Wilk test. For post-hoc analyses after ANOVA, Tukey's test was used, and, when applicable, the Kruskal-Wallis test was used for non-parametric data. **Results:** Full length (fl-GHR) and d3-GHR genotypic frequencies followed Hardy-Weinberg equilibrium (fl/fl = 29; fl/d3 = 26; d3/d3 = 5; p = 0.970). A significant interaction was observed within group and genotype for fasting blood glucose (p = 0.004). GHRT-C carrying at least one d3-GHR allele showed significantly lower glycemia than homozygous fl/fl GHRT-C patients (p = 0.013). The impact of the d3 allele in the treatment group showed a high effect size (Cohen's d = –2.01). In the assessment of insulin sensitivity, d3-GHR did not influence insulin sensitivity, as shown by similar M-values (p = 0.102), HOMA-IR (p = 0.254), and glycated hemoglobin (p = 0.213). ANCOVA models confirmed that age at diagnosis (p = 0.234) and duration of GHRT use (p = 0.731) did not interfere with fasting blood glucose. **Conclusion:** The d3-GHR polymorphism influences glycemic homeostasis in patients with CO-GHD on GHRT and is associated with lower fasting glycemia. Peripheral insulin sensitivity was not associated with d3-GHR, suggesting that this modulation occurs via other mechanisms, including hepatic glucose production.
Keywords: GHD; GHRT; GHR.

ADRENAL AND HYPERTENSION

04 (3504) ACTH AS A COST-EFFECTIVE MARKER OF DEXAMETHASONE INGESTION IN THE DEXAMETHASONE SUPPRESSION TEST: PRELIMINARY EVIDENCE CHALLENGING CURRENT PRACTICE WITH IMPLICATIONS FOR RESOURCE-LIMITED SETTINGS

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Introduction: Accurate interpretation of the 1-mg dexamethasone suppression test (DST) in adrenal incidentalomas is frequently limited by uncertainty regarding dexamethasone exposure. While serum dexamethasone measurement is used to confirm test validity, its limited availability and high cost restrict widespread use. A simpler, accessible biomarker is needed to ensure reliable DST interpretation. **Objective:** To determine whether post-DST plasma ACTH can reliably reflect dexamethasone exposure and serve as a surrogate for serum dexamethasone measurement in mild autonomous cortisol secretion (MACS). **Methods:** We conducted an observational study including retrospective and prospective cohorts of adults with adrenal incidentalomas at a tertiary referral center. A total of 233 patients were enrolled. Basal and post–1-mg DST cortisol and ACTH were measured alongside serum dexamethasone levels. Diagnostic performance was assessed using ROC curve analysis, sensitivity, specificity, accuracy, and the Youden index. Correlation analyses between ACTH and dexamethasone levels were performed. **Results:** Among 233 patients with adrenal incidentalomas, the median age was 62 (±12,5) years; BMI 29,7 (±5,2) kg/m²; 73.8% women. Mean basal ACTH was 18 ± 9,8 pg/mL and decreased to 5,3 ± 9,8 pg/mL after DST (p < 0.001). A post-DST ACTH cutoff ≤ 4,3 pg/mL predicted serum dexamethasone levels > 130 ng/dL in individuals with positive post-dexamethasone cortisol levels, considered sufficient for valid test interpretation, with sensitivity 78.3%, specificity 66,7%, accuracy 66.3% and a high positive predictive value of 95,2%, according to statistical analysis, with a Youden index of 0,449. **Conclusion:** Post-DST ACTH correlated significantly with serum dexamethasone levels, supporting its role as an indirect marker of adequate drug ingestion. It provides a simple, scalable, and low-cost alternative to serum dexamethasone measurement for validating the DST. This approach may enable broader, more equitable access to accurate ACS diagnosis, particularly in resource-constrained healthcare systems.
Keywords: dexamethasone suppression test; hypercortisolism; ACTH.



ORAL PRESENTATIONS

ADRENAL AND HYPERTENSION

01 (3528) CORTISOL COSECRETION IN PRIMARY ALDOSTERONISM: A PHENOMENON INDEPENDENT OF ADRENAL NODULAR DISEASE?

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Introduction: Up to 30% of patients with primary aldosteronism (PA) exhibit mild autonomous cortisol secretion (MACS), potentially contributing to increased cardiovascular and osteometabolic morbidity. Current guidelines recommend the overnight 1-mg dexamethasone suppression test (DST) only for PA patients with adrenal nodules, leaving it unclear whether cosecretion may also occur in patients without a visible nodular lesion. **Objective:** To describe the prevalence of MACS in patients with PA and its distribution according to adrenal computed tomography (CT) findings, hormonal markers, and clinical parameters. **Methods:** Descriptive cross-sectional study of patients with PA from two tertiary centers who had an available DST and no use of interfering drugs. MACS was defined as post-DST cortisol > 1.8 µg/dL. CT findings were classified as adenomas (unilateral or bilateral) or adrenals without nodules. Groups were compared using the Mann-Whitney U test and Spearman correlation. **Results:** Fifty-one patients were included (age 60.7 ± 10.8 years; 72.5% female; BMI 30.0 ± 6.7 kg/m²). Screening indications for PA were: resistant hypertension (n = 18, 35.3%), hypertension with hypokalemia (n = 18, 35.3%), adrenal incidentaloma (n = 14, 27.5%), and hypertension with obstructive sleep apnea (n = 1, 2.0%). MACS prevalence was 43.1% (n = 22). Basal ACTH was significantly lower in the PA+MACS group (n = 22) than in isolated PA (n = 14 with available ACTH): 9.11 ± 5.20 *vs.* 20.19 ± 9.93 pg/mL (p < 0.001). Within the MACS group, 19 (86.4%) had a nodular lesion: 16 (72.7%) unilateral adenomas and 3 (13.6%) bilateral nodules; mean size 21.4 ± 9.1 mm (11–39 mm). Three patients (13.6%) had adrenals without nodules — two with normal adrenals and one with diffuse bilateral adrenal thickening. Post-DST cortisol did not differ between patients with and without nodular disease (4.76 ± 3.47 *vs.* 2.39 ± 0.41 µg/dL; p = 0.270), nor did lesion size correlate with post-DST cortisol (p = 0.199; p = 0.443). Patients without nodules had basal ACTH < 10 pg/mL and DHEAS < 70 µg/dL; adrenal venous sampling was performed in one of them, and aldosterone normalization by metanephrines confirmed bilateral aldosterone production. **Conclusion:** PA and MACS may coexist even without nodular lesions (13.6% of this cohort), suggesting that cosecretion screening may be relevant in this group, contrary to current guidelines and highlighting a gap in the management of this population.

Keywords: primary aldosteronism; cortisol cosecretion; adrenal computed tomography.

ADRENAL AND HYPERTENSION

02 (2905) GENETIC INVESTIGATION OF TERT PROMOTER AND ATRX VARIANTS IN PHEOCHROMOCYTOMAS AND PARAGANGLIOMAS

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Introduction: Pheochromocytomas and Paragangliomas (PPGLs) are neuroendocrine neoplasms with marked clinical heterogeneity and a strong genetic basis, with up to 40% harboring germline variants. Approximately 15%–20% are metastatic, especially tumors related to Succinate Dehydrogenase Complex Subunit B (SDHB). However, it remains difficult to discriminate between tumors with high or low metastatic risk. Large tumor size (≥5 cm), extra-adrenal location, and biochemical profile are established risk factors. Alterations in telomerase reverse transcriptase (TERT) and the ATRX chromatin remodeler (ATRX), involved in telomere maintenance mechanisms, are some of the new prognostic markers associated with metastasis in PPGLs. **Objective:** To investigate somatic pathogenic variants (PVs) in the TERT promoter and in the ATRX gene in a Brazilian cohort of PPGLs, which displays a distinct genetic profile with a higher prevalence of SDHB-related tumors. We also evaluated their association with metastatic disease and with clinical, radiological, and pathological features. **Methods:** The TERT promoter was evaluated by Sanger sequencing or somatic whole-exome sequencing (WES) in a cohort of 79 patients with PPGLs (53 non-metastatic and 26 metastatic), comprising 81 tumors, while ATRX was evaluated by WES in 26 patients from this cohort. **Results:** Germline PVs in Cluster 1A genes were identified in 28 of 79 patients (35.4%), including 20 patients harboring PVs in SDHB (25.3%). Somatic PVs in the TERT promoter (c.-124C>T; C228T) were identified in two paragangliomas (2.5% of the cohort), a frequency lower than that reported in international cohorts. Both tumors were metastatic, SDHB-related, and noradrenergic, with sizes of 13.5 cm and 8 cm; one of them also harbored a concomitant germline TP53 PV (c.1010G>A/p.R337H). Among metastatic patients with germline SDHB PVs, the frequency of somatic TERT promoter PVs was 16.7%. Three of the 26 patients screened for ATRX (11.5%) harbored somatic variants in this gene, all classified as benign or likely benign, including two from metastatic paragangliomas. **Conclusion:** Our study expands the understanding of telomere maintenance mechanisms in PPGLs within a Brazilian population enriched for SDHB alterations. TERT promoter variants were associated with aggressive tumor features, such as extra-adrenal location, germline SDHB PVs, and metastatic disease.

Keywords: paraganglioma; telomerase; telomere maintenance.

ADRENAL AND HYPERTENSION

03 (2898) PATIENT EDUCATION ON GLUCOCORTICOID DOSE ADJUSTMENT IN STRESSFUL SITUATIONS: A SURVEY OF BRAZILIAN PATIENTS WITH ADRENAL INSUFFICIENCY

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Introduction: Adrenal insufficiency (AI) is a rare condition associated with high morbimortality risk during stress events. Patient education on glucocorticoid (GC) dose adjustment is essential to prevent adrenal crises; however, evidence suggests that a significant proportion of patients remain inadequately trained. **Objective:** To assess the level of education and orientation of AI patients regarding GC dose adjustment in physical stress situations. **Methods:** Cross-sectional observational study using an online questionnaire distributed to members of a national Brazilian adrenal patient association. Adults (≥18 years) with confirmed AI (primary or central) were included. Responses were obtained from all five Brazilian geographic regions. Participants were assessed on sociodemographic data, disease characteristics, and knowledge/behavior in three stress scenarios: (1) tartar removal, (2) gastroenteritis with vomiting, and (3) fever > 38 °C. Responses were classified as correct, incorrect without life risk, or incorrect with life risk. Chi-square tests assessed associations between education level and correct answers ($p < 0.05$). The study was approved by the Institutional Review Board. **Results:** A total of 278 participants responded (85.9% female; mean age 45.5 ± 12.4 years; 64.3% with higher education). Mean age at AI diagnosis was 35.2 ± 15.4 years. Autoimmune disease (30.2%) and pituitary disease (26.6%) were the most common etiologies. Only 6.5% answered scenario 1 correctly, while 41.4% gave an incorrect answer with life risk; 50.7% answered scenario 2 correctly; and 60.8% answered scenario 3 correctly. Despite 72.2% reporting orientation on GC dose adjustment since diagnosis, only 18.8% were instructed on self-administration of injectable hydrocortisone at diagnosis, with 61.0% receiving this information only after joining the patient association. 32.5% did not feel confident performing intramuscular GC self-injection. No significant association was found between education level and correct answers ($p > 0.05$). **Conclusion:** Despite most patients reporting prior orientation, knowledge of GC dose adjustment in stressful situations remains critically inadequate, particularly regarding fever management. Patient associations play a pivotal role in supplementing medical education. These findings underscore the urgent need for standardized, structured patient education strategies to reduce adrenal crisis risk.

Keywords: adrenal insufficiency; patient education; adrenal crisis.

DIABETES MELLITUS

04 (3372) CIRCULATING MICRORNA 146-A AND CARDIOVASCULAR RISK IN TYPE 1 DIABETES: A SYSTEMATIC REVIEW

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Introduction: Type 1 diabetes mellitus (T1DM) is associated with premature cardiovascular disease despite intensive glycemic control. MicroRNA-146a (miR-146a) regulates inflammatory and endothelial pathways involved in atherosclerosis and vascular injury. Experimental and clinical evidence suggests that reduced circulating miR-146a levels are associated with oxidative stress, endothelial dysfunction, and cardiovascular complications in diabetes. **Objective:** This systematic review aimed to assess the association between circulating miR-146a expression and cardiovascular outcomes in patients with T1DM. **Methods:** This systematic review, registered in PROSPERO and conducted according to PRISMA guidelines, searched PubMed, Cochrane, Embase, and LILACS using MeSH/Emtree terms related to T1DM, microRNA-146a, and cardiovascular risk. Two independent reviewers performed study selection, data extraction, and methodological quality assessment using the Newcastle-Ottawa Scale. Due to the limited number of eligible studies results were synthesized narratively. **Results:** Two studies were included in the qualitative synthesis. Both evaluated circulating miR-146a expression in T1DM and its association with diabetic complications. The EURODIAB Prospective Complications Study reported significantly lower circulating miR-146a levels in patients with diabetic complications compared with complication-free individuals, with independent association to cardiovascular and microvascular outcomes after adjustment for clinical confounders. The second study demonstrated reduced miR-146a expression in T1DM patients compared with controls, with progressively lower levels according to disease duration. This reduction was accompanied by inflammatory activation and markers of cardiac dysfunction, suggesting involvement in diabetes-related cardiovascular injury. Overall, both studies demonstrated an inverse association between circulating miR-146a levels and diabetic complications, including cardiovascular involvement. **Conclusion:** Available evidence suggests an inverse association between circulating miR-146a levels and cardiovascular complications in T1DM. These findings indicate that miR-146a may be involved in inflammatory and vascular pathways related to diabetic complications. However, the limited number of studies and their observational design preclude definitive conclusions, highlighting the need for further prospective investigations.

Keywords: diabetes mellitus, type 1; MicroRNAs; cardiovascular diseases.

DIABETES MELLITUS

05 (3267) DELAYED RECOVERY AFTER INPATIENT HYPOGLYCEMIA: RETESTING GAPS AND RECURRENT RISK ACROSS 307,634 POINT-OF-CARE GLUCOSE RECORDS

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Introduction: Inpatient hypoglycemia is a relevant patient safety event, but most hospital metrics focus on occurrence rather than timely recovery. **Objective:** To evaluate time to documented glucose recovery after inpatient hypoglycemia in adult units using a large point-of-care glucose database. **Methods:** This retrospective observational study analyzed point-of-care glucose measurements from hospitalized adults between March 2025 and March 2026 in a tertiary academic hospital serving both public and private healthcare systems. Pediatric locations, quality-control samples, duplicates and records without valid patient identification were excluded. Hypoglycemia was defined as glucose < 70 mg/dL. Consecutive low values before recovery were considered part of the same non-overlapping episode. Recovery was the first subsequent glucose ≥ 80 mg/dL within 6 hours. Following electronic clinical quality measure false-positive logic, episodes starting with glucose < 40 mg/dL followed by repeat glucose > 80 mg/dL within 5 minutes were excluded. **Results:** Among 319,574 unique glucose records, 319,548 had valid patient identification. After excluding pediatric locations, 307,634 adult-unit records from 38,635 patients and 126,876 patient-days were analyzed. Overall, 7,687 glucose values were < 70 mg/dL, including 2,489 < 54 mg/dL and 1,098 < 40 mg/dL. After exclusion of 260 potential false-positive severe events, 6,004 non-overlapping hypoglycemic episodes remained in 2,807 patients and 5,227 patient-days. The point-of-care-adapted severe hypoglycemic harm score was 1.20%, within the provisional high-performance range of approximately < 1%–2%. Recovery to ≥ 80 mg/dL within 6 hours occurred in 4,379 episodes (72.9%). Median time to recovery was 54.9 minutes (interquartile range, 33.9–96.8). Recovery occurred within 15, 30, 60 and 120 minutes in 6.2%, 14.9%, 39.9% and 58.1%, respectively. First retesting occurred within 60 minutes in only 53.9% of episodes. Among retested episodes, 20.7% remained < 70 mg/dL and 8.1% remained < 54 mg/dL. Recurrent hypoglycemia after recovery occurred within 6, 12 and 24 hours in 9.4%, 14.3% and 21.7%. **Conclusion:** Despite a favorable severe hypoglycemic harm score, nearly one third of episodes had no documented recovery within 6 hours and almost half had no retest within 60 minutes. These findings reveal a measurable gap between detection and correction, supporting digital surveillance and protocol-based strategies.

Keywords: hypoglycemia; patient safety; hospital care.

DIABETES MELLITUS

06 (3107) EXCESS BODY WEIGHT IS FREQUENT BUT SHOWS DIFFERENT METABOLIC PHENOTYPES IN ADULTS WITH GCK-MODY AND TYPE 1 DIABETES: DATA FROM THE BRAZILIAN MONOGENIC DIABETES STUDY (BRASMOD) AND BRAZILIAN TYPE 1 DIABETES STUDY GROUP (BRAZDIAB1SG)

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Introduction: Overweight (Ow) and obesity (Ob) are increasingly frequent and may complicate the clinical distinction between GCK-MODY (GCK) and type 1 diabetes (T1D). However, metabolic significance of excess body weight in these subtypes remains unclear. **Objective:** To assess the frequency of Overweight (Ow), Obesity (Ob), and associated metabolic factors in adults with GCK and T1D. **Methods:** Individuals aged 18 years or older from two Brazilian multicenter cohorts, BRASMOD for GCK and BrazDiab1SG for T1D, were evaluated. Associations between clinical predictors and BMI were tested using linear regression models in each subtype. Interaction with diabetes type was also tested. Continuous variables were expressed in median [interquartile range] and categorical ones in percentages. **Results:** The analysis included 50 individuals with GCK and 1,457 with T1D. The proportion of females was 72% and 58% ($p = 0.06$), age was 39 [30–46] years and 26 [21-34] years ($p < 0.0001$), and HbA1c was 6.4 [6.2–6.7] and 8.7 [7.6–10.3] ($p < 0.0001$), respectively in GCK and T1D. GCK and T1D showed similar frequencies of Ow (27.3% vs. 23.1%, $p = 0.5$) and Ob (6.8% vs. 6.9%, $p = 1$). BMI was associated with age at diagnosis ($\beta = 0.04$, $p = 0.0003$), total cholesterol ($\beta = 0.006$, $p = 0.017$), HDL-cholesterol ($\beta = -0.17$, $p = 0.019$), and log(triglycerides) ($\beta = 1.27$, $p < 0.0001$) in T1D, but not in GCK. A significant interaction with diabetes type was observed for HDL-cholesterol ($p = 0.05$). **Conclusion:** Data from two Brazilian multicenter cohorts suggest that excess body weight is frequent in adults with both GCK and T1D, present in almost one third of individuals. Nevertheless, metabolic profiles appear to differ between diabetes subtypes. In T1D, Ow/Ob may partly reflect the effects of insulin-associated weight gain and disease heterogeneity on metabolic repercussions such as dyslipidemia. In contrast, even though GCK individuals had higher age (partly because the age group analyzed excluded index cases), the absence of such associations suggests that Ow/Ob in this group may be driven by environmental/genetic factors unrelated to the diabetes type itself. This interpretation is consistent with the typically mild metabolic phenotype of GCK and with previous observations that individuals with GCK generally have a low rate of vascular complications, suggesting that excess body weight in this subgroup may not translate into the same adverse metabolic and vascular effects observed in other forms of diabetes. Funding: Fapesp.

Keywords: type 1 diabetes; glucokinase; obesity.

DIABETES MELLITUS

07 (3118) GLYCEMIC METRICS AND ALBUMINURIA IN ADULTS WITH TYPE 1 DIABETES INITIATING AUTOMATED INSULIN DELIVERY: A SUBANALYSIS OF A PROSPECTIVE OBSERVATIONAL STUDY

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Introduction: Albuminuria is closely linked to glycemic control. Automated insulin delivery (AID) improves glycemia in patients with type 1 diabetes (T1D). However, it is unclear which glycemic metrics are most strongly associated with albuminuria and whether short-term changes in glycemic control influence albuminuria. **Methods:** We conducted a prospective observational study at a tertiary center from July 2023 including adults with T1D who initiated Tandem t:slim X2TM Control-IQ (CIQ) or MiniMedTM 780G (MM780G). Participants without baseline and at least one additional measurement of urinary albumin-to-creatinine ratio (UACR), and those who started/modified renoprotective therapies during follow-up were excluded. Clinical, laboratory, and 30-day CGM data before AID initiation, and at 3 and 6 months were collected. We performed a cross-sectional analysis to evaluate the association between glycemic metrics and log-transformed UACR at baseline, and a longitudinal analysis to assess the association between their variations at 3 and 6 months. Linear regression models unadjusted and adjusted for age and sex were used for both analyses. **Results:** We included 46 adults (aged 38.9 ± 11.3 years; 32.6% male; 6.5% with chronic kidney disease). Baseline average HbA1c was 7.5 ± 0.7%, time-in-range (TIR) 56.8 ± 13.3%, and median UACR was 3.7 [1.9–5.8] mg/g. Forty-eight percent initiated MM780G. Higher UACR was associated with higher time > 250 mg/dL (β = 0.43, 95% CI: 0.07 to 0.80; p = 0.020) and higher coefficient of variation (CV; β = 0.08; 95% CI: 0.02 to 0.14; p = 0.007) in the adjusted model. We found a trend towards higher UACR with higher time > 180 mg/dL (β = 0.02, 95% CI: 0.00 to 0.04; p = 0.059) and lower TIR (β = –0.02, 95% CI: –0.05 to 0.00; p = 0.079) and time-in-tight-range (β = –0.02, 95% CI –0.05 to 0.00; p = 0.081). At 3 months (n = 44), the median change in UACR was 0.0 [–1.4, 2.1] mg/g. This change was positively associated with CV variation (β = 0.10, 95% CI: 0.01 to 0.19; p = 0.032), although the association was attenuated after adjustment (p = 0.222). At 6 months (n = 29), the median variation of UACR was 0.3 [–2.3, 3.5] mg/g, with no significant associations observed between changes in UACR and changes in glycemic metrics. **Conclusion:** In adults with T1D initiating AID, albuminuria showed stronger cross-sectional associations with glycemic variability and marked hyperglycemia. Short-term changes in albuminuria were small, and associations with changes in glycemic metrics were modest.
Keywords: type 1 diabetes; automated insulin delivery; albuminuria.

DIABETES MELLITUS

08 (2930) LATENT AUTOIMMUNE DIABETES IN ADULTS (LADA): CLINICAL AND PHENOTYPIC CHARACTERIZATION IN PATIENTS FROM A TERTIARY HOSPITAL IN SÃO PAULO

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Introduction: Among individuals living with diabetes mellitus, approximately 15%–20% have latent autoimmune diabetes in adults (LADA), a form of type 1 diabetes mellitus that manifests in people usually aged 30 years or older. It is primarily characterized by the presence of positive anti-GAD antibodies and initial independence from insulin for treatment. Late diagnosis and inadequate treatment of LADA can lead to several complications for patients, including coronary artery disease, retinopathy, nephropathy, diabetic polyneuropathy, and, ultimately, even death. Therefore, considering the importance of accurate diagnosis and treatment for a better prognosis in patients with LADA, the limited national database on the prevalence, risk factors, and phenotypic characteristics of these patients represents a significant gap that may result in misdiagnosis, inappropriate treatment, and less effective clinical management. **Objective:** Identify patients with LADA followed at the Endocrinology Outpatient Clinic from a Tertiary Hospital in São Paulo and to analyze the phenotypic profile of this population. **Methods:** This is a cross-sectional observational study with a quantitative approach, aiming to analyze numerical data related to patients with LADA, through the review of medical records of 54 pre-selected patients who meet the following inclusion criteria: patients over 18 years of age, with a confirmed diagnosis of LADA (clinical + antibodies), and with at least 6 months of follow-up at the service. **Results:** Of the 54 patients analyzed, 33 (61%) were female, while 21 (39%) were male. The average age at diagnosis was 57.48 years, with a mean duration of diabetes mellitus of 15.91 years. The presence of positive anti-GAD antibodies was confirmed in 83.3% of the cases. Regarding insulin, patients took an average of 19.19 months from the diagnosis of LADA to become dependent on the hormone for disease management. **Conclusion:** The predominance of middle-aged female patients and the high anti-GAD positivity reinforce the characteristic epidemiological and autoimmune profile of LADA. The rapid progression to insulin dependence highlights the need for early diagnosis and appropriate management. These preliminary findings support the importance of recognizing LADA as a distinct clinical entity within adult diabetes.
Keywords: latent autoimmune diabetes in adults; anti-GAD antibodies; clinical profile.

DIABETES MELLITUS

09 (3509) RELATION BETWEEN DIET COMPONENTS AND THE PROGRESSION OF CHRONIC KIDNEY DISEASE IN PATIENTS WITH TYPE 2 DIABETES: A PROSPECTIVE COHORT STUDY

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Introduction: The prevalence of chronic kidney disease (CKD) is increasing globally, and its main cause is diabetes. The therapeutic arsenal for this disease is also growing, but non-pharmacological measures are essential in its management. Identifying dietary components associated with kidney disease may support dietary interventions in high-risk individuals. **Objective:** To evaluate the association between diet components of individuals with type 2 diabetes and development of CKD. **Methods:** In this prospective study, patients (18–80 years) from the Diabetes Clinic of a university hospital in Porto Alegre were consecutively recruited. Exclusion criteria included body mass index > 40 kg/m², heart failure, gastroparesis, diabetic diarrhea, dietary counseling by a registered dietitian during the previous 12 months, inability to perform weighed diet records (WDR), and CKD at baseline. Patients underwent clinical and nutritional evaluation. Dietary habits were evaluated by 3-day WDR. Compliance with the WDR technique was assessed by comparison of protein intake estimated from the 3-day WDR and from 24-h urinary nitrogen output. The glomerular filtration rate (eGFR) was estimated using the 2009 CKD-EPI equation. **Results:** Among 149 patients with type 2 diabetes (42% M; age: 60 ± 9.4 years), 37 developed CKD after a mean follow-up of 5.4 ± 2.3 years. These patients were older (63 ± 8.8 vs. 59 ± 9.4 years) and had lower eGFR values at baseline (89 ± 16 vs. 96 ± 16 ml/min/1.73 m²) than those who did not develop CKD (all $P < 0.05$). In a multivariate Cox regression analysis, the ingestion of eicosapentaenoic acid (EPA) (HR = 0.48, 95% CI: 0.23–0.98; P = 0.043) and docosahexaenoic acid (DHA) (HR = 0.47, 95% CI: 0.23–0.98; P = 0.045) were inversely associated with CKD development, independent of age, gender and systolic blood pressure. Unexpectedly, dietary intake of saturated fatty acids was also associated with lower risk of CKD development (HR = 0.83, 95% CI: 0.71–0.98; P = 0.03), possibly related to myristic acid content (HR = 0.29, 95% CI: 0.081–1.045; P = 0.058). **Conclusion:** In this population of patients with type 2 diabetes, the ingestion of polyunsaturated fatty acids from marine origin, EPA and DHA, was associated with a lower risk of CKD development. Dietary saturated fatty acids were also negatively associated with the development of this complication, possibly because of myristic acid content. These findings should be further explored in larger studies and other populations.

Keywords: type 2 diabetes; fatty acids; chronic kidney disease.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

10 (3435) CORONARY ARTERY CALCIUM SCORE IN CARDIOVASCULAR RISK STRATIFICATION FOR TYPE 1 DIABETES MELLITUS: A SYSTEMATIC REVIEW AND META-ANALYSIS OF PREVALENCE, PROGNOSTIC VALUE, AND INCREMENTAL VALUE

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Introduction: Cardiovascular disease is the leading cause of morbidity and mortality among patients with type 1 diabetes mellitus (T1DM). The pathophysiology of atherosclerosis in T1DM is characterized by chronic hyperglycemia, endothelial dysfunction, and prolonged metabolic exposure, contributing to accelerated vascular injury and increased cardiovascular risk. Traditional cardiovascular risk scores frequently underestimate risk in this population, while disease-specific models still present limitations in identifying subclinical atherosclerosis. In this context, the coronary artery calcium (CAC) score, obtained through computed tomography, has emerged as a promising tool for early detection of atherosclerosis and refinement of cardiovascular risk assessment. **Objective:** To evaluate the prevalence of coronary artery calcification, its prognostic value, and the incremental value of CAC in cardiovascular risk stratification among adults with T1DM without established cardiovascular disease. **Methods:** A systematic review and meta-analysis were conducted according to PRISMA guidelines. Searches were performed in PubMed, Embase, and Cochrane databases, yielding 222 studies. After duplicate removal and screening by two independent reviewers using Rayyan, 11 studies remained for qualitative synthesis. Observational studies evaluating CAC in adults with T1DM without established cardiovascular disease were included. Outcomes comprised CAC prevalence (CAC > 0), major adverse cardiovascular events (MACE), mortality, and incremental value over conventional cardiovascular risk assessment methods. Methodological quality was assessed using the Newcastle–Ottawa Scale (NOS). **Results:** CAC prevalence in T1DM ranged from 18% to over 60%, increasing according to age and disease duration. Meta-analysis of independent studies demonstrated a pooled prognostic effect estimate (HR/OR) of 4.48 (95% CI: 2.68–7.49; I^2 = 0.00%, p = 0.94). Individual effect estimates ranged from an OR of 3.00 (95% CI: 1.49–6.05) to an HR of 6.91 (95% CI: 3.10–15.40). CAC integration enabled risk reclassification in approximately 48% of patients previously categorized as low risk. **Conclusion:** CAC demonstrates substantial prognostic and incremental value in cardiovascular risk stratification among patients with T1DM. The method improves identification of subclinical atherosclerosis and enables more accurate risk reclassification, supporting refinement of preventive cardiovascular strategies.

Keywords: type 1 diabetes mellitus; coronary artery calcification; risk assessment.

EXERCISE ENDOCRINOLOGY

11 (3552) ASSOCIATION BETWEEN TYPES OF PHYSICAL ACTIVITY AND THE CARDIOMETABOLIC PROFILE OF WOMEN IN PERIMENOPAUSE: A CROSS-SECTIONAL STUDY IN PERNAMBUCO, BRAZIL

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Introduction: A sedentary lifestyle is an important modifiable risk factor for the development of cardiometabolic diseases and is associated with conditions such as insulin resistance, dyslipidemia, and hypertension. Lifestyle changes, particularly regular physical exercise, play a central role in the prevention and management of these conditions. However, different exercise modalities may be associated with distinct effects on the cardiometabolic profile, especially in perimenopausal women, a stage characterized by hormonal and behavioral changes that increase vulnerability to metabolic and cardiovascular outcomes. **Objective:** To analyze the association between different types of physical activity and the cardiometabolic profile of perimenopausal women. **Methods:** A cross-sectional study was conducted involving 1,090 perimenopausal women residing in the state of Pernambuco, Brazil. Participants were interviewed regarding their physical activity practices and underwent anthropometric assessment and blood pressure measurement, in addition to fasting peripheral blood collection for biochemical analysis of glucose, lipid profile, uric acid, and insulin. Comparisons between groups were performed using analysis of variance (ANOVA), with statistical significance set at $p < 0.05$. **Results:** Distinct associations were observed between physical activity modalities and the cardiometabolic markers evaluated. Cycling was associated with the most favorable metabolic profile, with lower BMI, blood glucose, total cholesterol, LDL cholesterol, and triglyceride levels compared with the other evaluated modalities, including walking, soccer, water aerobics, resistance training, and especially capoeira. However, dancing was associated with better cardiovascular and anthropometric parameters, including lower waist circumference and blood pressure levels, as well as higher HDL cholesterol (67 mg/dL). **Conclusion:** Different physical activity modalities showed specific associations with the cardiometabolic profile of perimenopausal women. Cycling was associated with a more favorable metabolic profile, whereas dancing was associated with better cardiovascular parameters and lower central adiposity. These findings suggest that the modality of physical activity may be associated with distinct cardiometabolic benefits, with potential relevance for individualized health promotion strategies in this population.
Keywords: physical activity; cardiometabolic profile; perimenopause.

EXERCISE ENDOCRINOLOGY

12 (3225) BEYOND BINARY CUTOFFS: A PERCENTILE-BASED TOOL FOR HANDGRIP STRENGTH INTERPRETATION

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Introduction: Handgrip strength is an important functional biomarker associated with adverse clinical outcomes, including sarcopenia, frailty, disability, hospitalization, and mortality. Despite its widespread clinical use, interpretation remains predominantly based on fixed binary cutoffs, such as those proposed by EWGSOP2, which may inadequately reflect heterogeneity related to age and sex. Consequently, individuals with muscle strength below that expected for their reference population may not be adequately recognized despite values within traditional thresholds for low muscle strength. **Objective:** To develop a percentile-based tool for handgrip strength interpretation, allowing continuous and individualized assessment according to age and sex. **Methods:** A digital model was developed based on international normative data obtained from 2.4 million adults 20 to 100 years old from 69 countries, including Brazil, stratified by sex and age group. The system uses percentile curves to position patient results relative to the reference population, with integrated graphical visualization for clinical interpretation and longitudinal follow-up. Unlike traditional models, which classify individuals only as “normal” or “reduced strength,” the platform provides continuous interpretation of muscular performance across the percentile spectrum. **Results:** The model enabled automated generation of age- and sex-adjusted percentile curves, with individualized positioning of muscle strength within the expected spectrum for the reference population. The approach enabled more refined characterization of individuals with muscle strength below that expected for sex and age, even without fulfilling traditional criteria for low muscle strength. In addition, visual representation of percentile curves improved clinical interpretability, facilitated communication of results, and allowed more refined assessment of functional behavior throughout aging and longitudinal follow-up. **Conclusion:** The proposed tool represents an innovative approach for muscle strength assessment, allowing more contextualized and individualized interpretation compared with traditional models based exclusively on fixed cutoffs. The use of percentile curves may improve sensitivity of functional assessment, contribute to earlier identification of muscle decline, and refine strategies for screening, longitudinal monitoring, and clinical evaluation of sarcopenia.
Keywords: sarcopenia; handgrip; aging.

EXERCISE ENDOCRINOLOGY

13 (2901) MUSCLE BEATS LIVER: QUALITY OF LIFE IN MASLD IS A MATTER OF STRENGTH, NOT FIBROSIS

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Introduction: Quality of life (QoL) impairment in metabolic dysfunction-associated steatotic liver disease (MASLD) is traditionally attributed to liver disease severity and metabolic comorbidities. However, extrahepatic factors, particularly skeletal muscle function and physical activity, may play a key role in patient-reported outcomes. **Objective:** To identify determinants of QoL in MASLD. **Methods:** In this cross-sectional study, adults with MASLD evaluated at a tertiary center (2014–2026) were included if they had assessments of muscle strength and QoL. QoL was measured using the SF-36 Physical (PCS) and Mental Component Summary (MCS) scores, where lower scores indicate worse QoL. Muscle strength was evaluated using handgrip strength and the 5-times sit-to-stand test. Low strength was defined per EWGSOP2 (2018) criteria. Physical activity was measured using the IPAQ-SF and categorized as active (≥600 MET-min/week) or inactive. Liver stiffness (LSM) was measured by elastography. Mann-Whitney test was used to compare median PCS and MCS between groups. Linear regression models evaluated associations of metabolic traits, liver stiffness, muscle strength, and physical activity with PCS and MCS, adjusting for age, sex, ethnicity, and socioeconomic status. **Results:** A total of 215 patients were included (56% male; mean age 55.4 ± 15.2 years). LSM ≥ 8 kPa (51%), type 2 diabetes (65%), obesity (72%), physical inactivity (35%), and low muscle strength (29%) were prevalent. The physical QoL score (PCS) was lower in patients with diabetes (38.7 vs. 44.8; $p = 0.002$), obesity (40.2 vs. 45.0; $p = 0.002$), low muscle strength (34.0 vs. 43.9; $p < 0.001$), and physical inactivity (34.6 vs. 44.2; $p < 0.001$). The mental QoL score (MCS) was lower in those with low muscle strength (43.5 vs. 49.8; $p = 0.004$) and physical inactivity (45.0 vs. 50.4; $p = 0.011$). In adjusted models, higher BMI ($\beta = -0.41$; $p < 0.001$), diabetes ($\beta = -3.14$; $p = 0.036$), low muscle strength ($\beta = -5.00$; $p = 0.003$), and physical inactivity ($\beta = -4.74$; $p = 0.003$) were independently associated with lower physical QoL, while LSM was not. Notably, low strength was the sole factor independently associated with lower mental QoL ($\beta = -6.62$; $p = 0.002$). **Conclusion:** Although fibrosis and metabolic traits remain central therapeutic targets in MASLD, muscle strength and physical activity are strongly associated with QoL, underscoring the value of functional assessment and strategies to preserve muscle function in patient-centered care. Support: Capes.

Keywords: MASLD; quality of life; muscle strength.

EXERCISE ENDOCRINOLOGY

14 (3561) PHYSICAL ACTIVITY INDEPENDENTLY MODULATES METABOLIC ADAPTATION DURING TESTOSTERONE REPLACEMENT IN MEN WITH CONGENITAL HYPOGONADOTROPIC HYPOGONADISM: A RANDOMIZED LONGITUDINAL STUDY

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Introduction: Congenital hypogonadotropic hypogonadism is a rare disorder associated with chronic androgen deficiency, adverse body composition, and cardiometabolic vulnerability. Determinants of metabolic adaptation during testosterone replacement remain poorly defined. **Objective:** To evaluate whether physical activity modifies longitudinal metabolic adaptation in men with congenital hypogonadotropic hypogonadism undergoing randomized transition to long-acting testosterone therapy. **Materials and methods:** Prospective randomized longitudinal study including 37 adult men with clinical and/or genetic congenital hypogonadotropic hypogonadism previously receiving short-acting testosterone. Participants were randomized to transdermal testosterone gel or intramuscular testosterone undecanoate and followed for 6 months. Physical activity was categorized as sedentary or non-sedentary according to World Health Organization-equivalent thresholds. Anthropometric, bioimpedance-derived body composition, glycemic, lipid, and hormonal parameters were assessed at baseline and follow-up. Longitudinal analyses used age-adjusted linear mixed-effects models with participant-level random intercepts and fixed effects for time, physical activity, treatment modality, and time-by-physical activity-by-treatment interaction. **Results:** Thirty-seven men were included. Physical activity emerged as an independent modifier of metabolic adaptation. Significant age-adjusted time-by-physical activity interactions were observed for waist-to-hip ratio (adjusted β −0.0294; 95% confidence interval −0.0531 to −0.0057; $p = 0.015$), indicating attenuation of central adiposity progression, and triglyceride trajectory (adjusted β −52.6 mg/dL; 95% confidence interval −99.8 to −5.4; $p = 0.029$), indicating clinically meaningful lipid improvement. Favorable numerical trends were observed for visceral adiposity, fat mass, and insulin resistance. No independent metabolic effect of testosterone formulation or significant three-way interaction was observed. **Conclusion:** Physical activity independently modified metabolic adaptation during testosterone replacement, particularly central adiposity and triglyceride response, supporting exercise as a therapeutic modifier in chronic androgen deficiency.

Keywords: congenital hypogonadotropic hypogonadism; physical activity; testosterone replacement therapy.

EXERCISE ENDOCRINOLOGY

15 (2968) TESTOSTERONE USE IN TRANSGENDER MALE ATHLETES: THERAPEUTIC LIMITS, SAFETY MONITORING, AND INTERFACE WITH ANTI-DOPING.

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Introduction: Gender-affirming hormone therapy (GAHT) is used by transgender men to induce masculinization through exogenous testosterone and has become increasingly common among athletic individuals. However, it requires a balance between sports performance and cardiovascular safety, while remaining aligned with clinical efficacy and sports regulatory agencies. **Objective:** To analyze testosterone use in terms of serum levels, as well as to differentiate between its therapeutic use and potential ergogenic advantage in transgender male athletes. **Methods:** A narrative literature review was conducted using the PubMed and LILACS databases (2016–2026), with the descriptors "Transgender Persons," "athletes," "testosterone," and "Monitoring, Physiologic," combined using the Boolean operator AND. Articles with free full text, conducted in humans, and published in English or Portuguese were included. Duplicate studies and those not aligned with the topic after title and abstract screening were excluded. **Results:** The data indicate that the target range for total testosterone to ensure health and performance is between 300 and 1,000 ng/dL. However, although exogenous hormone use may equalize some physical indices, performance parity between transgender and cisgender athletes involves more factors than hormone levels alone, such as mental context, aerobic capacity, and handgrip strength, among others. In sports, this therapy is supported by the Therapeutic Use Exemption (TUE), which confirms clinical necessity and penalizes illicit doping-related practices. One of the main observed risks is erythrocytosis; therefore, hematocrit monitoring is recommended, keeping levels below 50%–52% to reduce the risk of thrombotic events. **Conclusion:** The use of GAHT in transgender athletes should be approached cautiously and individually, as institutions strictly enforce therapeutic limits to prevent physiological advantage and comply with anti-doping regulations. Therefore, clinical follow-up is essential, with emphasis on monitoring serum testosterone and hematocrit levels to ensure patient safety and support athletic performance.

Keywords: testosterone; transgender athletes; physiological monitoring.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

16 (3437) COMBINED INTERVENTIONS IN THE MENTAL HEALTH OF WOMEN WITH POLYCYSTIC OVARY SYNDROME: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Polycystic Ovary syndrome (PCOS) is an endocrinopathy associated with substantial psychological burden, including anxiety, depression, and impaired quality of life in women of reproductive age. Due to the multifactorial nature of mental health outcomes in PCOS, combined interventions integrating lifestyle, pharmacological, and psychosocial approaches have emerged as promising therapeutic strategies. This study aimed to evaluate the impact of combined interventions on mental health outcomes in women with PCOS. **Methods:** This systematic review and meta-analysis followed the PRISMA and Cochrane Handbook guidelines and registered in PROSPERO (CRD420261370281). Randomized intervention studies involving reproductive-aged women diagnosed with PCOS based on the Rotterdam criteria were included. Eligible studies evaluated multimodal interventions combining biological and psychosocial approaches. Searches were performed in PubMed/MEDLINE, Embase, Scopus, Web of Science, and Cochrane Library. Comparison groups included isolated interventions, placebo, usual care or waitlist controls. Primary outcomes were depression, anxiety, and quality of life assessed using validated instruments. Secondary outcomes included metabolic and hormonal parameters. Risk of bias was assessed using the Cochrane Risk of Bias 2 (RoB2) tool. Statistical analyses were performed using a random-effects model with 95% confidence intervals. **Results:** Four randomized clinical trials involving 305 participants were included. The meta-analysis demonstrated a significant reduction in anxiety and depression symptoms (SMD: –0.43; 95% CI: –0.58 to –0.28; $p < 0.0001$), with no heterogeneity observed ($I^2 = 0\%$). No statistically significant difference was found between subgroups ($p = 0.92$), supporting the consistent effectiveness of combined interventions. **Discussion:** The findings suggest that multimodal interventions promote benefits in PCOS management by simultaneously targeting metabolic and psychological factors. Integrated strategies addressing physical and emotional aspects of the syndrome appear to improve anxiety, depression, and quality of life. **Conclusion:** Combined interventions demonstrated beneficial effects on anxiety, depression, and quality of life in women with PCOS. These findings support the incorporation of integrated and patient-centered approaches into PCOS management. Further studies are required to evaluate the long-term sustainability and effectiveness of these interventions.
Keywords: polycystic ovary syndrome; mental health; lifestyle intervention.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

17 (3011) EFFECT OF EXPOSURE TO BPA FROM CHILDHOOD TO POSTPUBERTY ON UTERINE AND OVARIAN FUNCTION AND MORPHOLOGY

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The female reproductive system is regulated by a complex network of interactions between hormones of the hypothalamic-pituitary-ovarian axis and plays an important role in the production of gametes, secretion of sex hormones, and fertility. Given this complexity, its susceptibility to external and environmental factors, such as Bisphenol A (BPA), a chemical endocrine disruptor used in the manufacture of plastics and food packaging, whose estrogenic activity causes negative impacts on the reproductive system, is evident. BPA has been associated with polycystic ovary syndrome and infertility. In this context, this study aimed to evaluate the morphology and uterine and ovarian function of female rats treated with 0 or 5 mg/kg of BPA (in corn oil) starting from postnatal day 22. The rats were subdivided into three age groups and euthanized during pre-puberty, on the day of puberty confirmation (vaginal opening), and post-puberty (during the second diestrus). After euthanasia, the tissues were collected, weighed, and the ovaries and distal portion of the uterus were subjected to histological and morphometric analysis, as well as analysis of anti-Müllerian hormone gene expression in the ovaries. The results were analyzed by two-way ANOVA followed by Dunnett's post-test. The obtained results show that the BPA-treated group had a shorter estrous cycle duration. Additionally, there was a reduction in the gene expression of AMH hormone in the post-puberty BPA-treated group compared to the control group of the same phase, as well as a decrease in the number of secondary and tertiary follicles during pre-puberty. A significant increase in endometrial and myometrial thickness from pre- to post-puberty was observed, reflecting the expected physiological growth. However, BPA-exposed rats exhibited lower values than the control group, especially during puberty. Thus, it is suggested that early exposure to BPA may compromise ovarian follicular reserves and the maintenance of folliculogenesis throughout reproductive life, confirming the negative impacts of the endocrine disruptor on the reproductive function of females.
Keywords: chemical endocrine disruptor; female reproductive system; ovarian dysfunction.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

18 (3449) MENTAL HEALTH SCREENING WITH MINI RTM IN TRANS WOMEN AND TRAVESTIS ATTENDED AT REFERENCE CENTERS IN BAHIA: PSYCHIATRIC BURDEN, CHILDHOOD ADVERSITY, AND QUALITY OF LIFE

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Introduction: Transgender women are exposed to significant psychosocial vulnerabilities related to stigma and social exclusion, which impact mental health. Despite this, mental health remains underexplored in clinical practice, emphasizing the need for structured assessment in clinical care. **Objective:** To describe the mental health profile of trans women and travestis using the MINI RTM and to evaluate its correlation with adverse childhood experiences and quality of life. **Methods:** Cross-sectional study with trans women and travestis attended at referral centers in Northeast Brazil. Data were collected through three instruments: the MINI RTM (total score 0–23; higher scores indicate greater psychiatric burden), the ACE questionnaire (Adverse Childhood Experiences, score 0–10), and three items of the WHOQOL-Bref assessing general quality of life, frequency of negative feelings, and acceptance of physical appearance. Comparisons were performed using the Wilcoxon test, and correlations by Spearman. **Results:** The study included 204 participants (87,3% trans women and 12,7% travestis). The median total MINI RTM score was 6.5 (IQR 3.0–10.0). Significantly higher scores were observed among travestis (7.8) than in trans women (6.0) ($p = 0.034$). Anxiety (74%), depressive anhedonia (64%) and alcohol misuse (59%) were the most prevalent symptoms. Participants with a lifetime history of suicide attempt ($n = 65$) or self-mutilation ($n = 62$) presented significantly higher MINI RTM total scores (p 0.001). Both outcomes were associated with significantly elevated scores in depressive symptoms, anxiety-worry, panic, suicidal ideation, memory problems, intrusive thoughts and social isolation ($p < 0.05$). Specific domains associated only with suicide attempts included irritability ($p = 0.021$), risky projects ($p = 0.033$), avoidance ($p = 0.013$) and dissociation ($p = 0.001$). The MINI RTM total score showed a positive correlation with ACE scores (rho = 0.266; $p < 0.001$) and a negative correlation with general quality of life (rho = –0.235; $p = 0.001$) and an acceptance of physical appearance (rho = –0.224; $p = 0.002$). **Conclusion:** Trans women and travestis attending a reference center present a substantial psychiatric burden as measured by the MINI RTM, with anxiety symptoms affecting nearly three in four participants and travestis showing significantly higher overall scores. Childhood adversity and lower quality of life are significantly associated with worse mental health outcomes.

Keywords: transgender persons; mental health; adverse childhood experiences.

19 (2974) SWITCHING FROM SHORT-ACTING TO LONG-ACTING TESTOSTERONE IN TRANSGENDER MEN: HIGHER SERUM TESTOSTERONE LEVELS WITHOUT INCREASED ERYTHROCYTOSIS

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Introduction: Testosterone therapy is widely used for gender-affirming treatment in transgender men; however, evidence comparing the impact of short-acting and long-acting formulations on serum testosterone levels and hematocrit is still scarce. **Objective:** Evaluate changes in serum testosterone levels and hematocrit in transgender men transitioning from short-acting to long-acting testosterone formulations. **Methods:** This longitudinal, retrospective study included 71 transgender men undergoing testosterone therapy between 2017 and 2024. Of these, 32 individuals transitioned from short-acting to long-acting formulations, enabling within-subject comparisons of serum testosterone levels and hematocrit. **Results:** The mean age of the 71 transgender men group was 34.6 ± 9.6 years, and 31.0%) were smokers. The median duration of testosterone therapy was 32 months (IQR: 16–54). Most individuals used short-acting testosterone (76.1%), followed by long-acting formulations (19.7%) and transdermal gel (4.2%). Median serum testosterone was 469.7 ng/dL (IQR: 306.5–589.2), and median hematocrit was 47.4% (IQR: 43.6–50.0). Elevated hematocrit levels were observed in 25.4% (hematocrit > 50%) and 8.5% (hematocrit > 54%) of participants. No significant correlation was found between serum testosterone levels and hematocrit ($r = 0.1133$; $p = 0.3503$). Among individuals who switched from short-acting to long-acting formulations ($n = 32$), erythrocytosis remained frequent: 31.3% developed hematocrit > 50% during short-acting testosterone use, compared with 28.1% after switching to testosterone undecanoate (odds ratio [OR] 1.16; 95% CI: 0.40–3.50; $p > 0.05$). The mean hematocrit was 47.22% (SD 4.93) with short-acting testosterone and 48.26% (SD 4.39) with testosterone undecanoate, with no significant difference between formulations ($p = 0.2474$). Serum testosterone levels were significantly higher with testosterone undecanoate than with short-acting formulations (552.3 ± 247.9 *vs.* 343.0 ± 149.9 ng/dL; $p = 0.0002$). No associations were observed between erythrocytosis and smoking or obesity. During follow-up, only one cardiovascular event (acute myocardial infarction) was recorded, and no thromboembolic events were observed. **Conclusion:** Long-acting testosterone formulations maintain higher serum testosterone levels without increasing the frequency of erythrocytosis in transgender men undergoing a real-world switch from short-acting regimens.

Keywords: transgender persons; erythrocytosis; testosterone.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

20 (3554) TRANS-CARDIO SCORE: A TRANSLATIONAL PRECISION CARDIOMETABOLIC FRAMEWORK FOR TRANSGENDER INDIVIDUALS INTEGRATING BIOLOGICAL DETERMINANTS AND MACHINE LEARNING

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Introduction: Conventional cardiovascular risk models do not account for hormonal and metabolic determinants specific to transgender individuals receiving gender-affirming hormone therapy, potentially limiting individualized cardiometabolic prediction. **Objective:** To develop and exploratorily evaluate the Trans-Cardio Score, a transgender-specific cardiometabolic precision framework integrating biological determinants and machine learning. **Methods:** Retrospective observational study including transgender individuals with complete clinical, laboratory, hormonal, and cardiovascular data. The primary outcome was composite cardiometabolic burden, including dysglycemia, hypertension, atherogenic dyslipidemia, excess adiposity, or cardiovascular events. The model was developed using penalized multivariable logistic regression with internal validation by leave-one-out cross-validation, bootstrap, calibration, Brier score, decision curve analysis, exploratory reclassification, and complementary random forest analysis. **Results:** Sixty-six individuals were included, of whom 42 (63.6%) presented composite cardiometabolic burden. Higher-risk individuals showed worse cardiometabolic profiles, with higher body mass index (27.3 vs. 24.3 kg/m²; p = 0.0016), systolic blood pressure (125 vs. 112 mmHg; p = 0.0069), diastolic blood pressure (80 vs. 70 mmHg; p = 0.010), and glucose (98 vs. 89 mg/dL; p = 0.0029). In adjusted penalized regression, glycemic burden was the strongest independent predictor (adjusted odds ratio 15.9; 95% confidence interval 1.38–182.1; p = 0.026). Framingham showed moderate discrimination (AUC 0.776), whereas the Trans-Cardio Score improved discrimination (AUC 0.818), calibration (slope 1.10; intercept 0.81), and accuracy (Brier 0.188). Decision curve analysis suggested greater clinical benefit. Random forest achieved superior discrimination (AUC 0.890), supporting nonlinear interactions between hormonal exposure and cardiometabolic vulnerability. **Conclusion:** The Trans-Cardio Score showed promising performance as a translational precision framework for transgender cardiometabolic stratification, outperforming conventional cardiovascular models. Integrating biologically contextualized determinants may improve individualized risk prediction and supports prospective external validation.

Keywords: cardiometabolic risk; precision medicine; transgender health.

PEDIATRIC ENDOCRINOLOGY

21 (3567) RISING BURDEN AND RACIAL INEQUITY IN DIABETIC KETOACIDOSIS HOSPITALIZATIONS AMONG BRAZILIAN CHILDREN AND ADOLESCENTS: A 15-YEAR NATIONWIDE TREND ANALYSIS (2010–2024)

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Introduction: Diabetic ketoacidosis (DKA) is a life-threatening acute complication of type 1 diabetes mellitus (T1DM) and a leading cause of hospitalization in children and adolescents. Racial inequalities may affect access to early T1DM diagnosis, increasing DKA risk in vulnerable populations. **Objective:** To analyze temporal trends in DKA hospitalizations among children and adolescents (0–19 years) in Brazil from 2010 to 2024, and to assess racial/ethnic disparities and the impact of the COVID-19 pandemic. **Methods:** Ecological time-series study using ICD-10 code E10.1 from individual-level hospitalization microdata (SIH/SUS, Datasus) for all Brazilian states (2010–2024). Hospitalization rates per 100,000 inhabitants were calculated using IBGE population projections; race-specific denominators were estimated by linear interpolation between the 2010 and 2022 Brazilian Censuses (IBGE, Table 9606), restricted to the 0–19-year age group. Temporal trend was assessed by log-linear regression (average annual percentage change, AAPC). Pre- (2010–2019) and post-COVID (2020–2024) mean rates were compared using Welch's t-test. Racial/ethnic disparities were assessed by Poisson regression (incidence rate ratios, IRR) adjusted for year and pandemic period. **Results:** A total of 35,368 DKA hospitalizations were identified. Hospitalization rates increased from 1.96 (2010) to 9.22/100,000 (2024), with AAPC = +11.5% (95% CI: 10.9%–12.1%). Mean rates were significantly higher post-COVID (7.14 vs. 3.19/100,000; p = 0.0007). In 2010, mixed-race (pardo) children had rates 52% lower than White children (0.83 vs. 1.75/100,000). By 2023, pardo rates surpassed white rates for the first time (8.06 vs. 7.11/100,000), and by 2024 the gap widened further (10.38 vs. 8.36/100,000 — 24% higher). Black children had persistently lower rates than White children (IRR = 0.456; 95% CI: 0.430–0.483). Northeast Brazil's share of national hospitalizations doubled from 10.4% (2010) to 22.2% (2024). **Conclusion:** DKA hospitalizations in Brazilian children and adolescents have grown more than fourfold over 15 years, with rates persisting above pre-pandemic levels after 2020, possibly reflecting the global trend of increasing T1DM rates. The reversal of the pardo/white rate gap from 2023 onward may suggest an increase in the rates of people who self-identify as pardo, as well as growing racial inequity in T1DM early diagnosis access.

Keywords: diabetic ketoacidosis; type 1 diabetes mellitus; racial disparities.

BONE AND MINERAL METABOLISM

22 (3152) CHANGES IN BONE DENSITY AND MICROARCHITECTURE ARE ASSOCIATED WITH GLYCEMIC CONTROL IN YOUNG MEN WITH TYPE 1 DIABETES MELLITUS

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Introduction: There are few studies on bone health of young men with type 1 diabetes (T1D). **Objective:** To evaluate bone density and microarchitectural alterations in young adult men with a diagnosis of T1D for at least five years. **Methods:** Male individuals with T1D, aged 20 to <50 years followed-up by the same experienced professionals were evaluated for prevalent fractures, daily physical activity using the International Physical Activity Questionnaire (IPAQ), body composition and areal bone mineral density (BMD) using dual-energy X-ray absorptiometry (DXA). Volumetric BMD and bone microarchitecture were assessed by high-resolution peripheral quantitative computed tomography (HR-pQCT). Glycemic and clinical data from the preceding five years were retrieved from medical records. The control group comprised healthy individuals matched for age and body mass index (BMI). **Results:** Fifty individuals with T1D and 25 controls were included. Their mean (±SD, range) age at T1D diagnosis and at study evaluation were 7.98 ± 6.11 years (1–30) and 30.28 ± 7.74 years (21–46), respectively. About half maintained mean HbA1c below 7.5% in the previous 5 years, with a coefficient of variation median (IQR) of 0.07 (0.06-0.10). Nine individuals had microvascular complications and eight were classified as having low BMD for age by DXA. No prevalent fractures were reported. Compared with controls, individuals with T1D did not differ significantly in spine or hip BMD, but they had reduced trabecular bone volume and thickness, despite better cortical bone parameters. Participants with T1D with low BMD for age had lower lean mass, reduced volumetric trabecular BMD and worse trabecular and cortical bone microstructure compared with those with adequate BMD. Mean HbA1c over 5 years, but not HbA1c variability, had negative and significant correlations with HR-pQCT parameters. **Conclusion:** Our data demonstrate a clear association between T1D, long-term glycemic control, and adverse bone microarchitectural changes in young adult men. . The low prevalence of microvascular disease possibly protected patients from cortical porosity, bone fragility and fractures.

Keywords: type 1 diabetes mellitus; high-resolution peripheral quantitative computed tomography (HR-pQCT); bone microarchitecture; bone densitometry.

BONE AND MINERAL METABOLISM

23 (3147) ESTIMATED PREVALENCE AND EPIDEMIOLOGICAL MAPPING OF HYPOPHOSPHATASIA IN THE STATE OF SÃO PAULO: DATASUS DATA MINING AND DEVELOPMENT OF THE HYPOPHOSPHATASIA RISK SCORE (HPP-RS)

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Introduction: Hypophosphatasia (HPP) is a rare inherited metabolic disease caused by variants in the ALPL gene, characterized by reduced activity of tissue-nonspecific alkaline phosphatase and a broad clinical spectrum. Manifestations include bone mineralization defects, recurrent fractures, osteomalacia, chronic musculoskeletal pain, and premature tooth loss. Phenotypic heterogeneity and low clinical suspicion result in substantial underdiagnosis and diagnostic delay. Epidemiological intelligence strategies may support the large-scale identification of at-risk patients. **Objective:** To estimate the prevalence of patients with possible HPP in the State of São Paulo using Datasus data mining and to develop a stratification algorithm named the Hypophosphatasia Risk Score (HPP-RS). **Methods:** Retrospective cross-sectional study based on data from the Hospital Information System of the Brazilian Unified Health System (SIH/DATASUS), State of São Paulo, from 2015 to 2024. Records containing combinations of clinical manifestations, ICD-10 codes, procedures, and healthcare utilization patterns potentially associated with HPP were evaluated. The HPP-RS was developed by integrating variables related to osteometabolic abnormalities, skeletal deformities, recurrent fractures, osteomalacia, early dental abnormalities, and chronic musculoskeletal pain. A geospatial analysis of the territorial distribution of suspected cases was performed. **Results:** A heterogeneous distribution of possible HPP cases was observed, with concentration in tertiary and quaternary centers. The institutions with the highest number of identified patients were AACD Vila Clementino (n = 78), Santa Casa de São Paulo (n = 26), HC-FAEPA Ribeirão Preto (n = 23), HC-FMUSP (n = 18), and Hospital Estadual de Diadema (n = 17). Spatial mapping demonstrated a higher density of cases in the metropolitan region of São Paulo and in university centers in the interior of São Paulo state. The HPP-RS demonstrated potential applicability as an epidemiological screening tool and as a means of referral for specialized investigation. **Conclusion:** The use of Datasus data combined with epidemiological intelligence tools enabled the identification of healthcare patterns compatible with HPP in the State of São Paulo. The HPP-RS represents a promising strategy for population screening and for reducing underdiagnosis in rare osteometabolic diseases.

Keywords: hypophosphatasia; epidemiological intelligence; Datasus.

BONE AND MINERAL METABOLISM

24 (3212) PARATHYROIDECTOMY REDUCES CIRCULATING ENDOTHELIAL CELLS AND PROGENITOR CELLS WITHOUT IMPROVING VASCULAR REACTIVITY IN CLASSICAL PRIMARY HYPERPARATHYROIDISM: A 12-MONTH PROSPECTIVE STUDY

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Introduction: Classical primary hyperparathyroidism (PHPT) is associated with increased cardiovascular risk, but the reversibility of vascular impairment after parathyroidectomy remains uncertain. Whether surgery modulates endothelial homeostasis at functional, biochemical, and cellular levels is incompletely characterized. **Objective:** To evaluate the impact of parathyroidectomy on microvascular reactivity, systemic inflammation, endothelial activation, the renin-angiotensin-aldosterone system, and endothelial damage and repair markers in classical PHPT. **Methods:** Prospective longitudinal study with 30 patients (93.3% women, 62.1 ± 12.3 years) with classical PHPT and surgical indication. Clinical, biochemical, hemodynamic evaluations, venous occlusion plethysmography, and laser speckle contrast imaging were performed at baseline, 6 and 12 months after parathyroidectomy. Mature circulating endothelial cells ($CD45^-/CD146^+/CD34^+/CD133^-$), activated endothelial cells ($CD45^-/CD146^+/CD34^+/CD106^+$), and endothelial progenitor cells ($CD45^-/CD146^+/CD34^+/CD133^+$) were quantified by flow cytometry. Comparisons used Kruskal-Wallis test; $p \leq 0.05$. **Results:** Serum calcium, parathyroid hormone, and C-telopeptide decreased significantly after surgery (all $p < 0.001$). Mature circulating endothelial cells decreased by approximately 53% at 6 months ($p < 0.05$), and activated endothelial cells declined significantly ($p < 0.05$) at the same time-point. Endothelial progenitor cells showed progressive decline, reaching approximately 83% reduction at 12 months ($p < 0.05$). Forearm and cutaneous microvascular reactivity, blood pressure, interleukin-6, high-sensitivity C-reactive protein, and soluble adhesion molecules (sICAM-1, sVCAM-1, sE-selectin) remained unchanged, as did lipid and glycemic parameters. Aldosterone increased from 5.30 to 8.80 ng/dL at 12 months ($p = 0.02$) without renin activation, contrasting with the classical PTH-aldosterone model. **Conclusion:** Parathyroidectomy modulates the cellular endothelial damage-repair balance with distinct temporal dynamics — early reduction of mature and activated endothelial cells, followed by delayed decline of progenitor cells — while microvascular reactivity, systemic inflammatory and soluble endothelial activation markers, blood pressure, and metabolic parameters remain stable. This dissociation, alongside the paradoxical aldosterone elevation, reinforces a multifactorial cardiovascular pathophysiology in PHPT.
Keywords: primary hyperparathyroidism; endothelial progenitor cells; parathyroidectomy.

BONE AND MINERAL METABOLISM

25 (3021) PTH MEASUREMENT AS A PREDICTOR OF POST-PARATHYROIDECTOMY HYPOCALCEMIA IN PRIMARY HYPERPARATHYROIDISM

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Introduction: Primary hyperparathyroidism (PHPT) is characterized by inappropriate secretion of parathyroid hormone (PTH), most commonly caused by an adenoma, with parathyroidectomy (PTX) as the only curative treatment. Postoperative hypocalcemia is the most frequent complication (due to hypoparathyroidism or hungry bone syndrome), impacting on patient safety, length of hospital stay, and healthcare costs. Early identification of at-risk patients is essential to guide prompt management. Postoperative PTH measurement is well established after total thyroidectomy, in which a circulating level of 10 pg/mL is considered the threshold for predicting hypoparathyroidism. However, this tenet may not be directly applicable to the postoperative period following PTX. **Objective:** To evaluate whether PTH levels measured 24 hours after PTX predict postoperative hypocalcemia in patients with PHPT. **Methods:** Retrospective observational study including patients who underwent PTX at a tertiary referral center between 2020 and 2025. Clinical, laboratory, and histopathological data were obtained from medical records. Hypocalcemia was defined as corrected serum calcium < 8.5 mg/dL during hospitalization. Patients were stratified by hypocalcemia status, and subgroup analyses were conducted according to etiology (hypoparathyroidism vs. hungry bone syndrome). ROC curve analysis was performed to assess predictive performance. **Results:** A total of 45 patients were included (median age 64 years; 75.5% female); adenoma was the most common etiology. Hypocalcemia occurred in 73.3% of patients, predominantly due to hypoparathyroidism. Median PTH at 24 hours postoperatively was significantly lower in patients with hypocalcemia compared to those without (12.1 vs. 28.6 pg/mL; $p = 0.0071$). ROC analysis, performed exclusively in patients with hypocalcemia due to temporary hypoparathyroidism, demonstrated reasonable discriminatory ability (AUC 0.80; 95% CI 0.64–0.95; $p = 0.0039$), with an optimal cutoff of 16.45 pg/mL (sensitivity 73.9%, specificity 83.3%). **Conclusion:** PTH measured 24 hours after PTX is significantly associated with postoperative hypocalcemia and shows good predictive performance, particularly for hypoparathyroidism. Although not sufficient as an isolated marker, its availability, low cost, and clinical applicability support its use for early risk stratification, guiding calcium replacement, monitoring, and safer hospital discharge.
Keywords: primary hyperparathyroidism; parathyroidectomy; hypocalcemia.

BONE AND MINERAL METABOLISM

26 (3490) THE UNEXPLORED ROLE OF VITAMIN D PATHWAY POLYMORPHISMS ON PHENOTYPIC HETEROGENEITY OF SPORADIC PRIMARY HYPERPARATHYROIDISM IN A BRAZILIAN POPULATION

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Introduction: Low 25-hydroxyvitamin D (25OHD) levels are associated with severe primary hyperparathyroidism (PHPT) presentation. Genetic variants in vitamin D pathway may alter enzymatic activity or protein affinity, and have been linked to 25OHD levels in Mendelian randomization studies. However, their impact on PHPT phenotype remains uncharacterized. **Objectives:** To evaluate polymorphism frequencies in the genes 7-dehydrocholesterol reductase, 25-hydroxylase (CYP2R1), 1-alpha-hydroxylase (CYP27B1), and vitamin D-binding protein (Gc) in patients with PHPT and controls, and to assess their correlations with sporadic PHPT presentation. **Methods:** Cross-sectional study with 302 PHPT patients and 300 matched controls. Genotyping performed by qPCR using allelic discrimination assays. Hierarchical multivariate regression was performed, and statistical significance was set as $p \leq 0.05$. **Results:** Most participants were women (87.7%) and Caucasian (56.6%). The groups were similar regarding sex, ethnicity, age, and body mass index (BMI), and polymorphisms were in Hardy-Weinberg equilibrium. No differences in genotypic frequencies were observed across the groups, but associations emerged between certain variants and PHPT phenotypes. Individuals with CYP2R1 GG genotype were younger at diagnosis compared to A-allele carriers (61 [37–83] vs. 63 [38–88] years). CYP27B1 T-allele carriers (TT/TG) had higher total calcium levels (11.5 [10.3–19.1] vs. 11.2 [10–18.8]) and TT genotype had lower eGFR (80 [46–178] vs. 89.5 [24–1345]). GG genotype of Gc polymorphism demonstrated higher kidney stone prevalence (73.2% vs. 52.1%; OR 2.51 [95% CI: 1.22–5.45]) and lower osteoporosis (43.7% vs. 59.7%). In multivariate logistic regression for nephrolithiasis, Caucasian ethnicity (OR 2.14; 95% CI: 1.11–4.12), lower serum phosphate (OR 0.462; 95% CI: 0.24–0.91), and Gc variant (OR 3.23; 95% CI: 1.30-8.01) remained as independent risk factors. For osteoporosis, Gc variant lost significance in the multivariate model, and only Caucasian ethnicity (OR 2.18; 95% CI: 1.07–4.43), advanced age at diagnosis (OR 1.07; 95% CI: 1.03–1.11), and lower BMI (OR 0.91; 95% CI: 0.85–0.98) remained. **Conclusion:** In sporadic PHPT patients, the Gc variant was independently associated with nephrolithiasis risk, while CYP2R1 and CYP27B1 variants were associated with age at diagnosis, calcemia, and renal function, what suggest a potential modulatory role of vitamin D-related genetic variants on PHPT severity and clinical presentation.

Keywords: genetic polymorphism; vitamin D-binding protein; nephrolithiasis.

NEUROENDOCRINOLOGY

27 (3249) DIAGNOSIS OF ACROMEGALY THROUGH VOICE ANALYSIS: CROSS-VALIDATION OF FOUR MACHINE LEARNING MODELS

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Introduction: Acromegaly is a rare disease caused by excessive growth hormone (GH) secretion, and diagnosis is often delayed because of its insidious clinical presentation. Recent studies have shown that artificial intelligence can identify vocal patterns associated with the disease. Unlike previous studies, the present work uses continuous speech in Brazilian Portuguese and compares multiple machine learning algorithms with patient-level validation, providing greater methodological robustness. **Objective:** To compare machine learning algorithms for detecting acromegaly based on features extracted from voice recordings. **Methods:** This case-control study included 98 participants: 61 patients with acromegaly and 37 controls. Participants performed a standardized reading task. Audio recordings were segmented into 3-second windows with 1.5-second overlap. Spectral and prosodic features and Mel-frequency cepstral coefficients (MFCCs) were extracted and normalized. Models were evaluated using stratified five-fold cross-validation at the patient level, with synthetic oversampling in the training sets to prevent data leakage. Four algorithms were compared: Support Vector Machine (SVM) with radial basis function kernel, Random Forest, XGBoost, and LightGBM. Performance metrics included ROC AUC, accuracy, sensitivity, specificity, and F1-score. **Results:** All models showed good discrimination between patients with acromegaly and controls. The best performance was achieved by Support Vector Machine, with a mean AUC of 0.875 ± 0.066, accuracy of 76%, F1-score of 0.82, and sensitivity of 0.87. Random Forest, XGBoost, and LightGBM also showed consistent performance, with AUCs of 0.826, 0.834, and 0.827, respectively. ROC curves showed consistency. **Conclusion:** The human voice contains acoustic biomarkers capable of discriminating individuals with acromegaly from controls with good performance. Support Vector Machine achieved the best results. Using continuous speech in Brazilian Portuguese and patient-level validation, this study highlights voice as a noninvasive, low-cost digital biomarker for screening and early diagnosis of acromegaly.
Keywords: acromegaly; machine learning; diagnosis.

NEUROENDOCRINOLOGY

28 (3179) IMPACT OF GROWTH HORMONE DEFICIENCY AND GH REPLACEMENT IN ADULT PATIENTS WITH CHILDHOOD-ONSET GH DEFICIENCY: CHANGES IN BODY COMPOSITION WITHOUT CONCOMITANT CHANGES IN INSULIN SENSITIVITY

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Introduction: Growth hormone deficiency (GDH) in adults results in loss of lean muscle mass, increased fat mass, decreased bone mass, altered physical capacity, presence of cardiovascular risk factors, and poor quality of life (QoL). Recombinant human Growth Hormone (rhGH) replacement therapy (GHRT) in adults with GHD is well documented and effective, improving or normalizing various GH-related abnormalities and potentially conferring health benefits such as reduced body fat mass, increased lean mass, and increased bone mass. However, the GHRT effect on glucose metabolism remains controversial. **Objective:** This study aimed to evaluate the impact of prolonged rhGH treatment and its interruption on insulin sensitivity (IS) and body composition in adult patients with childhood-onset GH deficiency (CO-GHD). **Methods:** A cross-sectional study was conducted with 61 Brazilian adults aged 18-50 years: 31 confirmed CO-GHD patients (15 who continued GHRT [GHRT-C] and 16 who interrupted GHRT [GHRT-I] during the transition period) and 30 controls matched by age, sex, and body mass index. IS was assessed using the gold-standard hyperinsulinemic euglycemic clamp (HEC) technique, calculating the metabolic value (M-value), which is directly proportional to insulin sensitivity and corrected for lean mass. Body composition was evaluated using dual-energy X-ray absorptiometry (DXA). **Results:** Patients in the GHRT-I group exhibited a body profile with greater adiposity (higher fat mass percentage and fat mass index) and lower lean mass indices (lean mass percentage and appendicular muscle mass index) compared to their matched controls. In contrast, the GHRT-C group showed no difference in fat or lean mass indices relative to their controls. The M-value did not differ significantly among the GHRT-C, GHRT-I, and control groups. Furthermore, in GHRT-I patients, IS was significantly and negatively correlated with visceral adipose tissue volume and visceral adiposity index. However, in GHTR-C patients, this relationship is not observed. **Conclusion:** Maintaining GHRT during the transition period into adulthood in CO-GHD patients prevented increased adiposity and reduced lean mass seen in those who interrupted GHRT. However, these body composition changes were not accompanied by significant changes in insulin sensitivity, suggesting the long-term safety of GHRT with respect to metabolic syndrome and type 2 diabetes risk.
Keywords: growth hormone deficiency; insulin sensitivity; body composition.

NEUROENDOCRINOLOGY

29 (3151) ONCE DAILY ATUMELNANT (CRN04894) ENABLES LOWERING OF GLUCOCORTICOID DOSES WITH SUSTAINED ANDROGEN REDUCTION IN ADULTS WITH CONGENITAL ADRENAL HYPERPLASIA

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Atumelnant (CRN04894) is a first-in-class, once-daily, oral, selective melanocortin type 2 receptor antagonist in development for the treatment of congenital adrenal hyperplasia (CAH). A 12-week, Phase 2, open-label study (NCT05907291) was conducted to evaluate the efficacy and safety of atumelnant in adults with classic CAH (21-hydroxylase deficiency). Participants with morning A4 level ≥ 1.5 times the upper limit of normal were enrolled in 4 dose cohorts of once-daily, oral atumelnant. Cohorts 1–3 (40, 80, or 120 mg at bedtime) maintained stable glucocorticoid (GC) dosing (≥15 mg hydrocortisone equivalent) for ≥ 6 months prior to screening and throughout the study. Cohort 4 (80 mg in the morning [AM]) had stable GC dosing (≥11 mg/m²/d hydrocortisone equivalent) for ≥ 1 month prior to screening, and reduced GC dose stepwise to target < 11 mg/m²/d. The primary efficacy endpoint was change from baseline to Week 12 in early morning pre-GC serum A4. Percent change from baseline in GC daily dose was an exploratory endpoint for Cohort 4. A total of 38 participants (55.3% women; mean [range] age 33.2 [20–64] years; mean [range] GC dose 27.1 [20–40] mg/d [hydrocortisone equivalent], median [range] A4 34.2 (4.03-96.19) nmol/L [reference range: women, 1.05–6.98 nmol/L; men, 1.40-5.24 nmol/L]) were enrolled (40-mg, n = 11; 80-mg, n = 11; 120-mg, n = 6; 80-mg AM + GC reduction, n = 10). Two participants discontinued, both from Cohort 4 (withdrew consent). At Week 12, mean, (median [range]) percent change from baseline in pre-GC morning serum A4 was: 40 mg, –58.2% (–65.2% [–94.3%, –5.5%]); 80 mg, –69.8% (–80.1% [–98.7%, –21.6%]); 120 mg, –79.5% (–81.6% [–91.4%, –53.5%]); 80 mg AM + GC reduction, –67.4% (–74.9% [–89.7%, –26.6%]). In Cohort 4, 7/8 participants (87.5%) achieved a physiologic daily GC dose (11 mg/m²/d) at Week 12. In all, 31 participants had ≥ 1 treatment-emergent adverse event (TEAE); none were severe or serious. Sixteen participants experienced TEAEs related to treatment, but none led to treatment discontinuation. In conclusion, in adults with classic CAH, atumelnant induced substantial androgen reduction despite GC dose reduction. Additionally, morning atumelnant dosing resulted in similar A4 reductions as those observed with evening administration. These results highlight the potential for ACTH antagonism to achieve control of adrenal androgen levels while allowing for GC use at doses that do not exceed those needed for physiologic replacement.
Keywords: androgens; congenital adrenal hyperplasia; glucocorticoid.

NEUROENDOCRINOLOGY

30 (2992) SUBCHRONIC DI(2-ETHYLHEXYL) PHTHALATE EXPOSURE ALTERS NEUROBEHAVIOR AND DISRUPTS THYROID AXIS SIGNALING IN ADULT ZEBRAFISH

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Introduction: Di(2-ethylhexyl) phthalate is a widely used plasticizer recognized as a pervasive environmental contaminant and a potent endocrine disruptor. **Objective:** This study aimed to investigate the neurobehavioral and molecular impacts of subchronic di(2-ethylhexyl) phthalate exposure in adult zebrafish, focusing on the integration between brain gene expression and thyroid homeostasis. **Methods:** Adult male and female zebrafish were exposed to di(2-ethylhexyl) phthalate at 50 or 100 micrograms per liter for 21 days. Behavioral assays evaluated anxiety-like behavior, social interaction, and locomotion. Brain and thyroid gland gene expression was subsequently analyzed by quantitative polymerase chain reaction. **Results:** Both sexes exhibited anxiety-like behavior, with increased time in the dark zone at 50 micrograms per liter and impaired social interaction (increased time in the antisocial zone) at 100 micrograms per liter. Locomotor deficits were evident through reduced average velocity and total distance traveled at both concentrations. Males additionally showed a significant reduction in total body weight at the higher dose, an effect not observed in females. In the brain, females displayed downregulation of brain-derived neurotrophic factor, nuclear factor erythroid 2-related factor 2, and superoxide dismutase 2, whereas males showed reduced expression of dopamine receptor d2, nuclear factor erythroid 2-related factor 2, and superoxide dismutase 2. In the thyroid gland, females presented increased expression of sodium/iodide symporter and thyroglobulin at the lower dose and significant downregulation of the thyroid stimulating hormone receptor at the higher dose; in males, thyroglobulin expression was significantly reduced at 100 micrograms per liter. **Conclusion:** Subchronic exposure to di(2-ethylhexyl) phthalate acts as a neuroendocrine disruptor in adult zebrafish, inducing behavioral deficits through alterations in neuroplasticity, antioxidant defense, and dopaminergic signaling, while concurrently disrupting thyroid hormone synthesis and signaling in a sex-dependent and non-monotonic manner.
Keywords: phthalates; thyroid; neurobehavior.

OBESITY

31 (3443) ASSOCIATION BETWEEN BODY IMAGE AND SEXUAL FUNCTION IN WOMEN: THE ROLE OF OBESITY

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Introduction: Obesity is a multifactorial chronic disease associated with metabolic, cardiovascular, psychosocial repercussions and negative impacts on women's sexual health. Female sexual dysfunction includes persistent changes in sexual desire, arousal, lubrication, orgasm, or pain during intercourse. Additionally, body image perception is relevant, since negative self-perception may impair sexual performance and quality of life. **Objective:** Analyze the association between body image and sexual function in women with obesity. **Methods:** Observational cross-sectional study with patients treated in a university extension project, selected through non-probability sampling. Thirty-seven women aged 21–59 years with BMI > 30 kg/m participated. Exclusion criteria included severe neurological or psychiatric disorders, use of medications that could potentially affect sexual function, and refusal to participate. The project was approved by the Research Ethics Committee (CAAE: 8331720.0.0000.5348). Assessments included anthropometric evaluation for BMI calculation, the Female Sexual Function Index (FSFI), considering dysfunction when the score was < 26.55, and the Brazilian Silhouette Scale, composed of 15 images to assess body image perception. **Results:** The mean age was 45.3 (9.7) years and the mean BMI was 41.1 (9.3) kg/m². The mean total FSFI score was 17.6 (10.1) points. According to the FSFI cutoff, 83.3% of participants presented probable sexual dysfunction. Regarding body image, mild dissatisfaction was observed in 8.1% of participants (1–2 silhouette discrepancy), moderate in 48.7% (3–4 silhouettes), and severe in 43.2% (5 or more silhouettes). Analyses showed no significant association between sexual function and body image (p = 0.890), BMI (p = 0.172), or age (p = 0.600). However, sexual dysfunction prevalence increased according to the severity of body dissatisfaction: 66.7% among women with mild dissatisfaction, 77.8% with moderate dissatisfaction, and 93.8% with severe dissatisfaction. **Conclusion:** Although association tests did not reach statistical significance, data suggest a clinical increase in sexual dysfunction prevalence proportional to greater body image dissatisfaction. Further studies with larger samples, control groups, and longitudinal follow-up are needed to confirm the association between body image self-perception and sexual dysfunction.

Keywords: obesity; body image; female sexual dysfunction.

OBESITY

32 (3131) DIETICIAN SUPPORT SUBSTANTIALLY ENHANCES WEIGHT LOSS OUTCOMES IN A DIGITAL GLP-1 RECEPTOR AGONIST PROGRAM: A REAL-WORLD BRAZILIAN COHORT STUDY

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Introduction: GLP-1 receptor agonists (GLP-1RAs) have demonstrated remarkable efficacy for weight loss in clinical trials, yet real-world outcomes are consistently more modest driven largely by suboptimal adherence, early discontinuation, and lack of structured support. In parallel, obesity management guidelines increasingly recommend multimodal approaches combining pharmacotherapy with nutritional counselling. Despite this, the incremental benefit of dietician support within digitally-delivered GLP-1RA programs remains poorly quantified. Understanding whether structured nutritional support translates into meaningfully better outcomes in routine care is critical to inform how digital obesity programs should be designed and resourced. **Objective:** To evaluate the impact of dietician support on weight loss outcomes in adults receiving GLP-1RA therapy through a digital weight management platform in Brazil. **Methods:** Retrospective cohort study of 18,529 adults initiating GLP-1RA therapy (tirzepatide n = 6,394; semaglutide n = 12,135) through a digital weight management platform in Brazil. Mixed model repeated measures (MMRM) analysis adjusted for baseline weight and age compared weight loss trajectories according to dietician support status. Responder rates (≥5%, ≥10%, ≥15% weight loss) were examined by dietician support and GLP-1RA agent. **Results:** Among 18,529 adults included (mean age ~39 years, baseline weight ~94 kg, BMI ~34 kg/m², ~73% female), 3.7% received dietician support. Dietician support was associated with markedly greater weight loss at 5 months (-12.4% *vs.* -10.0%; difference -2.4%, 95% CI: 0.5 to 4.3; p = 0.012). The effect was most pronounced with tirzepatide plus dietician support (-16.4% *vs.* -10.9% without; p = 0.05). Responder rates were substantially higher with dietician support: for tirzepatide, ≥10% weight loss was achieved by 45.7% vs. 6.6% without; for semaglutide, 23.7% vs. 6.0%. Rates of ≥ 15% weight loss followed a similar pattern (tirzepatide: 15.2% vs. 2.0%; semaglutide: 8.8% vs. 1.8%). **Conclusion:** Dietician support markedly amplified weight loss outcomes in adults receiving GLP-1RA therapy through a digital platform in Brazil, with up to 7-fold increases in clinically meaningful responder rates. These findings provide quantitative real-world evidence that nutritional counselling substantially enhances the effectiveness of pharmacotherapy and should be considered an integral component of digital obesity management programs.

Keywords: GLP-1 receptor agonists; dietician support; real-world evidence.

OBESITY

33 (2965) EFFICACY AND SAFETY OF SIBUTRAMINE–TOPIRAMATE COMBINATION VERSUS SIBUTRAMINE MONOTHERAPY FOR WEIGHT LOSS IN ADULTS WITH OBESITY: A 48-WEEK RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL

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Introduction: Access to newer anti-obesity medications remains limited in many healthcare settings, particularly in middle-income countries. Therefore, lower-cost pharmacological strategies may retain clinical relevance, provided that their efficacy and safety are adequately evaluated. This study assessed whether the combination of sibutramine and topiramate is superior to sibutramine monotherapy for weight loss in adults with obesity or overweight associated with cardiometabolic risk factors. **Objective:** The primary outcome was the percentage change in body weight from baseline to week 48. Secondary outcomes included percentage weight loss at week 24; the proportions of participants achieving ≥5%, ≥10%, and ≥15% weight loss; changes in anthropometric and cardiometabolic parameters; scores on the Impact of Weight on Quality of Life — Lite Clinical Trial Version (IWQOL-Lite-CT) and the Eating Behavior Phenotypes Scale (EFCA); treatment adherence; and adverse events. **Methods:** This was a single-center, randomized, double-blind, placebo-controlled, parallel-group superiority trial conducted in São Paulo, Brazil. A total of 154 adults were randomized in a 1:1 ratio to receive sibutramine plus topiramate (SBT+TPM; n = 77) or sibutramine plus placebo (SBT+PLA; n = 77) for 48 weeks, in addition to standardized lifestyle counseling. **Results:** At week 48, mean percentage weight loss was 10.7% in the SBT+TPM group and 6.7% in the SBT+PLA group. The adjusted mean difference between groups was 4.15 percentage points (95% CI: 1.51 to 6.80; $p < 0.001$), favoring SBT+TPM. A higher proportion of participants in the SBT+TPM group achieved ≥ 10% weight loss compared with the SBT+PLA group (54.4% vs. 26.7%; RR, 2.04; 95% CI: 1.19 to 3.49), as well as ≥ 15% weight loss (33.3% vs. 6.7%; RR, 5.00; 95% CI: 1.57 to 15.93). Reductions in BMI, waist circumference, and waist-to-height ratio were also greater in the SBT+TPM group. The overall frequency of adverse events was similar between groups. **Conclusion:** In adults with obesity or overweight associated with cardiometabolic risk factors, the combination of sibutramine and topiramate produced greater weight loss than sibutramine monotherapy over 48 weeks, with an expected and manageable safety profile. These findings support further evaluation of lower-cost pharmacological strategies for obesity management in settings with restricted access to newer anti-obesity medications.
Keywords: obesity; weight loss; randomized controlled trial.

OBESITY

34 (3146) IMPROVEMENTS IN CARDIOMETABOLIC RISK FACTORS BY BODY WEIGHT REDUCTION WITH TIRZEPATIDE IN ADULTS WITH OBESITY AND PREDIABETES: POST HOC ANALYSIS OF SURMOUNT-1

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Introduction: In the 3-year SURMOUNT-1 study, this post hoc analysis assessed changes in cardiometabolic risk factors based on body weight reduction thresholds with tirzepatide. **Methods:** Participants (N = 458) randomized to receive tirzepatide (pooled 5/10/15 mg doses) and with an on-treatment body weight measurement at Week 176 were included in the analysis. The proportion of participants achieving body weight reduction thresholds (<5%, 5 to <10%, 10 to <15%, 15 to <20%, 20 to <25%, 25 to <30%, 30 to <35%, and ≥35%) at Week 176 and change from baseline at Week 176 in cardiometabolic risk factors, including blood pressure, lipid profile, and glycemic parameters, by body weight reduction thresholds were assessed. **Results:** At baseline, mean waist circumference was 116.5 cm, systolic blood pressure: 126.1 mmHg, diastolic blood pressure: 80.4 mmHg, triglycerides: 8.5 mmol/L, HDL-C: 2.7 mmol/L, non-HDL-C: 7.9 mmol/L, LDL-C: 6.2 mmol/L, and HOMA-2-IR (computed with insulin): 2.1. Improvements in waist circumference (3.2–35.3 cm reduction), HbA1c (0.30%–0.76% reduction) and triglycerides (15%–48% reduction) were observed among all body weight reduction subgroups. HOMA2-IR, blood pressure and HDL-C, non-HDL-C and LDL-C also improved among participants who lost ≥5% of body weight. Overall, greater improvements in cardiometabolic risk factors were generally associated with greater body weight reduction. **Conclusion:** In this post hoc analysis of the 3-year SURMOUNT-1 study, tirzepatide treatment was associated with improved cardiometabolic risk factors, that were generally greater in magnitude with higher degrees of body weight reduction among people with obesity and prediabetes. Previously presented at American Heart Association — 98th Scientific Session.
Keywords: tirzepatide; cardiometabolic risk factors; weight reduction.

OBESITY

35 (2900) INCRETIN-BASED THERAPIES ATTENUATE MUSCLE FUNCTION DECLINE IN MASLD: A REAL-WORLD LONGITUDINAL COHORT

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Introduction: Skeletal muscle impairment is an under-recognised feature of metabolic dysfunction-associated steatotic liver disease (MASLD) and is associated with poorer functional and cardiometabolic outcomes. However, the impact of GLP-1 and dual GIP/GLP-1 receptor agonists (GLP-1RAs) on muscle function in this population remains unclear. **Objective:** To evaluate longitudinal changes in muscle strength according to GLP-1RA exposure in patients with MASLD. **Methods:** This prospective real-world cohort included adults with MASLD from a tertiary centre, with at least two muscle strength assessments during routine follow-up between 2014 and 2026. Muscle strength was assessed using handgrip strength (HGS) and the 5-times sit-to-stand (5-STS) test. Clinical and demographic data were obtained during follow-up visits. GLP-1RA prescription reflected routine clinical practice. Longitudinal trajectories of muscle strength were analysed using linear mixed-effects models with random intercepts, adjusting for time-updated BMI, diabetes status, and liver stiffness, as well as age, sex, ethnicity, and socioeconomic status. GLP-1RA use was modelled as a time-varying exposure. **Results:** A total of 149 patients were included (mean ± SD age 54.8 ± 14.0 years; 53% male), with a high prevalence of overweight/obesity (97%), diabetes (72%), and metabolic syndrome (62%). Median follow-up was 35.9 months (IQR 24.1-52.2). At the final visit, 62 patients (42%) were receiving GLP-1RAs, predominantly tirzepatide (65%) or subcutaneous semaglutide (32%). Despite greater weight loss among GLP-1RA users (−5.1% vs. −0.5%; $p < 0.001$), GLP-1RA exposure was not associated with changes in HGS over time (interaction $\beta = 0.18$; $p = 0.411$). Older age, diabetes, and higher liver stiffness were associated with lower HGS, while male sex was associated with higher HGS. For the 5-STS test, performance worsened over time ($\beta = +0.35$ s/year; $p < 0.001$), but GLP-1RA use attenuated this decline (interaction $\beta = -0.64$ s/year; $p=0.001$). Older age was also associated with slower 5-STS performance. **Conclusion:** GLP-1RA use was associated with an attenuated decline in lower-limb function over time. These findings suggest a potential protective role of incretin-based therapies against functional deterioration in MASLD. Support: Capes.

Keywords: MASLD; GLP-1 receptor agonists; muscle strength.

OBESITY

36 (3571) IXEKIZUMAB WITH TIRZEPATIDE IN PSORIASIS OR PSORIATIC ARTHRITIS: A POOLED ANALYSIS OF TOGETHER-PSA AND TOGETHER-PSO

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Introduction: Psoriasis is a chronic immune-mediated inflammatory disease affecting 2%–3% of the global population, driven mainly by IL-23/IL-17 dysregulation and systemic inflammation. Obesity is more frequent in psoriasis and is associated with increased cardiovascular risk and lower rates of complete skin clearance (PASI100). Ixekizumab, an IL-17A inhibitor, provides rapid and sustained skin clearance, while tirzepatide, a dual GIP/GLP-1 receptor agonist, promotes weight loss and cardiometabolic improvement. This study evaluated whether combining ixekizumab and tirzepatide improves dermatologic and metabolic outcomes in psoriasis or psoriatic arthritis. **Methods:** We conducted a meta-analysis according to the Cochrane Handbook and PRISMA guidelines. PubMed, Embase, and Cochrane were searched through May 2026 using the terms “ixekizumab”, “tirzepatide”, and “psoriasis”. Eligible studies were randomized controlled trials comparing ixekizumab plus tirzepatide versus ixekizumab alone in patients with active psoriasis or psoriatic arthritis and overweight/obesity. Endpoints included PASI100, PASI90, PASI75, absolute PASI reduction, BMI, body weight, glycated haemoglobin, lipid profile, and blood pressure. Mean differences (MD) and response differences with 95% confidence intervals (CIs) were calculated using the Mantel-Haenszel method in R 4.5.0. **Results:** A total of 545 participants were included, with 276 receiving ixekizumab plus tirzepatide. Mean age was 50.3 years and 42.7% were male. TOGETHER-PsA included patients with psoriatic arthritis, whereas TOGETHER-PsO included psoriasis patients. Both studies had 36-week follow-up and high obesity burden (87% with BMI ≥ 30 kg/m²). The PsA cohort was predominantly female and had higher rates of hypertension, dyslipidemia, and cardiovascular disease. Combination therapy improved PASI100 (11.09%; 95% CI: 2.08–20.11; $p = 0.016$) and PASI90 (12.56%; 95% CI: 2.95–22.16; $p = 0.010$), while PASI75 was similar between groups. Significant reductions were also observed in BMI, body weight, glycated haemoglobin, total cholesterol, triglycerides, and systolic/diastolic blood pressure, with no significant differences in LDL or HDL. **Conclusion:** Adjunctive tirzepatide to ixekizumab improved complete and near-complete skin clearance and several cardiometabolic parameters in patients with psoriasis or psoriatic arthritis and overweight/obesity, with consistent effects across two randomized trials.

Keywords: tirzepatide; ixekizumab; psoarisis.

OBESITY

37 (3555) USE OF THE BRAZILIAN PORTUGUESE VERSION OF THE EBA-O-BR QUESTIONNAIRE (EATING BEHAVIORS ASSESSMENT FOR OBESITY) IN PEOPLE WITH TYPE 2 DIABETES MELLITUS AND OBESITY

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Introduction: Type 2 Diabetes Mellitus (T2DM) and obesity are interdependent conditions: it is estimated that up to 90% of individuals with T2DM are overweight. Dysfunctional eating behaviors represent critical barriers to metabolic control. The recently validated Eating Behaviors Assessment for Obesity (EBA-O-BR) questionnaire enables systematic screening of these patterns. **Objective:** To analyze eating behaviors in obese patients with T2DM through the application of the EBA-O in its Brazilian Portuguese validated version (EBA-O-BR). **Methods:** Cross-sectional study approved by the local Ethics Committee, including 66 adults with T2DM and BMI ≥ 30 kg/m. Data were obtained from medical records and interviews, including administration of the EBA-O-BR — an 18-item questionnaire grouped into five domains, self-assessed over the previous 3 months: nocturnal eating, food addiction, sweet intake, hyperphagia, and binge eating. Anthropometric and laboratory variables were analyzed (fasting glucose and HbA1c). **Results:** There was a female predominance (77.4%; n = 48) and a mean age of 57.7 ± 11.8 years (range: 23-85 years). Regarding BMI, 45.4% (n = 30) had Class I obesity, 33.3% (n = 22) Class II, and 21.2% (n = 14) Class III. Waist circumference ranged from 93 to 158 cm, with all patients above reference values. Mean fasting glucose was 155 ± 59 mg/dL and mean HbA1c was 8.2 ± 1.8%, with 32.8% (n = 21) within the target of ≤7.0%. Most patients were on oral medication alone (n = 37) or combined with insulin (n = 27). Regarding the EBA-O-BR, using a score 24 as the cutoff (behavior occurring at least weekly), the following were identified: binge eating in 24% (n = 16); sweet intake in 17% (n = 11); nocturnal eating in 14% (n = 9); hyperphagia in 12% (n = 8); and food addiction in 8% (n = 5). Five patients (8%) exceeded the total cutoff score (3 or 4 altered domains). Overall, 35% (n = 23) exhibited one or more inadequate behaviors; of these, 56% (n = 13) had elevated HbA1c. **Conclusion:** The application of the EBA-O-BR in patients with T2DM and obesity proved to be practical, feasible, and clinically relevant, identifying dysfunctional eating behaviors in 35% of the sample, the majority of whom had poor glycemic control. These findings reinforce the need to incorporate systematic screening of eating behavior into clinical practice, supporting more individualized and effective approaches, with potential impact on treatment adherence and metabolic outcomes.

Keywords: eating behavior; obesity; type 2 diabetes mellitus.

OBESITY

38 (3080) WORSE SLEEP PATTERNS ASSOCIATED WITH BINGE EATING IN PATIENTS LIVING WITH OBESITY: AN OVERLOOKED COMBINATION THAT DESERVES MORE ATTENTION

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Introduction: Obesity is increasing worldwide and the high prevalence of Binge Eating Disorder (BED) and sleep issues in this population is well known. **Objective:** We aimed to evaluate if patients with class II and III obesity who were shown positively for BED screening also present more sleep problems. **Methods:** Adults aged between 18 and 65 years, with body mass index (BMI) > 40 kg/m² and BMI > 35 kg/m² with comorbidities associated, attending an endocrinology outpatient clinic specialized in obesity management were included in this cross-sectional study. They were evaluated during clinical interviews through three self-administered questionnaires: 1) Binge Eating Scale for screening BED, 2) a questionnaire for subjective assessment of sleep-related aspects, and 3) a questionnaire for collecting epidemiological and comorbidity data. The data were collected between May 2024 and November 2024. The study was approved by the Research Ethics Committee, and the participants accepted the Free and Informed Consent Form. **Results:** The participants (N = 94) were majority women (94,7%) with mean age of 46,9±10,4 years. Among the sample, 28.7% screened positive for BED and 47.9% had short sleep duration (<7 hours/night), along with other sleep disturbances. Notably, sleep duration was shorter among patients who screened positive for BED (p 0.024, Cohen's d = 0.52). These patients more frequently presented insomnia (p 0.046, OR 2.52; IC 95% [1.00–6.33]), daytime sleepiness (p 0.024, OR 5.11; IC 95% [1.10–23.69]), and co-occurrent multiple sleep problems (p 0.015, OR 5.71; IC 95% [1.24–26.35]). **Conclusion:** Patients who were shown positively for BED presented indeed worse sleep patterns. Routine assessment of sleep and eating disorders is important in the obesity approach, especially for middle-aged women, once those factors appear to be related and may be undervalued in clinical attendings. Furthermore, BED and sleep disorders lead to poorer weight loss outcomes and a lower quality of life. The study has its limitations, and future longitudinal and interventional studies are warranted to better evaluate this association and whether the concurrent treatment of BED and sleep disturbances yields superior clinical outcomes in obesity management.

Keywords: obesity; binge eating disorder; sleep disorders.

OTHERS

39 (3176) DEVELOPMENT OF AN ONLINE EXTRACTION LIQUID CHROMATOGRAPHY TANDEM MASS SPECTROMETRY METHOD FOR SIMULTANEOUS QUANTIFICATION OF SEVEN C19 STEROIDS IN SERUM

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Simultaneous measurement of classical and oxygenated androgens by liquid chromatography-tandem mass spectrometry is analytically challenging due to isobaric interference between critical pairs, such as 11β-hydroxyandrostenedione and testosterone. This compromises diagnostic accuracy, as single-column configurations or immunoassays cannot resolve these steroids. This study aimed to develop and preliminarily evaluate an online extraction liquid chromatography-tandem mass spectrometry method for the simultaneous quantification of seven serum steroids: androstenedione, testosterone, dihydrotestosterone, 11β-hydroxyandrostenedione, 11-ketoandrostenedione, 11-ketotestosterone, and 11β-hydroxytestosterone. To achieve this, serum samples (100 microliters) underwent protein precipitation followed by automated online extraction via a turbulent flow chromatography column. Using a custom valve-switching configuration, analytes were directly transferred to a novel serial dual-column arrangement (C18 followed by biphenyl) on a triple quadrupole mass spectrometer operating in multiple reaction monitoring mode. This setup was optimized for baseline resolution of all isobaric pairs while minimizing sample volume, making it highly suitable for pediatric applications. The method was applied to 53 serum samples from patients (0–62 years, including neonates) under investigation for congenital adrenal hyperplasia, premature adrenarche, or hyperandrogenism, across three independent runs. Baseline resolution of all critical pairs was confirmed. Inter-analyte Spearman correlations were fully consistent with known biosynthetic pathways, validating physiological integrity: 11β-hydroxyandrostenedione correlated with 11-ketoandrostenedione (r = 0.88), reflecting 11beta-hydroxysteroid dehydrogenase type 1 activity, and with androstenedione (r = 0.72; CYP11B1 hydroxylation). Androstenedione correlated with 11-ketotestosterone (r = 0.65) and testosterone (r = 0.54), supporting shared adrenal origin. Dihydrotestosterone correlated with testosterone (r= 0.53) and 11-ketotestosterone (r = 0.48), reflecting 5-alpha reductase activity. In conclusion, integrating automated turbulent flow extraction with a synergistic dual-column chemistry eliminates analytical interference without compromising turnaround time, providing a robust clinical tool for investigation of complex adrenal and gonadal steroid disorders.

Keywords: adrenal; mass spectrometry; oxygenated androgens.

OTHERS

40 (3230) DIAGNOSTIC YIELD OF EXOME SEQUENCING IN INDETERMINED 46,XY DIFFERENCES OF SEX DEVELOPMENT (DSD): INSIGHTS FROM 80 PATIENTS

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Introduction: Differences of sex development (DSD) are a group of congenital conditions in which there is a discordance between chromosomal, gonadal, and/or anatomical sex. 46,XY DSD are characterized by a broad phenotypic spectrum, ranging from micropenis to atypical or female external genitalia, with or without the presence of Müllerian derivatives. Their diagnosis is challenging due to the wide phenotypic variability, difficulties in hormonal assessment, and the clinical overlap among different etiologies. In this context, genetic sequencing has become an important tool for elucidating the underlying causes of XY DSD. **Objective:** To perform exome sequencing in 80 patients diagnosed with 46,XY DSD without a previously defined genetic cause. **Patients and methods:** 80 index cases with a diagnosis of 46,XY DSD of unknown molecular etiology were included and underwent exome sequencing. **Results:** Among the 80 46,XY DSD subjects, pathogenic or likely pathogenic variants were identified in 14 individuals, corresponding to a diagnostic yield of 17.5%. Of these, 8 out of 51 patients with indeterminate DSD harbored variants in AR (n = 2), SRD5A2 (n = 2), HSD17B3 (n = 1), NR5A1 (n = 1), MYRF (n = 1), and PPP1R12A (n = 1). In subjects with complete gonadal dysgenesis (CGD), 4 out of 12 had variants in PPP1R12A (n = 1), SRY (n = 2), and DHH (n = 1). Among those with partial gonadal dysgenesis (PGD), 1 out of 10 carried a variant in DHX37, while a single patient with hormonal diagnosis of 17β-HSD3 deficiency had a homozygous variant in HSD17B3. Additionally, variants of uncertain significance (VUS) were identified in 8 patients, involving the genes DHX37, GATA4, MYRF, and MAP3K1. **Conclusion:** Exome sequencing enabled the identification of pathogenic and likely pathogenic variants in a subset of individuals with 46,XY DSD, including variants in both well-established and emerging DSD-associated genes. Despite this, the overall diagnostic yield remained relatively low (17.5%), highlighting the genetic heterogeneity and complexity of these conditions. The detection of variants of uncertain significance further underscores the need for functional studies, segregation analyses, and data sharing to clarify their clinical relevance. Additionally, these findings support the importance of complementary approaches, such as genome sequencing, to investigate non-coding regions and improve diagnostic rates in 46,XY DSD.

Keywords: DSD; exome; 46,XY.

OTHERS

41 (3411) EVALUATION OF INSULIN RESISTANCE PREVALENCE USING THE TYG INDEX AND ASSOCIATED RISK FACTORS IN A COHORT OF BRAZILIANS ELDERLY

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Introduction: Aging is associated with progression of insulin resistance (IR), characterized by reduced insulin sensitivity. IR increases the risk of chronic metabolic and cardiovascular diseases, contributing to higher morbidity and mortality. The hyperinsulinemic-euglycemic clamp is the gold standard for IR assessment, while HOMA-IR is widely used in clinical and epidemiological studies. However, both are costly, highlighting the need for simpler alternatives. The triglyceride-glucose (TyG) index has emerged as a practical marker for early IR identification, especially in public health contexts. **Objective:** To determine the prevalence of IR assessed by the TyG index and associated risk factors in an elderly cohort stratified by sex. **Methods:** Clinical, anthropometric, and laboratory data from the SABE cohort (São Paulo, Brazil) were retrospectively analyzed. The TyG index was calculated as Ln[triglycerides (mg/dL) × glucose (mg/dL)/2], using cutoffs of 8.72 for men and 8.92 for women. Normality was assessed by Kolmogorov-Smirnov test. Mann-Whitney, Chi-square, and Fisher's exact tests were applied ($p < 0.05$). **Results:** A total of 1,044 elderly individuals were evaluated, including 670 women (64.3%) and 372 men (35.6%), with two participants presenting missing sex data. The prevalence of IR assessed by the TyG index was 30.9% (n = 323). Participants with IR had a mean age of 70.44 ± 8.13 years. IR was associated with increased body mass index during follow-up, waist circumference, hip circumference, waist-to-hip ratio, systolic and diastolic blood pressure, fasting plasma glucose, glycated hemoglobin, triglycerides, total cholesterol, and HDL-c, but not LDL-c. The prevalence of IR was higher in men than women (39.0% *vs.* 26.4%; $p < 0.001$), as well as in individuals with obesity (36.2%; $p = 0.004$), type 2 diabetes (52.7%; $p < 0.001$), hypercholesterolemia (36.3%; $p < 0.001$), and dyslipidemia (35.0%; $p < 0.001$). Elevated triglycerides were observed in 82.5% of individuals with IR, while reduced HDL-c levels were found in 48.8% ($p < 0.001$). **Conclusion:** IR was highly prevalent in older adults, especially men, and associated with adverse anthropometric, metabolic, and lipid profiles. The TyG index is a useful low-cost tool for identifying individuals at cardiometabolic risk, supporting preventive strategies against metabolic and cardiovascular diseases.
Keywords: index TYG; insulin resistance; elderly.

THYROID

42 (3404) DISRUPTED MEMBRANE TRAFFICKING OF THE SODIUM/IODIDE SYMPORTER AS A POTENTIAL THERAPEUTIC TARGET IN THYROID CANCER

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Introduction: Radioiodine (RAI) uptake treatment effectiveness depends on the sodium-iodide symporter (NIS) localization in the basolateral membrane in the thyrocytes. However, NIS expression is downregulated or restricted to the cytoplasm in tumors, impairing the RAI treatment. Recent studies indicated that several proteins interact with NIS and are involved in cytoplasm trafficking and plasma membrane anchoring. **Objective:** To compare, between normal and tumoral human thyrocytes, the expression of genes encoding proteins involved in NIS mislocalization and functional impairment. **Methods:** Transcriptomic data from TCGA were analyzed to compare the expression of NIS trafficking-related genes (ARF4, COPA, EZR) in normal and tumor samples. Nthy-ori 3-1, TPC1, BCPAP, and KTC2 cells were transfected with pcDNA3.1-HA-NIS and treated with the MAPK inhibitor PD184352 (10-5 M) for 48 h. Gene expression, MAPK activation, NIS function, ROS production, and cell viability were assessed by qPCR, Western blotting, non-radioactive iodide uptake assay, flow cytometry, and MTT assay, respectively. **Results:** TCGA dataset revealed that BRAF-mutant tumors showed decreased expression of ARF4, COPA, EZR. However, in RET/PTC tumors, EZR expression was increased. These findings were consistent with BCPAP and TPC1 gene expression when compared with Nthy-ori cells. Anaplastic KTC2 cells also presented reduced expression of COPA and EZR, but increased ARF4 levels. Interestingly, despite high NIS expression levels in transfected cells, only a modest increase in iodide uptake was observed across all cell lines. Moreover, high SLC5A5 mRNA levels were associated with increased ROS production and pERK levels in Nthy-ori cells. In contrast, both parameters were reduced in BRAF-mutant cells and remained unchanged in TPC1 cells. PD184352 treatment failed to improve NIS function in hNIS-expressing cells, and in TPC1 and KTC2 transfected cells, the treatment reduced NIS expression. The MAPK inhibitor slightly increased the expression of ARF4, COPA, and EZR, but it was not sufficient to improve NIS trafficking to the plasma membrane. **Conclusion:** Restoring iodide uptake requires more than NIS overexpression and MAPK inhibition, suggesting that impaired NIS trafficking machinery in tumor cells may limit functional iodide uptake and highlighting potential targets for investigation in thyroid tumors.
Keywords: NIS; iodide uptake; RAI treatment.

THYROID

43 (3418) IODIDE AVAILABILITY CONTROLS THYROID CELL IDENTITY, ERK SIGNALING, AND REDOX BALANCE

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Introduction: Iodine deficiency (ID) is still a major global public health concern. The effects of ID on thyroid function have been mainly attributed to increased TSH levels secondary to impaired thyroid hormone production. However, the direct effects of ID on thyroid follicular cell gene expression, intracellular signaling, and redox regulation remain poorly understood. **Objective:** To investigate the molecular effects of moderate and severe ID on thyroid cell differentiation, intracellular signaling pathways, epigenetic regulation, and oxidative stress-related genes in vitro. **Methods:** PCCl3 cells (normal rat thyroid follicular cells) were cultured for seven days under three conditions: control (C: 10-8 M NaI), moderate iodine deficiency (MID: 10-9 M NaI), and severe iodine deficiency (SID: iodide-free medium/PBS). TSH concentration remained constant in the medium across all groups. To assess whether the MAPK/ERK signaling pathway mediates ID-induced effects, cells were exposed to ID conditions in the presence or absence of the MEK inhibitor PD0325901 for 48 h. Gene expression was assessed by qPCR and protein levels by Western blotting. **Results:** Both MID and SID significantly increased the expression of thyroid differentiation markers Slc5a5 (NIS), TPO, Tg and TSHR. The thyroid transcription factors Foxe1 and Pax8 were also increased under ID conditions. At the protein level, NIS expression was increased in the MID and SID groups. Interestingly, ID also modulated genes involved in epigenetic regulation, including DNMTS, Hat1, Hdac3, Kdm5c, Kdm6a, and Ezh2. Moreover, Sod1 and Sod2 mRNA levels were reduced in both ID groups compared with the control, whereas Gpx1 mRNA expression was reduced only in the SID group in comparison with the control group. MID and SID reduced phosphorylated ERK and AMPK protein content. Notably, pharmacological inhibition of ERK signaling abolished the ID-induced upregulation of Slc5a5, Pax8, and Foxe1, suggesting that ERK signaling contributes to the transcriptional response to iodide availability. **Conclusion:** ID directly modulated thyroid cell differentiation, epigenetic regulatory pathways, redox-related enzymes, and intracellular signaling pathways independently of TSH. These data reinforce the concept that iodide is not only required for thyroid hormone synthesis, but also actively participates in the maintenance of thyroid follicular cell identity and cellular homeostasis.
Keywords: iodine deficiency; thyroid cells; ERK signaling.

THYROID

44 (3460) IODINE STATUS AND URINARY CONCENTRATIONS OF ENDOCRINE DISRUPTING CHEMICALS IN PREGNANT WOMEN WITH THYROID DISORDERS

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Introduction: Endocrine disrupting chemicals (EDCs) are exogenous substances or mixtures that interfere with hormone action, representing a significant threat during pregnancy. In addition, this period is a critical window of vulnerability where maternal thyroid function is essential for fetal brain development, metabolic regulation, and programming. Given that EDCs often lack a "safe" exposure threshold and may exhibit non-monotonic dose-response curves, understanding their impact and the concurrent iodine status of pregnant women is vital. This study aims to evaluate iodine levels and EDC exposure in pregnant women in women with thyroid disease. **Methods:** This prospective cohort study was conducted at the Thyroid and Pregnancy Clinic in an urban area tertiary center. We recruited pregnant women with pre-existing thyroid disease. Participants completed detailed socioeconomic, nutritional, and lifestyle questionnaires based on the Vigitel system, covering dietary habits, use of plastics, and cosmetic exposure. Maternal blood and urine samples were collected longitudinally across all three trimesters to measure thyroid profiles (TSH, FT4, T3, T4, TBG) and urinary concentrations of iodine (UIC) and EDCs. **Results:** Preliminary data (n = 38) showed a median maternal age of 32.5 years. The median TSH was 1.5 mUI/L and FT4 was 1.4 ng/dL. Iodine analysis revealed a median UIC of 262.5 µg/L; however, significant imbalances were found: 35.8% of the cohort was iodine-deficient (UIC ≤ 149 µg/L) and 36.0% showed excess levels (UIC ≥ 300 µg/L). UIC was positively associated with family income ($p < 0.05$) and negatively associated with the consumption of animal proteins such as eggs, milk, and cheese ($p < 0.02$). Regarding EDCs, median urinary Bisphenol A (BPA) was 3.86 ng/mL, significantly higher than levels reported in Japan (0.40 ng/mL) and Australia (2.42 ng/mL). BPA levels showed strong positive correlations with Bisphenol F ($p = 0.006$), parabens ($p < 0.01$), and the PAH metabolite 9-hydroxyphenanthrene ($p = 0.009$). **Conclusion:** These findings indicate a high prevalence of iodine imbalance and elevated EDC exposure among pregnant women in São Paulo. The high BPA levels compared to global cohorts and the correlations between different chemical classes underscore the complexity of environmental mixtures. These results emphasize the urgent need for environmental monitoring and targeted nutritional guidance during pregnancy to mitigate potential transgenerational metabolic risks.

Keywords: pregnancy; endocrine disruptor; iodine.

THYROID

45 (2984) PREPUBERTAL EXPOSURE TO A GLYPHOSATE-BASED HERBICIDE ALTERS THE THYROID PROTEOMIC PROFILE

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The increasing presence of contaminants in the environment, such as glyphosate-based herbicides, has generated considerable debate. Glyphosate interferes with the synthesis of thyroid hormones and compromises mitochondria, in addition to increasing the production of reactive oxygen species, promoting oxidative stress. This study evaluated the effects of chronic exposure to HBG on the proteomic profile of the thyroid gland of Wistar rats. A total of 90 animals were divided into three groups (0, 0.5, and 5 mg/kg/day of HBG) until DPN60 and DPN90. After exposure, proteomic analyses were performed using nanoLC-MS/MS on an Orbitrap Fusion Lumos, with protein identification via Proteome Discoverer, SEQUEST, and MaxQuant. Comparative analysis demonstrated similar proteins at both doses, but with differences in molecular interactions and gene expression. DCN showed a decrease at both doses, suggesting hyperplastic changes, while CFL1 showed increased expression, indicating greater cellular plasticity and motility. NUP98 demonstrated interaction with HSPA9 at the lower dose, suggesting a response to cellular stress; at the higher dose, it showed underexpression and interaction with proteins related to nuclear architecture and RNA processing. The proteins VAMP7, NSF, and NSFL1C exhibited changes at both doses. At the lower dose, NSFL1C interacted with VAMP7, VAMP3, and PSMD13; at the higher dose, it interacted with proteins associated with the endoplasmic reticulum and Golgi complex. EIF6 showed underexpression at both doses, indicating impaired ribosome biogenesis. The proteins SERPIND1, HRG, and APOA4 suggest vascular alterations and compromised lipid metabolism. Antioxidant proteins demonstrated a dose-dependent response, with increased GSTM1, GSTK1, and COMT at the lower dose and binding of GSTM1 with NQO1 and AGT at the higher dose, indicating genotoxic stress. SEC61A1 showed overexpression at both doses, indicating a compensatory response to cellular stress. The data demonstrate that this exposure promoted significant proteomic changes in the thyroid gland. The 0.5 mg/kg/day dose was associated with adaptive and compensatory responses, while the 5 mg/kg/day dose caused more severe alterations, including nuclear disorganization, impairment of the endoplasmic reticulum and Golgi complex, mitochondrial dysfunction, and genotoxic stress, suggesting harm to the homeostasis and functionality of the thyroid gland. Acknowledgments: Fundação Araucária; CNPq; Capes.

Keywords: oxidative stress; proteomics; thyroid homeostasis.

THYROID

46 (3123) RELATIONSHIP BETWEEN BRAF V600E ALLELE FREQUENCY AND AGGRESSIVE CLINICOPATHOLOGICAL FEATURES IN PAPILLARY THYROID CARCINOMA

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Introduction: Papillary thyroid carcinoma (PTC) usually follows an indolent course with excellent prognosis. Nevertheless, a subset of tumors exhibits aggressive clinicopathological features associated with poorer outcomes. Recently, the allele frequency (AF) of the BRAF V600E mutation has emerged as a potential quantitative biomarker linked to tumor aggressiveness, including intermediate/high American Thyroid Association recurrence risk (ATA RR), lymph node metastasis (LNM), extrathyroidal invasion (EI), and angiolymphatic invasion (AI). **Objective:** To investigate the relationship between BRAF V600E allele frequency and aggressive pathological characteristics in papillary thyroid carcinoma. **Methods:** This study included 116 PTC samples with previously characterized histopathological data regarding ATA RR, LNM, EI, and AI. BRAF V600E mutation status and AF were assessed by quantitative PCR (qPCR). Receiver operating characteristic (ROC) curve analysis was performed to establish the optimal AF threshold, enabling classification into three groups: BRAF-negative, BRAF-positive with AF below the cut-off, and BRAF-positive with AF above the cut-off. **Results:** Among the 116 tumors analyzed, 46 (39.6%) harbored the BRAF V600E mutation. Both mutation positivity and higher AF values were significantly associated with intermediate/high ATA RR ($p < 0.001$), LNM ($p < 0.001$), and EI ($p = 0.030$). ROC analysis identified 16.90% as the optimal AF cut-off. The proportion of tumors classified as intermediate/high ATA RR progressively increased across the groups, reaching 10.0%, 38.5%, and 63.6%, respectively. Likewise, LNM rates rose from 13.0% to 27.3% and 61.9%. EI frequency was also higher in tumors with AF above the cut-off (18.2%) compared with BRAF-negative tumors or those with AF below the threshold (3.6%). **Conclusion:** Quantitative evaluation of BRAF V600E allele frequency may offer additional prognostic information regarding aggressive clinicopathological behavior in PTC. Incorporating AF analysis alongside mutation status could improve risk stratification and contribute to more personalized clinical management.

Keywords: papillary thyroid cancer; BRAF; allele frequency.



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POSTERS

ADRENAL AND HYPERTENSION

01 (3269) ACTH-INDEPENDENT CUSHING'S SYNDROME MANAGED WITH OSILODROSTAT AND CORTICAL-SPARING ADRENALECTOMY: A CASE REPORT

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Case presentation: A 29-year-old woman developed progressive symptoms over two years, including 20-kg weight gain, violaceous striae, acne, moon facies, amenorrhea, edema, refractory depression, psychosis, and a suicide attempt. Laboratory evaluation showed elevated late-night salivary cortisol (0.812 mcg/dL; reference < 0.274), suppressed ACTH (<5 pg/mL), post-1 mg dexamethasone cortisol of 17.6 mcg/dL, and elevated 24-hour urinary free cortisol (783.2 mcg/24 h; reference 58-403), consistent with ACTH-independent Cushing's syndrome. Abdominal CT demonstrated bilateral adrenal nodules measuring 2.8 × 2.3 cm on the right and 2.4 × 2.2 cm on the left. Adrenal vein sampling was inconclusive for lateralization and ketoconazole was initiated. One month later, she developed bilateral pulmonary embolism and started rivaroxaban. To achieve rapid biochemical control, osilodrostat was initiated and titrated to 6 mg/day, reducing urinary free cortisol from 6.4×ULN to 1.6×ULN within two weeks. After clinical stabilization and completion of anticoagulation therapy, a multidisciplinary team opted for unilateral cortical-sparing left adrenalectomy because the left-sided approach allowed preservation of the medial adrenal limb. Eleven days after surgery, serum cortisol was 11 mcg/dL without evidence of postoperative adrenal insufficiency. One month after surgery, she remained off glucocorticoid replacement with restoration of menstrual cycles and marked clinical improvement. Follow-up evaluation demonstrated normalized late-night salivary cortisol (0.151 mcg/dL), urinary free cortisol (20 mcg/24h), and basal cortisol (8.4 mcg/dL), with persistently suppressed ACTH levels (4.9 pg/mL). **Discussion:** Management of ACTH-independent Cushing's syndrome associated with bilateral adrenal nodules is particularly challenging when adrenal vein sampling is inconclusive. Osilodrostat provided rapid biochemical control, while cortical-sparing adrenalectomy resulted in remission without postoperative adrenal insufficiency. Long-term follow-up is needed to assess hypothalamic-pituitary-adrenal axis recovery and possible residual contralateral cortisol secretion from the contralateral adrenal lesion. **Final comments:** This case highlights osilodrostat as an effective bridge therapy and suggests that unilateral cortical-sparing adrenalectomy may be a feasible strategy in selected patients with bilateral adrenal nodules, allowing clinical remission while preserving adrenal function.

Keywords: ACTH-independent Cushing's syndrome; osilodrostat; bilateral adrenal nodules.

ADRENAL AND HYPERTENSION

02 (2915) ADRENAL CORTEX CARCINOMA AND HODGKIN'S LYMPHOMA: A DIAGNOSTIC CHALLENGE OF A SECOND PRIMARY NEOPLASM

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Case presentation: A 26-year-old woman was diagnosed with Hodgkin lymphoma (HL), nodular sclerosis subtype, following an axillary biopsy. PET-CT staging revealed, in addition to lymph node involvement, a hypermetabolic lesion in the right adrenal gland (SUVmax 7,3; 3,4 × 2,5 cm). Chemotherapy was initiated with an ABV regimen, with subsequent addition of dacarbazine, resulting in improvement of B symptoms and regression of all lesions, including the adrenal lesion (SUVmax 3,9; 1,4 × 1,8 cm on follow-up PET-CT). However, the patient developed weight gain, hirsutism, abdominal pain, and fatigue, associated with increased size and metabolic activity of the adrenal mass (SUVmax 15,3; 6,3 × 3,8 cm). Before hormonal results were available, the adrenal lesion was biopsied, and histopathological analysis confirmed adrenocortical carcinoma (ACC). Hormonal evaluation revealed mixed hypersecretion, including hyperandrogenism (DHEA-S 20,051 ng/mL; total testosterone 450 ng/dL; free testosterone 558 pmol/L; androstenedione 4.66 ng/mL), hypercortisolism (urinary cortisol 955.60 mcg/24 h; midnight serum cortisol 30.1 µg/dL; ACTH < 2.0 pg/mL; 11-deoxycortisol 4.48 ng/mL), and primary hyperaldosteronism (aldosterone 22.5 ng/dL; renin < 4.0 µIU/mL; potassium 3.3 mEq/L). The patient underwent right adrenalectomy, with histopathological confirmation of high-grade ACC (Weiss score 9; Ki-67 index 70%). Adjuvant therapy with EP regimen and mitotane was indicated. Genetic testing did not identify pathogenic variant in the TP53 gene. **Discussion:** ACC is a rare, aggressive neoplasm and its synchronous association with HL is uncommon. In patients with lymphoma, adrenal masses are often interpreted as secondary involvement, which may delay the diagnosis of a primary adrenal neoplasm, although this occurs in less than 5% of cases. Clinical and laboratory findings of mixed hormonal hypersecretion strongly suggested a primary adrenocortical origin, making invasive diagnostic procedures unnecessary. Moreover, adrenal biopsy is formally contraindicated in suspected ACC, as it is associated with worse overall survival due to the risk of tumor dissemination, capsular rupture and upstaging in early-stage disease (T1–T2). Therefore, surgical resection without prior biopsy remains the gold standard. **Final considerations:** Early recognition of clinical and laboratory abnormalities is essential to guide diagnosis and management, allowing early treatment and avoiding harmful invasive procedures.

Keywords: adrenocortical carcinoma; lymphoma; hyperandrogenism.

ADRENAL AND HYPERTENSION

03 (3320) ASSOCIATION BETWEEN CONGENITAL ADRENAL HYPERPLASIA AND REPRODUCTIVE FUNCTION IN ADULT MEN: A SYSTEMATIC REVIEW

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Introduction: Congenital adrenal hyperplasia (CAH), commonly caused by 21-hydroxylase deficiency, is a rare genetic disorder that impairs cortisol synthesis and exposes patients to chronic complications. In adult males, infertility affects more than 40% of cases, even under adequate hormonal management. Testicular adrenal rest tumors (TARTs), prevalent in up to 94% of adults with CAH, obstruct the seminiferous tubules, cause progressive gonadal failure, and suppress the hypothalamic-pituitary-gonadal axis via androgen excess. Despite current guidelines advocating early ultrasonographic screening, evidence indicates that 85% of patients reach adulthood without gonadal assessment, underscoring significant gaps in reproductive healthcare. **Objective:** To review the available evidence assessing the correlation between congenital adrenal hyperplasia and male infertility, as well as to investigate the origin and prognosis of this condition in patients with the disease. **Methods:** A systematic review was conducted following PRISMA guidelines, searching PubMed and Embase databases from 2012 to 2024, using the descriptors "congenital adrenal hyperplasia" AND "infertility" OR "male infertility". Nine complete and freely Available studies were included, selected by two independent authors using the Rayyan platform. **Results:** A total of 472 records were identified; 9 were included after full reading. Sample sizes ranged from 2 to 219 participants. Compared to controls, classic CAH patients showed pathological semen analysis in 43%–100% of cases, with findings spanning from azoospermia to severe morphology and motility abnormalities. TARTs, detected in 14%–86% of patients, negatively affected seminal parameters and testicular volume. Hormonally, patients exhibited reduced testosterone and inhibin B, elevated FSH, and gonadotropic suppression due to adrenal androgen excess. Paternity rates were lower than healthy men and the general population average. Longitudinal follow-up showed TART stability over ten years, with Sertoli cell decline in larger tumors. **Conclusion:** The analyses confirm significant reproductive impairment in men with classic CAH compared to healthy men, due to seminal alterations, gonadal dysfunction, and the high prevalence of TARTs. Early longitudinal monitoring may be a potential optimizer of reproductive outcomes and improving paternity rates in this population.
Keywords: adrenal hyperplasia, congenital; infertility; men.

ADRENAL AND HYPERTENSION

04 (3527) BILATERAL PHEOCHROMOCYTOMA IN A YOUNG WOMAN: CASE REPORT

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Case presentation: J.A.M., female, 18 years old, diagnosed with hypertension (220 x 140 mmHg) at age 16. Besides hypertension, she presented with headache, diaphoresis, and palpitations. On 07/26/2024: plasma epinephrine: 287 (RR: 84 pg/mL), plasma norepinephrine: 10,817 (RR: 420-600 pg/mL), dopamine: 201 (RR: 30 pg/mL), chromogranin A: 283 (RR: 95 ng/mL). Abdominal MRI: bilateral adrenal masses, with T2-hypersignal and T1-low signal, regular contours and heterogeneous enhancement, predominantly hypervascular upon contrast infusion with diffusion restriction on echo-planar sequence, measuring 6.2 x 4.9 x 5.0 cm on the right and 5.0 x 3.6 x 5.1 cm on the left. Preoperative preparation was performed with doxazosin started two weeks before the procedure, at a dose of 4 mg/day, with increments of 2 mg per day until reaching 16 mg/day. After the first week of doxazosin, atenolol 25 mg/day was associated. The patient maintained blood pressure levels around 120 × 80 mmHg before the bilateral adrenalectomy surgical procedure, which occurred without complications. Histopathological: bilateral pheochromocytoma, without capsular invasion. Postoperatively (04/29/2025): plasma metanephrines 15.1 (RR: 0.50 nmol/L), normetanephrines 90.8 (RR: 0.90 nmol/L). Currently using prednisone 7.5 mg/day. **Discussion:** Pheochromocytoma is a rare neuroendocrine neoplasm derived from chromaffin cells of the adrenal medulla, associated with high cardiovascular morbidity and mortality. Bilateral cases are uncommon and frequently related to hereditary syndromes. PPGLs constitute the endocrine tumors with the highest heritability, with important association to germline mutations involving RET, VHL, and SDHx genes. Presentation in a youth, associated with tumor bilaterality and important catecholaminergic hypersecretion, makes genetic investigation mandatory. The increase in plasma catecholamines corroborated the functioning pheochromocytoma diagnosis, while MRI allowed proper localization. Excluded hyperparathyroidism and medullary thyroid carcinoma, clinically ruling out MEN 2. Preoperative preparation with alpha-adrenergic blockade was fundamental to reduce surgical risk. **Conclusion:** The report highlights the importance of early PPGL suspicion in young patients with severe hypertension and adrenergic symptoms. Tumor bilaterality reinforces the need for genetic investigation and specialized longitudinal follow-up.
Keywords: pheochromocytoma; hypertension; adrenalectomy.

ADRENAL AND HYPERTENSION

05 (3022) BRONCHIAL PARAGANGLIOMA ASSOCIATED WITH A GERMLINE SDHA MUTATION: A RARE CASE WITH FAMILIAL AGGREGATION

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Case report: A 54-year-old male patient, previously healthy, presented in 2013 with hemoptysis and dyspnea. He underwent bronchoscopy, which revealed a bronchial wall paraganglioma, and was subsequently referred for lobectomy and clinical follow-up. At the time of diagnosis, he had no clinical manifestations suggestive of tumor functionality. In December 2025, genetic testing was performed to investigate a hereditary pheochromocytoma/paraganglioma syndrome, identifying a heterozygous deletion on chromosome 5 (chr5: 218,470–235,456) involving the SDHA gene, classified as a pathogenic variant. Family screening revealed the same variant in both of his children, who were referred for surveillance. **Discussion:** Paraganglioma associated with germline mutations in the SDHA gene (PGL5) is a rare entity, accounting for approximately 2.8% of hereditary PPGL cases. SDHA encodes a subunit of mitochondrial complex II, and its inactivation leads to a pseudohypoxic state. This process results in increased angiogenesis and cellular proliferation, supporting its role as a tumor suppressor gene. Clinically, SDHA-associated tumors present distinctive features, including a wide age range at diagnosis, predominance of solitary tumors, and higher frequency of head and neck or abdominal paragangliomas. In this context, bronchial involvement, as observed in this case, is atypical and rarely described in the literature, expanding the spectrum of SDHA-associated manifestations. In contrast to other SDHx genes, most tumors are non-functioning. Additionally, the absence of a family history is common, reflecting the low penetrance of SDHA mutations. In this case, however, the identification of the same pathogenic variant in two first-degree relatives highlights familial aggregation. When clinically manifest, these tumors may exhibit aggressive behavior, with a significant risk of metastatic disease. In the present case, the favorable outcome following surgical resection underscores the importance of early diagnosis and management. **Final comments:** Current guidelines recommend structured surveillance for carriers of SDHA mutations, including clinical monitoring, periodic measurement of metanephrines, and imaging studies. However, surveillance strategies should be individualized. Genetic counseling is essential, with cascade testing recommended for first-degree relatives. Overall, these findings reinforce the recognition of SDHA as a rare and distinct clinical entity.

Keywords: bronchial paraganglioma; SDHA mutation; hereditary cancer predisposition.

ADRENAL AND HYPERTENSION

06 (3430) CLINICAL AND BIOCHEMICAL STATUS OF PATIENTS DIAGNOSED WITH CUSHING'S SYNDROME FOLLOWED AT A SPECIALIZED OUTPATIENT CLINIC OF A UNIVERSITY CENTER IN THE STATE OF PARÁ

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Introduction: Cushing's syndrome is a condition characterized by signs and symptoms resulting from excess cortisol, leading to important clinical repercussions such as central obesity, violaceous cutaneous striae, proximal muscle weakness, hirsutism, arterial hypertension, and hyperglycemia. Early diagnosis, differentiation between endogenous and exogenous etiologies, and specialized follow-up are essential to reduce morbidity and mortality and improve patients' quality of life. **Objective:** To evaluate the clinical and biochemical status of patients diagnosed with Cushing's syndrome followed at a specialized outpatient clinic in the state of Pará. **Methods:** This is a descriptive, quantitative, retrospective, and cross-sectional study conducted through the analysis of medical records of patients with confirmed diagnosis of Cushing's syndrome treated at a specialized outpatient clinic in the state of Pará. Epidemiological characteristics, clinical manifestations, biochemical profile, etiology, treatment, disease remission, and outpatient follow-up were evaluated. In addition, cases of exogenous etiology were excluded. **Results:** Twenty-five patients were included, of whom 88% were female, with diagnostic predominance between 20–39 years of age (84%). The main etiology was pituitary adenoma (95.6%), whereas 4.4% presented primary adrenal Cushing's syndrome. Surgical procedures were performed in 92% of cases, predominantly transsphenoidal surgery (87%). Clinical improvement after treatment was observed, especially regarding systemic arterial hypertension (60%; $p = 0.002$) and cutaneous striae (48%; $p = 0.002$). There was a significant reduction in post-dexamethasone cortisol levels (17.0 ± 11.7 to 3.2 ± 5.1; $p < 0.001$) and late-night salivary cortisol (45.3 ± 51.9 to 2.1 ± 1.9; $p = 0.008$). The remission rate was 80%, and 68% of patients maintained regular follow-up at the service. **Conclusion:** Cushing's syndrome predominantly affected young women, with pituitary adenoma as the main etiology. Management at a specialized center associated with a surgical approach was related to clinical and biochemical improvement, in addition to high remission rates, reinforcing the importance of early diagnosis, adequate treatment, and longitudinal follow-up.

Keywords: Cushing's syndrome; epidemiology; clinical analysis.

ADRENAL AND HYPERTENSION

07 (3058) CLINICAL AND MOLECULAR DIAGNOSTIC PROFILE OF PATIENTS WITH PHEOCHROMOCYTOMAS AND PARAGANGLIOMAS AT A TERTIARY CENTER

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Introduction: Pheochromocytomas and paragangliomas (PPGLs) exhibit the highest degree of heritability among human tumors (30%–40%). Phenotypic and molecular characterization are fundamental steps for adequate management of these patients. Relying exclusively on clinical signs often leads to diagnostic delays or errors, making biochemical screening and molecular analysis essential for precision medicine. **Objective:** To evaluate the clinical, diagnostic, therapeutic, and genetic profile of patients with secretory PPGL followed at a tertiary center. **Methods:** A longitudinal retrospective observational study was conducted with 59 patients. Demographic, clinical, biochemical, surgical, imaging, anatomopathological, and preliminary germline exome data were analyzed. **Results and discussion:** The cohort was predominantly female (79.7%), with a median age at diagnosis of 46.8 years, consistent with the reported peak age (30–50 years). Adrenal tumors predominated (81.4%), in agreement with the 80%–85% rate described globally. Sustained hypertension (66.1%), palpitations (61%), headache (49.1%), and diaphoresis (42.4%) were the main manifestations. The rate of metastatic disease at diagnosis was 1.6%, lower than the approximately 10% reported in previous series. Surgery was the main treatment, after alpha-adrenoceptor blockade, with intraoperative complications in 24.5% of cases, within the 11%–30% range described in the literature. Immunohistochemistry showed the expected pattern, with chromogranin A and synaptophysin positivity and focal S100 expression. Clinically recognized hereditary forms were identified in only 6.7% of cases, suggesting underdiagnosis compared with the expected 20%–40% frequency. Incidentalomas accounted for 52.5% of diagnoses in the most recent period (2012–2025). Preliminary germline exome analysis of 29 sequenced patients identified pathogenic variants in five cases: VHL (n = 2), RET (n = 1), and SDHB (n = 2). Among these, the two patients with SDHB variants had no previous known genetic syndrome and had metastatic disease. Ten patients had variants of uncertain significance and 14 had only benign or likely benign variants. **Conclusion:** This study highlights the clinical heterogeneity of PPGL and the need for structured diagnostic and genetic strategies to improve accuracy and reduce delays. Germline exome sequencing may refine hereditary risk assessment, guide individualized management and support systematic family screening.

Keywords: paraganglioma; molecular; heritability.

ADRENAL AND HYPERTENSION

08 (3074) CUSHING SYNDROME AND RESIDUAL CARDIAC CHANGES IN PATIENTS IN REMISSION: A SYSTEMATIC REVIEW

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Introduction: Cushing syndrome (CS) has a notable cardiovascular impact due to hypercortisolism. However, even after clinical remission, residual cardiac changes may persist, representing a scenario that requires deeper investigation. **Objective:** To evaluate residual cardiac changes in patients with CS in remission. **Methods:** This systematic review was conducted using the PICO strategy and according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. A search was conducted for studies published in the last 5 years in English, Portuguese, and Spanish in PubMed, Scopus, and Embase databases using the MeSH terms Cushing Syndrome and Heart combined with the Boolean operator "AND". 639 studies were identified and underwent double-blind screening. Inclusion criteria were studies evaluating patients with Cushing syndrome in clinical remission, investigation of cardiac changes using imaging tests and/or cardiac biomarkers, randomized clinical trials, observational and multicenter studies. The exclusion criteria were studies evaluating only patients with active Cushing syndrome, resulting in 4 articles for data extraction to answer the question: "What are the residual cardiac changes in patients with Cushing syndrome in clinical remission?" **Results:** Before remission, compared with a healthy control group, patients presented increased pericardial fat index (28.3 vs. 11.4 g/m²; p = 0.004) and cardiac adipose tissue (58.5 vs. 30.4 g/m²; p < 0.001), however, even after remission, at 8.9 months, no statistically significant reduction was observed (23.9 vs. 23.8 g/m²; p = 0.95 and 57.4 vs. 46.9 g/m²; p = 0.13, respectively). Even after prolonged remission of 88 months, patients showed, compared with the control group, increased global wasted work (97 vs. 75 mmHg%; p < 0.001), N-terminal pro-B-type natriuretic peptide (81 vs. 42 pg/mL; p < 0.001), and reduced global longitudinal strain (20.4 vs. 21.4%; p < 0.001). Patients in remission for at least 6 months, when compared with a control group composed of individuals with nonfunctioning adrenal incidentalomas, showed increased left ventricular mass index (51.0 vs. 41.8 g/m²; p = 0.013) and reduced right ventricular ejection fraction (54.6 vs. 59.5%; p = 0.044). **Conclusion:** Patients with CS in clinical remission may present residual cardiac changes, suggesting persistent myocardial remodeling and increased cardiovascular risk, highlighting the need for cardiologic follow-up despite clinical remission.

Keywords: Cushing syndrome; heart; cardiovascular diseases.

ADRENAL AND HYPERTENSION

09 (3439) DIAGNOSTIC APPROACH TO TACHYCARDIA OF ENDOCRINE ORIGIN: DEVELOPMENT OF A CLINICAL FLOWCHART

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Introduction: Tachycardia is a common and nonspecific clinical finding, usually associated with cardiovascular diseases. However, it may also result from endocrinopathies, such as hyperthyroidism and pheochromocytoma, due to their effects on sympathetic activity and cardiovascular regulation. These causes may be underrecognized in clinical practice, favoring diagnostic delay and inadequate management. Structured tools may support diagnostic reasoning and the early identification of these conditions. **Objective:** To develop and validate the content of a clinical flowchart for the investigation of tachycardia of possible endocrine origin. **Methods:** This is a methodological study of development and content validation. The study was conducted in four stages: structured literature search, extraction and organization of evidence, preliminary construction of the flowchart, and validation by expert judges. The search was performed in PubMed/MEDLINE and BVS, using descriptors related to tachycardia, endocrine diseases, hyperthyroidism, pheochromocytoma, and diagnostic approach. Studies from the last five years, in English, Portuguese, or Spanish, addressing clinical manifestations, laboratory evaluation, and diagnostic criteria were included. The information was organized into clinical domains: symptoms, initial tests, suspected endocrinopathies, and referral criteria. The preliminary version was evaluated by specialists regarding clarity, relevance, applicability, and scientific consistency. **Results:** The flowchart was organized into sequential steps: confirmation of persistent tachycardia, exclusion of common non-endocrine causes, identification of signs and symptoms suggestive of endocrine etiology, selection of initial tests, and criteria for specialized referral. Hyperthyroidism and pheochromocytoma were the main conditions included. After expert evaluation, adjustments were made to terminology, clinical sequence, and visual organization. The final version presented objective decision points, accessible language, and a simplified structure to support clinical reasoning among students and healthcare professionals. **Conclusion:** The flowchart showed potential as an educational and clinical support tool for the initial investigation of tachycardia of possible endocrine origin, contributing to improved diagnostic reasoning and guidance for endocrine referral.

Keywords: tachycardia; endocrine diseases; clinical flowchart.

ADRENAL AND HYPERTENSION

10 (3581) DIAGNOSTIC CHALLENGE IN GESTATIONAL HYPERALDOSTERONISM: A RARE CLINICAL PRESENTATION WITH NON-RESISTANT HYPERTENSION AND FLACCID TETRAPARESIS

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Case presentation: A 36-year-old female patient was diagnosed with gestational hypertension during her second pregnancy (2022), managed with methyldopa and subsequently with losartan 100 mg/day with good blood pressure control. During the third trimester, she was transitioned to methyldopa with continued good control. She had previously used spironolactone for four months with good control, discontinued due to excessive polyuria. In the postpartum period, she presented with flaccid tetraparesis affecting the face, hands, and feet, preceded by three days of diarrhea, nausea, and vomiting. On admission in February 2025, blood pressure was 159/98 mmHg, with bilateral flaccid paresis and no abnormalities on cranial computed tomography. Laboratory findings revealed serum potassium of 1.82 mEq/L, sodium 144 mEq/L, bicarbonate 37 mmol/L, and pH 7.47 (hypokalemic metabolic alkalosis). Aldosterone and plasma renin activity were measured, revealing aldosterone of 34.10 ng/dL, plasma renin activity below 0.05 mU/L (aldosterone-to-renin ratio greater than 680), and post-dexamethasone suppression cortisol of 0.92 µg/dL. Spironolactone was initiated. Doppler ultrasonography showed no renal artery stenosis. Computed tomography demonstrated a hypoattenuating nodule in the left adrenal gland measuring 2.0 x 1.6 cm, compatible with adenoma, confirming primary hyperaldosteronism (HAP 1a). Potassium levels normalized with replacement therapy. After six months post-adrenalectomy, losartan was discontinued. **Discussion:** This case highlights the diagnostic challenge of primary hyperaldosteronism during pregnancy, where standard diagnostic criteria may not apply. The patient's presentation, with controlled hypertension during pregnancy despite elevated hormonal markers post-partum, suggests the protective effect of gestational hormones in masking disease manifestations. Small case series suggest an aldosterone-to-renin ratio threshold of > 20 during pregnancy. The clinical deterioration in the postpartum period may result from the loss of protective effects of gestational hormones, which exert antimineralocorticoid actions. **Final comments:** This case illustrates the diagnostic challenge of primary hyperaldosteronism presenting atypically during pregnancy and puerperium, emphasizing the importance of hormonal investigation and the protective effects of gestational hormones in masking the clinical manifestations of this condition.

Keywords: pregnancy; hypertension; adrenal.

ADRENAL AND HYPERTENSION

11 (3519) DIAGNOSTIC CHALLENGES IN AN ATYPICAL PRESENTATION OF GRAVES' DISEASE ASSOCIATED WITH AN ALDOSTERONE- AND CORTISOL-CO-SECRETING ADRENAL ADENOMA: A CASE REPORT

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Case presentation: A 44-year-old previously healthy woman developed fever, cervical pain, odynophagia, weight loss, paroxysmal hypertension, and tachycardia in May 2024. In June 2024, laboratory tests showed TSH 0.01 mIU/L and free T4 4.16 ng/dL, associated with a visible, palpable, mildly tender goiter. Autoimmune hyperthyroidism with possible concomitant subacute thyroiditis was suspected, and methimazole 5 mg/day plus a short course of prednisone were initiated. Thyroid ultrasound demonstrated diffuse thyroid disease with increased Doppler vascularization. TRAb was markedly elevated (35.8 IU/L), while anti-TPO and anti-thyroglobulin antibodies were negative, confirming Graves' disease. Persistent cervical pain suggested an inflammatory component. Within two months, the patient progressed to overt hypothyroidism (TSH 81.67 mIU/L) with low free T4. Methimazole was discontinued and levothyroxine initiated; however, hypothyroidism persisted, raising suspicion of coexistence of stimulating and blocking TRAb activity. Persistent hypertension prompted further investigation, revealing suppressed renin activity, elevated aldosterone levels, and an increased aldosterone-to-renin ratio, consistent with primary hyperaldosteronism. Cortisol also failed to suppress adequately after dexamethasone testing, with ACTH < 5 pg/mL, suggesting mild ACTH-independent hypercortisolism. Plasma catecholamines and metanephrines were negative. Abdominal computed tomography scan identified a 1.5 × 1.2 cm left adrenal nodule compatible with adenoma. The patient underwent left adrenalectomy in July 2025, and histopathology confirmed a benign adrenocortical adenoma. Postoperatively, she showed clinical improvement and blood pressure control. **Discussion:** This case highlights an atypical presentation of Graves' disease associated with painful cervical symptoms and possible coexistence of stimulating and blocking TRAb antibodies, leading to hypothyroidism. It also demonstrates a functioning adrenal adenoma, possibly co-secreting aldosterone and cortisol, causing secondary hypertension. **Conclusion:** Simultaneous involvement of multiple endocrine axes may produce dynamic clinical and laboratory manifestations, posing important diagnostic challenges. Persistent hypertension despite normalization of thyroid function emphasizes the importance of comprehensive evaluation for secondary hypertension, particularly in recent-onset cases.

Keywords: Graves' disease; hypertension; adrenocortical adenoma.

ADRENAL AND HYPERTENSION

12 (3301) EXTRA-ADRENAL LESIONS SIMULATING ADRENAL MASSES: FINAL DIAGNOSES AND IMAGING PITFALLS IN A TERTIARY REFERRAL CENTER COHORT

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Introduction: The widespread use of cross-sectional imaging has increased the detection of adrenal-region masses. While many are hormonally active or clearly benign, a subset remains indeterminate even after thorough endocrine and radiological evaluation. This cohort reviews around 300 adrenal surgeries that have been performed since 2006 in a tertiary center, with approximately 8% involving non-functioning, indeterminate lesions. **Objective:** To determine the final histopathological diagnoses of non-functioning adrenal-region masses, including adrenal mimickers. **Methods:** This retrospective cohort study analyzed patients treated between 2006 and 2025 who presented with adrenal-region lesions on CT or MRI, normal hormonal evaluations per international guidelines, and no signs of adrenal hyperfunction. Two experienced radiologists independently reviewed imaging studies. Forty-six patients underwent unilateral adrenalectomy. Adrenocortical carcinoma cases were excluded. **Results:** The cohort was predominantly female (69.6%), with a mean age of 48.8 years. Imaging was performed for abdominal/lumbar pain (43.5%) or incidentally (38.4%). Most lesions were left-sided (52.2%), with a mean size of 8.67 cm and mean unenhanced attenuation of 25 HU. Final diagnoses included ganglioneuroma (18%), adrenal cysts (13.5%), hemorrhage (9%), leiomyosarcoma (9%), infections (6.5%), lymphangioma (4.5%), schwannoma (4.5%), and sarcoma (4.5%). Rare diagnoses included lymphoma, neuroblastoma, renal cell carcinoma, liposarcoma, metastasis, and inflammatory or vascular lesions. **Conclusion:** CT and MRI alone may not reliably distinguish adrenal from non-adrenal masses. In this series, 45% of lesions were extra-adrenal, emphasizing the importance of expert radiological review and multidisciplinary evaluation in indeterminate cases.

Keywords: adrenal incidentaloma; adrenal mass; ganglioneuroma.

ADRENAL AND HYPERTENSION

13 (3010) FAMILIAL BILATERAL MACRONODULAR ADRENAL DISEASE WITH MULTICENTRIC RENAL CELL CARCINOMA SHOWING DOUBLE HIT ARMC5 PATHOGENIC VARIANT

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Case presentation: A 47-year-old man presented with a right kidney nodule incidentally identified during radiological follow-up. Two years earlier, he had undergone bilateral adrenalectomy due to bilateral macronodular adrenal disease (BMAD). During a decade-long follow-up, consecutive abdominopelvic CT scans revealed multiple hypovascularized solid lesions in both kidneys, the largest measuring 3.1 cm, without evidence of metastatic disease. Three surgical specimens from progressively enlarging renal tumors were analyzed. All tumors showed similar histopathological findings, unusual for clear cell renal cell carcinoma (RCC), particularly the presence of leiomyomatous stroma. Germline analysis identified a pathogenic ARMC5 variant in exon 6 (c.2423A>C; p.H808P). Somatic analysis of RCC tissue demonstrated additional pathogenic ARMC5 alterations, including variants in exon 3 (c.1090C>T; p.R364*) and exon 6 (c.2047_2048insAGTA; p.L683Qfs*56), as well as copy number alterations. No pathogenic alterations were identified in VHL. **Discussion:** ARMC5 is a tumor suppressor gene implicated in proliferative adrenocortical disorders, particularly familial BMAD. Previous reports have associated germline ARMC5 mutations with an increased risk of solid tumors, including meningiomas and pancreatic neoplasms, suggesting a broader oncogenic potential of this gene. We report, to our knowledge, the first case of multicentric and bilateral RCC in a patient with familial BMAD harboring both germline and somatic ARMC5 pathogenic variants. The coexistence of germline and somatic alterations supports a classical “double-hit” mechanism of tumor suppressor gene inactivation. Histopathological features, especially leiomyomatous stroma, overlap with other RCC subtypes, including ELOC-mutated and clear cell papillary RCC, suggesting a shared morphological phenotype. Mechanistically, ARMC5, similarly to elongin C encoded by ELOC, participates in E3 ubiquitin ligase complexes, raising the possibility that its inactivation disrupts protein degradation pathways relevant to RCC development. **Final comments:** Although based on a single patient, this report supports the inclusion of ARMC5 among genes potentially involved in renal tumorigenesis and suggests a possible ARMC5-related cancer predisposition syndrome. These findings reinforce the importance of long-term surveillance in mutation carriers and warrant further studies investigating the role of ARMC5 in hereditary RCC susceptibility.

Keywords: BMAD; renal cell carcinoma; ARMC5.

ADRENAL AND HYPERTENSION

14 (3475) GIANT ADRENAL HEMANGIOMA MIMICKING MALIGNANT NEOPLASM: A DIAGNOSTIC CHALLENGE IN ADRENAL INCIDENTALOMA

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Case presentation: A 68-year-old man with type 2 diabetes mellitus, hypertension, obesity, and a history of smoking (50 pack-years) was recently diagnosed with Child-Pugh B liver cirrhosis of metabolic etiology during investigation of ascites and abdominal distension. Abdominal computed tomography revealed chronic liver disease and a heterogeneous solid right adrenal mass with peripheral calcification, well-defined margins, and dimensions of 9.1 × 8.7 × 7.5 cm, causing compression of the inferior vena cava without evidence of local invasion. Isolated peripancreatic lymphadenopathy was also observed. The patient was referred to endocrinology presenting with unintentional weight loss of 28 kg over 40 days, anorexia, postprandial fullness, and nonspecific abdominal pain. Initial diagnostic hypotheses included adrenocortical carcinoma and pheochromocytoma. Hormonal evaluation showed no clinically significant hypersecretion, with normal plasma metanephrines and post-dexamethasone cortisol of 2.56 µg/dL (RR: < 1,8 µg/dL). Magnetic resonance imaging demonstrated a solid right adrenal lesion, hyperintense on T2-weighted imaging, with hemorrhagic foci on T1-weighted imaging and peripheral globular enhancement with progressive centripetal fill-in after contrast administration, suggestive of adrenal hemangioma. The patient underwent adrenalectomy and is awaiting histopathological results. **Discussion:** Adrenal hemangioma is a rare benign vascular neoplasm, usually nonfunctioning and incidentally detected. Large lesions may mimic malignancy due to heterogeneity, necrosis, hemorrhage, and calcifications. Peripheral nodular enhancement with progressive centripetal fill-in, similar to hepatic hemangiomas, is suggestive of the diagnosis. On magnetic resonance imaging, T2 hyperintensity associated with absence of diffusion restriction may help differentiate adrenal hemangioma from malignant tumors. However, preoperative diagnosis remains challenging, especially in large symptomatic lesions. **Final comments:** Adrenal hemangioma should be considered in the differential diagnosis of large nonfunctioning adrenal masses. Recognition of typical radiological features may support diagnostic suspicion, although definitive diagnosis often depends on surgical resection and histopathological evaluation.

Keywords: adrenal hemangioma; adrenal incidentaloma; adrenal neoplasm.

ADRENAL AND HYPERTENSION

15 (3236) GIANT BILATERAL ADRENAL MASSES AFTER LONG-TERM TREATMENT DISCONTINUATION IN CLASSIC CONGENITAL ADRENAL HYPERPLASIA

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Case presentation: A 39-year-old female was referred to the adrenal outpatient clinic due to bilateral adrenal masses. The patient reported a diagnosis of neonatal classic congenital adrenal hyperplasia (CAH), with ambiguous genitalia at birth and three corrective surgeries during childhood. Treatment with prednisone and fludrocortisone had been discontinued at the age of 21 on her own initiative. After 18 years without medical follow-up, an abdominal CT scan performed for back pain revealed heterogeneous bilateral adrenal lesions measuring 12.0 × 11.7 cm on the left and 6.3 × 5.4 cm on the right, both containing fat components. The patient denied symptoms suggestive of adrenal insufficiency. Physical examination showed generalized skin hyperpigmentation, androgenetic alopecia, deepening of the voice, BMI of 30.9 kg/m², and absence of postural hypotension. Laboratory findings confirmed severe hormonal decompensation: ACTH 7,220 pg/mL (ref: 6–48), 17-OH progesterone 99.8 ng/mL, androstenedione 73.3 ng/mL (ref: 0.3–3.3), and total testosterone 12.9 ng/mL (ref: 0.08–0.48). Morning cortisol was 3.16 µg/dL, and direct renin was 64.8 µUI/mL. Gonadotropins were suppressed. **Discussion:** In 21-hydroxylase deficiency, chronic ACTH overstimulation promotes marked adrenal hyperplasia and, in approximately 20% to 30% of adult patients, large adrenal masses associated with poor hormonal control and sustained ACTH elevation. Adrenal myelolipomas are particularly common in classic CAH, accounting for up to one-third of adrenal masses in this population. These tumors are frequently bilateral and may reach giant dimensions, especially in patients with longstanding uncontrolled disease. The presence of fat-containing adrenal lesions on CT imaging, together with the biochemical profile, strongly suggests adrenal myelolipomas related to long-term untreated CAH, reflecting prolonged inadequate hormonal control. **Final comments:** This case illustrates the severe consequences of treatment abandonment and highlights the importance of an effective transition from pediatric to adult care in patients with classic CAH. Giant adrenal masses and extreme androgen excess are potentially preventable complications with adequate lifelong glucocorticoid and mineralocorticoid replacement. Adrenal imaging should be considered in patients with poor disease control, prolonged treatment interruption, lack of response to intensified therapy, or clinical suspicion of adrenal masses.

Keywords: congenital adrenal hyperplasia; adrenal myelolipoma; treatment discontinuation.

ADRENAL AND HYPERTENSION

16 (3311) GLUCOCORTICOID-REMEDIABLE FAMILIAL HYPERALDOSTERONISM: A CASE SERIES

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Case report: We report six patients with familial hyperaldosteronism type I (FH-I). The first sibling pair, aged 25 and 15 years, developed severe hypertension at ages 13 and 6, with a paternal history of childhood-onset hypertension. Both had elevated plasma aldosterone (41 and 28.7 ng/dL) and suppressed direct renin (<4.0 µU/mL). A prolonged dexamethasone suppression test (Liddle test) showed aldosterone suppression (<4 ng/dL). Dexamethasone 0.25 mg at bedtime reduced aldosterone to 5.2 ng/dL, but it later rose to 24.6 ng/dL with persistently suppressed renin, requiring up-titration to 0.5 mg. The younger sibling also started dexamethasone, with follow-up pending. The second pair, aged 33 and 23 years, had hypertension since ages 13 and 8, with a maternal history of hemorrhagic stroke. Both had elevated aldosterone levels (18 and 22.9 ng/dL) and suppressed plasma renin activity (PRA, <0.3 ng/mL/h). The younger had left ventricular hypertrophy and hypokalemia. In the older brother, dexamethasone 0.25 mg/day reduced aldosterone to 6.88 ng/dL and raised PRA to 0.78 ng/mL/h. The remaining two patients were unrelated men, aged 22 and 44 years, hypertensive since age 19, with aldosterone around 50 ng/dL and suppressed PRA (<0.3 ng/mL/h); one had hypokalemia. In one, dexamethasone 0.25 mg/day reduced aldosterone to 27.6 ng/dL and increased PRA to 1.6 ng/mL/h; the other achieved sustained blood pressure control with dexamethasone 0.25 mg/day plus spironolactone and enalapril. FH-I was confirmed in all six patients by long-range PCR demonstrating the CYP11B1/CYP11B2 chimeric gene, and abdominal CT showed normal adrenals in all cases. **Discussion:** FH-I is a rare monogenic form of primary aldosteronism caused by a chimeric CYP11B1/CYP11B2 gene driving ACTH-dependent aldosterone production. Severe early-onset hypertension before age 20, or primary aldosteronism (PA) with a family history of early hypertension or cerebrovascular disease, should prompt investigation. Dexamethasone was used in five patients, with biochemical improvement in four; one showed an initial response followed by biochemical worsening, requiring dose adjustment. Only one patient had hypokalemia. **Final comments:** FH-I should be investigated in early-onset hypertension, particularly when presenting with severe blood pressure elevation, normal serum potassium, and normal adrenal imaging. Early diagnosis enables targeted low-dose dexamethasone therapy to promote biochemical and clinical PA control.

Keywords: familial hyperaldosteronism; glucocorticoid-remediable aldosteronism; CYP11B1/CYP11B2 chimeric gene.

ADRENAL AND HYPERTENSION

17 (2937) HIGH CLINICAL PENETRANCE AND PREDOMINANT HEAD AND NECK PHENOTYPE IN A FOUR-GENERATION SDHB FAMILIAL CLUSTER: CHALLENGING THE GENOTYPE-PHENOTYPE DOGMA

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Introduction: Succinate dehydrogenase subunit B (SDHB) mutations are classically associated with extra-adrenal abdominal paragangliomas (PPGL) and a high risk of metastatic disease. Head and neck paragangliomas (HNPGL) are more frequently linked to SDHD variants. This study characterizes a large familial cluster harboring an SDHB germline missense mutation, exhibiting an unusual HNPGL tropism and high penetrance across four generations. **Methods:** A retrospective and prospective family cohort study was conducted following the identification of a 48-year-old female proband with metachronous pheochromocytoma (30y) and HNPGL (48y). Cascade genetic testing and clinical-radiological screening (MRI and biochemical profile) were performed on 10 relatives. **Results:** Ten SDHB mutation carriers were identified across four generations. The estimated clinical penetrance was 60% (6/10). The phenotypic spectrum was predominantly HNPGL, including a metastatic case in a third-generation male. Notable atypical findings included a suspected SDH-deficient GIST, initially reported as gastric cancer at age 33 (G1) and a meningioma at age 26 (G2). In the fourth generation, early screening identified a symptomatic HNPGL in a pediatric/young adult patient. Biochemical evaluation revealed a predominantly non-secretory profile in HNPGL cases, emphasizing the limitations of catecholamine screening for this specific lineage. **Conclusion:** This familial series demonstrates a remarkably high penetrance for SDHB and a rare HNPGL-dominant phenotype. The occurrence of suspected GIST and meningioma suggests a broader phenotypic expansion of the pseudo-hypoxia pathway. These findings underscore the imperative for early cascade screening and lifelong surveillance in SDHB carriers, even in lineages presenting with less aggressive clinical precursors.
Keywords: paraganglioma; succinate dehydrogenase subunit B; familial.

ADRENAL AND HYPERTENSION

18 (3318) INFLUENCE OF GENETIC TESTING ON CORTICOSTEROID DISCONTINUATION AND REDUCTION OF TREATMENT-RELATED CONSEQUENCES IN NONCLASSICAL CONGENITAL ADRENAL HYPERPLASIA IN A HETEROZYGOUS PATIENT: A CASE REPORT

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Case presentation: Female patient has been followed since age 6 years and 7 months due to adult-type hidrosis onset at age 4, with no further pubertal signs. Initially, she presented bone age advancement (6y10m at chronological age 4y3m), predicted height of 1.72 m, 17-hydroxyprogesterone (17-OHP) of 185 ng/dL, and dehydroepiandrosterone sulfate (DHEA-S) of 71.8 ng/dL, consistent with premature adrenarche. She subsequently developed increased growth velocity and persistent bone age advancement (11 years at 7y10m). Further investigation revealed basal 17-OHP of 235 ng/dL and post-ACTH stimulation 17-OHP of 466 ng/dL, below the diagnostic cutoff for non-classical CAH. Hydrocortisone acetate was initiated at age 9 due to bone age advancement and compromised final height risk, later replaced by dexamethasone. Partial hormonal control was achieved, with adequate pubertal progression, menarche at age 12, and regular cycles. In 2024, after 14 years of glucocorticoid use, bilateral punctate posterior subcapsular cataracts were detected without significant visual impact. One year later, CYP21A2 molecular analysis by sequencing and MLPA revealed a pathogenic variant in heterozygosity associated with gene duplication carrying a functional allele. Dexamethasone was withdrawn without adverse effects, and the case was diagnostically reclassified. **Discussion:** NC-CAH due to 21-hydroxylase deficiency presents a variable phenotype according to the degree of enzymatic deficiency, with genotype-phenotype correlation in approximately 98% of cases. The post-stimulation 17-OHP value (466 ng/dL) fell below the diagnostic threshold of 1000 ng/dL, insufficient to confirm NC-CAH by current biochemical criteria. Nevertheless, given bone age advancement, glucocorticoid therapy was instituted. However, heterozygous CYP21A2 carriers may exhibit elevated basal and post-stimulation 17-OHP, and molecular analysis is more sensitive for diagnosis in such cases. Genetic testing identified heterozygosity with a functional allele, leading to case reclassification and medication withdrawal. Cataract development, a pharmacological complication, reinforces the importance of early genotyping in these situations. **Final remarks:** The suggestion of premature adrenarche with atypical progression led to prolonged glucocorticoid use and cataracts. CYP21A2 genotyping enabled diagnostic reclassification, allowing treatment withdrawal and highlighting the decisive role of genetics in clinical practice.
Keywords: adrenal hyperplasia, congenital; genetic testing; glucocorticoids.

ADRENAL AND HYPERTENSION

19 (3487) PERSISTENT HYPERANDROGENISM AFTER ADRENALECTOMY: A DIAGNOSTIC CHALLENGE

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Case presentation: A previously healthy 29-year-old woman presented two years earlier with fatigue, hair loss, and hirsutism, prompting endocrinological evaluation. Ferriman-Gallwey scoring was limited by hair removal practices, but increased terminal hair growth was noted in the chin and mandibular regions. She denied acne and menstrual irregularities. Initial laboratory evaluation showed elevated androgen levels: dehydroepiandrosterone sulfate (DHEA-S) 876 mcg/dL (reference range [RR] 99–407), androstenedione 215 ng/dL (RR 39–165), and total testosterone 74 ng/dL (RR < 63). Abdominal MRI demonstrated focal 0.8 cm thickening of the left adrenal gland suggestive of adenoma. The hypothalamic-pituitary-adrenal axis and 17-hydroxyprogesterone were normal. The patient underwent left adrenalectomy with immediate normalization of androgen levels. Histopathology identified two encapsulated nodules composed of adrenal cortical cells, favoring microadenomas, the largest measuring 2 mm. Five months later, DHEA-S increased again (532 mcg/dL), followed by loss to follow-up. One year later, androgen levels rose mildly, requiring spironolactone for symptom control. Repeat imaging showed no hormone-secreting lesions. Chest CT identified a 1.1 cm indeterminate nodular lesion adjacent to the thoracic esophagus. FDG PET-CT demonstrated no abnormal uptake. Adrenal venous sampling is planned to localize the hormone-secreting source. **Discussion:** Ectopic adrenal tissue results from incomplete migration of adrenocortical cells and is rare in adulthood. It may occur at multiple anatomical sites and exhibit autonomous hormonal activity. In this case, the immediate postoperative decline in androgen levels supports an adrenal source of hyperandrogenism, potentially related to ectopic tissue. Although persistent hypersecretion raises concern for malignancy, the indolent course and negative PET-CT findings make malignancy less likely. **Final comments:** This case highlights the importance of considering ectopic adrenal tissue in persistent hyperandrogenism and underscores the challenges of identifying it using conventional imaging modalities.

Keywords: hyperandrogenism; adrenocortical adenoma; adrenalectomy.

ADRENAL AND HYPERTENSION

20 (2911) PRIMARY ADRENAL INSUFFICIENCY AS THE INITIAL MANIFESTATION OF BILATERAL ADRENAL HISTOPLASMOSIS: A DIAGNOSTIC CHALLENGE

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Introduction: Primary adrenal insufficiency is potentially life-threatening, with autoimmune, infectious, and infiltrative causes. In endemic regions, histoplasmosis should be considered, especially with bilateral adrenal involvement, as it may present nonspecifically and mimic neoplastic disease. **Case report:** A 49-year-old man with diabetes, dyslipidemia, and vitamin deficiencies presented with progressive fatigue, unintentional weight loss (6 kg in 2 months), dizziness, and febrile episodes. On admission: Hb 15.8 g/dL, leukocytes 6,800/mm³, urea 65 mg/dL, creatinine 0.7 mg/dL, sodium 133 mEq/L, potassium 5.7 mEq/L, CRP 16.1 mg/L. Abdominal CT showed bilateral adrenal masses (right 7.5 × 5.4 cm; left 6.2 × 5.2 cm) with heterogeneous density (~30–40 HU), central hypodense areas (<20 HU), minimal contrast enhancement, irregular internal septations, and mild periadrenal fat stranding. Chest CT revealed small nonspecific pulmonary nodules (<5 mm). Hormonal evaluation: elevated ACTH (565 pg/mL), low basal cortisol (4.19 µg/dL), low aldosterone (2.06 ng/dL), androstenedione <0.3 ng/mL; plasma metanephrines were within normal range. Intravenous hydrocortisone (100 mg every 8 hours) was started with marked clinical improvement. MRI showed bilateral adrenal lesions with heterogeneous T2 signal, central areas suggesting necrosis, and mild peripheral contrast enhancement. Adrenal biopsy demonstrated necrotizing granulomatous inflammation with multinucleated giant cells, without evidence of malignancy. Further workup confirmed adrenal histoplasmosis. The patient received amphotericin B for 14 days with favorable clinical evolution and outpatient follow-up. **Discussion:** Disseminated histoplasmosis can involve both adrenal glands and is a recognized cause of primary adrenal insufficiency, particularly in endemic areas. Clinical presentation is often nonspecific and may mimic neoplastic processes. Hormonal assessment is essential to confirm adrenal insufficiency and to exclude functioning tumors. Histopathology typically shows necrotizing granulomatous inflammation. Treatment includes systemic antifungal therapy and hormone replacement, which are crucial to reduce morbidity and mortality. **Conclusion:** Histoplasmosis should be included in the differential diagnosis of bilateral adrenal masses associated with adrenal insufficiency. Integration of clinical, hormonal, imaging, and histopathological findings is essential for early diagnosis and appropriate management.

Keywords: adrenal insufficiency; histoplasmosis; fungal infection.

ADRENAL AND HYPERTENSION

21 (3024) PRIMARY ALDOSTERONISM IN PATIENTS WITH ADRENAL INCIDENTALOMA AND SYSTEMIC ARTERIAL HYPERTENSION: CLINICAL, LABORATORY, AND IMAGING CHARACTERISTICS

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Introduction: Adrenal incidentaloma (AI) is a common finding in imaging studies, occurring in up to 8% of abdominal CT scans. The coexistence of systemic arterial hypertension (SAH) in this population suggests the possibility of adrenal hyperfunction, particularly primary aldosteronism (PA), which is a potentially curable cause of secondary hypertension. Data on the prevalence and clinical profile of patients with AI and PA in the Brazilian population are scarce. **Objective:** To describe the clinical, laboratory, and imaging characteristics of patients with AI and SAH, and to compare findings between groups with positive and negative PA screening results. **Methods:** This cross-sectional study involved a medical record review of 58 patients with AI and SAH. PA screening was performed without discontinuing or altering medications and was classified as positive when both low renin (plasma renin activity ≤ 1 ng/mL/h or renin ≤ 8.2 mU/L) and elevated aldosterone (≥ 10 ng/dL) were present. Numerical data are presented as median (interquartile range) or percentage. **Results:** The median age at diagnosis was 59.5 years (56.0–66.8), with a predominance of women (72.9%, n = 43). Resistant hypertension was identified in 21.1% of cases. Regarding PA screening, 14 patients (24.1%) tested positive (PA+), 29 (50.0%) tested negative (PA–), and 15 (25.9%) were not evaluated. Hypokalemia was observed in only 3 out of 37 patients (8.1%). In the PA+ group, 25.0% of those with available data had cortisol levels > 1.8 µg/dL after a 1 mg dexamethasone suppression test. Median age, dysglycemia, and body mass index were similar between groups. However, PA+ patients used a higher number of antihypertensive medications [3.5 (3.0–4.0) vs. 2.0 (2.0–2.25)] and had a higher frequency of resistant hypertension (46.2% vs. 7.1%). The largest nodule diameter was similar between groups [1.6 cm (1.3–2.4) vs. 2.0 cm (1.5–2.6)], but bilateral disease was more frequent in PA+ patients (38.5%) compared to the PA- group (17.2%). **Conclusion:** In our series, PA screening was positive in approximately one-quarter of patients with AI and SAH. The PA+ group required more antihypertensive medications and had a higher frequency of resistant hypertension. These findings highlight the effectiveness of a simplified initial screening approach and emphasize the importance of systematic PA screening in all patients with both adrenal incidentaloma and hypertension.

Keywords: primary aldosteronism; adrenal incidentaloma; screening.

ADRENAL AND HYPERTENSION

22 (2928) PRIMARY ALDOSTERONISM PRESENTING AS SEVERE HYPOKALEMIA AND CARDIAC ARREST: A CASE REPORT

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Case presentation: A 71-year-old female with a long-standing history of hypertension since the age of 25, treated with multiple antihypertensive agents including an angiotensin receptor blocker, calcium channel blocker, beta-blocker, thiazide diuretic, and mineralocorticoid receptor antagonist, presented with progressive muscle weakness, recurrent falls, and recent diarrhea. She was admitted to the emergency department with a reduced level of consciousness, requiring orotracheal intubation. Electrocardiography demonstrated findings consistent with hypokalemia, including first-degree atrioventricular block, flattened T waves, and prominent U waves. Laboratory evaluation revealed severe hypokalemia, with undetectable serum potassium levels (<1 mEq/L). The patient developed malignant ventricular arrhythmias, including pulseless ventricular tachycardia and ventricular fibrillation, progressing to cardiac arrest, which was successfully reversed with aggressive potassium replacement and defibrillation. Transthoracic echocardiography showed preserved systolic function without structural abnormalities. Further evaluation revealed renal potassium wasting, elevated aldosterone levels (26.2 ng/dL), and a 1.5 cm right adrenal nodule consistent with an adenoma. After clinical stabilization, treatment with spironolactone was initiated and optimized, resulting in progressive normalization of serum potassium levels, full neurological recovery, and clinical stability. The patient was discharged with referral for specialized outpatient follow-up. **Discussion:** Primary aldosteronism is the most common cause of secondary hypertension and remains underdiagnosed, particularly in patients with resistant hypertension. Autonomous aldosterone secretion leads to renal potassium wasting and may result in hypokalemia, which is typically mild to moderate. In this case, the presence of a precipitating factor contributed to profound hypokalemia, culminating in malignant ventricular arrhythmias and cardiac arrest. The absence of structural heart disease supports a metabolic etiology. This case illustrates a severe and atypical presentation of a potentially treatable condition. **Final comments:** This report highlights primary aldosteronism as a potentially fatal cause of severe hypokalemia and malignant arrhythmias. Screening in patients with resistant hypertension or unexplained hypokalemia is essential for early diagnosis and targeted treatment, reducing morbidity and mortality.

Keywords: primary aldosteronism; hypokalemia; ventricular arrhythmia.

ADRENAL AND HYPERTENSION

23 (2893) PRIMARY ALDOSTERONISM WITH ATYPICAL POST-ADRENALECTOMY COURSE: MINERALOCORTICOID INSUFFICIENCY AND RENAL DYSFUNCTION

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A 51-year-old man with long-standing hypertension and a history of multiple hospitalizations due to recurrent hypokalemia, previously without a definitive diagnosis, was evaluated for secondary hypertension. Laboratory investigation revealed suppressed renin, elevated aldosterone (108 ng/dL), and an aldosterone-to-renin ratio of approximately 490, confirming primary aldosteronism. Computed tomography demonstrated a right adrenal nodule. Preoperative assessment showed a post-dexamethasone cortisol level of 2.26 µg/dL, suggestive of mild autonomous cortisol secretion, in addition to a recent diagnosis of diabetes mellitus and a baseline creatinine of 1.4 mg/dL. The patient underwent right adrenalectomy, with initial normalization of serum potassium. However, approximately two weeks later, he developed severe hyperkalemia (6.8 mEq/L) associated with worsening renal function (creatinine 2.4 mg/dL), requiring emergency management. Aldosterone levels were reduced, consistent with postoperative mineralocorticoid insufficiency. Fludrocortisone replacement was initiated, with partial response. Adrenalectomy for primary aldosteronism typically results in resolution of hypokalemia and improvement in blood pressure control. However, postoperative mineralocorticoid insufficiency is a rare and often underrecognized complication. Chronic suppression of the renin-angiotensin-aldosterone system, combined with long-standing disease, may impair functional recovery of the contralateral adrenal gland. In this case, pre-existing renal dysfunction and aldosterone-induced hyperfiltration may have masked underlying structural kidney disease, which became evident after surgical correction. Possible cortisol co-secretion may also have contributed to the unfavorable metabolic profile and increased clinical complexity. This case highlights an atypical course of primary aldosteronism following surgical treatment, with development of mineralocorticoid insufficiency and severe hyperkalemia. It underscores the importance of early monitoring of electrolytes and renal function in the postoperative period, particularly in patients with prolonged suppression of the renin-angiotensin-aldosterone system and a history of recurrent hypokalemia. Early recognition of this complication is essential for timely management and prevention of adverse outcomes.

Keywords: primary aldosteronism; adrenalectomy; mineralocorticoid insufficiency.

ADRENAL AND HYPERTENSION

24 (2959) PRIMARY HYPERALDOSTERONISM WITHOUT EVIDENT ADRENAL NODULE IN A PATIENT WITH EARLY-ONSET ARTERIAL HYPERTENSION: A CASE REPORT

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Case presentation: A 48-year-old male patient with a diagnosis of systemic arterial hypertension since the age of 13 was referred to the endocrinology outpatient clinic only in 2021 for etiological investigation. During the diagnostic workup, secondary causes such as pheochromocytoma and Cushing's syndrome were excluded, with normal plasma metanephrines and suppressed post-dexamethasone cortisol levels. In May 2023, laboratory evaluation showed plasma aldosterone of 9.6 ng/dL, plasma renin activity < 0.04 ng/mL/h, and an aldosterone-to-renin ratio > 30, consistent with primary hyperaldosteronism. These tests were repeated and confirmed in the absence of mineralocorticoid receptor antagonist therapy. The furosemide test was positive, without associated hypokalemia. Abdominal CT scan performed in February 2023 demonstrated bilateral adrenal thickening, whereas a previous examination from 2004 had shown adrenal glands of normal size, without identifiable nodules. At outpatient follow-up on May 8, 2026, the patient reported recurrent episodes of vertigo and drowsiness, without syncope (minimum BP 110/50 mmHg). He also reported recently starting regular physical activity, with good adherence. He denied chest pain, dyspnea, or palpitations. Current medications included losartan 50 mg twice daily and spironolactone 50 mg/day. On physical examination, he was normochromic, hydrated, and normotensive. Weight was 101 kg and heart rate 87 bpm. Recent laboratory tests showed creatinine 0.90 mg/dL, potassium 3.7 mEq/L, glucose 92 mg/dL. Given the clinical stability and risk of hypotension, the decision was made to reduce losartan to once daily, and maintain spironolactone therapy. **Discussion:** Primary Hyperaldosteronism is currently recognized as one of the leading causes of Secondary Hypertension, with an estimated prevalence of 5%–10% among hypertensive patients and up to 25% in cases of resistant hypertension. Nevertheless, it remains underdiagnosed, particularly in patients without evident adrenal nodules on imaging studies. **Final comments:** This case illustrates a typical presentation of long-standing primary hyperaldosteronism without evident adrenal adenoma, reinforcing that bilateral functional adrenal alterations may occur even in the absence of identifiable nodules. It highlights the importance of systematic screening in patients with early-onset and difficult-to-control hypertension, aiming at early diagnosis and treatment.

Keywords: secondary hypertension; primary hyperaldosteronism; adrenal glands.

ADRENAL AND HYPERTENSION

25 (3038) PROLONGED ZONA GLOMERULOSA INSUFFICIENCY AFTER UNILATERAL ADRENALECTOMY: A CASE REPORT

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Presentation of case: A 66-year-old woman with hypertension and stage IV chronic kidney disease under conservative treatment was investigated for chronic hypokalemia and diagnosed with primary aldosteronism. Laboratory findings demonstrated elevated aldosterone levels (395 ng/dL) and suppressed plasma renin activity (<0.3 ng/mL/h), with an aldosterone-to-renin ratio of 987.5. Abdominal CT revealed a 4-cm right adrenal nodule, and she underwent uncomplicated right laparoscopic adrenalectomy. Histopathological examination confirmed an adrenocortical adenoma. One month later, she was readmitted with severe hyperkalemia (7.7 mEq/L) and worsening renal function. This patient also developed hyponatremia (120 mEq/L), requiring temporary hemodialysis and hypertonic saline. Normal basal cortisol (17 µg/dL) excluded adrenal insufficiency. Given the unavailability of repeat aldosterone and plasma renin activity testing and high clinical suspicion of prolonged postoperative mineralocorticoid insufficiency, fludrocortisone (0.1 mg/day) was initiated, resulting in significant improvement in serum potassium and sodium levels within 7 days (5.0 mEq/L and 130 mEq/L, respectively). Mineralocorticoid replacement remained necessary after 9 months of the surgical procedure. **Discussion:** The pathophysiology behind postoperative mineralocorticoid insufficiency after unilateral adrenalectomy results from the contralateral suppression of the renin-angiotensin-aldosterone system, which can result in severe hyperkalemia and metabolic acidosis. Persistent cases are poorly described in the medical literature as most cases are transient. Recent studies suggest delayed recovery of aldosterone secretion after surgery, while renin tends to normalize earlier. In this case, severe late postoperative hyperkalemia and renal deterioration required prompt fludrocortisone replacement and temporary hemodialysis. **Final comments:** Prolonged mineralocorticoid insufficiency is a rare but potentially severe postoperative complication of surgical correction of primary aldosteronism. Early recognition and individualized management are essential for the prevention and treatment of life-threatening electrolyte disturbances.

Keywords: hypoaldosteronism; adrenalectomy; primary aldosteronism.

ADRENAL AND HYPERTENSION

26 (2907) RARE ONCOCYTIC ADRENOCORTICAL TUMOR PRESENTING AS A FUNCTIONAL INCIDENTALOMA

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Case presentation: A 39-year-old woman was referred for evaluation of a right adrenal mass incidentally identified during the diagnostic evaluation for a urinary tract infection. She presented with difficult-to-control hypertension, marked hirsutism, and progressive hair thinning. Computed tomography demonstrated a well-defined, heterogeneous right adrenal lesion of 10 cm at its largest diameter, with heterogeneous contrast enhancement and internal hypodense areas. Hormonal evaluation revealed androgen hypersecretion, with elevated androstenedione: 10 ng/mL (0.3–3.7), dehydroepiandrosterone sulfate (DHEA-S): 2,607 µg/dL (23–266), total testosterone: 288 ng/dL (7.21–79.31) and 17-OHprogesterone: 370 ng/dL (<80). Autonomous cortisol secretion was also observed, confirmed by lack of suppression on the 1 mg overnight dexamethasone suppression test (cortisol: 13.7 µg/dL) and an ACTH level of 5 ng/mL. Primary aldosteronism was also identified based on suppressed plasma renin activity (<0.14 ng/mL/h) and an aldosterone level of 11.3 ng/dL. Pheochromocytoma was excluded based on normal plasma metanephrine levels. Given the suspicion of malignancy, the patient underwent open right adrenalectomy. Histopathological and immunohistochemical analyses demonstrated an oncocytic adrenocortical neoplasm. **Discussion:** Adrenal incidentalomas are common findings and require evaluation for hormonal activity and malignant potential. Oncocytic adrenocortical neoplasms are rare, and diagnosis can be challenging, particularly in large and functioning tumors. Preoperative staging with abdominal and chest computed tomography did not reveal metastatic lesions. Despite the absence of major criteria for malignancy, a minor criterion according to the Lin-Weiss-Bisceglia system was present due to tumor size greater than 10 cm, allowing classification as an oncocytic adrenocortical neoplasm of uncertain malignant potential. During postoperative follow-up, hormonal normalization was observed, along with improved blood pressure control with a reduced need for antihypertensive therapy, partial regression of hirsutism and improvement in hair thinning. **Final comments:** Oncocytic adrenocortical neoplasms are a diagnostic challenge. The lack of standardized postoperative follow-up recommendations, along with possible late recurrence or progression to metastatic adrenocortical carcinoma, highlights the need for prolonged, continuous, individualized clinical, hormonal, and radiological surveillance.

Keywords: adrenocortical carcinoma; adrenocortical oncocytic neoplasms; adrenal incidentaloma.

ADRENAL AND HYPERTENSION

27 (3125) REDOX PATHWAY DYSREGULATION DEFINES TUMOUR AGGRESSIVENESS AND MITOTANE RESPONSE IN ADRENOCORTICAL TUMOURS

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Introduction: The glutathione system is a major cellular antioxidant pathway that preserves redox homeostasis in the adrenal gland. Mitotane, the therapy for advanced adrenocortical cancer (ACC) patients, interferes with the redox balance. The role of glutathione-related pathways in tumor aggressiveness and clinical outcomes associated with mitotane treatment remains poorly understood in pediatric (pACT) and adult adrenocortical tumors (aACT). **Objective:** To evaluate the association between glutathione-related antioxidant pathways, ACT aggressiveness, and response to mitotane in pACT and aACT. **Patients and methods:** Global tumor DNA methylation (EPIC array) and global mRNA expression (RNA-seq) of antioxidant-related genes were evaluated in 43 pACT samples and 76 aACC. Associations with metastatic status, disease-free survival (DFS), and overall survival (OS) were analyzed using Kaplan-Meier curves and false discovery rate correction. Functional studies in human H295R adrenal ACC cells assessed oxidative stress, mitochondrial activity, GPX1 expression, and cell proliferation upon mitotane exposure, the antioxidant N-acetylcysteine exposure, and GPX1 knockdown. **Results:** Metastatic pACT exhibited lower expression of REDOX-associated genes: TXNRD2 ($p = 0.01$), PRDX3 ($p = 0.01$), and GPX1 ($p = 0.05$). Reduced PRDX3 expression associated with shorter DFS ($p = 0.04$). Increased GPX1 methylation associated with progression to death ($p = 2.8 \times 10$). In aACC, increased glutathione pathway activity was associated with reduced DFS ($p = 0.013$) and, among mitotane-treated patients, with worse OS ($p = 0.034$) and DFS ($p = 0.021$). Elevated GPX1 expression was also associated with reduced OS in the overall cohort ($p = 0.024$) and with worse OS ($p = 0.0034$) and DFS ($p = 0.021$) in mitotane-treated patients. *In vitro*, mitotane increased oxidative stress ($p < 0.0001$), impaired mitochondrial activity ($p = 0.006$), and induced GPX1 expression ($p < 0.0001$) in H295R ACC cells. N-acetylcysteine partially attenuated the antiproliferative effects of mitotane ($p = 0.0004$), while GPX1 knockdown reduced tumor proliferation ($p < 0.0001$). **Conclusion:** In pACT, reduced antioxidant pathway expression was associated with aggressive disease, whereas in adult ACC, increased glutathione activity and GPX1 expression were associated with poorer outcomes in mitotane-treated patients, supporting a role in resistance to mitotane-induced oxidative stress. Thus, redox homeostasis is a potential biomarker and predicts therapeutic response in ACC.
Keywords: redox; adrenocortical tumours; mitotane.

ADRENAL AND HYPERTENSION

28 (3027) RETROPERITONEAL PARAGANGLIOMA REVEALING A LIKELY PATHOGENIC FH VARIANT AND HLRCC-RELATED FEATURES: 14-YEAR FOLLOW-UP

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Case report: A 35-year-old woman was incidentally found to have a right adrenal region mass on imaging performed for abdominal pain. MRI characterized a 6.3 × 5.1 cm heterogeneous lesion with intense contrast enhancement. She denied hypertensive crises, hyperhidrosis, or virilization. Biochemical workup revealed elevated 24-hour urinary fractionated normetanephrines, and ^{123}I-MIBG scintigraphy was positive, consistent with a catecholamine-secreting neuroendocrine tumor. She underwent right adrenalectomy with ipsilateral nephrectomy due to intraoperative tumor encasement of the renal artery. Histopathology confirmed a retroperitoneal paraganglioma (10 × 5 × 5 cm) with focal necrosis, one mitosis per 20 high-power fields, and positive circumferential margins. Germline testing identified a heterozygous likely pathogenic variant in the FH gene (c.151C>T; p.Arg51Trp). Prior hysterectomy for uterine leiomyoma suggested phenotypic overlap with HLRCC. First-degree relatives tested negative. Over 14 years, annual surveillance with plasma metanephrines and abdominal MRI has shown no recurrence, metastatic disease, or new tumor development. **Discussion:** The FH gene encodes fumarate hydratase, a Krebs cycle enzyme whose germline variants are classically associated with HLRCC, an autosomal dominant syndrome predisposing to leiomyomas and renal cell carcinoma. FH-related pheochromocytoma/paraganglioma (PCC/PGL) is a rare, increasingly recognized entity driven by fumarate accumulation and pseudohypoxic signaling. The histological features observed, necrosis, mitotic activity, and positive margins, indicated malignant potential and guided the decision for prolonged surveillance. Phenotypic overlap between FH-related PCC/PGL and HLRCC has been reported, with uterine leiomyoma as the most frequent co-occurring feature. The variant p.Arg51Trp has been previously described in HLRCC; its specific association with PCC/PGL remains incompletely characterized. The absence of the variant in first-degree relatives raises the possibility of a de novo mutation or reduced penetrance, both documented in FH-related disease. **Final comments:** This case illustrates genotype-phenotype variability in FH-related disease, with paraganglioma and HLRCC features co-occurring in a single patient. It reinforces the inclusion of FH in PCC/PGL germline panels, particularly when leiomyomatous disease is present, and underscores the need for lifelong, multisystem surveillance in FH variant carriers.
Keywords: paraganglioma retroperitoneal; FH gene germline; hereditary leiomyomatosis and renal cell carcinoma.

ADRENAL AND HYPERTENSION

29 (2960) SEIZURES AS THE INITIAL MANIFESTATION OF ADRENAL INSUFFICIENCY IN AUTOIMMUNE POLYGLANDULAR SYNDROME TYPE 2: A CASE REPORT

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Case presentation: A 53-year-old woman presented with severe asthenia, progressive fatigue, weight loss, dizziness, and episodes of arterial hypotension. She also reported sweating, tremors, and recurrent episodes of mental confusion. The patient evolved with generalized tonic-clonic seizures preceded by decreased level of consciousness, requiring hospitalization. On hospital admission, hypoglycemia of 55 mg/dL was documented. Physical examination revealed diffuse cutaneous hyperpigmentation, including the oral mucosa. Laboratory investigation demonstrated basal cortisol of 0.3 µg/dL and ACTH > 1,250 pg/mL, confirming primary adrenal insufficiency. She also presented elevated TSH, reduced free T4, and positive anti-thyroglobulin antibodies, compatible with Hashimoto's thyroiditis, previously diagnosed and under treatment with levothyroxine 100 mcg/day. The association between primary adrenal insufficiency and autoimmune thyroid disease confirmed the diagnosis of autoimmune polyglandular syndrome type 2 (APS-2). **Discussion:** Primary adrenal insufficiency classically manifests with asthenia, weight loss, arterial hypotension, and hyperpigmentation. Hypocortisolism predisposes to hypoglycemia by reducing hepatic gluconeogenesis and impairing the counterregulatory response to metabolic stress. In severe cases, hypoglycemia may progress to neuroglycopenia and seizures. The association between autoimmune adrenal insufficiency and autoimmune thyroid disease characterizes APS-2, a rare condition that mainly affects middle-aged women. The syndrome may also be associated with type 1 diabetes mellitus, reinforcing the need to investigate other autoimmune endocrinopathies. **Final comments:** This case illustrates a severe presentation of primary adrenal insufficiency, characterized by intense asthenia, hypoglycemia, and seizures as initial manifestations. It highlights the importance of clinical suspicion in the presence of hyperpigmentation and arterial hypotension, especially in patients with previous autoimmune endocrinopathies. Early diagnosis of APS-2 is essential to prevent complications and ensure appropriate treatment.

Keywords: primary adrenal insufficiency; autoimmune polyglandular syndrome type 2; seizures.

ADRENAL AND HYPERTENSION

30 (2987) SEVERE INSULIN-REQUIRING DIABETES MELLITUS SECONDARY TO ADRENAL AND EXTRA-ADRENAL PARAGANGLIOMA WITH COMPLETE REMISSION AFTER TUMOR RESECTION: TWO CASE REPORTS

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Case presentation: We report two cases of severe diabetes secondary to adrenal and extra-adrenal paraganglioma (PGL), with complete remission after surgical resection. Case 1: A 22-year-old woman presented 2 years ago with paroxysmal hypertension, functional bowel obstruction, and newly diagnosed type 1 Diabetes mellitus (HbA1c 11.0%) before referral to our center. Glycemic control required a basal-bolus insulin regimen totaling 53 units/day (1.02 units/kg/day). Imaging and biochemical investigation revealed a 5.2 cm para-aortic PGL with plasma metanephrine 0.3 nmol/L (reference < 0.6) and normetanephrine 18.0 nmol/L (reference < 0.9). Case 2: A 42-year-old man presented with 18 kg weight loss (BMI 17.9 kg/m²), diaphoresis, and hypertension for 2 years. Concurrently, he was diagnosed with Diabetes mellitus (HbA1c 8.9%) requiring intensive basal-bolus insulin therapy totaling 64 units/day (1.1 units/kg/day). Investigation revealed a 10.5 cm adrenal PGL with plasma metanephrine 22.6 nmol/L and normetanephrine 14.3 nmol/L. Following α1-blockade and surgical resection (PASS score 5 and 6, respectively), both patients achieved normalization of blood pressure and glycemia and were discharged without antihypertensive or glucose-lowering therapy. Two months after surgery, HbA1c levels were < 5.7% with preserved normal C-peptide levels in both cases. **Discussion:** Adrenal and extra-adrenal PGLs are rare catecholamine-producing neuroendocrine neoplasms. Although catecholamines are known to impact glucose metabolism, PGL-related insulin-requiring Diabetes mellitus remains uncommon. Secondary diabetes results from a complex interplay between catecholamine-induced insulin resistance and impaired insulin secretion. The pathophysiology is phenotype-dependent: excess catecholamines stimulate glycogenolysis and lipolysis, while α2-adrenergic receptor activation in pancreatic β-cells directly inhibits insulin secretion. The rapid post-surgical metabolic normalization in both cases confirms that the metabolic disturbances were mediated by catecholamine excess rather than permanent structural damage. **Final comments:** Diabetes mellitus secondary to PGLs can mimic severe type 1 or type 2 Diabetes mellitus. Identifying clinical phenotypes suggestive of PGL-related syndromes is essential, as appropriate treatment may allow permanent discontinuation of insulin therapy, significantly improving prognosis and quality of life.

Keywords: pheochromocytoma; paraganglioma; diabetes mellitus.

ADRENAL AND HYPERTENSION

31 (2889) SLEEP QUALITY AND ITS ASSOCIATION WITH HYPERTENSION AND DIABETES MELLITUS: A POPULATION-BASED STUDY

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Introduction: Sleep quality has been increasingly recognized as an important factor influencing metabolic and cardiovascular health. Furthermore, poor sleep has been associated with conditions such as hypertension and diabetes mellitus, highly prevalent chronic diseases and major public health concerns. **Objective:** To evaluate sleep quality among individuals with and without hypertension and/or diabetes in the populations of Taubaté and Tremembé. **Methods:** This cross-sectional, quantitative and exploratory study in which a questionnaire was applied to 2,469 individuals at the event "36th Hypertension and Diabetes Prevention Week", in Tremembé and Taubaté, São Paulo, Brazil. The questionnaire included general information about diabetes and hypertension, and the Pittsburgh Sleep Quality Index (PSQI). Data collection was conducted following approval by the Human Research Ethics Committee and the participants' signing of the informed consent form. Associations were analyzed using the Chi-square test, with a significance level set at 5%. **Results:** A significant association was observed between hypertension and/or diabetes and sleep quality ($p = 0.006$). Participants with both conditions showed higher rates of unsatisfactory sleep (42%) and sleep disorders (11%) compared with those without comorbidities (33% and 8%, respectively). Similarly, hypertension alone also showed higher frequencies of unsatisfactory sleep (34%) and sleep disorders (11%), although the increase in unsatisfactory sleep was smaller. On the other hand, diabetes alone showed lower rates of unsatisfactory sleep (32%) and a higher prevalence of satisfactory sleep (59%) than those with hypertension alone (55%) or both conditions (48%), suggesting a weaker association with impaired sleep quality. **Conclusion:** Hypertension showed a stronger association with poorer sleep quality, whereas diabetes alone appeared to have a weaker association. Also individuals with both comorbidities presented the worst sleep profile, with the highest rates of unsatisfactory sleep. Therefore, the findings highlight the relevance of cardiometabolic conditions for sleep health and reinforce the importance of understanding the mechanisms affecting sleep, which may help allocate resources and improve quality of life in patients with chronic diseases.

Keywords: hypertension; diabetes; sleep.

ADRENAL AND HYPERTENSION

32 (3391) SYSTEMATIC SCREENING FOR PRIMARY ALDOSTERONISM IN RESISTANT HYPERTENSION: IMPACT ON DIAGNOSTIC RATES AND BLOOD PRESSURE CONTROL

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Introduction: Primary aldosteronism is the most common cause of secondary hypertension but remains underdiagnosed. In resistant hypertension, its prevalence may reach 20%; however, screening based solely on clinical suspicion often misses cases, resulting in inadequate treatment and higher cardiovascular risk. **Objective:** To evaluate whether the implementation of a systematic screening protocol for primary aldosteronism, using the aldosterone-to-renin ratio, increases diagnostic accuracy and improves blood pressure control in adults with resistant hypertension compared to conventional clinical management. **Methods:** A systematic review was conducted following the PICO strategy. Searches were performed in PubMed, Embase, and the Cochrane Library for studies published between 2015 and 2024. Inclusion criteria were: adults with resistant hypertension (blood pressure above 140/90 mmHg on three or more medications); Intervention: systematic screening via aldosterone-to-renin ratio; comparison: usual care or clinical suspicion; outcomes: prevalence of primary aldosteronism and systolic blood pressure reduction after targeted therapy with mineralocorticoid receptor antagonists or adrenalectomy. **Results:** Data from twelve high-quality studies involving four thousand five hundred and eighty patients were synthesized. Systematic screening identified primary aldosteronism in eighteen point six to twenty-two point four percent of resistant hypertension cohorts, whereas conventional screening detected less than two percent of cases in the same populations. Patients diagnosed through systematic protocols who received targeted therapy achieved a mean reduction in systolic blood pressure of fourteen point two millimeters of mercury, compared to five point eight millimeters of mercury in those maintained on standard antihypertensive therapy. Additionally, thirty-two percent of patients in the systematic screening group required fewer antihypertensive medications to achieve blood pressure targets after twelve months. **Conclusion:** Systematic screening for primary aldosteronism in patients with resistant hypertension significantly increases diagnostic accuracy and enables superior blood pressure control through targeted interventions. These findings support the implementation of universal screening via the aldosterone-to-renin ratio in all patients with resistant hypertension as a standard of care to mitigate cardiovascular morbidity.

Keywords: primary aldosteronism; resistant hypertension; mass screening.

ADRENAL AND HYPERTENSION

33 (3427) THE POTENTIAL OF PREDICTION MODELS FOR SCREENING PRIMARY ALDOSTERONISM IN PATIENTS WITH HYPERTENSION: A SYSTEMATIC REVIEW

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Introduction: Primary aldosteronism (PA) is the leading cause of secondary hypertension, accounting for 5%–10% of hypertension cases and up to 20% among patients with resistant hypertension. Screening is mainly based on the Aldosterone-to-Renin Ratio (ARR), whose accuracy may be affected by antihypertensive drugs, potassium levels, circadian rhythm, and patient posture. In this context, prediction models, especially those based on machine learning (ML), have emerged as promising tools for optimizing PA screening. **Objective:** To evaluate the diagnostic performance and clinical applicability of prediction models for PA screening in adult hypertensive patients. **Methods:** This systematic review was conducted according to PRISMA 2020 recommendations. Searches were performed in PubMed, Embase, and Cochrane databases using descriptors related to PA and predictive models. Two independent reviewers screened and selected studies. Observational studies involving adult hypertensive patients assessed through clinical prediction models, nomograms, diagnostic scores, or ML algorithms applied to PA screening were included. Literature reviews, case reports, preclinical studies, and studies without full text were excluded. From 280 identified records, eleven studies were included after duplicate removal and eligibility assessment. Risk of bias and applicability were evaluated using PROBAST. **Results:** The eleven studies included more than 28,000 participants. Five studies evaluated ML algorithms, four assessed clinical nomograms, and two developed conventional diagnostic scores. Models predominantly used serum potassium, plasma renin activity, aldosterone levels, and systolic blood pressure. ML models showed better performance than conventional methods in several studies. Sensitivity ranged from 71.4% to 96.6%, specificity from 48.0% to 98.8%, PPV from 82.4% to 90.5%, and NPV from 90.4% to 97%. ML-based models also demonstrated satisfactory overall performance with elevated AUC values. **Conclusion:** ML-based prediction models appear promising for PA screening, showing superior diagnostic performance compared with conventional methods in part of the included studies. However, methodological heterogeneity remains significant, highlighting the need for prospective multicenter studies to provide external validation and improve clinical applicability.

Keywords: primary aldosteronism; hypertension; mass screening.

ADRENAL AND HYPERTENSION

34 (3454) TRANSIENT HYPOKALEMIC PARALYSIS REVEALING PRIMARY HYPERALDOSTERONISM AND CARDIOMYOPATHY

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Case presentation: A 46-year-old woman with hypertension since age 38 and prior hypokalemic periodic paralysis, requiring hospitalization in July 2022 for severe hypokalemia (K = 1.5 mEq/L). Subsequent endocrine investigation confirmed hyporeninemic hyperaldosteronism, with aldosterone > 100 and renin activity of 0.17, secondary to a left adrenal tumor measuring 2.5 × 1.6 cm. Imaging revealed bilateral nephrolithiasis with nephrocalcinosis, and medullary sponge kidney. Cardiac magnetic resonance showed severe left ventricular systolic dysfunction, ejection fraction of 38%, and mild aortic regurgitation, without myocardial fibrosis. Current treatment includes spironolactone 100 mg, potassium chloride 1,200 mg/day, carvedilol, losartan, dapagliflozin, hydralazine, and isosorbide mononitrate. Adrenalectomy is also considered. After adequate potassium replacement, the patient reported marked improvement of muscle paresis. A recent echocardiogram showed recovery of global systolic function, with an ejection fraction of 64%. **Discussion:** Primary hyperaldosteronism is a recognized cause of secondary hypertension; however, initial presentation as hypokalemic periodic paralysis is extremely rare, particularly in non-Asian women. Chronic aldosterone excess combined with profound hypokalemia can contribute to target-organ damage, as seen in this patient with severe but reversible cardiomyopathy and medullary nephrocalcinosis. Improvement in left ventricular function after optimized medical therapy and electrolyte correction highlights the potential reversibility of aldosterone-induced cardiotoxicity when myocardial fibrosis is absent. **Final comments:** This case emphasizes the need for rigorous screening for secondary causes of hypertension in young patients, especially with severe hypokalemia, structural cardiac dysfunction, and resistant hypertension. Early diagnosis and aggressive multidisciplinary management are essential to reverse life-threatening cardiovascular complications, such as severe cardiomyopathy, and prevent renal disease progression.

Keywords: hyperaldosteronism; hypokalemia; hypertension.

DIABETES MELLITUS

35 (3508) AGE AT DIAGNOSIS AS A PREDICTOR OF GLYCEMIC RESPONSE TO CONTINUOUS GLUCOSE MONITORING IN TYPE 1 DIABETES

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Introduction: Continuous glucose monitoring (CGM) has demonstrated glycemic benefits for type 1 diabetes (T1D), yet access in Brazil remains limited and fragmented, with isolated initiatives that often prioritize specific subgroups. Identifying predictors of response may support more equitable prioritization strategies. **Objective:** To identify clinical and sociodemographic predictors of glycemic response to CGM use in individuals with T1D. **Methods:** A 6-month longitudinal study enrolled 53 individuals with T1D from a public endocrinology clinic in Northeastern Brazil. Participants were classified as responders if they achieved HbA1c equal to or below 7% and/or time in range equal to or above 70% at the end of follow-up. Individuals without final HbA1c were classified based on time in range alone. Univariable analyses were performed for clinical, sociodemographic, and CGM engagement variables. Independent predictors were identified using Firth penalized logistic regression. **Results:** The cohort was predominantly female (64.2%), mostly adults (69.8%), with mean diabetes duration of 11±8.72 years and total daily insulin dose of 0.8 ± 0.4 IU/kg. Of 53 participants, 21 were classified as responders and 32 as non-responders. On univariable analysis, responders had lower baseline HbA1c [7.6% (6.6–8.4) vs. 9.8% (9.0–11.2); $p < 0.001$] and older age at diagnosis [18.0 (9.0–24.0) vs. 11.0 years (6.8–15.0); $p = 0.006$], as well as higher scan frequency and greater sensor active time. On multivariable analysis, only baseline HbA1c (OR 0.18, 95% CI 0.06-0.57; $p = 0.003$) and age at diagnosis (OR 2.96, 95% CI 1.19-7.38; $p = 0.020$) remained independent predictors. Each additional year of age at diagnosis was associated with approximately 3-fold higher odds of response. No associations were found with sex, BMI, depressive symptoms, or private health insurance. **Conclusion:** Older age at T1D diagnosis and lower baseline HbA1c independently predicted glycemic response to CGM, suggesting that prioritization policies should consider adults with later-onset T1D, a group often overlooked in favor of pediatric populations. The predictive value of baseline HbA1c, however, should be interpreted with caution, as it also served as part of the response criterion.
Keywords: type 1 diabetes; continuous glucose monitoring; glycemic response.

DIABETES MELLITUS

36 (3387) ANALYTICAL PERFORMANCE OF ELISA 3 SCREEN ISLET CELL AUTOANTIBODY ASSAY IN QUALITY-CONTROL SAMPLES FROM INDIVIDUALS WITH TYPE 1 DIABETES

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Introduction: Type 1 diabetes (T1D) screening with specific autoantibodies measurement before the onset of hyperglycemia has clinical advantages, but a high cost. ELISA RSR 3 Screen Islet Cell Autoantibody assay (RSR 3AB) is a cheaper test that enables detection of 1 or more antibodies in a single sample, but does not discriminate GADA (glutamic acid decarboxylase antibody), Anti-IA-2 (islet antigen-2 tyrosine phosphatase antibody), or Anti-ZnT8 (zinc transporter 8 antibody) positivity. It might be used as an initial screening tool to identify individuals who need individual antibody testing, to reduce screening costs. **Objective:** To evaluate the analytical performance of RSR 3AB compared to reference standard tests, to evaluate its applicability as a low-cost screening tool for T1D. **Methods:** This exploratory study analyzed 15 previously characterized quality-control samples with known autoantibody profiles, determined by chemiluminescence and/or ELISA, from individuals with T1D followed at a tertiary center. Samples included positive and negative controls. Samples were tested in triplicate with RSR 3AB at 450 nm and 405 nm readings. Diagnostic performance was assessed (sensitivity, specificity, predictive values, accuracy). **Results:** The assay demonstrated high concordance with reference methods and excellent reproducibility among replicates. Using the manufacturer-recommended cutoff value (≥20 U/mL), sensitivity of 100%, accuracy of 86.7%, and positive predictive value of 86.7% were observed. Specificity and negative predictive value were inconclusive because of the limited number of negative samples (n = 2). Known positive samples showed substantially higher titers than known negative samples in RSR 3AB, allowing clear differentiation between groups. Negative samples had reactivity slightly above the manufacturer's cutoff, although markedly lower than observed in positive samples. In exploratory analyses, a higher cutoff (≥80 U/mL) improved sensitivity, specificity, positive and negative predictive values and accuracy to 100% at 450 nm. **Conclusion:** RSR 3AB assay demonstrated excellent analytical performance, particularly at 450 nm, in this preliminary sample set. Although a clear distinction between positive and negative samples was observed, a different cutoff may be more appropriate in this population. Further studies in larger cohorts are necessary. These findings suggest that RSR 3AB assay may be a useful low-cost initial screening strategy for T1D.
Keywords: type 1 diabetes; RSR 3AB; autoantibodies.

DIABETES MELLITUS

37 (3069) ASSOCIATION BETWEEN ADMISSION GLYCATED HEMOGLOBIN AND CLINICAL OUTCOMES IN PATIENTS WITH ATHEROSCLEROTIC PERIPHERAL ARTERIAL DISEASE UNDERGOING VASCULAR INTERVENTION

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Introduction: diabetes mellitus (DM) is highly prevalent among patients with peripheral arterial disease (PAD) and may influence outcomes following vascular interventions. Glycated hemoglobin (HbA1c) reflects long-term glycemic control; however, evidence regarding its prognostic impact remains inconsistent. **Objective:** To evaluate the association between HbA1c levels and outcomes such as amputation, infection, major adverse cardiovascular events (MACE), and mortality in patients with PAD undergoing vascular intervention. Additionally, to describe the epidemiological profile and the prevalence of HbA1c > 7%. **Methods:** A retrospective cohort study (January 2023–October 2025) was conducted at a tertiary hospital in Curitiba, Brazil. Adult patients with confirmed atherosclerotic PAD and available HbA1c measurements (obtained up to 3 months before admission or within the first days of hospitalization) were included. Exclusion criteria comprised conditions affecting HbA1c levels, non-atherosclerotic etiologies, and incomplete medical records. HbA1c was dichotomized as ≤7% and >7% for analysis. The primary outcomes were in-hospital and 30-day post-discharge mortality, MACE, infections, and amputations. Secondary analyses included the patients' metabolic and epidemiological characteristics. **Results:** Of the 176 medical records screened, 136 patients were included. Mean age was 67.6 ± 11.5 years, 70.6% were male, and 67.6% had pre-existing DM. HbA1c was ≤7% in 41.9% (n = 57) and >7% in 58.1% (n = 79). Emergency procedures predominated (66.9%), with no significant difference between groups (p = 0.266). No significant association was observed between HbA1c levels and MACE (8.8% vs. 2.5%; p = 0.130), amputation (8.8% vs. 16.5%; p = 0.213), infection (3.5% vs. 6.3%; p = 0.699), or mortality (10.5% vs. 7.6%; p = 0.558). **Conclusion:** Elevated HbA1c at admission was not associated with adverse in-hospital or 30-day outcomes in patients with PAD undergoing vascular intervention. Prospective studies are needed, particularly in elective settings.

Keywords: diabetes mellitus; atherosclerosis; cardiovascular diseases.

DIABETES MELLITUS

38 (3133) ASSOCIATION BETWEEN EATING DISORDERS, DIABETES DISTRESS, AND GLYCEMIC CONTROL IN ADULTS WITH TYPE 1 DIABETES: A CROSS-SECTIONAL STUDY

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Introduction: Eating disorders and diabetes distress are common yet often overlooked complications in type 1 diabetes (T1D) that can negatively affect glycemic control. **Objective:** To investigate the prevalence of typical eating disorder symptoms and diabetes-related distress in adults with T1D, and to analyze how these factors relate to clinical parameters. **Methods:** This cross-sectional study included patients with T1D aged ≥ 12 years. Participants completed the Diabetes Eating Problem Survey-Revised (DEPS-R) and Problem Areas in Diabetes scale (PAID-20) questionnaires. Eating disorder symptoms were identified with a DEPS-R score of 20 or higher. Diabetes-related distress levels were categorized as low (0–16), moderate (17–39), or high (40 or above). Data collected encompassed demographics, diabetes duration, body mass index (BMI), and glycated hemoglobin (HbA1c). Statistical analysis involved correlation and multiple regression. **Results:** The study involved 56 participants, with a median age of 27 (20–4) years, of whom 62.5% were female. Participants had been living with diabetes for a median of 14 (8–21) years, had a median BMI of 23.9 (21.4–27.2) kg/m², and an HbA1c of 9.3% (8.1%–11.1%). Symptoms of eating disorders were present in 28.6% of the participants, with a median DEPS-R score of 14 (9–21). Diabetes distress levels were classified as low in 17.9%, moderate in 37.5%, and high in 44.6%, with a median PAID-20 score of 42 (28–59). The DEPS-R score was positively correlated with HbA1c (r = 0.4, p 0.001) and nearly significant with PAID-20 (r = 0.38, p = 0.058). PAID-20 also showed a correlation with HbA1c (r = 0.3, p = 0.02). Participants with diabetes distress exhibited higher HbA1c levels than those without distress [9.5% (8.3%–11.5%) vs. 7.8% (6.8%–8.3%), p = 0.009], respectively. HbA1c was identified as an independent predictor of both eating disorder symptoms (β = 0.56, p = 0.004) and diabetes distress (β = 0.67, p = 0.027). Conclusion: Symptoms of eating disorders and moderate/high diabetes distress are very common among patients with T1D, affecting nearly one-third and over 80%, respectively. Both conditions are strongly associated with poorer glycemic control. These findings emphasize the need for routine psychosocial screening using tools like DEPS-R and PAID-20 in T1D care. Adopting integrated strategies that address both metabolic and psychological issues is crucial for improving health outcomes.

Keywords: type 1 diabetes; diabetes distress; eating disorders.

DIABETES MELLITUS

39 (3578) ASSOCIATION BETWEEN SLEEP DISORDERS AND INSULIN RESISTANCE IN ADULTS WITH TYPE 2 DIABETES OR PREDIABETES: A SYSTEMATIC REVIEW

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Introduction: Sleep disorders affect glucose metabolism regulation. Alterations in sleep quality and duration favor insulin resistance, central to the progression of prediabetes and type 2 diabetes. This review synthesizes evidence on the association between sleep disorders and insulin resistance in adults. **Objective:** To evaluate the association between sleep disorders and insulin resistance in adults with type 2 diabetes or prediabetes. **Methods:** Systematic review conducted according to PRISMA guidelines. Searches were performed in PubMed, Embase, Virtual Health Library, and Cochrane Library (2010–2026) for studies in adults ≥18 years with type 2 diabetes or prediabetes evaluating sleep disorders (insomnia, sleep apnea, inadequate duration/quality) and insulin resistance (HOMA-IR, fasting glucose, HbA1c, or insulin sensitivity). Excluded: minors, type 1 diabetes, case reports, and conference abstracts. From 754 records, after duplicate removal and independent screening by three reviewers in Rayyan, 15 studies were included. **Results:** 11.847 participants from 15 studies were analyzed, predominantly cross-sectional (53.3%). Insomnia was associated with elevated HbA1c (7.72 ± 1.74% vs. 7.50 ± 1.46%, p = 0.031), with important gender difference: men with insomnia showed HbA1c 7.79% versus 7.45% without insomnia (p = 0.005), while women showed no difference. Insomnia alone was independently associated with 1.08% higher HbA1c compared to isolated sleep apnea (p = 0.007). Severe sleep apnea (AHI > 30) showed HbA1c 8.91% versus 7.32% in mild cases (p < 0.001) and HOMA-IR 8.2 versus 4.5 (p < 0.001), evidencing dose-response. CPAP treatment reduced HbA1c from 7.68% to 6.82% (p < 0.05), demonstrating reversibility and suggesting causality. Fasting glucose increased 23% in insomnia patients and 9% with sleep fragmentation. Sleep duration showed curvilinear relationship with HOMA-IR: very short (<5h) or long (>9h) sleep elevated HOMA-IR; optimal duration 7-8 hours. Elevated PSQI was independently associated with higher HbA1c after adjustment for BMI and depression (p = 0.041). **Conclusion:** Insomnia, inadequate sleep duration, and sleep apnea are associated with worse glycemic control and insulin resistance in type 2 diabetes or prediabetes. HbA1c improvement with CPAP reinforces the metabolic impact of sleep treatment, highlighting the importance of sleep assessment in managing these patients.
Keywords: sleep disorders; insulin resistance; type 2 diabetes.

DIABETES MELLITUS

40 (2877) ASSOCIATIONS BETWEEN DXA-DERIVED BODY COMPOSITION, BONE MASS, AND METABOLIC PARAMETERS IN INDIVIDUALS WITH LONG-STANDING TYPE 2 DIABETES

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Introduction: Type 2 diabetes (T2D) is associated with increased fracture risk despite normal or elevated bone mineral density (BMD), suggesting impaired bone quality. Chronic hyperglycemia, insulin resistance, and unfavorable body composition may contribute to skeletal fragility. **Objective:** To investigate associations between DXA-derived body composition, BMD, and clinical-metabolic variables in individuals with long-standing T2D. **Methods:** Cross-sectional study conducted between 2023–2025 at a tertiary outpatient clinic. Adults with confirmed T2D were consecutively enrolled. All participants provided written informed consent. Clinical, anthropometric, laboratory, and DXA data were collected. Fat Mass Index (FMI) was calculated as fat mass/height^2. Statistical analyses were performed using GraphPad Prism 10.0. Correlations, group comparisons, and multivariable linear regression were applied. **Results:** A total of 137 participants were analyzed (67.1% women; mean age 65.0 ± 7.5 years; diabetes duration 19.0 ± 11.5 years). FMI correlated inversely with diabetes duration (r = –0.432, p = 0.0002) and positively with systolic (r = 0.442, p = 0.0002) and diastolic blood pressure (r = 0.395, p = 0.001). Femoral neck T-score showed negative correlations with HbA1c at diagnosis (r = –0.774, p < 0.0001), waist circumference (r = –0.448, p = 0.0003), total body fat percentage (r = –0.269, p = 0.0021), and lower limb fat percentage (r = –0.395, p = 0.0006), and a positive correlation with appendicular lean mass (r = 0.240, p = 0.006). Serum creatinine was not associated with BMD at any skeletal site. Nineteen participants (13.9%) had osteoporosis and showed significantly lower femoral neck and total femur BMD (p < 0.000001), body weight (p = 0.017), and fat mass (p = 0.028). Multivariable analysis did not identify independent predictors of BMD. **Conclusion:** In individuals with long-standing T2D, progressive alterations in body composition are associated with cardiometabolic risk and skeletal health. Glycemic control and regional adiposity significantly influence femoral neck BMD. DXA provides a comprehensive tool for integrated assessment of fracture risk in T2D.
Keywords: osteoporosis; diabetes mellitus; DXA.

DIABETES MELLITUS

41 (3285) AUTOIMMUNE DIABETES IN GENETICALLY CONFIRMED CONGENITAL GENERALIZED LIPODYSTROPHY TYPE 1 DUE TO AGPAT2 VARIANTS

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Case presentation: A 25-year-old male was diagnosed with diabetes at the age of 16, presenting with polyuric symptoms, without ketoacidosis, requiring insulin therapy since diagnosis, and with positive anti-glutamic acid decarboxylase antibodies. During follow-up, acanthosis nigricans was noted despite normal weight. Physical examination also revealed marked reduction of subcutaneous adipose tissue and prominent musculature, raising suspicion for lipodystrophy. Genetic testing confirmed biallelic pathogenic AGPAT2 variants, an intragenic deletion spanning exons 3–4 and nonsense variant p.Lys216*, establishing Congenital Generalized Lipodystrophy type 1 (CGL1). Despite classical phenotype, metabolic profile was atypical: triglycerides remained normal across all assessments, and serial C-peptide confirmed severe beta-cell dysfunction (0.1 ng/mL). The patient requires basal-bolus insulin (1.1 IU/kg/day) and has no evidence of hepatic steatosis. **Discussion:** CGL1 typically presents with severe insulin resistance, preserved or elevated C-peptide levels, and hypertriglyceridemia. In contrast, this patient exhibited marked insulin deficiency and strongly positive autoimmune markers, supporting autoimmune beta-cell destruction as the predominant diabetogenic mechanism rather than insulin resistance alone. Furthermore, triglyceride levels remained persistently normal, an uncommon finding in CGL1. This atypical metabolic profile may reflect reduced hepatic lipogenesis secondary to severe insulin deficiency, rather than the classical hyperinsulinemic-lipotoxic state usually observed in congenital generalized lipodystrophy. Experimental AGPAT2-deficient murine models demonstrate preserved islet mass with compensatory hypertrophy despite hyperglycemia, suggesting that marked beta-cell loss is not expected as a consequence of lipodystrophy-related metabolic stress alone. **Conclusion:** This case describes the coexistence of genetically confirmed CGL1 and autoimmune diabetes in a single patient, illustrating the overlap of two distinct diabetogenic mechanisms. The underlying basis of this association remains uncertain and warrants further investigation. Importantly, this report underscores the need for broader etiological evaluation in atypical presentations of diabetes, as accurate diagnosis is fundamental for precision medicine and individualized management.
Keywords: type 1 diabetes; lipodystrophy; AGPAT2.

DIABETES MELLITUS

42 (3290) AUTOMATED INSULIN DELIVERY IN THE BRAZILIAN PUBLIC HEALTH SYSTEM: THE TECHNOLOGY WORKS, THE CONTINUOUS SUPPLY DOES NOT

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Introduction: Continuous subcutaneous insulin infusion (CSII) and automated insulin delivery (AID) systems improve glycemic control and reduce hypoglycemia in individuals with type 1 diabetes (T1D). In Brazilian public health system (SUS), access to these technologies occurs mainly through litigation and real-world data remain scarce. **Objective:** To analyze the glucose profile of patients with T1D using CSII with or without AID followed at a tertiary hospital. **Methods:** Cross-sectional observational study included patients with T1D using CSII. Clinical data and glucose profile in continuous glucose monitoring and capillary blood tests were obtained from the manufacturer software and clinical charts. Time in range (TIR, 70–180 mg/dL), time above range (TAR > 180; > 250 mg/dL), time below range (TBR < 70; < 54 mg/dL) and coefficient of variation (CV) were evaluated. Patients were categorized into Groups A and B (AID and manual mode, respectively). In B, the percentage of capillary glucose readings within target (GIR) was also evaluated. Statistical analyses included Mann-Whitney and Spearman correlation tests; $p < 0.05$ considered statistically significant. **Results:** 41 patients were included (mean age and diabetes duration 35.8 and 23.2 years, respectively; 64.3% female). Devices included MiniMed 780G (n = 32), MiniMed 640G (n = 9), and Medtrum (n = 1). In Group A (n = 23), mean TIR, TAR > 180, TAR > 250, TBR < 70, TBR < 54 and CV were 74.0% (±8.4), 20.4%, 5.2%, 4.4%, 1.4% and 36%, respectively. Recommended targets for TIR ≥ 70%, TBR < 70 ≤ 4%, and CV < 36% were achieved by 65%, 70%, and 41% of patients, respectively. Mean HbA1c was 7.2% in Group A and 7.6% in Group B ($p = 0.13$). Mean time in automated mode was 73.3%. Only 2 (8.7%) maintained automation ≥ 95% of the time, whereas 4 (17%) used automation during < 50% of the analyzed period, mainly due to interruptions in sensor supply. Mean sensor use was 11.3 of 14 days. Time in automation was directly correlated to TIR ($R = 0.48$; $p = 0.029$). Mean GIR in Group B was 60.5%. **Conclusion:** Patients with T1D using CSII in automated mode achieved satisfactory glycemic control, supporting the feasibility of this treatment within SUS, but had similar HbA1c than patients in manual mode. Greater time in automation with regular supply was consistently associated with better glycemic outcomes, reinforcing the importance of uninterrupted access to sensors and supplies to maximize the benefits of AID systems in routine clinical practice.
Keywords: automated insulin delivery; type 1 diabetes; glycemic control.

DIABETES MELLITUS

43 (3369) EVALUATION OF THE URIC ACID/HDL RATIO IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ITS RELATIONSHIP WITH RENAL PROGNOSIS

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Introduction: Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disease characterized by hyperglycemia and associated with obesity, sedentary lifestyle, and an unhealthy diet. Among its complications, Diabetic Kidney Disease (DKD) stands out, affecting up to 40% of patients and having a significant impact on cardiovascular mortality. Biomarkers such as serum uric acid (SUA) and its ratios with HDL and creatinine have been studied as predictors of metabolic and renal risk. **Methods:** A descriptive, retrospective study was conducted using secondary data from 1,232 patients with T2DM (HbA1c ≥7.0%), of both sexes, aged 40–70 years. Blood glucose, HbA1c, creatinine, lipid profile, and SUA were analyzed. The uric acid/HDL ratio (UA/HDL) and uric acid/creatinine ratio (UA/Cr) were calculated. Spearman's correlation and multiple linear regression analyses were performed. **Results:** The median HbA1c was 8.6%, and estimated blood glucose was 200 mg/dL. A negative correlation was observed between HbA1c and the UA/HDL ratio ($\rho = -0.150$; $p < 0.001$) and the UA/Cr ratio ($\rho = -0.202$; $p < 0.001$). Adjusted models showed a negative association between HbA1c and both ratios. Male sex was associated with higher UA/HDL and lower UA/Cr ratios. Age showed a negative association only with the UA/Cr ratio. The models had low explanatory power ($R^2 < 0.10$). **Discussion:** The metabolic ratios showed an inverse association with glycemic control, suggesting a potential role as complementary biomarkers. The UA/Cr ratio demonstrated greater sensitivity to HbA1c and an influence of age, whereas the UA/HDL ratio varied according to sex. These findings reflect the complex interaction between glucose metabolism, lipid metabolism, and renal function in T2DM.
Keywords: type 2 diabetes mellitus; uric acid; chronic kidney disease.

DIABETES MELLITUS

44 (3345) BEYOND OBESITY: DIAGNOSTIC JOURNEY OF FAMILIAL PARTIAL LIPODYSTROPHY TYPE X IN A PATIENT WITH YOUNG-ONSET TYPE 2 DIABETES MELLITUS

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Case presentation: A 32-year-old woman was referred to a tertiary endocrinology clinic in Northeastern Brazil after eight years of uncontrolled diabetes mellitus initially managed as type 2 diabetes mellitus (T2DM). Physical examination revealed a lipodystrophic phenotype despite obesity (BMI 32 kg/m²), raising suspicion for lipodystrophic diabetes. Although treated with metformin, gliclazide, and insulin at 42 IU/day (0.5 IU/kg/day), HbA1c remained 9.6% and fasting glucose 218 mg/dL. Laboratory evaluation showed triglycerides 153 mg/dL, LDL-c 119 mg/dL, and ApoB 125 mg/dL. Dual-energy X-ray absorptiometry (DXA) demonstrated visceral adipose tissue of 1,393 g, lower limb fat mass 23%, and fat mass ratio (FMR) 1.33. Anthropometric parameters revealed waist-to-hip ratio 1.05, thigh skinfold 31 mm, and Köb index 2.34. Genetic testing was unavailable. Following phenotypic diagnosis, pioglitazone and dapagliflozin were introduced, resulting in HbA1c reduction from 9.6% to 7.3%, complete insulin withdrawal, triglyceride reduction from 153 to 97 mg/dL, and LDL-c reduction from 119 to 64 mg/dL. **Discussion:** Familial partial lipodystrophy is characterized by impaired adipose tissue expandability, ectopic fat deposition, severe insulin resistance, and cardiometabolic dysfunction. In patients with obesity phenotype, central adiposity associated with peripheral fat depletion may delay recognition for years. In this case, suspicion of lipodystrophic diabetes prompted DXA evaluation, which demonstrated lower limb fat below 25%, FMR above 1.2, and Köb index lower than 3.477, supporting abnormal fat redistribution compatible with FPLD type X (FPLDx). The diagnosis of FPLDx was favored by the combination of severe metabolic dysfunction disproportionate to overall adiposity, peripheral lipoatrophy and Köb index below the threshold typically associated with FPLD type 1. This reinforces the relevance of DXA-derived body composition analysis in identifying concealed lipodystrophic phenotypes that may otherwise be classified as conventional T2DM **Final comments:** Recognition of disproportionate metabolic dysfunction and abnormal body fat partitioning enabled appropriate therapeutic adjustment and substantial metabolic improvement. This case reinforces that FPLDx may be identified when DXA fulfills established phenotypic criteria, allowing more appropriate therapeutic strategies in patients with concealed lipodystrophic phenotypes.
Keywords: diabetes mellitus; lipodistrofia parcial familiar; dislipidemia.

DIABETES MELLITUS

45 (2945) CARDIOMETABOLIC MULTIMORBIDITY AND HIDDEN DYSGLYCEMIA IDENTIFIED THROUGH COMMUNITY SCREENING DURING WORLD DIABETES DAY 2025 IN SÃO PAULO, BRAZIL

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Introduction: Prediabetes and type 2 diabetes (T2D) frequently coexist with hypertension (HTN), dyslipidemia (DLP), and cardiovascular disease (CVD), but many individuals remain undiagnosed until advanced cardiometabolic burden develops. We evaluated dysglycemia and cardiometabolic multimorbidity in a community-based screening initiative in São Paulo, Brazil. **Methods:** Cross-sectional study conducted during the 2025 World Diabetes Day campaign. Participants underwent random capillary glucose testing and structured clinical assessment. Hyperglycemia was defined as capillary glucose ≥140 mg/dL. Cardiometabolic multimorbidity was defined as at least two among HTN, DLP, CVD, chronic kidney disease (CKD), and diabetic retinopathy (DR). Diabetes status was categorized as no diabetes, prediabetes, T2D, or T1D. Multinomial logistic regression evaluated factors associated with dysglycemia progression. **Results:** A total of 796 individuals were evaluated (mean age 56.5 ± 16.2 years; 52.4% female). Prediabetes and T2D were identified in 25.3% and 16.7% of participants, respectively. Mean blood glucose was 124.5 ± 57.7 mg/dL. HTN was present in 27.3%, DLP in 17.8%, CVD in 3.0%, CKD in 1.8%, and DR in 0.5%. Importantly, 12.1% of individuals without known diabetes or prediabetes presented hyperglycemia during screening, suggesting previously unrecognized dysglycemia. Cardiometabolic multimorbidity was frequent: 84 participants presented two comorbidities and 17 presented three or more. Additionally, 43.3% of participants already reported previous episodes of abnormal glucose levels. Multinomial regression demonstrated that previous awareness of abnormal glucose levels was the strongest factor associated with progression from normoglycemia to prediabetes and T2D (all $p < 0.001$). Hypertension also showed a positive association with dysglycemia severity. Nearly half of participants (48.2%) reported > 30 minutes/day of physical activity despite the elevated cardiometabolic burden observed. **Conclusion:** Community screening identified a substantial burden of hidden dysglycemia and cardiometabolic multimorbidity in São Paulo. More than one in ten individuals without previous diagnosis of dysglycemia presented abnormal glucose levels during screening. These findings reinforce major gaps in early cardiometabolic detection and support broader population-based screening strategies targeting prediabetes and T2D before advanced multimorbidity develops.

Keywords: diabetes; community screening; management gap.

DIABETES MELLITUS

46 (3277) CARDIOVASCULAR RISK PROFILES IN CHRONIC DIABETIC FOOT SYNDROME: A CROSS-SECTIONAL ANALYSIS

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Introduction: diabetes mellitus (DM) is a leading cause of morbidity and mortality worldwide, affecting 589 million adults globally. Diabetic foot syndrome is one of its most severe complications: studies report a 5-year mortality rate of 49.1% following the diagnosis of a diabetic foot ulcer, with cardiovascular disease as the primary cause of death. Therefore, elucidating the cardiovascular profile of these patients may contribute to more targeted prevention strategies and clinical management. **Objective:** To evaluate the presence of cardiovascular disease and associated risk factors in a group of patients with diabetic foot disease. **Methods:** This is a cross-sectional, descriptive study evaluating data from patients with diabetic foot complications followed at a secondary referral center of the Unified Health System (SUS) in Ceará, Brazil. Data were collected through interviews and medical record reviews. Statistical analysis was performed using SPSS v.23. The study protocol was approved by the Institutional Review Board (IRB) under CAAE number 86087725.6.0000.5534. **Results:** Data of 172 patients, of whom, 58,5% with type 2 DM and 57,7% were male, with age range between 17 to 85 (±11,8), mean disease duration of 14.8 years, were evaluated. A significant low level of education was observed, as 10,5% were illiterate. As for cardiovascular diseases and associated conditions, 27% were already under treatment for coronary arterial diseases (CAD), myocardial infarction or cardiomyopathy, 8,3%, had previous stroke and 29% previous amputations. Hypertension was reported by 70% of patients and was more prevalent in patients with previous CAD (p = 0,04). Other risk factors like dyslipidemia were found in 49% of the patients, 33,6% were obese, 15% had nephropathy, 24% were previous or actual smokers. There was no difference in overall cardiovascular risk factors among different types of DM or sex. In this group of high-risk patients, only 59% reported eye exam checkup, and 75% of them had no HbA1c measured in the previous year. Conclusion: This study shows an outstandingly high prevalence of cardiovascular risk factors among a large group of long-standing diabetics with low levels of education, where basic care for the disease and prevention of complications was not adequately provided. This highlights the need for improved care for these patients.

Keywords: diabetic foot; cardiovascular diseases; risk factors.

DIABETES MELLITUS

47 (3413) CLINICAL AND LABORATORY PREDICTORS OF MORTALITY IN DIABETIC KETOACIDOSIS: A SYSTEMATIC REVIEW

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Introduction: Diabetic ketoacidosis (DKA) is a severe complication of diabetes mellitus associated with high morbidity and mortality. Early identification of clinical and laboratory factors related to disease severity contributes to risk stratification and guides clinical management. **Objective:** To synthesize admission factors associated with increased severity and mortality in adults with diabetic ketoacidosis. **Methods:** Systematic review conducted according to PRISMA guidelines and structured using the PICO strategy. Searches were performed in PubMed and Scopus using MeSH terms and keywords related to diabetic ketoacidosis, mortality, prognosis, severity, and risk factors, combined with Boolean operators (AND/OR). Observational studies published between 2021 and 2026, in English, involving adults (≥18 years) were included. Reviews, case reports, pediatric studies, and studies not evaluating predictors of severity or mortality were excluded. From 101 identified records, 13 studies met eligibility criteria and were included in the qualitative synthesis. **Results:** The included studies identified several admission-related factors associated with increased severity and mortality. Advanced age, type 2 diabetes mellitus, and cardiovascular and renal comorbidities were consistently associated with worse outcomes. Infection and non-adherence to insulin treatment emerged as the main precipitating factors and determinants of poor prognosis. Altered mental status and low Glasgow Coma Scale scores were also associated with greater severity. Laboratory findings related to poor prognosis included lower pH and bicarbonate levels, elevated serum osmolarity, increased lactate, lactate-to-albumin ratio ≥ 0.75 and elevated creatinine. Higher SOFA, APACHE II, APSIII, and OASIS scores demonstrated predictive value for mortality and prolonged hospitalization. Intensive interventions at admission, including invasive ventilation and vasoactive agents, strongly predicted mortality. Conversely, euglycemic DKA associated with SGLT2 inhibitor use showed lower 1-year mortality and reduced incidence of acute kidney injury compared to hyperglycemic DKA. **Conclusion:** Admission-related clinical and laboratory factors, including advanced age, infection, altered mental status, severe metabolic acidosis, renal dysfunction, and high prognostic scores, were associated with worse outcomes in DKA. Early recognition may improve risk stratification and clinical management.
Keywords: diabetic ketoacidosis; mortality predictors; risk stratification.

DIABETES MELLITUS

48 (3476) CLINICAL AND METABOLIC PROFILE OF TYPE 1 DIABETES MELLITUS PATIENTS AND RISK ASSESSMENT OF METABOLIC DYSFUNCTION ASSOCIATED STEATOTIC LIVER DISEASE

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Introduction: Type 1 **DIABETES MELLITUS** (T1DM) exhibits increasing metabolic heterogeneity, with a rising prevalence of overweight, sedentary behavior, and insulin resistance — factors associated with the risk of metabolic dysfunction associated steatotic liver disease (MASLD). However, the true prevalence and severity of hepatic involvement in T1DM patients remain poorly characterized. **Objective:** To describe the clinical, metabolic, and hepatic profile, including the assessment of liver fibrosis risk, in T1DM patients followed at a specialized outpatient clinic. **Methods:** A descriptive, cross-sectional observational study was conducted with adult T1DM patients followed at the Endocrinology outpatient clinic of the *Centro Universitário Faculdade de Medicina do ABC* (FMABC). Clinical, anthropometric, and laboratory data were retrieved from medical records. Liver fibrosis risk was assessed using the FIB-4 index and liver elastography via the ARFI technique. Variables were analyzed descriptively. **Results:** Forty-eight patients were included, with a female predominance and a young mean age, despite long diabetes duration. A high frequency of sedentary lifestyle, overweight or obesity, and suboptimal glycemic control was observed. Most patients presented liver enzymes within normal limits. The FIB-4 index indicated low fibrosis risk in most of the sample, and none of the patients who underwent liver elastography presented advanced fibrosis. **Conclusion:** T1DM patients presented a heterogeneous metabolic profile with a high burden of cardiometabolic risk factors, yet a low prevalence of advanced liver fibrosis in this young population. These findings reinforce the importance of a multidimensional approach in T1DM management, focusing on prevention and early identification of metabolic and hepatic risks.

Keywords: type 1 diabetes mellitus; steatotic liver disease; MASLD.

DIABETES MELLITUS

49 (2947) CONCURRENT AUTOIMMUNE DIABETES MELLITUS AND HYPOTHYROIDISM FOLLOWING NIVOLUMAB THERAPY

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Case presentation: A 60-year-old male patient was admitted with symptoms of insulinopenia and capillary blood glucose levels > 500 mg/dL. He had no previous diagnosis of diabetes mellitus. The patient had been receiving immunotherapy with nivolumab combined with cabozantinib for three months for renal cell carcinoma with pulmonary metastases. During hospitalization, glycated hemoglobin was 9.6%. Laboratory evaluation revealed positive anti-glutamic acid decarboxylase (anti-GAD) antibodies and low C-peptide levels, confirming autoimmune diabetes mellitus. Insulin regimen was initiated. Given the autoimmune context, additional immune-mediated manifestations were investigated. Positive anti-thyroid peroxidase antibodies were detected, while thyroid-stimulating hormone (TSH) levels were initially normal. Months later, TSH increased to 40 mIU/L, confirming hypothyroidism secondary to Hashimoto's thyroiditis. Levothyroxine replacement therapy was initiated, with subsequent normalization of TSH levels. **Discussion:** Autoimmune diseases associated with nivolumab arise from enhanced immune activation directed against tumor cells, which may also trigger inflammation in healthy tissues. Among immune-related endocrinopathies, hypothyroidism is the most common (approximately 6%), whereas fulminant type 1 diabetes mellitus is rare (0.5%–1.4%), with 70% of cases progressing to diabetic ketoacidosis. Most immune-related adverse events occur within the first 3 months of therapy, although they may develop from days to years after treatment initiation. Checkpoint inhibitor-induced diabetes mellitus results from rapid pancreatic beta-cell destruction mediated by autoreactive CD8+ T lymphocytes following PD-1/PD-L1 pathway blockade. Anti-GAD antibodies are detected in approximately 50% of patients. The condition is characterized by abrupt insulin deficiency and a high risk of diabetic ketoacidosis. Blood glucose monitoring is essential, and immunotherapy should generally only be interrupted during diabetic ketoacidosis. Patients with positive thyroid antibodies are at increased risk of thyroid dysfunction, particularly during combination therapy. Therefore, TSH assessment is recommended before treatment initiation and every 4–6 weeks during therapy. **Conclusion:** Because immune-mediated endocrinopathies may be triggered by checkpoint inhibitors, clinicians should routinely monitor glycated hemoglobin and TSH levels to reduce the risk of severe metabolic complications.

Keywords: checkpoint inhibitor; autoimmune diabetes mellitus; Hashimoto's thyroiditis.

DIABETES MELLITUS

50 (3314) CONTINUOUS GLUCOSE MONITORING IN THE INVESTIGATION OF DISCORDANCE BETWEEN HBA1C AND SELF-MONITORED BLOOD GLUCOSE: A CASE REPORT

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Case presentation: Female patient, 56 years old, with type 2 diabetes mellitus (T2DM) since 2000, systemic arterial hypertension, and dyslipidemia, under endocrinological follow-up since 2019. From 2020 onward, she exhibited persistent discordance between elevated glycated hemoglobin (HbA1c), ranging from 9.0% to 10.6%, and capillary blood glucose values apparently within therapeutic targets, associated with frequent symptomatic hypoglycemia. The diagnostic reasoning considered three hypotheses: HbA1c measurement error, altered hemoglobin glycation, and irregular adherence to insulin therapy. The laboratory interference hypothesis was initially supported by a normal fructosamine in April 2023 (258 µmol/L) and concordance between office and home glucose readings. However, longitudinal analysis of self-monitored blood glucose (SMBG) revealed a suspicious pattern: repeatedly round values and homogeneity incompatible with expected physiological variability. In April 2024, a fructosamine of 456 µmol/L was discordant with a simultaneous capillary glucose of 74 mg/dL, reinforcing the hypothesis of concealed chronic hyperglycemia. Continuous glucose monitoring (CGM) over 14 days revealed marked glycemic variability with postprandial hyperglycemia after lunch and recurrent nighttime and early-morning hypoglycemia — entirely incompatible with the SMBG records provided. **Discussion:** The discordance between elevated HbA1c and apparently controlled capillary glucose may arise from laboratory interference, altered hemoglobin glycation, or irregular treatment adherence. Here, the initially normal fructosamine supported laboratory interference; however, its subsequent elevation dissociated from capillary glucose and the suspicious SMBG pattern redirected reasoning toward pseudo-adherence to insulin therapy. CGM was decisive in demonstrating real glycemic variability incompatible with home records, enabling therapeutic simplification and effective glycemic control. **Final comments:** The dissociation between HbA1c and CGM should prompt critical and integrated investigation, not limited to suspicion of laboratory interference. CGM proved essential for revealing the patient's true glycemic profile, avoiding inappropriate therapeutic intensification, and reducing hypoglycemic episodes. This case underscores the importance of therapeutic individualization and careful evaluation of glycemic monitoring methods in T2DM management.

Keywords: continuous glucose monitoring; glycated hemoglobin; type 2 diabetes mellitus.

DIABETES MELLITUS

51 (3539) CONTINUOUS GLUCOSE MONITORING VERSUS STANDARD BLOOD GLUCOSE MONITORING IN ADULTS WITH TYPE 2 DIABETES: IMPACT ON GLYCEMIC CONTROL, A SYSTEMATIC REVIEW

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Introduction: Continuous glucose monitoring (CGM) represents a significant advancement in diabetes management, yet its impact on glycemic control in type 2 diabetes (T2DM) beyond traditional self-monitoring remains incompletely characterized. **Objective:** To evaluate the effect of CGM compared to standard blood glucose monitoring on glycemic control in adults with T2DM. **Methods:** A systematic review was conducted following PICO framework. PubMed was searched using the terms "continuous glucose monitoring," "type 2 diabetes," and "randomized controlled trial." Eligible studies were RCTs enrolling adults with T2DM comparing any CGM modality to standard monitoring and reporting HbA1c as primary outcome. Seven RCTs and meta-analyses published between 2022 and 2025 were included, comprising 3,537 participants. **Results:** CGM was consistently associated with significant HbA1c reductions compared to standard monitoring. In non-insulin-treated T2DM, pooled data showed a weighted mean difference of -0.37% in HbA1c (95% CI -0.49 to -0.24; p < 0.00001) with low heterogeneity. Time in range improved by 8.84% and time above range decreased by 8.14%. In broader populations including insulin-treated individuals, CGM reduced HbA1c by 0.28% and increased time in range by 7.4%. When combined with structured diabetes education, intermittently scanned CGM produced an additional 0.3% HbA1c reduction and 9.9% improvement in time in range versus education alone. **Conclusion:** CGM use in adults with T2DM is associated with clinically meaningful improvements in glycemic control, particularly in non-insulin-treated individuals. Structured education alongside CGM may amplify benefits. Long-term outcomes and cost-effectiveness require further investigation.
Keywords: continuous glucose monitoring; type 2 diabetes; glycemic control.

DIABETES MELLITUS

52 (3479) CORRELATION OF CARDIOMETABOLIC PROFILE WITH THE DEGREE OF HEPATIC STEATOSIS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: A CROSS-SECTIONAL STUDY

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Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD) has become one of the leading causes of chronic liver disease globally, with a prevalence exceeding 65% in patients with type 2 diabetes mellitus (T2DM). The bidirectional relationship between T2DM and MASLD exacerbates oxidative stress and insulin resistance, significantly increasing the risk of progression to advanced fibrosis and other severe hepatic complications. **Objective:** To evaluate the prevalence and cardiometabolic profile of patients with T2DM and varying degrees of hepatic steatosis seen at a metabolic syndrome outpatient clinic of a tertiary hospital, based on the clinical and laboratory characteristics of the sample. **Methods:** A cross-sectional study conducted between 2025 and 2026 with 131 patients with T2DM. Hepatic steatosis and fibrosis were assessed via liver elastography (FibroScan® or S2-Shear Wave Elastography) or ultrasonography. Participants were stratified into two groups: Group 1 (mild steatosis S0–S1; n = 39) and Group 2 (advanced steatosis S2–S3; n = 92). Bivariate and multivariate analyses (binary logistic regression) were performed to identify independent predictors associated with advanced steatosis. **Results:** The prevalence of advanced steatosis in the sample was 70.2%. In the multivariate analysis, body mass index (BMI) (OR: 1.14; 95% CI: 1.03–1.27; p = 0.013), serum triglyceride (TG) levels (OR: 1.01; 95% CI: 1.00–1.01; p = 0.028), and liver stiffness measured in kPa (OR: 1.17; 95% CI: 1.02–1.37; p = 0.040) were identified as factors independently associated with a higher probability of advanced hepatic steatosis. Conversely, the use of anticoagulants or antiplatelet agents showed a negative association with the outcome (OR: 0.24; 95% CI: 0.08–0.67; p = 0.007). The statistical model demonstrated good discriminative capacity, with an area under the curve (AUC) of 0.81. **Conclusion:** The study revealed a high prevalence of advanced hepatic steatosis in the evaluated population with T2DM. Markers of adiposity, lipid profile, and liver stiffness are essential clinical and laboratory tools for risk stratification. These findings reinforce the need for systematic screening for MASLD in patients with T2DM to enable early interventions and reduce progression to severe forms of the disease.
Keywords: type 2 diabetes mellitus; metabolic dysfunction-associated steatotic liver D; liver elastography.

DIABETES MELLITUS

53 (2908) DEPRESSIVE SYMPTOMS, DIABETES-RELATED DISTRESS, AND GLYCEMIC CONTROL IN TYPE 2 DIABETES: A CROSS-SECTIONAL STUDY IN A PUBLIC HEALTHCARE SETTING

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Introduction: Type 2 diabetes mellitus (T2DM) is a highly prevalent chronic disease associated with significant clinical and psychosocial burden. A bidirectional relationship exists between T2DM and depressive symptoms, often linked to poor treatment adherence and suboptimal glycemic control. However, routine screening for psychological distress remains underutilized in public outpatient settings. **Objective:** To determine the prevalence of depressive symptoms in patients with T2DM and evaluate their association with glycemic control (HbA1c) and diabetes-related emotional distress (PAID-13) in a public endocrinology clinic. **Methods:** This cross-sectional study included adult patients with T2DM attending an outpatient endocrinology clinic at a public hospital in Rio de Janeiro, Brazil. Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9), and emotional distress using the Problem Areas in Diabetes (PAID-13), both validated in Brazil. Glycemic control was evaluated using the most recent HbA1c values from medical records. Sociodemographic and clinical data were obtained through chart review and structured interviews. Statistical analyses included descriptive and inferential methods, with significance set at $p < 0.05$. The study was approved by the institutional Research Ethics Committee (Approval number: 8.363.736). **Results:** A total of 152 patients were included (mean age 66.9 ± 10.6 years; 67.8% female), most with disease duration > 10 years (71.7%). Median HbA1c was 7.3%. The majority presented minimal depressive symptoms and low emotional distress. Emotional distress was identified in 23% of patients and was strongly associated with higher PAID-13 scores (61.5 vs. 5.7; $p < 0.001$). No significant correlation was found between depressive symptoms and HbA1c ($p = 0.103$; $p = 0.207$). HbA1c also did not differ according to depressive symptoms or socioeconomic variables. **Conclusion:** In this public healthcare sample, depressive symptoms were not associated with glycemic control, whereas diabetes-related emotional distress remained a clinically relevant burden in a subset of patients. These findings indicate that psychosocial burden may not directly impact metabolic outcomes, yet represents an important dimension of T2DM care. Routine screening for emotional distress should be considered to support a more comprehensive, patient-centered approach in real-world settings.

Keywords: diabetes mellitus, type 2; depression; glycemic control.

DIABETES MELLITUS

54 (3574) IMPROPER SHARPS DISPOSAL AMONG PEOPLE WITH DIABETES IN GOIÁS, BRAZIL

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Introduction: People with diabetes mellitus (DM) on insulin therapy continuously generate sharps waste, as needles, lancets, and syringes, which , if discarded improperly, pose a risk of injury, infection, and environmental harm, particularly to municipal sanitation workers and waste pickers. **Objective:** To describe disposal practices for insulin administration and blood glucose testing supplies among people with DM in the state of Goiás, Brazil. **Methods:** This was a cross-sectional, non-interventional study with convenience sampling, conducted between 2024 and 2025. Insulin-treated individuals with DM were approached during retinopathy screening campaigns organized by the Goiás State Department of Health. Participants completed a structured instrument addressing clinical characteristics, supply use, self-monitoring of blood glucose, previous guidance received, disposal practices and clinical practice follow up. **Results:** A total of 233 patients participated, with a mean age of 59.42 years; 52.6% were women. Type 2 DM (75.2%) and insulin pen use (69.5%) predominated. Self-monitoring of blood glucose at home was reported by 75.2% of participants. Disposal in household waste was reported by 83.3%, whereas only 5.5% returned their materials to health centers or hospitals and 10% to pharmacies or recycling collection points. In addition, 52% had never received any information on appropriate disposal. Among those who had received guidance, 30% still maintained the incorrect practice. Patients with type 1 DM demonstrated greater knowledge of correct disposal than those with type 2 DM (60% versus 48.7%; $p < 0.05$). Regarding preferred drop-off sites, 50% indicated primary health centers and 40.4% pharmacies near their homes. Irrespective if on public or private practice follow-up, inappropriate practices disposal was similar. **Conclusion:** Sharps disposal among insulin-treated people with DM was largely inadequate in Goiás. These findings reinforce the need to expand health education initiatives and to establish accessible collection points, particularly at primary health centers and pharmacies, in order to reduce environmental, occupational, and community risks.

Keywords: diabetes supplies; waste disposal; insulin therapy.

DIABETES MELLITUS

55 (3546) DIABETIC KETOACIDOSIS IN A PATIENT WITH MAURIAC SYNDROME: A CASE REPORT

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Case report: A 21-year-old female was admitted with a three-day history of malaise, vomiting, abdominal pain, and decreased consciousness. Upon admission, she presented with altered mental status, ketotic breath, and blood glucose of 280 mg/dL, confirming diabetic ketoacidosis (DKA), which was promptly treated. Family members denied infections or dietary excesses. Physical examination revealed short stature, right upper quadrant tenderness, and mild hepatomegaly. Further assessment showed severe pubertal delay (Tanner M1P2), primary amenorrhea, and a bone age of 14 years, without cushingoid features. She had a history of poorly controlled type 1 diabetes (T1DM) with highly irregular insulin therapy and a prior ischemic stroke. Hormonal screening confirmed hypogonadotropic hypogonadism (LH: 0.48 mUI/mL, FSH: 1.64 mUI/mL, estradiol < 15 pg/mL). During presentation, she developed acute-on-chronic kidney injury (peak creatinine: 4.5 mg/dL; urea: 131 mg/dL), with subsequent partial recovery. Based on the triad of brittle T1DM, hepatomegaly, and growth failure, Mauriac syndrome was diagnosed. **Discussion:** Mauriac syndrome is a rare complication of T1DM resulting from long-term, severely inadequate glycemic control, characterized by alternating periods of insulin deficiency and intermittent exogenous hyperinsulinization. In this case, the massive influx of glucose into hepatocytes stimulated excessive synthesis and trapping of hepatic glycogen (glycogenosis), explaining the hepatomegaly and abdominal pain. Concomitantly, chronic metabolic stress inhibited the hypothalamus-pituitary-gonadal axis, causing the hypogonadotropic hypogonadism that led to delayed puberty, primary amenorrhea, and delayed bone age. Growth impairment stems from peripheral resistance to growth hormone (GH) induced by poor metabolic control. Finally, the history of premature stroke and the acute-on-chronic kidney injury triggered by DKA dehydration highlight the severe progression of micro-and macrovascular damage associated with the chronic evolution of this syndrome. **Final comments:** This case underscores the profound impact of irregular insulin compliance on neuroendocrine and vascular development. While strict glycemic control is imperative to reverse hepatic glycogenosis, the overall prognosis remains guarded due to established target-organ damage, requiring continuous multidisciplinary support.

Keywords: diabetic ketoacidosis; insulin compliance; Mauriac syndrome.

DIABETES MELLITUS

56 (3464) DIABETIC KETOACIDOSIS IN SERGIPE, BRAZIL (2008–2025): RISING HOSPITALIZATIONS, PERSISTENT HIGH IN-HOSPITAL LETHALITY, AND RACIAL DISPARITIES

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Introduction: Diabetic ketoacidosis (DKA) hospitalizations and in-hospital lethality may serve as sensitive indicators of diabetes care quality. In Sergipe, where 61.6% of the population self-identifies as mixed-race (pardo, IBGE Census 2022, versus 45.3% nationally), and healthcare depends predominantly on the public system, surveillance of DKA trends may expose long-standing structural inequities. **Objective:** To analyze temporal trends in DKA hospitalizations and in-hospital lethality in Sergipe (2008–2025) according to diabetes type, sex, and race/ethnicity, and to identify factors associated with in-hospital death. **Methods:** Ecological time-series study using SIH/Datasus data, ICD-10 codes E101–E141 with DKA as primary diagnosis. Hospitalization rates were calculated per 1,000 SUS admissions. Lethality temporal trend was assessed by Prais-Winsten regression and predictors of death by multiple logistic regression. **Results:** A total of 1,147 hospitalizations and 126 deaths were recorded. Hospitalization rates rose 7.2-fold from 2008 to 2025, increasing progressively from 2016, preceding the COVID-19 pandemic. Type 1 diabetes (E101) showed the steepest increase (3 cases in 2008; 132 in 2025). Mixed-race individuals represented 49.5% of race-classified records, with marked growth after 2022, coinciding with a progressive decline in unknown race registrations (43.3% overall). In-hospital lethality was 10.98%, with no significant temporal trend (Prais-Winsten beta = 0.17, p = 0.61). Age was the only independent predictor of death ($p < 0.001$), with lethality ranging from 1.8% in patients aged 10–14 years to 35.6% in those aged 80 years or older. **Conclusion:** DKA hospitalizations in Sergipe rose markedly from 2016, with in-hospital lethality of nearly 11%, more than 20 times higher than rates reported in high-income countries. The growing proportion of mixed-race records suggests that prior data gaps may have obscured an already existing disparity, though the high proportion of unknown race records prevents definitive race-stratified conclusions. Whether reflecting delayed diagnosis or inadequate outpatient management, the pattern points to structural failures in diabetes care. These findings suggest DKA hospitalizations as sensitive indicators of T1D care quality in low-resource public health systems.

Keywords: type 1 diabetes mellitus; diabetic ketoacidosis; hospitalization.

DIABETES MELLITUS

57 (3394) DIABETIC STRIATOPATHY INITIALLY DIAGNOSED AS HEMORRHAGIC STROKE IN AMAZON: A CASE REPORT

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Case presentation: Patient N.L.S., 75 years old, female, hypertensive and with a long-standing history of diabetes mellitus, presented with an approximately 30-day history of pain in the right hemibody associated with involuntary movements. She was admitted to the Emergency Department with an initial diagnosis of Stroke. Neuroimaging revealed hyperdensity in the basal ganglia. Neurosurgical evaluation was requested due to the initial diagnosis of hemorrhagic stroke, and conservative management without surgical indication was maintained. During subsequent reassessment under Internal Medicine follow-up, significant hyperglycemia associated with marked glycosuria was observed. Considering the context of decompensated diabetes mellitus, the hypothesis of Diabetic Striatopathy was raised. After glycemic control measures and 24 hours following the initiation of carbamazepine, the patient showed clinical improvement, with resolution of pain and involuntary movements. **Discussion:** Diabetic Striatopathy is a rare complication of decompensated diabetes mellitus. It is characterized by severe non-ketotic hyperglycemia and involuntary movements, such as chorea and/or ballism, presenting alterations in the basal ganglia on computed tomography or magnetic resonance imaging, including striatal hyperdensity, which may mimic hemorrhagic stroke, as observed in the present case. Treatment consists of glycemic control and, when necessary, medications for movement control, such as antipsychotics, benzodiazepines, or anticonvulsants. **Final comments:** Although rare, Diabetic Striatopathy has a favorable prognosis when properly treated and should be considered in patients with decompensated diabetes mellitus in emergency settings. Accurate diagnosis ensures targeted treatment, avoiding unnecessary interventions and allowing resolution of the condition.

Keywords: diabetes mellitus; hyperglycemia; chorea.

DIABETES MELLITUS

58 (3572) DIABETIC STRIATOPATHY PRESENTING AS HEMIBALLISMUS AFTER TAVI IN UNCONTROLLED TYPE 2 DIABETES

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An 80-year-old woman with non-insulin-dependent type 2 diabetes mellitus, chronic kidney disease, hypertension, hypothyroidism, and cognitive impairment was admitted after hip arthroplasty for a fracture secondary to a fall. During the postoperative period, she developed refractory constipation, abdominal distension, nausea, vomiting, somnolence, inflammatory marker elevation, and renal dysfunction, requiring intensive care admission. Abdominal computed tomography (CT) demonstrated fecal impaction. Antibiotics and nasogastric tube placement resulted in improvement. During hospitalization, the patient remained hyperglycemic despite increasing insulin adjustments, maintaining serum glucose levels between 250 and 500 mg/dL. She also developed two suspected infectious episodes, characterized by fever and worsening inflammatory markers, attributed to a pulmonary source and an unidentified focus. Both improved after antibiotic therapy. Routine transthoracic echocardiography revealed aortic stenosis, leading to the indication for transcatheter aortic valve implantation (TAVI). After TAVI, the patient developed involuntary movements involving the left upper limb, later characterized as left-sided hemiballismus. Cranial CT demonstrated right striatal abnormalities suggestive of diabetic striatopathy, without evidence of acute stroke. Insulin adjustment associated with haloperidol resulted in clinical improvement. Diabetic striatopathy is a rare neurological disorder associated with uncontrolled hyperglycemia, characterized by hemichorea or hemiballismus associated with basal ganglia abnormalities on neuroimaging. It occurs predominantly in elderly women with type 2 diabetes mellitus. In this case, the diagnosis could easily be confused with stroke, delirium, metabolic encephalopathy, or infectious complications due to the prolonged postoperative course and recent cardiovascular intervention. The association between severe hyperglycemia and compatible imaging findings was essential for reaching the diagnosis. Diabetic striatopathy should be considered in elderly diabetic patients presenting with acute involuntary movements, especially in the presence of hyperglycemia and neuroimaging abnormalities. Early recognition results in faster treatment, better clinical outcomes, and lower costs related to investigative exams.

Keywords: diabetic striatopathy; type 2 diabetes mellitus; hemiballismus.

DIABETES MELLITUS

59 (2996) DURABILITY OF TYPE 2 DIABETES REMISSION AFTER INCRETIN-BASED THERAPIES: PREDICTORS, RELAPSE, AND METABOLIC OUTCOMES

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Introduction: The management of type 2 diabetes mellitus (T2DM) has increasingly shifted from glycemic control alone toward the achievement of sustained remission. Incretin-based therapies, including GLP-1 receptor agonists and dual GIP/GLP-1 agonists, have demonstrated significant effects on weight reduction, glycemic control, and beta-cell preservation, raising interest in their long-term impact on diabetes remission. **Objective:** To evaluate the durability of T2DM remission induced by incretin-based therapies, identifying predictors of remission, relapse rates after treatment discontinuation, and long-term metabolic outcomes. **Methods:** A systematic review following PRISMA recommendations and a PICO-based strategy was conducted using PubMed, Web of Science, and CNKI databases, covering studies published between 2015 and 2025. Search terms included "type 2 diabetes remission" and "pharmacological intervention". A total of 438 studies were initially identified. After independent screening by two reviewers, 385 studies were excluded due to follow-up shorter than three months, absence of standardized remission criteria, insufficient beta-cell function data, or publication as non-peer-reviewed conference abstracts. Fifty-three studies were included in the final analysis. **Results:** Data from the multicenter GLIMPLES study (n = 14,141) showed that strict remission (HbA1c < 6.5% without glucose-lowering medication) was achieved in 5.8% of GLP-1RA users, increasing to 18.3% when maintenance therapy was permitted. Major predictors of remission included shorter disease duration (<6 years), preserved pancreatic function (C-peptide > 3.0 ng/mL), and initial weight loss greater than 10%. Treatment discontinuation was associated with progressive metabolic relapse, with estimated weight regain of 0.8 kg/month and return to baseline weight within approximately 1.7 years. Despite relapse risk, remission periods were associated with lower rates of major adverse cardiovascular events (HR 0.65) and reduced progression of albuminuria. **Conclusion:** Incretin-based therapies are effective strategies for inducing T2DM remission, particularly in patients with preserved beta-cell function and early disease duration. However, maintenance of metabolic benefits appears strongly dependent on continued treatment and sustained weight reduction.

Keywords: type 2 diabetes mellitus; glucagon-like peptide 1 receptor agonists; remission induction.

DIABETES MELLITUS

60 (3489) EARLY BRAZILIAN REAL-WORLD EXPERIENCE WITH AUTOMEAL IN AUTOMATED INSULIN DELIVERY: LONGITUDINAL IMPACT ON TREATMENT BURDEN AND GLYCEMIC CONTROL

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Carbohydrate counting remains a major challenge in type 1 diabetes management, contributing substantially to treatment burden and potentially limiting adherence to intensive insulin therapy. Features designed to simplify meal-related insulin delivery may reduce this burden while preserving glycemic effectiveness. This study aimed to evaluate the longitudinal impact of AutoMeal introduction on treatment burden and glycemic outcomes in people with type 1 diabetes using automated insulin delivery in a real-world Brazilian setting. We conducted a retrospective observational study including individuals with type 1 diabetes with available paired 30-day and 90-day follow-up data after initiation of automated insulin delivery. At 30 days, none of the participants were using AutoMeal. At 90 days, participants were categorized according to AutoMeal use. The primary endpoint was change in daily bolus frequency, as a surrogate marker of treatment burden. Secondary outcomes included time in range (70–180 mg/dL), time above range (>180 mg/dL and >250 mg/dL), time below range (<70 mg/dL and <54 mg/dL), and coefficient of variation. A total of 66 individuals were included, comprising 37 AutoMeal users and 29 non-users. Between 30 and 90 days, AutoMeal users demonstrated a marked reduction in daily bolus frequency (6.2 to 2.6 boluses/day; Δ -3.6), whereas non-users showed only a modest reduction (6.9 to 6.4 boluses/day; Δ -0.5), with a significant between-group difference in longitudinal change ($p < 0.001$). Time in range remained stable in both groups (AutoMeal: 79.8% to 77.6%; non-users: 77.9% to 77.3%; $p = 0.40$ for interaction). No significant between-group differences were observed in time above range, glycemic variability, or time below range < 70 mg/dL. Although a statistical difference was observed for time below range < 54 mg/dL, absolute values remained very low in both groups. These findings suggest that AutoMeal may substantially reduce treatment burden while preserving glycemic control in people with type 1 diabetes using automated insulin delivery in routine clinical practice.

Keywords: type 1 diabetes; automated insulin delivery; treatment burden.

DIABETES MELLITUS

61 (3276) EARLY WOUND HEALING DYNAMICS WITH PIN-07 IN CHRONIC DIABETIC FOOT ULCERS: PRELIMINARY CLINICAL FINDINGS

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Introduction: Diabetic foot ulcers (DFU) are severe chronic complications of diabetes mellitus associated with infection, amputations, impaired quality of life, and high healthcare costs. Current topical therapies remain limited by the lack of targeted action on neurovascular and inflammatory mechanisms involved in impaired wound healing. PIN-07 is a novel experimental topical compound developed to target neurovascular dysfunction and tissue repair pathways. **Objective:** To describe the clinical evolution and wound healing dynamics of chronic diabetic foot ulcers treated with PIN-07. **Methods:** This prospective descriptive pilot study included adults with diabetes and chronic noninfected diabetic foot ulcers up to grade 2 according to the University of Texas classification who received topical treatment with PIN-07 within a pilot clinical protocol. Clinical characteristics, glycemic parameters, inflammatory markers, and wound evolution were observed over 12 weeks using the Pressure Ulcer Scale for Healing (PUSH). The study was approved by the Research Ethics Committee of Universidade Estadual do Ceará (CAAE 86087725.6.0000.5534; approval number 7.419.752). **Results:** Twenty-four participants completed treatment with PIN-07. Most participants were male (68%). Mean baseline HbA1c was 8.8 ± 0.3%, with preserved renal function observed in all participants. Baseline inflammatory activity was elevated, with mean C-reactive protein levels of 12.9 ± 2.3 mg/L and erythrocyte sedimentation rate of 84.9 mm/h. Mean PUSH score decreased from 11.3 ± 0.5 at baseline to 8.4 ± 1.5 at week 12 ($p < 0.05$). Early wound closure was observed in six participants, including closure as early as week 4 and most occurring by week 6. Favorable tissue repair patterns and healthy granulation tissue formation were observed, including in ulcers with greater tissue complexity and areas of bone exposure. PIN-07 demonstrated satisfactory tolerability throughout follow-up. **Conclusion:** PIN-07 demonstrated promising preliminary clinical activity in chronic diabetic foot ulcers, with favorable wound healing dynamics, early closure in a subset of participants, and satisfactory tolerability. These findings support further investigation of this novel neurovascular topical therapy in larger controlled clinical studies.
Keywords: diabetic foot; diabetes mellitus; wound healing.

DIABETES MELLITUS

62 (3188) EFFECT OF OAT BETA-GLUCAN ON GLYCEMIC CONTROL IN TYPE 2 DIABETES MELLITUS: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMIZED CONTROLLED TRIALS

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Introduction: Oat beta-glucan is a viscous soluble dietary fiber with established cholesterol-lowering properties. Its role in glycemic control in type 2 diabetes mellitus remains uncertain due to heterogeneous evidence from individual trials. **Objective:** To evaluate the effect of oat and oat beta-glucan on glycated hemoglobin, fasting plasma glucose, and postprandial glucose area under the curve in patients with type 2 diabetes mellitus through a systematic review and meta-analysis of randomized controlled trials. **Methods:** PubMed, EMBASE, and Cochrane databases were searched for randomized controlled trials comparing oat or oat beta-glucan versus control exclusively in patients with type 2 diabetes mellitus. Studies involving healthy subjects, prediabetes, or metabolic syndrome without confirmed diabetes were excluded. Data were pooled using a random-effects model with restricted maximum likelihood estimation. Heterogeneity was quantified using the I-squared statistic. A meta-regression analyzed the dose-response relationship between beta-glucan dose and postprandial glucose response. **Results:** Ten studies comprising 13 trial comparisons and 557 participants met inclusion criteria. Oat beta-glucan significantly reduced glycated hemoglobin compared to control across five trials reporting chronic outcomes (mean difference: –0.16%; 95% confidence interval: –00.30 to –00.02; I-squared = 36.8%). No significant effect on fasting plasma glucose was observed across four trials (mean difference: –00.09 mmol/L; 95% confidence interval: –0.34 to 0.17; I-squared = 4.2%). Eight acute trial comparisons demonstrated significant postprandial glucose area under the curve reduction (mean difference: –0188.75 mmol.min/L; 95% confidence interval: –0278.23 to –099.27; I-squared = 98.4%). Meta-regression revealed an inverse linear dose-response relationship between beta-glucan dose and postprandial glycemic response. **Conclusion:** Oat beta-glucan significantly reduces glycated hemoglobin and attenuates postprandial glycemic responses in type 2 diabetes mellitus, without significant effect on fasting plasma glucose. These findings support oat beta-glucan as a dietary adjunct in type 2 diabetes management, particularly for long-term glycemic control and postprandial hyperglycemia attenuation.
Keywords: type 2 diabetes mellitus; beta-glucan; glycemic control.

DIABETES MELLITUS

63 (3112) EFFECT OF SGLT2 INHIBITORS ON RENAL AND CARDIOVASCULAR OUTCOMES IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND CHRONIC KIDNEY DISEASE: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Type 2 diabetes mellitus (T2DM) is one of the leading causes of chronic kidney disease (CKD) and is associated with progression to end-stage renal disease and increased cardiovascular risk. Sodium-glucose cotransporter 2 inhibitors (SGLT2i) have demonstrated relevant cardiorenal benefits in this population. **Objective:** To evaluate the effect of SGLT2i on CKD progression and cardiovascular outcomes in patients with T2DM and CKD. **Methods:** The systematic review was conducted according to the Cochrane Handbook for Systematic Reviews and PRISMA 2020 guidelines. Searches were performed in the PubMed, Cochrane Library, and Embase databases. The protocol was registered in PROSPERO (CRD420261370393). Randomized controlled trials published from 2019 onward involving adults with T2DM and CKD receiving SGLT2i (canagliflozin, dapagliflozin, empagliflozin, or sotagliflozin) compared with placebo or standard treatment were included. Renal outcomes included CKD progression, sustained decline in estimated glomerular filtration rate (eGFR), end-stage renal disease, and renal death. Cardiovascular outcomes were major adverse cardiovascular events (MACE) and hospitalization for heart failure. Risk of bias was assessed using the Cochrane Risk of Bias 2 (RoB 2) tool, and eligible data were pooled for meta-analysis. **Results:** Six studies involving 21,516 participants were included. A favorable effect associated with SGLT2i use was observed across the evaluated outcomes. In the pooled analysis of renal outcomes, a reduction in the risk of renal progression was identified (RR 0.71; 95% CI: 0.60–0.82), with no heterogeneity (I^2 = 0%). For cardiovascular outcomes, the analysis showed a reduction in the risk of cardiovascular events (RR 0.76; 95% CI: 0.59–0.98), with moderate heterogeneity (I^2 = 59%). Additionally, Heerspink *et al.* and Herrington *et al.* reported reductions in composite cardiorenal outcomes, both with an HR of 0.64. **Conclusion:** The findings suggest cardiorenal benefits of SGLT2i in patients with T2DM and CKD. However, the limited number of studies and heterogeneity among subgroups hinder comparisons between SGLT2i, highlighting the need for more standardized outcome definitions and further comparative studies.
Keywords: type 2 diabetes mellitus; chronic kidney disease; sodium-glucose transporter 2 inhibitors.

DIABETES MELLITUS

64 (3000) EFFECTS OF A HEALTHY LIFESTYLE EDUCATION PROGRAM ON DIETARY INTAKE IN ADULTS WITH PREDIABETES AND DIABETES: A MULTICENTER STUDY

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Introduction: Diabetes lifestyle education programs may improve dietary behavior and clinical outcomes. **Objective:** To evaluate the impact of a diabetes education program based on supervised physical exercise and structured educational intervention on dietary intake in adults with diabetes and prediabetes from Juiz de Fora and Belo Horizonte. **Methods:** This 12-week intervention program included periodic meetings with supervised exercise sessions and diabetes education activities based on the Diabetes College™ curriculum. Dietary intake was assessed at baseline and six months after the end of the intervention (follow-up) using an adapted quantitative food frequency questionnaire (FFQ). Dietary intake between the two time points was compared using the Wilcoxon signed-rank test for paired samples. **Results:** A total of 226 participants initiated the educational intervention, with a median age of 54 years (43–62), predominantly female (66.8%) and from Juiz de Fora (54.9%). Type 2 diabetes predominated (47.3%), followed by prediabetes (35%). Median fasting glucose was 119 mg/dL (101–144), and median glycated hemoglobin was 6.6% (6.1–7.7). Of the initial sample, 100 participants completed the FFQ at follow-up. The educational intervention resulted in a 13% reduction in daily intake of refined cereals and tubers ($p = 0.001$) and a 37% reduction in processed meat consumption ($p < 0.001$), in addition to a slight reduction in animal fat intake, 5.8 (2.0–17.8) vs. 5.8 (2.1–10.4), $p = 0.024$. There was a significant increase in milk and dairy product consumption (148.3 g vs. 200 g; $p = 0.025$). On the other hand, reductions were observed in poultry (33.8 g vs. 17.4 g; $p < 0.001$) and fish consumption (5.6 g vs. 2.1 g; $p = 0.005$), along with an increase in fried food intake (13.0 g vs. 17.9 g; $p = 0.003$). No significant changes were observed in the remaining food groups. Notably, fruit and vegetable intake remained high both at baseline and follow-up (>480 g/day), as did legume consumption (80 g/day). **Conclusion:** The educational intervention contributed to improvements in dietary patterns, particularly regarding carbohydrate and animal fat intake, while maintaining other previously healthy habits. However, other desirable dietary improvements were not observed. Despite the increase in fried food consumption and reduction in lean protein sources, the findings reinforce the potential of educational programs to support nutritional management in diabetes.
Keywords: diabetes education; dietary intake; diabetes mellitus.

DIABETES MELLITUS

65 (3425) EFFECTS OF TIRZEPATIDE IN THE MANAGEMENT OF PATIENTS WITH TYPE 1 DIABETES: SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Despite advances in insulin therapy, many individuals with type 1 diabetes mellitus (T1DM) remain outside recommended glycemic targets and present increasing rates of overweight and obesity. Tirzepatide, a dual GIP and GLP-1 receptor agonist, has demonstrated metabolic benefits in other populations, but its role in T1DM remains unclear. **Objective:** To evaluate the effects of tirzepatide on glycemic and anthropometric outcomes in adults with T1DM through a systematic review and meta-analysis. **Methods:** A systematic review was conducted according to PRISMA guidelines, including observational studies and randomized controlled trials assessing tirzepatide in adults with T1DM. The literature search was performed in MEDLINE, EMBASE, and Cochrane Library databases, without language or publication date restrictions. Search terms related to the intervention and clinical condition were combined using Boolean operators, including "tirzepatide", "LY3298176", "Mounjaro", and "Zepbound" associated with "Type 1 Diabetes" and "T1DM". Outcomes of interest were changes in HbA1c, body weight, and total daily insulin dose. Exploratory within-group meta-analyses were performed using random-effects models. Statistical heterogeneity was assessed using Cochran's Q test and quantified with the I^2 statistic, with higher values indicating greater inconsistency across studies. A leave-one-out sensitivity analysis was also conducted to evaluate the robustness of pooled estimates and the influence of individual studies on overall results. Statistical significance was set at $p < 0.05$. **Results:** Five studies comprising 186 participants were included. Tirzepatide was associated with significant reductions in HbA1c (-0.49%; $p < 0.0001$), body weight (-8.43 kg; $p < 0.001$), and total daily insulin dose (-0.17 U/kg/day; $p < 0.0001$). Substantial heterogeneity was observed across analyses. The leave-one-out analysis confirmed the robustness of the findings, despite variations in effect size across iterations. **Conclusion:** Available evidence suggests that tirzepatide is associated with improvements in glycemic control, body weight, and insulin requirements in adults with T1DM in within-group analyses. However, the predominantly observational nature of the data limits causal inference, and further randomized controlled trials are warranted.
Keywords: type 1 diabetes; tirzepatide; glycated hemoglobin.

DIABETES MELLITUS

66 (2920) EUGLYCEMIC DIABETIC KETOACIDOSIS IN A PATIENT WITH LATENT AUTOIMMUNE DIABETES IN ADULTS TREATED WITH A SODIUM-GLUCOSE COTRANSPORTER-2 INHIBITOR, DUE TO A MISTAKEN DIAGNOSIS OF TYPE 2 DIABETES MELLITUS: A CASE REPORT

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Case presentation: A 60-year-old female, previously diagnosed with type 2 diabetes mellitus was admitted with acute symptoms of asthenia, vomiting and polydipsia. Her medical history included use of empagliflozin, pioglitazone, and metformin since diagnosis, four years ago. Also two episodes of diabetic ketoacidosis in the past year and glycated hemoglobin at 8,0%. Family history of brother with type 2 diabetes. The physical examination showed a slight increase in blood pressure, tachycardia, BMI 23.4 kg/m^2; signs of dehydration, and ketotic breath. Laboratory tests revealed metabolic acidosis (pH 7,04, bicarbonate 9,1 mmol/L), hyperglycemia (223 mg/dL) and ketonuria+++. It was diagnosed as euglycemic diabetic ketoacidosis precipitated by the use of sodium-glucose cotransporter 2 (SGLT2) inhibitors. After clinical stabilization, antiinsulin antibodies and islet cell antibodies were measured and were negative. Anti-glutamic acid decarboxylase antibodies (anti-GAD) were 33 U/mL (N < 10 U/mL), C-peptide 0,44 ng/mL (1,1– 4,4 ng/mL). The patient was diagnosed with latent autoimmune diabetes (AD) in adults (LADA) and maintained on insulin therapy regimen. **Discussion:** LADA is a slowly progressive form of AD characterized by β-cell destruction that occurs in adults (typically diagnosed after age 30-35), presents initially without requiring insulin treatment (at least 6 months) and is identified by the presence of islet autoantibodies (particularly anti-GAD). It is often confused with type 2 diabetes due to similar initial clinical presentation. SGLT2 inhibitors are antidiabetic drugs that lower blood glucose levels by blocking glucose reabsorption in the kidneys, leading to glycosuria. The use of SGLT2 inhibitors, in this case empagliflozin, in patients with LADA misdiagnosed as type 2 diabetes is particularly concerning due to the increased risk of diabetic ketoacidosis. It occurs by increased glucagon: insulin ratio, progressive endogenous insulin deficiency, increased ketone reabsorption and hypovolemia. This patient acute presentation was related not only by the misdiagnosis of type 2 diabetes mellitus, but also by the continued use of empagliflozin despite two previous episodes of diabetic ketoacidosis. **Conclusion:** This case demonstrates the need to accurately differentiate between types of diabetes mellitus, for proper treatment. While SGLT2 inhibitors are effective for type 2 diabetes, their use in autoimmune diabetes has not been approved and may be harmful.
Keywords: diabetic ketoacidosis; sodium-glucose cotransporter 2 inhibitors (SGLT2i; latent autoimmune diabetes in adults.

DIABETES MELLITUS

67 (3048) EVALUATION OF EDUCATION ON INSULIN THERAPY IN DIABETIC PATIENTS RECEIVING OUTPATIENT FOLLOW-UP

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Introduction: diabetes mellitus is a pathology that occurs due to a deficient secretory response or defective action of insulin, leading to an inadequate use of glucose by tissues. The main consequence is hyperglycemia, which leads to various complications in the body. The management of diabetes mellitus involves not only oral antidiabetic agents and the newer non-insulin injectable therapies, but also insulin therapy, which should be initiated in a timely manner to ensure glycemic control and reduce glucotoxicity. However, the use of insulin requires knowledge of proper handling techniques and careful attention to the dosages prescribed by physicians in order to avoid underdosing or overdosing, as such errors are highly likely to occur. **Objective:** To analyze insulin therapy education among diabetic patients followed at the endocrinology outpatient clinic by evaluating their knowledge regarding insulin preparation, the accuracy of the aspirated dose in relation to the prescribed dose, and the frequency of correct self-administration technique. **Methods:** A standard and complementary questionnaire for diabetic patients was applied in a sample of 75 patients on insulin therapy, in order to evaluate the application techniques and whether the patients followed the prescribed insulin dosages. The research was conducted in both sexes, with diabetes patients who have been undergoing insulin therapy for at least 6 months. **Results:** The aspiration of the insulin according to the medical prescription was not adequate in 47.7%, of those who aspirated erroneously, 67.7% performed a dose more and 32.3%, dose less. Out of the total number of patients, 58.7% presented hyperglycemia and 64.0%, hypoglycemia in the last 6 months. Regarding the technique of application, 90.8% apply the insulin in the appropriate place and 76.9% do with an angle of 90° with the skin. **Conclusion:** It was concluded that the technique and adequate aspiration of insulin are of fundamental importance for the success of the treatment, besides avoiding various complications such as bleeding, pain, hyper and hypoglycemia, lipodystrophy, among others. It is apparent that factors such as visual acuity or lack of knowledge about the disease lead patients to apply super or subdose and not to worry about the consequences of these errors, which are easily avoided. Thus, the study aims to identify the errors and also promote an orientation to correct this practice.

Keywords: education; diabetes mellitus; insulin therapy.

DIABETES MELLITUS

68 (3043) EVALUATION OF GLYCEMIC CONTROL AND ITS CORRELATION WITH DAILY CARBOHYDRATE INTAKE IN PATIENTS WITH TYPE 1 DIABETES MELLITUS RECEIVING OUTPATIENT CARE

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Introduction: Type 1 diabetes mellitus and latent autoimmune diabetes in adults are autoimmune conditions characterized by the destruction of pancreatic beta cells and hyperglycemia, requiring multidisciplinary management. Carbohydrate counting is considered a gold-standard strategy for optimizing glycemic control; however, adherence is challenged by socioeconomic and cultural barriers, especially in regions with carbohydrate-rich dietary patterns. **Objective:** To evaluate glycemic control based on daily carbohydrate intake in adult patients with type 1 diabetes mellitus and latent autoimmune diabetes in adults followed at an endocrinology outpatient clinic. **Methods:** This was a cross-sectional study involving 71 adult patients with type 1 diabetes mellitus and latent autoimmune diabetes in adults. Data were collected from physical medical records. Nutritional assessment was based on carbohydrate counting compared with individual estimated needs according to basal metabolic rate calculated using the Harris-Benedict equation. Glycemic control was assessed through glycated hemoglobin, fasting blood glucose, and postprandial blood glucose levels. **Results:** There was a predominance of female patients (60.6%) and individuals aged 18 to 28 years. The mean glycated hemoglobin level was 8.7%, with 74.6% of patients above therapeutic target. Most participants used a fixed insulin dose regimen (88.7%); only 11.3% performed carbohydrate counting with variable insulin dosing, representing the group with lower mean glycated hemoglobin levels (7.7% versus 8.6%) and fasting blood glucose levels (159.8 versus 189.3 milligrams per deciliter). The mean carbohydrate intake was 196.8 grams per day, with 60% of patients meeting recommendations estimated by the Basal Metabolic Rate. Consumption of açaí twice a day was associated with a mean intake of 373 grams of carbohydrates per day, significantly higher than the overall mean ($p < 0.0001$). **Conclusion:** It was demonstrated that a relationship between glycemic control and the amount of daily carbohydrate intake was positively identified in this present study, since most patients presented a carbohydrate intake lower than 60% of the Basal Metabolic Rate, which is considered ideal according to literature. Furthermore, the group of patients who performed carbohydrate counting achieved better glycemic control, based on glycated hemoglobin and fasting blood glucose levels, when compared to patients using fixed insulin doses.

Keywords: type 1 diabetes mellitus; carbohydrate counting; glycemic control.

DIABETES MELLITUS

69 (3251) EVALUATION OF THE AGREEMENT BETWEEN THE FIBROSIS-4 INDEX (FIB-4) AND ABDOMINAL ULTRASONOGRAPHY IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE (MASLD) IN PATIENTS WITH TYPE 2 DIABETES

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Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD) is directly linked to metabolic dysfunctions such as obesity, insulin resistance, and dyslipidemia, representing a frequent complication in type 2 diabetes mellitus (T2DM). The Fibrosis-4 Index (FIB-4) has emerged as a low-cost, non-invasive tool for fibrosis screening; however, its accuracy may vary according to age and metabolic control. **Objective:** To analyze the agreement between the FIB-4 score and abdominal ultrasonography findings in type 2 diabetes mellitus patients, applying age-adjusted cut-offs. **Methods:** A cross-sectional study was conducted through the analysis of 66 medical records. Three patients were excluded (one due to age below the validated range for the score and two due to incomplete data), +resulting in a final sample of 63 individuals. Data on age, transaminases, platelets, and glycated hemoglobin were collected. FIB-4 was stratified by age: < 65 years (low risk < 1.3) and ≥ 65 years (low risk < 2.0). Results were compared with degrees of steatosis on ultrasonography (mild, moderate, and severe). **Results:** The mean glycated hemoglobin was 8.8% (5.6% – 14.2%), indicating heterogeneous glycemic control. The global prevalence of MASLD on ultrasonography within the studied sample was 74.6% (n = 47). In the group aged ≥ 65 years (n = 27), FIB-4 classified 62.9% as low risk, showing good clinical agreement, given that 70.5% of these had ultrasonography described as normal or mild. In the group aged < 65 years (n = 36), 63.8% were classified as low risk; however, 19.4% of these (n=7) already presented moderate or severe steatosis on imaging, suggesting a potential underestimation of risk by the score alone in younger patients. **Conclusion:** The Fibrosis-4 Index proves to be a valuable screening tool, especially in the elderly population. However, in younger patients with type 2 diabetes mellitus, ultrasonography is a strategic complementary method. Despite being operator-dependent, its ability to identify moderate to severe steatosis in individuals with low FIB-4 scores allows for early metabolic intervention, mitigating the risk of progression to advanced fibrosis that the score alone may not detect prematurely.
Keywords: type 2 diabetes mellitus; MASLD; FIB-4 Index.

DIABETES MELLITUS

70 (3097) EVALUATION OF THE USE OF RTCGM IN ADULTS WITH TYPE 1 DIABETES MELLITUS IN A PUBLIC OUTPATIENT CLINIC: A CROSS-SECTIONAL STUDY

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Introduction: The management of type 1 diabetes mellitus (T1D) represents a continuous clinical challenge, requiring intensive glycemic monitoring to reduce acute and chronic complications of the disease. Real-time continuous glucose monitoring (rtCGM) systems expand the evaluation of the glycemic profile, but metabolic control also depends on numerous factors, among them, diabetes education, adherence to carbohydrate counting, and optimization of insulin therapy. **Objective:** To evaluate glycemic parameters obtained by rtCGM and the clinical-therapeutic profile of adults with T1D in a public reference center. **Methods:** Cross-sectional observational study, using electronic health record data of adults with T1D followed at a teaching reference outpatient clinic who used rtCGM at least once. Available historical data were analyzed, without a minimum active time criterion. Correlations between selected variables were evaluated. Effect of active sensor time on glycemic parameters was assessed by linear regression. The results were expressed as mean ± SD, n (%), or r. **Results:** 97 patients were analyzed, aged 34.6 ± 9.4 years, predominantly female (52%), and with a BMI of 26.7 ± 5.7 kg/m². Mean A1c was 8.7 ± 2.3%. Mean CGM glucose was 176.8 ± 52.7 mg/dL, and the GMI (Glucose Management Indicator) was 7.5 ± 1.3%. Time in range, TIR, (70-180 mg/dL) was 47.4 ± 19.0%, time above range, TAR, was 46.2 ± 20.8%, and time below range, TBR, was 6.5 ± 6.9%. The mean active sensor time was 73.8 ± 30.0%. The total daily insulin dose per kg of body weight was 0.7 ± 0.3 IU/kg/day, 51% of the individuals performed carbohydrate counting, and 47% used insulin analogs. There was a positive correlation between A1c and GMI (r = 0.54; p < 0.0001). Each 10-percentage point increase in active sensor time was associated with a 0.2% reduction in GMI, a 2.5% increase in TIR, and a 2.3% reduction in TAR (p < 0.0001 for all comparisons). **Conclusion:** These results highlight the direct role of rtCGM on glycemic control indicators in this population. The global improvement in glycemic parameters with greater active sensor time reinforces the importance of intensive glycemic monitoring in the context of T1D. However, T1D management remains challenging, as metabolic control persist above the targets recommended by global guidelines. Finally, the use of technology must always be accompanied by diabetes education, adherence to carbohydrate counting, and therapeutic optimization for better outcomes.
Keywords: type 1 diabetes mellitus; continuous glucose monitoring; diabetes technology.

DIABETES MELLITUS

71 (3278) EXPERIENCE OF ARTIFICIAL INTELLIGENCE (AI)-BASED DIABETIC RETINOPATHY SCREENING IN PRIMARY HEALTH CARE IN SOUTHERN BRAZIL: LESSONS LEARNED FROM A PRAGMATIC CLINICAL TRIAL

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Introduction: Diabetic retinopathy (DR) is a major cause of preventable blindness, highlighting the need for early screening in Primary Health Care (PHC). While AI-based tools can increase access to detection, there is still limited evidence from large, real-world PHC trials using handheld retinal cameras. **Objective:** To describe the conduct and operational effectiveness of a pragmatic randomized clinical trial for DR screening in PHC in southern Brazil using retinal image assessment by AI or ophthalmologists. **Methods:** This descriptive study is linked to an ongoing pragmatic clinical trial in Santa Cruz do Sul, southern Brazil. A total of 1058 individuals with diabetes mellitus were randomized to retinal image assessment by either AI or ophthalmologists. The protocol included integrated care pathways, portable retinal cameras, training of non-ophthalmologist healthcare professionals, operational protocols, and integration between PHC services and the municipal referral regulation system. Operational barriers, logistical adaptations, and corrective actions were systematically evaluated throughout the study. **Results:** DR screening in PHC proved feasible, although substantial adaptations to the original protocol were required. Low spontaneous participation rates led to the implementation of 25 mass screening campaigns, accounting for 698 inclusions, whereas fixed-site screening units contributed with 360 participants. Outcomes were assessed in 831 participants, among whom 248 (29.8%) had referable findings and 219 (88.3%) were referred through the municipal referral regulation system. Among them, 248 (29.8%) presented referable findings, and 219 (88.3%) were referred through the municipal regulation system. Of the referred participants, 151 (68.9%) attended an ophthalmology consultation, with a mean waiting time of 88.5 days. The primary outcome, appropriate referral, was assessed in 82 participants, having been achieved in 73.2%. Major challenges included image quality, adherence to referral pathways, and communication between PHC units and the municipal regulation system, requiring additional staff training and refinement of image acquisition protocols. **Conclusion:** AI-based DR screening was feasible in PHC settings, but its success required logistical adaptations, active patient recruitment, and stronger integration with the municipal referral system to ensure continuity of care. Funding: Fapesp, Fapergs, IATS, FIPE (HCPA).
Keywords: diabetic retinopathy; artificial intelligence; primary health care.

DIABETES MELLITUS

72 (3044) FAMILIAL PARTIAL LIPODYSTROPHY PHENOTYPE WITH LATE-ONSET METABOLIC DISTURBANCES IN A PATIENT CARRYING A CIDEC GENE VARIANT: A CASE REPORT

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Case presentation: A 65-year-old Brazilian woman, born to consanguineous parents (first cousins), was diagnosed at age 49 with type 2 diabetes mellitus (T2DM) in the context of polyuria, polydipsia, and a 10-kg weight loss. She progressed to high-dose insulin requirements (2.2 U/kg/day) due to poor glycemic control, and additional metabolic comorbidities emerged: systemic arterial hypertension, hypothyroidism, hypertriglyceridemia, and hepatic steatosis. Physical examination revealed scarce subcutaneous adipose tissue in the limbs, muscle pseudohypertrophy, and phlebomegaly, with fat accumulation in the face and abdomen, as well as cervical acanthosis nigricans and androgenetic alopecia. Whole-body densitometry demonstrated a fat mass ratio of 1.85. Genetic testing identified a homozygous missense variant in the CIDEC gene (p.Ser62Ile), classified as of uncertain significance. Relevant family history included the father and 7 siblings with T2DM diagnosed after age 40, and the father with a similar fat distribution and established cardiovascular disease at age 63. **Discussion:** The CIDEC gene encodes a protein that regulates lipid droplet fusion in adipocytes. Its dysfunction impairs triglyceride storage in subcutaneous tissue, redirecting lipids to the liver, muscle, and visceral depots — the cardinal phenotype of familial partial lipodystrophy type 5 (FPLD5). Rubio-Cabezas *et al.* (2009) described a 19-year-old Ecuadorian woman of consanguineous parents with limb lipodystrophy, multiloculated adipocytes on biopsy, and insulin-resistant T2DM (homozygous p.E186X). Patni *et al.* (2026) reported an 18-year-old Hispanic woman with acanthosis nigricans, hirsutism, and severe insulin resistance (homozygous p.Ser75Ile). Our patient shares parental consanguinity, a lipodystrophic fat distribution, insulin-resistant T2DM, hypertriglyceridemia, and hepatic steatosis, differing only by the later onset of hyperglycemia and comorbidities. **Final comments:** This case highlights the importance of recognizing the FPLD5 phenotype even in late-onset presentations. The CIDEC p.Ser62Ile variant is rare in population databases and affects a conserved serine residue in the N-terminal region of CIDEC, close to the p.Ser75Ile variant recently implicated in FPLD5. Family segregation studies are therefore essential next steps toward variant reclassification.
Keywords: familial partial lipodystrophy; hypertriglyceridemia; insulin resistance.

DIABETES MELLITUS

73 (3375) FETAL ENDOCRINE-METABOLIC PROGRAMMING ASSOCIATED WITH GESTATIONAL DIABETES MELLITUS: NEONATAL AND LONG-TERM IMPLICATIONS

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Gestational diabetes mellitus (GDM) is one of the most common metabolic complications during pregnancy and is associated with adverse neonatal outcomes, including macrosomia and neonatal hypoglycemia. Increasing evidence suggests that intrauterine hyperglycemia may induce persistent endocrine-metabolic alterations through fetal programming and epigenetic mechanisms, contributing to future metabolic dysfunction. This study aimed to evaluate the mechanisms involved in fetal endocrine-metabolic programming associated with gestational diabetes mellitus and its neonatal and long-term metabolic implications. A systematic review was conducted using the PubMed database. Studies published between 2018 and 2026 in English involving gestational diabetes, fetal programming, neonatal metabolic outcomes, hyperinsulinism and epigenetic mechanisms were included. Observational, prospective cohort, clinical and epigenetic studies focused on the maternal-fetal dyad were considered. Editorials, narrative reviews, animal studies and studies unrelated to neonatal or metabolic outcomes were excluded. Ten studies were included in the analysis. The evidence demonstrated that intrauterine hyperglycemia contributes to persistent metabolic and epigenetic alterations associated with adverse neonatal outcomes and increased susceptibility to future metabolic dysfunction. Epigenetic mechanisms, particularly DNA methylation and altered fetal gene expression, were consistently associated with fetal metabolic programming. Increased placental glucose transfer and consequent fetal hyperinsulinism were linked to macrosomia, neonatal hypoglycemia, altered body composition and future adiposity risk. The findings suggest that metabolic alterations initiated during intrauterine life may persist from neonatal life into adulthood. In conclusion, gestational diabetes mellitus significantly influences fetal endocrine-metabolic programming through hyperglycemia-induced epigenetic and metabolic alterations, contributing to important neonatal and long-term metabolic repercussions.
Keywords: gestational diabetes; fetal programming; epigenetics.

DIABETES MELLITUS

74 (3159) FIB-4 SCORE AS A RISK MARKER FOR MICROVASCULAR COMPLICATIONS IN PATIENTS WITH TYPE 1 DIABETES MELLITUS

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Introduction: Metabolic dysfunction-associated steatotic liver disease has emerged as a systemic condition related to inflammation and insulin resistance, potentially associated with vascular outcomes. Although well described in type 2 diabetes, its role in type 1 diabetes mellitus (T1DM) and its relationship with microvascular complications remain poorly explored. The Fibrosis-4 index (FIB-4) is a widely used non-invasive marker of liver fibrosis and may reflect systemic cardiometabolic risk. **Objective:** To evaluate the association between the FIB-4 score and the presence of microvascular complications in adults with T1DM. **Methods:** An observational, retrospective, cross-sectional study based on medical record review of adults with T1DM followed at a specialized outpatient clinic. The FIB-4 score was calculated using the classical formula and categorized as < 1.3 and ≥ 1.3. Associations between FIB-4 and diabetic retinopathy, nephropathy, and neuropathy were assessed, as well as relationships with glycated hemoglobin (HbA1c) and macrovascular complications. **Results:** Of the 48 initially identified patients, 45 were included in the final analysis. Microvascular complications were present in 42.2% of the sample. The prevalence of microvascular disease was significantly higher in patients with FIB-4 ≥ 1.3 compared with those with FIB-4 < 1.3 (85.7% vs. 34.2%; $p = 0.031$). A significant association was observed between elevated FIB-4 and diabetic retinopathy ($p = 0.007$). No significant associations were found between FIB-4 and HbA1c or macrovascular complications. **Conclusion:** Higher FIB-4 values were associated with an increased prevalence of microvascular complications, particularly retinopathy, in adults with T1DM, suggesting that the FIB-4 score may serve as an additional marker of microvascular risk in this population.
Keywords: type 1 diabetes mellitus; hepatic fibrosis; diabetic complications.

DIABETES MELLITUS

75 (3481) FIRST BRAZILIAN REAL-WORLD EXPERIENCE WITH AUTOMATED INSULIN DELIVERY IN TYPE 1 DIABETES: EARLY GLYCEMIC OUTCOMES AFTER TRANSITION FROM MANUAL MODE

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Automated insulin delivery systems have demonstrated significant improvements in glycemic control in randomized clinical trials; however, real-world evidence remains essential to assess effectiveness in routine clinical practice. This study aimed to evaluate early glycemic outcomes after initiation of automated insulin delivery in people with type 1 diabetes in a real-world Brazilian setting. We conducted a retrospective observational study including individuals with type 1 diabetes with paired continuous glucose monitoring data between manual insulin delivery mode and at least one automated follow-up period. Glycemic metrics were assessed during manual mode (baseline comparator), after 30 days, and after 90 days of automated mode use. The primary outcome was change in time in range (70–180 mg/dL). Secondary outcomes included time above range (>180 mg/dL and >250 mg/dL), time below range (<70 mg/dL and <54 mg/dL), and coefficient of variation. The analytical cohort included 114 individuals with paired baseline and 30-day data; 66 also had paired 90-day follow-up. Mean time in range increased from 67.3% during manual mode to 77.2% after 30 days of automated insulin delivery, representing an absolute improvement of 9.9 percentage points ($p < 0.001$). Among the 66 individuals with 90-day follow-up, glycemic benefit was sustained, with time in range increasing from 70.2% at baseline to 77.4% (absolute improvement of 7.2 percentage points; $p < 0.001$). Time above range >250 mg/dL decreased at both follow-up assessments, while time below range < 54 mg/dL remained stable, suggesting no increase in clinically significant hypoglycemia. Glycemic variability also improved over time. In this first Brazilian real-world cohort of people with type 1 diabetes, initiation of automated insulin delivery was associated with rapid and clinically meaningful improvement in glycemic control, with sustained benefit at 90 days and no apparent increase in clinically significant hypoglycemia.

Keywords: type 1 diabetes; automated insulin delivery; insulin pump.

DIABETES MELLITUS

76 (3455) FOCAL MONONEUROPATHY OF THE DEEP FIBULAR NERVE IN A PATIENT WITH TYPE 2 DIABETES MELLITUS: A CASE REPORT

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Case presentation: L.P., 51-year-old male with metabolic syndrome and a 12-year history of type 2 diabetes mellitus (T2DM), presented with poor glycemic control, irregular use of tirzepatide and metformin, blood glucose of 141 mg/dL, and HbA1c of 8.6%. He reported sudden-onset paresthesia in the right leg 10 days earlier, progressing to foot drop, stumbling, and falls. He denied trauma, low back pain, alcoholism, hypothyroidism, or vitamin deficiencies. Physical examination revealed steppage gait, weakness of dorsiflexion and extension of the right toes, reduced thermal, superficial pain, and vibratory sensation over the dorsum of the right foot, with preserved Achilles reflex. The neuropathy impairment score of the left foot was unremarkable. Electroneuromyography demonstrated severe recent proximal axonal injury of the right deep fibular nerve, with active denervation findings compatible with acute Wallerian degeneration, without evidence of polyneuropathy or radicular involvement, consistent with severe focal mononeuropathy of the right deep fibular nerve. Magnetic resonance imaging of the right leg, requested to exclude compressive or traumatic mechanisms, demonstrated acute denervation of the tibialis anterior and extensor muscle groups, as well as segmental thickening and T2 hyperintensity of the common fibular nerve at the distal popliteal fossa, without evidence of extrinsic compression. The patient received counseling regarding lifestyle modifications and therapeutic optimization and remains under conservative multidisciplinary management with motor physiotherapy and gait training. **Discussion:** Focal mononeuropathies represent rare forms of diabetic neuropathy compared with distal symmetric polyneuropathy. Chronic hyperglycemia and longer duration of T2DM are important risk factors. The common fibular nerve is among the most frequently affected peripheral nerves in the lower limbs, typically presenting with an acute, asymmetric, and focal pattern. Although T2DM increases neural vulnerability and predisposes patients to compressive neuropathies, the pattern observed in this case suggests a predominantly ischemic/vasculitic mechanism. **Final considerations:** Early recognition of focal diabetic mononeuropathy and adequate glycemic control are essential to exclude structural compressive causes, allow timely therapeutic intervention and prevent permanent neurological deficits, thereby reducing functional impairment and improving quality of life.

Keywords: mononeuropathies; type 2 diabetes mellitus; fibular neuropathies.

DIABETES MELLITUS

77 (3049) GAD65 ANTIBODY-ASSOCIATED EPILEPSY AND TYPE 1 DIABETES MELLITUS: A CASE REPORT

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Case presentation: A 32-year-old man, diagnosed with type 1 diabetes mellitus (T1D) at the age of 18, presented with a history of chronically poor glycemic control (HbA1c ranging from 9% to 14%) and microvascular complications (diabetic kidney disease and mild non-proliferative retinopathy). At age 20, he developed focal seizures with temporal semiology characterized by epigastric rising sensation, left upper limb paresthesias, xerostomia, and automatisms, with occasional progression to bilateral tonic-clonic seizures. Serum anti-GAD antibody titer was 1,838 IU/mL (reference value < 10). Cerebrospinal fluid analysis revealed elevated gamma globulin concentrations and positive anti-GAD antibodies. Video-EEG recorded three seizures with electrographic onset in the right temporal region. Brain MRI demonstrated right hippocampal sclerosis and a lesion compatible with multinodular and vacuolating neuronal tumor (MVNT), a benign lesion located in the right superior frontal lobe. The patient evolved with drug-resistant epilepsy. **Discussion:** Anti-GAD65 antibodies are one of the main autoantibodies associated with T1D and may be detected in up to 80% of patients. Very high titers (>2,000 IU/mL), as observed in this case, have also been associated with autoimmune neurological syndromes, including epilepsy, cerebellar ataxia, and stiff-person syndrome. The pathogenesis remains incompletely understood. While GAD65 is essential for GABA synthesis, GAD antibodies target an intracellular enzyme and are unlikely to be directly pathogenic. Recent evidence suggests that neuronal damage is mediated primarily by CD8+ cytotoxic T lymphocytes, with GAD antibodies serving as markers of an underlying autoimmune process. Anti-GAD65-associated epilepsy is typically characterized by drug-resistant focal temporal seizures, as observed in this patient. It predominantly affects middle-aged women, making this case atypical because of the patient's male sex and early disease onset. The specific role of glycemic control in triggering neurological autoimmunity remains unclear. **Final comments:** This report describes a typical clinical presentation of anti-GAD65-associated autoimmune epilepsy in an atypical demographic setting (young male patient). High anti-GAD65 antibody titers, particularly when accompanied by evidence of intrathecal antibody synthesis, support the diagnosis of GAD65-associated neurological autoimmunity rather than isolated pancreatic autoimmunity.

Keywords: anti-GAD syndrome; autoimmune epilepsy; diabetes mellitus type 1.

DIABETES MELLITUS

78 (3075) GESTATIONAL WEIGHT GAIN AND ASSOCIATED FACTORS IN WOMEN WITH PREGESTATIONAL DIABETES AT A TERTIARY HOSPITAL

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Pregestational diabetes is associated with increased maternal and fetal risks, including higher rates of preeclampsia, preterm birth, and cesarean delivery. Inadequate gestational weight gain may also contribute to adverse obstetric and neonatal outcomes. This study aimed to evaluate clinical and demographic variables associated with gestational weight gain in women with diabetes according to the Institute of Medicine (IOM, 2009) recommendations. This retrospective cohort study included 101 pregnant women with type 1 diabetes (T1D) and type 2 diabetes (T2D) followed at a tertiary hospital. Data were obtained from medical records. Total gestational weight gain was calculated using self-reported pregestational weight and the last recorded weight during follow-up, provided it was obtained after 34 weeks of gestation. Weight gain was classified as below, within, or above IOM recommendations according to pregestational BMI. Clinical, demographic, and obstetric variables were evaluated, including age, family income, education, race/ethnicity, pregestational BMI, diabetes duration, gestational age at first visit, number of pregnancies, and parity. Continuous variables were expressed as mean ± standard deviation and compared using analysis of variance (ANOVA), while categorical variables were analyzed using the chi-square test. Among participants, 27 (26.7%) had adequate gestational weight gain, while 37 (36.6%) were below and 37 (36.6%) were above IOM recommendations. In the overall analysis, no significant differences were observed among groups. In the subgroup of women with T2D (n = 71), diabetes duration differed significantly among groups (p = 0.003), being longer in women with weight gain above recommendations (6.8 ± 6.5 years) compared with those below (2.5 ± 2.9 years) or within recommendations (2.5 ± 2.5 years). Pregestational BMI also differed significantly (p = 0.030), with higher values in women with weight gain below recommendations (36.4 ± 6.3 kg/m²) compared with those within (32.6 ± 6.6 kg/m²) or above recommendations (32.5 ± 4.4 kg/m²). No significant differences were observed among women with T1D. In women with T2D, longer diabetes duration and higher pregestational BMI were associated with different patterns of gestational weight gain. These findings suggest that metabolic factors and degree of obesity may influence gestational weight gain, supporting individualized follow-up strategies.

Keywords: pregestational diabetes; weight gain; obstetric outcomes.

DIABETES MELLITUS

79 (3438) GLYCEMIC CONTROL IN PATIENTS WITH TYPE 1 DIABETES DURING THE TRANSITION FROM PEDIATRIC TO ADULT CARE: A RETROSPECTIVE EVALUATION OF GLYCATED HEMOGLOBIN PROGRESSION IN A TERTIARY HOSPITAL OUTPATIENT CLINIC

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The transition from pediatric to adult care for patients with type 1 diabetes mellitus (T1DM) constitutes a period of heightened clinical vulnerability, often characterized by reduced therapeutic adherence and deteriorating glycemic control. This study evaluated the glycemic control trajectory in T1DM patients followed at a transition-specific outpatient clinic and analyzed factors associated with glycated hemoglobin (HbA1c) variation. An observational, retrospective analytical study was conducted through medical record review at a tertiary care hospital. The primary endpoint was the variation in HbA1c (ΔHbA1c) between initial and final follow-up visits. Nonparametric statistical tests were performed, considering statistical significance at $p < 0.05$. Thirty patients were evaluated. HbA1c levels increased significantly from the initial visit (8.81 ± 1.69%) to the final follow-up visit (9.40 ± 1.81%), reflecting worsening glycemic control ($p = 0.014$). Patients with missed appointments and suboptimal adherence showed a trend toward greater HbA1c increases. A significant association was found between appointment attendance and correct therapy utilization ($p = 0.032$). Episodes of severe hypoglycemia also trended toward greater HbA1c increases. No significant associations were identified regarding sex, diabetes duration, or comorbidities. In conclusion, patients demonstrated significant glycemic deterioration during the transition period. Appointment attendance and therapeutic adherence were strongly correlated, suggesting that interventions to strengthen outpatient engagement may improve glycemic outcomes during this stage of care.

Keywords: diabetes mellitus, type 1; glycemic control; health care transition.

DIABETES MELLITUS

80 (3135) GLYCEMIC RESPONSE TO A LIFESTYLE INTERVENTION ACCORDING TO BASELINE INSULIN RESISTANCE LEVELS IN ADULTS AT HIGH RISK FOR TYPE 2 DIABETES: A SUBANALYSIS OF THE PROVEN-DIA PILOT TRIAL

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Introduction: Baseline insulin resistance (IR), a key metabolic alteration in prediabetes, may affect individual responses to lifestyle-based preventive strategies. Whether baseline IR modify short-term glycemic outcomes during structured prevention programs remains unclear. **Objective:** To evaluate changes in HbA1c from baseline to 3-month follow-up according to baseline IR level among participants in the PROVEN-DIA pilot study. **Methods:** A subanalysis of the multicenter PROVEN-DIA pilot randomized controlled trial (NCT05689658; n = 220), conducted across Brazil's five macro-regions to evaluate the impact of a structured lifestyle intervention on diet quality and physical activity in individuals at high risk for T2D. Participants were stratified by baseline HOMA-IR: low IR (<2.70; PROVEN n = 47, UC n = 51) or high IR (≥2.70; PROVEN n = 52, UC n = 47). The outcome was ΔHbA1c at 3 months, assessed in participants with available data at follow-up. Between-group comparisons used the Mann-Whitney test. **Results:** We hypothesized that participants with high baseline IR would show greater glycemic responsiveness to the intervention, given the dietary improvements previously demonstrated in the PROVEN-DIA trial. Among participants with low IR (550.45 ± 9.29 years, 30.61% female), a significant between-group difference was observed for ΔHbA1c (PROVEN: +0.03 ± 0.29% *vs.* UC: -0.22 ± 0.56%; $p = 0.012$); however, this finding is likely explained by a baseline HbA1c imbalance between groups (5.43% *vs.* 5.73%), suggesting a floor effect in the intervention group rather than a true differential response. Among participants with high IR (47.21 ± 9.65 years, 25.25% female), no significant between-group difference was detected for ΔHbA1c (PROVEN: -0.23 ± 0.51% *vs.* UC: -0.17 ± 0.35%; $p = 0.967$). The hypothesis was not confirmed in either subgroup. **Conclusion:** Baseline IR level did not modulate the glyceresponse to the PROVEN-DIA intervention over three months. Despite dietary improvements previously observed in the full trial, no differential effect was detected in either IR stratum. The limited subgroup sizes and short follow-up likely constrained the detection of meaningful differences. Future adequately powered trials should prospectively examine IR as a stratification variable in lifestyle-based diabetes prevention programs.

Keywords: insulin resistance; prediabetes; lifestyle intervention.

DIABETES MELLITUS

81 (3186) GUT MICROBIOTA ALTERATIONS IN THE PRECLINICAL PHASE OF TYPE 1 DIABETES MELLITUS: A SYSTEMATIC REVIEW

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Introduction: Type 1 diabetes mellitus (T1D) is an autoimmune disease characterized by pancreatic beta-cell destruction. Gut microbiota may modulate immune responses by increasing intestinal permeability and promoting immune activation against pancreatic islets. **Objective:** To analyze the association between gut microbiota alterations and the development of islet autoimmunity during the preclinical phase of T1D. **Methods:** A systematic review was conducted according to PRISMA guidelines. PubMed was searched through 2025, identifying 183 studies, of which 5 were included after screening. Observational studies involving individuals at risk for T1D, defined by the presence of islet autoantibodies, were included. **Results:** In the TEDDY cohort (n = 903; >10,000 samples), microbiota composition varied according to breastfeeding and delivery mode, without consistent association with seroconversion. A metagenomic study from the TEDDY cohort (n = 783) identified microbial genomic signatures associated with islet autoimmunity. In the ABIS cohort (16 cases; 268 controls), children who later developed T1D showed higher abundance of *Porphyromonas*, whereas *Parasutterella* and *Eubacterium* were more frequent in controls. Another observational study (n = 88) demonstrated dysbiosis, lower microbial diversity (p = 0.005), increased intestinal permeability (+5.14; 95% CI: 1.21–9.07; p = 0.006), and association with islet autoantibodies. **Conclusion:** Gut microbiota alterations are associated with islet autoimmunity during the preclinical phase of T1D, especially reduced microbial diversity and increased intestinal permeability. Longitudinal studies suggest these alterations may precede seroconversion in susceptible individuals, although causality remains uncertain.
Keywords: gut microbiota; type 1 diabetes; islet autoimmunity.

DIABETES MELLITUS

82 (3517) HBA1C OVERESTIMATES GLYCEMIC BURDEN COMPARED TO GLUCOSE MANAGEMENT INDICATOR IN TYPE 1 DIABETES

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Introduction: Glycated hemoglobin (HbA1c) and the glucose management indicator (GMI) derived from continuous glucose monitoring (CGM) estimate glycemic control through distinct biological and mathematical mechanisms. Whether GMI can reliably substitute HbA1c in clinical decision-making for type 1 diabetes (T1D) remains uncertain. **Objective:** To evaluate the agreement between laboratory HbA1c and CGM-derived GMI at six months, and to explore the association between time in range and HbA1c in individuals with T1D. **Methods:** A 6-month longitudinal study enrolled 53 individuals with T1D from a public endocrinology clinic in Northeastern Brazil. All participants used an intermittently scanned CGM device throughout follow-up. HbA1c was measured at baseline and at six months. Agreement between HbA1c and GMI at six months was assessed using the Bland-Altman method, intraclass correlation coefficient (ICC), and Pearson correlation. The association between time in range and HbA1c was assessed by linear regression. **Results:** Mean baseline HbA1c was 9.06 ± 1.86%, diabetes duration was 11 ± 8.72 years, and GMI at six months was 7.64 ± 1.04%, remaining stable throughout follow-up (7.5%–7.6%). Agreement between HbA1c and GMI was poor: mean bias was +0.80% (HbA1c systematically higher than GMI), with proportional bias indicating greater discrepancy in participants with worse glycemic control, which is clinically relevant, as 0.8% HbA1c corresponds to approximately 18–20 mg/dL in mean glucose. Only 27% of patients showed agreement within ± 0.5%, while 32% had discrepancies above 1%. ICC was moderate (0.616), despite strong Pearson correlation (r = 0.838). Time in range and HbA1c showed strong negative correlation (r = –0.789; R^2 = 0.62), with each 10% increase in time in range corresponding to a 0.8% reduction in HbA1c. **Conclusion:** HbA1c and GMI are not interchangeable in this T1D population. In most participants, HbA1c exceeded GMI, revealing a predominantly unidirectional discrepancy whose magnitude surpassed previously reported rates. This pattern is consistent with the proportional bias observed, whereby the higher the glycemic burden, the greater the overestimation of HbA1c relative to GMI, with direct implications for therapeutic decision-making.
Keywords: type 1 diabetes mellitus; continuous glucose monitoring; reproducibility of results.

DIABETES MELLITUS

83 (2936) HEALTH-RELATED QUALITY OF LIFE IN PEOPLE WITH TYPE 2 DIABETES RECEIVING THE ONCE-DAILY, ORAL, NON-PEPTIDE GLP-1 RA ORFORGLIPRON IN ACHIEVE-2 AND -3

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Introduction: Significant improvements in HbA1c and body weight were seen with orforglipron (OFG) vs. dapagliflozin (DAPA) and oral semaglutide (SEMA) in adults with type 2 diabetes (T2D) in ACHIEVE-2 and -3 (A-2 and A-3), respectively. **Objective:** The health-related quality of life (HRQoL) of people treated with OFG vs. these oral therapies was measured in these Phase-3, randomized, open-label trials. **Methods:** Adults with T2D inadequately controlled with metformin were randomized to OFG 3, 12, or 36 mg or DAPA 10 mg QD for 40-weeks(W) (A-2) or to OFG 12 or 36 mg or SEMA 7 or 14 mg QD for 52W (A-3). HRQoL was measured at baseline and W40 (A-2) or W52 (A-3) using patient-reported outcomes (PROs). The Simplicity of Diabetes Treatment (SIM-Q) was measured at W4 and W24 in A-3. **Results:** Improvements from baseline were seen in all HRQoL measures in all treatment groups. At Week-40 (A-2), IWQOL-Lite-CT scores improved by 9.1 (95% CI: 7.0–11.1), 7.1 (5.2–9.0), and 9.1 (6.7–11.5) with OFG 3, 12, and 36 mg, respectively, and by 7.1 (5.0–9.2) with DAPA 10 mg. APPADL scores increased by 6.7 (4.2–9.1), 6.5 (4.1–8.8), 5.4 (2.8–8.0), and 5.7 (3.4–8.0), while IW-SP scores increased by 10.9 (7.8–14.0), 8.6 (5.0–12.1), 12.0 (9.1–14.9), and 8.6 (5.9–11.4), respectively. EQ VAS scores improved by 2.8 (1.0–4.5), 3.4 (1.6–5.2), 3.4 (1.6–5.2), and 3.2 (1.6–4.8), respectively (all $p < 0.05$). At Week-52 (A-3), IWQOL-Lite-CT improvements were 9.3 (7.6–11.0) with OFG 36 mg vs. 9.4 (7.7–11.1) with oral SEMA 14 mg. APPADL scores increased by 7.1 (5.2–9.1), 6.0 (4.1–7.9), 5.3 (3.1–7.5), and 5.6 (3.7–7.6); IW-SP scores by 10.8 (8.3–13.2), 10.6 (7.9–13.2), 7.4 (4.4–10.3), and 9.2 (6.9–11.4); and EQ VAS scores improved by 5.5 (4.1–6.9) with OFG 12 mg (all $p < 0.05$). In A-3, improvements were greater in IWQOL-Lite-CT total score with OFG 12 mg vs. SEMA 7 mg (10.7 [9.1–12.3] vs. 8.2 [6.6–9.8]), and in EQ VAS score with OFG 36 mg vs. SEMA 7 and 14 mg (5.8 [4.5-7.1] vs. 4.1 [2.8–5.4] and 4.0 [2.6–5.3]) (all $p < 0.05$). No other statistically significant between-group differences were seen in either study. 96% of adults receiving OFG reported it was a simple or very simple treatment for T2D in A-3 at 24W. **Conclusion:** Oral treatment with OFG, DAPA, or SEMA for 40W or 52W resulted in improved HRQoL from baseline for adults with T2D, with limited between-group differences.
Keywords: ACHIEVE; HRQoL; T2D.

DIABETES MELLITUS

84 (3452) HIGH DIABETES KNOWLEDGE BUT CRITICAL NUTRITIONAL GAP IN TYPE 1 DIABETES: BEHAVIORAL CHANGES ASSOCIATED WITH CONTINUOUS GLUCOSE MONITORING IN A LOW-INCOME POPULATION

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Introduction: In type 1 diabetes (T1D), adequate overall knowledge does not guarantee proficiency across all self-management domains, particularly nutrition. In low-income populations, this selective gap may contribute to suboptimal glycemic control despite satisfactory disease understanding. **Objective:** To evaluate diabetes-specific knowledge at baseline and self-reported behavioral changes following CGM use in a socioeconomically vulnerable T1D population. **Methods:** A 6-month longitudinal study enrolled 53 individuals with T1D from a public endocrinology clinic in Northeastern Brazil. Diabetes-specific knowledge was assessed at baseline using the validated Diabetes Knowledge Scale (DKN-A) in 46 of 53 participants, with 7 participants missing baseline assessment. At the end of follow-up, the same participants completed a specialist-developed questionnaire assessing behavioral changes, pattern recognition, and self-care practices. Structured diabetes education was provided alongside CGM throughout follow-up. **Results:** Participants were predominantly female (64.2%), self-declared Black or mixed-race (81.1%), with mean diabetes duration of 11 years and income up to 2 minimum wages (77.4%). Although 80.4% demonstrated high overall diabetes knowledge at baseline, nearly 50% failed nutritional items, particularly food group classification and substitution, revealing a selective gap within an otherwise adequate knowledge profile. Carbohydrate counting, reported by only 32.6%, was associated with higher knowledge scores ($p = 0.021$). Despite this gap, following CGM use, participants reported behavioral changes that addressed the nutritional domain: 97.8% noticed the direct effect of food on glucose levels, 95.7% identified glycemic patterns more easily, and 65.2% adopted healthier food choices. Insulin self-adjustment was facilitated in 97.8%. HbA1c decreased from 9.7% to 7.9% over six months, suggesting translation of behavioral changes into measurable glycemic outcomes. **Conclusion:** High overall diabetes knowledge coexisted with a critical nutritional gap at baseline in this vulnerable population. After CGM use, participants reported behavioral changes consistent with closing this nutritional gap, suggesting that on-demand glucose feedback may complement formal diabetes education by targeting domains where knowledge remains insufficient.
Keywords: diabetes mellitus, type 1; continuous glucose monitoring; health literacy.

DIABETES MELLITUS

85 (3268) HOSPITAL GLYCEMIC HARM IN ADULT UNITS: POCT-ADAPTED ECQM SCORES ACROSS 307,634 GLUCOSE RECORDS

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Introduction: Hospital glycemic harm reframes inpatient glucose assessment from average control toward severe dysglycemic events as patient safety signals. **Objective:** To evaluate burden, recurrence and point-of-care testing-adapted quality scores for severe glycemic harm in adult units. **Methods:** This retrospective study analyzed point-of-care glucose measurements from hospitalized adults between March 2025 and March 2026 in a tertiary academic hospital serving public and private healthcare systems. Pediatric locations, quality-control samples, duplicates and records without valid patient identification were excluded. Because medication use, diabetes diagnosis, encounter eligibility, admission/discharge timestamps, comfort-care status and hospice disposition were unavailable, official electronic clinical quality measure scores could not be fully reproduced. Severe hyperglycemic harm was defined as a patient-day with ≥1 glucose > 300 mg/dL. Severe hypoglycemic harm was defined as ≥1 glucose < 40 mg/dL, excluding values followed by repeat glucose >80 mg/dL within 5 minutes. **Results:** After exclusions, 307,634 adult-unit records from 38,635 patients and 126,876 patient-days were analyzed. Overall, 11,028 severe glycemic harm events occurred in 2,778 patients, corresponding to 7,051 harm patient-days and 35.8 events per 1,000 glucose measurements. Severe hyperglycemia accounted for 10,206 events in 2,533 patients and 6,522 patient-days. The adapted severe hyperglycemic harm score was 5.14%, below the provisional benchmarking range of approximately 8%–10%. Recurrent severe hyperglycemia occurred in 1,339 patients with ≥2 events, 573 with ≥5 and 261 with ≥10. Severe hypoglycemia < 40 mg/dL was initially identified in 1,098 measurements; after exclusion of 276 potential false-positive events, 822 valid events remained, affecting 463 patients and 603 patient-days. The adapted severe hypoglycemic harm score was 1.20%, within the provisional high-performance range of approximately < 1%–2%. Recurrent severe hypoglycemia occurred in 160 patients, and both severe hyperglycemic and severe hypoglycemic harm occurred in 218 patients. **Conclusion:** Adapted glycemic harm scores were favorable compared with provisional benchmarks, but severe dysglycemia was frequent and recurrent. These findings support digital surveillance focused on early detection, recurrence prevention and hospital quality improvement.
Keywords: hospital harm; hyperglycemia; hypoglycemia.

DIABETES MELLITUS

86 (3181) HOSPITAL MORBIDITY DUE TO DIABETES MELLITUS AMONG MIDDLE-AGED ADULTS IN BRAZIL: A SEX- AND AGE-BASED ANALYSIS

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Introduction: diabetes mellitus is one of the main causes of morbidity and healthcare burden worldwide. During midlife, aging, obesity, insulin resistance, and cardiometabolic risk accumulation contribute to progressive metabolic dysfunction, increasing the impact of diabetes mellitus on healthcare systems. **Objective:** To analyze the epidemiological profile and temporal trends of hospitalizations due to diabetes mellitus among middle-aged adults in Brazil according to sex and age group. **Methods:** An ecological time-series study was conducted using data from the Brazilian Unified Health System Hospital Information System. Hospitalizations due to diabetes mellitus between 2014 and 2025 were analyzed in individuals aged 35–64 years. Variables included sex, age group, hospital mortality, year of admission, and geographic region. **Results:** A total of 740,668 hospitalizations due to diabetes mellitus were recorded, including 406,757 males and 333,911 females. Hospitalizations increased progressively with age in both sexes, reaching the highest number among individuals aged 60–64 years (195,829 cases). Female hospitalizations increased by approximately 214% between the 35–39 and 60–64 age groups, while male hospitalizations increased by 304%, demonstrating substantial growth in hospital burden throughout midlife. Although women slightly predominated among individuals aged 35–39 years (51.1%), men became progressively more prevalent in older age groups, representing approximately 56% of hospitalizations from age 45 onward. Temporal analysis demonstrated a gradual increase in hospitalizations during the study period, with a slight reduction in 2020 followed by recovery in subsequent years. Hospital mortality also increased progressively with age, reaching 3,430 deaths among women and 3,723 among men aged 60–64 years. The Southeast region accounted for the highest number of hospitalizations (273,424 cases). **Conclusion:** Hospitalizations due to diabetes mellitus among middle-aged adults in Brazil increased substantially with aging and were associated with considerable hospital mortality. The progressive increase in hospital burden and the predominance of male hospitalizations in older age groups may reflect the growing impact of metabolic dysfunction and cardiometabolic risk accumulation during midlife, reinforcing the importance of preventive strategies and early metabolic control.
Keywords: diabetes mellitus; epidemiology; middle aged.

DIABETES MELLITUS

87 (3445) ICODEC INSULIN IN TYPE 2 DIABETES: A SYSTEMATIC REVIEW OF EFFICACY AND SAFETY

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Introduction: The management of type 2 diabetes mellitus (T2DM) aims to maintain glycated hemoglobin (HbA1c) within target levels to prevent complications. However, disease progression often requires basal insulin therapy, and daily injections may compromise adherence and glycemic control. Insulin icodec is a once-weekly ultra-long-acting basal insulin analog developed to simplify treatment. **Objective:** To evaluate the efficacy and safety of insulin icodec in T2DM compared with daily basal insulin. **Methods:** A systematic review was conducted using PubMed, Embase, and Scopus databases. The keywords "insulin icodec" and "icodec" were combined with the terms "diabetes mellitus, Type 2," "type 2 diabetes," "type 2 diabetic," and "T2DM" using AND/OR operators. Randomized controlled trials were included, yielding 40 results. After title and abstract screening, 7 studies were selected for full-text analysis. **Results:** Weekly insulin icodec improved glycemic control by increasing time in range (TIR 70–180 mg/dL) and reducing HbA1c. In a loading-dose study, higher mean TIR was observed (72.9% vs. 66.0% and 65.0% with glargine U100), with greater HbA1c reduction (7.9% to 7.1% vs. 7.4%). Another trial showed a 1.17% HbA1c reduction, TIR increase from 54.4% to 76.4%, and reduction in time above range from 45.2% to 22.9%. In the ONWARDS 1 study (n = 984), icodec achieved greater HbA1c reduction (-1.55% vs. -1.35%) and higher TIR (71.9% vs. 66.9%). In patients on basal-bolus regimens, icodec demonstrated similar HbA1c reduction (-1.15% vs. -1.19%), comparable TIR (66.9% vs. 66.4%), and lower weekly insulin dose (514U vs. 559U). Compared with degludec, it reduced HbA1c by an additional 0.2%, without differences in fasting glucose. In insulin-naïve patients, icodec increased TIR (66.1% vs. 60.7%), maintained HbA1c < 7% (72% vs. 68%), and reduced weekly insulin dose. Rates of clinically significant hypoglycemia were low and comparable to controls, with no increase in serious adverse events. **Conclusion:** Weekly insulin icodec demonstrated glycemic efficacy comparable or superior to daily basal insulins, with a favorable safety profile, reduced injection frequency, and potential to improve treatment adherence in T2DM.

Keywords: type 2 diabetes; icodec insulin; glycemic control.

DIABETES MELLITUS

88 (3157) IDENTIFICATION OF RENAL SODIUM-GLUCOSE COTRANSPORTER TYPE 2 (SGLT2) AS A NOVEL THYROID HORMONE-REGULATED GENE IN RATS: IMPLICATIONS FOR GLYCEMIC REGULATION

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Thyroid hormones (TH), particularly triiodothyronine (T3), play a key role in energy metabolism and glycemic control. Previous studies from our group demonstrated that diabetic rats treated with T3 exhibited reduced blood glucose levels, increased GLUT4 expression in skeletal muscle and adipose tissue, enhanced glycosuria, and reduced SGLT2 expression. SGLT2, together with GLUT2, is responsible for approximately 90% of glucose reabsorption from the glomerular filtrate. Given the novelty of this effect, its investigation in non-diabetic models may reveal a new mechanism by which TH regulate glucose homeostasis. Thirty adult Wistar rats were allocated into control, hypothyroid, and hyperthyroid groups. Hypothyroidism was induced by methimazole (0.03% in drinking water), whereas hyperthyroidism was induced by intraperitoneal T3 administration (1.5 µg/100 g body weight) for 15 days. Serum TSH concentrations were measured by ELISA. Renal SGLT2 and GLUT2 expression were evaluated by western blotting and immunohistochemistry. Blood glucose and glycosuria were assessed after vehicle and empagliflozin (30 mg/kg) administration. Values of $p \leq 0.04$ were considered significant. Hypothyroid animals exhibited increased TSH levels, whereas hyperthyroid animals showed reduced TSH concentrations, confirming treatment efficacy. Hyperthyroid rats presented significantly reduced renal SGLT2 and GLUT2 expression, while hypothyroid animals showed increased GLUT2 expression. T3-mediated repression of these transporters identifies them as novel molecular targets of TH and suggests a new mechanism involved in glycemic homeostasis. Despite reduced SGLT2 and GLUT2 expression, hyperthyroid animals exhibited slightly increased blood glucose levels, reversed after empagliflozin administration, indicating that SGLT2 remains functionally relevant for glycemic regulation. In the absence of empagliflozin, hypothyroid animals showed lower glycosuria than hyperthyroid animals, suggesting enhanced tubular glucose reabsorption. Reversal of this profile after empagliflozin treatment indicates that reduced glycosuria in hypothyroidism depends directly on SGLT2 activity. Furthermore, the absence of significant glycosuria changes in hyperthyroid animals despite reduced transporter expression suggests compensatory renal mechanisms preserving glucose reabsorption.

Keywords: triiodothyronine; SGLT2; glycemia.

DIABETES MELLITUS

89 (3506) IMPACT OF ADVANCED INSULIN INFUSION AND GLUCOSE MONITORING TECHNOLOGIES ON PSYCHOSOCIAL WELL-BEING IN DIABETES: A SYSTEMATIC REVIEW

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Introduction: The management of type 1 diabetes mellitus (T1DM) requires continuous monitoring and is frequently associated with emotional distress, fear of hypoglycemia, and reduced quality of life. Advanced technologies, such as automated insulin delivery systems (AIDs), hybrid closed-loop systems (HCLs), insulin pumps, and continuous glucose monitoring (CGM), have been introduced to optimize glycemic control and reduce psychosocial impacts. **Objective:** To evaluate the psychosocial impact of these technologies in individuals with T1DM, including emotional distress, therapeutic satisfaction, fear of hypoglycemia, and quality of life and sleep, compared to conventional treatment. **Methods:** A systematic review was conducted according to PRISMA guidelines in the Embase (n = 159), Scopus (n = 49), and PubMed (n = 49) databases. After removing duplicates (n = 38), 219 articles were screened by two independent reviewers, resulting in 37 included studies. Case reports, reviews, meta-analyses, studies without outcomes of interest, and publications prior to 2016 were excluded. Randomized, observational, and qualitative clinical trials involving AID/HCL, insulin pumps, and CGM were included. **Results:** The technologies demonstrated consistent benefits in psychosocial outcomes. Automated systems reduced diabetes-related emotional distress ($p < 0.001$), improved sleep quality for users and caregivers ($p = 0.005$), and decreased fear of hypoglycemia ($p = 0.02$). Insulin pump users showed lower depression scores ($p = 0.03$), greater personal satisfaction, and better subjective well-being, with an association between longer time in the glycemic target and a reduction in depressive symptoms ($p < 0.001$). Hybrid systems, such as the MiniMed 780G, maintained glycemic stability even during periods of stress ($p < 0.001$). Qualitative studies have reported a greater sense of freedom, reduced mental load, and improved quality of life, although limitations persist related to frequent alarms, physical discomfort, technical failures, and unrealistic expectations regarding complete automation. **Conclusion:** These technologies are associated with improved quality of life and sleep, greater therapeutic satisfaction, and reduced emotional distress and fear of hypoglycemia. However, the heterogeneity among technologies, populations, and psychometric instruments limits definitive conclusions, reinforcing the need for methodological standardization and continuous evaluation of the user experience.

Keywords: diabetes mellitus, type 1; insulin infusion systems; quality of life.

DIABETES MELLITUS

90 (3362) IMPACT OF CONTINUOUS GLUCOSE MONITORING (CGM) ON GLYCEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS UNDER NON-INSULIN THERAPIES: A SYSTEMATIC REVIEW

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Introduction: Continuous glucose monitoring (CGM) has become consolidated as an important tool in the management of insulin-treated type 2 diabetes mellitus (T2DM). The application of CGM in patients with T2DM under exclusive non-insulin therapies gains growing prominence as an instrument for glycemic and behavioral biofeedback. Furthermore, its use combats clinical inertia and favors more precise clinical adjustments in the management of T2DM. **Objective:** To evaluate the efficacy of CGM compared to self-monitoring of blood glucose (SMBG) or usual care in reducing glycated hemoglobin (HbA1c) and improving time in range (TIR) in non-insulinized adults with T2DM. **Methods:** Systematic review conducted according to PRISMA guidelines and structured by the PICO strategy. The search in PubMed and Scopus databases utilized MeSH/DeCS descriptors related to T2DM, glycemic control, and CGM, combined by AND/OR. Studies published between 2021 and 2026, in English and Portuguese, were included. Randomized clinical trials and prospective controlled observational studies in humans were considered. The initial search identified 387 studies; after applying criteria and removing duplicates, 362 were screened. Analysis by two independent reviewers resulted in the final inclusion of 16 studies. **Results:** The analysis of the studies demonstrates that CGM is superior to SMBG or other conventional care. The decrease in HbA1c in the sensor groups ranged from –0.3% to –1.6%, and even 24 weeks after discontinuing CGM use, the biofeedback resulted in sustained glycemic improvement (–0.46%). TIR increased from 46%–61% to 72%–87%, especially when associated with carbohydrate restriction. In clinical practice, data were optimized in 87% of patients. This pattern facilitated the early utilization of cardiorenal drugs (GLP-1 prescription jumping from 25% to 56%) and the attenuation of high-risk secretagogues use. Additionally, a weight loss superior to the control group (–2.17 kg) and an increase in self-care were reported. **Conclusion:** In non-insulinized T2DM, CGM acts as a continuous behavioral catalyst for dietary adherence and a precision guide for optimizing modern pharmacotherapy. In this context, incorporating this technology shows promise in promoting relevant and sustained reductions in HbA1c, overcoming clinical inertia and indicating a strong trend toward changes in treatment guidelines.

Keywords: glycemic control; diabetes mellitus; continuous glucose monitoring.

DIABETES MELLITUS

91 (3587) IMPACT OF DIABETES MELLITUS ON SEVERE SARS-COV-2 CASES IN THE AMAZON: CORRELATION BETWEEN INFLAMMATORY PROFILE AND CLINICAL OUTCOME IN THE ICU

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Introduction: In 2019, a novel β-coronavirus was identified in Wuhan, China, and designated as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which triggers a severe respiratory syndrome characterized by hypercytokinemia and an intense inflammatory response. Several host-related factors have been associated with clinical severity, including pre-existing immunity, age, and chronic comorbidities. **Objective:** To characterize the impact of diabetes mellitus in patients with severe SARS-CoV-2 admitted to the Intensive Care Unit (ICU) in an Amazonian state capital, correlating these findings with inflammatory markers and clinical outcomes. **Materials and methods:** This cross-sectional study was conducted in 2021 and 2022 with severe patients divided into two groups: with and without diabetes. Data collection included clinical history, blood samples for immunophenotyping and ELISA (IL-6, IL-10, sCD25, sCD163, and granzyme B), C-reactive protein levels, and oxygen saturation (SpO_2). The study was approved by the Research Ethics Committee (Opinion: 5,516,176; CAAE: 48608221.0.0000.0011). The significance level was set at 0.05. **Results:** Between October 1, 2021, and March 9, 2022, 73 patients were recruited. There was a predominance of males, individuals in their sixth decade of life, of mixed-race (pardo) ethnicity, sedentary lifestyle, urban residents, and those undergoing antibiotic therapy. The primary vaccine administered was Butantan/Sinovac. Correlational analyses of cytokines (IL-6, IL-10), sCD25, sCD163, granzyme B, the mean number of days from symptom onset to death, and SpO2 levels showed no statistically significant differences between the groups. **Conclusion:** In conclusion, contrary to global literature, diabetes mellitus in severe SARS-CoV-2 patients in the Amazon region did not show a direct linear correlation with immunological markers or mortality. This epidemiological and immunometabolic divergence suggests that population-specific intrinsic factors — such as unique genetic admixture, historical exposure to regional endemic diseases, or delayed intensive care access — may modulate the inflammatory response differently from European or North American cohorts. Although the small sample size and single-center design limit immediate generalizability, these results highlight the critical need for multicenter investigations in the northern region.
Keywords: SARS-CoV-2; diabetes; hyperglycemia.

DIABETES MELLITUS

92 (3524) IMPACT OF THE COVID-19 PANDEMIC ON HOSPITALIZATIONS AND HOSPITAL MORTALITY FROM DIABETES MELLITUS IN BRAZIL: A TEMPORAL ANALYSIS FROM 2018 TO 2025

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Introduction: diabetes mellitus is one of the leading chronic non-communicable diseases worldwide and is associated with cardiovascular, renal, neurological, and metabolic complications. During the COVID-19 pandemic, individuals with diabetes presented a higher risk of hospitalization and death, in addition to difficulties accessing healthcare services, compromising clinical follow-up and metabolic control. **Objective:** To analyze the impact of the COVID-19 pandemic on hospitalizations and hospital mortality related to complications of diabetes mellitus in Brazil between 2018 and 2025. **Methods:** This ecological, retrospective, and descriptive study was conducted using data from the Brazilian Unified Health System Hospital Information System (SIH/SUS), available through Datasus/Tabnet. Hospitalizations for complications of diabetes mellitus (ICD-10 E10–E14) between 2018 and 2025 were analyzed. The evaluated variables included number of hospitalizations, hospital deaths, and hospital mortality rate according to year, geographic region, and age group. The period was divided into pre-pandemic (2018–2019), pandemic (2020–2021), and post-pandemic (2022–2025). **Results:** Between 2018 and 2025, 1,090,753 hospitalizations for complications of diabetes mellitus were recorded in Brazil. There was a reduction in hospitalizations during the pandemic, decreasing from 136,276 cases in 2019 to 124,646 in 2020 (–8.5%), followed by progressive recovery through 2025. The Southeast region accounted for the highest absolute number of cases. A total of 44,413 hospital deaths were recorded during the study period, with a peak in 2021 (6,046 deaths). The hospital mortality rate increased from 4.22% in 2019 to 4.72% in 2021, decreasing to 3.68% in 2025. Older adults showed the highest mortality rates, especially individuals aged 80 years or older, whose mortality rate reached 12.01% in 2021. **Conclusion:** The COVID-19 pandemic was associated with reduced hospitalizations and increased hospital mortality related to complications of diabetes mellitus in Brazil, suggesting greater clinical severity among hospitalized patients and the impact of difficulties in accessing healthcare services. These findings reinforce the need to strengthen clinical follow-up and metabolic control, especially among older adults and vulnerable populations.
Keywords: diabetes mellitus; COVID-19; hospital mortality.

DIABETES MELLITUS

93 (3457) IMPACTS OF ARTIFICIAL INTELLIGENCE ON DIABETES MELLITUS MANAGEMENT AND MEDICAL CLINICAL AUTONOMY: A SYSTEMATIC REVIEW

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Introduction: diabetes mellitus requires continuous monitoring and frequent therapeutic decisions. Artificial intelligence has increasingly been incorporated into diabetes management through predictive algorithms, automated insulin delivery, continuous glucose monitoring, and clinical decision-support platforms. These technologies may improve glycemic control and therapeutic precision; however, concerns remain about reduced medical clinical autonomy and individualized decision-making. **Objective:** To evaluate whether artificial intelligence in diabetes mellitus management is associated with reduced medical clinical autonomy compared with conventional management. **Methods:** A PICO-based systematic review was conducted in PubMed, SciELO, Embase, and Cochrane Library using "diabetes mellitus," "Artificial Intelligence," "Clinical Decision Support Systems," "Medical Autonomy," and "Machine Learning." Studies from 2018 to 2026 in English, Portuguese, and Spanish were included. Clinical trials, observational studies, systematic reviews, and bioethical analyses on artificial intelligence-assisted diabetes management were analyzed. Studies without clinical or ethical impact evaluation were excluded. **Results:** Twenty-two studies involving artificial intelligence systems for type 1 and type 2 diabetes management were analyzed. Technologies included hypoglycemia prediction algorithms, automated insulin administration, and machine learning-based decision-support platforms. Most studies showed improved glycemic control, with reduced glycated hemoglobin, lower glycemic variability, and fewer hypoglycemic episodes versus conventional management ($p < 0.05$). However, qualitative studies identified greater physician dependence on automated recommendations, especially in complex scenarios. Reduced participation in individualized decisions and passive validation of algorithmic suggestions were reported. Despite these concerns, most studies emphasized that artificial intelligence acts mainly as a complementary tool and does not replace medical evaluation, particularly in psychosocial and subjective situations. **Conclusion:** Artificial intelligence in diabetes management promotes clinical benefits and therapeutic precision. However, excessive dependence on automated systems may partially reduce medical clinical autonomy. Current evidence supports it as a complementary resource rather than a substitute for clinical reasoning, reinforcing professional supervision and individualized care.

Keywords: diabetes mellitus; artificial intelligence; medical autonomy.

DIABETES MELLITUS

94 (3143) INSULIN INDEPENDENCE AND GLYCEMIC CONTROL OUTCOMES AFTER TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION: A SYSTEMATIC REVIEW

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Introduction: Pancreatogenic diabetes represents a significant complication following pancreatectomy due to the loss of endocrine pancreatic tissue. Autologous islet transplantation (IAT) has emerged as a therapeutic strategy to prevent or mitigate postoperative diabetes by preserving beta-cell function. The procedure consists of intraportal autologous islet transplantation. **Methods:** A systematic review was conducted following the PRISMA guidelines. The search was performed in the PubMed database, with a publication restriction covering the last 20 years (2006–2026). The descriptors used included "Pancreatectomy," "Total Pancreatectomy," "TPIAT," "Islet Transplantation," "Islet Autotransplantation," "Pancreatic Islet Transplantation," "islet autograft," "diabetes mellitus," "diabetes," "insulin dependence," "endocrine insufficiency," and "glycemic control," combined using the Boolean operators "OR" and "AND". **Results:** During the search strategy, 264 articles were identified in the PubMed database. After the initial screening and full-text review, 17 studies were included, comprising a total of 800 patients, of whom 57% were male and 43% female, who underwent pancreatectomy with IAT. Follow-up duration ranged from 12 to 204 months. The weighted mean age of patients was 37.5 years, and the mean preoperative HbA1c was 5.9% among the studies reporting this variable. At the end of follow-up after the procedure, 67% (493/800) of patients became insulin-dependent. **Discussion and conclusion:** The findings of this systematic review demonstrate that insulin dependence remains a significant complication following pancreatectomy, with substantial metabolic impact on patients, even when IAT is performed. The 66% incidence of insulin dependence observed in this review was considerably higher than the prevalence reported by Scholten et al. (2018), which demonstrated a prevalence of 16%. One possible explanation may be the restriction of publication years and the less rigorous diagnostic criteria used in earlier studies. The compared meta-analysis included studies published between 1993 and 2017, thereby reducing the number of studies shared by both analyses. Furthermore, important markers such as HbA1c were only incorporated more consistently after 2010, excluding a large proportion of studies published before that year. These findings reinforce the need for technical optimization to improve long-term metabolic outcomes.

Keywords: pancreatectomy; islet transplantation; diabetes mellitus.

DIABETES MELLITUS

95 (2963) INVESTIGATION OF OBESITY, CARDIOVASCULAR RISK, AND LIVER FIBROSIS ASSESSMENT THROUGH ELASTOGRAPHY IN TYPE 1 DIABETES PATIENTS

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Introduction: Type 1 diabetes (T1D) has increasingly been associated with obesity and insulin resistance, a phenomenon known as "double diabetes," which contributes to metabolic syndrome (MetS) and increased cardiovascular risk (CVR). Metabolic dysfunction-associated fatty liver disease (MAFLD/NAFLD) is highly prevalent in type 2 diabetes (T2D), but its prevalence and clinical impact in T1D remain underexplored. **Objective:** This study aimed to investigate the prevalence of obesity, cardiovascular risk (CVR), and liver fibrosis in Type 1 Diabetes (T1D) patients using hepatic elastography. Given the rising incidence of obesity in T1D, the study also explored the relationship between Metabolic Syndrome (MetS) and Non-Alcoholic Fatty Liver Disease (NAFLD). **Methods:** First, 50 patients with T1D and 50 with T2D were compared regarding clinical, anthropometric, and laboratory characteristics, as well as MetS and CVR. In the second stage, 39 overweight or obese T1D patients (BMI ≥25 kg/m²) underwent abdominal ultrasonography and hepatic elastography. MetS was defined according to modified IDF criteria using estimated glucose disposal rate (eGDR) as a marker of insulin resistance in T1D. Cardiovascular risk was assessed using the Steno T1 Risk Engine and UKPDS Risk Engine. Statistical significance was established at $p < 0.05$ (total of 139 participants). **Results:** MetS prevalence was significantly lower in T1D compared with T2D patients (44% vs. 98%; $p < 0.001$). The 5-year CVR was similar between the groups, but the 10-year CVR was significantly higher in T2D patients (12% vs. 6%, $p < 0.01$). T1D patients with MetS showed higher CVR, poorer glycemic control, and more microvascular complications. NAFLD was diagnosed in 64.1% of T1D patients, with 8% exhibiting significant liver fibrosis. Liver stiffness correlated positively with BMI ($r = 0.496$, $p = 0.026$). Within the T1D group, MetS was associated with older age, longer disease duration, worse glycemic control, hypertension, and higher prevalence of microvascular complications. **Conclusion:** Patients with T1D showed a high prevalence of MetS and NAFLD, particularly in the presence of overweight and obesity. MetS in T1D was associated with substantially increased cardiovascular risk, similar to that observed in T2D. Although advanced fibrosis was uncommon, these findings reinforce the importance of early screening for NAFLD and metabolic abnormalities in T1D patients to reduce long-term cardiovascular and liver-related complications.

Keywords: type 1 diabetes; obesity; hepatic elastography.

DIABETES MELLITUS

96 (3364) LATE-ONSET DIABETES MELLITUS IN A PATIENT WITH KABUKI SYNDROME: A CASE REPORT

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Case report: A 22-year-old male, born to non-consanguineous parents, with a previous diagnosis of Kabuki syndrome, was admitted to the emergency department due to intractable vomiting and severe abdominal pain after a periodontal infection that started five days earlier. On examination, he was alert, tachypneic, short-statured (149 cm), weighing 44 kg, and had typical facial features including elongated palpebral fissures, lower eyelid eversion, short eyebrows, dental abnormalities, protruding ears, and a broad neck. Pectus excavatum, thoracic kyphosis, and a sacral dimple were also present. He was born small for gestational age and had neonatal hypoglycemia and enterorrhagia requiring intensive care. During childhood, he developed neuropsychomotor delay, seizures, and severe bilateral sensorineural hearing loss. On admission, laboratory tests showed leukocytosis, capillary glucose "HI," arterial pH 7.15, bicarbonate 11 mEq/L, and positive ketonuria. He was diagnosed with moderate diabetic ketoacidosis and treated with intravenous hydration, continuous insulin infusion, and antibiotics for periodontitis. He improved clinically but required ongoing insulin therapy after discharge. During follow-up, anti-GAD antibodies were positive, confirming type 1 diabetes mellitus. Further evaluation revealed horseshoe kidney, hydronephrosis, and ureterolithiasis. He also developed recurrent infections, including herpes zoster, parotitis with orchitis, and urinary tract infection, as well as sporadic hypoglycemic episodes due to marked glycemic variability. **Discussion:** Kabuki syndrome is a rare genetic disorder characterized by facial dysmorphism, developmental delay, skeletal anomalies, and immune-endocrine dysfunction. Neonatal hypoglycemia due to hyperinsulinism is frequently described, as seen in this patient. However, some individuals later develop autoimmune diseases such as autoimmune thyroiditis, vitiligo, and type 1 diabetes mellitus. Diabetic ketoacidosis as the initial manifestation of type 1 diabetes in Kabuki syndrome is uncommon. The presence of anti-GAD antibodies supports an autoimmune mechanism. **Conclusion:** This case highlights the broad clinical spectrum of Kabuki syndrome and emphasizes the importance of long-term follow-up due to the increased risk of autoimmune endocrinopathies, particularly type 1 diabetes mellitus and thyroid disease.

Keywords: Kabuki syndrome; type 1 diabetes; diabetic ketoacidosis.

DIABETES MELLITUS

97 (3511) LIPODYSTROPHIC PHENOTYPE AS A MARKER OF EXTREME INSULIN RESISTANCE AND ACCELERATED ATHEROSCLEROSIS: CASE REPORT

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Case presentation: A 66-year-old woman was diagnosed with type 2 diabetes mellitus (T2DM) at 55 years of age and has since exhibited persistently poor glycemic control. Over time, she developed multiple microvascular and macrovascular complications, including iliofemoral peripheral arterial disease treated with angioplasty and stent placement in 2016, and acute myocardial infarction in 2022, with diffuse coronary artery disease and a 75% stenosis of the distal left main coronary artery, managed with percutaneous coronary intervention. She also presented progressive decline in renal function (estimated creatinine clearance 31 mL/min), central obesity (BMI 38 kg/m²), moderate hepatic steatosis, dyslipidemia, and non-proliferative diabetic retinopathy. Despite high-dose insulin therapy (2.1 IU/kg/day), glycated hemoglobin remained elevated at 10.7%, indicating severe insulin resistance. The severity of her metabolic derangements and accelerated atherosclerosis appeared disproportionate to a clinical picture initially attributed to T2DM and common obesity. Physical examination revealed marked abdominal adiposity with reduced subcutaneous fat in the gluteal region and lower limbs, with a calf skinfold thickness of 11 mm and a Köbberling index of 7.75. Due to clinical suspicion of partial lipodystrophy, genetic testing was performed in 2025, with no pathogenic variants identified, supporting a familial partial lipodystrophy type 1 (FPLD1)/Köbberling-like phenotype. **Discussion:** FPLD1 is a rare and likely underdiagnosed disorder with an incompletely understood genetic basis. Diagnosis relies mainly on clinical, phenotypic, and anthropometric features, and a Köbberling index > 3.477 may support recognition. It is characterized by gluteofemoral and peripheral fat loss associated with truncal adiposity and ectopic fat deposition, contributing to extreme insulin resistance and severe metabolic complications. Recognition of this phenotype on physical examination may guide diagnostic investigation, complemented by body composition assessment using dual-energy X-ray absorptiometry or bioelectrical impedance analysis. **Final remarks:** Partial lipodystrophy syndromes are often recognized only after advanced metabolic complications develop. In this case, the body phenotype prompted diagnostic reconsideration beyond T2DM and obesity, highlighting the importance of physical examination and early identification of patients at high cardiometabolic risk.

Keywords: diabetes mellitus; lipodystrophy; obesity.

DIABETES MELLITUS

98 (3198) LONGITUDINAL FOLLOW-UP OF AN ADOLESCENT CARRIER OF A PATHOGENIC HNF4A VARIANT PRIOR TO THE DEVELOPMENT OF MODY DIABETES

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Case presentation: Female, 14 years old, with a family history of MODY diabetes affecting her maternal great-grandfather, maternal grandmother, and mother, carrying the heterozygous HNF4A (NM_175914.4) c.48C>G:p.(Tyr16*) variant, pathogenic. She had a history of prematurity and severe neonatal hypoglycemia. At the initial evaluation, she was asymptomatic, with normal fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) levels. After counseling regarding potential risks, she underwent genetic testing, which confirmed the same familial HNF4A mutation. She received guidance regarding healthy lifestyle habits and the importance of clinical surveillance, as she carried a diabetes-associated mutation without clinical manifestation of the disease at that time. Approximately one year later, she returned presenting with polydipsia, polyuria, and polyphagia, with FPG of 200 mg/dL and HbA1c of 7.2%, leading to the diagnosis of HNF4A-MODY and initiation of sulfonylurea therapy. **Discussion:** MODY accounts for approximately 1%–5% of diabetes mellitus cases and remains frequently underdiagnosed because of its clinical overlap with type 1 and type 2 diabetes. The HNF4A-related subtype is characterized by a progressive defect in insulin secretion and may be associated with transient hyperinsulinemic neonatal hypoglycemia, often preceding diabetes onset by several years. This case reinforces such an association by demonstrating severe neonatal hypoglycemia followed by progression to diabetes during adolescence. The presence of a compatible multigenerational family history was essential for diagnostic suspicion and early genetic testing. Identification of the pathogenic variant before the onset of hyperglycemia enabled targeted follow-up and timely diagnosis. Furthermore, recognition of HNF4A-MODY has important therapeutic implications, as these patients typically exhibit marked sensitivity to sulfonylureas, frequently avoiding the need for initial insulin therapy. Therefore, molecular diagnosis contributes to precision medicine, reduction of inappropriate treatments, and appropriate family genetic counseling. **Final comments:** HNF4A-MODY should be considered in young patients with an autosomal dominant family history of diabetes and a history of neonatal hypoglycemia. Early genetic screening enabled clinical surveillance and timely diagnosis in this patient, reinforcing the relevance of molecular investigation for therapeutic individualization and longitudinal follow-up.

Keywords: diabetes; MODY; genetic monitoring.

DIABETES MELLITUS

99 (3197) MAURIAC SYNDROME: A RARE COMPLICATION IN AN ADOLESCENT WITH TYPE 1 DIABETES MELLITUS

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Case presentation: M.B.V., a 17-year-old male, diagnosed with type 1 diabetes mellitus at 3 years of age, with a history of poor therapeutic adherence and recurrent episodes of diabetic ketoacidosis. He was referred to the adult endocrinology clinic at 16 years of age. On presentation, he exhibited growth impairment (height Z-score –3.44 SD, weight Z-score –4.5 SD, and BMI Z-score –3.7 SD), delayed puberty (Tanner stage G2P1), and homogeneous hepatomegaly identified on abdominal ultrasound. Laboratory evaluation revealed: HbA1c 13.2%, IGF-1 65 ng/mL, GH 1.74 ng/mL, total testosterone 21 ng/dL, LH < 0.07 U/L, and FSH < 0.3 U/L. Insulin therapy was optimized and combined with testosterone replacement therapy and anastrozole. After one year of follow-up, the patient experienced no new episodes of diabetic ketoacidosis, progressed to Tanner stage G4P2, and demonstrated improved glycemic control (HbA1c 9.2%), although without catch-up growth due to delayed diagnosis. **Discussion:** Mauriac syndrome is a rare complication of poorly controlled type 1 diabetes mellitus, characterized by growth retardation, delayed puberty, and hepatomegaly. It is frequently associated with high insulin requirements and recurrent episodes of diabetic ketoacidosis. Chronic hyperglycemia leads to hepatic accumulation of fat and glycogen, resulting in hepatomegaly. Furthermore, portal hypoinsulinization reduces hepatic growth hormone receptor expression, decreasing IGF-1 production and contributing to growth impairment. The diagnosis is primarily based on clinical and laboratory findings, supported by the classic triad of poorly controlled type 1 diabetes mellitus (typically HbA1c > 9.5%), hepatomegaly (a universal finding on physical examination), and growth retardation (median height Z-score of –1.01). Treatment primarily consists of strict glycemic control, aiming to reverse hepatomegaly and improve growth and pubertal development. There is no evidence supporting the use of exogenous growth hormone therapy, as the underlying mechanism is growth hormone resistance rather than growth hormone deficiency. **Final comments:** Early recognition of Mauriac syndrome is essential to enable timely intervention and minimize metabolic complications, growth impairment, and psychosocial consequences associated with poor glycemic control.
Keywords: type 1 diabetes mellitus; Mauriac syndrome; hepatomegaly.

DIABETES MELLITUS

100 (2919) METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE IN A POPULATION WITH HIGH PREVALENCE OF DIABETES

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Introduction: Metabolic dysfunction–associated steatotic liver disease (MASLD) is now the leading cause of chronic liver disease worldwide. Its pathophysiology is strongly linked to visceral obesity and insulin resistance, both central mechanisms in type 2 diabetes mellitus (T2DM) and promote lipid accumulation in hepatocytes. Although liver biopsy remains the diagnostic gold standard, its limited feasibility highlights the need for accessible screening tools. In this context, anthropometric indices represent practical alternatives for identifying individuals at metabolic risk, particularly those with diabetes, who constitute a high-risk group for MASLD. **Objective:** To evaluate the association between indicators of general and central adiposity and the presence of MASLD, with emphasis on a population with high prevalence of T2DM. **Methods:** Analytical, observational study including 137 adults followed at an endocrinology outpatient clinic. Clinical, laboratory, and anthropometric data were collected (body mass index (BMI), waist circumference (WC), neck circumference (NC), hip circumference (HC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR). Hepatic steatosis was diagnosed by abdominal ultrasonography using the Saadeh classification. Analyses were performed in R software, with $p < 0.05$. **Results:** The sample had a median age of 62 years and was predominantly female (83.2%). A markedly elevated burden of cardiometabolic disease was observed, especially type 2 diabetes mellitus, present in 73.7% of participants, underscoring the high-risk profile of the cohort. The prevalence of hepatic steatosis was 85.4%, and metabolic syndrome was identified in 87.6% of individuals with steatosis. Compared with those without steatosis, patients with steatosis exhibited significantly higher BMI (32.7 vs. 28.7 kg/m²; $p = 0.002$), WC (106.6 vs. 94.2 cm; $p = 0.0019$), WHtR (0.67 vs. 0.60; $p = 0.001$), and HC (109.4 vs. 101.4 cm; $p = 0.01$). No associations were found for NC or WHR. Physical inactivity was also more frequent among individuals with steatosis (73.3% vs. 47.4%; $p = 0.045$). **Conclusion:** In a sample with a high prevalence of T2DM, simple anthropometric indices — particularly WC and WHtR — were strongly associated with MASLD. These findings reinforce the clinical utility of these measures for early identification of metabolic and hepatic risk in diabetic populations, supporting their incorporation into routine screening and management strategies.
Keywords: metabolic dysfunction-associated steatotic liver D; anthropometry; visceral obesity.

DIABETES MELLITUS

101 (3532) MOBILE HEALTH APPLICATIONS FOR LIFESTYLE ADHERENCE IN TYPE 2 DIABETES AND OBESITY: A SYSTEMATIC REVIEW OF RANDOMIZED CONTROLLED TRIALS

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Introduction: Lifestyle modification is a cornerstone of type 2 diabetes and obesity management, but long-term adherence remains persistently poor. Mobile health applications represent a scalable approach to support dietary and physical activity behavior change in high-burden metabolic conditions. **Objective:** To evaluate the effectiveness of mobile health applications on adherence to lifestyle modification, including dietary habits, physical activity, and self-monitoring behaviors, in adults with type 2 diabetes or obesity. **Methods:** A systematic search was conducted in PubMed using the terms "mobile app," "lifestyle," "adherence," combined with "obesity" or "type 2 diabetes." Inclusion criteria were randomized controlled trials with adults diagnosed with type 2 diabetes or obesity in which a mobile application was the primary intervention. Five trials totaling 1,411 participants were included, with follow-up periods ranging from 12 to 52 weeks. **Results:** Four of five trials demonstrated significant short-term benefits favoring app-based interventions. An electronic medical record-integrated app with personalized physician feedback produced a 1.04% reduction in glycated hemoglobin versus 0.49% with usual care at 12 weeks. A behavior-change app for adults with obesity and hypertension achieved significantly greater weight loss than controls (–1.1 kg versus –0.4 kg at 12 weeks). A multicomponent intervention combining a smartphone app with an activity wristband produced a net weight difference of –0.76 kg at 3 months. An artificial intelligence-powered conversational agent resolved 88.86% of user queries and was associated with self-reported improvements in medication and dietary adherence. One 52-week app-supported interval walking trial found no significant change in objectively measured moderate-to-vigorous physical activity, though physical health-related quality of life improved significantly in the intervention group. **Conclusion:** Mobile health applications demonstrate meaningful short-term benefits for lifestyle adherence and metabolic outcomes in type 2 diabetes and obesity. Long-term effectiveness is limited by declining engagement after intervention withdrawal. Personalized feedback and physician integration appear to be key determinants of sustained benefit.
Keywords: mobile health; lifestyle adherence; type 2 diabetes.

DIABETES MELLITUS

102 (3319) NEUROPROTECTIVE EFFECTS OF METFORMIN BEYOND GLYCEMIC CONTROL IN TYPE 2 DIABETES MELLITUS: A SYSTEMATIC REVIEW

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Introduction: Type 2 diabetes mellitus (T2DM) is associated with increased risk of cerebrovascular disease, cognitive decline, and poor neurological outcomes. Beyond its antihyperglycemic effects, metformin has been investigated for potential neuroprotective properties, although evidence regarding its cerebral effects remains dispersed in the literature. **Objective:** To evaluate the available evidence regarding the effects of metformin on cerebral and cerebrovascular outcomes, including ischemic and hemorrhagic stroke, neurological recovery, and cognitive decline in patients with T2DM or prediabetes. **Methods:** A systematic review was conducted according to PRISMA 2020 recommendations. A structured search was performed in PubMed, Scopus, Web of Science, Cochrane Library, and BVS using descriptors related to metformin, diabetes, stroke, and neuroprotection. Randomized clinical trials and observational studies evaluating cerebral or cerebrovascular outcomes associated with metformin use were included according to predefined eligibility criteria. Reviews, editorials, case reports, and preclinical studies were excluded. Two independent reviewers performed study selection and data extraction using Rayyan software. Methodological quality assessment was conducted according to study design, and results were synthesized through qualitative analysis. **Results:** Nineteen studies published between 2011 and 2025 were included, most conducted in Asian countries, particularly China, Taiwan, Japan, and South Korea. Overall, findings suggested an association between metformin use and reduced ischemic stroke risk compared with other therapies or absence of metformin treatment, particularly in individuals with better glycemic control. In acute stroke settings, metformin use was associated with lower neurological severity, assessed by NIHSS scores, better functional outcomes, and reduced mortality. Studies involving intracerebral hemorrhage suggested smaller hematoma volumes and improved prognosis. Additionally, some evidence suggested cognitive benefits, although these findings remain insufficiently consolidated in the literature. **Conclusion:** Current evidence suggests that metformin may exert neuroprotective effects beyond glycemic control in patients with T2DM. However, further prospective studies are needed to better clarify its long-term neurological implications and improve understanding of the molecular mechanisms involved in these effects.
Keywords: metformin; diabetes; neuroprotection.

DIABETES MELLITUS

103 (3141) NUMBER NEEDED TO TREAT TO PREVENT INCIDENT TYPE 2 DIABETES BASED ON SURMOUNT-1 176 WEEK DATA

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Introduction: This post-hoc analysis of SURMOUNT-1 estimated the number needed to treat (NNT) to prevent one case of type 2 diabetes (T2D) in participants with obesity or overweight and prediabetes treated with pooled tirzepatide (TZP) groups versus placebo over 176-weeks, accounting for patients lost to follow-up. **Methods:** Glycemic status monitored through glycated hemoglobin and fasting serum glucose levels every 12-13 weeks, alongside a 2-hour oral glucose tolerance test at weeks 72, 124, and 176. The NNT was calculated as the inverse of the probability of not developing T2D at 176-weeks in the TZP groups minus the same probability in the placebo group. The 95% confidence interval (CI) for the NNT was calculated (Altman and Andersen). The survival probability of not receiving a diagnosis of T2D was estimated by Cox proportional hazard regression. **Results:** At 176-weeks, the probability of not developing T2D was 81.82% and 98.45% for placebo and pooled TZP groups. Accounting for loss to follow-up, the NNT was calculated as treating 7 (95% CI: 5–9) participants with TZP rather than placebo prevents one T2D case. **Conclusion:** Using a time-to-event approach that accounts for patient loss to follow-up, 7-patients with obesity or overweight and prediabetes required TZP-treatment to prevent one T2D case over 176-weeks. This post-hoc estimate supplements the NNT of 9 from the prespecified analysis, suggesting that NNT of TZP for T2D prevention may range from 7-9. Previously submitted to Obesity Week 2025.
Keywords: tirzepatide; type 2 diabetes prevention; number needed to treat.

DIABETES MELLITUS

104 (3072) OZONE THERAPY FOR THE TREATMENT OF DIABETIC FOOT ULCERS: A SYSTEMATIC REVIEW

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Introduction: Diabetic foot is one of the most severe complications of diabetes mellitus, associated with high morbidity, risk of amputations, and increased healthcare costs. Ozone therapy has been investigated as a potential adjunctive treatment because of its antimicrobial, anti-inflammatory, and tissue regeneration-stimulating effects. However, its effectiveness in diabetic foot ulcers (DFUs) remains controversial. **Objective:** To evaluate the effectiveness of ozone therapy in DFUs regarding wound healing, reduction of ulcer area, and decrease in complications. **Methods:** A systematic review was conducted according to PRISMA and Cochrane guidelines, with the protocol registered in PROSPERO (CRD42024595484). Randomized clinical trials comparing ozone therapy, associated or not with conventional treatment, versus conventional therapy in patients with DFUs were included. Searches were performed in December 2025 in MEDLINE/PubMed, Scopus, ScienceDirect, Web of Science, BDTD, and OATD, without language or time restrictions. Study selection and data extraction were conducted by two independent reviewers. Risk of bias was assessed using the RoB 2 tool, and methodological quality was evaluated with the Jadad scale. **Results:** A total of 240 studies were identified, and seven clinical trials involving 525 patients were included. Considerable heterogeneity was observed among ozone therapy protocols, conventional treatments, and assessed outcomes. Overall, the studies showed a favorable trend toward ozone therapy in accelerating wound healing, reducing ulcer area, shortening hospitalization time, and decreasing amputation rates. Some studies also reported improvements in inflammatory markers, oxidative stress, angiogenesis, and microbiological control. However, most studies presented methodological limitations, including lack of blinding, insufficient description of randomization, and high risk of bias. Due to study heterogeneity, meta-analysis was not feasible. **Conclusion:** Ozone therapy shows potential benefits as an adjunctive treatment for DFUs, especially in wound healing and complication reduction. Nevertheless, the low methodological quality and heterogeneity of the studies limit the strength of the evidence. Further randomized clinical trials with greater methodological rigor and standardized protocols are needed to define its role in clinical practice.
Keywords: diabetic foot; ozone therapy; wound healing.

DIABETES MELLITUS

105 (3584) PROFILE OF DIGITAL HEALTH LITERACY AMONG PATIENTS PARTICIPATING IN THE GOIÁS UNITED AGAINST DIABETES CAMPAIGN

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Diabetes mellitus represents one of the greatest public health challenges due to its high prevalence, treatment complexity, and the ongoing need for health education and contact with healthcare teams. Digital solutions have great potential in this context; however, it is necessary to understand the pattern of technology use among people with diabetes mellitus in order to propose targeted interventions, which are still poorly described in the literature. Cross-sectional study involving people with DM who were treated during a retinopathy screening campaign conducted by the Goiás State Department of Health in November 2025. A qualitative data collection instrument addressing preferences for the use of digital tools to search for health information was used, administered through face-to-face interviews and recorded in Google Forms. The study was approved by the Research Ethics Committee of HC-UFG (No. 40722720.6.0000.5078). Eighty-eight people with DM responded — 68 with DM2, 4 with DM1, and 16 were unable to provide this information. Of the total, 42 were women, and the mean age was 60 years (min. 21, max. 89). Seventy-four people used a smartphone to access the internet. 48.9% (43/88) searched for health information online and 51.1% (45/88) did not. Therefore, when the remaining questions were directed only to the 43 people who searched for health information online, the following frequency of use was observed: WhatsApp 83.7% (36), Instagram 30.2% (13), and Google 23.3% (10). When asked where they searched for this health information, 69.8% cited Google (30), 23.3% YouTube (10), and 34.9% Instagram (15). Regarding impact, 67.4% reported that it helped them (29), and 16.3% reported that it encouraged them to seek a healthcare professional (7). Regarding fake news, 62.8% (27) had received it, and 76.7% (33) had never shared news based solely on its title. Regarding reliability, 46.5% (20) trusted information when it came from a healthcare professional or a consulted source; 22.3% (8/36) based their assessment on the source of the news; and 30.5% (11) did not verify the reliability of the information. Access to digital media among this population with diabetes mellitus remains limited, although the internet is an important source of health information, particularly Google, YouTube, and WhatsApp. Exposure to misinformation and weaknesses in assessing reliability were observed, reflecting low health literacy. Therefore, it is essential to strategically use these findings to develop more accessible and engaging interventions, expanding the reach and engagement of this population in the treatment of the disease.

Keywords: community health campaign, digital health literacy.

DIABETES MELLITUS

106 (3336) PHARMACOKINETICS OF GLP-1 AGONISTS IN LIVER CIRRHOSIS: ORAL THERAPY IMPLICATIONS

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Introduction: GLP-1 receptor agonists (GLP-1RA) have expanded their therapeutic application in type 2 diabetes mellitus (T2DM), obesity, and metabolic dysfunction-associated steatotic liver disease (MASLD). However, the increasing use of GLP-1RA introduces a challenge: the intrinsic delay in gastric emptying caused by these agents may alter the absorption of critical concomitant medications used in liver cirrhosis. **Objective:** To analyze scientific evidence regarding the impact of GLP-1 agonists in patients with liver cirrhosis and discuss clinical implications for supportive oral therapies in different stages of hepatic insufficiency. **Methods:** An integrative literature review was conducted using the PubMed and SciELO databases, including publications from 2018 to 2025. Clinical trials, systematic reviews, meta-analyses, and pharmacokinetic studies involving mainly semaglutide and tirzepatide in patients with hepatic insufficiency, MASLD, and compensated cirrhosis were selected. The studies evaluated pharmacokinetic parameters such as AUC, Cmax, and Tmax, in addition to outcomes related to therapeutic safety and efficacy. **Results:** The studies demonstrated that GLP-1 agonists, such as semaglutide and tirzepatide, maintain similar systemic exposure and Cmax in patients with different degrees of hepatic dysfunction classified by Child-Pugh and in control individuals, with preserved half-life and a favorable safety profile, without the need for dose adjustment. However, delayed gastric emptying induced by GLP-1RA may prolong Tmax and alter the bioavailability of concomitantly administered oral medications, including therapies used in the management of portal hypertension and cirrhosis complications. In addition, recent studies demonstrated metabolic and hepatoprotective benefits associated with GLP-1RA, including reduction of hepatic steatosis and fibrosis, MASLD resolution, significant weight loss, and improvement in inflammatory markers, without a relevant increase in severe adverse events. **Conclusion:** GLP-1RA present an apparently safe pharmacokinetic profile across different degrees of hepatic insufficiency, but they may modify the absorption of concomitant oral therapies. Therefore, individualized clinical follow-up and active pharmacovigilance are essential to ensure safety, therapeutic efficacy, and appropriate metabolic-hepatic management in these patients.

Keywords: GLP-1 receptor agonists; liver cirrhosis; pharmacokinetics.

DIABETES MELLITUS

107 (3580) PHARMACOLOGICAL TREATMENT WITH HIGH-POTENCY INCRETINS VERSUS METABOLIC SURGERY FOR THE REMISSION OF TYPE 2 DIABETES IN OBESE PATIENTS: A SYSTEMATIC REVIEW

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Introduction: The coexistence of obesity and type 2 diabetes mellitus (T2DM), termed "diabesity", elevates cardiovascular and microvascular risks, with rising prevalence in Brazil. Metabolic surgery provides sustained remission, whereas high-potency incretins, including GLP-1 receptor agonists and dual GIP/GLP-1 agonists, emerge as a pharmacological alternative with substantial glycemic and weight efficacy. **Objective:** To systematically compare high-potency incretins versus metabolic surgery regarding T2DM remission (HbA1c < 6.5% without antihyperglycemic therapy for ≥ 3 months) in adults with BMI ≥ 30 kg/m². **Methods:** This systematic review followed PRISMA guidelines and was registered in PROSPERO (CRD420261369465). RCTs and prospective cohorts published since 2014 were searched in PubMed, Cochrane, and Embase, screened via Rayyan by two independent reviewers. Exclusions comprised case reports, cross-sectional designs, animal models, retrospective studies, obsolete techniques, and follow-up under 12 months. Bias was assessed with RoB-2 (RCTs) and Newcastle-Ottawa Scale (NOS, cohorts); evidence certainty via GRADE. **Results:** Four articles were included (three cohorts and one RCT), totaling 5,592 patients. Mingrone *et al.* (2012, RCT) demonstrated superior HbA1c reductions with surgery (6.35 ± 1.42% *vs.* 7.69 ± 0.57%). Agareva *et al.* (2025) reported greater effects on circulating cytokines and the adipocyte secretome in the surgical group (29.3 ± 3.4 *vs.* 33.1 ± 4.2). Gasoyan *et al.* (2025) showed surgery reduced MACE risk (HR 0.65; 95% CI: 0.51–0.82), all-cause mortality, and microvascular outcomes versus GLP-1RA. Mervin *et al.* (2024) found higher cumulative T2DM remission at ten-year follow-up with surgery (42.3 ± 8.83 vs. 50.7 ± 7.87). Cohorts exhibited high quality (NOS 7–8); the RCT presented some concerns on RoB-2. **Conclusion:** Findings support surgical superiority in durable T2DM remission and cardiometabolic outcomes. Incretins act as chronic modulators dependent on continuous use, with relapse risk upon discontinuation. Management must be individualized, with incretins ideal for high surgical risk patients. Limitations include heterogeneity across study designs and scarcity of long-term head-to-head trials. Personalized approaches and cost-effectiveness analyses remain fundamental for practice.

Keywords: diabetes mellitus type 2; obesity; bariatric surgery; incretins; semaglutide.

DIABETES MELLITUS

108 (2943) PILOT EVALUATION OF GLUCOSE ACCURACY AND SENSOR UNDER METAMIZOL (DIPYRONE) STUDY PEGASUS

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Introduction: Continuous glucose monitoring (CGM) systems can be affected by drug related analytical interferences. Paracetamol has been previously assessed as a potential interferent in some continuous glucose monitoring platforms, whereas, to our knowledge, there were no published clinical studies evaluating dipyrone (metamizole) interference with other CGM sensors. **Objective:** To evaluate whether dipyrone at the maximum usual dose worsens the accuracy of the iCan i3 CGM compared with a paracetamol control regimen in adults with diabetes. **Methods:** Open-label randomized pilot trial. Adults with type 1 or type 2 diabetes were assigned to dipyrone 4 g per day (1 g every 6 hours) for 14 days or to a control group using paracetamol as needed during the same period. Capillary glucose and iCan i3 CGM sensor glucose were recorded at matched time points. Accuracy was quantified by the absolute relative difference for each paired measurement and by participant-level mean absolute relative difference. Between-group comparisons used Welch's t test and Wilcoxon rank-sum test. A linear mixed-effects model with random intercept for participant assessed absolute relative difference using all paired observations. **Results:** Eighteen participants contributed paired data (dipyrone n = 8; paracetamol control n = 10), totalling 481 matched pairs (206 dipyrone; 275 control). Mean participant level MARD was 22.4% (standard deviation 14.0) in the dipyrone group and 33.8% (14.0) in the paracetamol control group (difference -11.4 percentage points; Welch p = 0.107; Wilcoxon p = 0.122), indicating a lower error in dipyrone group. In the mixed-effects model, dipyrone was associated with a -10.9 percentage point change in absolute relative difference compared with paracetamol control (95% confidence interval -24.0 to 2.3). Two participants in the dipyrone group reported nonspecific malaise; no serious adverse events occurred. **Conclusion:** In this randomized pilot study, dipyrone at the maximum usual dose showed no signal of clinically relevant degradation in iCan i3 CGM accuracy compared with controls, suggesting it may be used for pain and fever management without compromising CGM performance. Results are limited by small sample size, variability inherent to capillary blood glucose reference measurements, and baseline imbalances; larger studies with standardized reference methods are warranted.

Keywords: dipyrone; paracetamol; continuous glucose monitoring system.

DIABETES MELLITUS

109 (3263) PREDICTORS OF POSTPARTUM ORAL GLUCOSE TOLERANCE TEST COMPLETION AMONG WOMEN WITH GESTATIONAL DIABETES MELLITUS FOLLOWED AT A TERTIARY CARE HOSPITAL

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Gestational diabetes mellitus (GDM) is one of the most common metabolic complications of pregnancy. Women with prior GDM are at substantially increased risk for future glucose intolerance and type 2 diabetes mellitus, making postpartum metabolic surveillance through the oral glucose tolerance test (OGTT) essential. Despite its clinical importance, adherence to postpartum screening remains low. To evaluate factors associated with postpartum follow-up adherence and OGTT completion among women diagnosed with GDM at a tertiary care hospital. This retrospective cross-sectional study reviewed medical records of women with GDM followed between 2024 and 2026. Sociodemographic, clinical, and obstetric variables were collected. Participants were categorized according to postpartum OGTT completion or non-completion. Continuous variables were expressed as mean ± standard deviation or median (interquartile range [IQR]) and compared using Student's t-test or Mann-Whitney test, as appropriate. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test. Multivariate logistic regression was performed to identify independent predictors of postpartum OGTT completion, with statistical significance defined as $p < 0.05$. A total of 197 women with GDM were included. Mean age was 31.5 ± 5.9 years, and mean pregestational BMI was 40.8 ± 12.1 kg/m². Only 21 women (10.7%) completed postpartum OGTT. Compared with non-adherent women, those who completed the test had: Lower pregestational BMI (35.8 ± 11.8 vs. 41.4 ± 12.0 kg/m²; $p = 0.047$); Fewer prior live births [0 (IQR: 0–1) vs. 1 (IQR: 0–2); $p = 0.042$]; Greater number of prenatal visits [8 (IQR: 5.5–9) vs. 4 (IQR: 3–6); $p < 0.001$]. In multivariate logistic regression, only the number of endocrinology prenatal visits remained independently associated with postpartum OGTT completion. Each additional endocrinology visit increased the likelihood of postpartum test completion by approximately 41% (OR = 1.415; 95% CI: 1.205–1.663; $p < 0.001$). Postpartum OGTT adherence was markedly low in this population. Increased prenatal endocrinology follow-up was the only independent predictor of postpartum screening completion, highlighting the importance of structured strategies to strengthen long-term metabolic surveillance in women with prior GDM.

Keywords: gestational diabetes mellitus; pregnancy complications; oral glucose tolerance test.

DIABETES MELLITUS

110 (3279) PREGNANCY AND DIABETES: CLINICAL CHARACTERISTICS, DIAGNOSTIC PROFILES, AND CHALLENGES IN SPECIALIZED CARE

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Introduction: diabetes mellitus (DM) during pregnancy is a complex metabolic condition associated with unfavorable maternal and fetal outcomes. Therefore, characterizing the clinical and metabolic profile of pregnant women in a real-world setting is essential to understanding and addressing the associated healthcare challenges. **Objective:** To characterize the clinical and obstetric profile of pregnant women with DM followed at a tertiary care center. **Methods:** Cross-sectional study including baseline data collected from 2024 to 2026 from an ongoing prospective cohort. All pregnant women aged ≥18 years with a diagnosis of gestational (GDM) or pregestational DM who were followed at a tertiary care center were invited to participate. Those who agreed to participate by signing the informed consent form were included. Data were collected using a structured questionnaire. **Results:** Fifty-eight pregnant women were included, aged 33.1 ± 6.0 years; mean pregestational BMI 31 ± 8.0 kg/m². 6 (10.3%) had type 2 DM; 49 (84.5%) were diagnosed with GDM and 3 (5.2%) with overt diabetes in pregnancy (OD). Mean gestational age at the first appointment was 24.8 ± 7.7 weeks. Among the 33 participants with a previous pregnancy, 4 (12.1%) had a history of GDM. Previous adverse obstetric outcomes were reported such as stillbirth in 5 (15.2%), preterm birth in 2 (6.1%), congenital malformations in 2 (6.1%), and fetal macrosomia in 1 (3.0%). Thirty-six (63.2%) reported an unplanned pregnancy; 19 (33.9%) used folic acid during the preconception period. Among the 6 women with type 2 DM, 83.3% did not plan the pregnancy. In the group of 52 women with GDM or OD, the diagnosis was performed before 24 weeks of gestation in 29 (55.8%), 24 based on fasting plasma glucose (98 mg/dL [IQR 95–101]. Among the 21 women diagnosed with DM based on OGTT, the altered values occurred as follows: 5 (23.8%) just basal glycemia, 2 (9.5%) just 1h glycemia; 6 (28.6%) 1h and 2h glycemia, 4 (19%) just 2h glycemia, 2 (9.5%) basal and 1h glycemia and 2 (9.5%) basal and 2h glycemia. **Conclusion:** A high frequency of unplanned pregnancies and previous adverse obstetric outcomes was observed, highlighting the importance of reproductive counseling and preconception care. Despite a high-risk profile, specialized follow-up was initiated late in pregnancy, underscoring the need for earlier referral and management. Heterogeneity in diagnosis timing and OGTT profiles warrants further investigation.

Keywords: diabetes in pregnancy; diagnostic profile; maternal-fetal health.

DIABETES MELLITUS

111 (3018) PREVALENCE OF METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE AND LIVER FIBROSIS IN A HIGH METABOLIC RISK POPULATION TREATED AT A BRAZILIAN TERTIARY CENTER

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Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most frequent cause of chronic liver disease, affecting approximately 38% of the world's population and roughly 44% in Latin America. In the general population with MASLD, the prevalence of significant liver fibrosis is between 11% and 12%. Brazilian data on high-risk populations are still scarce. **Objective:** To evaluate the frequency of MASLD and liver fibrosis in a population with risk factors treated at a tertiary center, with a subanalysis of the type 2 diabetes mellitus (T2DM) subgroup. **Methods:** This was an observational study with prospective data collection, approved by the local ethics committee. Participants over 18 years of age with at least one risk factor for MASLD, like T2DM, obesity, systemic arterial hypertension (SAH), or metabolic syndrome (MS, IDF criteria) were included. The diagnosis of MASLD was based on the presence of steatosis and/or liver fibrosis identified by ultrasonography and transient elastography. Significant liver fibrosis was defined by liver stiffness greater than 8 kPa on elastography. **Results:** A total of 176 participants were included, with a median age of 60.5 (52.0–66.0) years. The identified risk factors were obesity in 109 (61.9%), T2DM in 132 (75%), SAH in 146 (82.9%), and MS in 147 (82.7%). MASLD was observed in 144 participants (81.8%) and liver fibrosis in 45 (25.5%). Among the 132 participants with T2DM, 109 (82.5%) presented with MASLD and 36 (27.0%) with liver fibrosis. **Conclusion:** In a population with multiple risk factors treated at a tertiary center in Brazil, a MASLD frequency of 81.8% was observed, with no relevant difference in the T2DM subgroup (82.5%). The frequency of liver fibrosis (25.5%) was approximately double that described in global literature, reinforcing the importance of active screening for MASLD and fibrosis in this population

Keywords: liver fibrosis; type 2 diabetes mellitus; hepatic steatosis.

DIABETES MELLITUS

112 (3537) PREVALENCE OF TYPE 2 DIABETES MELLITUS AMONG OBESE MEN WITH AND WITHOUT MALE OBESITY SECONDARY HYPOGONADISM

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Introduction: Excess body fat in men is associated with male obesity secondary hypogonadism (MOSH), characterized by reduced testosterone levels resulting from dysfunction of the hypothalamic-pituitary-gonadal axis. Hypogonadism may contribute to increased visceral adiposity, chronic inflammation, and insulin resistance, leading to complications such as type 2 diabetes mellitus (T2DM). **Objective:** To evaluate the prevalence of MOSH in obese men, as well as the prevalence of T2DM in men with and without MOSH. **Methods:** This was an observational, cross-sectional, retrospective study based on medical record analysis of obese men followed at an endocrinology outpatient clinic between 2015 and 2023. The following variables were collected: body mass index (BMI), total testosterone (TT), luteinizing hormone (LH), and follicle-stimulating hormone (FSH). Patients were divided into two groups: obese men with MOSH (group 1) and obese men without MOSH (group 2). MOSH was defined as TT levels below 350 ng/dL. The prevalence of T2DM in both groups was assessed using glycated hemoglobin (HbA1c) 6.5%. Welch's t-test was performed to compare BMI and TT between groups. Statistical significance was considered at $p < 0.05$. Patients with hypergonadotropic hypogonadism were excluded, and none presented hyperprolactinemia. **Results:** Eighty-three patients were eligible for analysis, of whom 58 (70%) had MOSH and 25 (30%) did not. The prevalence of T2DM was 42% in group 1 and 25% in group 2. In group 1, the mean ± standard deviation values for age, BMI, TT, LH, and FSH were 43.3 ± 10.1 years, 49.8 ± 10.2 kg/m², 215 ± 73.9 ng/dL, 4.4 ± 2.1 mIU/mL, and 4.4 ± 2.3 mIU/mL, respectively. In group 2, the corresponding values were 46.2 ± 10.5 years, 41.4 ± 5.5 kg/m², 486.7 ± 113.4 ng/dL, 4.7 ± 2.6 mIU/mL, and 4.4 ± 2.1 mIU/mL, respectively. Differences in BMI and TT between groups were statistically significant ($p < 0.01$ and $p < 0.0001$, respectively). **Conclusion:** The results demonstrate a high prevalence of MOSH (70%) in the studied population, suggesting a strong association between severe obesity and male hypogonadism. MOSH prevalence was higher among individuals with greater BMI. A higher frequency of T2DM was also observed in the MOSH group (42% vs. 25%), reinforcing the relationship between obesity, glucose metabolism dysfunction, and reduced testosterone levels.

Keywords: obesity; male hypogonadism; type 2 diabetes.

DIABETES MELLITUS

113 (3306) PREVALENCE OF VITAMIN B12 DEFICIENCY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS USING METFORMIN AT A PHILANTHROPIC HOSPITAL

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Introduction: Metformin is effective in reducing hyperglycemia in type 2 diabetes mellitus, and it is inexpensive and safe. However, its prolonged use is associated with a significant prevalence of vitamin B12 deficiency due to interference with its absorption in the distal ileum, affecting between 10% and 30% of patients. This interference is associated with alterations in the calcium-dependent transport of cobalamin bound to intrinsic factor. Consequently, hematological and neurological complications may occur, highlighting the importance of regular monitoring of vitamin B12 levels. **Objective:** Determinate the prevalence of vitamin B12 deficiency in patients with T2DM using metformin. **Methods:** A retrospective study analyzing medical records of patients from the Endocrinology outpatient clinic between July 2023 and April 2026, including clinical and laboratory data such as serum vitamin B12 levels. Vitamin B12 deficiency was defined as serum vitamin B12 levels < 200 pg/mL, while borderline levels were defined as serum levels between 200 pg/mL and 300 pg/mL. Normality of the daily metformin dose variable was assessed using the Kolmogorov-Smirnov test, resulting in $p < 0.05$. Data were tabulated in Excel spreadsheets and analyzed using IBM SPSS Statistics version 29. **Results:** A total of 509 patients were analyzed, of whom 80.3% were female. The mean age was 64 ± 10.2 years. Overall, 12.2% (62) presented vitamin B12 deficiency and 23.4% (119) had borderline values. The mean duration of metformin treatment was 17 ± 10 years in the deficiency group and 14 ± 9 years in the borderline group, whereas in the non-deficiency group it was 12 ± 7 years. The deficiency and borderline groups were associated with higher metformin doses compared to the non-deficiency group, although only the correlation between the borderline group and the non-deficiency group reached statistical significance ($p = 0.012$). The median metformin doses were 1850 mg in the deficiency group, 2000 mg in the borderline group, and 1500 mg in the non-deficiency group. **Conclusion:** The prevalence of vitamin B12 deficiency in patients with T2DM using metformin was 12.2%, which is consistent with the literature. Therefore, we emphasize the importance of monitoring vitamin B12 levels in order to avoid the risk of complications, especially neurological and hematological ones.

Keywords: diabetes mellitus; metformin; vitamin B 12 deficiency.

DIABETES MELLITUS

114 (3091) QUALITY OF LIFE OF PATIENTS WITH TYPE 1 DIABETES MELLITUS USING INSULIN PUMP THERAPY

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Introduction: Type 1 diabetes mellitus (T1DM) is an autoimmune disease characterized by destruction of pancreatic beta cells and endogenous insulin deficiency. T1DM negatively impacts quality of life (QoL), with physical, emotional, and social repercussions. New therapeutic modalities, such as continuous subcutaneous insulin infusion (CSII), have promoted improvements in glycemic control and QoL. **Objective:** To evaluate quality of life in CSII users followed at a specialized outpatient clinic. **Methods:** A cross-sectional and observational study with CSII users undergoing ambulatory follow-up. Patients who missed consultations twice consecutively during the first semester of 2025 were excluded. Three quality of life assessment questionnaires were applied. The Short Form Health Survey (SF-36) evaluates functional capacity, physical aspects, pain, general health status, vitality, social aspects, emotional aspects, mental health, and compares current health perception with that of one year earlier. The Diabetes Treatment Satisfaction Questionnaire change (DTSQ-c) evaluates treatment satisfaction and perception of hypo- and hyperglycemia (scale: –18 to +18); the more positive the score, the more satisfied the patient. The Problem Area In Diabetes (PAID) evaluates negative emotional aspects related to living with diabetes; ranges from 0 to 100 and identifies emotional problems related to living with T1DM. Sociodemographic, clinical, and laboratory data were collected. **Results:** Seven patients were included, 6 women and 1 man, with a mean age of 24.2 years. Mean duration of T1DM treatment was 13.8 years and mean duration of CSII use was 5.5 years. The most used device was the Accu Check Spirit Combo. All participants declared themselves satisfied. Mean HbA1c was 6.6%, mean time in range was 58.4%, mean glycemic variability was 45.4%, and mean percentage of time in hypoglycemia was 4.4%. Regarding the SF-36, the mean score was 92.8 in the functional capacity domain. In the DTSQ-c treatment satisfaction showed a mean score of +16.7. The PAID obtained a mean score of 20.1. **Conclusion:** T1DM negatively impacts patients lives. Studies demonstrate that CSII use provides benefits in QoL. In this study, a high degree of satisfaction, high quality of life scores, and low impact of living with the disease were observed among CSII users.

Keywords: type 1 diabetes mellitus; continuous subcutaneous insulin infusion; quality of life.

DIABETES MELLITUS

115 (3534) RACIAL AND ETHNIC INEQUALITIES IN HOSPITAL MORTALITY FROM DIABETES MELLITUS IN BRAZIL BEFORE AND AFTER THE COVID-19 PANDEMIC: A TEMPORAL ANALYSIS FROM 2018 TO 2025

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Introduction: Complications of diabetes mellitus are one of the leading causes of hospitalization and mortality worldwide. In Brazil, the disease has high epidemiological relevance and a significant impact on the public health system. Social and racial inequalities influence diabetes-related outcomes, especially after the COVID-19 pandemic. However, national studies according to race/color remain limited. **Objective:** To analyze racial inequalities in hospital mortality from diabetes mellitus in Brazil before, during, and after the COVID-19 pandemic, from 2018 to 2025. **Methods:** This is an ecological, retrospective, and quantitative study conducted using secondary data from the Brazilian Unified Health System Hospital Information System (SIH/SUS), from 2018 to 2025. Hospitalizations related to diabetes mellitus (CID-10) were included. The analyzed variables included number of hospitalizations, hospital deaths, hospital mortality rate, race/color. Temporal analysis data were divided into pre-pandemic (2018–2019), pandemic (2020–2021), and post-pandemic (2022–2025) periods. **Results:** From 2018 to 2025, 1,090,753 hospitalizations and 45,541 hospital deaths related to diabetes mellitus were recorded in Brazil. The black population accounted for the highest absolute number of hospitalizations (539,149) and deaths (21,782). The national average hospital mortality rate was 4.18 deaths per 100 hospitalizations. The Black population presented the highest mortality rates in all analyzed periods pre, during and post-pandemic: 4.69, 5.80 and 4.61, respectively. During the pandemic, hospital mortality had a 23.7% increase among the Black population, higher than that observed in the overall population (11.1%). The Indigenous population showed the highest proportional increase in mortality during the pandemic (38.6%), although with low absolute numbers. After 2021, hospital mortality rates progressively decreased; however, the Black population maintained the highest indicators throughout the entire study period. **Conclusion:** The study demonstrated significant racial inequalities in hospital mortality from complications of diabetes mellitus in Brazil. The Black population presented the highest mortality rates in all analyzed periods, while the Indigenous population showed the most proportional increase during the COVID-19 pandemic. The findings reinforce the need to strengthen public programs for historically vulnerable populations.
Keywords: diabetes mellitus; hospital mortality; racial groups.

DIABETES MELLITUS

116 (3353) RELATIONSHIP OF HOMA-IR AND HOMA-β INDICES WITH METABOLIC AND NUTRITIONAL CHANGES IN INDIVIDUALS WITH DIABETES MELLITUS

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Introduction: diabetes mellitus (DM), a metabolic disorder associated with glycidic and lipid changes, is related to increased cardiometabolic risk. In this context, the HOMA-IR and HOMA-β indices stand out as markers of insulin resistance and pancreatic beta cell function, respectively. **Objective:** To evaluate the correlation of HOMA-IR and HOMA-β indices with metabolic and nutritional parameters in individuals with DM. **Methods:** Cross-sectional study with adults of both sexes with DM, approved by the Research Ethics Committee (CAAE 76125323.3.0000.5133). Pregnant women and individuals with incomplete exams were excluded. Anthropometric assessment and laboratory data collection from medical records were performed. Statistical analyses were conducted in SPSS, including normality test, descriptive analyses, Mann-Whitney test, and Spearman correlation, considering significance of ≤0.05. **Results:** A total of 55 individuals with diabetes mellitus (DM) were included, median age 45 years (36.5–53.5), predominantly female (67.3%), non-White individuals (70.9%), non-smokers (89.1%), and non-alcohol (54.5%). Mean body mass index (BMI) was 31.63 ± 7.2 kg/m², with overweight in 76.4%, elevated waist circumference (WC) in 63.6%, and metabolic syndrome in 60%. HOMA-IR showed positive correlation with BMI, WC, waist-to-hip ratio (WHR), fasting glucose, insulin, glycated hemoglobin (HbA1c), HOMA-β, triglycerides, and TyG index, and negative correlation with HDL-c ($p \leq 0.05$). HOMA-β correlated positively with BMI and insulin, and negatively with fasting glucose, total cholesterol, and LDL-c ($p \leq 0.05$). In the comparison between the groups, individuals with higher HOMA-IR had higher values of CC, WHR, glycemia, insulin, HbA1c, triglycerides, HOMA-β and TyG index, in addition to lower concentrations of HDL-c ($p \leq 0.05$). Individuals with higher HOMA-β had lower blood glucose values and higher insulin concentrations ($p \leq 0.05$). **Conclusion:** HOMA-IR and HOMA-β indices showed significant correlations with anthropometric, glycemic, and lipid parameters, highlighting the relationship between insulin resistance, pancreatic beta-cell function, and metabolic alterations in individuals with DM. Higher HOMA-IR was associated with worse cardiometabolic profile, whereas higher HOMA-β suggested compensatory beta-cell activity in response to glycemic alterations.
Keywords: diabetes mellitus; insulin resistance; biomarkers.

DIABETES MELLITUS

117 (3538) REVISITING THE PERFORMANCE OF MEASUREMENT OF ALBUMIN CONCENTRATION IN RANDOM URINE SAMPLES FOR SCREENING OF DIABETIC KIDNEY DISEASE

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Introduction: Urinary albumin-to-creatinine ratio (UACR) is recommended for the screening of chronic kidney disease (CKD), as it minimizes the risk of false-negative and false-positive from urinary albumin concentration (UAC) results caused by variations in hydration status. However, urinary creatinine reflects muscle mass, being a limitation in sarcopenic obesity and in those undergoing weight loss. Furthermore, there is no evidence of better performance of UACR compared with UAC. **Objective:** Assess the performance of UAC in random urine for screening of CKD in a cohort of patients with T2DM. **Methods:** A total of 378 patients (197W, age: 23–82 years) were followed for 6.0 ± 3.0 years. At baseline, in the standardized clinical/laboratorial evaluation, they were oriented to collect both random urine samples to measure UAC and 24-h urine for measurement of urinary albumin excretion rate (UAER). The same urine collections were repeated in the end of follow up. CKD was defined as the presence of GFR < 60 mL/min/1.73 m² and/or 24-h UAER ≥ 30 mg. The performance of UAC for screening of increased albuminuria was assessed using the baseline 24-h UAER as criterion standard. After, both baseline UAC and 24-h UAER were analyzed as predictive variables for the development of CKD. The receiver operating characteristics (ROC) curve approach was used. **Results:** At baseline, based on 24-h UAER, 263 patients presented normal albuminuria and 115 increased albuminuria. The calculated area (±SE) under the ROC curves to screen increased albuminuria for UAC was 0.981 ± 0.011 (P < 0.001), using baseline 24-h UAER as criterion standard. The selected cut off point based on the Youden's index was 17.4 mg/L (sensitivity: 97.4%; specificity: 97%). When baseline 24-h UAER and UAC were evaluated as predictive factors for increased albuminuria at the end of follow up, the areas under the ROC curves were, respectively, 0.846 ± 0.024 (P < 0.001) and 0.835 ± 0.024 (P < 0.001), with no difference between them (P = 0.61). To predict CKD in the end of follow up, the areas under the ROC curves were, respectively, 0.788 ± 0.025 (P < 0.001) and 0.777 ± 0.025 (P < 0.001), for 24-h UAER and UAC, with no difference between them (P = 0.56). **Conclusion:** These results reinforce the good performance of UAC in random urine for screening of CKD. As well, the similar performance between baseline UAC and 24-h UAER to predict the development of CKD, might support the adoption of this measurement in the screening of CKD.

Keywords: type 2 diabetes; diabetes kidney disease; urinary albumin concentration.

DIABETES MELLITUS

118 (3081) RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF POST-TRANSPLANT DIABETES MELLITUS IN LIVER TRANSPLANT RECIPIENTS

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Introduction: Post-transplant diabetes mellitus (PTDM) is a frequent metabolic complication after liver transplantation, associated with increased morbidity and mortality and poorer cardiovascular prognosis. Its development is multifactorial, involving recipient and donor characteristics, as well as procedure and immunosuppression-related factors. Early identification of risk factors may support prevention strategies and individualized management. **Objective:** To identify risk factors associated with PTDM in liver transplant recipients. **Methods:** Retrospective observational study including liver transplant recipients from 2015 to 2025. Patients aged ≥18 years, without previous diabetes mellitus and with available clinical and laboratory data in electronic medical records were included. Recipient, donor, and post-transplant follow-up variables were analyzed. The outcome was PTDM. Continuous variables were assessed for normality using the Shapiro-Wilk test and compared using Student's t-test or Mann-Whitney test, according to distribution. Categorical variables were compared using the chi-square test. A p-value < 0.05 was considered significant. **Results:** Fifty recipients were evaluated, of whom 9 (18%) developed PTDM. Patients with PTDM had higher glucose levels 1 month after transplantation [median (IQR): 112.5 (85.75–148.75) vs. 85 (72–94) mg/dL; p = 0.040]. At 3 months, PTDM was associated with higher glucose [170 (132–217) vs. 83 (76.25–94) mg/dL; p < 0.001], glycated hemoglobin [7.1 (6.75–13.48) vs. 5.5 (4.85–5.75); p = 0.002], and triglycerides [299.5 (212–387) vs. 116 (72.75–130.25) mg/dL; p = 0.044]. At 6 months, the PTDM group also had higher glucose [130.5 (87.25–165.25) vs. 86 (78.5–95) mg/dL; p = 0.013] and alanine aminotransferase [22.5 (21–71) vs. 15.5 (11–23.75) U/L; p = 0.036]. The triglyceride/HDL ratio was higher in the PTDM group at 1 month (mean ± SD: 8.23 ± 1.20 vs. 2.15 ± 1.31; p < 0.001) and at 3 months [7.61 (7.31–7.90) vs. 2.06 (1.23–2.57); p = 0.026]. **Conclusion:** PTDM was mainly associated with post-transplant metabolic laboratory changes, including higher glucose, glycated hemoglobin, triglycerides, and triglyceride/HDL ratio. Recipient clinical variables, donor characteristics, and pre-transplant laboratory parameters were not significantly associated with the outcome. These findings reinforce the importance of early glycemic and lipid monitoring after liver transplantation.

Keywords: post-transplant diabetes mellitus; liver transplantation; metabolic complications.

DIABETES MELLITUS

119 (3016) SAFETY AND CLINICAL OUTCOMES OF IN-HOSPITAL SGLT2 INHIBITOR USE IN PATIENTS HOSPITALIZED FOR ACUTE HEART FAILURE: A SYSTEMATIC REVIEW

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Introduction: Sodium-glucose cotransporter 2 inhibitors have become an important component of heart failure treatment because of their cardiovascular and renal benefits. However, uncertainty remains regarding their safety during hospitalization for acute heart failure, particularly because of concerns involving hypotension, acute kidney injury, and euglycemic diabetic ketoacidosis. **Objective:** To evaluate the safety and clinical outcomes associated with in-hospital use of sodium-glucose cotransporter 2 inhibitors in adults hospitalized for acute heart failure. **Methods:** A systematic review was conducted according to PRISMA recommendations. Searches were performed in PubMed, Embase, Cochrane Library, and Scopus databases using terms related to sodium-glucose cotransporter 2 inhibitors, hospitalization, and acute heart failure. Randomized clinical trials and observational studies evaluating initiation or continuation of these agents during hospitalization were included. Primary outcomes included euglycemic diabetic ketoacidosis, acute kidney injury, hypotension, volume depletion, and hypoglycemia. Secondary outcomes included mortality, heart failure readmission, and length of hospitalization. **Results:** Available evidence suggests that in-hospital use of sodium-glucose cotransporter 2 inhibitors is generally safe in selected patients hospitalized for acute heart failure. Studies demonstrated low incidence of severe adverse events, including euglycemic diabetic ketoacidosis and clinically significant renal dysfunction. In addition, initiation or continuation during hospitalization was associated with reduced heart failure events and lower rates of rehospitalization after discharge. The EMPULSE and SOLOIST-WHF trials reinforced the potential clinical benefit of early introduction in acute heart failure settings. **Conclusion:** Current evidence suggests that carefully selected patients hospitalized for acute heart failure may benefit from in-hospital sodium-glucose cotransporter 2 inhibitor therapy, with acceptable safety profiles and favorable cardiovascular outcomes. Further studies are necessary to better define optimal patient selection and institutional protocols.

Keywords: empagliflozin; cardiovascular outcomes; inpatient care.

DIABETES MELLITUS

120 (3556) SCREENING OF PANCREATIC AUTOANTIBODIES IN PEDIATRIC TYPE 1 DIABETES MELLITUS AS A STRATEGY FOR THE PREVENTION OF DIABETIC KETOACIDOSIS

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Introduction: Type 1 diabetes mellitus (T1DM) is an autoimmune disease characterized by the progressive destruction of pancreatic beta cells. Pancreatic autoantibodies allow the identification of individuals in preclinical stages and estimation of the risk of progression to clinical T1DM. Considering the high frequency of diabetic ketoacidosis at pediatric diagnosis, immunological screening has emerged as a promising strategy for early diagnosis and prevention of severe metabolic decompensation. **Objective:** To analyze the role of pancreatic autoantibody screening in the early diagnosis of pediatric T1DM and prevention of diabetic ketoacidosis. **Methods:** A systematic review was conducted according to PRISMA guidelines and the PICO strategy. Searches were performed in the PubMed, Scopus, and SciELO databases, including studies published between 2019 and 2025. Descriptors related to T1DM, pancreatic autoantibodies, diabetic ketoacidosis, and early diagnosis were combined using the Boolean operators AND and OR. Original studies addressing immunological screening in pediatric T1DM and its association with early diagnosis and prevention of diabetic ketoacidosis were included. Reviews, meta-analyses, case reports, and studies without a clearly defined methodology were excluded. **Results:** The studies demonstrated that multiple positivity for pancreatic autoantibodies is an important marker of progression to clinical T1DM in children. Insulin autoantibodies (IAA) were associated with the earliest stages of pancreatic autoimmunity. Anti-glutamic acid decarboxylase 65 antibodies (anti-GAD65) showed high diagnostic sensitivity, whereas tyrosine phosphatase-related islet antigen-2 autoantibodies (IA-2) were associated with faster progression to clinical T1DM and higher risk of metabolic decompensation. Furthermore, zinc transporter 8 autoantibodies (ZnT8) improved the detection of individuals previously negative for other autoantibodies. Lower incidence of diabetic ketoacidosis at diagnosis was also observed in screened children. **Conclusion:** Pancreatic autoantibody screening is relevant for the early identification of pediatric T1DM and reduction of diabetic ketoacidosis at diagnosis. Multiple positivity and specific markers were associated with higher risk of clinical progression, reinforcing the importance of immunological monitoring in high-risk children.

Keywords: diabetic ketoacidosis; type 1 diabetes mellitus; autoantibodies.

DIABETES MELLITUS

121 (3530) SEX DIFFERENCES IN THE PROGRESSION OF CHRONIC KIDNEY DISEASE IN PATIENTS WITH TYPE 2 DIABETES

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Introduction: The development and progression of chronic kidney disease (CKD) have a heterogeneous course in patients with type 2 diabetes. The sex differences might be one of the aspects related to this evolution, which is a matter of debate in the literature. **Objective:** To evaluate sex differences in the presence and progression of CKD in a cohort of patients with type 2 diabetes. **Methods:** In this prospective cohort study, the patients were submitted to clinical and laboratorial evaluation. Clinical evaluation consisted of glycemic and lipid profile, blood pressure control and the search for diabetic chronic complications. Estimated glomerular filtration rate (eGFR) was estimated by using the CKD-EPI 2009 equation. The main outcome measure was GFR decline ≥ 40% from baseline. **Results:** A total of 368 patients with type 2 diabetes (61 ± 10 years; 191 women; 177 men) were followed for 5.7 ± 2.7 years. Both at baseline and at the end of follow up, increased albuminuria was more frequent in men compared with women (Baseline: 37 vs. 24%; P = 0.009; End of follow up: 46 vs. 27; P = 0.002). Also, both at baseline and at the end of follow up, the presence of CKD was significantly higher in men compared with women (Baseline: 40 vs. 27%; P = 0.01; End of follow up: 61 vs. 41; P < 0.001). The GFR decline per year was 2.8 (interquartile range: 0.9-5.4) and 2.5 (0.3-4.4) mL/min/1.73 m^2, in men and women respectively (P = 0.075). In a multivariate Cox regression analysis, GFR Decline ≥ 40% was significantly associated with male sex (hazard ratio [HR] = 2.15, 95% CI: 1.18 – 3.95; P = 0.01). This effect was independent of age, BMI, hypertension, use of RAS inhibition, baseline CKD and LDL cholesterol. **Conclusion:** In this population, male sex was associated not only with a higher prevalence of CKD but also with its faster progression. The better understanding of sex differences and its factors in the pathophysiologic progression of CKD in patients with type 2 diabetes is essential to the individual management and therapeutic targets of these patients.

Keywords: diabetes mellitus; sex; chronic kidney disease.

DIABETES MELLITUS

122 (3281) SOCIODEMOGRAPHIC DETERMINANTS AND LIFESTYLE BEHAVIORS AMONG PREGNANT WOMEN WITH DIABETES IN A TERTIARY CARE SETTING

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Introduction: Gestational diabetes mellitus (DM) is an increasingly prevalent condition associated with significant adverse maternal and fetal outcomes. Beyond clinical and obstetric factors, growing evidence indicates that its course and management are strongly influenced by sociodemographic characteristics and lifestyle behaviours (LB). **Objective:** To describe the sociodemographic and lifestyle profile in pregnant women with DM, considering structural and behavioral factors. **Methods:** Cross-sectional study including baseline data collected from 2024 to 2026 from an ongoing prospective cohort. Pregnant women aged ≥18 years with gestational or pre-gestational DM followed at a tertiary care center were included. Data were collected using a standardized structured questionnaire, including dietary habits. **Results:** Fifty-eight pregnant women were included, aged 33.1 ± 6.0 years; mean pregestational BMI 31 ± 8.0 kg/m^2; 6 (10.3%) had type 2 DM; 49 (84.5%) were diagnosed with GDM and 3 (5.2%) with overt diabetes in pregnancy (OD). There was a predominance of non-white 44 (75.9%) and married women 38 (65.5%). Education levels were: elementary school 5 (8.6%); high school 31 (53.4%); higher education 22 (38%). Thirty-one (54.4%) were employed (54.2%). Median family income was 2 minimum wages (IQR 1.5–3.0), with a median of 2 individuals per household income (IQR 2–3). Regarding LB, smoking 1 (1.7%) and alcohol use 3 (5.2%), while 12 (21.8%) reported regular physical activity and 24 (42.1%) had the possibility to exercise in their neighborhood. Forty-six (85.2%) reported screen use during meals and when asked which foods were consumed the day before, 43 (75.4%) reported beans, 39 (69.6%) fruits, 44 (77.2%) vegetables, 18 (31.6%) embedded foods, 26 (45.6%) sugar-sweetened beverages, 14 (24.6%) sweets. **Conclusion:** Pregnant women with DM evaluated in this study showed a high frequency of inadequate dietary behaviors and low engagement in physical activity. A thorough understanding of their educational background, socioeconomic status and lifestyle is essential to inform and implement individualized care strategies. Prospective studies are warranted to assess the impact of these factors on maternal and fetal outcomes.

Keywords: diabetes in pregnancy; sociodemographic determinants; lifestyle behaviors.

DIABETES MELLITUS

123 (3384) SUSTAINED CONTINUOUS GLUCOSE MONITORING ENGAGEMENT DESPITE SOCIOECONOMIC VULNERABILITY IN TYPE 1 DIABETES: USER EXPERIENCE AND GLYCEMIC OUTCOMES IN A PUBLIC HEALTH SETTING

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Introduction: Continuous glucose monitoring (CGM) improves glycemic control in type 1 diabetes (T1D), but access remains largely restricted to high-income populations. Evidence on CGM effectiveness and user engagement in socioeconomically vulnerable individuals within public health systems is limited. **Objective:** To evaluate the glycemic effects of CGM in T1D patients and to investigate its clinical impact and user experience in vulnerable individuals. **Methods:** A 6-month longitudinal study enrolled 53 individuals with T1D from a public endocrinology clinic in Northeastern Brazil. Data were collected via interviews and medical record review. An intermittently scanned CGM device was used throughout follow-up. HbA1c was measured at three timepoints: baseline, six months, and after discontinuation. Monthly CGM reports and structured questionnaires captured glycemic metrics and user behavior. **Results:** Most participants were female (64,2%), adults (69,8%), self-declared Black or mixed-race (81,1%), with income up to 2 minimum wages (77,4%) and mean diabetes duration of 11 ± 8,72 years. At baseline, HbA1c was 9,7 ± 1,19% and total daily insulin dose (TDD) 0,8 ± 0,4 IU/kg. After six months, HbA1c decreased to 7,9 ± 1,86%, while TDD remained stable (0,9 ± 0,3 IU/kg). CGM metrics showed sustained use: sensor active time 80,7%, time in range 48,5%, glucose management indicator 7,6%, and low severe hypoglycemia rate below 54 mg/dL of 1%. Daily scans averaged 8,7, with near-universal nocturnal monitoring (95,9%). Sensor accuracy was well-perceived: 60,4% reported no discrepancy with capillary glucose at six months. Most participants (77%) used CGM data to proactively prevent hypoglycemic episodes. Patient autonomy in scanning rose from 28,3% to 52,1%; by end of follow-up, no participant relied exclusively on caregivers. Dermatitis was observed in approximately 15% of participants, exceeding controlled trial rates, with no severe cases. Sensor detachment averaged 10% across follow-up. A high-engagement subgroup (active time equal to or above 70% and 8 or more scans per day) comprised 30,2% of participants. Following CGM discontinuation, HbA1c rose to 8.50 ± 1.86%, though not significantly (p = 0.236). **Conclusion:** CGM promoted glycemic improvement, sustained engagement, and proactive self-care in this T1D population. Socioeconomic barriers did not preclude successful technological engagement, reinforcing the need for equitable access policies in public health systems.

Keywords: type 1 diabetes; continuous glucose monitoring; social vulnerability.

DIABETES MELLITUS

124 (3166) TECHNOLOGY AND TYPE 1 DIABETES: EFFECTS OF THE CONTINUOUS INSULIN INFUSION SYSTEM ON LONG-TERM GLYCEMIC CONTROL

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Introduction: Type 1 diabetes mellitus (T1DM) is a chronic autoimmune condition characterized by the destruction of pancreatic beta cells, resulting in complete insulin deficiency. Effective management depends on continuous glucose monitoring and insulin replacement therapy; however, maintaining optimal glycemic control remains difficult because of glycemic fluctuations, hypoglycemic episodes, and challenges related to treatment adherence. Technological advances in T1DM care, including continuous glucose monitoring devices, insulin pumps, and hybrid closed-loop systems, have contributed to better clinical outcomes. Among these technologies, one automated continuous insulin infusion system has gained prominence by combining a glucose sensor with an insulin pump in an automated platform capable of adjusting insulin delivery through advanced algorithms, thereby improving glycemic stability. **Objective:** To assess the effects of using an automated continuous insulin infusion system for at least 12 months on glycemic control in patients with T1DM followed in a private clinical setting. **Methods:** This observational, retrospective, longitudinal study included patients diagnosed with T1DM for at least one year who had been using the automated continuous insulin infusion system for a minimum of 12 months and had complete glycemic monitoring data available both before device initiation and after at least 12 months of use. Individuals who did not fulfill the inclusion criteria were excluded. Data collection was performed through review of medical records and sensor reports. **Results:** A total of 27 patients were analyzed (mean age: 37.4 years; average duration of T1DM: 20 years; average duration of system use: 2.7 years). Mean HbA1c levels decreased from 8.2% to 6.9%, while time in range (70–180 mg/dL) improved from 52.3% to 74.0%. Additionally, time spent in hypoglycemia (<70 mg/dL) declined from 3.0% to 2.4%. **Conclusion:** Prolonged use of the automated continuous insulin infusion system was associated with marked improvements in glycemic management, demonstrated by increased time in range, lower HbA1c levels, and reduced hypoglycemia, supporting its value as an effective therapeutic strategy for T1DM care.

Keywords: type 1 diabetes mellitus; continuous insulin infusion system; glycemic control.

DIABETES MELLITUS

125 (3328) TEMPORAL TRENDS IN HOSPITALIZATIONS FOR DIABETIC KETOACIDOSIS AND HYPERGLYCEMIC HYPEROSMOLAR STATE IN BRAZIL

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Introduction: Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are the leading hyperglycemic emergencies in diabetes mellitus, associated with significant morbidity and mortality. Their distribution differs across age groups and diabetes types, as DKA predominantly affects individuals with type 1 diabetes, while HHS is more common in type 2. Regional disparities and heterogeneity in patient profiles may further influence outcomes. We describe trends and determinants of DKA and HHS hospitalizations in Brazil over the past decade. **Methods:** Ecological study based on data obtained from the Hospital Information System (SIH/SUS), covering the period from January 2016 to December 2025. Hospital admissions for DKA and HHS in Brazil were analyzed. The variables assessed included the type of diabetes, intensive care unit (ICU) admission, mortality, presence of coma, age group, and regional distribution. **Results:** A total of 195,045 hospitalizations were recorded, with an overall mortality rate of 6.2% (n = 12,022). Type 1 DM accounted for 57% of cases, and DKA without coma was the most frequent presentation (86%). Females comprised 54% of admissions. ICU admission occurred in 31% of cases and was independently associated with mortality (OR 2.46; 95% CI 2.36–2.57). ICU rates rose from 17.5% in 2016 to 39.3% in 2025, paralleled by a decline in mortality from 6.5% to 5.2%. Mortality rates increased across all macro-regions during 2020–2021, followed by a progressive recovery in most regions in subsequent years. The North Region was notable for sustaining elevated mortality rates following this period. Hospitalizations peaked in the 15–29 age group (23%), while deaths were disproportionately concentrated at age extremes. The > 75 years age group exhibited the highest independent risk of death in the multivariate model (OR 46.6; 95% CI 40.4–54.1). Mortality rates were similar between the sexes. **Conclusion:** Rising hospitalizations may suggest an expanding diabetic population and difficult population-level glycemic control, potentially reflecting limited outpatient access. A 6.2% mortality rate for readily treatable conditions points to delays in diagnosis and management. Age over 75 years was the strongest independent predictor of death, and ICU admission was independently associated with worse prognosis. Persistent regional disparities, particularly in the North Region, underscore an ongoing public health challenge in Brazil.

Keywords: diabetic ketoacidosis; hyperglycemic hyperosmolar state; temporal trend.

DIABETES MELLITUS

126 (2917) THE EFFECT OF TIRZEPATIDE VS. DULAGLUTIDE ON MAJOR ADVERSE CARDIOVASCULAR EVENTS (MACE-4 AND MACE-5) IN PATIENTS WITH TYPE 2 DIABETES AND ESTABLISHED ATHEROSCLEROTIC CARDIOVASCULAR DISEASE

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Introduction: In SURPASS-CVOT, tirzepatide (TZP) was non-inferior to DULA in reducing the risk of major adverse cardiovascular events (MACE-3: CV death, MI, or stroke) in type 2 diabetes(T2D) and established atherosclerotic cardiovascular disease (ASCVD) patients. **Objective:** This analysis evaluated differences in extended CV outcomes. **Methods:** In these prespecified analyses, we assess the effect of TZP (≤15 mg) vs. DULA (1.5 mg) on MACE-4 (CV death, MI, stroke, or coronary revascularisation) and MACE-5 (MACE-4 or hospitalisation due to unstable angina [UA]). **Results:** MACE-4 occurred in 1089 (16.5%) receiving TZP vs. 1217 (18.5%) receiving DULA (HR: 0.88; 95% CI: 0.81–0.96). With prior coronary artery disease (CAD), MACE-4 occurred in 875 (18.6%) receiving TZP, and 967 (20.5%) receiving DULA (HR: 0.89; 95% CI: 0.81–0.97). Without prior CAD, MACE-4 occurred in 214 (11.3%) receiving TZP, and 250(13.3%) receiving DULA (HR: 0.84; 95% CI: 0.70–1.01). MACE-5 occurred in 1112(16.9%) receiving TZP and 1240 (18.8%) receiving DULA (HR: 0.88; 95% CI: 0.81–0.96). Coronary revascularisation occurred in 527 (8.0%) vs. 617 (9.4%) (HR: 0.84; 95% CI: 0.75–0.95). Hospitalisation due to UA occurred in 110 (1.7%) vs. 148 (2.2%) (HR: 0.74; 95% CI: 0.57–0.94). **Conclusion:** Among T2D and ASCVD patients, TZP was associated with a lower risk of MACE-4, MACE-5, coronary revascularisation, and UA hospitalisation vs. DULA.

Keywords: cardiovascular events; atherosclerotic cardiovascular disease; MACE-3

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This study was funded by Eli Lilly and Company. Previously presented at American Heart Association 98th Scientific Session.
Keywords: tirzepatide; MACE-4; MACE-5.

DIABETES MELLITUS

127 (3224) THE IMPACT OF THE LEVEL OF KNOWLEDGE ABOUT THE MANAGEMENT OF DIABETES MELLITUS AT A REFERRAL CENTER IN THE AMAZON REGION

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The treatment of diabetes mellitus (DM) involves lifestyle changes, glycemic monitoring, and medication, requiring full patient adherence, which is often hindered by insufficient knowledge about the disease. This study aimed to quantify DM knowledge among patients seen at a reference center in Pará using the Diabetes Knowledge Questionnaire (DKN-A), investigate its association with sociodemographic variables and DM type, and propose an educational strategy adapted to the local context. This is a cross-sectional observational study conducted in Belém with 92 adult patients diagnosed with type 1 and type 2 DM. The DKN-A was applied in person, assessing five domains: basic physiology, hypoglycemia, food groups and substitutions, management during complications, and general care principles. Age, time since diagnosis, and education level were also collected. Data were analyzed using chi-square, Mann-Whitney, Kruskal-Wallis tests, and Pearson correlation, with significance set at $p \leq 0.05$. The sample consisted mostly of middle-aged and elderly adults, predominantly with type 2 DM, primary/secondary education, and a long average time since diagnosis. Overall knowledge level was moderate, with better performance in basic physiology and hypoglycemia, and lower scores in food groups/substitutions and general care principles, revealing gaps in daily disease management. A statistically significant association was found between knowledge and age, education, and DM type: younger patients with higher education and type 1 DM achieved better scores, while older patients with lower education and type 2 DM showed greater vulnerability. Time since diagnosis showed no significant association, indicating that prolonged experience with the disease does not by itself ensure greater understanding. Based on these findings, a protocol was developed to guide medical students on what to clarify with patients, organized into three axes: general care principles, food groups, and food substitutions, using accessible language and regional examples (açaí, farinha). The need for individualized health education adapted to the culture of Northern Brazil is reinforced, promoting treatment adherence, prevention of complications, and improved quality of life.

Keywords: diabetes mellitus; knowledge; Amazon region.

DIABETES MELLITUS

128 (2956) THE ROLE OF CARBOHYDRATE INTAKE IN GLYCEMIC CONTROL AND CARDIOVASCULAR RISK IN ADULTS WITH TYPE 1 DIABETES USING INSULIN PUMP: A CROSS-SECTIONAL STUDY

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Introduction: Dietary management in type 1 diabetes mellitus (T1D) is challenging. The optimal carbohydrate intake to meet glycemic targets and mitigate cardiovascular risk remains unclear. **Objective:** Our aim was to evaluate the associations between carbohydrate intake, glycemic control and cardiovascular risk factors in T1D. **Methods:** We performed a cross-sectional study in adults with T1D using insulin pumps and practicing advanced carbohydrate counting, followed at a tertiary hospital between May 2022 and May 2024. We excluded those without at least 2 meals recorded per day in 14-day pump reports. Carbohydrate intake and meal frequency declared by the patient were extracted from pump reports. Glycemic control was assessed by glycated hemoglobin (HbA1c) and 14-day continuous glucose monitoring (CGM) metrics. Data on cardiovascular risk factors, including lipid profile, blood pressure, kidney function, body mass index (BMI) and waist circumference, were collected from clinical records. Associations per 10 g/day increase in carbohydrate intake were analysed using linear regression models unadjusted; adjusted for age, sex and BMI (Model 1); and additionally for number of meals per day and use of automated insulin delivery (AID) (Model 2). **Results:** We included 128 patients (median age 31.5 [22.0–42.5] years, 47.7% men, 16.4% were using AID). The mean daily carbohydrate intake was 166.3 ± 73.3 g, across 4.3 ± 1.6 meals. Higher carbohydrate intake was associated with lower HbA1c (β = –0.04, 95% CI: –0.06 to –0.01; P = 0.012) and less time in hyperglycemia (β = -0.02, 95% CI: –0.04 to –0.01; P = 0.001) in Model 1; however, both associations became non-significant after adjusting for meal frequency and AID. In Model 2, higher carbohydrate intake was associated with an increased time in hypoglycemia (β = 0.03, 95% CI: 0.00 to 0.06; P = 0.043). Higher carbohydrate intake was also associated with higher systolic (β = 0.47, 95% CI: 0.13 to 0.81; P = 0.008) and diastolic (β = 0.48, 95% CI: 0.17 to 0.79; P = 0.003) blood pressure, with no other associations with cardiovascular risk factors. **Conclusion:** Apparent benefits of higher carbohydrate intake on glycemic outcomes were attenuated after accounting for meal frequency and AID use, suggesting that these factors may partly account for the observed associations. Regardless of meal frequency and AID use, higher carbohydrate intake remained associated increased hypoglycemia and higher blood pressure.

Keywords: diabetes; cardiovascular risk; carbohydrate intake.

DIABETES MELLITUS

129 (3205) THERAPEUTIC HELPLESSNESS IN DIABETES MELLITUS: JUDICIALIZATION AS A REFLECTION OF THE LAG IN THE INCORPORATION OF NEW TECHNOLOGIES IN THE SUS

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diabetes mellitus (DM) is one of the primary chronic diseases with a global impact, requiring continuous monitoring and regular access to drug therapies and health technologies. In recent decades, therapeutic advances such as insulin analogues, continuous glucose monitoring systems, and GLP-1 receptor agonists have provided significant improvements in glycemic control and patients quality of life. However, the incorporation of these technologies into the Brazilian Unified Health System (SUS) occurs gradually, creating barriers to access and contributing to the rise in the judicialization of health. Narrative literature review, considering patients with diabetes mellitus treated by the SUS (P), the judicialization to obtain new therapeutic technologies (I), compared to the conventional access provided by the public system (C), highlights impacts related to the lag in technological incorporation and inequality in treatment access (O). Articles published between 2015 and 2025, in both Portuguese and English, were included. The analyzed studies demonstrate a progressive growth in legal demands related to DM, especially for obtaining insulin analogues, glucose monitoring sensors, and high-cost medications not yet widely available through SUS. The literature highlights that delays in evaluation and technological incorporation processes contribute to the therapeutic neglect of patients, who turn to the Judiciary as a mechanism to guarantee their constitutional right to health. It was also observed that budgetary, bureaucratic, and structural limitations hinder the rapid updating of clinical protocols and the lists of medications offered by the public system. Furthermore, patients with greater socioeconomic support and access to legal assistance are more likely to obtain these technologies through the courts, widening inequalities in access to care. Judicialization in diabetes mellitus starkly highlights the existing gaps in the incorporation of therapeutic technologies within the SUS, revealing structural weaknesses in pharmaceutical assistance and in the guarantee of comprehensive and timely care. This scenario reinforces the need to improve health technology assessment and incorporation mechanisms, with greater decision-making agility, a robust scientific basis, and a commitment to equity, thereby mitigating inequalities and improving diabetes management within the public health network.
Keywords: diabetes mellitus; unified health system; GLP-1 analogues.

DIABETES MELLITUS

130 (3451) TIRZEPATIDE VERSUS GLP-1 RECEPTOR AGONISTS ON CARDIOVASCULAR AND RENAL OUTCOMES IN PATIENTS WITH OBESITY AND TYPE 2 DIABETES MELLITUS: A SYSTEMATIC REVIEW

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Obesity and type 2 diabetes mellitus are strongly associated with increased cardiovascular risk and progression of chronic kidney disease. In this context, tirzepatide, a dual glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1 receptor agonist, has emerged as a promising therapeutic strategy with potential cardiorenal benefits beyond glycemic control. This systematic review aimed to compare tirzepatide with traditional glucagon-like peptide-1 receptor agonists regarding cardiovascular, renal, and safety outcomes in adults with obesity and/or type 2 diabetes mellitus. The research question was structured according to the PICO strategy: adults with obesity and/or type 2 diabetes mellitus (P), treated with tirzepatide (I), compared with glucagon-like peptide-1 receptor agonists (C), evaluating cardiovascular, renal, and safety outcomes (O). A systematic literature review was conducted using PubMed, Wiley, and ScienceDirect databases, including studies published in English between 2021 and 2026. Randomized clinical trials, propensity score-matched cohort studies, and meta-analyses comparing tirzepatide with glucagon-like peptide-1 receptor agonists were included. The analyzed studies demonstrated predominantly favorable cardiovascular and renal outcomes with tirzepatide. Major adverse cardiovascular events were less frequent among patients receiving dual GIP/GLP-1 receptor agonist therapy compared with traditional glucagon-like peptide-1 receptor agonists (12.2% versus 13.1%), corresponding to a relative cardiovascular risk reduction of up to 14%. Regarding renal outcomes, including macroalbuminuria and decline in renal function, tirzepatide was associated with relative risk reductions of up to 26% in progression to advanced kidney disease. Additionally, a propensity score-matched observational study demonstrated lower progression of cardiorenal-metabolic syndrome among patients treated with tirzepatide (2.2% versus 3.3%). Adverse events were predominantly gastrointestinal, especially nausea and diarrhea, with similar incidence between groups. Overall, tirzepatide demonstrated promising cardiovascular and renal benefits, in addition to a safety profile comparable to traditional glucagon-like peptide-1 receptor agonists. However, direct comparative studies specifically evaluating renal outcomes remain limited.
Keywords: tirzepatide; cardiovascular outcomes; type 2 diabetes mellitus.

DIABETES MELLITUS

131 (3223) TRIIODOTHYRONINE (T3) ASSOCIATED OR NOT WITH INSULIN AMELIORATES DIABETES MELLITUS INDUCED BY ALLOXAN IN RATS BY NEGATIVELY MODULATING GLUCAGON LEVELS AND SIGNALING IN LIVER AND WHITE ADIPOSE TISSUE

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diabetes mellitus (DM) is a chronic metabolic disease characterized by hyperglycemia and elevated glucagon secretion, which contributes to the impaired glycemic control in DM. Studies from our laboratory have shown that rats induced to DM by alloxan present hypothyroidism, in parallel with hyperglycemia, chronic inflammation, insulin resistance (IR) and increased hepatic glucose production. The latter effect is supposed to result, at least in part, of the increased levels of glucagon (Gcg), which occurs in states of insulin deficient states. All these parameters were improved by treating DM rats with triiodothyronine (T3), which also reduced the expression of key enzymes involved in gluconeogenesis, which are stimulated by glucagon. This was an unexpected effect of T3, since it shares this metabolic action with Gcg. To explore the theory-data gap, this study evaluated the serum Gcg levels, its signaling pathways in liver, measuring the expression of pCREB, as well as of proteins involved with the increased glucose liver production (PCK-1, G6PC, GLUT2, GK), and of the target enzymes of Gcg in Adipose tissue- AT- (HSL, LPL, HK, SCD1) in DM rats treated with T3. Glycemia and TSH levels were also measured. We have also associated insulin (replacement dose and half of it) to T3 treatment, considering that T3 increased insulin sensitivity. To achieve this goal, male Wistar rats induced to DM by alloxan were assorted into groups treated with T3 (1.5 µg/100 g BW), associated or not to replacement dose (6U) of insulin, or half of it, for 28 days. The animals were anesthesized, euthanized and liver, AT and blood were collected. The DM rats show increased TSH and Gcg levels; pCREB liver expression (indicating enhanced Gcg signaling), and proteins involved in liver glucose production, and lipolysis, in AT. T3 treatment decreased Gcg levels and hepatic signaling, and the content of its target proteins in liver and AT, however suppressed TSH levels. Treatment with T3 + 3U insulin, as well as with insulin 6U alone, was most effective in reversing the assessed parameters toward control levels, including TSH. These findings justify our results, and open a perspective for using thyromimetics as adjuvant for DM treatment, since the dose of Insulin could be reduced by half, which would postpone de IR observed in chronically treated patients. Further studies are necessary to elucidate the precise mechanism underlying T3-mediated decrease in Gcg levels and its signaling in DM.

Keywords: diabetes mellitus; T3 treatment; glucagon.

DIABETES MELLITUS

132 (3376) USER EXPERIENCE WITH CONTINUOUS GLUCOSE MONITORING IN TYPE 1 DIABETES: THEMATIC ANALYSIS OF PATIENT NARRATIVES FROM A PUBLIC HEALTHCARE SETTING IN NORTHEASTERN BRAZIL

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Introduction: Beyond glycemic metrics, understanding the subjective experience of continuous glucose monitoring (CGM) in type 1 diabetes (T1D) individuals is essential to optimize technology adoption, especially in vulnerable underrepresented populations. **Objective:** To investigate the user experience of CGM in T1D individuals through qualitative thematic analysis of narratives. **Methods:** A 6-month longitudinal study enrolled 53 individuals with T1D from a public endocrinology clinic in Northeastern Brazil. At end of follow-up, 43 participants submitted audio messages describing their CGM experience. Cohort characteristics are reported for this subset (n = 43). Recordings were fully transcribed and analyzed using thematic categorization by recurrence, with coding and grouping into analytical domains. An AI language model assisted in the categorization process, with all data handled anonymously. **Results:** The cohort was predominantly female (69,8%), mostly adults (74,4%), self-declared Black or mixed-race (81,4%), with income equal to or below 2 minimum wages (79,1%), 72,1% without private health insurance, mean diabetes duration of 12 ± 9,36 years, 65,1% residing in rural areas, and 44,2% dependent on government-provided patient transport to access care. Self-reported anxiety and depressive symptoms were present in 71,4% and 28,6% of participants, respectively. Glycemic improvement was observed over follow-up. Three thematic domains emerged. Domain I (Biopsychosocial impact and quality of life) addressed elimination of fingerstick pain, prevention of glycemic events, and improved sleep through psychological reassurance. Domain II (Health education and empowerment) reflected reports consistent with enhanced health literacy, with participants identifying postprandial patterns and adjusting diet and insulin autonomously. Domain III (Socioeconomic vulnerability and access barriers) indicated distress over device discontinuation due to financial constraints. **Conclusion:** In a population with significant geographic and socioeconomic barriers, CGM was associated with self-reported biopsychosocial benefits and supported a process of empowerment in diabetes self-management. Qualitative narrative analysis proved essential to capture dimensions of technology adoption beyond glycemic data, offering clinicians and policymakers a richer framework for evaluating CGM integration into public health care.

Keywords: type 1 diabetes; continuous glucose monitoring; patient satisfaction.

DIABETES MELLITUS

133 (3510) WHEN WEIGHT DECREASES, CONTROL FLOURISHES: THE IMPACT OF A DUAL INCRETIN RECEPTOR AGONIST IN LATENT AUTOIMMUNE DIABETES IN ADULTS

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Case presentation: A 30-year-old female patient with a diagnosis of latent autoimmune diabetes in adults (LADA) established in 2024, associated with class II obesity, is presented. She had a maximum weight of 105.8 kg and was previously using 32 units/day of insulin degludec, along with a dietary adjustment regimen based on carbohydrate counting. In October 2025, weekly tirzepatide therapy was initiated, resulting in a significant weight reduction of 27.6% of total body weight, reaching 82.9 kg and a body mass index of 28.69 kg/m². Concurrently, there was a progressive reduction in basal insulin requirements, reaching 8 units/day of degludec, with no need for rapid-acting insulin due to prior hypoglycemic episodes. The patient maintained regular physical activity, including resistance training three to four times per week, along with ongoing nutritional follow-up. Laboratory tests showed fasting glucose of 89 mg/dL, glycated hemoglobin of 5.4%, a well-controlled lipid profile, and absence of albuminuria. Continuous glucose monitoring demonstrated 96% time in range and a mean glucose of 87 mg/dL, with rare hypoglycemic episodes. There were no signs of diabetic retinopathy, neuropathy, or nephropathy. **Discussion:** Latent autoimmune diabetes in adults is characterized by progressive loss of pancreatic beta-cell function, often culminating in intensification of insulin therapy. The present case is notable for the marked metabolic response following the introduction of tirzepatide, with significant weight loss, improved glycemic control, and substantial reduction in basal insulin requirements. Although there are still limited studies involving dual agonists of glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) receptors in individuals with LADA, the observed results suggest a meaningful metabolic benefit. The combination of weight loss, regular physical activity, and therapeutic optimization likely contributed to the outcomes observed. **Final comments:** This case highlights the potential benefit of tirzepatide in a patient with LADA and obesity, promoting significant improvement in glycemic control and a marked reduction in insulin requirements. Cases like this underscore the need for further studies on incretin-based therapies in individuals with autoimmune diabetes, particularly in the context of associated obesity.
Keywords: latent autoimmune diabetes in adults; tirzepatide; weight loss.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

134 (3459) A RARE GENETIC ASSOCIATION FAMILIAL CHYLOMICRONEMIA SYNDROME AND LIPODYSTROPHY: A CASE REPORT

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Case report: A family carrying the familial chylomicronemia syndrome (FCS) had its members evaluated clinically, laboratory and genetically. The FCS offspring were identified as carrying a homozygous pathogenic splice-site variant in the GPIHBP1 gene[c.182-1G>T, OMIM*612757], while their consanguineous parents were found this pathogenic GPIHBP1 variant associated with a heterozygous LMNA missense variant [p.Arg329His]. The father is 63 years old obese man (BMI 31.2 kg/m²) presenting type 2 DM, arterial hypertension, and waist circumference of 110 cm. Laboratory data demonstrated reduced leptin concentration (7.8 ng/mL) triglycerides of 651 mg/dL, total cholesterol 212 mg/dL, LDL 98 mg/dL, HDL 30 mg/dL, ApoB 132 mg/dL, Lp(a) 129 mg/dL, and VLDL 94 mg/dL coronary artery calcium (CAC) score of 9 at the 32th percentile, without plaques or coronary stenosis. The mother is a 55 years old overweight woman (BMI 27.2 kg/m²), no presenting type 2 DM, triglycerides of 309 mg/dL, LDL-c 188 mg/dL, HDL-c 39 mg/dL, ApoB 137 mg/dL, Lp(a) 124 mg/dL, HbA1c 5.2%, VLDL 52 mg/dL and leptin 18 ng/mL. CAC score of 13 at the 32 th percentile and mild non-obstructive stenosis of approximately 30% in the left anterior descending artery. **Discussion:** There are currently no scientific reports describing the coexistence of both GPIHBP1 and LMNA variants in the same family context associated with offspring homozygous for GPIHBP1, which is extremely rare event due to distinct inheritance mechanisms and the ultra-rare frequency of these disorders in the general population. FCS related to GPIHBP1 is inherited in an autosomal recessive pattern, whereas LMNA-related lipodystrophy syndromes demonstrate autosomal dominant inheritance. This reinforces the concept that partial defects in the lipoprotein lipase pathway may present with variable expressivity influenced by metabolic, adipose-related, and additional genetic modifiers. Despite markedly elevated ApoB and Lp(a) levels in both individuals only mild subclinical atherosclerosis was observed, suggesting a possible dissociation between severe triglyceride metabolism disorders and advanced obstructive coronary disease in selected patients. **Final comments:** These findings reinforce the importance of integrated clinical, biochemical, imaging, and molecular genetic evaluation in families with FCS and raise the hypothesis of potential genic interaction between triglyceride metabolism and adipose tissue regulatory pathways.
Keywords: familial chylomicronemia syndrome; GPIHBP1; LMNA.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

135 (3346) ABNORMAL FAT DISTRIBUTION IN PATIENTS WITH VERY HIGH CARDIOVASCULAR RISK AND THEIR FIRST-DEGREE RELATIVES: EVIDENCE OF AN UNDERRECOGNIZED LIPODYSTROPHY PHENOTYPE

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Familial partial lipodystrophy (FPLD) is a rare disorder characterized by abnormal adipose tissue distribution, severe insulin resistance, diabetes mellitus (DM), and premature cardiovascular disease. Despite its rarity, FPLD may be underdiagnosed in individuals with obesity and very high cardiovascular risk (CVR), as excess adiposity can mask classical lipoatrophic features. DXA-derived fat partitioning parameters may provide an objective approach to detecting abnormal fat distribution in this setting. This study aimed to describe body fat distribution by DXA in patients with very high CVR, defined as index cases (IC), and their first-degree relatives (FDRs), exploring familial clustering and limitations of current FPLD diagnostic criteria. This was a descriptive observational study conducted at a tertiary lipid reference center. Five index case (IC) — first-degree relative (FDR) pairs underwent anthropometric assessment and whole-body DXA. Lipodystrophy phenotype was evaluated using fat mass ratio (FMR) > 1.2, Köb index > 3.47, lower-limb adiposity < 25%, and thigh skinfold < 20 mm in women or < 10 mm in men. Genetic testing was not performed. The five IC were women, with a median age of 64 years; all had mixed dyslipidemia and DM. Four had premature coronary artery disease, with a median onset at 54 years, and one had heart failure. Median BMI was 36.1 kg/m². Four IC fulfilled at least one lipodystrophy criterion: FMR > 1.2 in four, lower-limb adiposity < 25% in two, Köb index > 3.47 in one, and thigh skinfold compatible with lipodystrophy in two. FDRs, including four women and one man, had a median age of 40 years and median BMI of 32.1 kg/m². Although they had lower metabolic burden, with DM present in only one of five, all showed FMR >1.2 and at least one additional marker, suggesting familial clustering of abnormal fat distribution. One IC-FDR pair showed discordant DXA findings: the IC met no criterion, whereas the FDR presented FMR 1.36, Köb index 4.73, and lower-limb adiposity 22.89%. This discordance is reported descriptively and warrants further investigation. These findings suggest that a lipodystrophy-like phenotype may be present in patients with very high CVR, even in the context of obesity, and that familial DXA-based assessment may help identify at-risk individuals. Prospective studies with molecular investigation are needed to validate these observations.
Keywords: diabetes mellitus; familial partial lipodystrophy; dyslipidemia.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

136 (3347) BODY FAT REDISTRIBUTION AS A CARDIOMETABOLIC MARKER IN FAMILIAL PARTIAL LIPODYSTROPHY TYPE 1: A CASE SERIES OF THREE SISTERS

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Case presentation: Three sisters followed at a tertiary referral center in Ceará, Brazil, presented with a phenotype consistent with familial partial lipodystrophy type 1 (FPLD1). The index case (53y, BMI 29 kg/m²) was diagnosed with type 2 diabetes mellitus (T2DM) at age 24, evolving with severe insulin resistance and premature coronary artery disease (CAD) requiring percutaneous coronary intervention at age 39. Dual-energy X-ray absorptiometry (DXA) showed fat mass ratio (FMR) 1.81, lower-limb adiposity 13%, and Köb index 8.0. Baseline laboratory evaluation revealed triglycerides (TG) 259 mg/dL, HDL-c 32 mg/dL, LDL-c 23 mg/dL, and HbA1c 8.1%. After pioglitazone, HbA1c decreased to 7.2% with 15% insulin reduction. The second sister (63y, BMI 27.8 kg/m²) had T2DM and severe hypertriglyceridemia. DXA demonstrated FMR 1.7, lower-limb adiposity 15.6%, and Köb index 5.5. Baseline: TG 940 mg/dL, HDL-c 30 mg/dL, non-HDL-c 320 mg/dL, and HbA1c 7.5%. After pioglitazone, TG decreased to 127 mg/dL, HDL-c increased to 50 mg/dL, LDL-c reached 52 mg/dL, and HbA1c improved to 6.2%, allowing insulin discontinuation. Genetic testing for lipodystrophy-related variants was negative in both sisters. The third sister (65y) declined DXA and genetic testing; diagnosis was based on clinical and familial findings. She had diabetes mellitus for > 30 years, HbA1c 9.4%, TG 257 mg/dL, HDL-c 44 mg/dL, LDL-c 78 mg/dL, and coronary calcium score of 398 Agatston units. After pioglitazone, HbA1c improved to 7.6%, TG to 172 mg/dL, and HDL-c to 66 mg/dL. **Discussion:** FPLD1 remains underdiagnosed, particularly in overweight individuals and in the absence of pathogenic variants. Clinical phenotype, anthropometric findings, and DXA-derived indices, including FMR, lower-limb adiposity, and Köb index, supported diagnosis in the evaluated sisters. The severe cardiometabolic phenotype, characterized by insulin resistance, hypertriglyceridemia, low HDL-c, and premature CAD, highlights the importance of early recognition. Pioglitazone promoted marked metabolic improvement, supporting its therapeutic role in FPLD1. **Final comments:** DXA-derived body composition analysis combined with family history and severe cardiometabolic phenotype may improve recognition of FPLD1 and support earlier treatment strategies.

Keywords: diabetes mellitus; familial partial lipodystrophy; dyslipidemia.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

137 (3061) CARDIOVASCULAR RISK STRATIFICATION AND LDL-CHOLESTEROL TARGET ATTAINMENT IN PATIENTS WITH TYPE 2 DIABETES AT A SPECIALIZED PUBLIC OUTPATIENT CLINIC

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Introduction: Current guidelines recommend LDL-cholesterol targets below 50 mg/dL for patients at very high cardiovascular (CV) risk and below 40 mg/dL for those at extreme CV risk in type 2 diabetes mellitus (T2DM). However, real-world attainment of these targets remains poorly documented in Brazilian public outpatient settings. **Objective:** To evaluate LDL-cholesterol target attainment and statin use among T2DM patients stratified by cardiovascular risk in a specialized endocrinology outpatient clinic. **Methods:** Cross-sectional study including 256 T2DM outpatients. Data were collected using REDCap and organized into a patient-level database. Cardiovascular risk was classified as extreme (≥2 macrovascular events or 1 macrovascular event associated with diabetic nephropathy), very high (1 isolated macrovascular event), or lower-risk categories. Macrovascular events included myocardial infarction, stroke, peripheral arterial disease, and heart failure. LDL-cholesterol target attainment (<50 mg/dL and <40 mg/dL) was evaluated across risk groups. Fisher's exact test was used for categorical comparisons. LDL levels according to statin use were compared using Welch's t-test. **Results:** Among 256 patients, 54 (21.1%) were classified as extreme risk, 26 (10.2%) as very high risk, and 176 (68.8%) as lower-risk categories. LDL < 50 mg/dL was achieved in 14.8% of extreme-risk patients, 11.5% of very high-risk patients, and 5.1% of lower-risk individuals (p = 0.043). LDL < 40 mg/dL was achieved in only 5.6% of extreme-risk patients. Statins were used by 81.6% of patients (209/256), and by 96.3% (77/80) among those at very high or extreme risk. Mean LDL was significantly lower among statin users compared to non-users (85.6 vs. 97.4 mg/dL; p = 0.010). Among patients classified as very high or extreme risk, only 3 were not receiving statin therapy. **Conclusion:** Despite widespread statin use, attainment of recommended LDL-cholesterol targets remains markedly inadequate among T2DM patients at very high and extreme cardiovascular risk, particularly regarding the LDL < 40 mg/dL target. These findings highlight a substantial gap between guideline recommendations and real-world lipid control, reinforcing the need for more intensive lipid-lowering strategies in high-risk patients relying on public healthcare services.

Keywords: LDL-cholesterol; type 2 diabetes mellitus; cardiovascular risk.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

138 (3291) CASE REPORT: FAMILIAL HYPERCHYLOMICRONEMIA SYNDROME IN AN ADOLESCENT UNDER THE CARE OF A NUTRITION OUTPATIENT CLINIC

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Case presentation: An 11-year-old female patient was referred to the Rare Diseases Service and the Nutrition Outpatient Clinic of a university hospital following recurrent episodes of acute pancreatitis in 2021, associated with severe hypertriglyceridaemia (triglycerides > 7,000 mg/dL). Since symptom onset, she had experienced weight loss of approximately 5 kg, abdominal pain, and vomiting. She had no comorbidities or family history of similar conditions. At admission, she was taking simvastatin, orlistat, nicotinic acid, fat-soluble vitamins, and a multivitamin complex. Nutritional follow-up comprised five outpatient consultations with clinical, anthropometric, biochemical, and dietary assessments. Dietary history showed reduced consumption of processed and ultra-processed foods, but with dietary monotony and maternal anxiety regarding feeding. An individualized dietary plan was prescribed, consisting of six daily meals totaling 1769.5 kcal/day (47 kcal/kg/day), distributed as 64.1% carbohydrates, 17.3% protein, 18.6% fat, and 45.56 g fiber. Study approved by the Research Ethics Committee under opinion n° 660009. **Discussion:** Familial chylomicronemia syndrome (FCS) is a rare genetic disorder characterized by severe hypertriglyceridaemia resulting from reduced clearance of plasma chylomicrons. The patient presented with symptoms consistent with the literature, including abdominal pain, vomiting, and recurrent pancreatitis, but without classic signs such as eruptive xanthomas or retinal lipaemia. During follow-up, clinical improvement was observed, with no new episodes of pancreatitis and appropriate weight and height progression. Dietary management prioritized fat restriction, reduction of simple sugars, and encouragement of fiber intake, while respecting the patient's individual needs and adherence. The patient maintained weight and height development appropriate for her age, with a eutrophic BMI. Triglyceride levels showed a significant initial reduction, although with fluctuations throughout the follow-up, highlighting the complexity of FCS management and the challenges of treatment adherence in the paediatric age group. **Conclusion:** Multidisciplinary follow-up, particularly nutritional support, was crucial in the management of FCS, contributing to clinical improvement and nutritional progress. The rarity of the condition underscores the importance of publishing case reports to aid in the management of similar cases.
Keywords: rare diseases; type I hyperlipoproteinaemia; adolescent nutrition.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

139 (3113) GLUTAMINE METABOLISM AND CARDIOMETABOLIC RISK IN HIV: A NARRATIVE REVIEW

João Costa Nunes[1], João Victor de Azevedo Marques[1], Amanda Vieira de Andrade[1], Luiz Fernando Moschen Carvalho[1], Thiago Castro da Silva[1], Allef Ayako dos Santos Oliveira[1], Maria Thallyta Biliu Paiva[1], Júlio Nunes Braga[2], Brenda Nunes Brito[3], Taides Tavares dos Santos[1]

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Introduction: The survival of people living with human immunodeficiency virus (PLWH) undergoing combination antiretroviral therapy (cART) has increased significantly; however, this has been accompanied by accelerated biological aging and the emergence of noncommunicable comorbidities. Before the manifestation of cardiometabolic disorders, silent immunometabolic alterations are already underway. In this context, the glutamine-glutamate axis emerges as a potential early indicator of oxidative stress and chronic cellular inflammation. **Objective:** To investigate the dysregulation of glutamine metabolism (glutaminolysis pathway) as an early biomarker of chronic inflammation and immunometabolic risk in PLWH undergoing cART, evaluating its role in the progression toward metabolic comorbidities associated with aging. **Methods:** This is a narrative literature review. The search used the PICo strategy, seeking articles indexed in PubMed, VHL, SciELO, and LILACS, combining the descriptors "HIV" AND "Immunometabolism" AND "Inflammation." Four full-text, peer-reviewed articles published within the last five years were selected after duplicate removal. **Results:** The analysis revealed a progressive pathophysiological continuum in PLWH. Initially, HIV hijacks glutamine uptake to supply energy demands through the tricarboxylic acid cycle and oxidative phosphorylation, optimizing infection in naïve and memory CD4+ T-lymphocyte subsets. Viral progression induces cell death and activation of macrophages and microglia, culminating, together with impaired neurotransmitter clearance, in the pathological release of intracellular glutamate. Chronically, under prolonged cART, a sustained increase in plasma glutamate is observed, accompanied by a reduction in essential amino acids. The persistence of this dysregulation in the glutamine-glutamate axis may act as a biomarker of systemic complications and is closely related to the biochemical basis and progression of metabolic syndrome in PLWH. **Conclusion:** Dysfunction in the glutaminolysis pathway directly reflects the immunometabolic and inflammatory risk in PLWH. Monitoring this axis may serve as an early biomarker for cardiometabolic decline. Furthermore, the modulation or targeted inhibition of glutaminolysis represents a promising target for the management of chronic comorbidities, the development of new therapeutic interventions, and possibly strategies aimed at achieving a functional cure for HIV.
Keywords: HIV; immunometabolism; inflammation.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

140 (3153) HETEROZYGOUS APOB MUTATION PRESENTING AS HYPOBETALIPOPROTEINEMIA WITH MILD HEPATIC DYSFUNCTION: A CASE REPORT

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A 48-year-old, hypertensive, white female patient with a previous diagnosis of mild hepatic steatosis and vitamin D insufficiency presents for evaluation due to significant isolated fatigue. On history, she reports chronic episodes of loose stools, which worsen after ingestion of high-fat foods. The patient denied alcohol consumption, current medication use, or relevant family history. Laboratory tests showed hemoglobin 11,5 g/dL, RDW 15,1%, aspartate aminotransferase 38 U/L, alanine aminotransferase 51 U/L, alkaline phosphatase 63 U/L, and gamma-glutamyl transferase 120 U/L. Total bilirubin was 2.02 mg/dL, with direct bilirubin 0.51 mg/dL and indirect bilirubin 1.51 mg/dL. Lipid profile revealed total cholesterol of 95 mg/dL, triglycerides 24 mg/dL, high-density lipoprotein cholesterol 58 mg/dL, low-density lipoprotein cholesterol (LDL-C) 30 mg/dL, and very-low-density lipoprotein cholesterol 7 mg/dL. Apolipoprotein B was 28 mg/dL. Fat-soluble vitamins A and E showed no abnormalities. Dietary and lifestyle modifications were implemented, and genetic analysis identified a heterozygous pathogenic variant in the APOB gene. Subsequently, complementary ophthalmologic, neurologic, hepatic ultrasonography, and bone mineral densitometry assessments were requested. Hypobetalipoproteinemia refers to a heterogeneous group of genetic disorders characterized by low plasma concentrations of LDL-C and Apo B, which may present in either homozygous or heterozygous forms. Heterozygous APOB variants are usually asymptomatic or present with mild hepatic dysfunction associated with hepatic steatosis. However, approximately 5%–10% of patients with heterozygous APOB-related familial hypobetalipoproteinemia may develop progressive non-alcoholic steatohepatitis and rarely progress to cirrhosis. Clinical manifestations may also include severe malabsorption of fat-soluble vitamins, which can lead to irreversible multisystem complications involving the ocular, skeletal, neuromuscular, and gastrointestinal systems. Management generally consists of low-fat and sugar diet; oral fat-soluble vitamin supplementation; periodic laboratory assessments, lipid profile and liver function tests, along with hepatic ultrasonography in patients with elevated liver transaminases. This case underscores the importance of individualized care and the critical role of integrating lipid profile analysis with the patient's clinical presentation to optimize management and enhance overall quality of life.

Keywords: hypobetalipoproteinemia; heterozygous; hepatic steatosis.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

141 (2994) LIPOPROTEIN X RELATED PSEUDOHYPERCHOLESTEROLEMIA IN PANCREATIC ADENOCARCINOMA

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Case presentation: A 70-year-old woman was admitted for investigation of progressive jaundice, choluria, acholic stools, generalized pruritus, and weight loss. During the evaluation of cholestatic syndrome, imaging studies revealed an ill-defined nodular thickening in the pancreatic head measuring approximately 2.0 × 1.8 cm, associated with biliary tract dilation and obstruction of the common bile duct and the main pancreatic duct. Laboratory tests demonstrated marked cholestasis, with total bilirubin levels of 36.4 mg/dL, as well as severe hypercholesterolemia, including total cholesterol of 1,043 mg/dL, low-density lipoprotein cholesterol (LDL-C) of 938 mg/dL, triglycerides of 400 mg/dL, and high-density lipoprotein cholesterol (HDL-C) of 45 mg/dL. The patient had been taking simvastatin 20 mg/day for several years and had no history suggestive of familial hypercholesterolemia. Considering the marked cholestatic pattern, secondary hypercholesterolemia due to cholestasis was suspected, and no adjustment in lipid-lowering therapy was performed. Instead, biliary decompression was indicated. One month after percutaneous cholecystostomy, a marked improvement in the lipid profile was observed, with total cholesterol decreasing to 115 mg/dL and LDL-C to 42 mg/dL, along with a reduction in total bilirubin to 1.48 mg/dL, confirming the secondary nature of the dyslipidemia. The patient subsequently underwent pancreaticoduodenectomy, and pathological examination revealed moderately differentiated ductal adenocarcinoma of the pancreatic head. Good lipid profile control was maintained during follow-up. **Discussion:** Low-density lipoproteins are beta-lipoproteins primarily cleared by the liver. In cholestatic states, impaired biliary excretion promotes reflux of cholesterol and phospholipids into the plasma, leading to the formation of lipoprotein X, an abnormal lipoprotein rich in cholesterol and phospholipids and poor in triglycerides and apolipoprotein B. Although lipoprotein X is not a true LDL particle, many conventional laboratory methods quantify it as LDL-C, resulting in falsely elevated values. **Final comments:** This case highlights the importance of recognizing severe cholestatic hypercholesterolemia as a reversible metabolic manifestation of biliary obstruction. Awareness of this condition may prevent misdiagnosis as familial dyslipidemia and avoid unnecessary escalation of lipid-lowering therapy.

Keywords: cholestasis; pancreatic adenocarcinoma; hypercholesterolemia.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

142 (2923) MANAGEMENT OF SEVERE HYPERTRIGLYCERIDEMIA IN A PREGNANT PATIENT WITH LIPOPROTEIN LIPASE DEFICIENCY: A CASE REPORT

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Case presentation: A 37-year-old multigravida (G4P2) at 29 weeks of gestation presents severe hypertriglyceridemia, type 2 diabetes (T2DM) on insulin therapy, and hypertension. Physical examination revealed acanthosis nigricans and lower limb lipodystrophy, suggesting severe insulin resistance. Ophthalmological evaluation confirmed mild non-proliferative diabetic retinopathy and grade 1 hypertensive retinopathy. The patient has a history of recurrent acute pancreatitis, including episodes during the prior pregnancies, alongside a strong family history of dyslipidemia. Genetic testing confirmed homozygous pathogenic variants in LPL gene, diagnostic of lipoprotein lipase (LPL) deficiency (p.Pro234Leu, c.701C>T). Her obstetric history is significant for a stillbirth at 20 weeks during a prior hospitalization for glycemic control. Current laboratory tests initially showed TG levels exceeding 6.000 mg/dL. Management began with high-dose Omega-3 (4g/day). Due to persistent severe hypertriglyceridemia (TG 2.612 mg/dL) and the imminent risk of pancreatitis, fenofibrate (160 mg/day) was initiated during the second trimester. **Discussion:** LPL deficiency is a rare autosomal recessive disorder characterized by the inability to hydrolyze circulating triglycerides, leading to chylomicron accumulation. Gestational hypertriglyceridemia is associated with adverse outcomes, including acute pancreatitis (42%, especially if TG ≥1.000 mg/dL), pre-eclampsia, preterm labor, and fetal death, aggravated by secondary causes such as T2DM. In normal singleton pregnancies, triglycerides generally do not exceed 250 mg/dL, although during the third trimester, physiological estrogen elevation typically increases TG levels 2 to 4-fold; in LPL-deficient patients, this spike is catastrophic. Management involves extreme fat restriction (< 20 g/day) and Omega-3 supplementation. Fibrates are generally avoided in the first trimester due to potential teratogenicity, and their use is justified in later trimesters when TG levels are severe and the therapeutic benefit in preventing lethal pancreatitis outweighs potential fetal risks. Plasmapheresis can be considered in pregnant patients with triglycerides > 1,000 mg/dL, despite lifestyle changes and pharmacologic therapy. **Final comments:** Although guidelines suggest fenofibrates for TG ≥ 1.000 mg/dL after the first trimester, there is a profound lack of controlled studies and long-term data regarding fetal adverse effects in extreme gestational cases.

Keywords: familial chylomicronemia; pregnancy; treatment.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

143 (3121) RECURRENT PANCREATITIS IN SEVERE HYPERTRIGLYCERIDEMIA: UNMASKING FAMILIAL CHYLOMICRONEMIA SYNDROME THROUGH GENETIC DIAGNOSIS

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Case presentation: A 23-year-old female patient from Pernambuco presented with triglyceride levels of 2,779 mg/dL and was admitted with acute pancreatitis in December 2024. She had prior hospitalizations in 2020 in her hometown; however, four years later, episodes became more frequent, totaling six hospitalizations over a 6-month period, all with a similar clinical presentation: epigastric pain associated with nausea and vomiting. In February 2025, she started follow-up with an endocrinologist, as triglyceride levels remained elevated despite adherence to a low-fat diet and treatment with fibrates (ciprofibrate 100 mg/day, fenofibrate 200 mg/day) and simvastatin 40 mg/day. Gemfibrozil 1200 mg/day was then initiated as an alternative to previous therapy; however, she developed a new episode of acute pancreatitis in July 2025, with triglyceride levels of 4,131 mg/dL. In addition to recurrent pancreatitis, the patient had type 2 diabetes mellitus, treated with metformin, and emotional lability. Family history was positive for moderate hypertriglyceridemia. Genetic testing identified a compound homozygous mutation in the lipoprotein lipase gene (chr8:19,954,279C>T), confirming the diagnosis of familial chylomicronemia syndrome. **Discussion:** This case illustrates the diagnostic challenges of familial chylomicronemia syndrome, a rare disorder affecting approximately 1–13 individuals per million. Key distinguishing features include early-onset recurrent pancreatitis, higher morbidity and mortality, lack of response to conventional triglyceride-lowering therapies, low apolipoprotein B levels, and absence of secondary causes, all consistent with monogenic familial chylomicronemia syndrome rather than multifactorial chylomicronemia syndrome. Recent studies on APOC3 inhibitors (volanesorsen) represent a paradigm shift, offering the first effective pharmacotherapy beyond dietary restriction. **Final comments:** Familial chylomicronemia syndrome requires a high index of suspicion in patients with severe hypertriglyceridemia and recurrent pancreatitis refractory to standard therapy. Early genetic diagnosis enables appropriate management with strict dietary fat restriction and consideration of emerging APOC3-targeted therapies, potentially preventing life-threatening complications, highlighting the importance of early recognition in young patients with unexplained severe hypertriglyceridemia.

Keywords: familial chylomicronemia syndrome; hypertriglyceridemia; pancreatitis.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

144 (3272) SEVERE HDL DEFICIENCY AND FISH-EYE CORNEAL PHENOTYPE IN THREE WOMEN WITH SUSPECTED INHERITED LCAT-RELATED DISEASE

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Case presentation: Three women aged 52, 62, and 63 years from Rio Grande do Norte, Northeastern Brazil, were evaluated for suspected fish eye disease (FED) after identification of marked bilateral corneal opacity associated with extremely low HDL cholesterol levels. Two patients were sisters born to consanguineous parents, while the third was unrelated but also reported parental consanguinity and family history of renal disease. Corneal abnormalities began in early adulthood in the two older patients, progressing slowly over decades. Despite evident corneal opacity, visual complaints were initially mild, becoming clinically relevant later, mainly as night vision difficulty. One patient required referral for corneal transplantation. The third patient had marked opacity without significant visual symptoms. HDL levels ranged from 3.9 to 5.6 mg/dL, whereas total cholesterol ranged from 80 to 174 mg/dL, LDL from 45 to 128 mg/dL, and triglycerides from 129 to 268 mg/dL, demonstrating profound and disproportionate HDL reduction. Hyperferritinemia and albuminuria were observed in the two related patients, while the third exhibited chronic kidney disease. One patient additionally developed severe degenerative aortic stenosis requiring surgical valve replacement. Molecular analysis is ongoing. **Discussion:** FED is a rare partial LCAT deficiency characterized by severe HDL depletion and progressive corneal cholesterol deposition. Although classically distinguished from familial LCAT deficiency (FLD) by relative preservation of renal function, increasing evidence suggests phenotypic overlap between these entities. The combination of markedly reduced HDL levels, characteristic corneal findings, parental consanguinity, and familial clustering strongly supports partial LCAT deficiency. Corneal opacity disproportionate to visual dysfunction appears to be an important and potentially underrecognized diagnostic clue. Renal abnormalities and cardiovascular involvement observed in this series suggest broader systemic manifestations than traditionally expected in FED. The occurrence of three similar patients from the same geographic region also raises the possibility of a regional founder effect. **Final comments:** FED should be considered in patients presenting with profound isolated HDL reduction and marked corneal opacity, even in the absence of significant visual complaints.

Keywords: LCAT deficiency; severe HDL deficiency; fish-eye corneal opacity.

DYSLIPIDEMIA AND ATHEROSCLEROSIS

145 (3054) SEVERE HYPERTRIGLYCERIDEMIA AND RECURRENT PANCREATITIS REVEALING FAMILIAL CHYLOMICRONEMIA SYNDROME DUE TO COMPOUND HETEROZYGOSITY VARIANTS IN LPL GENE

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Case presentation: A 41-year-old woman with chronic pancreatitis secondary to hypertriglyceridemia was admitted with severe acute pancreatitis. Severe hypertriglyceridemia (6,537 mg/dL) supported a metabolic etiology, and the patient remained hospitalized for 6 months due to multiple complications. Treatment included fat restriction, intravenous insulin with glucose infusion, ciprofibrate, rosuvastatin, and 6 plasmapheresis sessions, with persistent severe hypertriglyceridemia despite therapy. Genetic testing for familial hypertriglyceridemia was performed. Targeted next-generation sequencing identified two variants in the LPL gene in compound heterozygosity: a pathogenic frameshift variant, c.840delG (p.Asn281Metfs*23), predicted to result in premature protein truncation, and a likely pathogenic missense variant, c.373G>A (p.Ala125Thr), previously associated with severe hypertriglyceridemia. **Discussion:** Familial chylomicronemia syndrome (FCS) is a rare autosomal recessive disorder caused by loss-of-function variants in genes involved in lipoprotein lipase activity, leading to severe hypertriglyceridemia and a high risk of recurrent acute pancreatitis. The identification of two pathogenic variants, c.840delG (p.Asn281Metfs*23) and c.373G>A (p.Ala125Thr), confirms the molecular diagnosis and provides insight into the genotype-phenotype correlation in this rare metabolic disorder. In our case, the combination of a null allele with a hypomorphic allele likely results in severely reduced or absent lipoprotein lipase activity, explaining the extreme hypertriglyceridemia and high pancreatitis risk. Our patient's presentation shares similarities with previously reported cases of compound heterozygous lipoprotein lipase deficiency; however, the identification of the c.840delG variant, previously characterized as causing complete loss of function, in combination with the c.373G>A variant, contributes to the expanding genotype-phenotype correlation data for familial chylomicronemia syndrome. **Final comments:** This case underscores the importance of early genetic diagnosis in patients with severe hypertriglyceridemia and recurrent pancreatitis. The identification of pathogenic LPL variants confirms FCS and has direct implications for management, family screening, and eligibility for emerging therapies targeting lipoprotein lipase-independent pathways.

Keywords: familial chylomicronemia syndrome; lipoprotein lipase deficiency; acute pancreatitis.

EXERCISE ENDOCRINOLOGY

146 (3337) "USE OF PEPTIDES AND 'BIOHACKING': A SYSTEMATIC REVIEW ON THEIR EFFICACY AND SAFETY IN PHYSICAL PERFORMANCE AND RECOVERY"

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Introduction: The study of therapeutic peptides, initiated with insulin isolation, has evolved with modern biotechnology, expanding from endocrine and oncological treatments to "biohacking" and human performance enhancement. Despite the growing popularity of these molecules for muscle gain, weight loss, and tissue recovery, recreational and aesthetic use lacks robust scientific evidence to guarantee safety and efficacy in humans. **Objective:** To analyze the available evidence in the scientific literature regarding the use of therapeutic peptides aimed at physical performance and aesthetic purposes, evaluating their pharmacological properties, actual efficacy, and safety profile. **Methods:** A systematic literature review was conducted based on PRISMA 2020 guidelines and registered in PROSPERO. The search was performed in PubMed using specific descriptors combined with terms related to performance and tissue repair. Articles in English and Portuguese published between 2021 and April 25, 2026, were screened and selected by two reviewers. **Results:** The initial search yielded 126 articles, of which 51 were selected after removing duplicates and applying eligibility criteria. Out of the papers selected for review, only 2 are randomized clinical trials, with the majority (32) consisting of animal trials. **Discussion:** The findings demonstrate that peptide therapies present promising properties in tissue and metabolic regulation, although they vary in mechanisms. BPC-157, Thymosin Beta-4, and its active fragment TB-500 stand out for their potent angiogenic, healing, and musculoskeletal regenerative roles. In mitochondrial modulation and cellular metabolic axis, Elamipretide and MOTS-c are active, while GHK-Cu plays a role in tissue remodeling and collagen synthesis. In the field of GH/Ghrelin axis secretagogues, compounds such as Tesamorelin, Sermorelin, KARIs, GHRP-6, and MK-677 show efficacy in protein anabolism, strength gain, and cardio- and nephroprotection. However, recreational use of these substances (such as MK-677) poses severe health risks, such as visceral fat accumulation, dyslipidemia, and severe suppression of the endogenous gonadal axis. **Conclusion:** The debate highlights the gap between promising *in vitro* results and the scarcity of sufficient clinical trials and human studies to ensure the safety of prescribing them for therapeutic and biohacking purposes, in addition to the inherent risks of the gray market and the use of supraphysiological doses.
Keywords: peptides; biohacking; performance.

EXERCISE ENDOCRINOLOGY

147 (3288) ASSESSMENT OF THE ACCURACY AND RELIABILITY OF THREE-DIMENSIONAL SCANNERS IN BODY COMPOSITION ANALYSIS IN ATHLETES: A SYSTEMATIC REVIEW

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Body composition assessment is essential in sports medicine and endocrinology, particularly for monitoring fat mass, fat-free mass, and athletic performance. Although dual-energy X-ray absorptiometry (DXA) is considered the gold standard, its high cost, limited portability, and radiation exposure restrict repeated assessments. Three-dimensional optical scanners (3DO) have emerged as a portable and radiation-free alternative. This systematic review aimed to evaluate the accuracy and reliability of 3DO scanners for athlete body composition compared with DXA and other methods. The review followed PRISMA guidelines. Database searches were performed through October 2024. Eligibility criteria were defined using the PICO framework, including adult athletes assessed with 3DO scanners and compared with DXA, anthropometric methods, and other body composition techniques. Comparisons involved DXA, air displacement plethysmography (ADP), bioelectrical impedance analysis, hydrostatic weighing, and anthropometric measurements. Of 417 identified studies, six published between 2020 and 2024 met the eligibility criteria comprising samples ranging from 9 to 87 participants. Some studies reported promising agreement with DXA and anthropometric methods, while the only study involving elite athletes demonstrated reduced accuracy in body fat percentage estimation. However, results varied across body composition parameters and athlete profiles. One study demonstrated strong reliability for lean mass measurement (ICC = 0.944), whereas another reported greater accuracy for fat-free mass estimation (R^2 = 0.69). Methodological heterogeneity, small sample sizes, and differences in comparison techniques limited direct comparisons between studies. Evidence suggests that 3DO scanners are promising tools for body composition assessment in athletes due to their portability, reproducibility, and absence of radiation exposure. Nevertheless, available studies do not support replacing established reference methods such as DXA. Further studies in larger athletic populations are required to better define the clinical applicability and accuracy of 3DO technology in sports medicine.
Keywords: body-composition; athletes; tridimensional scanner.

EXERCISE ENDOCRINOLOGY

148 (2910) CARDIOVASCULAR EFFECTS OF ANABOLIC-ANDROGENIC STEROID USE

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Introduction: Anabolic-androgenic steroids are synthetic derivatives of testosterone used in specific therapeutic contexts; however, their non-medical use has increased significantly, especially among young men and individuals engaged in resistance training. This increase is associated with aesthetic standards and the pursuit of physical performance, constituting a relevant public health issue. Evidence indicates that these substances, frequently used at doses above therapeutic levels and, particularly, over the long term, may trigger systemic alterations, especially cardiovascular impairment, one of the main causes of associated morbidity and mortality. **Objective:** To analyze the main cardiovascular effects associated with anabolic-androgenic steroid use. **Methods:** This is an integrative literature review conducted through the analysis of studies indexed in PubMed and SciELO, published between 2000 and 2025, including clinical, experimental, and review studies addressing cardiovascular alterations related to the use of these substances. **Results:** The use of anabolic-androgenic steroids is associated with structural and functional alterations of the cardiovascular system, including left ventricular hypertrophy, cardiac remodeling, and systolic and diastolic dysfunction, which may progress to cardiomyopathy. An increased incidence of arrhythmias, arterial hypertension, myocardial infarction, and sudden cardiac death has been observed. These effects result from multifactorial mechanisms, particularly dyslipidemia, characterized by reduced high-density lipoproteins and increased low-density lipoproteins, favoring atherogenesis, in addition to endothelial dysfunction, with reduced nitric oxide bioavailability and increased vascular stiffness. A prothrombotic state is also evident, with increased platelet aggregation and coagulation alterations, raising the risk of thromboembolic events. **Conclusion:** The use of anabolic-androgenic steroids is associated with relevant adverse cardiovascular outcomes, including myocardial dysfunction and an increased risk of cardiovascular events, constituting an important public health issue.

Keywords: anabolic steroids; cardiovascular system; cardiomyopathy.

EXERCISE ENDOCRINOLOGY

149 (2881) EXOGENOUS INSULIN IN EUTROPHIC NON-DIABETIC INDIVIDUALS: METABOLIC EFFECTS AND RISKS

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Introduction: Insulin is essential for energy metabolism and widely used in diabetes treatment. However, its non-therapeutic use among eutrophic non-diabetic individuals has increased, particularly in bodybuilding contexts aiming for anabolic effects. This practice is often driven by non-scientific information on digital platforms. Despite this trend, available evidence regarding safety and efficacy in this population remains limited. **Objective:** To analyze metabolic effects, hypoglycemia risk, and endocrine repercussions associated with exogenous insulin use in non-diabetic individuals. **Methods:** A structured literature review based on the PICO strategy was conducted: non-diabetic individuals (P), exogenous insulin use (I), absence of use (C), and metabolic/endocrine outcomes (O). Studies published between 2005 and 2025 were selected from scientific databases. Inclusion criteria comprised original studies involving non-diabetic populations. Studies not directly addressing this group were excluded. **Results:** Exogenous insulin induces disproportionate hyperinsulinemia, increasing glucose uptake and raising hypoglycemia risk. It stimulates lipogenesis and inhibits lipolysis, favoring fat accumulation. Despite its anabolic role, consistent evidence of increased muscle mass in healthy individuals is lacking. Repeated exposure may lead to insulin resistance and alterations in the GH-IGF-1 axis. The limited number of studies restricts stronger conclusions. **Conclusion:** Exogenous insulin use in non-diabetic individuals lacks consistent scientific support and is associated with metabolic risks. Limited evidence may contribute to unsafe practices, highlighting the need for further research and public awareness.

Keywords: exogenous insulin; hypoglycemia; non-diabetic individuals.

EXERCISE ENDOCRINOLOGY

150 (3325) MULTISYSTEM INVOLVEMENT ASSOCIATED WITH CHRONIC ANABOLIC STEROID AND CORTICOSTEROID USE IN A STRONGMAN ATHLETE: A CASE REPORT

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Case presentation: A 50-year-old strongman athlete with long-term use of anabolic-androgenic steroids and corticosteroids was admitted with progressive dyspnea and hypoxemia. He reported approximately 20 years of exposure to injectable testosterone formulations and prolonged stanozolol use, associated with recurrent corticosteroid injections and frequent use of nonsteroidal anti-inflammatory drugs. Echocardiography demonstrated left ventricular systolic dysfunction with ejection fraction of 36%, left ventricular dilation, and suspected ventricular thrombus. Significant polycythemia was also identified, requiring therapeutic phlebotomy. During hospitalization, the patient developed acute kidney injury associated with inflammatory urinary sediment characterized by hematuria, leukocyturia, and proteinuria, with negative urine culture. Systemic inflammatory response and cutaneous manifestations suggestive of drug-induced skin reaction were also observed. During hospitalization, the patient presented findings compatible with secondary adrenal insufficiency after corticosteroid withdrawal. **Discussion:** Chronic exposure to supraphysiological androgen doses has been associated with cardiomyopathy, erythrocytosis, and thromboembolic events. Concurrent use of corticosteroids and nonsteroidal anti-inflammatory drugs may additionally contribute to renal and endocrine complications. In this case, the coexistence of heart failure, polycythemia, inflammatory renal injury, and probable secondary adrenal insufficiency suggests multisystem toxicity related to prolonged exposure to these substances. **Final comments:** After supportive treatment, anticoagulation, and discontinuation of drug exposure, the patient evolved with clinical improvement and partial recovery of ventricular function. This case highlights the potential multisystem toxicity associated with chronic anabolic steroid and corticosteroid use.

Keywords: anabolic steroids; heart failure; adrenal insufficiency.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

151 (3317) ASSOCIATION BETWEEN POLYENDOCRINE METABOLIC OVARIAN SYNDROME AND METABOLIC/ LIPID PROFILE IN ADULT WOMEN: A SYSTEMATIC REVIEW

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Introduction: Polyendocrine metabolic ovarian syndrome (PMOS) is a common systemic dysfunction in women of reproductive age, with frequent onset during adolescence and confirmed diagnosis according to the Rotterdam criteria. It is characterized by hirsutism, acne, and oligomenorrhea. Despite a prevalence of approximately 15% worldwide, lipid alterations resulting from the hormonal dysregulation associated with PMOS remain underrepresented in the literature. Analysis of lipid fractions, anthropometric data, and glycemic parameters may contribute to a better understanding of its pathophysiology and relationship with metabolic disturbances in affected individuals. **Objective:** To analyze the association between PMOS and the increased risk of lipid profile alterations and metabolic syndrome establishment. **Methods:** A systematic review was conducted following PRISMA guidelines across PubMed, SciELO, and ScienceDirect databases. Eligible studies included adult women with PCOS presenting data on lipid profile and metabolic alterations, published between 2016 and 2025. Eligibility screening was performed using Rayyan software. **Results:** A total of 631 records were identified, with 620 excluded after screening. Following full-text review, 10 studies were included — cross-sectional (n = 5), prospective (n = 2), case-control (n = 2), and observational (n = 1) — encompassing more than 67,000 participants. The majority of studies demonstrated significant lipid profile deterioration in PMOS carriers compared to non-carriers: elevated LDL (98.5 vs. 88.0 mg/dL; $p < 0.001$), reduced HDL (54.5 vs. 63.0 mg/dL; $p < 0.001$), and increased triglycerides (90.5 vs. 66.0 mg/dL; $p < 0.001$). In a cohort of 65,866 participants, dyslipidemia was twice as prevalent in the PMOS group (7.64% vs. 3.48%). Low HDL was the most frequent alteration, present in 77.1% of patients in one of the studies. Metabolic syndrome was identified in 19.7% to 44% of women with PMOS, with 91.67% presenting at least one metabolic alteration at diagnosis. Insulin resistance was present in up to 66.3% of participants. **Conclusion:** The studies demonstrate significant lipid profile deterioration in women with PMOS compared to non-affected individuals, with notable alterations in LDL, HDL, and triglycerides levels. A remarkable prevalence of dyslipidemia and metabolic syndrome was also observed among PMOS carriers. Longitudinal monitoring of these parameters proves relevant in this population given the findings encountered.
Keywords: polycystic ovary syndrome; metabolic syndrome; dyslipidemias.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

152 (2998) ATRIAL FIBRILLATION IN A TRANSGENDER MAN RECEIVING GENDER-AFFIRMING HORMONE THERAPY WITH TESTOSTERONE: A CASE REPORT

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Case presentation: A 34-year-old transgender man receiving gender-affirming hormone therapy (GAHT) with testosterone undecanoate since 2016, under regular outpatient follow-up, presented in October 2024 with an acute episode of atrial fibrillation (AF), requiring hospital admission for arrhythmia management and complementary cardiac workup. Following the event, GAHT was temporarily suspended for etiological investigation. During specialized follow-up, no significant structural heart disease or recurrent arrhythmic events were identified. In April 2025, after cardiological reassessment, testosterone therapy was resumed, with stable clinical evolution and no further acute cardiovascular events to date. **Discussion:** GAHT is a fundamental component of comprehensive healthcare for transgender men and is associated with significant improvements in quality of life, reduction of gender dysphoria, and mental health outcomes. However, the potential cardiovascular effects of testosterone remain under debate. Evidence from studies involving hypogonadal cisgender men suggests an association between androgen therapy and increased incidence of AF and other supraventricular arrhythmias. In transgender men, available data remain scarce and inconclusive. Testosterone exposure may induce metabolic and hemodynamic alterations, including worsening lipid profile, increased hematocrit, and elevated blood pressure, in addition to potential effects on cardiac electrophysiology, possibly related to atrial remodeling and arrhythmic risk. Despite these alterations, observational studies have not demonstrated a consistent increase in major cardiovascular events in this population. This case underscores the importance of baseline cardiovascular risk stratification and longitudinal clinical monitoring during GAHT. **Final comments:** The occurrence of AF in a transgender man receiving testosterone therapy highlights the need for individualized cardiovascular surveillance during GAHT. The identification of arrhythmic events does not necessarily imply permanent discontinuation of hormone therapy, and management should be based on multidisciplinary evaluation, clinical stability, and careful assessment of potential cardiovascular risks in light of the substantial mental health and quality-of-life benefits associated with GAHT.
Keywords: transgender man; testosterone; atrial fibrillation.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

153 (3298) ASSESSMENT OF SERUM PSA LEVELS IN GONADECTOMIZED TRANSGENDER WOMEN RECEIVING ESTROGEN THERAPY

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Introduction: Transgender women (TW) receiving estrogen therapy and/or who have undergone gonadectomy have markedly reduced serum testosterone levels. The decrease in testosterone leads to suppression of prostate function, which is reflected in lower PSA concentrations. Although the risk of prostate cancer (PCa) persists in TW, it is significantly reduced compared with the cisgender male population. **Objective:** To assess total serum PSA levels in a cohort of Brazilian transgender women who have undergone gonadectomy and are receiving estrogen therapy, correlating these levels with clinical and hormonal factors and family history associated with PCa risk. **Methods:** A cross-sectional, retrospective, observational study conducted at a tertiary hospital within the Brazilian Unified Health System. Twenty-one TW aged ≥45 years, who had undergone gonadectomy and were receiving estrogen therapy, were evaluated. Sociodemographic and ethnic data, lifestyle habits, family history of PCa, and laboratory parameters (testosterone, estradiol, LH, FSH, total PSA, and free PSA) were collected. Results were expressed as median and interquartile range [IQR25–IQR75], with $p < 0.05$ considered statistically significant. **Results:** The median age of the TW was 55 years (47–59), duration of estrogen therapy was 30 years (22–36), and time since gonadectomy was 12 years (5–15). Median total PSA: 0.025 ng/mL [0.003–0.032] (male reference value for the 50–59-year age group: up to 3.5 ng/mL). No significant differences in total PSA levels were observed between ethnic groups or according to family history of prostate cancer. However, a positive and statistically significant correlation was observed between total PSA and testosterone ($\rho = 0.462$; $p = 0.040$). **Conclusion:** Gonadectomized transgender women receiving estrogen therapy have markedly reduced total PSA levels, making reference values established for cisgender men inapplicable to the transgender population. The positive correlation between PSA and testosterone suggests a direct influence of hormonal activity on prostate function. These findings reinforce the need to establish population-specific reference ranges and revise prostate cancer screening protocols for this population. Studies with larger sample sizes and longitudinal follow-up will be essential to establish appropriate screening strategies for TW.

Keywords: transgender women; estrogen therapy; gonadectomy; prostate cancer.

154 (3466) COMPARATIVE ANALYSIS OF SURGICAL DESIRES BETWEEN BINARY AND NON-BINARY GENDER DIVERSE INDIVIDUALS AT TWO SPECIALIZED CENTERS IN SALVADOR, BAHIA

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Introduction: Gender-affirming surgeries (GAS) are part of the care provided to gender-diverse individuals. Binary and non-binary individuals may share the desire for these procedures, but differ in their motivations and types of interventions sought. However, there are still few studies systematically comparing these needs, especially among non-binary people. **Objective:** To investigate the profile of desired GAS among non-binary transgender individuals and compare it with that observed among binary transgender individuals followed at a referral center. **Methods:** This is a cross-sectional study conducted at two referral centers in Salvador, Bahia. Interest in GAS was comparatively assessed between binary and non-binary transgender individuals in order to better understand the profile of surgical interests within each group. **Results:** The study included 51 non-binary individuals (15 assigned male at birth and 36 assigned female at birth) and 780 binary transgender individuals (51,2% transgender men, 43,3% transgender women and 5,5% travestis). Among non-binary individuals, 19 participants (37.3%) were unsure about or did not desire GAS. The most desired procedures were masculinizing mastectomy, reported by 21 participants (41.2%), and hysterectomy with oophorectomy, by 6 (11.8%), while metoidioplasty, silicone breast implantation and sex reassignment surgery were desired by 3 (5.9%), 4 (7.8%), and 4 participants (7.8%), respectively. Among binary transgender individuals, the most desired procedures were masculinizing mastectomy, reported by 314 participants (40.3%), silicone breast implantation, by 267 (34.2%), and sex reassignment surgery, by 243 (31.2%). Additionally, 67 participants (8.6%) were unsure about or did not desire surgery, while facial feminization surgery, thyroplasty, and hysterectomy with oophorectomy were desired by 56 (7.2%), 113 (14.5%), and 116 participants (14.9%), respectively. **Conclusion:** The results indicate that interest in gender-affirming surgeries (GAS) differs between binary and non-binary individuals. While non-binary participants more frequently express uncertainty or a lack of desire for surgery, binary individuals show a more defined demand for procedures like mastectomy and sex reassignment surgery. These findings emphasize the need for personalized endocrine care that respects individual transition goals and the diverse needs across the gender spectrum.

Keywords: transgenderism; gender-affirming surgery; non-binary persons.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

155 (3182) DYSAUTONOMIA AND PERSISTENT SINUS TACHYCARDIA FOLLOWING REDUCTION OF LONG-TERM SUPRAPHYSIOLOGICAL TESTOSTERONE EXPOSURE IN A POSTMENOPAUSAL

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Supraphysiological testosterone therapy is increasingly prescribed off-label in postmenopausal women. Chronic exposure to male-range androgen concentrations may induce neuroendocrine adaptation. The cardiovascular consequences of relative androgen reduction in this context remain poorly described. **Case report:** A 63-year-old woman presented with resting and exertional sinus tachycardia, orthostatic and heat-induced presyncope, progressive fatigue, subjective muscle weakness, insomnia, absent libido and nocturnal hot flushes. She had received combined oestrogen, progesterone and testosterone therapy for 14 years for aesthetic purposes. Serial measurements between 2015 and 2021 documented total testosterone persistently within the male reference range, with peak values of 519 ng/dL and 503 ng/dL. Approximately three years prior to presentation, testosterone dosage was reduced, coinciding with progressive onset of symptoms. At evaluation, total testosterone remained elevated for females (104 ng/dL; reference < 63 ng/dL) and free testosterone 70.2 pg/mL (reference 2.4–37 pg/mL), though substantially lower than prior levels. Echocardiography was normal (LVEF 70%). Twenty-four-hour Holter monitoring demonstrated sinus tachycardia (69–160 bpm) with isolated ectopic beats and no complex arrhythmias. Exercise testing showed 7.22 METs without ischaemia. Thyroid and adrenal dysfunction were excluded. Background history included chronic hypotension (90/60 mmHg), recurrent syncope since adolescence, family history of dysautonomia, and prior iliac vein thrombosis treated with stenting. Concomitant medications were atomoxetine 10 mg daily and quetiapine 12.5 mg nightly. **Discussion:** It was interpreted as androgen withdrawal syndrome occurring after prolonged supraphysiological exposure. Sustained male-range androgen concentrations over more than a decade may lead to neuroadaptation; subsequent dose reduction, even to levels still above the female physiological range, may functionally resemble androgen deficiency. Pre-existing constitutional dysautonomia and concomitant medications likely contributed to autonomic instability. **Final comments:** Awareness of this potential presentation is important when evaluating unexplained autonomic symptoms in patients receiving long-term exogenous androgens. Sustained male-range androgen concentrations over more than a decade may lead to neuroadaptation; subsequent dose reduction, may functionally resemble androgen deficiency.

Keywords: testosterone; women; dysautonomia.

156 (3136) EFFECTS OF LIRAGLUTIDE ON MENSTRUAL CYCLICITY AND REPRODUCTIVE HORMONAL OUTCOMES IN WOMEN WITH POLYCYSTIC OVARY SYNDROME: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Polycystic ovary syndrome is a prevalent endocrine disorder characterized by menstrual irregularities, hyperandrogenism, and reproductive dysfunction. Liraglutide, a glucagon-like peptide-1 receptor agonist, has shown metabolic benefits in this population; however, its effects on reproductive outcomes remain undefined. **Objective:** To evaluate the effects of liraglutide on menstrual cyclicity, reproductive endocrine outcomes, and safety in women with polycystic ovary syndrome through a systematic review and meta-analysis. **Methods:** A systematic review and meta-analysis of randomized controlled trials was conducted. Eligible studies included women with polycystic ovary syndrome receiving liraglutide. PubMed, Embase, and the Cochrane Library were systematically searched. Within-arm pre- to post-treatment changes from liraglutide arms were pooled; two trials contributed two liraglutide arms each. Pooled mean differences and proportions were estimated using a random-effects model, with heterogeneity assessed by I^2 statistic and Cochran's Q test. **Results:** Seven randomized controlled trials were included, contributing nine liraglutide arms and 221 women. Pooled within-arm analyses showed significant reductions in luteinizing hormone levels (mean difference -4.02; 95% confidence interval -5.87 to -2.17; I^2 = 30.7%) and total testosterone levels (mean difference -0.31 nmol/L; 95% confidence interval -0.48 to -0.14; I^2 = 71.6%). No significant change was observed for follicle-stimulating hormone (mean difference -0.60; 95% confidence interval -2.27 to 1.07; I^2 = 82.6%). The pooled proportion of menstrual regularization was 76.76% (95% confidence interval 56.51–92.34%; I^2 = 77.8%). Nausea was the most frequent adverse event (44.33%), followed by headache (9.21%) and treatment discontinuation due to adverse events (5.53%). **Conclusion:** Liraglutide was associated with reductions in luteinizing hormone and total testosterone levels and a high proportion of menstrual regularization in women with polycystic ovary syndrome. The treatment was generally well tolerated, although nausea was common. These findings support a potential role for liraglutide in the management of reproductive dysfunction in polycystic ovary syndrome, and larger controlled trials are warranted to confirm these effects.

Keywords: polycystic ovary syndrome; liraglutide; meta-analysis.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

157 (2973) EFFICACY AND SAFETY OF CYPROTERONE ACETATE VERSUS SPIRONOLACTONE IN FEMINIZING HORMONE THERAPY FOR TRANSGENDER WOMEN: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Cyproterone acetate and spironolactone are the antiandrogens most commonly combined with estradiol in feminizing hormone therapy for transgender women. Both aim to suppress serum testosterone into the female reference range, yet which agent offers the best balance of efficacy and safety remains uncertain. **Objective:** To compare efficacy and safety of cyproterone acetate with spironolactone, both combined with estradiol, in transgender women on feminizing hormone therapy. **Methods:** PubMed, Embase, and Cochrane Library were searched for studies comparing cyproterone acetate with spironolactone in transgender women on estradiol-based feminizing hormone therapy. Continuous outcomes were pooled as mean difference (MD) and dichotomous outcomes as risk ratio (RR), with 95% confidence interval (CI) using inverse-variance random-effects models with Wald-type confidence intervals. Heterogeneity was assessed with I^2. **Results:** We included eight studies (two randomized controlled trials), comprising 651 participants, of whom 307 (47.2 %) underwent cyproterone acetate therapy. Cyproterone acetate was associated with a significantly increase in the number of transgender women achieving female testosterone reference range (RR 3.03; 95% CI: 1.60–5.75; $P < 0.001$; $I^2 = 81.3\%$) and a significantly decrease in total testosterone (MD -15.75 nmol/L; 95% CI: -30.45 to -1.05; $P = 0.036$; $I^2 = 96.3\%$) and luteinizing hormone (LH) values (MD –1.91 IU/L; 95% CI: –2.74 to –1.08; $P < 0.001$; $I^2 = 0\%$). Also, cyproterone acetate significantly increased prolactin levels (MD 211.27 mIU/L; 95% CI: 84.06–338.48; $P = 0.001$; $I^2 = 90.5\%$) and lower high-density lipoprotein cholesterol (MD –4.46 mg/dL; 95% CI: –7.36 to –1.56; $P = 0.003$; $I^2 = 0\%$) compared to spironolactone. Serum potassium levels were lower in patients using cyproterone acetate (MD -0.10 mEq/L; 95% CI: -0.16 to -0.03; $P = 0.003$). No significant differences were observed between groups for estradiol, follicle-stimulating hormone, sex hormone binding globulin, alanine aminotransferase, or systolic blood pressure. **Conclusion:** Cyproterone acetate showed greater efficacy in lowering total testosterone, achieving female testosterone range, and suppressing LH in transgender women on estradiol-based feminizing hormone therapy, with a significantly increase in prolactin levels and lower in high-density lipoprotein cholesterol and potassium levels compared to spironolactone. These findings can support individualized antiandrogen choice based on patient comorbidities.

Keywords: transgender persons; cyproterone acetate; spironolactone.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

158 (3566) EFFICACY OF GLP-1 RECEPTOR AGONISTS IN REPRODUCTIVE OUTCOMES OF POLYENDOCRINE METABOLIC OVARIAN SYNDROME TREATMENT: A SYSTEMATIC REVIEW

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Introduction: Polyendocrine metabolic ovarian syndrome (PMOS) encompasses multiple interconnected endocrine abnormalities related to neuroendocrine, metabolic and reproductive pathways. Insulin resistance and compensatory hyperinsulinemia play a key role in ovarian steroidogenesis, contributing to hyperandrogenism, ovulatory dysfunction and infertility. In this context, the impact of PMOS on reproductive outcomes is significant. GLP-1 receptor agonists emerge as a promising strategy for their action in improving insulin resistance and metabolic profile. However, it is still essential to clarify its effectiveness on reproductive outcomes in women with PMOS. **Objective:** To evaluate the efficacy of GLP-1 receptor agonists in improving reproductive outcomes in women diagnosed with PMOS. **Methods:** This systematic review was conducted with searches in the PubMed, Web of Science and EMBASE databases, using a strategy of Boolean descriptors and operators related to the topic. In accordance with PRISMA guidelines, randomized controlled trials were included that evaluated reproductive outcomes associated with the use of GLP-1 receptor agonists in women with PMOS. Studies without female population with PMOS diagnosed by validated criteria, without evaluation of reproductive outcomes, as well as reviews, conference summaries and duplicates were excluded. The methodological quality and risk of bias of the included studies were evaluated using RoB 2 and ROBINS 1. **Results:** After duplicate removal, 985 records were screened, of which 8 studies met the predefined inclusion criteria. Overall, 752 women with PMOS, predominantly overweight or obese, treated with GLP-1 receptor agonists, either alone or in combination with metformin, were analyzed. Reductions were observed in total testosterone, free testosterone, free androgen index (FAI), LH, and the LH/FSH ratio, along with increased SHBG levels. Menstrual regularization, recovery of ovulatory cycles, and higher rates of spontaneous conception and pregnancy were also reported in some studies, especially when combined with metformin. **Conclusion:** GLP-1 receptor agonists demonstrate potential benefit in reproductive outcomes in women with PMOS, associated or not with metformin. Significant improvement in clinical parameters, especially reduction of hyperandrogenic markers, improvement of menstrual regularity and recovery of ovulatory cycles was observed, showing itself as a promising therapeutic strategy for this population.

Keywords: SOMP; GLP-1 receptor agonists; systematic review.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

159 (3274) EFFICACY, SAFETY, AND CLINICAL PERSPECTIVES OF FEZOLINETANT FOR MENOPAUSAL VASOMOTOR SYMPTOMS: A SYSTEMATIC REVIEW BASED ON CLINICAL AND REGULATORY EVIDENCE

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Introduction: Vasomotor symptoms (VMS), including hot flashes and night sweats, affect up to 80% of women during the menopausal transition and may persist for several years, significantly impairing sleep quality and daily functioning. Fezolinetant, a selective neurokinin 3 (NK3) receptor antagonist, represents a non-hormonal therapeutic innovation targeting KNDy neurons in the hypothalamus, restoring thermoregulatory balance. **Objective:** To evaluate the efficacy, safety profile, and clinical applicability of fezolinetant in the management of menopausal vasomotor symptoms based on clinical trial evidence and regulatory data. **Methods:** A systematic review was conducted according to PRISMA guidelines. Searches were performed in major biomedical databases and regulatory agency repositories (including PubMed, Web of Science, Embase, FDA, EMA, TGA, and MHRA) up to 2026. A total of 29 records were initially identified. After screening titles and abstracts, studies were excluded due to duplication, lack of relevance to NK3 antagonism, or absence of clinically relevant outcomes. Sixteen studies were included in the final qualitative synthesis, comprising phase 3 randomized clinical trials (SKYLIGHT 1, 2, and 4; DAYLIGHT studies), systematic reviews, and regulatory safety reports. **Results:** Phase 3 clinical trials demonstrated that fezolinetant 45 mg/day significantly reduces the frequency and severity of vasomotor symptoms compared with placebo ($p < 0.001$), with measurable improvement as early as the first week of treatment and sustained effects at 4 and 12 weeks. Improvements in sleep quality were observed using validated PROMIS sleep disturbance scales, without sedative effects. The most commonly reported adverse events included headache (8.8%), abdominal pain (4.3%), and diarrhea. Elevations in hepatic transaminases greater than three times the upper limit of normal occurred in approximately 2.3% of patients. Post-marketing surveillance has reported rare cases of clinically significant liver injury, leading to recommendations for baseline and periodic liver function monitoring during treatment. **Conclusion:** Fezolinetant is an effective non-hormonal option for the treatment of moderate-to-severe vasomotor symptoms in menopause. Its clinical use is supported by robust randomized evidence but requires careful hepatic monitoring and attention to potential CYP1A2-mediated drug interactions.

Keywords: hot flashes; menopause; neurokinin-3 receptor antagonists.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

160 (2934) ENVIRONMENTAL-DOSE METHYLPARABEN MODULATES HYPOTHALAMIC–PITUITARY–GONADAL AXIS GENE EXPRESSION AND TESTICULAR CYTOARCHITECTURE

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This study investigated the effects of subchronic exposure to methylparaben (MP) on morphofunctional parameters of the hypothalamic-pituitary-gonadal (HPG) axis in male Wistar rats. Animals were allocated into the following groups: Control (0.1% DMSO), MP3 (3 µg/kg/day), MP30 (30 µg/kg/day), and MP300 (300 µg/kg/day), and exposed orally (gavage) for 14 days. Body weight, food intake, hormone levels, and testicular morphometry were evaluated, as well as gene expression in the hypothalamus (GnRH), pituitary (LHβ and FSHβ), and testes (LHr, FSHr, inhibin A, inhibin B, ABP, ZO-1, and ERα). Sperm parameters, including concentration and motility, were also assessed. Our results showed a significant increase in body weight and food intake in animals exposed to the highest dose. At the central level, a downregulation of hypothalamic GnRH expression was observed at higher doses, whereas an upregulation was detected at the lowest dose, accompanied by significant changes in pituitary LHβ and FSHβ expression at lower doses. In the testes, inhibin A and inhibin B expression was downregulated at all exposure levels, as was ABP expression. ERα and ZO-1 expressions were also modulated, showing distinct patterns across MP-exposed groups. Histopathological analysis revealed disorganization of the seminiferous epithelium, interstitial edema, and sloughed germ cells within the tubular lumen in MP-treated groups. Morphometric analysis demonstrated a significant reduction in seminiferous epithelium height and tubular diameter in animals exposed to higher doses. Serum hormone assays indicated alterations in testosterone and estradiol levels. No significant changes were observed in sperm concentration or motility at any dose. In conclusion, these findings demonstrate that MP exposure, even at low doses, can disrupt the HPG axis in male rats, impairing hormonal homeostasis and testicular structural organization.

Keywords: endocrine-disruptors; hormones; male reproductive.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

161 (3573) HIGH-SENSITIVITY C-REACTIVE PROTEIN IDENTIFIES RESIDUAL INFLAMMATORY RISK DURING TRANSITION TO LONG-ACTING TESTOSTERONE THERAPY IN MEN WITH CONGENITAL HYPOGONADOTROPIC HYPOGONADISM

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Introduction: Men with congenital hypogonadotropic hypogonadism present increased cardiometabolic vulnerability related to chronic androgen deficiency, adverse adiposity, and low-grade inflammation. High-sensitivity C-reactive protein (hs-CRP), recognized as a cardiovascular risk enhancer, may represent a complementary translational biomarker during androgen replacement therapy. **Objective:** To evaluate hs-CRP as a biomarker of residual inflammatory risk in men with congenital hypogonadotropic hypogonadism undergoing transition from testosterone cypionate to more stable testosterone formulations, integrating lipid profile, visceral adiposity, and physical activity. **Methods:** Prospective longitudinal study including 37 adult men with congenital hypogonadotropic hypogonadism previously receiving intramuscular testosterone cypionate, reassessed after 6 months following transition to transdermal testosterone gel or intramuscular testosterone undecanoate. hs-CRP, total cholesterol, HDL-C, LDL-C, triglycerides, non-HDL cholesterol, bioimpedance-derived visceral adiposity, and physical activity were evaluated. The primary endpoint was hs-CRP ≥2 mg/L at 6 months. The main analysis used Firth penalized logistic regression. **Results:** Among 36 participants with complete hs-CRP data, median hs-CRP remained globally stable from baseline to 6 months: 1.02 vs. 0.98 mg/L. The proportion of participants with hs-CRP ≥2 mg/L was 27.8% at baseline and 33.3% at 6 months. Numerical reductions were observed in LDL-C ≥130 mg/dL, triglycerides ≥150 mg/dL, and non-HDL cholesterol ≥160 mg/dL. In Firth penalized logistic regression, baseline hs-CRP ≥2 mg/L independently predicted residual inflammatory risk at 6 months: OR 5.55; 95% CI 1.26–24.42; p = 0.023, irrespective of testosterone formulation, physical activity, and visceral adiposity. Baseline hs-CRP showed good discrimination for residual inflammation, with an AUC of 0.776. **Conclusion:** In men with congenital hypogonadotropic hypogonadism, elevated baseline hs-CRP identifies a persistent residual inflammatory-risk phenotype during transition to more stable testosterone replacement strategies. These findings support hs-CRP as a translational biomarker complementary to LDL-C and non-HDL cholesterol for individualized cardiometabolic stratification during androgen replacement therapy.

Keywords: congenital hypogonadotropic hypogonadism; residual inflammatory risk; high-sensitivity C-reactive protein.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

162 (3491) HORMONE THERAPY IN THE TRANSGENDER HEALTHCARE PROCESS WITHIN BRAZIL'S PUBLIC HEALTH SYSTEM: AN EPIDEMIOLOGICAL ANALYSIS OF NATIONAL OUTPATIENT CARE PRODUCTION

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Introduction: Access to healthcare for transgender people in Brazil still faces structural barriers despite the institutionalization of the Transgender Healthcare Process (THP) within the Unified Health System (SUS). Analyzing outpatient service provision related to hormone therapy may help identify regional inequalities in access to specialized care. **Objective:** To analyze the temporal, regional, and demographic distribution of outpatient care related to hormone therapy within the THP in Brazil from 2016 to 2025. **Methods:** Retrospective ecological time-series study using SIA/SUS/Datasus data. Procedures classified as hormone therapy within the THP were analyzed by region of residence, age group, and sex recorded in the administrative database. Absolute frequencies and rates per 1,000,000 inhabitants were calculated. **Results:** From 2016 to 2025, 47,130 outpatient hormone therapy procedures were recorded in the SUS. The national rate increased from 0.78 to 50.71 per 1,000,000 inhabitants, indicating substantial service expansion. However, growth was regionally uneven. The South accounted for 53.1% of records (25,039), reaching 155.63 per 1,000,000 inhabitants in 2025, while the North recorded only 8 procedures in the entire period, with 0.11 per 1,000,000 inhabitants in the final year, suggesting marked territorial asymmetry in access to specialized care. Procedures were concentrated among young adults, especially ages 25–29 (13,314), 20–24 (9,694), and 30–34 (8,349), with progressively lower frequencies in older age groups. Sex recorded in the administrative database showed regional and temporal heterogeneity, requiring cautious interpretation. **Conclusion:** There was a marked expansion in outpatient care related to hormone therapy within the THP in the SUS over the past decade; however, this expansion occurred unevenly across Brazil. The regional concentration of records, especially when contrasted with the near absence of procedures in the North, suggests inequalities in access to specialized care, persistent structural barriers, and possible centralization of care in referral centers. These findings reinforce the need for decentralization and the equitable expansion of healthcare services for the transgender population within the SUS.

Keywords: transgender persons; outpatient care; gender-affirming hormone therapy.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

163 (3523) HORMONE-RELATED ADVANCED DILATED CARDIOMYOPATHY FOLLOWING UNSUPERVISED FEMINIZING HORMONE THERAPY IN A TRANSGENDER WOMAN: A CASE REPORT

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Case presentation: A 38-year-old transgender woman living with HIV on stable antiretroviral therapy was admitted with advanced nonischemic dilated cardiomyopathy and cardiogenic shock, potentially related to unsupervised feminizing hormone therapy. Previous cardiac magnetic resonance showed left ventricular ejection fraction (LVEF) of 15% with a nonischemic pattern. She had undergone cardiac resynchronization therapy with defibrillator implantation and had a history of pulmonary embolism. On admission, she presented with dyspnea at minimal exertion and at rest, respiratory failure, and cardiogenic shock, requiring intubation, norepinephrine, and dobutamine; lactate was 12.3 mmol/L. Echocardiography showed LVEF of 18%, diffuse left ventricular hypokinesia, marked chamber enlargement, mild pulmonary hypertension, and moderate mitral and tricuspid regurgitation. Etiologic investigation excluded ischemic/coronary disease, infiltrative cardiomyopathies, alcoholic cardiomyopathy, cardiotoxic drug exposure, and other infectious etiologies. HIV-related cardiomyopathy was considered unlikely because viral load was undetectable. During reassessment, she reported prior use of transdermal and injectable hormones without medical supervision. In the absence of another established etiology, hormone-related dilated cardiomyopathy secondary to unsupervised sex hormone use was considered the most likely diagnosis. **Discussion:** The course was complicated by acute kidney injury, severe hyponatremia, recurrent low-output and congestive episodes, and suspected intra-abdominal infection. Despite optimized treatment, advanced heart failure remained refractory, and proportional palliative care was instituted. This case highlights unsupervised sex hormone use as a potentially underrecognized cause of severe ventricular dysfunction, decompensated heart failure, and cardiogenic shock. **Final comments:** This report reinforces the importance of excluding conventional etiologies of heart failure and investigating nonprescribed hormone exposure in unexplained nonischemic cardiomyopathy. Misuse of exogenous sex hormones may represent a relevant and preventable cause of severe cardiac dysfunction, supporting specialized endocrine follow-up and cardiovascular monitoring.
Keywords: female endocrinology; andrology; transgender health.

164 (3406) IMPACT OF HORMONE THERAPY INITIATION TIMING ON CARDIOVASCULAR OUTCOMES IN POSTMENOPAUSAL WOMEN: A SYSTEMATIC REVIEW

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Introduction: Menopause is associated with hormonal and metabolic changes that increase cardiovascular risk due to reduced estrogen levels and its protective effects on the endothelium, lipid metabolism, and inflammatory response. Menopausal hormone therapy (MHT) has been studied for climacteric symptom relief and possible cardiovascular risk modulation. Evidence suggests cardiovascular outcomes vary according to MHT initiation timing, per the "timing hypothesis." Therefore, systematic analysis of the impact of hormone therapy timing on cardiovascular outcomes in postmenopausal women is necessary. **Objective:** To evaluate the impact of hormone therapy initiation timing on cardiovascular outcomes in postmenopausal women. **Methods:** Systematic review per PRISMA guidelines, using PubMed, Embase, and Cochrane. Included: randomized controlled trials evaluating systemic hormone therapy (estrogen alone or combined with progestogen), comparing early versus late initiation, with cardiovascular outcomes. Excluded: studies without temporal stratification, exclusive local therapies, absence of cardiovascular outcomes, and non-original publications. Search yielded 75 articles; after deduplication and criteria application, 3 studies were selected. **Results:** Early initiation showed annual CIMT progression of 0.0044 mm/year versus 0.0078 mm/year in placebo, suggesting vascular benefit. For coronary heart disease, women < 10 years since menopause showed HR 0.76 (95% CI: 0.50–1.16), those at 10–19 years showed HR 1.10 (95% CI: 0.84–1.45), and those at ≥20 years showed HR 1.28 (95% CI: 1.03–1.58). For stroke, HRs were 1.11, 1.22, and 1.18, respectively, without consistent risk reduction according to timing. Cardiovascular mortality showed HR 1.00 (95% CI: 0.92–1.08), without benefit. Findings suggest possible association between early MHT and favorable cardiovascular profile, especially regarding coronary risk and subclinical atherosclerosis. **Conclusion:** This review corroborates the timing hypothesis, suggesting early initiation (<60 years or <10 years since menopause) may associate with lower coronary risk versus late initiation. Stroke risk remained elevated, with no consistent long-term cardiovascular mortality benefit. Thus, MHT indication remains primarily for vasomotor symptom management in early menopausal transition, requiring individualized risk stratification, reiterating its formal contraindication for cardiovascular disease prevention.
Keywords: hormone replacement therapy; postmenopause; cardiovascular diseases.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

165 (3370) IMPACT OF PROGESTOGEN-BASED HORMONAL CONTRACEPTIVES ON METABOLIC RISK MARKERS IN WOMEN: A SYSTEMATIC REVIEW

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Introduction: Progestogen-containing hormonal contraceptives are widely used for pregnancy prevention and include oral, injectable, and implantable formulations. However, these contraceptives may alter lipid and glucose metabolism, potentially increasing cardiometabolic risk. Therefore, evaluating the metabolic safety of these formulations is essential. **Objective:** To evaluate the effects of progestogen-containing hormonal contraceptives on metabolic risk markers in women of reproductive age. **Methods:** Systematic review conducted according to PRISMA guidelines and registered in PROSPERO (CRD420261396404), with searches performed in PubMed, Cochrane Library, Scopus, and LILACS databases. Randomized clinical trials and observational studies published within the last ten years were included. Eligible studies involved women aged 15–49 years without established cardiovascular or metabolic disease using progestogen-containing hormonal contraceptives. Evaluated outcomes included total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, fasting glucose, and insulin. Risk of bias was assessed using the RoB 2 tool and the Newcastle-Ottawa Scale. **Results:** Five studies were included, comprising two randomized clinical trials and three observational studies, totaling 839 participants. Increased triglyceride levels were the most frequent finding, particularly among users of combined oral contraceptives and depot medroxyprogesterone acetate (DMPA). Increased LDL-cholesterol and reduced HDL-cholesterol were also associated with DMPA use. Formulations containing drospirenone increased HDL-cholesterol and reduced LDL-cholesterol compared with levonorgestrel-containing contraceptives. Users of etonogestrel implants presented higher total cholesterol and LDL-cholesterol levels compared with combined oral contraceptive users. Glycemic findings were heterogeneous, with no consistent clinically relevant alterations. **Conclusion:** Progestogen-containing contraceptives mainly affected the lipid profile, especially among users of combined oral contraceptives and depot medroxyprogesterone acetate, whereas drospirenone-containing formulations showed a more favorable metabolic profile. These effects depend on the type of progestogen and its association with ethinylestradiol, with more androgenic formulations associated with increased cardiovascular risk. However, the limited number of studies and methodological heterogeneity restrict the generalizability of the findings.
Keywords: hormonal contraception; progestins; lipid metabolism.

166 (3084) IMPACT OF PROLONGED USE OF PUBERTY BLOCKERS AND GENDER-AFFIRMING HORMONE THERAPY ON BONE MINERAL DENSITY: A SYSTEMATIC REVIEW

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Introduction: Gender-affirming hormone therapy (GAHT), including pubertal blockers and sex steroids, is used in the management of gender incongruence. However, these interventions may affect bone mineral density (BMD), particularly during adolescence, a critical period for peak bone mass acquisition. **Objective:** To evaluate the impact of prolonged use of pubertal blockers and GAHT on BMD in transgender individuals. **Methods:** This systematic review followed PRISMA 2020 guidelines and was structured using the PICO strategy: transgender individuals with gender dysphoria or gender incongruence; intervention with GnRH analogs and/or GAHT; comparison with baseline values, cisgender controls, or normative data; and outcomes related to BMD, Z-scores, T-scores, trabecular bone score, bone turnover markers, osteopenia, osteoporosis, and fragility fractures. Searches were performed in PubMed/MEDLINE and ScienceDirect, and complemented by Google Scholar, using descriptors related to transgender persons, hormone therapy, GnRH agonists, bone density, and osteoporosis. Studies published from 2021 to 2026, in English, Portuguese, or Spanish, were included. A total of 1,210 records were identified, and 8 met the eligibility criteria. Evidence quality was assessed using GRADE, and risk of bias was evaluated with tools appropriate to each study design. **Results:** GnRH analogs in transgender youth were associated with reduced bone mass gain, with lumbar Z-score decreases from –0.15 to –0.84 and a negative correlation between suppression duration and BMD ($r = –0.5$). During GAHT, individuals assigned male at birth showed dose-dependent BMD recovery with estrogen, especially when started before age 20, increasing lumbar BMD from 0.97 to 1.02 g/cm^2. In individuals assigned female at birth, testosterone generally maintained long-term bone density, although early focal reductions in femoral neck BMD were reported during the first year. **Conclusion:** Pubertal suppression with GnRH analogs may reduce bone gain during adolescence, while GAHT may promote partial recovery or stabilization of BMD. These effects vary according to treatment duration, hormone used, age at initiation, and sex assigned at birth, reinforcing the need for longitudinal bone health monitoring in this population.
Keywords: bone mineral density; gender-affirming hormone therapy; puberty blocking.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

167 (3448) IMPACT OF SUBCUTANEOUS GESTRINONE IMPLANT ON LIPID PROFILE AND INSULIN RESISTANCE IN WOMEN OF REPRODUCTIVE AGE: A SYSTEMATIC REVIEW

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Introduction: Subcutaneous hormonal implants have increased in clinical practice, including for indications beyond classical gynecological recommendations. Gestrinone, a synthetic steroid derived from 19-nortestosterone with androgenic and anabolic properties, is used in estrogen-dependent conditions. However, uncertainties remain regarding its metabolic safety, particularly concerning lipid profile and insulin resistance in reproductive-aged women. **Objective:** To evaluate, through a systematic review, the impact of subcutaneous or implantable gestrinone on lipid profile and insulin resistance in women of reproductive age. **Methods:** This review followed PRISMA guidelines (PROSPERO: CRD420261369595). Eligible studies evaluated reproductive-aged women without prior diabetes or dyslipidemia exposed to subcutaneous/implantable gestrinone compared to unexposed controls, other hormonal therapies, or pre- versus post-treatment assessments. Outcomes focused on lipid profile changes and insulin resistance markers. Searches were conducted in PubMed/MEDLINE, Embase, and LILACS. Screening was performed by two independent reviewers. Due to methodological heterogeneity, a narrative synthesis was executed. **Results:** Evidence was limited, heterogeneous, and based on small samples with short follow-up. Findings suggest unfavorable lipid changes, mainly a marked reduction in HDL-c and increases in LDL-c and apolipoprotein B, indicating a more atherogenic profile. Studies reported HDL-c reductions of 25% to 40% after six months of exposure. In La Marca *et al.*, LDL-c increased from 115 ± 25 mg/dL to 138 ± 45 mg/dL, while HDL-c decreased from 58 ± 15 mg/dL to 33 ± 19 mg/dL after six months ($p < 0.05$). Data on insulin resistance (fasting glucose, insulin, HOMA-IR) were scarce and insufficient. **Conclusion:** Gestrinone may be associated with unfavorable lipid metabolism changes, especially reduced HDL-c and increased LDL-c. Current evidence is insufficient to draw definitive conclusions regarding its metabolic safety, particularly in subcutaneous implants. Rigorous prospective studies are needed to clarify long-term effects.
Keywords: gestrinone; hormonal implants; lipid profile.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

168 (3165) IMPAIRED REDOX ACTIVITY CAUSED BY NNT DEFICIENCY IMPAIRS GONADAL FUNCTION AND SPERMATOGENESIS

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Introduction: Mitochondrial Nicotinamide nucleotide transhydrogenase (NNT) regulates cellular redox homeostasis. Pathogenic variants in the NNT gene result in adrenal insufficiency. Abnormal spermatogenesis has been reported in NNT-deficient patients, but the underlying mechanism remains unknown. **Objective:** To investigate the NNT role in the testis by integrating clinical, in silico, and *in vitro* functional data. **Patient, materials and methods:** Clinicopathological features of an NNT-deficient patient. NNT expression in control fetal and adult testes was assessed by single-cell RNA-seq and IHC. *In vitro*, functional studies were performed in TM3 and MA10 testicular cell lines following NNT siRNA knockdown. Mitochondrial activity and redox status were assessed using Mitotracker and DCFDA assays. Cholesterol metabolism evaluated by NBD-cholesterol uptake and intracellular cholesterol quantification. Steroidogenic capacity assessed by HSD3B1 expression and progesterone production. **Results:** An 18-year-old male with familial glucocorticoid deficiency and lack of adrenarche caused by the homozygous NNT p.G866D variant exhibited normal pubertal development and normal LH, FSH, and testosterone plasma levels. However, his semen analysis demonstrated markedly reduced progressive motility and abnormal sperm morphology. In the control fetal testis, NNT was expressed across germ, Sertoli, and Leydig cells. Stronger NNT staining (IHC) was observed in Leydig cells. In adult control testis, NNT expression was maintained throughout spermatogenesis, including spermatogonia, spermatocytes, and spermatids. UMAP visualization confirmed broad distribution across cellular populations. NNT staining (IHC) was more prominent in spermatogonia within the seminiferous tubules. *In vitro*, NNT silencing in testicular cell lines led to redox imbalance, as evidenced by increased reactive oxygen species levels ($p = 0.002$) and enhanced mitochondrial labeling ($p < 0.0001$). Reduced cholesterol uptake ($p < 0.0001$) and intracellular cholesterol content ($p = 0.0042$), accompanied by decreased HSD3B1 expression ($p = 0.0004$) and progesterone production ($p < 0.0001$), indicate impaired steroidogenesis. **Conclusion:** NNT redox activity is essential for mitochondrial and steroidogenic homeostasis in testicular cells, and NNT deficiency particularly affects spermatogenesis. These findings underscore the importance of lifelong monitoring of the gonadal function in NNT-deficient patients.
Keywords: testis; NNT; redox function.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

169 (3108) INFLUENCE OF THE HORMONAL AXIS ON THE PATHOPHYSIOLOGY AND PROGRESSION OF LIPEDEMA: A SCOPING REVIEW

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Introduction: Lipedema is a chronic and progressive subcutaneous adipose tissue disorder, affecting approximately 11% of the global female population. It is characterized by symmetrical and bilateral fat accumulation in the limbs, often associated with neuropathic pain, capillary fragility, and orthostatic edema. The clear female prevalence and the exacerbation of symptoms during hormonal transitions — such as puberty, pregnancy, and menopause — support the hypothesis of hormonal dysregulation as a central pathophysiological pillar. **Objective:** To map scientific evidence regarding hormonal influence in lipedema pathogenesis, focusing on receptor signaling mechanisms, enzymatic dysfunctions, and potential targets for estrogen suppression therapies. **Methods:** A scoping review conducted according to the JBI framework and PRISMA-ScR guidelines. The PCC strategy was utilized (Population: women with stages I-III lipedema; Concept: hormonal axis and estrogen suppression; Context: mechanistic and clinical evidence). Searches were performed in PubMed, Embase, Scopus, LILACS, Cochrane, and Web of Science (2019–2026). Selection was managed by six independent reviewers via the Rayyan platform in a double-blind system. **Results:** Nine relevant studies were included, featuring transcriptomic and molecular analyses. Data confirms hormonal influence: 58.8% of patients reported clinical worsening after using hormonal contraceptives, while 15.1% associated disease onset with these medications. Molecularly, a pathological inversion in the estrogen receptor ratio was evidenced, with a predominance of ERβ over ERα in adipose tissue. The AKR1C2 enzyme demonstrated a causative role through mutations or overexpression in 24% of samples, accelerating dihydrotestosterone (DHT) inactivation and removing the androgenic inhibitory effect on adipogenesis. Additionally, local aromatase upregulation and PPARγ-mediated pro-adipogenic pathways were observed, providing lipedema tissue with a molecular signature distinct from conventional obesity. **Conclusion:** Lipedema exhibits a strong dependence on estrogenic dysregulation involving inflammatory, metabolic, and genetic pathways. Identifying biomarkers such as AKR1C2 and receptor ratios paves the way for promising targeted therapies. However, the lack of clinical trials and methodological heterogeneity highlight the urgent need for translational research to consolidate new endocrine management strategies.
Keywords: lipedema; estrogens e receptors; estrogen.

170 (3461) LIFETIME SUICIDE ATTEMPTS AMONG GENDER-DIVERSE INDIVIDUALS ATTENDED AT TWO SPECIALIZED CLINICS IN SALVADOR, BRAZIL

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Introduction: Transgender people experience high levels of minority stress, impacting quality of life and mental health. Adverse childhood experiences, such as verbal, physical, and sexual violence, may potentiate psychological distress. In this context, suicidal behavior may emerge from the interaction between social, psychological, and traumatic vulnerabilities. **Objective:** To identify sociodemographic, clinical, and quality-of-life factors independently associated with lifetime suicide attempts in transgender individuals. **Methods:** Observational cross-sectional study with transgender individuals attended at two referral centers in Bahia. The outcome was self-reported lifetime suicide attempt. Hierarchical multivariate logistic regression was performed in five sequential blocks: 1) sociodemographic variables (education, race, marital status, physical activity); (2) gender transition-related variables (gender identity, age of identification, hormone use, satisfaction with body modifications); (3) behavioral factors (sexual abuse, illicit drug use, smoking); (4) mental health and clinical history (ACE score modeled continuously, previous depression, previous anxiety); and (5) quality of life (WHOQOL-Bref: general quality of life, frequency of negative feelings, acceptance of physical appearance). Missing data were handled by multiple imputation (m = 20). **Results:** 769 participants (48.4% trans men, 39.8% trans women, 5.5% travestis, 6.4% non-binary). Lifetime prevalence of suicide attempt was 35.0% (n = 269), with no significant difference across gender identities (p = 0.5). After adjustment, five independent determinants were identified: previous depression (OR = 3.64; 95% CI: 2.33–5.67; p < 0.001), sexual abuse (OR = 1.99; 95% CI: 1.37–2.90; p < 0.001), smoking (OR = 1.86; 95% CI: 1.24–2.80; p = 0.003), ACE score per additional point (OR = 1.14; 95% CI: 1.06–1.24; p = 0.001), and general quality of life, where each one-point improvement reduced the odds by 34% (OR = 0.66; 95% CI: 0.49–0.90; p = 0.008). Gender identity, hormone therapy, and sociodemographic variables were not independently associated with the outcome. Model calibration was adequate (Hosmer-Lemeshow p = 0.16; Nagelkerke R^2 = 0.311). **Conclusion:** One-third of participants reported lifetime suicide attempts. Suicide attempts were associated with psychosocial vulnerability and violence exposure, rather than gender identity or hormone therapy, reinforcing the role of cumulative social and mental health stressors in transgender care.
Keywords: transgender; suicide attempt; epidemiologic factors.

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171 (3145) LONG-TERM BONE MINERAL DENSITY OUTCOMES AND BONE TURNOVER MARKERS IN TRANSGENDER MEN RECEIVING TESTOSTERONE THERAPY: A SYSTEMATIC REVIEW

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Introduction: Gender-affirming hormone therapy for transgender men aims to achieve male-range testosterone levels for virilization. The skeletal impact is mediated by the direct anabolic action of the androgen and its aromatization into estradiol. However, uncertainties remain regarding long-term osteometabolic health outcomes and the effect of gonadectomy on bone mineral density in this population. **Objective:** To evaluate long-term changes in bone mineral density and turnover markers in transgender men using testosterone through a systematic review of high-quality evidence. **Methods:** A systematic review was conducted across Virtual Health Library, PubMed, and Cochrane Library databases. The strategy was based on the PICO framework: Population (transgender men), Intervention (long-term testosterone), Comparison (controls), and Outcome (bone density and markers). Medical Subject Headings terms (Transgender Persons, Testosterone, Bone Density) and the Boolean operator AND were utilized. Inclusion criteria consisted of longitudinal studies with a minimum follow-up of two years. Duplicate articles and studies without a direct relationship to the topic were excluded. **Results:** Twenty-one studies met the eligibility criteria. In the analysis of lumbar spine mineral density, long-term hormone therapy led to a statistically significant mean increase of 2.1%, with a 95% confidence interval between 1.2% and 3.0%, indicating a favorable anabolic response to testosterone aromatization. No statistically significant longitudinal variations were observed in the femoral neck and total hip, confirming the preservation of cortical bone. Bone turnover markers (collagen type 1 cross-linked C-telopeptide and procollagen type 1 N-terminal propeptide) showed a sustained trend toward suppression of bone resorption. In the post-gonadectomy subgroup, osteoprotection was contingent upon serum testosterone levels maintained strictly within the physiological male range of 300–1,000 ng/dL. No clinical incidence of fragility fractures was documented. **Conclusion:** Testosterone therapy is effective in maintaining bone health in transgender men over the long term by stabilizing bone turnover. Bone mass preservation is highly dependent on achieving therapeutic levels and aromatization. Monitoring via dual-energy X-ray absorptiometry remains the clinical gold standard.

Keywords: transgender persons; testosterone; bone density.

172 (3130) METABOLIC AND HORMONAL HETEROGENEITY IN HYPERANDROGENIC WOMEN: A PRELIMINARY PHENOTYPIC ANALYSIS ACCORDING TO OBESITY STATUS

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Hyperandrogenic syndrome in women is frequently associated with metabolic abnormalities and insulin resistance, conditions influenced by obesity and androgen bioavailability. However, clinical and laboratory manifestations of hyperandrogenism demonstrate substantial heterogeneity, even among women with similar body mass index (BMI) and clinical presentation, suggesting the involvement of distinct metabolic and hormonal mechanisms. This study aimed to evaluate metabolic and hormonal variability in reproductive-aged women with clinical signs of hyperandrogenism, investigating the influence of obesity and sex hormone-binding globulin (SHBG) on phenotypic expression. This observational, analytical, cross-sectional study included a planned sample of 75 participants divided into three groups: women with hyperandrogenic manifestations and normal BMI, women with hyperandrogenic manifestations and obesity, and a control group. Anthropometric, clinical, metabolic, and hormonal parameters were assessed, including fasting glucose, insulin, lipid profile, testosterone, and SHBG levels. Preliminary findings demonstrated marked clinical and laboratory variability among participants with hyperandrogenic manifestations. A higher frequency of acanthosis nigricans, hirsutism, and hair loss was observed in women with obesity. Nevertheless, distinct metabolic and hormonal profiles were identified even among participants with similar BMI and clinical presentation. Some women exhibited pronounced clinical manifestations despite the absence of significant hormonal abnormalities, whereas others demonstrated hyperinsulinemia, reduced SHBG levels, and notable metabolic alterations with less evident clinical expression, suggesting a modulatory role of SHBG in androgen bioavailability and phenotypic variability. These findings suggest that hyperandrogenic manifestations may not be fully explained by traditionally assessed hormonal parameters alone. Obesity and associated metabolic factors appear to influence clinical expression, possibly through mechanisms related to androgen bioavailability and peripheral androgen sensitivity. Detailed phenotypic and metabolic characterization may therefore contribute to a broader understanding of hyperandrogenic syndrome, even in the absence of classic laboratory abnormalities.

Keywords: hyperandrogenism; obesity; insulin resistance.

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173 (3456) METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE (MASLD) AND FEMALE INFERTILITY: A SYSTEMATIC REVIEW OF REPRODUCTIVE OUTCOMES

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Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), affects approximately 40% of the global adult population. Among women of reproductive age, 14% to 19.8% of pregnant women present the condition. Evidence indicates MASLD as an independent risk factor for maternal-fetal complications, significantly increasing the chances of developing gestational diabetes mellitus, pre-eclampsia, preterm birth, and large-for-gestational-age newborns. A direct correlation between steatosis severity and the magnitude of clinical risks is also observed. **Objective:** To evaluate the implications of MASLD on reproductive and obstetric outcomes in women of reproductive age, with emphasis on maternal-fetal complications and fertility parameters. **Methods:** This is a systematic review conducted in accordance with PRISMA guidelines and structured by the PICO strategy, with searches in PubMed, Embase, and Science Direct (2016–2026). Observational studies and clinical trials involving women of reproductive age diagnosed with MASLD by imaging, liver biopsy, or validated biomarkers, reporting adverse reproductive outcomes, were included. Records were screened by two independent reviewers on the Rayyan platform, and 7 studies were included in the narrative synthesis. **Results:** MASLD is an independent predictor of maternal-fetal complications, significantly increasing the risk of gestational diabetes mellitus (GDM), pre-eclampsia, and gestational hypertension, even in women with normal pre-gestational weight. Steatosis severity correlates with higher rates of preterm birth in some populations, though evidence remains heterogeneous across studies. Large-for-gestational-age (LGA) newborns are consistently associated with MASLD across multiple cohorts. A high prevalence of MASLD is observed in patients with PCOS, with hyperandrogenism conferring an OR of 3.31 (95% CI 2.58–4.24) for MASLD development, worsened by obesity and insulin resistance. These results reinforce the importance of early screening and metabolic follow-up in prenatal care. **Conclusion:** Studies demonstrate a significant association with risk of maternal-fetal complications in eutrophic women with MASLD and, predominantly, in patients also diagnosed with PCOS and hyperandrogenism due to compounded metabolic outcomes. Early and longitudinal prenatal follow-up proves relevant in this group, given the vulnerabilities identified.

Keywords: MASLD; reproductive outcomes; maternal-fetal complications.

174 (3241) METFORMIN ALONE VERSUS METFORMIN COMBINED WITH INOSITOLS IN POLYCYSTIC OVARIAN METABOLIC SYNDROME: A SYSTEMATIC REVIEW OF EFFECTS ON REPRODUCTIVE, METABOLIC, AND HORMONAL OUTCOMES

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Introduction: Polycystic ovarian metabolic syndrome (PMOS) affects 4%–20% of women of reproductive age, characterized by hyperandrogenism, insulin resistance, and ovulatory dysfunction. Metformin is the reference insulin sensitizer, while inositols — myo-inositol (MI) and D-chiro-inositol (DCI) — have emerged as complementary agents with potential synergistic effects on ovarian function and metabolic profile. **Objective:** To evaluate the efficacy of metformin combined with inositols versus metformin alone on reproductive, metabolic, and hormonal outcomes in Rotterdam-criteria PMOS. **Methods:** Systematic review (PRISMA) searching PubMed and Embase (2016–2025) using: ("polycystic ovary syndrome" OR "PCOS") AND ("inositol" OR "myo-inositol") AND "metformin". Eligible studies included reproductive-age women with Rotterdam-criteria PMOS comparing metformin plus MI and/or DCI versus metformin monotherapy. Exclusion criteria: observational studies, case reports, animal studies, and publications without a metformin monotherapy comparator. Screening by two independent reviewers using Rayyan. **Results:** Of 229 identified records, 98 were duplicates; 25 underwent full-text review, yielding 9 included studies. Combined therapy was superior in reproductive outcomes: greater menstrual cycle regularity (RR 1.56; 95% CI: 1.01–2.41; p = 0.04), with 79.2% vs. 62.6% achieving regular cycles in a 6-month RCT. In infertile women undergoing ovulation induction, clinical pregnancy rates (63.3% vs. 33.3%) and live birth rates (55% vs. 26.67%) were significantly higher. For hormonal outcomes, combined therapy reduced LH (p = 0.002), LH/FSH ratio (p = 0.007; MD -0.13; 95% CI: -0.24 to -0.03), hirsutism score (MD -0.97; p < 0.01), and free testosterone by -34.9%. Metabolic outcomes were heterogeneous: one double-blind RCT showed superiority in reducing HOMA-IR and increasing HDL; however, Kelly *et al.*'s meta-analysis found no significant differences for HOMA-IR (p = 0.25), BMI (p = 0.13), or fasting glucose (p = 0.07). **Conclusion:** The addition of inositols to metformin produces a synergistic effect with superiority in reproductive and hormonal outcomes — menstrual regularisation, LH/FSH ratio reduction, hirsutism improvement, and higher pregnancy rates — without consistent additional metabolic benefit on HOMA-IR or BMI. These findings support preferential use of combined therapy in women with PMOS and reproductive goals. Methodological heterogeneity underscores the need for multicentre trials.

Keywords: polycystic ovary syndrome; inositol; metformin.

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175 (3368) MULTIPLE FOCAL NODULAR HYPERPLASIA IN A TRANSGENDER MAN

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Sex hormones may influence the genesis and growth of hepatic lesions, raising important considerations for the maintenance of hormone therapy in transgender individuals. Testosterone, depending on its formulation and route of administration, may exert distinct effects on hepatic metabolism and cellular architecture. **Case report:** We present a 39-yo transgender man, menarche at 15 yrs, who initiated cross-sex hormone therapy with IM testosterone cypionate at age 28. At 32, mild elevation of liver enzymes prompted imaging, revealing hepatic nodules. MRI findings suggested focal nodular hyperplasia (FNH) versus adenoma. After discontinuation of therapy, testosterone undecanoate was reintroduced at age 34. At 39, CT demonstrated stability of the nodules. Given diagnostic uncertainty, videolaparoscopic nodulectomy was performed, confirming FNH. Hormone therapy was subsequently modified to transdermal testosterone. **Discussion:** Oral testosterone formulations, particularly 17α-alkylated derivatives, are well known for their hepatotoxic potential, including risks of cholestasis, adenoma, and hepatocellular carcinoma. This toxicity is linked to intense hepatic first-pass metabolism. In contrast, injectable formulations (e.g., cypionate, undecanoate) bypass first-pass metabolism but deliver supraphysiologic peaks, which may stress hepatocellular function and contribute to nodular hyperplasia. Transdermal testosterone provides more stable serum levels, minimizing hepatic impact. Histopathological findings in this case (hepatocellular proliferation with fibrous septa, inflammatory infiltrate) illustrate cellular modifications that may be potentiated by androgenic stimulation. **Conclusion:** This case highlights the relevance of testosterone's pharmacokinetics and tissue-level effects in the evaluation of hepatic lesions in transgender individuals. The choice of formulation and route of administration is critical for minimizing hepatic risk and guiding long-term therapy.

Keywords: transgender man; testosterone; multiple focal nodular hyperplasia.

176 (3001) NORMOSMIC CONGENITAL HYPOGONADOTROPIC HYPOGONADISM ASSOCIATED WITH A RARE VARIANT IN THE FGFR1 GENE IN AN ADULT WOMAN WITH FAMILIAL PRIMARY AMENORRHEA

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Case presentation: A 26-year-old woman was referred for investigation of primary amenorrhea and absent pubertal development. She reported gynecological follow-up since the age of 15 years without a definitive diagnosis. She had used combined oral contraceptives until the age of 20 years, remaining amenorrheic. She denied menarche and presented absence of breast and pubic hair development. Family history was positive for a 19-year-old sister with a similar condition. Parents were non-consanguineous. Physical examination revealed Tanner stage M3 breasts and P3 pubic hair. Investigation demonstrated a hypoplastic uterus, reduced ovaries, atrophic endometrium, and normal pituitary magnetic resonance imaging. Hormonal evaluation revealed estradiol below 20 pg/mL, FSH 0.51 mIU/mL, and LH below 0.1 mIU/mL, compatible with hypogonadotropic hypogonadism. Karyotype was 46,XX. Considering the association of primary amenorrhea, persistently low gonadotropins, normal pituitary imaging, and positive family history, a next-generation sequencing panel was performed, identifying a heterozygous c.232C>T (p.Arg78Cys) variant in the FGFR1 gene, classified as pathogenic. After diagnostic confirmation, pubertal induction with progressive doses of transdermal estradiol was initiated, with subsequent mild pubertal development and increased serum estradiol levels. **Discussion:** Congenital hypogonadotropic hypogonadism is a rare disorder caused by deficient secretion or action of GnRH. Variants in the FGFR1 gene are classically associated with Kallmann syndrome but may also occur in normosmic patients. In this case, the severe phenotype, absence of spontaneous puberty until adulthood, and marked familial aggregation without olfactory abnormalities or associated malformations were noteworthy. Variants in FGFR1 represent an important cause of congenital hypogonadotropic hypogonadism and are associated with broad phenotypic heterogeneity, reinforcing the importance of molecular investigation for etiological definition, genetic counseling, and therapeutic management. **Final comments:** This report highlights the importance of genetic investigation in patients with primary amenorrhea and hypogonadotropic hypogonadism, especially in the presence of positive family history. Identification of the rare variant in the FGFR1 gene enabled definitive diagnosis, prognostic guidance, and appropriate pubertal induction.

Keywords: congenital hypogonadotropic hypogonadism; primary amenorrhea; FGFR1.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

177 (3392) NOVEL FRAMESHIFT VARIANT IN THE ANDROGEN RECEPTOR GENE LINKED TO COMPLETE ANDROGEN INSENSITIVITY SYNDROME

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Case presentation: The androgen receptor (AR) gene, located on the X chromosome, encodes a ligand-dependent nuclear receptor essential for androgen signaling. Variants affecting AR may impair androgen action and lead to Androgen Insensitivity Syndrome (AIS). The present report describes a 36-year-old patient with a 46,XY karyotype, initially followed for Partial Gonadal Dysgenesis, and so addressed to our service. However, after getting back to the investigation of primary amenorrhea and identification of inguinal gonads by magnetic resonance imaging at the age of sixteen, the diagnostic hypothesis was revised to complete androgen insensitivity syndrome (CAIS). Imaging and anatomopathological examinations demonstrated the absence of the prostate and uterus, presence of a blind-ending vagina, and tubular cystic formations suggestive of Müllerian remnants. Molecular analysis of the AR identified a 19-bp deletion in exon 1, resulting in a frameshift variant c.721_739del (p.Lys241Argfs*11). **Discussion:** The identified deletion is not previously described in the literature, but is consistent with the diagnosis of CAIS due to causing an altered frame reading and premature stop codon formation, probably with complete loss of androgen receptor function. This variant was classified as likely pathogenic according to ACMG/AMP criteria (PVS1, PM2_supporting, PP4). Because the variant occurs in exon 1, a major structural impairment of the protein is expected, including disruption of important functional domains such as AF-1, which is essential for transcriptional activation. The molecular finding is consistent with the phenotype of CAIS observed in the patient. Furthermore, as this variant has not been previously reported, this case expands the mutational spectrum associated with AIS and reinforces the relevance of molecular sequencing in the diagnostic investigation of 46,XY Differences of Sex Development (DSD). **Final comments:** Although we identified a likely pathogenic 19-bp AR deletion, ongoing functional studies and its correlation with immunohistochemical markers remain important to further support the pathogenicity of this variant. This case underscores the importance of genotypic analysis in individuals with 46,XY DSD to improve clinicomolecular correlation and emphasizes the relevance of a multidisciplinary approach in advancing the understanding of molecular heterogeneity associated with AIS.

Keywords: androgen-insensitivity syndrome; differences of sex development; intersex persons.

178 (3204) OFF-LABEL USE OF TESTOSTERONE FOR HYPOACTIVE SEXUAL DESIRE DISORDER IN POSTMENOPAUSAL WOMEN: A SYSTEMATIC REVIEW

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Introduction: Genitourinary syndrome of menopause affects 50% to 60% of postmenopausal women, compromising quality of life through urogenital and sexual symptoms, notably Hypoactive Sexual Desire Disorder (HSDD). Despite the physiological decline of testosterone with aging, its therapeutic use remains predominantly off-label due to the absence of female-specific formulations and limited long-term safety data, requiring caution regarding dosages and administration routes during the postmenopausal period. **Objective:** To evaluate the scientific evidence on the efficacy, safety, and indications of testosterone in postmenopausal women. **Methods:** A systematic review based on the PICO strategy (P: Postmenopausal women; I: Testosterone; C: Placebo or isolated estrogen; O: Sexual function and safety). Conducted according to PRISMA recommendations, the search was performed in PubMed/MEDLINE, SciELO, Scopus, and LILACS databases via the Virtual Health Library, covering the last 5 years. Randomized clinical trials, systematic reviews, and meta-analyses evaluating transdermal doses of approximately 300 mcg/day and efficacy/safety outcomes were included. **Results:** Therapy demonstrated a significant increase in libido, raising the mean number of satisfying sexual episodes from 2.92 to 4.92 in 4 weeks in the intervention group ($p = 0.003$). An improvement in Female Sexual Function Index (FSFI) scores was observed from a mean of 12.7 (standard deviation 10.0) to 18.2 (standard deviation 13.0) in 12 weeks. The transdermal route proved superior to the oral route by not negatively impacting the lipid profile (LDL and triglycerides). Common side effects include acne and mild hirsutism. There was no statistically significant increase in the risk of invasive breast cancer (HR 1.42; 95% CI 0.95-2.11) or serious cardiovascular events in short-term studies (up to 24 months). Comparatively, hyaluronic acid showed similar efficacy to estrogen for atrophy but was inferior to testosterone in the domains of desire and orgasm. **Conclusion:** Testosterone replacement at physiological doses is effective for the short-term treatment of HSDD in postmenopause. The transdermal route is preferable due to its metabolic safety. In the absence of evidence for improvement in cognition or general well-being, use should be restricted to proven sexual dysfunction, with treatment discontinuation if there is no clinical response within 6 months.

Keywords: testosterone; postmenopause; hypoactive sexual desire disorder.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

179 (3502) OVARIAN LEYDIG CELL TUMOR AS A RARE CAUSE OF POSTMENOPAUSAL HYPERANDROGENISM

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Case presentation: A 67-year-old postmenopausal woman with hypertension and overweight presented with progressive facial hirsutism and alopecia of late onset, without significant virilization signs. Laboratory investigation demonstrated persistent elevation of total testosterone levels, while dehydroepiandrosterone sulfate levels remained within the normal range, suggesting an ovarian source of hyperandrogenism. Androstenedione and 17-hydroxyprogesterone levels were unremarkable. Initial transvaginal ultrasound showed only a probable endometrial polyp, without significant ovarian abnormalities. Due to persistent hyperandrogenism, magnetic resonance imaging of the abdomen and pelvis was performed, revealing a 1.9 cm solid nodular lesion in the right ovary, classified as O-RADS 4, raising suspicion for a sex cord-stromal tumor. The patient underwent total hysterectomy with bilateral salpingo-oophorectomy by videolaparoscopy. Histopathological examination confirmed a Leydig cell tumor in the right ovary associated with contralateral Leydig cell hyperplasia. **Discussion:** Ovarian Leydig cell tumors belong to the sex cord-stromal tumor group and represent a rare cause of postmenopausal hyperandrogenism. These tumors are commonly associated with androgenic manifestations such as hirsutism and alopecia and may also present with virilization. This case is noteworthy because of its subtle clinical presentation and small tumor size, which initially hindered diagnosis. Normal dehydroepiandrosterone sulfate levels helped exclude adrenal hyperandrogenism and supported an ovarian etiology. Another relevant aspect was the absence of significant ovarian findings on initial ultrasound, making magnetic resonance imaging essential for lesion detection. **Final comments:** Late-onset hyperandrogenism in postmenopausal women should prompt investigation for androgen-secreting neoplasms, even in patients with mild clinical manifestations. Although rare, Leydig cell tumors should be considered in the differential diagnosis, particularly in cases of persistent testosterone elevation associated with normal dehydroepiandrosterone sulfate levels. This case highlights the importance of integrating clinical, laboratory, and imaging findings for early diagnosis of rare ovarian neoplasms.

Keywords: hyperandrogenism; postmenopause; Leydig cell tumor.

180 (3308) PREGNANCY IN TRANSGENDER MEN: THREE CASE REPORTS

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Introduction: Approximately one-third of transgender men using testosterone still ovulate, reinforcing the need for reproductive counseling during Gender-affirming hormone therapy (GAHT). We report three cases followed up in a multidisciplinary outpatient clinic at a public maternity hospital. Case 1: 27-year-old, BMI 31.5 kg/m², G1P0A0, had an unplanned pregnancy. GAHT had been discontinued for more than three years. Testosterone levels remained within the normal range during the third trimester (60.87 ng/dL). Transient grade 1 intrauterine growth restriction occurred. A female baby (2635 g, Apgar 8/9) was delivered by elective cesarean section at 39 weeks of gestation, based on the patient's preference, without evidence of virilization. Exclusive breastfeeding was initiated. After four months, the patient underwent intrauterine device insertion and resumed GAHT. Case 2: 26-year-old, BMI 28.3 kg/m², G2P1A1, had an unplanned pregnancy. GAHT had been discontinued for one year. Third-trimester tests showed total and free testosterone (100 ng/dL and 1.7 pg/mL), suggesting androgen exposure. Elective cesarean delivery, performed at the patient's request, resulted in a healthy female newborn (3310 g, Apgar 8/9) without signs of virilization. Exclusive breastfeeding was initiated, and postpartum free testosterone normalized. Case 3: 26-year-old, BMI 15.1 kg/m², G1P0A0, underwent a planned pregnancy after discontinuing GAHT six months before conception. Testosterone levels remained in female pattern during both the first and third trimesters. Gestational diabetes mellitus was diagnosed and managed with diet. The patient had appropriate gestational weight gain, female fetus is developing without complications at 28 weeks of gestational age. **Discussion:** These cases highlight the heterogeneity of transmasculine pregnancies and the role of a multidisciplinary team trained to care for transgender people. We did not observe triggers or worsening of gender dysphoria in any of these cases. All patients expressed a desire to carry and deliver a child. Free testosterone measurement should help to identify androgen exposure, as increased sex hormone-binding globulin levels impair total testosterone interpretation. **Final comment:** Transgender men can achieve favorable outcomes with inclusive care.

Keywords: transgender men; pregnancy; gender-affirming hormone therapy.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

181 (3305) PREVALENCE AND CLINICAL IMPACT OF EATING DISORDERS (ED) IN WOMEN AND ADOLESCENTS WITH POLYCYSTIC OVARY SYNDROME (PCOS): A SYSTEMATIC REVIEW

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Introduction: Polycystic ovary syndrome (PCOS) is one of the most common endocrinopathies among women of reproductive age and is associated with metabolic, hormonal, and psychosocial disturbances. Clinical manifestations such as obesity, hirsutism, and difficulty losing weight may contribute to body dissatisfaction and emotional distress, increasing vulnerability to eating disorders (EDs). However, evidence regarding this association remains heterogeneous. **Objective:** To evaluate the prevalence and clinical impact of EDs in women and adolescents with PCOS. **Methods:** This systematic review was conducted according to PRISMA guidelines. Literature searches were performed in the Cochrane, Embase, and Scopus databases using the following strategy: ("Polycystic Ovary Syndrome" OR "PCOS" OR "Polycystic Ovarian Syndrome") AND ("Eating Disorder" OR "Emotional Eating"). Inclusion criteria comprised English-language observational studies and randomized controlled trials published within the last five years. Review articles were excluded. **Results:** A total of 3,285 studies were identified, of which 8 met the eligibility criteria and were included in the review. Women and adolescents with PCOS showed a significantly higher prevalence of EDs compared with controls (25.5% vs. 2.7%). Rates of binge eating disorder (BED) ranged from 6.8% to 23.3%, while binge-eating symptoms affected up to 60% of patients. PCOS diagnosis increased the likelihood of EDs by more than sevenfold, with higher body mass index identified as a relevant risk factor. Binge-eating symptoms, uncontrolled eating, and emotional eating were associated with greater severity of postprandial reactive hypoglycemia symptoms, whereas overeating episodes correlated with clinical hyperandrogenism. The coexistence of PCOS and BED was associated with poorer health-related quality of life, anxiety, and depressive symptoms. **Conclusion:** Women with PCOS demonstrate high vulnerability to EDs, particularly BED, whose pathophysiology may involve hormonal alterations and glycemic fluctuations. Management strategies focused exclusively on caloric restriction and weight loss may reinforce dysfunctional eating behaviors. Therefore, routine screening for EDs in patients with PCOS is essential to optimize both metabolic outcomes and mental health.

Keywords: eating disorders; polycystic ovary syndrome; systematic review.

182 (3031) PSEUDO-HYPERANDROGENEMIA DUE TO PROBABLE TESTOSTERONE IMMUNOASSAY INTERFERENCE: A CASE REPORT

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A reproductive-age woman was referred for persistently elevated total serum testosterone measured by chemiluminescence immunoassay (CLIA). She had regular menses, no current hirsutism or virilization and denied exogenous androgen exposure. She reported acne and alopecia, treated with spironolactone 100 mg/day. Serial immunoassays showed repeatedly elevated total testosterone levels, reaching 157 ng/dL (reference range 10–75 ng/dL). Additional evaluation did not support adrenal, ovarian, pituitary, or cortisol-related disease (Table 1). Transvaginal pelvic ultrasound showed normal ovarian morphology and no adnexal mass. Because of the discrepancy between laboratory findings and clinical phenotype, assay interference was suspected and total testosterone was reassessed by liquid chromatography-tandem mass spectrometry (LC-MS/MS), with a normal result of 22 ng/dL (reference range ≤ 63 ng/dL). This case illustrates false biochemical hyperandrogenism caused by probable analytical interference in testosterone immunoassay. Testosterone measurement in women is technically challenging because circulating concentrations are low and automated immunoassays may be affected by limited sensitivity, cross-reactivity, and interferences such as heterophile antibodies and biotin-streptavidin interference. The absence of progressive symptoms, menstrual dysfunction, virilization, adrenal androgen excess, or ovarian mass argued against a pathological cause of hyperandrogenemia. The normal result obtained by LC-MS/MS, a more specific steroid assay, reclassified the previous elevations as spurious rather than true hyperandrogenemia. Once the patient denied using any supplement containing biotin, the presence of a heterophile antibody was the leading hypothesis in this case. Testosterone measurement without a clear clinical indication may lead to false results, unnecessary investigation, and patient anxiety. Discordance between biochemical findings and clinical phenotype should raise suspicion for immunoassay interference. Confirmation by LC-MS/MS is useful before extensive imaging, invasive investigation or treatment decisions in women with isolated testosterone elevation by immunoassay.

Keywords: hyperandrogenism; testosterone; immunoassay interference.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

183 (3486) RECURRENCE OF DEPRESSIVE SYMPTOMS AFTER INITIATION OF CLOMIPHENE CITRATE FOR FUNCTIONAL HYPOGONADISM: A CASE REPORT

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Case report: A 41-year-old overweight man (BMI 29 kg/m²; waist circumference 105 cm) presented with asthenia, sleep disturbances, decreased libido, and a 9-kg weight gain over the previous year. He sought testosterone therapy because of low total testosterone levels. He denied comorbidities, medication use, smoking, alcohol consumption, or illicit drug use. He had a previous depressive episode successfully treated with escitalopram six years earlier. Laboratory evaluation showed total testosterone levels of 267 ng/dL and 281 ng/dL on separate measurements, LH 2.0 mIU/mL, FSH 2.7 mIU/mL, and SHBG 42 nmol/L. Testicular ultrasound and pituitary MRI were unremarkable. Male obesity-associated secondary hypogonadism (MOSH) was diagnosed. Intensive lifestyle modification was recommended, but the patient declined anti-obesity pharmacotherapy. Clomiphene citrate 50 mg three times weekly was initiated. Approximately 15 days later, he developed marked worsening of depressive symptoms, initially reported by his spouse. Considering potential psychiatric adverse effects related to clomiphene, the medication was discontinued, escitalopram was restarted, and lifestyle interventions were reinforced. Psychiatric symptoms progressively improved thereafter. After six months, the patient showed overall clinical improvement and an increase in total testosterone levels to 431 ng/dL. **Discussion:** This case highlights important challenges in MOSH management, including symptom attribution solely to low testosterone levels, resistance to obesity treatment, and the increasing use of clomiphene citrate as an alternative to exogenous testosterone therapy. Although uncommon, psychiatric adverse effects associated with clomiphene — including depression, anxiety, panic symptoms, and suicidal ideation — have been reported, particularly in patients with previous psychiatric disorders. **Conclusion:** Recurrence or worsening of psychiatric symptoms should be recognized as a possible adverse effect of clomiphene citrate, especially in individuals with prior psychiatric history. Careful clinical assessment and monitoring are essential during treatment.
Keywords: male obesity-associated secondary hypogonadism; clomiphene citrate; depression.

184 (2909) RECURRENT NORMOPROLACTINEMIC GALACTORRHEA ASSOCIATED WITH SSRIS: A CASE REPORT

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A 20-year-old woman presented with bilateral galactorrhea for 7 months, associated with painless bilateral breast nodules. She denied menstrual irregularities and current or previous pregnancy. She reported a similar episode 3 years prior, when she underwent excision of a left breast nodule, with benign histopathology. Hormonal evaluation, including prolactin and thyroid-stimulating hormone (TSH), was within normal limits. Breast ultrasound revealed bilateral nodules with benign characteristics. The patient had a history of galactorrhea at age 17 during fluoxetine use, which resolved after discontinuation of the medication and use of a lactation-suppressing agent. Years later, after starting sertraline, the symptoms recurred. Based on the temporal association between antidepressant use and symptom onset, a diagnosis of antidepressant-induced galactorrhea was established. Clinical follow-up and therapeutic reassessment were performed, with a stable condition and no evidence of malignancy. Galactorrhea is defined as milk secretion outside of pregnancy or lactation, usually associated with hyperprolactinemia. However, the normoprolactinemic form occurs despite normal serum prolactin levels and is less well understood. Selective serotonin reuptake inhibitors (SSRIs) can induce galactorrhea, most frequently with elevated prolactin levels. The main characteristic of this case is the recurrence of symptoms associated with drug use, despite normal hormone levels. This atypical presentation suggests a functional mechanism, possibly involving modulation of the serotonin-dopamine axis and increased breast sensitivity to basal prolactin. The diagnosis was based on the exclusion of endocrine and structural causes, as well as the temporal relationship between drug exposure and symptoms, supporting an iatrogenic etiology. Physicians should recognize that galactorrhea can occur even with normal prolactin levels, as hormonal fluctuations can make it difficult to detect nocturnal peaks. The diagnosis is clinical and depends on correlation with pharmacological history, requiring an understanding of the serotonin-dopamine-prolactin axis and consideration of peripheral hypersensitivity to physiological hormonal levels.
Keywords: galactorrhea; normoprolactinemic; SSRIs.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

185 (3088) ROLE OF GUT MICROBIOTA IN INSULINA RESISTANCE ASSOCIATED WITH POLYCYSTIC OVARY SYNDROME

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Introduction: Polycystic ovary syndrome (PCOS) is a prevalent endocrinopathy among reproductive-aged women, commonly associated with insulin resistance, hyperandrogenism, and metabolic dysfunction. Evidence suggests that gut microbiota alterations contribute to PCOS pathophysiology through inflammatory modulation, increased intestinal permeability, and metabolic and hormonal dysregulation. **Objective:** To analyze the association between gut microbiota alterations and insulin resistance in women with polycystic ovary syndrome. **Methods:** This systematic review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines. The research question was structured using the PICO strategy: "Is there an association between gut microbiota alterations and insulin resistance in women with Polycystic Ovary Syndrome?" The literature search was performed in the PubMed/MEDLINE database (2021–2026) using Boolean operators and descriptors in English and Portuguese, including "Polycystic Ovary Syndrome," "Gut Microbiota," "Insulin Resistance," "Dysbiosis," and "Metabolic Syndrome." Article selection, data extraction, methodological quality and risk of bias assessment were independently conducted by reviewers. **Results:** The search identified 39 potentially relevant studies. After applying eligibility criteria, 11 articles were selected for full-text evaluation, and 5 studies were included in the final analysis. The findings demonstrated that women with PCOS exhibit significant alterations in gut microbiota composition, mainly dysbiosis and reduced bacterial diversity. These alterations were positively associated with insulin resistance, systemic inflammation, obesity, and metabolic syndrome. Furthermore, evidence suggested a possible interaction between gut microbiota imbalance and hyperandrogenism, reinforcing the involvement of dysbiosis in the metabolic and endocrine dysfunctions observed in PCOS. **Conclusion:** The analyzed evidence demonstrates a consistent association between gut microbiota alterations and insulin resistance in women with PCOS, suggesting that dysbiosis may contribute to the worsening of metabolic and endocrine abnormalities associated with the syndrome. These findings reinforce the potential role of gut microbiota in the pathophysiology of PCOS and highlight its potential as a therapeutic target. However, further studies are needed to better clarify the mechanisms underlying this relationship.
Keywords: polycystic ovary syndrome; gut microbiota; insulin resistance.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

186 (3564) SATISFACTION WITH BODY CHANGES AMONG TRANSFEMININE INDIVIDUALS UNDERGOING GENDER-AFFIRMING HORMONE THERAPY (GAHT)

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Introduction: Gender-Affirming Hormone Therapy (GAHT) is a cornerstone for secondary sex characteristics in transfeminine individuals. In Brazil, injectable estrogens/progestogens or oral estrogen with/without synthetic antiandrogens (cyproterone acetate) have historically been the most widespread regimens among transgender women. Only recently has this population gained access to transdermal estrogen-based GAHT. Nevertheless, literature lacks comparative data regarding satisfaction levels across different administration routes. **Objective:** To evaluate the perception of body changes and subjective satisfaction associated with different GAHT regimens among transfeminine individuals receiving care at two specialized public healthcare services. **Methods:** A descriptive cross-sectional study was conducted with 90 participants followed at two specialized public healthcare services within the Brazilian Unified Health System (SUS). Structured questionnaires investigated five perceived changes in breast development, skin, body hair, fat distribution/muscle mass, and mood. Satisfaction levels were compared across injectable, oral, transdermal, and antiandrogen-only regimens. **Results:** For breast augmentation, the injectable-only regimen yielded the highest satisfaction rate, 35/90 (38.8%). Regarding fat and muscle mass, fat redistribution (81.1%) and decreased physical strength (73.3%) were the most prominent changes, with greater satisfaction reported among participants using injectable regimens (25.5%). Concerning skin changes, thinning was the most reported effect (88.9%), followed by reductions in acne (56.7%) and oiliness (51.1%). For body hair, 33.3% of participants were indifferent to the regimen used. Regarding mood, emotional lability was identified through active inquiry in 54.3% of the sample, while 42.2% reported no perceived association between mood changes and the specific regimen employed. **Conclusion:** GAHT was associated with a high perception of desirable body changes. Greater subjective satisfaction was observed with injectable regimens, particularly concerning breast development and fat redistribution. In contrast, reductions in body hair and mood changes appeared less dependent on the specific hormonal regimen used.
Keywords: transgender; GATH; body satisfaction.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

187 (3468) SOCIODEMOGRAPHIC PROFILE OF TRANSGENDER INDIVIDUALS FOLLOWED AT TWO SPECIALIZED CENTERS IN SALVADOR, BRAZIL

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Introduction: Qualified assistance in referral centers is essential for the longitudinal care and health promotion of transgender individuals. In this context, the expansion of access to these services reveals a growing diversification in the users' profile, whose specificities directly condition the health needs and interventions required by this population. **Objective:** To describe the sociodemographic profile of transgender individuals followed at a referral center. **Methods:** This cross-sectional observational study included transgender individuals receiving care at two referral centers in Salvador, Bahia. The study analyzed the sociodemographic profile of the participants using data obtained through structured questionnaires in REDCap. The variables included age, gender identity, race/ethnicity, educational level, religion, sexual orientation and hormone use. **Results:** A total of 640 participants were included, with a median age of 28 [25–33] years. Trans men predominated (47.2%), followed by trans women (39.8%), non-binary individuals (7.2%), and travestis (5.8%). Most participants self-identified as Black (36.4%) or mixed-race (35.5%). Regarding religion, participants with no religious affiliation predominated (33.6%), followed by practitioners of Candomblé (21.0%) and agnostics (20.3%). In terms of educational attainment, most participants had incomplete higher education (37.3%) or completed secondary education (32.5%). Regarding sexual orientation, heterosexuality predominated (60.6%), followed by bisexuality (18.0%) and pansexuality (15.5%). Only 3.0% of participants self-identified as homosexual. Regarding hormone use, 47.3% reported exclusive use under medical prescription, whereas 19.5% reported self-medication and 24.8% reported both forms of use. **Conclusion:** The sociodemographic profile of transgender individuals in Bahia reveals a heterogeneous population, predominantly Black and mixed-race trans men, with high educational attainment, a predominance of heterosexual orientation, and notable rates of unsupervised hormone use, highlighting the need to expand and strengthen specialized transgender health services.

Keywords: gender identity; transgender health; sociodemographic factors.

188 (2975) SUBACUTE EXPOSURE TO BISPHENOL S AND/OR BISPHENOL F AT ENVIRONMENTALLY RELEVANT DOSES DISRUPTS THE MALE HYPOTHALAMIC-PITUITARY-GONADAL AXIS

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Bisphenols are endocrine-disrupting chemicals widely used in the plastics and resin industry. Following restrictions on bisphenol A (BPA), analogs such as bisphenol S (BPS) and bisphenol F (BPF) have been increasingly used as supposedly safer alternatives. However, evidence suggests that these compounds may dysregulate the hypothalamic-pituitary-gonadal (HPG) axis, which is responsible for regulating male reproductive function, including spermatogenesis and hormone production. Therefore, this study aimed to evaluate the effects of subacute exposure to BPS and/or BPF on morphological, molecular, and functional parameters of the HPG axis in young adult male rats. Young adult male Wistar rats were divided into four groups (n = 5/group): Control (10% ethanol), BPS (4 µg/kg), BPF (4 µg/kg), and BPS+BPF (4+4 µg/kg), treated via gavage for 15 days. After euthanasia, histological, morphometric, and gene expression analyses were performed, evaluating genes related to the HPG axis (GnRH, LHβ, FSHβ, and kisspeptin), testicular function (ABP, LH receptor, FSH receptor, 3β-HSD, DDX4, aromatase, and inhibins), inflammation (IL-1β, IL-10, NF-κB, and TNF-α), and blood-testis barrier integrity (ZO-1 and claudin). Finally, sperm motility and concentration parameters were evaluated. Histopathological analysis revealed vacuolization in the seminiferous epithelium, presence of polymorphonuclear cells, interstitial edema, and vascular congestion in animals treated with BPS and/or BPF. Gene expression analysis demonstrated increased GnRH expression and reduced LHb expression in all treated groups. LH receptor and inhibin A expression were increased, whereas inhibin B expression was reduced. ABP expression was increased only in the mixture group. Additionally, 3β-HSD expression was elevated in all treated groups. Claudin expression was increased in the BPF group and decreased in the mixture group. Finally, no significant changes were observed in kisspeptin, FSHβ, FSH receptor, DDX4, aromatase, ZO-1, TNF-α, NF-κB, IL-1β, or IL-10 expression. Sperm analysis showed reduced progressive motility and increased non-progressive motility in treated animals. Subacute exposure to BPS and/or BPF promoted testicular histopathological alterations, dysregulation of HPG axis-related genes, and impaired sperm quality. These findings suggest that exposure to BPS and BPF, even at low doses, is not biologically inert and may negatively impact male reproductive function.

Keywords: bisphenol S; bisphenol F; hypothalamic-pituitary-gonadal axis.

WOMEN ENDOCRINOLOGY, ANDROLOGY AND TRANSGENERITY

189 (3341) SUPPRESSION OF THE GONADOTROPIC AXIS AND MACROCYTIC ANEMIA DUE TO CHRONIC USE OF ANABOLIC-ANDROGENIC STEROIDS: A RELEVANT DIFFERENTIAL DIAGNOSIS

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Case presentation: A 36-year-old male patient was admitted for the investigation of dyspnea and anemia. He reported a 12-month history of chronic and abusive use of anabolic-androgenic steroids (trenbolone, testosterone, and boldenone), T3, and levothyroxine, which were discontinued one month prior to admission. He denied any comorbidities. Clinical examination revealed no abnormalities. Laboratory tests showed normocytic anemia (hemoglobin 8.8 g/dL, mean corpuscular volume 83 fL, RDW 20.8%), with polychromasia, macrocytosis, and significant reticulocytosis (8.93%; 284,000/mm³). Other findings included elevated lactate dehydrogenase (1032 U/L), elevated transaminases (aspartate aminotransferase 174 U/L, alanine aminotransferase 136 U/L), low serum iron (34 µg/dL), low vitamin B12 (154 UI/mL), and elevated folic acid. The hormonal profile demonstrated suppression of the hypothalamic-pituitary-gonadal axis: follicle-stimulating hormone (FSH) 0.17 mUI/mL, luteinizing hormone (LH) less than 0.12 mUI/mL, and total testosterone 268.7 ng/dL. Early morning basal cortisol was 10.6 µg/dL, a range within which adrenal insufficiency is clinically unlikely. Thyroid-stimulating hormone was normal; however, free thyroxine (FT4) was at the lower limit of normality, and total thyroglobulin was markedly elevated (1009.40 ng/dL). Insulin-like growth factor 1 was 115 ng/mL, within the expected range for his age. A bone marrow aspirate (myelogram) demonstrated megaloblastic erythropoiesis due to folate deficiency, with no evidence of hematological malignancy. **Discussion:** Prolonged anabolic-androgenic steroid (AAS) use suppresses the gonadotropic axis. Here, undetectable FSH/LH and low testosterone confirm hypogonadotropic hypogonadism, which may persist after chronic use. Recovery of the hypothalamic-pituitary-testicular axis post-steroids varies: <1 year of use usually normalizes within a year, but prolonged or high doses can cause delayed, incomplete, or persistent hypogonadism. Anemia showed megaloblastic (B12 deficiency) and mild hemolytic traits, alongside hepatotoxicity (17-alpha-alkylated AAS cause transaminase elevation). Isolated thyroglobulin elevation suggests a pre-existing thyroid disorder requiring outpatient evaluation. **Final remarks:** This case highlights the endocrine and hematological impacts of abusive AAS use. Early recognition is vital given rising youth use. Outpatient follow-up is crucial to monitor suppressed axes, as recovery can be delayed.

Keywords: anabolic agents; hypogonadism; anemia.

PEDIATRIC ENDOCRINOLOGY

190 (3235) ASSOCIATION BETWEEN EXPOSURE TO ENDOCRINE-DISRUPTING CHEMICALS AND EARLY PUBERTAL ONSET IN CHILDREN: A SYSTEMATIC REVIEW

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Introduction: Precocious puberty (PP) is defined as the development of secondary sexual characteristics before 8 years in girls and 9 years in boys. Pubertal onset has been increasingly observed worldwide, and is associated with genetic, nutritional, and environmental influences. Among these, endocrine-disrupting chemicals (EDCs) interfere with hormonal regulation, mimicking hormones and prematurely activating the hypothalamic-pituitary-gonadal (HPG) axis. **Objective:** To evaluate the association between exposure to environmental endocrine disruptors and precocious puberty or early pubertal onset in children and adolescents. **Methods:** A systematic review was conducted according to PRISMA-based in PubMed and Embase. Randomized clinical trials and observational studies involving individuals aged 0–18 years were included. Eligible studies assessed exposure to EDCs — such as bisphenol A, phthalates and dioxins — and their association with precocious puberty or early pubertal onset. The search strategy followed the PICO framework. A total of 180 records were identified; after removal of 88 duplicates & title/full-text screening, 10 studies were included in the final synthesis. **Results:** Exposure to EDCs is associated with earlier pubertal onset. Bisphenol A (BPA) increases the risk of central PP in girls by up to 9.08 times, and each doubling in BPA levels raises the risk of premature thelarche by 29%. Short-chain phthalates more than double the likelihood of early menarche (OR 2.22) and increase the risk of breast development (IRR 1.64). Frequent exposure to contaminated toys (slime) increases the likelihood of PP diagnosis by 5.5 times. Compounds such as 2,5-dichlorophenol may advance menarche by an average of 4.09 months. Finally, emerging agents such as parabens, per- and polyfluoroalkyl substances, and metals (zinc) have been identified as factors that disrupt the androgenic-glucocorticoid balance, accelerating sexual maturation through multiple hormonal pathways. **Conclusion:** Ubiquitous endocrine-disrupting chemicals, particularly BPA and phthalates, are significant risk factors for precocious puberty through neuroendocrine disruption. Despite variations depending on exposure timing, the observed risks (1.4- to 9-fold higher in exposed groups) highlight the urgent need for regulatory policies to limit these chemicals in consumer products. Reducing exposure is essential to prevent long-term consequences, including impaired final height and metabolic disorders.
Keywords: endocrine-disrupting chemicals; precocious puberty; hypothalamic-pituitary-gonadal axis.

PEDIATRIC ENDOCRINOLOGY

191 (2979) — CASE REPORT: LATE REPERCUSSIONS OF EXTREME PREMATURITY ON GROWTH, PUBERTAL DEVELOPMENT, AND GLUCOSE METABOLISM

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Case presentation: A 21-year-old female patient, born extremely premature at 27 weeks of gestation, with extremely low birth weight (580 g), delivered by cesarean section due to oligohydramnios and fetal distress. Her neonatal course was complicated by bronchopulmonary dysplasia, patent ductus arteriosus, anemia, grade I intraventricular hemorrhage, and grade I retinopathy of prematurity. She required prolonged neonatal hospitalization, including mechanical ventilation for 44 days. During early childhood, she developed clinical signs of insulin resistance and hyperandrogenism, including obesity, dyslipidemia, hepatic steatosis, acne, hirsutism, and glucose intolerance, which progressed to type 2 diabetes mellitus at the age of 15 years, requiring insulin therapy from 18 years of age onward. She also presented precocious puberty (pubarche and menarche at 7 years of age), with advanced bone age of 17 years at a chronological age of 9 years, resulting in reduced final adult height. **Discussion:** This case illustrates the medium and long-term systemic repercussions of extreme prematurity associated with low birth weight, as well as its metabolic implications. From a neuroendocrine perspective, emphasis should be placed on the role of neonatal hyperinsulinemia in the accelerated catch-up growth frequently observed in extremely preterm infants, which contributes to glucose intolerance and childhood obesity. These features ultimately culminate in premature activation of the pubertal axis. The patient's short final stature appears to be multifactorial, resulting from intrauterine growth restriction, alterations in the GH–IGF-1, early onset of puberty, and consequent premature epiphyseal closure. **Final comments:** Extreme prematurity induces adverse metabolic programming, frequently associated with accelerated catch-up growth, which is linked to insulin resistance and outcomes such as obesity and type 2 diabetes mellitus, thereby constituting a marker of cardiometabolic and endocrinological risk. Hyperinsulinemia and elevated leptin levels act as modulators of the hypothalamic-pituitary-gonadal axis, anticipating the onset of puberty. Therefore, early recognition of these individuals as a high-risk group is imperative, allowing for the implementation of preventive strategies from the neonatal period onward, in order to mitigate the progression of metabolic syndrome and potentially delay the onset of puberty.
Keywords: extreme prematurity; precocious puberty; insulin resistance.

PEDIATRIC ENDOCRINOLOGY

192 (3522) CLINICAL COMPLEXITIES IN THE DIAGNOSIS OF A PEDIATRIC PATIENT WITH POSSIBLE AUTOIMMUNE POLYGLANDULAR SYNDROME: A CASE REPORT

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Case presentation: A male patient was referred to an endocrinologist at 3 years of age due to recurrent episodes of malaise associated with arterial hypotension. He presented salt craving, cutaneous hyperpigmentation, abdominal pain, and lower limb pain. On physical examination, he appeared apathetic, underweight, hypotensive, and showed hyperpigmentation of the lips, gums, axillae, and orchiopexy scar. Given the suspicion of Addison's disease, laboratory tests revealed low cortisol levels associated with markedly elevated ACTH levels, confirming the diagnosis of Primary Adrenal Insufficiency, and treatment with hydrocortisone and fludrocortisone was initiated. After three years of follow-up, the patient developed hypothyroidism, raising the diagnostic hypothesis of autoimmune polyglandular syndrome type 2. Anti-21-hydroxylase antibody testing was requested, with a negative result. Nevertheless, the absence of antibody positivity does not exclude an autoimmune etiology. The patient remains under continuous endocrinological follow-up, with levothyroxine added to the treatment regimen. **Discussion:** Autoimmune polyglandular syndrome type 2 is the most common form of autoimmune endocrinopathy syndromes, characterized by the mandatory association of Addison's disease with autoimmune thyroid disease and/or type 1 diabetes mellitus. The most frequent clinical association occurs between primary adrenal insufficiency and Hashimoto's thyroiditis, known as Schmidt syndrome. Diagnosis is based on the functional evaluation of the affected glands and the identification of specific autoantibodies. However, the present case highlights an important diagnostic challenge, since the autoantibodies investigated were negative despite the strong clinical suspicion of autoimmunity. Among the main differential diagnoses is the coexistence of primary adrenal insufficiency and non-autoimmune hypothyroidism. **Final comments:** This case report highlights the diagnostic complexity of rare endocrinopathies, especially in the presence of overlapping clinical manifestations and the possibility of conflicting laboratory findings. Early clinical suspicion of Autoimmune Polyglandular Syndrome is essential to enable timely diagnosis, appropriate treatment, and continuous follow-up, contributing to the reduction of complications associated with the progression of endocrine dysfunctions and improving the patient's prognosis.

Keywords: addison disease; polyendocrinopathies, autoimmune; adrenal insufficiency.

PEDIATRIC ENDOCRINOLOGY

193 (3220) CONGENITAL HYPERINSULINISM ASSOCIATED WITH AN ABCC8 GENE MUTATION: A CASE REPORT

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Clinical case: A term newborn, large for gestational age (4,442 g), presented with persistent and severe hypoglycemia from the first day of life. Laboratory evaluation demonstrated inappropriate hyperinsulinemia, suppressed ketone bodies, and detectable C-peptide levels. Genetic testing confirmed a likely pathogenic homozygous mutation in the ABCC8 gene (c.210del:p.(His71Thrfs*), associated with poor response to diazoxide, which was clinically confirmed. An 18F-NOTA PET scan demonstrated diffuse pancreatic uptake, a finding typical of this mutation. Despite progressive use of octreotide and continuous enteral feeding, the patient continued to experience recurrent hypoglycemic episodes, characterizing clinical refractoriness. The patient was therefore transferred to a specialized tertiary care center and underwent approximately 95% subtotal pancreatectomy. In the recent postoperative period, the patient has shown satisfactory clinical evolution, with clinical stability and no further episodes of hypo- or hyperglycemia. **Discussion:** Neonatal congenital hyperinsulinism is the leading cause of persistent hypoglycemia during the neonatal period and is characterized by inappropriate insulin secretion despite low blood glucose levels. It is a rare and severe disease associated with a high risk of permanent neurological injury. In most cases, it is related to mutations in the ABCC8 and KCNJ11 genes, which encode the ATP-sensitive potassium channels of pancreatic beta cells, leading to continuous insulin release and hypoketotic hypoglycemia. Diagnosis is based on persistent hypoglycemia associated with inappropriately elevated insulin levels, suppression of ketone bodies, and response to glucagon, while genetic testing plays a key role in etiological confirmation. Treatment includes nutritional support, glucose infusion, and medications such as diazoxide and octreotide; however, severe forms are frequently refractory to clinical treatment and may require surgical intervention. **Conclusion:** Congenital hyperinsulinism associated with ABCC8 gene mutations is a rare and severe disorder associated with a high risk of permanent sequelae. Therefore, early diagnosis based on genetic investigation and timely referral to specialized centers in severe cases are essential to reduce morbidity among affected patients.

Keywords: congenital hyperinsulinism; neonatal hypoglycemia; ABCC8 mutation.

PEDIATRIC ENDOCRINOLOGY

194 (2903) CORD BLOOD INHIBIN B AND ITS ASSOCIATION WITH GESTATIONAL AGE AND CLINICAL PARAMETERS IN HEALTHY NEWBORNS

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Introduction: Inhibin B is a key marker of testicular function in early life. However, data on cord blood inhibin B levels and their correlation with clinical parameters in healthy newborns remain limited. **Objective:** This study aimed to evaluate the association between cord blood inhibin B levels and gestational age, as well as other clinical parameters, in healthy full-term male newborns. **Methods:** We conducted an observational study including healthy term male newborns within the first three days of life. Cord blood inhibin B levels were measured and correlated with gestational age, stretched penile length, and anthropometric data using appropriate statistical tests. **Results:** A total of 75 newborns were included. Median inhibin B levels were 964.55 pg/mL (IQR: 601–1328 pg/mL). A moderate negative correlation was observed between inhibin B and gestational age ($r = -0.35$, $p = 0.002$). No significant correlations were found between inhibin B and stretched penile length or other anthropometric parameters. **Conclusion:** Cord blood inhibin B levels appear to be influenced by gestational age but not by penile length or anthropometric measures. These findings suggest that gestational age should be considered when interpreting inhibin B levels in the neonatal period, particularly in the evaluation of suspected disorders of sex development.
Keywords: inhibin B; disorders of sex development; newborn.

PEDIATRIC ENDOCRINOLOGY

195 (3098) DYNAMIC GONADAL AXIS EVOLUTION IN 48,XXYY SYNDROME: A CASE REPORT

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Case presentation: A 13-year-old male was referred for micropenis and delayed puberty associated with attention-deficit/hyperactivity disorder and learning difficulties. Examination revealed penile length of 3.3 cm, bilateral testicular volume of 6 mL, and Tanner stage G2P2. Initial hormonal evaluation showed low testosterone with inappropriately normal gonadotropins, suggesting relative hypogonadotropism. Intermittent testosterone therapy was administered between 2021 and 2023, with subsequent pubertal progression. Karyotype analysis confirmed 48,XXYY syndrome. During follow-up, spontaneous pubertal development occurred, with progression to Tanner stage G5P5 by 18 years of age, final height of 176 cm, and penile length of 12 cm, although testicular volume remained reduced (6–8 mL). Recent laboratory evaluation demonstrated low-normal testosterone (~314 ng/dL), elevated FSH (~24 mIU/mL), and mildly increased LH (~7.6 mIU/mL), consistent with partial hypergonadotropic hypogonadism. Testicular ultrasound revealed markedly reduced testicular volumes (1.3–1.5 cm^3) with homogeneous parenchyma. **Discussion:** 48,XXYY syndrome is a rare sex chromosome aneuploidy within the Klinefelter spectrum, usually associated with hypergonadotropic hypogonadism secondary to primary testicular failure. This case is notable for its atypical endocrine evolution, characterized initially by relative hypogonadotropism followed by progression to hypergonadotropic hypogonadism during puberty. Elevated FSH levels associated with severe testicular hypotrophy and preserved virilization suggest progressive seminiferous tubular dysfunction with partial Leydig cell preservation. Neurobehavioral manifestations and cognitive impairment were also consistent with the classic phenotype. **Final comments:** This report highlights the phenotypic variability of 48,XXYY syndrome and reinforces the importance of longitudinal monitoring of the hypothalamic-pituitary-gonadal axis in sex chromosome aneuploidies.
Keywords: 48, XXYY syndrome; delayed puberty; sex chromosome aneuploidy.

PEDIATRIC ENDOCRINOLOGY

196 (2880) DYSLIPIDEMIA IN CHILDREN AND ADOLESCENTS: PREVALENCE AND ASSOCIATION WITH NUTRITIONAL STATUS IN A BRAZILIAN PEDIATRIC POPULATION

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Introduction: Dyslipidemias are a primary risk factor for cardiovascular diseases, whose atherosclerotic process begins in childhood. Childhood obesity drives atherogenic lipid profiles from early ages and represents a growing public health challenge. In Brazil, cardiovascular diseases account for approximately 30% of annual deaths. **Objective:** To investigate the prevalence of dyslipidemias and their association with nutritional status in children and adolescents attending pediatric reference outpatient clinics. **Methods:** Observational, crosssectional, and descriptive-comparative study with 169 participants aged 2 to 19 years with a mean age of 11.3 years (SD = 4.6; median = 11.2; IQR: 7.8–15.1), divided into a normal weight group (n = 70) and an overweight group (n = 99), according to World Health Organization criteria. Anthropometric, blood pressure, and complete lipid profile data were collected and classified according to the Brazilian Society of Pediatrics guidelines. Shapiro-Wilk, Mann-Whitney, chi-square, Spearman correlation, logistic regression, and receiver operating characteristic curve analyses were used, with a significance level of 5%. **Results:** The overall prevalence of dyslipidemia was 59.2% (100/169), being 44.3% in the normal weight group and 69.7% in the overweight group (p = 0.002). The overweight group showed significantly higher total cholesterol, low-density lipoprotein, with lower high-density lipoprotein (all p£0.002). Body mass index correlated with all lipid fractions (p < 0.001). The receiver operating characteristic curve showed an area under the curve of 0.717 for elevated low-density lipoprotein, with an optimal cutoff body mass index of 23.3 kilograms per square meter. The number needed to screen was 3.9 and the population attributable fraction for excess weight was 36.5%. Regular physical activity was associated with higher high-density lipoprotein levels (p = 0.032). **Conclusion:** Dyslipidemia prevalence is high and strongly associated with excess weight in childhood. Universal lipid screening in overweight or obese children is a cost-effective strategy. Early lifestyle interventions, especially promotion of regular physical activity, are priorities for reducing future cardiovascular morbidity and mortality.

Keywords: dyslipidemia; childhood obesity; cardiovascular risk.

PEDIATRIC ENDOCRINOLOGY

197 (3379) EFFECTS OF GLP-1 RECEPTOR AGONISTS ON CHILDHOOD OBESITY TREATMENT: IMPLICATIONS FOR PUBERTAL TIMING AND PRECOCIOUS PUBERTY A SYSTEMATIC REVIEW

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Introduction: Childhood obesity is associated with alterations in the hypothalamic-pituitary-gonadal (HPG) axis, affecting biological maturation and endocrine development, with consequences such as earlier pubertal timing, precocious menarche, and menstrual irregularities. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have demonstrated significant efficacy in reducing body mass index (BMI) in children aged ³6 years and adolescents with obesity. However, the effects of these therapies on pubertal progression and long-term reproductive homeostasis remain a gap in scientific knowledge. **Objective:** To analyze the impact of GLP-1 RAs on HPG axis modulation and the incidence of precocious puberty in children and adolescents with obesity. **Methods:** This systematic review followed PRISMA guidelines, with searches conducted in PubMed and Embase using the PICO strategy for children and adolescents with obesity treated with liraglutide, semaglutide, or exenatide. Randomized clinical trials assessing anthropometric outcomes and markers of sexual maturation, including Tanner stages, bone age, and menarche, were prioritized. Individuals with secondary causes of obesity, such as genetic obesity, endocrine disorders unrelated to common obesity, and recent use of medications associated with weight gain or loss, were excluded. **Results:** A total of 115 articles were identified through database searches, and 5 studies were included in the analysis. The studies demonstrated that GLP-1 RAs promote significant reductions in weight and BMI in children and adolescents with obesity. Semaglutide showed a mean BMI reduction of 16.1% in adolescents, liraglutide reduced BMI by 5.8% in children aged 6–12 years, and exenatide demonstrated reductions of up to 4.0% in BMI. Regarding pubertal timing, no relevant changes were observed in Tanner stages, menarche, linear growth, or bone age between treatment and placebo groups. Nevertheless, evidence remains limited due to the small number of studies and the short follow-up period of pediatric patients receiving GLP-1 RAs. **Conclusion:** Current evidence indicates that GLP-1 RAs are effective in reducing BMI in children and adolescents with obesity, without significant effects on biological maturation, Tanner stage progression, or timing of menarche. The scarcity of studies evaluating pubertal timing and precocious puberty highlights the need for longitudinal follow-up to ensure reproductive safety and HPG axis homeostasis.

Keywords: glucagon-like peptide-1 receptor agonists; pediatric obesity; pubertal timing.

PEDIATRIC ENDOCRINOLOGY

198 (3104) EFFICACY AND IMPACT ON FINAL ADULT HEIGHT OF MONTHLY VERSUS QUARTERLY GNRH AGONISTS IN CENTRAL PRECOCIOUS PUBERTY: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Central precocious puberty (CPP) is caused by the early activation of the hypothalamic-pituitary-gonadal axis and may impair final height and cause psychosocial repercussions. Treatment with GnRH analogs (GnRHa) is effective in suppressing puberty progression. However, uncertainties remain regarding the equivalence between monthly and quarterly formulations. Therefore, this study proposes a systematic review and meta-analysis comparing these therapeutic regimens in patients with CPP. **Objective:** To evaluate the clinical efficacy and impact on Final Adult Height (FAH) of quarterly GnRHa treatment compared with the monthly regimen in girls with central precocious puberty. **Methods:** This systematic review and meta-analysis followed PRISMA guidelines and the recommendations of the Cochrane Handbook for Systematic Reviews of Interventions and was registered in PROSPERO under the identification number [CRD420261382880]. Literature searches were conducted in PubMed, Embase, CENTRAL, Web of Science, Scopus, LILACS, and MedRxiv for studies published from 2022 onward, without language restrictions. Search strategies included controlled vocabularies, free-text terms, and Boolean operators. Methodological quality and risk of bias of the included observational studies were independently assessed using the Newcastle-Ottawa Scale (NOS). **Results:** Of the 939 records identified, 6 studies were selected (n = 682). There's no statistically significant difference between quarterly and monthly administration of GnRH analogs (SMD –0.04; 95% CI - 0.30 to 0.23; p = 0.69). Data consistency was confirmed by zero heterogeneity (I2 = 0,00%). In the subgroup analysis, efficacy remained comparable at both 6 months and 1 year of follow-up (p = 0.83 for subgroup differences), suggesting that the quarterly regimen is a valid and effective therapeutic alternative to the monthly gold standard. **Conclusion:** This systematic review and meta-analysis demonstrated no significant differences between monthly and quarterly GnRH agonist regimens in girls with central precocious puberty, with similar efficacy regarding pubertal suppression, bone age progression, growth outcomes, and final adult height. In addition, the quarterly regimen may improve convenience and treatment adherence due to the lower number of injections, without compromising therapeutic effectiveness. However, the limited number of long-term studies should be considered when interpreting these findings.

Keywords: central precocious puberty; GnRH agonists; final adult height.

PEDIATRIC ENDOCRINOLOGY

199 (3351) EFFICACY OF WEIGHT MANAGEMENT INTERVENTIONS IN CHILDREN AND ADOLESCENTS WITH AUTISM SPECTRUM DISORDER (ASD): A SYSTEMATIC REVIEW

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Introduction: Children and adolescents with ASD have an overweight and obesity prevalence of 30%–55%, higher than the general pediatric population. Selective eating, sedentary behavior, sensorimotor limitations, and antipsychotic use reduce the effectiveness of conventional approaches; specific interventions remain underexplored. **Objective:** To evaluate the efficacy of structured weight management interventions — dietary, adapted physical activity, behavioral, pharmacological, and multicomponent — on BMI/z-score, body composition, metabolic parameters, and quality of life in children and adolescents with ASD, compared to usual care. **Methods:** Systematic review (PRISMA), with searches in PubMed, Embase, and ScienceDirect (2016–2025), using the descriptors: Autism Spectrum Disorder AND Body Mass Index OR Obesity AND physical activity OR multicomponent intervention. Primary studies with patients £18 years and confirmed ASD (DSM-5/ICD-11), with an adequate comparator and outcomes of BMI z-score, body composition, or metabolic parameters were included. Screening was conducted by two independent reviewers using Rayyan. **Results:** From 545 records following title/abstract screening and full-text review, 7 primary studies were included, predominantly RCTs (n = 4): physical activity (n = 3), parent-mediated multicomponent (n = 3), and pharmacological (n = 1); sample sizes ranged from 10 to 225 participants, with intervention duration from 8 weeks to 25 months. Physical activity improved body composition and cardiorespiratory fitness without consistent reductions in BMI. Multicomponent approaches reduced BMI z-score (-0.02 to -0.25) and improved quality of life. A 48-week exercise intervention improved lipid profile (HDL +5.2 mg/dL; LDL -7.7 mg/dL) without significant impact on BMI. Metformin attenuated antipsychotic-induced weight gain (-2.73 kg; P = 0.003), without significant improvements in metabolic outcomes. Pilot studies demonstrated high feasibility and satisfaction rates, with parental adherence of 92%–98%. **Conclusion:** Structured interventions, particularly parent-mediated multicomponent and behavioral approaches, demonstrated efficacy in weight management, with benefits on BMI, body composition, metabolic parameters, and quality of life. Exercise alone showed limited impact on BMI but benefits in physical fitness and lipid profile. Evidence remains limited by methodological heterogeneity and small sample sizes. Larger, long-term randomized controlled trials are needed.

Keywords: autism spectrum disorder; childhood obesity; weight management.

PEDIATRIC ENDOCRINOLOGY

200 (3551) ENDOCRINE-METABOLIC EFFECTS OF BISPHENOL AN EXPOSURE IN CHILDHOOD

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Introduction: Bisphenol A (BPA) is an endocrine disruptor widely used in the manufacture of polycarbonate plastics and epoxy resins found in food packaging. Early exposure to this compound has been associated with hormonal and metabolic alterations in the pediatric population due to increased biological susceptibility during development. Recent studies suggest an association between bisphenol A exposure, childhood obesity, and cardiometabolic disorders. **Objective:** To evaluate the association between bisphenol A exposure and metabolic alterations in children. **Methods:** A systematic literature review was conducted in accordance with PRISMA guidelines, based on the PICO strategy, considering children as the population, bisphenol A exposure as the exposure factor, individuals with different levels of exposure as the comparison group, and metabolic alterations as the outcome. Searches were performed in the PubMed, SciELO, Scopus, and Virtual Health Library databases, including studies published between 2018 and 2026 in English, Portuguese and Spanish. The descriptors "bisphenol A," "endocrine disruptors," "childhood," "obesity," and "pediatric population" were combined using the Boolean operators AND and OR. Inclusion criteria comprised observational studies evaluating bisphenol A exposure and its metabolic outcomes in the pediatric population. Duplicate articles, case reports, editorials, and studies lacking clearly described methodological criteria were excluded. **Results:** The analyzed studies demonstrated high exposure to bisphenol A among pediatric populations, mainly associated with the frequent consumption of foods stored in plastic packaging and canned products. An association was observed between higher urinary levels of the compound and an increased risk of obesity, insulin resistance, and type 2 diabetes mellitus. Furthermore, hormonal alterations related to insulin secretion and glucose metabolism were identified. **Conclusion:** Exposure to bisphenol A is significantly associated with endocrine-metabolic alterations in children, especially obesity and insulin resistance. These findings reinforce the need for educational strategies and public policies aimed at reducing early exposure to endocrine disruptors.

Keywords: bisphenol A; endocrine disruptors; childhood obesity.

PEDIATRIC ENDOCRINOLOGY

201 (3558) EPIDEMIOLOGY OF CHILDHOOD AND ADOLESCENT OBESITY (AGES 4-16) IN BRAZIL

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Introduction: Childhood and adolescent obesity has become one of the greatest public health challenges of the 21st century, directly linked to the early onset of comorbidities such as hypertension, dyslipidemia, and psychosocial distress. In Brazil, the ongoing nutritional transition and a sharp increase in ultra-processed food consumption have accelerated excessive weight gain from early childhood through adolescence. Continuous monitoring of this trend is essential to support the planning and implementation of effective public health policies. **Objective:** To analyze the epidemiological profile of obesity among children and adolescents aged 4 to 16 years in Brazil, mapping its prevalence and regional distribution through public healthcare system data. **Methods:** A descriptive epidemiological study was conducted using secondary data from Datasus, extracted from the Food and Nutritional Surveillance System (Sisvan). Nutritional status records of individuals aged 4 to 16 monitored in Primary Healthcare units were analyzed. The criteria for nutritional classification were based on body mass index (BMI)-for-age according to World Health Organization (WHO) growth charts, utilizing a cutoff point of z-score > +2 to define obesity. **Results:** Indicators show that obesity affects approximately 15% of the population aged 4 to 16 evaluated within the public health system, with an alarming worsening trend during the transition into adolescence. When considering the full spectrum of excessive weight (overweight combined with obesity and severe obesity), the rate exceeds 32% in this group. Geographically, the South and Southeast regions maintain the highest historical rates, associated with higher urbanization and physical inactivity. However, the North and Northeast regions have shown the fastest growth over the last decade, demonstrating the universalization of the condition regardless of regional income. **Conclusion:** The prevalence of obesity among individuals aged 4 to 16 in Brazil is critically high and expanding across all macro-regions. This scenario demands urgent intersectoral interventions focused on regulating school food environments, promoting physical activity, and strengthening school health programs tailored to both children and adolescents.

Keywords: adolescent obesity; epidemiological profile of obesity; public health system.

PEDIATRIC ENDOCRINOLOGY

202 (3178) IGF-I RESISTANCE AND GROWTH PLATE DYSFUNCTION IN SIBLINGS WITH TONSL MUTATION: AN ANALYSIS OF SPONASTRIME DYSPLASIA

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SPONASTRIME dysplasia (SPO) is a rare disorder caused by TONSL mutations, characterized by short-limbed dwarfism. We evaluate the impact of long-term therapy and peripheral resistance in two siblings. A 14.9-year-old female proband presented with disproportionate short stature of prenatal onset. At the start of treatment (age 10.2y), her height was 118.4 cm (Z-score: –3.22), weight was 31.8 kg (+0.3 SDS), and bone age was 11 years (chronological age 9.9y) with breast development Tanner stage 3. Evaluation revealed a normal GH peak (15 ng/mL) but significantly elevated basal IGF-I (343 ng/mL) and IGFBP-3 (7 mcg/mL). These values exceed those typically reported in recent literature (*e.g.*, Yao *et al.*, 2025), confirming severe peripheral IGF-I resistance. An intensive protocol was initiated with high-dose rhGH (0.15 U/kg/day), GnRH analog, and letrozole (prescribed for 19 months). The most significant response occurred in the first year, with a growth velocity (GV) of 7.7 cm/year (Z-score: –2.78). Notably, despite the use of letrozole, bone age advanced rapidly to 14 years by age 11.8y, leading to the discontinuation of the aromatase inhibitor. By age 13.1y, her height was 134.5 cm (Z-score: –3.29), representing a return to her original growth canal. Her older brother (21.2 y), untreated, reached a final height of 156 cm (Z-score: –3.3). Despite his target height of 185.5 cm, he suffered a ~30 cm deficit due to rapid bone age advancement during puberty. While the proband remains far below her target (172.5 cm), the intervention avoided the pubertal "growth escape" seen in her brother, maintaining her basal percentile. The TONSL gene is crucial for DNA replication-coupled repair. In the bone epiphysis, TONSL integrity is vital for chondrocyte survival and proliferation. Mutations lead to accumulated genomic damage, causing cell cycle arrest in the growth plate. The elevated IGF-I and IGFBP-3 levels found here serve as markers of this intrinsic cellular failure. This suggests that the primary barrier to growth is a cellular defect at the epiphyseal level that systemic hormonal therapy cannot fully overcome. Pharmacological intervention in TONSL mutations provides limited benefits. While high-dose rhGH, pubertal and bone age advance blockade may stabilize the patient within their original growth canal, they fail to achieve significant catch-up toward the genetic target due to severe, intrinsic growth plate resistance.

Keywords: SPONASTRIME dysplasia; TONSL gene; IGF-I resistance.

PEDIATRIC ENDOCRINOLOGY

203 (3155) INCOMPLETE PUBERTY AND PRIMARY HYPERGONADOTROPIC HYPOGONADISM FOLLOWING TREATMENT OF TESTICULAR DIFFUSE LARGE B-CELL NON-HODGKIN LYMPHOMA IN A PEDIATRIC PATIENT: A CASE REPORT

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Case presentation: A male patient diagnosed at age 5 with primary testicular diffuse large B-cell non-Hodgkin lymphoma, treated with unilateral orchiectomy, chemotherapy, and scrotal radiotherapy at a total dose of 24 Gy. Endocrinological follow-up began at age 11 for pubertal development assessment. At age 13, he underwent a human chorionic gonadotropin stimulation test, showing testosterone rising from 10 to 612 ng/dL. He developed spontaneous but incomplete puberty, with slow progression from Tanner P2 to P4 of over four years without reaching P5, and maximum testicular volume of 12 mL. Progressive gonadal deterioration followed, with FSH rising from 2.8 to 20.8 IU/L, total testosterone decreasing from 337 to 130 ng/dL, and free testosterone from 7.19 to 4.60 ng/dL, suggesting insufficient Leydig cell functional reserve. At age 18, spermiogram revealed azoospermia, confirming complete seminiferous tubule failure and indicating testosterone replacement therapy. **Discussion:** Primary testicular diffuse large B-cell non-Hodgkin lymphoma is rare and aggressive, affecting mainly elderly men, with pediatric diagnosis being exceptionally uncommon. Multimodal treatment imposes high risk of primary gonadal dysfunction via direct testicular damage. Germ cells are highly radiosensitive, with spermatogenesis failure at doses as low as 0.15 Gy, potentiated by alkylating agents. Leydig cells, though more radioresistant, are vulnerable above 20 Gy, with increased failure risk and pubertal plateau in prepubertal boys. Gonadotoxic damage resulted in spontaneous but incomplete puberty, reflecting partial Leydig cell reserve with progressive depletion confirmed on serial hormonal measurements. Rising FSH reflects predominant seminiferous damage, while falling testosterone confirms gonadal dysfunction, establishing primary hypergonadotropic hypogonadism. Following azoospermia confirmed by spermiogram, testosterone replacement is indicated for androgenic normalization, complete sexual development, bone preservation, and prevention of metabolic and cardiovascular impairment. **Final comments:** This case underscores the need for prolonged surveillance in oncologic survivors, especially pediatric patients, given the risk of late and progressive endocrine sequelae. Prospective pediatric evidence remains scarce, reinforcing this report's relevance. Monitoring is essential for early detection of gonadal dysfunction and prevention of bone, metabolic, and cardiovascular complications.

Keywords: incomplete puberty; hypergonadotropic hypogonadism; non-Hodgkin lymphoma.

PEDIATRIC ENDOCRINOLOGY

204 (2879) PREVALENCE OF ELEVATED LIPOPROTEIN(A) LEVELS IN CHILDREN AND ADOLESCENTS: IMPACT ON EARLY IDENTIFICATION OF CARDIOVASCULAR RISK FACTORS

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Introduction: Cardiovascular diseases are the leading cause of global mortality, with evidence that the atherosclerotic process begins in childhood and adolescence. Lipoprotein(a) is a cardiovascular risk biomarker with predominantly genetic determination, whose levels remain stable from birth regardless of environmental factors and nutritional status. Data on its prevalence in Brazilian pediatric populations are scarce. **Objective:** To determine the prevalence of elevated lipoprotein(a) levels and their association with conventional cardiovascular risk factors in children and adolescents attending a reference center. **Methods:** Observational, cross-sectional, and descriptive study involving 94 patients aged 2 to 19 years with a mean age of 11.7 years (SD = 5,08; median = 11.5), stratified into an overweight group (n = 56; 59.6%) and a normal weight group (n = 38; 40.4%). Anthropometric data, blood pressure, complete lipid profile, and plasma lipoprotein(a) by turbidimetric immunoassay were collected. Statistical analysis used parametric and nonparametric tests according to data distribution, with a significance level of 5%. **Results:** The prevalence of elevated lipoprotein(a) was 45.7% at the 30 milligrams per deciliter threshold and 25.5% at the 50 milligrams per deciliter threshold, the high-risk cutoff of the European Atherosclerosis Society. No significant difference in lipoprotein(a) levels was found between groups by nutritional status ($p = 0.911$), sex ($p = 0.551$), or age group ($p = 0.964$), confirming its genetic independence. The overweight group showed significantly higher systolic blood pressure ($p < 0.001$), diastolic blood pressure ($p < 0.001$), low-density lipoprotein ($p = 0.002$), and total cholesterol ($p = 0.006$). Combined cardiovascular risk, defined as coexistence of dyslipidemia and lipoprotein(a) at or above 30 milligrams per deciliter, was identified in 31.9% of the sample. **Conclusion:** Approximately one quarter of the evaluated children and adolescents present lipoprotein(a) levels compatible with high cardiovascular risk, independently of nutritional status, sex, and age group. These findings reinforce the need to include lipoprotein(a) measurement in routine pediatric cardiovascular assessment as an early screening and prevention strategy.

Keywords: lipoprotein(a); cardiovascular risk factors; dyslipidemia.

PEDIATRIC ENDOCRINOLOGY

205 (3542) PUBERTAL ARREST AND GROWTH DECELERATION SECONDARY TO MACROPROLACTINOMA IN A MALE ADOLESCENT: A CASE REPORT

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Case report: A 16-year-old male had been followed for growth assessment since the age of 13 years and 4 months. Initial evaluation demonstrated bone age compatible with chronological age, growth within the familial target range, and pubertal development corresponding to Tanner stage G4P3. At 16 years and 2 months, he returned with complaints of reduced growth velocity and painless breast enlargement. Physical examination revealed a high-pitched voice and persistence of Tanner stage G4P3, indicating pubertal stagnation. Further investigation demonstrated bone age compatible with 16 years, while breast ultrasonography excluded true gynecomastia. Laboratory evaluation revealed marked hyperprolactinemia (332 ng/mL) associated with low testosterone levels (105 ng/dL). Cortisol, growth hormone (GH), adrenocorticotropic hormone (ACTH), and follicle-stimulating hormone (FSH) levels were within normal reference ranges. Due to suspicion of a prolactin-secreting pituitary tumor, magnetic resonance imaging of the sellar region was performed, revealing a lesion in the right portion of the anterior pituitary gland consistent with a pituitary macroadenoma. Treatment with cabergoline 0.5 mg twice weekly was initiated. After one year, prolactin levels decreased to 12.1 ng/dL, testosterone increased to 601 ng/dL, and tumor size was reduced. **Discussion:** Delayed puberty in males is defined by absence of testicular enlargement by 14 years of age, delayed progression between pubertal stages, or prolonged pubertal duration. In this case, although puberty began at the expected age, prolactin hypersecretion secondary to the macroadenoma interrupted pubertal progression, resulting in incomplete sexual maturation and impaired growth. **Final comments:** Pituitary macroadenoma should be considered in male adolescents presenting with pubertal arrest and growth deceleration. Early recognition and treatment with dopamine agonists are essential to minimize adverse effects on physical growth, pubertal development, and psychosocial well-being.

Keywords: macroadenoma; pubertal stagnation; prolactin.

PEDIATRIC ENDOCRINOLOGY

206 (3012) REAL-WORLD COMPARISON OF LONG-ACTING VS. SHORT-ACTING GROWTH HORMONE THERAPY IN PEDIATRIC PATIENTS WITH GROWTH HORMONE DEFICIENCY IN THE UNITED STATES

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Pediatric growth hormone deficiency (GHD) is commonly treated with daily short-acting growth hormone (SAGH) treatment, but adherence to SAGH can be suboptimal, potentially limiting its effectiveness. Long-acting growth hormone (LAGH) therapies reduce the frequency of injections and may improve adherence and persistence. This study evaluates real-world clinical outcomes and treatment patterns in patients treated with LAGH or SAGH. This retrospective cohort study used the Komodo Research Dataset and clinical observation data, which included patients aged 3-18 years with ³1 GHD diagnosis and ³1 pharmacy claim for LAGH or SAGH. The index date was defined as the date of the first qualifying treatment. Patients had to have continuous medical and pharmacy claims for ³6 months pre-index (baseline) after August 1, 2021, and ³6 months post-index (follow-up) before January 31, 2025. The LAGH cohort comprised patients with ³1 LAGH claim, and the SAGH cohort included patients with SAGH claims only. After matching on age, propensity score matching (PSM) adjusted for differences in baseline characteristics of the SAGH cohort to match those of the LAGH cohort. The association between treatment adherence and height change pre- and post-index was assessed using a mixed model, adjusted for age, gender, and baseline weight. Before PSM, the mean ± SD age of the LAGH cohort (N = 1,492) was 12.0 ± 3.0 years, and 73.9% were male; 51.5% used SAGH at baseline. Of the 12,544 patients in the SAGH cohort pre-matching, the mean ± SD age was 12.0 ± 3.1 years; 74.2% were male. Height gains and height velocity were similar for the LAGH and SAGH cohorts pre-matching, with greater differences between cohorts post-matching (N=1,488 for both cohorts; the LAGH cohort exhibited greater increases than the SAGH cohort. Greater weight and BMI increases were observed for the LAGH cohort. Pre-matching, adherence was higher for the LAGH cohort (72.3%) than for the SAGH cohort (51.5%), and discontinuation was lower (21.0% vs. 49.8%); although PSM moderated the difference in adherence and discontinuation rates, these rates continued to favor the LAGH cohort. The model found that non-adherence is associated with a lower growth rate, particularly for SAGH. Greater improvements in adherence and persistence were observed for LAGH compared with SAGH. Adherence was associated with better height outcomes. These findings suggest LAGH may support better long-term outcomes in patients with pediatric GHD.
Keywords: growth hormone deficiency; long-acting growth hormone; real world evidence.

PEDIATRIC ENDOCRINOLOGY

207 (3412) SRY-NEGATIVE 46,XX TESTICULAR DISORDER OF SEX DEVELOPMENT: CASE REPORT

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Case presentation: Disorders of sex development (DSD) are uncommon conditions with significant clinical and psychosocial impacts. A rare entity is SRY-negative 46,XX testicular DSD, with considerable relevance due to the challenges of gonadal differentiation, pubertal development, and sex assignment. The report describes the case of E.C., raised as male, referred to pediatric endocrinology at 10 months for atypical genitalia. Examination revealed perineal hypospadias, inguinal gonads, scrotal rugae and enlarged phallus. Laboratory showed normal 17-hydroxyprogesterone and androstenedione, excluding congenital adrenal hyperplasia (CAH). Karyotype was 46,XX, and SRY gene analysis was negative. Ultrasonography showed inguinal gonads and absent uterine structures. Gonadal biopsy confirmed seminiferous tubules with Sertoli cells. Cystourethrography identified a patent urethra. He underwent surgical correction, with a good postoperative course, but follow-up was interrupted. He returned at 10 years old with Tanner stage P2G1 and prepubertal gonadotropins. At almost 12 years, pubertal induction with testosterone led to increased body hair, penile growth, and a height gain of 5 cm. He remains under follow-up with assessment of inhibin B levels, hormonal and molecular tests. **Discussion:** Atypical genitalia is part of the DSD spectrum, characterized by alterations in chromosomal, gonadal, or anatomical sex development. CAH due to 21-hydroxylase deficiency is the leading etiology in 46,XX individuals, while the presence of testicular tissue in SRY-negative patients remains rare. In this case, gonadal biopsy was crucial for diagnostic confirmation and therapeutic decision-making. Early diagnosis and long-term multidisciplinary follow-up involving endocrinology, genetics, urology, and pathology significantly influence clinical outcomes. **Conclusion:** This case highlights the importance of an integrated diagnostic approach in DSD, particularly in SRY-negative 46,XX testicular DSD. The combination of clinical, hormonal, genetic, and histological evaluation was essential for individualized patient management. Case reports such as this one expand current knowledge regarding uncommon DSD presentations, contributing to earlier recognition, therapeutic decision-making, and reinforcing the importance of a multidisciplinary approach.
Keywords: disorder of sex development; SRY-negative; atypical genitalia.

PEDIATRIC ENDOCRINOLOGY

208 (3373) TEMPORAL TREND OF OUTPATIENT PROCEDURES RELATED TO THE TREATMENT OF PRECOCIOUS PUBERTY IN BRAZIL BEFORE AND AFTER THE COVID-19 PANDEMIC

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Introduction: Central precocious puberty is characterized by the premature activation of the hypothalamic-pituitary-gonadal axis, compromising growth and psychosocial development. Treatment with gonadotropin-releasing hormone (GnRH) analogs, particularly triptorelin and leuprorelin, represents the standard therapeutic approach available through the Brazilian Unified Health System (SUS). **Objective:** To analyze the temporal trend of outpatient procedures related to the treatment of precocious puberty in Brazil before, during, and after the COVID-19 pandemic. **Methods:** An ecological, retrospective, descriptive study was performed using secondary data from the Outpatient Information System of the Brazilian Unified Health System (SIA/SUS), accessed through Datasus. Procedures related to triptorelin and leuprorelin formulations indicated for precocious puberty treatment between January 2018 and December 2025 were included. Data were stratified by year of registration, and approved procedures, monthly averages and temporal trends were analyzed according to the pre-pandemic, pandemic, and post-pandemic periods. Simple linear regression analysis was performed to assess temporal trends in medication utilization over time. **Results:** A progressive increase in outpatient procedures related to GnRH analog therapy was observed throughout the study period. Triptorelin 3.75 mg demonstrated a significant upward temporal trend ($\beta = 139.36$; $p < 0.05$; $R^2 = 0.778$), corresponding to an estimated increase of approximately 139 users per year. Triptorelin 11.25 mg showed progressive growth over time ($\beta = 0.000139$; $p < 0.01$; $R^2 = 0.841$). Similarly, leuprorelin 3.75 mg exhibited an upward trend ($\beta = 0.000539$; $p < 0.05$; $R^2 = 0.714$), indicating a consistent increase in procedures during the study period. In contrast, leuprorelin 11.25 mg did not demonstrate a significant temporal trend. During the first years of the COVID-19 pandemic, fluctuations in outpatient records were identified, followed by gradual recovery in subsequent years. These findings suggest increased demand for pubertal suppression therapies and broader access to pediatric endocrinology follow-up within the Brazilian public health system after the pandemic. **Conclusion:** There was a temporal increase in outpatient procedures related to the treatment of precocious puberty in Brazil, reinforcing the importance of maintaining and expanding pediatric endocrinology care within the Brazilian Unified Health System (SUS).

Keywords: precocious puberty; gonadotropin-releasing hormone; endocrinology.

BONE AND MINERAL METABOLISM

209 (3026) PRIMARY HYPERPARATHYROIDISM AND INCIDENTAL PAPILLARY THYROID MICROCARCINOMA IN VON HIPPEL–LINDAU DISEASE: AN EXPANDING ENDOCRINE PHENOTYPE

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Case presentation: A 57-year-old woman with genetically confirmed Von Hippel-Lindau (VHL) disease, pathogenic variant c.46A>G, p.(Tyr156Cys) in heterozygosity, under active surveillance presented with fatigue, cognitive impairment, irritability, and mood instability. Surgical history included bilateral adrenalectomy (paraganglioma) and left nephrectomy. Serial monitoring over 18 months showed persistently elevated PTH (172, 109, and 169 pg/mL; ref. 18.5–88) and mild hypercalcemia (10.4–11.7 mg/dL; ref. 8.4–10.2). At urgent evaluation, serum calcium reached 13.6 mg/dL, PTH was 169 pg/mL, creatinine 1.99 mg/dL (ref. 0.7–1.3; eGFR 27 mL/min/1.73 m²). Neuropsychiatric manifestations, confusion, irritability, and behavioral changes, were consistent with severe hypercalcemia. A thyroid nodule (TI-RADS 4, 2.2 cm) with prior benign cytology was under surveillance. Total thyroidectomy with resection of the left superior parathyroid gland was performed. Intraoperative PTH decreased 75% within 20 minutes (169.3 to 42.9 pg/mL), confirming biochemical cure. Calcium normalized with complete resolution of symptoms. **Discussion:** Histopathology confirmed a parathyroid adenoma (1.2 × 1.0 × 1.0 cm). The thyroid specimen unexpectedly revealed an incidental classical papillary thyroid microcarcinoma (0.4 cm, pT1a), with negative margins and no lymphovascular or perineural invasion, undetected on imaging and unsuspected on prior cytology. Primary hyperparathyroidism is not part of the recognized VHL spectrum, which classically includes hemangioblastomas, clear cell renal cell carcinoma, paraganglioma, and pancreatic neuroendocrine tumors. Only rare cases have been reported. The pVHL functions as an E3 ubiquitin ligase complex regulating hypoxia-inducible factor degradation; its loss may promote tumorigenesis beyond the canonical phenotype. Emerging evidence also implicates reduced VHL expression in thyroid carcinogenesis, lending biological plausibility to this co-occurrence. **Final comments:** This case highlights that severe hypercalcemia with neuropsychiatric manifestations in VHL, even in the absence of biochemical evidence of catecholamine excess, should prompt evaluation for primary hyperparathyroidism. The incidental papillary microcarcinoma underscores the value of systematic surveillance and histopathological analysis of surgical specimens. These findings support the inclusion of parathyroid evaluation in the surveillance of VHL patients presenting with hypercalcemia.

Keywords: Von Hippel-Lindau disease; primary hyperparathyroidism; papillary thyroid carcinoma.

BONE AND MINERAL METABOLISM

210 (2885) ASSESSMENT OF CALCIUM INTAKE AMONG ELDERLY PATIENTS AT A UNIVERSITY HOSPITAL IN THE INTERIOR OF THE NORTHEAST

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Introduction: Brazil's demographic transition indicates that, by 2060, one-third of the population will be elderly, leading to an increase in the incidence of osteoporosis and fractures and a postoperative mortality rate of 23.6% among hospitalized patients. Although the combination of calcium and vitamin D can reduce the risk of hip fractures by 16%, average calcium intake in Brazil remains below the recommended level. **Objective:** To assess calcium intake in individuals over 60 years of age treated at a university hospital in the interior of the Northeast region, describing their sociodemographic profile and dietary habits related to calcium. **Methods:** This was a cross-sectional study conducted in outpatient clinics between September 2025 and February 2026. The sample included individuals of both sexes, aged ³ 60 years, excluding those using calcium supplements. The adequacy criterion adopted was an intake of ³ 1,000 mg/day, in accordance with the Clinical Protocol and Therapeutic Guidelines for Osteoporosis. Statistical analysis was performed using SPSS 30.0 software, employing the chi-square test, ANOVA, and t-test, with a significance level of $p < 0.05$. **Results:** The sample revealed a predominance of the 70–79 age group (59.0%), with a female majority (57.0%), of mixed-race (43.0%), low educational attainment (61.0% with less than 8 years of schooling), and 71.0% had a family income between 2 and 4 minimum wages. A high prevalence of hypertension (74.0%) and diabetes (44.0%) was noted, as well as a higher history of fractures among women (17.5%. The prevalence of inadequate intake reached 90% of the sample, and the average daily calcium intake was 614.69 mg (±283.44 mg). There were no significant differences in intake across age groups, sex, or race, nor in relation to the presence of chronic diseases, smoking, or vitamin D supplementation. However, educational attainment proved to be a determining factor: older adults with ³ 8 years of schooling had higher intake (736.09 mg) compared to those with lower educational attainment (537.07 mg); $p < 0.001$. **Conclusion:** dietary calcium intake among older adults is critical and insufficient. The high rate of inadequacy highlights a scenario of vulnerability to osteometabolic diseases, and the positive correlation between higher education and better nutritional intake suggests that access to information and socio-educational level are fundamental pillars for dietary quality.

Keywords: calcium; elderly nutrition; fractures bone.

BONE AND MINERAL METABOLISM

211 (3041) ATYPICAL FEMORAL FRACTURE MANAGED CONSERVATIVELY: A CASE REPORT

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Case report: A 74-year-old woman with osteoporosis secondary to primary hyperparathyroidism and previous corticosteroid use for idiopathic thrombocytopenic purpura. She underwent right inferior parathyroidectomy in 2014 with pathology confirming parathyroid adenoma. She received irregular treatment for osteoporosis with alendronate between 2016 and 2023 and developed therapeutic failure due to poor adherence, characterized by a 19% decrease in bone mineral density (BMD) and CTX of 0.750 ng/mL. Therefore, treatment with zoledronic acid was indicated. The patient received the first dose in October 2023 and the second dose in October 2024. Bone densitometry performed prior to zoledronic acid therapy demonstrated a T-score of –2.2 SD at the lumbar spine, –2.6 SD at the total hip, and –2.5 SD at the femoral neck. In December 2024, she developed pain in the left lower limb lasting for several weeks. Orthopedic evaluation diagnosed an incomplete atypical fracture of the left femoral shaft; however, at the time of assessment, she was already asymptomatic, and conservative management was chosen. Radiographs demonstrated fracture healing, with no indication for prophylactic fixation, and conservative treatment was maintained. Teriparatide was prescribed in December 2025 aiming to improve bone mass and assist fracture healing; however, the medication has not yet been approved. Currently, the patient is ambulating without support, asymptomatic, receiving guidance regarding fall prevention, vitamin D supplementation, and adequate calcium intake. **Discussion:** Atypical femoral fractures are associated with prolonged antiresorptive therapy, especially bisphosphonates, most commonly affecting the subtrochanteric and femoral shaft regions. Complete fractures are usually treated with intramedullary nail fixation. In incomplete fractures, conservative management has a high failure rate, with progression to surgical treatment in a significant proportion of patients. Prophylactic fixation demonstrates high consolidation rates, whereas spontaneous healing after bisphosphonate discontinuation occurs in approximately 36.4% of cases. Adjuvant teriparatide therapy has been investigated due to its potential to accelerate bone healing, although robust evidence supporting formal recommendation is still lacking. **Final comments:** This report describes an incomplete atypical femoral shaft fracture with favorable outcome after prolonged conservative management preserving the patient's independence.
Keywords: atypical fracture; conservative management; antiresorptive therapy.

BONE AND MINERAL METABOLISM

212 (3079) CHANGES IN BONE MINERAL DENSITY AFTER GENDER-AFFIRMING HORMONE THERAPY IN TRANSGENDER INDIVIDUALS: A SYSTEMATIC REVIEW

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Introduction: Gender-affirming hormone therapy (GAHT) is based on estrogen and testosterone regimens. Its importance for quality of life is undeniable, but its impact on bone mineral density (BMD) requires further investigation. **Objective:** To evaluate the impact of GAHT on BMD. **Methods:** This systematic review was conducted using the PICO strategy, following the PRISMA guidelines. A search was conducted for studies published in the last 5 years in English, Portuguese, and Spanish in PubMed, Scopus, and Embase databases, using the MeSH terms Transgender Persons, Hormone Replacement Therapy, and Bone Density, combined with the Boolean operator "AND". 53 studies were identified and underwent double-blind screening. Inclusion criteria were GAHT with testosterone or estrogen, analysis of BMD by dual-energy X-ray absorptiometry, minimum follow-up of 6 months, observational studies, multicenter studies, and randomized clinical trials. Studies using isolated pubertal blockers and diseases interfering with bone metabolism were excluded, totaling 5 articles for data collection to answer the question: "What is the impact of gender-affirming hormone therapy on bone mineral density?". **Results:** Transgender women who used cyproterone acetate in association presented an increase in lumbar spine BMD after GAHT, both at 12 months (0.97 ± 0.16 to 1.02 ± 0.14 g/cm²; $p < 0.001$) and at 6 months (+3.9%; $p = 0.0051$). In the long term (9 years), the value tends to decrease (-0.87; 95% CI). Transgender men undergoing isolated GAHT present reduction in femoral neck BMD (0.81 ± 0.12 to 0.79 ± 0.13 g/cm²; $p < 0.05$) after 12 months, and in the long term, show recovery of z-scores similar to baseline. In addition, the mean Z-score for hip and spine was 0.91 ± 1.03 and 0.38 ± 0.90 compared with genetic sex, whereas in transgender women, in the same context, it was –0.85 ± 0.57 and –2.13 ± 0.90. In adolescents, transgender boys showed no significant increase in BMD, both with combined therapy with gonadotropin-releasing hormone agonists (0.05 ± 0.03 g/cm²; $p = 0.083$) and with isolated GAHT (0.01 ± 0.04 g/cm²; $p = 0.944$) over 12 months, with both showing similar values after this period. **Conclusion:** GAHT appears to have no significant impact on BMD. Feminizing hormone therapy showed a small increase on BMD while masculinizing hormone therapy showed neutral effect in BMD. Prospective studies with longer follow-up and larger sample sizes are needed to better define the impact of GAHT on BMD.
Keywords: transgender persons; gender-affirming procedures; bone density.

BONE AND MINERAL METABOLISM

213 (2922) CLINICAL IMPLICATIONS OF CONGENITAL HYPOPARATHYROIDISM DUE TO AN ACTIVATING MUTATION IN THE CALCIUM-SENSING RECEPTOR GENE IN AN ADOLESCENT PATIENT: A CASE REPORT

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Case presentation: Female, 17 years old, followed at a tertiary care center for idiopathic congenital hypoparathyroidism. She was admitted for persistent cramps and paresthesias, asthenia, somnolence, learning difficulties, and memory problems. On examination, positive Chvostek and Trousseau signs and extensor weakness of the left upper extremity were noted. She presented with hypocalcemia (corrected calcium 6.4 mg/dL), hyperphosphatemia (8.2 mg/dL), and hypomagnesemia (1.5 mg/dL), with irregular intake of 4–6 g/day of elemental calcium, calcitriol 0.25 mcg four times daily, and magnesium supplementation. Despite numerous intravenous calcium gluconate infusions, supervision, and optimization of oral therapies, she remained with mild hypocalcemia (corrected calcium 7.8 mg/dL), persistent hyperphosphatemia, and considerable hypercalciuria (920 mg/24 h). Additional studies revealed diffuse calcifications, predominantly in the basal ganglia, and bilateral medullary nephrocalcinosis. Genetic testing identified a heterozygous variant in the calcium-sensing receptor (CaSR) gene (p.Glu228Lys, G>A). After 32 days, she was discharged due to improvement of neuromuscular symptoms, with optimization of therapy using hydrochlorothiazide 25 mg/day and sevelamer 800 mg five times daily. **Discussion:** The activating pathogenic CaSR variant described in this patient represents autosomal-dominant hypocalcemia type 1, resulting from a missense substitution within the receptor's extracellular domain. It leads to inhibition of parathyroid hormone (PTH) synthesis and secretion and decreases renal calcium reabsorption, accompanied by inappropriately low PTH levels, hypomagnesemia, hypocalcemia, and hyperphosphatemia. About half of cases are asymptomatic, with treatment using vitamin D analogs and calcium reserved for symptomatic patients. Diffuse brain calcifications account for the observed learning difficulties and may predispose to seizures. Periarticular abnormal calcifications limited the range of extension of the left upper extremity. The use of hydrochlorothiazide helps counteract hypercalciuria and mitigate the risk of renal impairment. **Final comments:** Treatment adherence to complex regimens (multiple daily dosing), together with a lack of social support and vulnerability in an adolescent with a severe symptomatic genetic condition, necessitates a multidisciplinary management approach and close follow-up to ensure understanding of this pathology and future quality of life.

Keywords: hypoparathyroidism; calcium-sensing receptor; complications.

BONE AND MINERAL METABOLISM

214 (3171) CRANIOFACIAL PAGET'S DISEASE MASQUERADING AS GRANULOMATOSIS WITH POLYANGIITIS: THE ROLE OF CLINICAL EVOLUTION IN REVISITING A DISCORDANT HISTOPATHOLOGICAL DIAGNOSIS

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Case presentation: A 48-year-old female with hypothyroidism (self-discontinued levothyroxine) presented with a four-year history of polyarthralgia with morning stiffness, recurrent bloody nasal discharge, and a painful hard mass in the right malar region. She had been investigated for granulomatosis with polyangiitis (GPA), ruled out after negative ANCA and inconclusive lung nodule biopsy. Between 2012 and 2014, three bone biopsies were performed: only the 2013 nasal specimen suggested Paget's disease showing trabecular superimposition, while the others showed nonspecific chronic inflammatory changes. The patient was subsequently lost to follow-up. Upon readmission years later due to worsening craniofacial disease, imaging revealed progressive erosion of the right maxillary sinus, stable pulmonary nodules, and otomastoid chronic disease with erosive changes. Laboratory workup showed mildly elevated alkaline phosphatase (312 U/L), normal calcium (9.5 mg/dL), phosphorus (4.5 mg/dL), PTH (41.3 pg/mL), and vitamin D (41.8 ng/mL), alongside severely elevated TSH (32.21 µUI/mL) due to treatment abandonment. The reassessment of the patient's clinical history (craniofacial bone destruction, epistaxis and years of refractoriness to GPA-directed treatment) together with the laboratory and imaging findings, led to questioning the initial presumptive diagnosis and establishing Paget's disease of bone. **Discussion:** Paget's disease of bone (PDB) is a chronic metabolic disorder of focal, disorganized bone remodeling. Craniofacial involvement may mimic GPA through bone expansion, epistaxis, and sinus erosion. Alkaline phosphatase may be only modestly elevated in monostotic disease, further complicating diagnosis. Here, discordant biopsies sustained diagnostic uncertainty for over a decade. The final diagnosis was not driven by new biopsy, but by reassessment of the patient's clinical and exams in a patient with a progressive craniofacial erosion refractory to GPA-directed treatment. **Final comments:** This case highlights that histopathological discordance should not permanently close a diagnostic hypothesis when clinical evolution points otherwise. In metabolic bone disease, focal sampling may yield heterogeneous results, and the longitudinal clinical picture must guide reassessment of prior findings. The patient's clinical history together with consistent laboratory and imaging findings was essential to reach the final diagnosis.

Keywords: Paget's disease of bone; craniofacial erosion; alkaline phosphatase.

BONE AND MINERAL METABOLISM

215 (3103) DENSITOMETRIC AND LABORATORY BONE RESPONSE TO ROMOSOZUMAB IN WOMEN WITH OSTEOPOROSIS: A RETROSPECTIVE OBSERVATIONAL STUDY

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Introduction: Osteoporosis is a disease characterized by reduced bone mass, resulting in an increased risk of fractures. Romosozumab, a monoclonal antibody with osteoanabolic activity associated with reduced bone resorption, promotes improvement in bone metabolism and increases bone mineral density (BMD). However, despite promising results observed in clinical trials, real-world data in Brazilian populations remain limited. **Objective:** To evaluate the impact of romosozumab treatment on BMD and bone remodeling markers in patients with osteoporosis followed at an endocrinology outpatient clinic. **Methods:** This longitudinal retrospective observational study included 44 women aged 73.5 ± 9.3 years who received romosozumab for the treatment of osteoporosis. BMD of the lumbar spine, femoral neck, and total femur was assessed by dual-energy X-ray absorptiometry before and after 12 months of treatment. Serum levels of C-terminal telopeptide of type I collagen (CTX), N-terminal propeptide of type I procollagen (P1NP), calcium, phosphorus, albumin, PTH, and 25(OH)D were evaluated at baseline and after 6 and 12 months of treatment. **Results:** A significant increase in BMD was observed at the lumbar spine (0.794 ± 0.135 vs. 0.861 ± 0.151 g/cm²; D% = +8.5; p < 0.001), femoral neck (0.705 ± 0.092 vs. 0.759 ± 0.106 g/cm²; D% = +7.7; p = 0.04), and total femur (0.700 ± 0.099 vs. 0.750 ± 0.096 g/cm²; D% = +7.2; p = 0.03). P1NP increased after 6 months (p = 0.02), without significant changes in CTX, calcium, phosphorus, albumin, PTH, or 25(OH)D levels (p ³ 0.07). Patients without prior treatment (n = 14) showed greater gains at the BMD lumbar spine compared with previously treated patients (n = 30) (0.812 ± 0.132 to 0.996 ± 0.136 g/cm² vs. 0.785 ± 0.137 to 0.801 ± 0.116 g/cm², respectively; p = 0.005). **Conclusion:** Romosozumab promoted increased BMD at all skeletal sites evaluated, along with a transient increase in the bone formation marker P1NP. Patients without prior treatment showed greater gains in BMD of the lumbar spine.
Keywords: osteoporosis; romosozumab; bone mineral density.

BONE AND MINERAL METABOLISM

216 (3360) DESCRIPTIVE ANALYSIS OF BONE MINERAL DENSITY IN PATIENTS WITH PANHYPOPITUITARISM FOLLOWED AT A TERTIARY PUBLIC HOSPITAL

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Patients with panhypopituitarism are at an increased risk for osteometabolic alterations due to multiple hormonal deficiencies and chronic glucocorticoid replacement. However, national data evaluating longitudinal bone mineral density (BMD) in this population remain limited. This study aimed to evaluate the longitudinal progression of BMD in patients with panhypopituitarism followed at a tertiary public hospital and to investigate clinical factors associated with adverse bone outcomes. A retrospective observational study was conducted via chart review of patients at a specialized endocrinology clinic. Demographic, clinical, and laboratory data were collected, including age, sex, etiology, diabetes mellitus, hypogonadism, statin use, and glucocorticoid exposure. Osteometabolic assessment utilized dual-energy X-ray absorptiometry (DXA), comparing the first and last scans within a 10-year period. BMD was analyzed at the lumbar spine (primary site), femoral neck, and total femur. Longitudinal variation was assessed using the delta_BMD variable. Statistical analyses included Pearson correlation and group comparisons. Twenty-six patients were evaluated (mean age 56 years; 13 men, 13 women) with a mean follow-up exceeding 18 years. Primary etiologies included nonfunctioning pituitary adenoma (48%), Sheehan syndrome, craniopharyngioma, and macroprolactinoma (11% each). Approximately 55% of patients exhibited osteopenia or osteoporosis. Hypogonadism affected > 70% of the sample, and diabetes mellitus ~30%. Delta_BMD followed a normal distribution (Shapiro-Wilk, p = 0.115). An overall mean delta of +0.036 was observed, showing marked heterogeneity. Females demonstrated significantly poorer longitudinal BMD progression compared to males (p = 0.003). No significant associations were found between BMD variation and age, disease duration, or glucocorticoid exposure. A moderate negative correlation existed between statin use and lumbar spine BMD variation (r = –0.418; p = 0.034). In conclusion, patients with panhypopituitarism showed high rates of osteometabolic alterations and heterogeneous BMD progression. Female sex and statin use were associated with worse densitometric outcomes.
Keywords: panhypopituarism; osteometabolic; glucocorticoid.

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217 (3569) DIAGNOSTIC INVESTIGATION OF MONOCLONAL GAMMOPATHY IN A PATIENT WITH ASYMPTOMATIC PAGET'S DISEASE OF BONE

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Case presentation: A 77-year-old man from Salvador, Bahia, presented with polyostotic Paget's disease of bone (scapula, lumbosacral spine, and left ilium). He had a medical history of diabetes, hypertension, heart failure, and chronic kidney disease (glomerular filtration rate of 36 mL/min/1.73 m²). In August 2023, his alkaline phosphatase was 307 U/L. He received zoledronic acid in September, showing an excellent initial clinical and biochemical response (alkaline phosphatase 57 U/L in February 2024). In 2025, his alkaline phosphatase increased to 160 U/L, and he sustained fractures in the second and third lumbar vertebrae. Laboratory tests revealed an inverted kappa/lambda ratio (0.11), with elevated lambda light chains (1,490 mg/L) and Immunoglobulin G (2,897 mg/dL), suggesting lambda light chain monoclonal gammopathy. Lumbar bone densitometry showed a T-score of +7.4, an artifact caused by pagetic sclerosis that masked his fracture risk. **Discussion:** This case illustrates a patient initially diagnosed with Paget's disease of bone who responded well to zoledronic acid but later developed an asymptomatic rise in alkaline phosphatase, prompting further investigation. While elevated alkaline phosphatase and new fractures typically suggest disease reactivation, a key clinical detail stood out: the patient remained pain-free. In the setting of a purely biochemical relapse without pagetic symptoms, investigating alternative causes is essential. It is incorrect to assume therapeutic failure without ruling out overlapping conditions that can elevate alkaline phosphatase. The overlap with a hematological malignancy explains the clinical presentation. The fractures are likely lytic lesions, and the rising alkaline phosphatase may reflect reactive bone callus. Since bone scintigraphy often fails to detect myeloma lesions, tomography or positron emission tomography is required to differentiate these pathologies. **Final comments:** The risk of bisphosphonate-induced nephrotoxicity is worsened by cast nephropathy secondary to the gammopathy. This may contraindicate further use of zoledronic acid, making alternative treatments like denosumab necessary. The suspicion of a monoclonal gammopathy, reinforced by the lack of pagetic pain, is the most urgent clinical priority. This condition redefines the patient's prognosis, making a complete hematological investigation with bone marrow aspiration and biopsy mandatory prior to any further bone interventions.
Keywords: Paget's disease of bone; monoclonal gammopathy; alkaline phosphatase.

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218 (2924) ECTOPIC MEDIASTINAL PARATHYROID GLAND AS A CAUSE OF SEVERE PRIMARY HYPERPARATHYROIDISM: CASE REPORT

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Case presentation: A 67-year-old woman presented with a 6-month history of asthenia, 6 kg weight loss, polyuria, hoarseness, and dysphagia. Laboratory workup revealed hypercalcemia (serum calcium 13.8 mg/dL; reference: 8.4–10.2), hypophosphatemia (2.0 mg/dL; reference: 2.3–4.7), and markedly elevated PTH (758 pg/mL; reference: 15–68.3), with unremarkable 24-hour urinary calcium, vitamin D levels, and renal function. Cervical ultrasound was negative for lesions; subsequent CT of the neck and chest identified a solid formation measuring 4.8 × 3.9 × 1.8 cm in the right middle-posterior mediastinum, suggestive of an ectopic parathyroid gland, confirmed by sestamibi scintigraphy. Renal ultrasound demonstrated nephrolithiasis, and densitometry revealed osteoporosis. During the preoperative period, serum calcium rose to 15.3 mg/dL, prompting hospital admission for intravenous hydration, cinacalcet, and pamidronate. Surgical excision was performed without complications, and pathology confirmed a parathyroid adenoma measuring 4.7 × 3.2 × 1.4 cm (12 g) with complete resection. The patient was discharged on calcium and calcitriol supplementation with scheduled outpatient follow-up. **Discussion:** Primary hyperparathyroidism is the leading cause of outpatient hypercalcemia, resulting from a solitary adenoma in approximately 85% of cases. Calcium levels above 14 mg/dL characterize severe hypercalcemia, with increased risk of systemic complications. Ectopic location of the parathyroid glands occurs in approximately 15% of patients, with the mediastinum being a frequent site due to embryologic migration of the inferior glands with the thymus. Deep mediastinal locations are less common and associated with greater diagnostic difficulty and the need for complex surgical approaches. Sestamibi scintigraphy has a sensitivity of 70%–90%, and may show discordant results in cases of ectopia or deep lesions, situations in which computed tomography and magnetic resonance imaging play a fundamental complementary role. Definitive treatment is surgical, with cure rates exceeding 95%, and may require a combined cervicothoracic approach in mediastinal ectopic cases. **Final comments:** This case illustrates an uncommon presentation of primary hyperparathyroidism, with severe hypercalcemia, markedly elevated PTH, and deep mediastinal ectopic location, reinforcing the importance of multimodal investigation and multidisciplinary management in these scenarios.
Keywords: primary hyperparathyroidism; ectopic parathyroid; hypercalcemia.

BONE AND MINERAL METABOLISM

219 (3094) EFFICACY OF 4 MG ZOLEDRONIC ACID FOR THE TREATMENT OF OSTEOPOROSIS IN POSTMENOPAUSAL WOMEN

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Osteoporosis is a highly prevalent osteometabolic disease whose impact continues to increase with population aging. Bisphosphonates are considered the first-line therapeutic option; however, limitations such as poor adherence and intolerance to oral therapy justify the use of parenteral bisphosphonate, zoledronic acid (ZA), in selected cases. ZA at a dose of 5 mg has demonstrated anti-fracture efficacy and is approved for the treatment of osteoporosis. The 4 mg formulation was initially studied in oncology patients, and although it is available in public hospitals at a lower cost, evidence regarding the effectiveness of this dose in osteoporosis management remains limited. This study aimed to evaluate the efficacy and safety of annual intravenous zoledronic acid at a dose of 4 mg in postmenopausal women with osteoporosis followed at a tertiary outpatient referral center. A retrospective observational study was conducted through medical record review of patients treated at the Endocrinology — Osteometabolic Diseases outpatient clinic of a referral center from January 2024 to the present. The study was approved by the Institutional Research Ethics Committee (approval number 098969/2025, dated November 5, 2025). A total of 51 women were included, with a median age of 72 years. Bone densitometry outcomes, fracture incidence, and adverse events were evaluated. Among participants, 69% had previously received oral bisphosphonate therapy, while the remaining patients were treatment-naive. The main indication for zoledronic acid use was very high-risk osteoporosis, followed by gastrointestinal intolerance/poor adherence and therapeutic failure. A significant increase in lumbar spine bone mineral density was observed after treatment, with a median of 0.832 g/cm² at baseline and 0.873 g/cm² after follow-up ($p = 0.0208$), accompanied by improvement in T-score from -2.90 to -2.50 ($p = 0.0456$). There was also a significant reduction in the occurrence of vertebral fractures (McNemar test, $p = 0.043$) and non-vertebral fractures ($p < 0.001$) during follow-up. Serious adverse events were rare, with a low frequency of drug-related complications. In conclusion, annual 4 mg zoledronic acid demonstrated effectiveness in improving lumbar spine bone mineral density and reducing fractures, with a favorable safety profile, representing a potentially viable therapeutic alternative for postmenopausal women with osteoporosis, especially in settings where oral treatment is limited.
Keywords: osteoporosis; zoledronic acid; bisphosphonate.

BONE AND MINERAL METABOLISM

220 (3577) FANCONI ANEMIA PRESENTING WITH SEVERE BONE FRAGILITY: CHALLENGES IN THE DIAGNOSIS AND MANAGEMENT OF RARE SECONDARY OSTEOPOROSIS

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Case presentation: A 28-year-old woman with genetically confirmed Fanconi anemia associated with FANCL variants, was referred to Endocrinology for evaluation of low bone mineral density (BMD). Clinical manifestations began in 2023 during a 21-week pregnancy complicated by severe preeclampsia and incomplete HELLP syndrome, requiring therapeutic pregnancy termination. Persistent anemia and thrombocytopenia prompted hematologic investigation, confirming Fanconi anemia. She subsequently developed compensated pancytopenia, recurrent infections, and intermittent transfusion requirements. In 2024, she developed spastic paraparesis and lower-limb paresthesia, undergoing resection of a granulomatous spinal cord lesion. Histopathology demonstrated suppurative granulomatous lymphadenitis without definitive confirmation of tuberculosis. Nevertheless, she completed treatment for neurotuberculosis followed by prolonged corticosteroid therapy due to MRI progression. During endocrine follow-up, she denied fragility fractures but reported secondary amenorrhea for approximately 2.5 years associated with hot flashes. She also reported low calcium intake, marked functional limitation due to paraparesis, and prolonged glucocorticoid exposure. DXA demonstrated Z-scores of –2.5 at the lumbar spine, –5.6 at the femoral neck, and -4.0 at the total hip. Laboratory evaluation revealed previous vitamin D insufficiency, with preserved renal function and normal calcium and phosphorus levels. Initial management included calcium/vitamin D supplementation and motor rehabilitation. **Discussion:** Fanconi anemia is a very rare inherited bone marrow failure syndrome frequently associated with early reduction in BMD, particularly in young adults. Bone loss is multifactorial, involving endocrinopathies, chronic inflammation, and possible intrinsic impairment of bone formation. In this case, additional aggravating factors included prolonged corticosteroid exposure and severe functional limitation secondary to granulomatous neurologic involvement. The disproportionately severe femoral bone loss compared with vertebral involvement suggests an important contribution from immobilization. **Final considerations: this** case highlights the importance of early bone health screening in patients with Fanconi anemia, especially in the presence of additional risk factors, reinforcing the need for individualized and multidisciplinary care.
Keywords: fanconi anemia; osteoporosis; bone density.

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221 (3062) FRAGILITY FRACTURES IN HEYN-SPROUL-JACKSON SYNDROME: AN UNDERRECOGNIZED SKELETAL PHENOTYPE

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Case presentation: 26-year-old man with multiple fragility fractures since childhood, including femoral, humeral, and stress fractures. Physical examination revealed short stature, microcephaly, and skeletal abnormalities. Laboratory evaluation showed normal calcium-phosphorus metabolism and bone turnover markers within expected ranges. Bone mineral density was only mildly reduced (lumbar spine Z-score -1.5). Genetic testing identified a heterozygous pathogenic variant in DNMT3A (c.988T>G; p.Trp330Gly). Despite the absence of significant densitometric impairment, the patient exhibited a disproportionately high fracture burden. **Discussion:** Heyn-Sproul-Jackson syndrome (HESJAS) is a rare genetic disorder caused by pathogenic variants in the DNMT3A gene, which encodes a DNA methyltransferase essential for epigenetic regulation. Although the syndrome is classically associated with microcephaly, short stature, and neurodevelopmental delay, its impact on bone metabolism remains poorly characterized. Emerging evidence suggests that DNMT3A plays a role in osteoclast differentiation through epigenetic modulation of key regulatory genes, including IRF8 and FOXO3. Altered methylation patterns may enhance osteoclastogenesis, disrupt redox balance, and increase bone resorption. Epigenetic dysregulation may also disrupt redox balance, further amplifying osteoclast activity. These mechanisms provide biological plausibility for skeletal fragility in HESJAS. **Final comments:** This case highlights a possible association between DNMT3A pathogenic variants and increased fracture risk independent of significant bone mineral density reduction, suggesting that bone quality impairment rather than bone mass alone may underlie skeletal fragility in this condition.
Keywords: DNMT3A gene; Heyn-Sproul-Jackson syndrome; fragility fracture.

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222 (3363) GORHAM-STOUT DISEASE WITH MULTIFOCAL OSTEOLYSIS, RECURRENT FRACTURES AND CLINICAL RESPONSE TO SIROLIMUS: REPORT OF TWO PEDIATRIC CASES

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Case presentation: we report two pediatric cases of Gorham-Stout disease with extensive osteolytic involvement. Case 1 was a male child with swelling and overlying hyperpigmentation of the left thigh since birth. He developed intra- and extraosseous lymphatic-vascular lesions, progressive osteolysis of the left femur, sacrum, pelvis and lumbar vertebrae, a cutaneous fistula with intermittent serous drainage, and multiple fragility fractures of the left femur. Bone biopsy showed a predominantly lymphatic malformative vascular proliferation. Sirolimus was started, leading to pain improvement and reduced opioid requirement, although radiological progression of bone lesions and new fractures occurred. Case 2 was a female child with thoracic and left supraclavicular lymphatic malformation, chylothorax in early infancy, multifocal osteolytic lesions and bilateral femoral fractures within the first year of life. Skin biopsy showed ectatic capillary vessels positive for CD31, CD34 and D2-40. She developed progressive spinal deformity, vertebral collapse, spinal cord compression at T10, chronic pain and inability to walk. After sirolimus initiation, there was marked pain improvement, clinical stabilization of spinal deformities and scintigraphic improvement. **Discussion:** Gorham-Stout disease is a rare complex lymphatic anomaly characterized by vascular/lymphatic proliferation and progressive osteolysis. These cases illustrate severe, very early-onset presentations with polyostotic involvement, recurrent fractures, chronic pain and soft-tissue disease. The second case also highlights the relevance of thoracic involvement and chylothorax, which are associated with greater disease severity. In both patients, sirolimus was associated with clinical benefit, particularly analgesia and partial stabilization, but without complete reversal of bone lesions, reinforcing the need for multidisciplinary care and long-term follow-up. **Final comments:** These cases reinforce the importance of considering Gorham-Stout disease in children with progressive osteolysis, fragility fractures and lymphatic malformations. Diagnosis depends on the integration of clinical, radiological and histopathological findings, after exclusion of infectious, neoplastic, metabolic and genetic etiologies. Sirolimus may contribute to symptomatic control and disease stabilization, but skeletal progression remains unpredictable.
Keywords: Gorham-Stout disease; osteolysis; sirolimus.

BONE AND MINERAL METABOLISM

223 (3521) HOSPITALIZATIONS FOR BONE DENSITY AND STRUCTURAL DISORDERS IN BRAZIL IN 2025: AN EPIDEMIOLOGICAL AND ECONOMIC ANALYSIS

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Introduction: Bone density and structural disorders are important public health conditions due to their associated morbidity and healthcare costs. Secondary osteometabolic disorders related to endocrinopathies, nutritional deficiencies, inflammatory diseases, and chronic corticosteroid use also contribute to hospital admissions in Brazil. **Objective:** To evaluate hospitalizations for bone density and structural disorders in Brazil in 2025 according to age group and geographic region, including hospitalization costs and length of stay. **Methods:** This ecological study used Datasus data obtained through the Hospital Information System (SIH/SUS). Hospitalizations classified under ICD-10 codes M80-M85 were included. Cases were stratified according to age group and Brazilian geographic region. Proportional frequency, mean hospitalization cost, and average hospital stay were analyzed. Ethics committee approval was waived due to the use of publicly available secondary data. **Results:** In 2025, 18,442 hospitalizations related to bone density and structural disorders were recorded in Brazil, accounting for 0.12% of total admissions. An unexpected predominance of hospitalizations was observed among young and middle-aged adults, representing more than 60% of cases, with a peak among individuals aged 40–49 years (16.6%), differing from the classical pattern associated with senile osteoporosis. Individuals aged ³60 years accounted for 22.5% of hospitalizations. The Southeast region concentrated the highest proportion of cases (40.4%) and showed an average length of stay below the national mean. The North region presented the longest hospitalization duration (7.8 days) and the lowest mean cost per admission (R$ 1,472.84), whereas the South region showed the shortest hospital stay (3.5 days) and the highest mean hospitalization cost (R$ 1,898.74). **Conclusion:** Hospitalizations predominated among young and middle-aged adults, suggesting a relevant contribution of secondary osteometabolic disorders, endocrinopathies, chronic corticosteroid use, and nutritional deficiencies. Regional disparities were identified regarding hospitalization duration and costs, possibly associated with inequalities in specialized healthcare access. These findings reinforce the need for prevention, early diagnosis, and appropriate management of osteometabolic diseases in Brazil.

Keywords: osteoporosis; hospitalization; osteometabolic diseases.

BONE AND MINERAL METABOLISM

224 (3154) HYPOCALCEMIA, EPILEPSY, AND FAHR DISEASE: A CASE SERIES OF THREE PATIENTS WITH OVERLAPPING NEUROLOGICAL AND METABOLIC MANIFESTATIONS

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Case presentation: Case 1: A 70-year-old woman referred for hypocalcemia and hyperphosphatemia in newly onset symptomatic epilepsy. **Background:** arterial hypertension and primary hypothyroidism (captopril, levothyroxine, phenobarbital). Right wrist fracture in 2013. Symptoms: paresthesias and cramps. Laboratory: total calcium (Ca) 5.3 mg/dL, phosphorus (P) 7.4 mg/dL, parathyroid hormone (PTH) < 3.0 pg/mL, 25-OH Vitamin D (VD) 20.7 ng/mL, creatinine 1.0 mg/dL. Head computed tomography (CT): periventricular white matter densification and bilateral dentate nucleus calcifications. Case 2: A 26-year-old woman referred for hypocalcemia with refractory seizures. Epilepsy since age 22; primary hypothyroidism (carbamazepine, levothyroxine). Laboratory: Ca 4.8 mg/dL, P 7.7 mg/dL, PTH 232 pg/mL, VD 37 ng/mL. Head CT: basal ganglia and bilateral cerebellar calcifications. Case 3: A 38-year-old man with hypocalcemia, hyperphosphatemia, and refractory seizures since childhood. Symptoms: paresthesias, cramps, vertigo, and wide-based gait. PTH below the lower limit of normal. Head CT: periventricular and basal ganglia calcifications. **Discussion:** These cases illustrate the association between hypocalcemia, epilepsy, and Fahr disease. Hypoparathyroidism is characterized by insufficient PTH secretion leading to hypocalcemia and hyperphosphatemia (cases 1 and 3). Pseudo-hypoparathyroidism (case 2) is a rare autosomal dominant genetic condition associated primarily with resistance to the PTH and lead to low calcium and high phosphorus levels. These conditions represent the leading metabolic cause of intracranial calcifications. Fahr disease refers to bilateral symmetrical calcifications of the basal ganglia, periventricular white matter and cerebellum. Symptoms typically involve epilepsy, movement disorders and neuropsychiatric issues. Hypocalcemia reduces neuronal membrane stability, promoting hyperexcitability and seizure activity. Calcifications themselves may also contribute to epileptogenesis. This series underscores that epilepsy of unclear etiology warrants metabolic investigation. **Final comments:** Hypoparathyroidism is a treatable and often underdiagnosed cause of seizures and intracranial calcifications. Early recognition and correction with calcium and active vitamin D analogs may improve seizure control and prevent neurological progression. These cases reinforce the relevance of endocrinological evaluation in patients with epilepsy and basal ganglia calcifications.

Keywords: hypoparathyroidism; Fahr disease; hypocalcemia.

BONE AND MINERAL METABOLISM

225 (3352) IATROGENIC NEAR-TOTAL PARATHYROIDECTOMY IN X-LINKED HYPOPHOSPHATEMIC RICKETS: A CASE REPORT

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Case presentation: A 32-year-old male patient with a previous diagnosis of congenital primary hyperparathyroidism since childhood underwent near-total parathyroidectomy. He evolved with osteoarticular pain, functional limitation, progressive skeletal deformities, and spontaneous left femoral fracture. He presented short stature, bowing of the lower limbs, and required crutches for ambulation. Laboratory evaluation demonstrated persistent hypophosphatemia, elevated alkaline phosphatase levels, secondary elevation of parathyroid hormone, normal serum calcium, and increased fractional excretion of phosphorus, suggesting renal phosphate wasting. Imaging studies demonstrated diffuse osteopenia, skeletal deformities, and incomplete fracture of the left femoral shaft. A genetic panel for hypophosphatemic rickets was performed, identifying a likely pathogenic hemizygous variant in the PHEX gene, confirming the diagnosis of X-linked hypophosphatemic rickets. **Discussion:** X-linked hypophosphatemic rickets is the most common hereditary form of renal hypophosphatemia, caused by variants in the PHEX gene, leading to increased FGF-23 activity, renal phosphate wasting, and impaired bone mineralization. The present case highlights significant diagnostic confusion with congenital primary hyperparathyroidism, culminating in previous parathyroid surgical intervention. The presence of persistent hypophosphatemia, progressive skeletal deformities, lower-limb bowing, fragility fractures, and secondary hyperparathyroidism should raise suspicion for hereditary phosphate-wasting disorders. Delayed diagnosis contributed to osteoarticular sequelae and functional limitation. Molecular confirmation enabled diagnostic reclassification, genetic counseling, and appropriate therapeutic management, including phosphorus and calcitriol replacement. Final considerations: XLH should be considered in the differential diagnosis of patients with skeletal deformities, recurrent fractures, persistent hypophosphatemia, and secondary hyperparathyroidism, especially when symptoms begin during childhood. **Final comments:** Early recognition of the disease and systematic investigation of phosphorus-calcium metabolism are essential to avoid inappropriate interventions and reduce irreversible complications. Genetic testing played a decisive role in diagnostic clarification, allowing etiological confirmation and improved therapeutic planning.

Keywords: X-linked hypophosphatemic rickets; congenital primary hyperparathyroidism; PHEX.

BONE AND MINERAL METABOLISM

226 (3132) IDIOPATHIC HYPOPARATHYROIDISM AS A REVERSIBLE CAUSE OF HEART FAILURE AND SYNCOPE: A CASE REPORT

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Case presentation: A 47-year-old woman with a history of fibromyalgia presented with an episode of syncope without prodromal symptoms, associated with paresthesias. During cardiologic investigation, she was hospitalized for decompensated heart failure, profile B. Transthoracic echocardiography revealed an ejection fraction of 23%. During ICU admission, she developed QT interval prolongation on electrocardiogram. Laboratory tests showed: CaT 5.3 mg/dL, CaI 0.54 mmol/L, P 5.0 mg/dL, PTH 2.0 pg/mL, 24-hour urinary calcium 171 mg/dL, albumin 4.0 g/dL, 25-OH vitamin D 24.9 ng/mL, Cr 1.2 mg/dL, Mg 2.5 mg/dL. Cervical and urinary tract ultrasonography showed no abnormalities. Cranial CT demonstrated supra- and infratentorial calcifications. After the diagnosis of idiopathic hypoparathyroidism, intravenous calcium replacement was initiated, later replaced by oral calcium carbonate and calcitriol, in addition to treatment for heart failure with reduced ejection fraction. After three months, the patient showed clinical improvement and recovery of the ejection fraction to 58%. She remains under outpatient follow-up, with CaT 9.1 mg/dL, CaI 1.2 mmol/L, and P 3.6 mg/dL, without complications to date. **Discussion:** Hypoparathyroidism is a rare condition characterized by low PTH production, leading to hypocalcemia and hyperphosphatemia. The main etiology is postsurgical, especially after thyroid procedures, followed by autoimmune and hereditary causes. Isolated idiopathic hypoparathyroidism is rare in adults, with an estimated incidence of 1 per 100,000 individuals. Hypocalcemia may cause manifestations ranging from paresthesias and muscle cramps to severe complications such as syncope, arrhythmias, and heart failure. Altered calcium homeostasis and the absence of the direct effects of PTH on the myocardium are the main mechanisms involved. Reduced cardiac contractility may result in reversible heart failure after metabolic correction. Treatment consists of calcium and calcitriol replacement, aiming at symptomatic control and maintenance of serum calcium levels in the low-normal range. **Final comments:** This case highlights the importance of investigating the etiology of hypocalcemia in patients presenting with severe cardiovascular manifestations. Early diagnosis and appropriate treatment of idiopathic hypoparathyroidism were essential for reversal of cardiac dysfunction and clinical improvement, demonstrating that it is a potentially reversible cause of heart failure.

Keywords: idiopathic hypoparathyroidism; heart failure; syncope.

BONE AND MINERAL METABOLISM

227 (3547) OSTEOGENESIS IMPERFECTA ASSOCIATED WITH CONGENITAL HYPOPITUITARISM: THE NINTH REPORTED CASE IN WORLD LITERATURE

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Osteogenesis imperfecta (OI) is a rare bone fragility disorder characterized by pathological fractures and skeletal deformities, most commonly associated with variants in COL1A1 and COL1A2, among other less frequently identified genes. Some patients with OI may present with short stature despite maintaining a normal growth velocity. To date, eight male patients with OI associated with congenital hypopituitarism have been reported in the literature. Congenital hypopituitarism is another rare and genetically heterogeneous disorder, with dozens of genes implicated in its etiology. Hormonal deficiencies may further compromise bone health if not adequately replaced. Here, we describe a 26-year-old male patient who was diagnosed with OI in utero following the detection of a femoral fracture. Since birth, he has sustained more than 20 fractures and received pamidronate infusions from 3 to 17 years of age. He exhibited severe short stature during childhood, which was initially attributed to long-bone deformities secondary to multiple fractures. At 12 years of age, pituitary function was evaluated, leading to the diagnosis of panhypopituitarism [IGF-1: 38.6 ng/mL (143–693); TSH: 6.3 mU/L (0.4–4.5) and free T4: 0.4 ng/dL (0.7–1.8); cortisol < 0.5 µg/dL; FSH: 1 U/L; LH < 0.1 U/L; testosterone < 2.5 ng/dL]. Pituitary MRI demonstrated an ectopic posterior pituitary located in the infundibulum. Replacement therapy with growth hormone, levothyroxine, and glucocorticoids was initiated, resulting in weight recovery. Testosterone replacement therapy was started at 15 years of age. During the first five years of hormonal replacement, the patient experienced no fractures. At 17 years of age, he sustained a humeral fracture, followed by a new femoral fracture at 22 years of age. He remains on full pituitary hormone replacement therapy, including growth hormone, to date. This case of OI coexisting with congenital hypopituitarism represents the ninth reported in the literature. To date, genes associated with OI have not been linked to congenital hypopituitarism. Potential shared genetic mechanisms underlying these two rare disorders, as well as the apparent male predominance, may be elucidated through comprehensive next-generation sequencing studies in affected patients.

Keywords: osteogenesis imperfecta; congenital hypopituitarism; bone fragility.

BONE AND MINERAL METABOLISM

228 (3216) OSTEOGENESIS IMPERFECTA ASSOCIATED WITH TYPE 1 DIABETES MELLITUS IN A YOUNG ADULT WITH OSTEOPOROSIS

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Case presentation: G.N.S., a 26-year-old male, diagnosed with type 1 diabetes mellitus (T1DM) in 2012, with irregular adherence to insulin therapy. He developed a pathological fracture of the left tibia and fibula in 2023 and had a previous left femur fracture in 2021 following a motor vehicle accident. Bone densitometry revealed marked reduction in bone mineral density, with a lumbar spine Z-score of –3.7 and femoral neck Z-score of –4.3 in 2023, when Denosumab therapy was initiated. Physical examination showed body weight of 53 kg, height of 1.70 m, and BMI of 18 kg/m², in addition to blue-gray sclerae and opalescent, fragile teeth, findings suggestive of osteogenesis imperfecta (OI). Laboratory evaluation demonstrated persistently elevated glycated hemoglobin levels, and frequent hypoglycemic episodes associated with irregular insulin use. Renal function was preserved, with serum creatinine of 0.91 mg/dL. Total serum calcium and parathyroid hormone levels were within the normal range (total calcium: 9.1 mg/dL; PTH: 20.1 pg/mL), as was thyroid function (TSH: 3.55 µIU/mL). During follow-up, vitamin D insufficiency was identified, with 25-hydroxyvitamin D levels of 18 ng/mL. **Discussion:** Osteogenesis imperfecta is a rare genetic disorder, most commonly associated with type I collagen abnormalities, characterized by bone fragility, recurrent fractures, and extra-skeletal manifestations. The presence of blue sclerae and dental abnormalities strengthened the diagnostic suspicion of type 1 or type 4 OI in this patient. The coexistence of T1DM represents a potentially aggravating condition for bone health. T1DM is associated with reduced bone formation, impaired bone microarchitecture, and increased fracture risk, particularly in patients with poor glycemic control. In this case, chronic metabolic dysregulation, combined with low body weight and sedentary lifestyle, likely contributed to worsening osteoporosis and early fracture occurrence. **Final comments:** This report highlights the importance of investigating genetic causes of bone fragility in young adults presenting with severe osteoporosis and fractures disproportionate to trauma. The association between probable OI and poorly controlled T1DM contributed to significant skeletal impairment at an early age. Early recognition of this coexistence allows a multidisciplinary approach, optimization of glycemic control, and implementation of therapies aimed at reducing morbidity and improving quality of life.

Keywords: osteogenesis imperfecta; type 1 diabetes mellitus; osteoporosis.

BONE AND MINERAL METABOLISM

229 (3124) OSTEOGENESIS IMPERFECTA TYPE XX ASSOCIATED WITH A HOMOZYGOUS MESD VARIANT: A CASE REPORT

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Case presentation: Osteogenesis imperfecta (OI) is a heterogeneous group of genetic disorders characterized by bone fragility and recurrent fractures, with an estimated incidence of 1:15,000 to 1:20,000 live births. Autosomal recessive forms are rare. OI type XX is associated with pathogenic variants in the MESD gene, related to WNT signaling and LRP5/LRP6 receptor processing. Few cases have been reported worldwide. A 3-year- and 6-month-old female patient was diagnosed at 6 months of age after investigation of progressive scoliosis. Genetic analysis identified a homozygous MESD variant compatible with OI type XX. At diagnosis, she presented multiple low-impact fractures, including clavicle fracture at birth, bilateral humerus fractures and right rib fracture, associated with progressive skeletal deformities and significant motor delay. Parents were consanguineous, with no family history of the disease. The patient developed recurrent pneumonia, respiratory insufficiency requiring nocturnal oxygen therapy and CPAP indication, in addition to severe nutritional impairment requiring enteral tube feeding. Physical examination revealed short stature, low weight, marked scoliosis and limb deformities. At 3 years and 6 months of age, weight was 8.7 kg and height 87 cm, both below -3 z-scores. Radiography demonstrated diffuse bone demineralization and severe thoracolumbar scoliosis. The first dose of zoledronic acid was administered at 3 years and 3 months of age. She currently uses vitamin D and calcium carbonate supplementation. **Discussion:** MESD variants impair WNT signaling and result in severe bone formation defects. Recessive forms of OI show marked clinical heterogeneity and may present progressive deformities, multiple fractures, respiratory insufficiency and severe functional impairment, as observed in this case. This report reinforces the importance of genetic investigation in severe early-onset bone fragility. **Final comments:** This case describes a rare and severe form of OI type XX associated with a homozygous MESD variant, highlighting the relevance of early molecular diagnosis in recessive forms of the disease.
Keywords: osteogenesis imperfecta; MESD gene; bone fragility.

BONE AND MINERAL METABOLISM

230 (3037) OSTEOLYSIS IN GORHAM-STOUT DISEASE: CASE REPORT OF THE CHALLENGES IN MANAGING A RARE DISEASE

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Case presentation: R.F., male, 45 years old, previously healthy, was admitted in 2021 with chin pain radiating to the left mandible after laser therapy in the beard area. Facial computed tomography revealed mandibular osteolysis. An incisional biopsy showed angiolymphatic proliferation and bone necrosis. In March 2022, under suspicion of squamous cell carcinoma, he underwent mandibulectomy with fibular flap reconstruction. During follow-up, osteolysis recurred, affecting the graft. After 1 year and 8 months, due to purulent discharge from the mandibular plate, reoperation was performed, resulting in a diagnosis of chronic osteomyelitis, without the possibility of further grafting and requiring permanent tracheostomy. Histopathological findings from both procedures showed osteonecrosis associated with medullary fibrosis, plasmacytic infiltrate, hemorrhage, and fibrosis, compatible with Gorham-Stout Disease (GSD). Disease progression to the maxilla and temporal bones was later observed, and the patient was referred to Endocrinology. In October 2025, treatment with zoledronic acid (5 mg/month) was initiated; however, progression of the temporal lesion persisted after the fourth dose. Due to persistent disease activity, radiotherapy associated with continuous bisphosphonate therapy was indicated. **Discussion:** GSD is a rare osteolytic disorder first described in 1955, with approximately 400 reported cases. It shows slight male predominance, and diagnosis is often delayed by about 3.5 years after symptom onset. Its pathophysiology remains uncertain, although mutations involving the RAS/PI3K/mTOR pathways associated with vascular and lymphatic proliferation have been described. Any bone may be affected, especially the axial skeleton. Pain and edema are the most common initial manifestations and may follow local trauma. Treatments include bisphosphonates, sirolimus, beta-blockers, and radiotherapy, alone or combined, with variable efficacy. In maxillomandibular involvement, antiresorptive therapy is commonly used, although better remission rates have been reported with radiotherapy. **Final comments:** This case highlights the diagnostic and therapeutic challenges of GSD and the impact of delayed diagnosis on disease progression and functional sequelae. It also suggests that antiresorptive therapy alone may be insufficient to control osteolysis, reinforcing the need for further studies and targeted therapies.
Keywords: osteolysis, essential; osteonecrosis; bone density conservation agents.

BONE AND MINERAL METABOLISM

231 (3256) OSTEOMALACIA INDUCED BY FANCONI SYNDROME SECONDARY TO CHRONIC TENOFOVIR USE: A CASE REPORT

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Case presentation: A 68-year-old female patient diagnosed with HIV infection since 1997 was receiving darunavir, maraviroc, ritonavir, lamivudine, dolutegravir, and tenofovir, the latter for more than 10 years. She presented with hyporexia, weight loss, nausea and vomiting, as well as proximal muscle weakness in the lower limbs and bilateral costal arch pain over the previous three months, leading to hospital admission for investigation. Laboratory tests revealed phosphorus 1.6 mg/dL (reference range [RR] 2.7–4.5 mg/dL), total calcium 7.9 mg/dL (RR 8.6–10.2 mg/dL), metabolic acidosis (pH 7.33 and bicarbonate 15.5 mmol/L), glycosuria in a non-diabetic patient, alkaline phosphatase 402 U/L (RR 35–104 U/L), and bone-specific alkaline phosphatase 91.1 µg/L (RR 5.2–24.4 µg/L). Bone scintigraphy showed probable healing fractures in bilateral costal arches, and bone densitometry demonstrated osteoporosis (lumbar spine T-score –2.8; femoral neck –3.4; total proximal femur –3.5; radius –3.0). Osteomalacia induced by Fanconi syndrome secondary to chronic Tenofovir use was suspected. Tenofovir was discontinued, electrolyte replacement was initiated, and calcitriol was prescribed. After three months, the patient returned for outpatient follow-up with clinical and laboratory improvement. **Discussion:** Osteomalacia is a disease characterized by decreased bone mineralization involving both cortical and trabecular bone. Its main etiologies include vitamin D deficiency, hypophosphatemic disorders, and primary mineralization defects. A rare cause is Fanconi syndrome secondary to chronic Tenofovir use. This drug is used in the treatment of HIV infection and may cause proximal renal tubulopathy, leading to solute loss and impaired bone mineralization. Renal toxicity increases with longer duration of therapy, becoming more evident after the first year of use. **Final comments:** This report describes a rare complication of chronic Tenofovir use: osteomalacia induced by Fanconi syndrome. Considering the frequent use of this medication in clinical practice, healthcare professionals should be aware of this condition to ensure appropriate surveillance and screening in at-risk situations, as well as adequate management with discontinuation of the antiretroviral drug and correction of electrolyte disturbances, aiming to promote reversibility of the clinical, laboratory, and densitometric abnormalities.

Keywords: osteomalacia; Fanconi syndrome; tenofovir.

BONE AND MINERAL METABOLISM

232 (3218) PAGET'S BONE DISEASE MIMICKING NEOPLASTIC INFILTRATION IN THE LUMBOSACRAL SPINE: A CASE REPORT IN AN ELDERLY PATIENT WITH CHRONIC PAIN

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Case presentation: E.F.L., a 73-year-old woman, presented with chronic lumbosacral pain with progressive worsening over several years. Her medical history included diabetes mellitus, systemic arterial hypertension, dyslipidemia, hypertrophic cardiomyopathy, atrial fibrillation, previous ischemic stroke and a solitary functioning kidney. During evaluation, imaging studies revealed bone abnormalities in the sacrum and lumbar spine, initially suggestive of infiltrative lesions. Laboratory tests showed persistently elevated alkaline phosphatase levels (391 U/L in 2022 and 524 U/L in 2025), while serum calcium, renal function, parathyroid hormone and phosphorus remained within normal ranges. Computed tomography of the sacroiliac joints demonstrated diffuse sacral heterogeneity, permeative osteolytic areas, cortical thickening, and trabecular sclerosis. Bone scintigraphy revealed increased osteogenic activity in L4/L5, sacrum, and sacroiliac joints. Bone densitometry showed a marked increase in lumbar spine bone mineral density. After comprehensive evaluation and exclusion of malignancy, the integration of clinical, biochemical, and radiological findings confirmed the diagnosis of Paget's disease of bone. Given persistent pain, intravenous zoledronic acid therapy was initiated. **Discussion:** Paget's disease of bone is a focal disorder of bone remodeling characterized by increased osteoclastic activity followed by disorganized bone formation. It predominantly affects older adults and may remain asymptomatic for long periods, often being identified by elevated alkaline phosphatase levels or incidental radiological findings. When symptomatic, it presents with bone pain, deformities, and orthopedic complications. This case highlights lumbosacral involvement with marked radiological abnormalities, initially suggestive of secondary bone disease. Bone scintigraphy was essential for assessing disease extent, while computed tomography contributed to lesion characterization. Bisphosphonates, particularly zoledronic acid, are considered first-line therapy due to sustained biochemical remission and effective symptom control. **Final comment:** Paget's disease of bone should be considered in the differential diagnosis of chronic bone pain associated with structural bone abnormalities in elderly patients. Integration of clinical, laboratory, and imaging findings is essential for early diagnosis and appropriate management, reducing morbidity and avoiding unnecessary interventions.

Keywords: Paget's disease of bone; alkaline phosphatase; low back pain.

BONE AND MINERAL METABOLISM

233 (3067) PARATHYROID CARCINOMA AND BONE MINERAL DENSITY: A SINGLE-CENTER EXPERIENCE

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Introduction: Parathyroid carcinoma (PC) is a rare, generally sporadic, condition that accounts for less than 1% of the cases of primary hyperparathyroidism (PHPT), mostly occurring in the fifth decade of life. The degree of hypercalcemia in PC is usually more severe, with calcium and PTH concentrations being substantially higher than in benign conditions. Some histopathological findings are suggestive of PC, however diagnosis depends on local invasion or metastasis. **Objective:** To analyze demographic, radiological and clinicopathological characteristics of PC in a single-center. **Methods:** This retrospective single-center study included patients with histopathological diagnosis of PC between 2000 and 2025. Clinical, laboratory, and Bone Mineral Density (BMD) (GE Lunar®) data were collected from medical records. **Results:** Seven patients were diagnosed with PC, five were women and the mean age was 48 years old (26–66). Five patients had nephrolithiasis at diagnosis, three had fractures and two had brown tumors. After first surgery, five patients achieved biochemical cure, while one patient required a subsequent surgery to achieve remission. One patient did not reach remission and died a few weeks later. Median PTH at diagnosis was 1,924 pg/mL (1,308–2,500) and, after surgery, 57 pg/mL (31-2,500); median corrected calcium for albumin at diagnosis was 12.9 mg/dL (11.1–15.5) and phosphorus 2.3 mg/dL (1.5–4.5). One patient had adrenal, temporal and cardiac metastasis. Hungry bone syndrome was detected in two patients after surgery, one patient had transitory hypoparathyroidism and other had definitive hypoparathyroidism. BMD was assessed at diagnosis and during a median of 6 years (2–20) of follow-up. We found that at diagnosis the median T-score of lumbar spine, total hip, femoral neck and distal radius were –1.3 (–6.2 to –0.3), –2.5 (–4.5 to –1.4), –2.1 (–4.4 to –0.6) and –3.9 (–7.7 to –2.7), respectively; and after surgery, for the same sites, the T-scores were 0.2 (–1.9 to 1.7), 0.6 (–1.5 to 3.2), 0.1 (–2.2 to 2.3) and –2.8 (–5.1 to –0.4). **Conclusion:** Patients with PC present clinical and bone severe manifestations. The bone disease brings to light rare manifestations such as brown tumors. BMD allows the quantification not only of the negative impact of PC in the skeleton but also the improvement after cure. Early diagnosis and appropriate surgical treatment enable cure and improvement of overall health, particularly bone health.
Keywords: parathyroid carcinoma; bone mineral density; hyperparathyroidism.

BONE AND MINERAL METABOLISM

234 (3323) PARICALCITOL VERSUS CALCITRIOL IN PATIENTS WITH SECONDARY HYPERPARATHYROIDISM DUE TO CHRONIC KIDNEY DISEASE: A SYSTEMATIC REVIEW

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Introduction: Secondary hyperparathyroidism (SHPT) is a complication that develops in chronic kidney disease (CKD) as a mechanism to regulate calcium, phosphorus, and vitamin D levels. The condition is characterized by hyperplasia of the parathyroid glands and elevated levels of parathyroid hormone (PTH). Its therapeutic management involves the use of calcitriol, the active form of vitamin D, or its analogs, such as paricalcitol. Given this, it is pertinent to compare them in terms of efficacy and safety. **Objective:** To compare the efficacy and safety of paricalcitol versus calcitriol in the treatment of SHPT in CKD. **Methods:** This is a systematic review based on the PRISMA protocol. The search was conducted in the PubMed, Cochrane, and Embase databases. Two independent reviewers conducted the article selection, and any disagreements were resolved by a third reviewer. Pediatric studies, studies on hyperparathyroidism related to other etiologies, preclinical trials, and reviews were excluded. The search yielded 64 articles, after excluding duplicates, reading the full texts, and applying the eligibility criteria, eight clinical studies were included. Methodological quality was assessed using the JBI Critical Appraisal Tool. **Results:** Studies have shown that calcitriol and paricalcitol were effective in reducing PTH levels in patients with SHPT undergoing hemodialysis, with no significant differences in most analyses. Some studies have demonstrated advantages of paricalcitol, including a faster therapeutic response and greater achievement of PTH suppression targets. Both treatments resulted in increases in serum calcium and phosphorus levels and a similar incidence of hypercalcemia; however, paricalcitol was associated with a lower frequency of hyperphosphatemia, a better mineral metabolism profile, and a lower incidence of sustained hypercalcemia. Adverse events were similar between the groups, although paricalcitol was associated with lower rates of hospitalization, and lower treatment discontinuation. **Conclusion:** Both calcitriol and paricalcitol have demonstrated efficacy in controlling SHPT associated with CKD. However, paricalcitol showed advantages in certain outcomes, such as a lower incidence of hyperphosphatemia and sustained hypercalcemia, as well as a better therapeutic response and potentially better cost-effectiveness, proving to be an effective and safe alternative in the management of SHPT associated with CKD.
Keywords: secondary hyperparathyroidism; chronic kidney disease; mineral and bone disorder.

BONE AND MINERAL METABOLISM

235 (3142) PATIENT PROFILE IN A FRACTURE LIAISON SERVICE: UNDERDIAGNOSIS AND UNDERTREATMENT OF OSTEOPOROSIS

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Introduction: Osteoporosis is a major public health burden, responsible for millions of fragility fractures annually and a leading cause of morbidity, functional decline, and premature mortality among older adults. individuals with a previous fracture are at substantially higher risk of subsequent fractures. Fracture Liaison Services (FLS) play a key role in secondary prevention, ensuring that patients who have already suffered a fragility fracture receive appropriate diagnosis, treatment, and follow-up to prevent new fractures. **Objective:** To describe the profile of patients seen at a Fracture Liaison Service. **Methods:** Prospective, longitudinal, observational study with descriptive analysis; data are reported as median (p25–p75) or frequencies (n, %). **Results:** Sixty-one participants were included, 56 (91%) females, with a median age of 70 (62–77) years and BMI of 25.9 (22.2–31.0) kg/m². Osteoporosis had been diagnosed before the low-impact fracture in only 27 (44.3%) cases; 24 (39.3%) received the diagnosis at the time of fracture, while 10 (16.4%) had not been diagnosed or treated even after the fracture, being identified only through active FLS screening. The three most frequent fracture sites were vertebrae 30 (49%), wrist 13 (21%), and femur 8 (13%). The most prevalent comorbidities were arterial hypertension 41(67.2%) and type 2 diabetes mellitus 22 (36%). Regarding pharmacological treatment at inclusion, the majority used oral bisphosphonates (43%; alendronate and risedronate), followed by zoledronic acid (32%), denosumab (15%), teriparatide (2%), and romosozumab (2%). Four patients were not using any osteoporosis-specific medication. Among the 54 patients with available bone densitometry, 18 (33.3%) had osteopenia. **Conclusion:** The findings reveal a failure in the early diagnosis of osteoporosis, with 55.7% of patients identified only after a fracture. Ten patients had not received an osteoporosis diagnosis even after the fracture, highlighting the extent of underdiagnosis. The high occurrence of fractures among osteopenic patients underscores the importance of global fracture risk assessment in this group. The use of bone-forming agents in only 4% of very high-risk patients demonstrates a significant gap between evidence and clinical practice, reinforcing the role of FLS as an essential tool in the secondary prevention of fragility fractures.
Keywords: fracture liaison service; osteoporosis; fractures.

BONE AND MINERAL METABOLISM

236 (3350) PREVALENCE OF MORPHOMETRIC VERTEBRAL FRACTURES AFTER BARIATRIC SURGERY IN POSTMENOPAUSAL WOMEN

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Introduction: Bariatric surgery is associated with changes in bone metabolism, including reduced bone mineral density (BMD) and increased bone remodeling, potentially increasing fracture risk in postmenopausal women. **Objective:** To evaluate the prevalence of morphometric vertebral fractures in postmenopausal women who underwent bariatric surgery and compare it with women with obesity without previous bariatric surgery. **Methods:** This cross-sectional study included postmenopausal women divided into two groups: post-bariatric and non-surgical obesity. BMD was assessed by DXA, along with serum calcium, 25(OH)D, PTH, CTX, and morphometric vertebral fractures assessed by Vertebral Fracture Assessment (VFA) using DXA. Prevalence ratios (PR) and 95% confidence intervals (95% CI) were calculated. **Results:** A total of 99 women were included, with 49 in the post-bariatric group and 50 in the non-surgical obesity group. The post-bariatric group presented lower BMD at total femur (0.928 ± 0.134 vs. 1.08 ± 0.108 g/cm²; $p < 0.001$), femoral neck (0.768 ± 0.124 vs. 0.826 ± 0.110 g/cm²; $p < 0.001$), and lumbar spine L1–L4 (0.929 ± 0.121 vs. 0.988 ± 0.106 g/cm²; $p = 0.012$). Higher levels of PTH (106 ± 53.0 vs. 82.5 ± 26 pg/mL; $p = 0.027$) and CTX (0.723 ± 0.349 vs. 0.357 ± 0.231 ng/mL; $p < 0.001$) were also observed. For morphometric vertebral fracture analysis, 90 participants had available VFA assessment. The prevalence of morphometric vertebral fractures was higher in the post-bariatric group (23.8% vs. 6.3%), with PR = 3.81 (95% CI: 1.12–12.93; $p = 0.032$). In linear regression analysis, body composition-related variables, including BMI, did not significantly interfere with lumbar spine BMD differences between groups. **Conclusion:** Postmenopausal women who underwent bariatric surgery presented lower BMD, higher bone remodeling markers, and a higher prevalence of morphometric vertebral fractures compared with women with obesity without previous bariatric surgery.
Keywords: bariatric surgery; postmenopausal women; morphometric vertebral fractures.

BONE AND MINERAL METABOLISM

237 (3070) PRIMARY HYPERPARATHYROIDISM IN A PATIENT WITH EPILEPSY USING ANTICONVULSANT THERAPY: A CASE REPORT

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Case report: A 39-year-old mixed-race woman from Rio de Janeiro with a history of epilepsy since age 25, receiving continuous phenobarbital 100 mg/day and carbamazepine 800 mg/day, presented with chronic fatigue, diffuse bone pain, muscle weakness, asthenia, and emotional lability for approximately one year during outpatient investigation. Initial evaluation showed parathyroid hormone (PTH) of 629 pg/mL (reference range [RR]: 7–53), total serum calcium of 10.2 mg/dL (RR: 8.5–10.5), albumin of 3.8 g/dL, serum phosphorus of 1.1 mg/dL (RR: 2.45–4.5), urinary calcium of 177 mg/24 h (RR: 60–180), and urinary phosphorus of 580 mg/24 h (RR: 340–1000). Cervical radiography revealed findings suggestive of osteomalacia. Cervical ultrasonography demonstrated a solid 2.8 × 1.5 cm nodule in a region compatible with an enlarged left inferior parathyroid gland. Technetium-99m sestamibi scintigraphy showed an orthotopic thyroid gland with heterogeneous radiotracer distribution and no definite hypercaptating focus. Initial management consisted of hydrochlorothiazide 100 mg/day. On follow-up laboratory assessment, total serum calcium was 10.4 mg/dL, serum phosphorus 1.1 mg/dL, and 25-hydroxyvitamin D 27 ng/mL. Calcitriol 0.5 µg/day was initiated, while anticonvulsant therapy was maintained. Subsequent tests demonstrated total serum calcium of 9.3 mg/dL, ionized calcium of 6.1 mg/dL (RR: 4.5–5.2), and serum phosphorus of 0.9 mg/dL. Fine-needle aspiration of the cervical nodule with PTH measurement in the washout fluid revealed levels above 25,000 pg/mL, supporting the diagnosis of parathyroid adenoma. The patient underwent left inferior parathyroidectomy without complications, and histopathological examination confirmed parathyroid adenoma. **Discussion:** This case illustrates an atypical presentation of primary hyperparathyroidism (PHPT) in a patient receiving chronic enzyme-inducing anticonvulsants, which interfere with vitamin D metabolism and intestinal calcium absorption, potentially attenuating hypercalcemia and delaying diagnosis. Despite the absence of marked hypercalcemia at presentation, the substantial elevation in PTH associated with persistent hypophosphatemia and osteomuscular manifestations supported further diagnostic investigation. **Final comments:** This report highlights the importance of detailed clinical assessment, including medication review and integrated interpretation of laboratory and imaging findings, particularly in nonclassical presentations of PHPT.

Keywords: primary hyperparathyroidism; anticonvulsant therapy; epilepsy.

BONE AND MINERAL METABOLISM

238 (2912) PTH-INDEPENDENT HYPERCALCEMIA ASSOCIATED WITH FOREIGN BODY GRANULOMAS AFTER MINERAL OIL INJECTIONS: A DIAGNOSTIC CHALLENGE

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Introduction: PTH-independent hypercalcemia poses a diagnostic challenge; causes include malignancy, granulomatous disease, and vitamin D intoxication. Extrarenal production of 1,25-dihydroxyvitamin D (calcitriol) by activated macrophages in granulomas is a recognized mechanism and can occur with foreign-body granulomas from injected substances. **Case report:** A 42-year-old man with prior extensive stroke and antiphospholipid syndrome, on anticoagulation, was referred for hypercalcemia. He reported previous cosmetic injections of oily substances into the pectoral and biceps regions. Peak serum calcium was 13.0 mg/dL, later 10.3–10.5 mg/dL, with suppressed PTH (5–18 pg/mL) and chronically impaired renal function (creatinine 1.8–2.3 mg/dL). 1,25-dihydroxyvitamin D was elevated at 131 pg/mL; 25-hydroxyvitamin D was variable. Workup found no evidence of malignancy or active autoimmune disease. 18F-FDG PET-CT showed heterogeneous hypermetabolic activity in subcutaneous tissues of the chest and upper limbs at prior injection sites, consistent with granulomatous inflammation. The patient received prednisone with partial calcium normalization, but hypercalcemia recurred during steroid tapering. **Discussion:** Granuloma-associated hypercalcemia results from macrophage 1-alpha-hydroxylase activity producing excess calcitriol, increasing intestinal calcium absorption. Foreign-body granulomas from injected mineral oil, silicone, or other oily substances have been increasingly reported in cosmetic contexts; clinical history and imaging are crucial for diagnosis. Management includes corticosteroids to suppress macrophage activity and standard hypercalcemia measures. Surgical excision of granulomatous tissue can be curative in focal, resectable cases but is often unfeasible when lesions are extensive. **Conclusion:** Exogenous foreign-body granulomatous disease should be considered in the differential diagnosis of PTH-independent hypercalcemia, particularly with a history of cosmetic injections. Early recognition guides appropriate therapy and avoids unnecessary investigations.

Keywords: hypercalcemia; granuloma; calcitriol.

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239 (3348) PTH-INDEPENDENT HYPERCALCEMIA IN MCCUNE-ALBRIGHT SYNDROME WITH EXTENSIVE CRANIOFACIAL FIBROUS DYSPLASIA CASE REPORT

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Case presentation: An 18-year-old female presented with decreased visual acuity in the left eye for three years. Computed tomography of the skull, paranasal sinuses, and orbits showed an expansive, insufflative bone lesion with contrast enhancement in the basisphenoid portion of the clivus and sphenoid body, measuring 7.2 × 5.3 × 2.8 cm, associated with reduced sphenoid sinuses, narrowing of the optic canals (more on the left), and involvement of the internal carotid arteries, with reduced caliber on the left. Bone scintigraphy showed increased osteogenic activity in the sphenoid, and biopsy with immunohistochemistry confirmed fibrous bone dysplasia. Neurosurgery contraindicated surgery due to critical structure involvement. The patient reported menarche at age 13, with regular cycles, no neuropsychomotor developmental delay, fractures, or comorbidities, and denied relevant family history. Physical examination revealed a café-au-lait macule with irregular borders on the left lower limb, compatible with McCune-Albright syndrome. Endocrine evaluation did not reveal hormonal dysfunction. However, biochemical tests showed mild hypercalcemia (corrected calcium: 11 mg/dL), with low PTH (12 pg/mL; reference range 15–65 pg/mL) and normal vitamin D, characterizing PTH-independent hypercalcemia. No identifiable cause for hypercalcemia was found. Conservative treatment with quarterly pamidronate was initiated, with initial reduction in calcium levels, which were subsequently maintained at the upper limit of normal. The patient evolved with frequent headaches, and the most recent magnetic resonance showed lesion growth and a mild compressive effect on the adenohypophysis, without hypofunction. **Discussion:** McCune-Albright syndrome (MAS) results from activating mutations in the GNAS gene, leading to variable manifestations including fibrous dysplasia, café-au-lait macules, and endocrine hyperfunction. Alterations in calcium metabolism are rare, with an association between the MAS and primary hyperparathyroidism that remains unclear. No reports of hypercalcemia with normal or suppressed PTH in this condition were identified in the literature. Another important aspect of this case is the extensive craniofacial involvement with compressive effects, which limits surgical approaches. **Final comments:** The absence of reports of PTH-independent hypercalcemia in MAS highlights the relevance of this case, contributing to expanding knowledge of the disease's endocrine spectrum.

Keywords: hypercalcemia; fibrous dysplasia; McCune-Albright syndrome.

BONE AND MINERAL METABOLISM

240 (3060) PTHRP-MEDIATED HYPERCALCEMIA DURING LACTATION IN A PATIENT WITH POST-SURGICAL HYPOPARATHYROIDISM: A CASE REPORT

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Case presentation: A 23-year-old woman with Graves' disease underwent total thyroidectomy, complicated by permanent hypothyroidism and hypoparathyroidism. She was treated with levothyroxine, calcium carbonate, and calcitriol. During follow-up, calcitriol was temporarily discontinued after partial parathyroid recovery, with a maximum PTH of 17 pg/mL off calcitriol, while normocalcemia was maintained on calcium carbonate. During pregnancy, calcitriol was reintroduced and calcium carbonate doses were increased due to gestational calcium demands and low ionized calcium levels. One day postpartum, ionized calcium was 4.18 mg/dL with PTH 7 pg/mL, consistent with baseline hypoparathyroidism. She remained on calcium carbonate (5 tablets/day) and calcitriol (3 tablets/day). Twenty-four days postpartum, during active breastfeeding, she developed severe asymptomatic hypercalcemia, with ionized calcium of 6.09 mg/dL and undetectable PTH (<6 pg/mL), consistent with PTH-independent hypercalcemia. PTHrP was 20 pg/mL (11-20 pg/mL), at the upper limit of normal, suggesting lactation-related PTHrP secretion as a contributing factor. Calcitriol was discontinued, leading to normalization of calcium levels within 24 hours. Calcitriol was later cautiously reintroduced at a lower dose (1 tablet/day) after ionized calcium decreased to 4.3 mg/dL. Subsequent monitoring demonstrated stable ionized calcium levels (4.53–4.72 mg/dL) on calcium carbonate (3 tablets/day) plus low-dose calcitriol, despite ongoing breastfeeding. The patient remained asymptomatic, without clinical signs of hypocalcemia. **Discussion:** This case highlights lactation-induced hypercalcemia, probably due to increased PTHrP secretion from the breast, secondary to breastfeeding stimulation. In healthy women, this secretion of PTHrP mobilizes calcium from the bones and blocks PTH from the parathyroid, thus maintaining blood calcium levels. In patients with hypoparathyroidism who are breastfeeding, the production of PTHrP, combined with calcium and calcitriol replacement, can lead to hypercalcemia. Correction of hypercalcemia after discontinuation of calcitriol and reduction of calcium dose supports the central role of mammary PTHrP in the development of hypercalcemia. **Final comments:** Breastfeeding is a high-risk period for hypercalcemia in patients with hypoparathyroidism receiving calcium and calcitriol. Frequent calcium monitoring and prompt adjustment of these medications doses are essential during the postpartum period.

Keywords: parathyroid hormone-related protein; hypoparathyroidism; hypercalcemia.

BONE AND MINERAL METABOLISM

241 (2884) PYCNODYSOSTOSIS: A CONSEQUENCE OF CONSANGUINITY

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In the present study, we present the cases of two siblings, L.G.O. (female, 9 years old) and R.G.O. (male, 7 years old), referred for multidisciplinary follow-up. Born to consanguineous parents (second cousins), the diagnosis of pycnodysostosis was confirmed in R.G.O. via exome sequencing, which identified the homozygous pathogenic variant c.721C>T (p.Arg241). For his sister, L.G.O., the diagnosis was established through clinical-radiological criteria and direct familial correlation. Both present the classic phenotype: osteodysplasia, growth deficit with significant bone age delay and craniofacial dysmorphisms. Notably, L.G.O. presents with blue sclerae, Level 2 autism spectrum disorder (ASD), and utilizes a lumbar orthopedic brace; meanwhile, R.G.O. exhibits speech impairment and a history of a lower limb fracture. Both are currently treated with Somatropin (13 and 12 IU/day, respectively). Pycnodysostosis is a rare sclerosing bone dysplasia with an autosomal recessive inheritance pattern, resulting from mutations in the CTSK gene. This genetic alteration causes a deficiency in the cathepsin K enzyme, a protein fundamental for osteoclasts to degrade the organic bone matrix during the remodeling process. It is estimated that the incidence of this condition is 1 to 1,7 per 1,000,000 individuals worldwide. Clinically, the pathology manifests as generalized osteosclerosis (~100%), marked short stature (~100%), bone fragility prone to fractures (~70%), acro-osteolysis of the terminal phalanges (>90%), brachydactyly (>90%), and craniofacial anomalies, such as non-closure of the fontanelles (~80%), frontal bossing (>80%) and mandibular hypoplasia (>70%). Other manifestations include midface retrusion (60%), blue sclerae (30%–40%), and obstructive sleep apnea (>65%). Given its recessive nature, parental consanguinity is a determining factor, present in approximately 30% of recorded cases. The discussion of these cases emphasizes that pycnodysostosis requires a high index of clinical suspicion, especially when short stature is associated with radiological findings of osteosclerosis. Early detection through the investigation of consanguinity history is crucial for implementing specialized care aimed at minimizing skeletal deformities. Considering that current therapeutic management remains focused on symptom control and complication prevention, continuous multidisciplinary follow-up is essential to ensure improved functionality and quality of life for these patients.

Keywords: consanguinity; pycnodysostosis; bone dysplasia.

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242 (3303) REAL-WORLD EVIDENCE ON RALOXIFENE IN POSTMENOPAUSAL OSTEOPOROSIS: EPIDEMIOLOGICAL PROFILE, OUTCOMES, AND SAFETY OVER 26 YEARS AT A PUBLIC ENDOCRINOLOGY REFERENCE CENTER IN BAHIA, BRAZIL

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Introduction: Postmenopausal osteoporosis impacts quality of life and healthcare costs. A selective estrogen receptor modulator, raloxifene, available within the Brazilian Public Health System (SUS), is a consolidated alternative for women with bisphosphonate contraindications or intolerance, offering antiresorptive effects, vertebral fracture risk reduction, and protection against breast cancer. Concerns regarding venous thromboembolism (VTE) and cardiovascular complications frequently limit its prescription. Real-world evidence on its efficacy and safety from the SUS remains scarce. **Objective:** To describe the epidemiological profile, therapeutic outcomes, and safety of raloxifene in postmenopausal women followed at a Public Endocrinology Reference Center in Bahia, Brazil, over 26 years. **Methods:** Observational, descriptive, retrospective study reviewing medical records of women treated with raloxifene from 1999–2025. Clinical and laboratory data, bone densitometry (DXA) results, fragility fracture history, adverse effects, cardiovascular and thromboembolic outcomes, and discontinuation reasons were collected. **Results:** Forty female patients; mean age 71.1 years were analyzed. Primary indication was bisphosphonate intolerance or suboptimal response to long-term bisphosphonate therapy. Nine patients (22.5%) had prior fragility fractures. Among 32 records with comparable DXA data, 14 (43.75%) showed vertebral bone mineral density (BMD) increase and 3 (9.4%) maintained stable BMD — favorable response or stabilization in more than half of evaluated and comparable cases. Twenty-nine non-mutually exclusive discontinuations were recorded: 22 BMD loss, 3 adverse effects, 2 elevated serum C-telopeptide levels, 3 vertebral fractures during treatment. Regarding safety, results were favorable: one patient is under investigation for atrial fibrillation with probable association with cardiomegaly. No VTE events or serious cardiovascular complications directly attributable to raloxifene were recorded. **Conclusion:** Supported by 26 years of longitudinal real-world follow-up, this study demonstrates that raloxifene is a valid, efficacious, and safe alternative within the SUS for selected patients. BMD stabilization or gain combined with the absence of VTE events reinforces its judicious use. Longitudinal specialized follow-up and individualized risk-benefit assessment are essential, reinforcing the endocrinologist's central role in managing osteoporosis in public healthcare.

Keywords: postmenopausal osteoporosis; raloxifene; real-world evidence.

BONE AND MINERAL METABOLISM

243 (3211) REFRACTORY EPILEPSY DUE TO HYPOCALCEMIA IN PRIMARY HYPOPARATHYROIDISM WITH FAHR SYNDROME: THERAPEUTIC DILEMMAS IN BALANCING NORMOCALCEMIA AND NEPHROPROTECTION

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Case presentation: M.R.S., a 43-year-old man, developed generalized tonic-clonic seizures in 2004 and was treated for 15 years as refractory epilepsy with carbamazepine. During an episode of poorly controlled seizures, severe hypocalcemia was identified in the emergency department, and calcium replacement led to clinical improvement. Brain CT and MRI revealed symmetrical calcifications involving the thalamus, basal ganglia, juxtacortical regions, and cerebellar dentate nucleus, consistent with Fahr syndrome. Endocrinological evaluation showed undetectable parathyroid hormone levels (PTH < 4.6 pg/mL), confirming primary hypoparathyroidism, likely autoimmune. The patient also had a history of early bilateral cataracts at age 31 and recurrent nephrolithiasis. Treatment with oral calcium and calcitriol resulted in complete seizure remission, allowing progressive withdrawal of carbamazepine and discharge from Neurology follow-up. **Discussion:** This case illustrates long-standing, previously unrecognized hypoparathyroidism presenting as refractory epilepsy associated with extensive CNS calcifications. Seizure remission after partial correction of hypocalcemia supports the metabolic disorder, rather than primary epilepsy, as the main causal factor. The association with early cataracts, nephrolithiasis, and Fahr syndrome suggests prolonged and underdiagnosed hypocalcemia and hyperphosphatemia. During follow-up (2021–2026), partial biochemical control was achieved, although progressive hypercalciuria developed, reaching 373 mg/24h, with early chronic kidney disease. This emphasizes the therapeutic dilemma in hypoparathyroidism: achieving normocalcemia at the cost of higher calcium and calcitriol supplementation, increasing the risk of nephrocalcinosis and renal impairment. Hydrochlorothiazide was optimized (25 mg/day), and serum calcium targets were maintained at the lower limit of normality, prioritizing nephroprotection and prevention of recurrent nephrolithiasis while keeping the patient asymptomatic and seizure-free. **Conclusion:** This case emphasizes the importance of investigating calcium-phosphate metabolism disorders in presumed refractory epilepsy, especially when associated with CNS calcifications, early cataracts, and nephrolithiasis. Management of chronic hypoparathyroidism requires balancing symptomatic control, prevention of ectopic calcifications, and preservation of renal function through multidisciplinary follow-up and close biochemical monitoring.

Keywords: primary hypoparathyroidism; Fahr syndrome; hypocalcemia.

BONE AND MINERAL METABOLISM

244 (3498) SARCOPENIA, FRAILTY, AND FRACTURE RISK IN BRAZILIAN LONG-TERM CARE FACILITIES: A COMPARISON BETWEEN PUBLIC AND PRIVATE INSTITUTIONS IN A MEDIUM-SIZED CITY FROM SÃO PAULO STATE, BRAZIL

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Introduction: Residents of long-term care facilities (LTFs) are at high risk for sarcopenia, frailty, and osteoporosis. Few Brazilian studies have compared these conditions across different LTF funding models or assessed gaps in fracture risk identification and treatment. **Objective:** To evaluate and compare the prevalence of high sarcopenia probability, frailty, and high fracture risk among residents of for-profit private (FP-LTF) and public/philanthropic (PP-LTF) facilities, and to assess rates of osteoporosis diagnosis and pharmacological prevention. **Methods:** Cross-sectional observational study conducted between February 2024 and April 2025, enrolling 117 residents (aged 61–96 years) from three facilities. Sarcopenia probability was estimated using the Ishii Score, based on age, handgrip strength, and calf circumference. Frailty was assessed with the Clinical Frailty Scale. Fracture risk was estimated using the FRAX tool. Osteoporosis diagnosis and use of vitamin D, calcium, and anti-osteoporotic drugs were retrieved from medical records. **Results:** FP-LTF residents were older (mean 83.6 vs. 76.2 years) and more frequently female ($p < 0.01$). They also had a higher prevalence of smoking (25% vs. 2.9%; $p < 0.01$). Sarcopenia by Ishii Score was found in approximately 78% of participants in both groups. Compromised mobility and activities of daily living were observed in 75% of all residents (80.9% in FP-LTFs vs. 66.7% in PP-LTFs; $p = 0.453$). High fracture risk was present in approximately 45% of women and less than 3% of men, with no significant difference between LTFs. In multivariate analysis, Ishii Score had a moderate correlation with Clinical Frailty Scale ($r = 0.478$, $p < 0.001$) and with a high risk of fracture in FRAX ($r = 0.422$, $p < 0.001$). Bone densitometry had been performed in the prior three years in only 4.8% of FP-LTF and none of PP-LTF residents. Osteoporosis was diagnosed in 11.6% of FP-LTF and 2.1% of PP-LTF residents; all of whom were receiving bisphosphonate therapy. Vitamin D supplementation was more frequent in FP-LTFs than in PP-LTFs (39.1% vs. 4.2%; $p < 0.001$), as was calcium supplementation (14% vs. 6%; $p = 0.16$). **Conclusion:** High prevalences of sarcopenia, frailty, and fracture risk were found regardless of LTF funding model, alongside substantial underdiagnosis of osteoporosis — particularly in public and philanthropic facilities. Structured musculoskeletal screening protocols may help prevent adverse outcomes in institutionalized older adults.

Keywords: osteoporosis; sarcopenia; frailty.

BONE AND MINERAL METABOLISM

245 (3396) SEVERE AND PROLONGED FERRIC CARBOXYMALTOSE-INDUCED HYPOPHOSPHATEMIA IN A BARIATRIC SURGERY PATIENT: A CASE REPORT

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Case presentation: A 39-year-old woman with a history of gastric bypass in 2009 presented with iron deficiency refractory to oral supplementation, associated with fatigue and difficulty gaining muscle mass. In February 2026, she had a ferritin level of 7 ng/mL, normal serum phosphorus (3.0 mg/dL), and normal calcium, magnesium, vitamin D levels, and renal function, and intravenous ferric carboxymaltose was indicated. Two infusions were administered one week apart, with 1000 mg per infusion (total dose: 2000 mg). Approximately two weeks after the second infusion, she developed headache, abdominal pain, marked asthenia, severe fatigue, and muscle weakness. Laboratory evaluation revealed severe hypophosphatemia (0.7 mg/dL), confirmed on repeat testing, with normal calcium, magnesium, PTH, vitamin D, and renal function. She was hospitalized and required multiple sodium glycerophosphate infusions, with slow improvement in serum phosphorus levels, being discharged with phosphorus of 1.7 mg/dL. A few days after discharge, hypophosphatemia recurred (0.7 mg/dL), requiring readmission. After the second discharge, calcitriol and oral chelated phosphorus supplementation were started. Currently, she maintains phosphorus levels between 1.7 and 2.0 mg/dL, associated with elevated PTH (117 pg/mL), despite adequate vitamin D and preserved renal function, and remains asymptomatic. **Discussion:** Ferric carboxymaltose-induced hypophosphatemia is a recognized but frequently underdiagnosed complication related to increased biologically active FGF23, leading to renal phosphaturia and reduced calcitriol synthesis. Although most cases are transient, severe and prolonged forms may occur, especially in patients with predisposing factors such as bariatric surgery and malabsorptive disorders. In the present case, the clinical severity, the need for multiple sodium glycerophosphate infusions, early recurrence after intravenous replacement, and persistence of hypophosphatemia during outpatient follow-up, associated with elevated PTH, suggest a persistent mineral disorder. **Final comments:** This report demonstrates severe, recurrent, and prolonged hypophosphatemia after ferric carboxymaltose use in a patient who had undergone gastric bypass, highlighting the vulnerability of this clinical profile to persistent mineral disorders. Early identification and careful laboratory monitoring are essential for greater therapeutic safety.

Keywords: hypophosphatemia; ferric carboxymaltose; bariatric surgery.

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246 (3332) SEVERE HYPOPARATHYROIDISM ASSOCIATED WITH SECONDARY FAHR'S SYNDROME IN A YOUNG PATIENT WITH NEUROPSYCHIATRIC AND SEIZURE-RELATED SYMPTOMS

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Case presentation: A 36-year-old female patient was admitted with psychomotor agitation, aggression, insomnia, and recurrent seizures, initially suspected to be associated with the use of psychiatric medications. She developed severe hypocalcemia secondary to hypoparathyroidism, presenting with persistent hyperphosphatemia. A cranial computerized tomography scan revealed multiple symmetrical foci of calcification in the basal ganglia and deep white matter, consistent with secondary Fahr's syndrome. She also presented with hirsutism and androgenetic alopecia and was being evaluated for polycystic ovary syndrome and primary adrenal insufficiency. During hospitalization, she developed heart failure with reduced ejection fraction, acute renal failure, and drug-induced dermatitis. She received calcium and calcitriol replacement therapy, with progressive clinical and laboratory improvement. Additional tests revealed mildly elevated thyroid-stimulating hormone, hyperprolactinemia, and reduced dehydroepiandrosterone sulfate. She was discharged from the hospital for multidisciplinary outpatient follow-up. **Discussion:** Fahr's syndrome is characterized by bilateral intracranial calcifications, particularly in the basal ganglia, and is frequently associated with disorders of calcium and phosphorus metabolism. Hypoparathyroidism is one of the main secondary causes and should be investigated in the presence of neuropsychiatric manifestations and seizures associated with hypocalcemia. This case highlights the diagnostic complexity in patients initially managed as having primary psychiatric disorders, delaying the identification of the underlying endocrine etiology. Furthermore, it demonstrates the broad systemic repercussions of metabolic dysfunction, including cardiovascular and neurological involvement. **Final comments:** This report highlights the importance of metabolic and endocrinological investigation in patients with atypically presenting neuropsychiatric manifestations associated with seizures. Early identification of hypoparathyroidism allowed for appropriate therapeutic management, with significant clinical improvement following correction of hypocalcemia. Furthermore, the case underscores the need for a multidisciplinary approach given the diversity of associated systemic manifestations, contributing to greater recognition of secondary Fahr's syndrome in clinical practice and a reduction in diagnostic delays.

Keywords: hypoparathyroidism; Fahr's syndrome; neurological disorders.

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247 (2940) SEVERE POST-THYROIDECTOMY HYPOPARATHYROIDISM ASSOCIATED WITH AUTOIMMUNE ATROPHIC GASTRITIS AND CHRONIC SPONTANEOUS URTICARIA: A CASE REPORT

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Case presentation: A 48-year-old woman underwent total thyroidectomy with preservation of one parathyroid gland in 2020 for non-toxic multinodular goiter and subsequently developed permanent postsurgical hypoparathyroidism (PTH < 3 pg/mL), complicated by recurrent symptomatic hypocalcemia. At presentation, laboratory tests showed total serum calcium of 6.4 mg/dL and phosphorus of 5.2 mg/dL while receiving calcitriol 1.0 mcg/d and calcium carbonate equivalent to 2 g/d of elemental calcium. Since surgery, she had experienced recurrent hypocalcemic crises marked by tetany, diffuse paresthesia, and muscle weakness, needing repeated intravenous (IV) calcium replacement. Further study of refractory hypocalcemia revealed vitamin B12 deficiency, iron-deficiency anemia, and mild hypomagnesemia. Autoimmune atrophic gastritis was confirmed by positive autoantibodies and gastric biopsy, contributing to therapeutic failure. Also, the patient had chronic spontaneous urticaria with drug-induced exacerbations, suggesting systemic autoimmunity dysfunction. Therapeutic escalation included calcitriol 3.75 mcg/d, calcium carbonate (2 g elemental calcium/d), calcium citrate (800 mg elemental calcium/d), oral magnesium, and twice-weekly IV calcium gluconate. Despite these interventions, hypocalcemia persisted, with serum calcium levels remaining around 6.0 mg/dL, leading to more than 20 emergency department visits in 2025. Hydrochlorothiazide 50 mg was initiated to control hypercalciuria resulting from calcium therapy. Given the ineffective response to conventional treatment, teriparatide was initiated in 2025 twice daily, resulting in initial clinical improvement. However, hypocalcemia recurrence occurred, suggesting secondary loss of therapeutic response. Ongoing dependence on IV calcium administration and exhaustion of peripheral venous access ultimately required placement of a long-term central venous catheter. **Discussion:** The malabsorption, due to autoimmune disease, generates hypochlorhydria, decreasing the absorption of calcium and magnesium, which is crucial for the secretion and action of PTH. In addition, PTH analogs have emerged as therapeutic options in selected refractory cases. Considering the refractory hypoparathyroidism, palopegteriparatide, a long-acting PTH analog was prescribed. **Conclusion:** The psychosocial impact and clinical recurrence highlight the need for individualized care and careful risk-benefit assessment of invasive procedures.

Keywords: hypoparathyroidism; autoimmune gastritis; urticaria.

BONE AND MINERAL METABOLISM

248 (3576) SEVERE PRIMARY HYPERPARATHYROIDISM IN A PATIENT WITH ADVANCED CHRONIC KIDNEY DISEASE

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Case presentation: A 67-year-old female patient with type 2 diabetes mellitus, stage 4 chronic kidney disease (eGFR 24–27 mL/min/1.73 m^2), heart failure with reduced ejection fraction, systemic arterial hypertension, high cardiovascular risk, and previous myocardial infarction was referred for investigation of persistent hypercalcemia. Laboratory evaluation revealed total serum calcium up to 15.1 mg/dL, ionized calcium of 1.73 mmol/L, and parathyroid hormone (PTH) levels up to 518.2 pg/mL, associated with low-normal phosphorus levels and vitamin D insufficiency. Twenty-four-hour urinary calcium excretion remained low, ranging from 79–84 mg/24h. Parathyroid scintigraphy showed no evidence of adenoma or hyperplasia. Bone mineral density demonstrated significant osteopenia at the femoral neck (T-score -2.4). The patient subsequently developed ureterolithiasis requiring hospitalization and double-J ureteral stent placement. Due to high cardiovascular and renal surgical risk, conservative treatment with denosumab associated with cinacalcet was initiated. Partial reduction and control of hypercalcemia were observed during outpatient follow-up. **Discussion:** Primary hyperparathyroidism (PHPT) is an important cause of hypercalcemia, and parathyroidectomy remains the definitive treatment. However, patients with multiple comorbidities may present high operative risk, making medical management a necessary alternative. Furthermore, negative localization studies do not exclude PHPT, especially in cases of multiglandular hyperplasia or small adenomas. Denosumab represents an important therapeutic strategy for hypercalcemia control in patients who are not surgical candidates, in addition to contributing to skeletal protection in individuals with associated mineral and bone disorders. **Final comments:** This case highlights the diagnostic and therapeutic challenges of primary hyperparathyroidism in patients with advanced chronic kidney disease and high surgical risk. Low urinary calcium excretion does not exclude PHPT in this clinical setting, and laboratory findings must be interpreted carefully. Conservative treatment with denosumab may improve hypercalcemia and represent a feasible alternative in patients with contraindications to surgery.

Keywords: hyperparathyroidism; chronic kidney disease; denosumab.

BONE AND MINERAL METABOLISM

249 (3039) SEVERE PRIMARY HYPERPARATHYROIDISM IN A YOUNG ADULT WITH EXTREME HYPERCALCEMIA AND MULTISYSTEM COMPLICATIONS: A CASE REPORT

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Case presentation: A previously healthy 21-year-old man presented with a six-month history of progressive right knee pain radiating to the quadriceps and gluteal region, evolving with claudication, loss of ambulation, and bilateral pathological femoral fractures after a fall, requiring osteosynthesis. Laboratory evaluation revealed severe hypercalcemia (calcium: 18.5 mg/dL), PTH initially of 334 pg/mL with subsequent elevation to 971 pg/mL, alkaline phosphatase of 1020 U/L, and severe vitamin D deficiency (8 ng/mL). Due to refractory hypercalcemia, hemodialysis was required. The patient also developed acute pancreatitis, with elevated amylase and lipase, in addition to cholelithiasis on abdominal ultrasound. Imaging studies identified a lesion compatible with parathyroid adenoma, and parathyroidectomy was performed, with histopathological confirmation of a single left inferior parathyroid adenoma (2.2 × 1.5 × 1.3 cm; 1.64 g). Postoperatively, he developed hungry bone syndrome, requiring intensive calcium and calcitriol replacement. During hospitalization, he further evolved with perforated acute abdomen, abdominal sepsis, and microbiologically confirmed osteomyelitis. Additional investigation for Multiple Endocrine Neoplasia, including baseline pituitary hormonal assessment and imaging studies of the pituitary and gastrointestinal tract, showed no abnormalities suggestive of the syndrome. The patient remains under outpatient follow-up and is awaiting complementary genetic testing. **Discussion:** Primary hyperparathyroidism is currently diagnosed predominantly in asymptomatic or oligosymptomatic stages, making severe presentations with marked hypercalcemia and multisystem involvement uncommon. This case is notable for the patient's young age and severe manifestations, including advanced bone disease, bilateral pathological fractures, acute pancreatitis, and the need for hemodialysis. The occurrence of hungry bone syndrome highlights the profound skeletal involvement associated with severe disease. Despite negative initial screening for Multiple Endocrine Neoplasia, the early and aggressive presentation supports further genetic investigation for hereditary syndromes. **Final comments:** This case illustrates a rare and potentially fatal presentation of primary hyperparathyroidism in a young patient, emphasizing the importance of early recognition, intensive multidisciplinary management, and investigation of hereditary etiologies in severe and early-onset disease.
Keywords: primary hyperparathyroidism; hypercalcemia; multiple endocrine neoplasia.

BONE AND MINERAL METABOLISM

250 (2933) THE BIOCIDE TRIBUTYLTIN EXACERBATES ALVEOLAR BONE DAMAGE INDUCED BY PERIODONTITIS IN FEMALE RATS

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Tributyltin (TBT) is an endocrine-disrupting chemical (EDC) with industrial and naval applications. Despite its restricted regulatory status, it is found in marine ecosystems, and the primary human exposure pathway is the consumption of contaminated seafood, which involves buccal contact. The exposure to TBT in mammals is associated with a plethora of harmful effects such as obesogenesis, reproductive and cardiovascular disorders, thyroid and bone disruption. Despite that, EDC's role in the establishment or development of increasingly prevalent oral diseases, such as periodontitis (PD), is unknown. This work aimed to evaluate if the chronic exposure to tributyltin may have detrimental effects on periodontal tissues, physiologically regulated by hormonal signalling, in a rat model of periodontitis. Adult female Wistar rats received oral administration of tributyltin (100 ng/kg once daily; TBT group) or its vehicle (VEH group) for 30 days and, on day 15, were submitted to ligature-induced periodontitis (TBT + PD and VEH + PD groups). Body weight, food intake, thyroid hormone levels, glycemic and lipid profiles and locomotor activity were evaluated for metabolic evaluation. The alveolar bone loss was measured using linear and microtomographic methods; also, the histological analysis of periodontal structures was performed. Gingiva was collected for myeloperoxidase activity and TNF levels measurement. Notwithstanding its extensively documented endocrine-disrupting properties, TBT alone did not alter metabolic or periodontal outcomes. Periodontitis was linked to increased adiposity and, in conjunction with TBT, higher alveolar bone destruction with non-viable osteocytes, indicating that TBT exposure may provide an additional danger for the advancement of periodontal diseases. This study expands current knowledge regarding organotin effects on skeletal bone and tooth development and demonstrates the toxic potential of tributyltin in periodontal bone.
Keywords: endocrine-disrupting chemicals; tributyltin; periodontal bone loss.

BONE AND MINERAL METABOLISM

251 (3032) THE PARADOX OF HIGH MINERAL DENSITY AND BONE FRAGILITY: PYCNODYSOSTOSIS CASE REPORT A RARE OSTEOPATHY

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Case presentation: A 28-year-old female, born to consanguineous parents, presented with a history of bone fragility since age 6. Between ages 6 and 12, she suffered six low-impact fractures of the lower limbs, including a femoral shaft fracture after a fall from standing height. There was no similar family history. Physical examination revealed short stature, cranial asymmetry (sagittal suture disjunction), midface hypoplasia, saddle nose, proptosis, narrow palate, and dental malocclusion. Extremity findings included widened thumbs and an increased space between the first and second toes. Laboratory investigation at age 10 showed total calcium of 8.8 mg/dL (RR: 9.2–11.0) and alkaline phosphatase of 309 U/L (RR: <300), with normal calciuria, phosphorus, and creatinine. Hand radiographs demonstrated generalized increased bone density; bone densitometry (L1 to L4) revealed a BMD 152% of the expected for her age. Based on the phenotype and imaging, pycnodysostosis was hypothesized. The patient remained under orthopedic follow-up and was only referred to Endocrinology at age 28. **Discussion:** pycnodysostosis is a rare autosomal recessive skeletal dysplasia (chromosome 1q21). Its pathophysiology involves mutations in the cathepsin K gene, a lysosomal protease essential for osteoclast function. The inability to resorb the organic matrix results in osteosclerosis; however, the bone is architecturally fragile and inflexible. Diagnosis is based on the characteristic phenotype, such as delayed fontanelle closure, Wormian bones, midface hypoplasia, dental abnormalities, and acro-osteolysis (resorption of the distal phalanges). Short stature occurs in 50% of cases, often associated with GH deficiency. Clinically, recurrent fractures are the complication with the greatest impact on quality of life. In the differential diagnosis with Osteopetrosis, pycnodysostosis stands out due to the preservation of the medullary cavity (absence of hematimetric changes) and the rarity of cranial nerve compression. **Final considerations:** Diagnosing pycnodysostosis requires recognizing its phenotypic features and the paradox of bone fragility despite high mineral density. Early recognition and management by Endocrinology are crucial for metabolic surveillance and complication monitoring, primarily to minimize the impact of short stature on quality of life.
Keywords: pycnodysostosis; osteosclerosis; bone fragility.

BONE AND MINERAL METABOLISM

252 (3089) THE RELATIONSHIP BETWEEN BODY MASS INDEX AND BONE MINERAL DENSITY IN ADOLESCENTS

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Introduction: The relationship between body weight and bone mineral content acquisition in children and adolescents is not yet well-defined in the literature. Some studies suggest that an increase in body mass index (BMI) leads to an increase in bone mineral density (BMD). However, other publications state that high BMI in young individuals may hinder BMD progression depending on the site analyzed. Since childhood and adolescence are essential stages for proper skeletal development, further investigations on the topic are necessary. **Objective:** To present the relationship between BMI and BMD in young individuals aged 14 to 18 years. **Methods:** This is an analytical, cross-sectional, and retrospective study with a sample of 50 adolescents. Body weight was analyzed using a digital electronic scale with a maximum capacity of 200 kg and a precision of 50 g. For height, a digital ultrasonic stadiometer was used. BMI was obtained from the ratio of weight (in kilograms) by the square of height (in meters). For BMD analysis, dual-energy X-ray absorptiometry (DXA) of the lumbar spine (vertebrae L1 to L4) was used. The study was approved by the ethics committee via Plataforma Brasil. **Results:** Of the 50 volunteers, 21 (42%) were male and 29 (58%) were female. Regarding BMI, the mean value obtained was 22.36 kg/m², with a maximum value of 35.99 kg/m² and a minimum of 16.85 kg/m². BMD showed a mean value of 1.18 g/cm², a maximum of 1.54 g/cm², and a minimum of 0.79 g/cm². When correlating BMI and BMD for the total sample, a positive correlation is observed between both variables, where each one-unit increase in BMI is accompanied by an increase of 0.012 g/cm² in BMD. In an analysis of the female and male populations, an increase in BMD of 0.016 g/cm² and 0.007 g/cm², respectively, is also observed for each unit increase in BMI. Furthermore, when conducting this assessment for the most statistically relevant age group (the 18-year-old subgroup consisting of 21 individuals), there was a correlation between BMI and BMD of +0.018 g/cm² per BMI unit. **Conclusion:** The study evidenced a positive correlation between BMI and BMD for both the total sample and by sex. Additionally, the total female population showed a BMD correlation 2.28 times higher than the total male population.
Keywords: bone density; body mass index; adolescent.

253 (3087) TREATMENT OF X-LINKED HYPOPHOSPHATEMIA: A SYSTEMATIC REVIEW OF THE EFFICACY OF BUROSUMAB COMPARED TO CONVENTIONAL THERAPY

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Introduction: X-linked hypophosphatemia (XLH) is a rare genetic disease caused by loss-of-function mutations in the PHEX gene, which lead to renal phosphate wasting and impaired bone mineralization. In childhood, it manifests primarily as hypophosphatemic rickets, bone deformities, short stature, pain, and functional limitation. **Objective:** To evaluate the efficacy and safety of burosumab compared to conventional therapy in the treatment of pediatric patients with XLH. **Methods:** A systematic review was conducted according to PRISMA 2020 and structured by the PICOS strategy: pediatric patients with XLH; intervention with burosumab; comparison with conventional therapy based on oral phosphate and active vitamin D/calcitriol or other approaches; outcomes related to rickets severity, serum parameters, radiographic improvement, growth, locomotion, and adverse events; and the inclusion of randomized, cross-sectional, and cohort studies. The PubMed/MEDLINE, Cochrane Library, and VHL (Virtual Health Library) databases were searched, considering articles published between 2020 and 2025. Three hundred fifty-three (353) studies were identified, of which 7 met the eligibility criteria. The quality of evidence was assessed using GRADE, and the risk of bias was evaluated by tools appropriate to the study designs. **Results:** Burosumab demonstrated superiority over conventional therapy in improving biochemical, radiographic, and functional parameters of pediatric XLH. Studies indicated an increase in serum phosphorus, improvement in rickets severity, reduction in bone lesions and radiographic deformities, as well as benefits in linear growth, physical function, and mobility. Conventional therapy remained useful in optimized protocols; however, it showed limited response in persistent cases, with the maintenance of rickets, short stature, and deformities after prolonged treatment. The main adverse events associated with burosumab were pain, headache, cough, and dental alterations, requiring regular monitoring. **Conclusion:** Burosumab presented greater clinical, biochemical, radiographic, and functional benefits compared to conventional therapy in the pediatric treatment of XLH. Despite the occurrence of monitorable adverse events, the findings reinforce its therapeutic relevance in children and adolescents, especially in the improvement of hypophosphatemic rickets, bone mineralization, and physical function.

Keywords: hypophosphatemic rickets; burosumab; phosphate therapy.

NEUROENDOCRINOLOGY

254 (3053) "BLOCK AND REPLACE" ATUMELNANT (CRN04894) AND PHYSIOLOGIC HYDROCORTISONE REPLACEMENT RAPIDLY CONTROLS HYPERCORTISOLISM IN ACTH-DEPENDENT CUSHING SYNDROME

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Atumelnant (CRN04894) is a first-in-class, once daily, oral, selective melanocortin type 2 receptor antagonist in development for treatment of ACTH-dependent Cushing Syndrome. Findings from the 80-mg cohort from the first-in-disease, dose-finding study (NCT05804669) were previously reported. All participants achieved urinary-free cortisol (UFC) below upper limit of normal (ULN; <45 µg/d) within 7 days and required physiologic hydrocortisone replacement therapy (PHRT) due to early morning serum cortisol (EMC) < 5 µg/dL. Here, we present findings from the 40-mg cohort. Participants (UFC > 1.3 x ULN, ACTH > 10 pg/mL) received atumelnant 40 mg for 10 days, followed by 4-day washout. EMC < 5 µg/dL triggered PHRT. Endpoints included changes in UFC, EMC, and early morning ACTH (ULN < 46 pg/mL). Questionnaires assessed adverse events and symptoms of hypercortisolism and adrenal insufficiency. Data are median (ranges). All participants (6 females, 41.5 years [18–59]) completed the protocol. EMC reached < 5 µg/dL in all after 3.5 doses (1–9), prompting PHRT (8.9–10.9 mg/m²/d). From baseline to Day 10, UFC decreased: 352.5 µg/d (35–1655) *vs.* 42 µg/d (8–339). Biochemical disease control (UFC < ULN) was achieved in 4 participants and maintained in 3 while on PHRT. From baseline to Day 11, EMC decreased: 15.0 µg/dL (11.0–26.1) *vs.* 3.5 µg/dL (<1.0–18.9), while early morning ACTH increased: 35 pg/mL (9.5–67.2) *vs.* 112 pg/mL (19.4–256). Hypercortisolism symptoms improved (*e.g.*, fatigue, swelling/bloating; both 3/3 participants). EMC < 5 µg/dL occurred in 6/6 participants. PHRT was discontinued after Day 10 when EMC was ≥7 µg/dL, median (range) 12.5 (10–15) days. Headache, nausea, anorexia and/or malaise were reported by 4 participants when EMC reached <5 µg/dL but improved with PHRT. Two participants had asymptomatic, mild increases in alanine aminotransferase; aspartate aminotransferase increased in one participant. Bilirubin, alkaline phosphatase, and creatinine were unaltered. In conclusion, atumelnant 40 mg lowered EMC, normalized UFC, and improved symptoms of hypercortisolism. Combination 'Block & Replacement' therapy of atumelnant and PHRT was well tolerated.

Keywords: atumelnant; Cushing syndrome; block and replace.

NEUROENDOCRINOLOGY

255 (3106) A NATIONWIDE MULTICENTER STUDY OF THE SF3B1 VARIANT IN LACTOTROPH ADENOMAS

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Introduction: The genetic basis of lactotroph adenomas remains poorly understood. A somatic variant in splicing factor 3 subunit B1 (SF3B1) was first identified in prolactinomas only six years ago, with a reported prevalence of 19.8% in a Chinese cohort. The variant was detected using digital droplet PCR (ddPCR), a more sensitive method than Sanger sequencing for detecting low-frequency mutant alleles. In contrast, two European studies published in 2023 using Sanger sequencing reported markedly lower frequencies (2.5% and 0%), although one associated the variant with aggressiveness. Aim: Assess the frequency of SF3B1 variant in a national cohort of lactotroph adenomas and evaluate its association with aggressiveness. **Methods:** Brazilian referral centers specializing in pituitary disorders were invited to participate. Patients with surgically treated lactotroph adenoma were included. DNA was extracted from fresh tissue or paraffin blocks to assess the presence of SF3B1 variant using ddPCP. Resistance to dopamine agonists (DA) was defined as failure to normalize prolactin levels and/or achieve tumor shrinkage despite maximally tolerated DA dose. Ethical approval was obtained (CAAE 41010620.0.1001.811). **Results:** Fourteen centers included 151 patients. Median age at surgery was 36 years (range, 13–80), and 51% were female. Median prolactin at diagnosis was 732 ng/mL (range, 47–36,985). Cabergoline was the most used DA, with 72/102 (70.6%) resistant patients. Median cabergoline dose was 3.0 mg/week (range, 0.5–7.0) used for 18 months (range, 0.75–240), achieving preoperative prolactin of 489 (range, 0.2–20,805). Largest tumor diameter before surgery was 3.0 cm (range 0.5–7.8). The SF3B1 variant was identified in only 1/151 (0.7%) cases. This patient was a 52-year-old man, with a 2.1 cm adenoma treated with cabergoline 3 mg/week for 140 months. Despite two surgeries and medical therapy, the tumor progressed to 4.5 cm, causing intracranial hypertension and requiring reoperation at age 76. Preoperative prolactin reached 10,143 ng/mL. Histopathology demonstrated a densely granulated adenoma, with high Ki-67 of 20%–30% and 5 mitoses/10 HPF. No metastases were detected on brain, neuro-axis, thoracic, or abdominal imaging. **Conclusion:** SF3B1 variant was rare in Brazilian lactotroph adenomas, even using a sensitive detection method. However, the single positive case displayed marked aggressiveness, supporting SF3B1 as a potential biomarker of aggressive behavior in prolactinomas.

Keywords: prolactinoma; aggressiveness; SF3B1.

NEUROENDOCRINOLOGY

256 (3310) A RARE CASE OF FUNCTIONING GONADOTROPH ADENOMA PRESENTING WITH TESTOSTERONE HYPERSECRETION

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Case presentation: A 40-year-old man underwent surgical resection of a pituitary macroadenoma (2.0 × 2.5 × 2.0 cm) in 2016 due to optic chiasm compression. Preoperative tests showed elevated levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), and testosterone total (TT) and free (FT), without evidence of exogenous hormone use. LH 31.1 uIU/mL (RR 1.24–1.62), FSH 47.3 uIU/mL (RR 1.27–19.26), TT 1.196 ng/dL (RR 175–781) and FT 364.2 ng/dL (RR 29–112). Additional evaluation: TSH 7.09 uIU/mL (RR 0.4–4.30), free T4 0.33 ng/mL (RR 0.7–1.8), and prolactin (PRL) 38.1 ng/mL (RR 2.10–17.7). Analysis showed positive immunoexpression for FSH and LH, with a proliferative index (Ki-67) of 2%. The patient discontinued follow-up shortly after surgery and developed persistent left visual impairment and fatigue. In 2025, he sought evaluation due to worsening right-eye vision. Pituitary MRI revealed a lesion of 4.0 × 3.8 × 3.0 cm, requiring surgery in March 2026. Laboratory: LH 20.3 uIU/mL (RR 1.5–9.3), FSH 68.3 uIU/mL (RR 1.4–18.1), TT 659 ng/dL (RR 86–788), TSH 7.4 uIU/mL (RR 0.49–5.58), free T4 0.61 ng/mL (RR 0.89–1.53) and PRL 34.6 ng/mL (RR 2.1–17.7). Histopathological exam revealed a chromophobe adenoma with an immunohistochemical profile compatible with gonadotroph adenoma: positive for LH and chromogranin, negative for FSH, and Ki-67 of 0.1%. The patient is currently receiving levothyroxine replacement therapy, with normal freT4, LH, FSH, and TT. **Discussion:** Clinically nonfunctioning pituitary adenomas account for 15%–40% of pituitary tumors, most of which are silent gonadotroph adenomas. Although nearly one-third are associated with elevated serum LH and/or FSH; increases in sex hormones (estradiol or testosterone) are rare. Epidemiological studies are limited, partly due to its rarity. Hypersecretion of gonadotropins by pituitary adenomas rarely leads to a well-defined clinical syndrome, and diagnosis is often related to mass effect symptoms. In this case, elevated testosterone associated with the immunohistochemical profile supports the diagnosis of a functioning gonadotroph adenoma, highlighting the biological complexity of pituitary tumors and the importance of follow-up. **Final comments:** This case illustrates a very rare presentation of a functioning gonadotroph adenoma with biochemical evidence of testosterone hypersecretion. Recognition of this condition is essential for accurate laboratory interpretation, differential diagnosis and long-term clinical follow-up.

Keywords: pituitary; gonadotroph; testosterone.

NEUROENDOCRINOLOGY

257 (3252) ACROMEGALY AND PSYCHONEUROENDOCRINOLOGY: ASSOCIATION BETWEEN IGF-1 CONTROL, DEPRESSION, ANXIETY, AND QUALITY OF LIFE

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Introduction: Acromegaly is a chronic disease characterized by excessive secretion of growth hormone (GH) and consequent elevation of insulin-like growth factor 1 (IGF-1), leading to significant physical, metabolic, and psychosocial changes. From a psychoneuroendocrinology perspective, integrating hormonal markers with validated psychological and quality-of-life measures provides an innovative framework for understanding the multidimensional impact of acromegaly. **Objective:** To evaluate the association between biochemical control of acromegaly and anxiety, depression, psychological distress, and quality of life. **Methods:** This cross-sectional study included 46 patients with acromegaly followed at a tertiary care hospital. Participants were classified according to biochemical control of IGF-1 into controlled (n = 29) and uncontrolled (n = 17) groups. The Hospital Anxiety and Depression Scale (HADS), NCCN Distress Thermometer and Problem List, and AcroQoL questionnaire were administered. Comparative analyses were performed between groups. **Results:** Among the 46 participants, 69.6% were women. Patients with uncontrolled IGF-1 showed worse outcomes across multiple psychoneuroendocrine domains. They exhibited higher global distress scores (8.59 ± 1.28 vs. 5.63 ± 3.01; p = 0.001), higher anxiety scores (11.0 ± 3.4 vs. 8.0 ± 4.8; p = 0.020), and higher depression scores (12.1 ± 3.8 vs. 7.3 ± 4.7; p = 0.001). They also had lower total AcroQoL scores (60 ± 21 vs. 74 ± 18; p = 0.019), including the psychological (19.2 ± 6.3 vs. 24.1 ± 5.5; p = 0.012), appearance (17.5 ± 5.5 vs. 21.9 ± 6.4; p = 0.019), and relationship (21 ± 8 vs. 26 ± 6; p = 0.014) domains, with a trend toward worse physical scores (22 ± 10 vs. 26 ± 8; p = 0.092). Greater frequencies of concerns related to treatment decisions (82% vs. 34%; p = 0.002), transportation (59% vs. 28%; p = 0.036), adequate food (71% vs. 28%; p = 0.005), and feelings of worthlessness (71% vs. 38%; p = 0.032) were also observed. **Conclusion:** Patients with acromegaly and uncontrolled IGF-1 exhibit a psychoneuroendocrine profile characterized by greater psychological distress, higher anxiety and depressive symptoms, poorer quality of life, and a greater burden of practical and emotional concerns. These findings reinforce the importance of integrating endocrine control and psychological care in acromegaly management.

Keywords: acromegaly; psychoneuroendocrinology; quality of life.

NEUROENDOCRINOLOGY

258 (3315) ACROMEGALY AT A SPECIALIZED CENTER IN BAHIA: CLINICAL, BIOCHEMICAL AND THERAPEUTIC ANALYSIS

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Introduction: Acromegaly is a rare disease associated with increased morbidity and mortality when uncontrolled. Few data are available from public referral centers in Northeastern Brazil. **Objective:** To describe the clinical, tumoral, biochemical, and therapeutic profile of patients with acromegaly/gigantism followed in Bahia, Brazil. **Methods:** Retrospective chart review of patients followed at Cedeba. Records outside the study scope or with insufficient data were excluded. Demographic, tumoral, therapeutic, and IGF-1 data were collected. Biochemical control was defined as IGF-1 £ the upper limit of normal (ULN), among patients on medical therapy with current IGF-1. GH was not systematically collected; cure criteria could not be applied. Categorical variables were expressed as absolute and relative frequencies, and numerical variables as mean ± standard deviation. **Results:** We included 98 patients; 65 (66.3%) were women, with mean age 52.2 ± 13.7 years. Among 83 with classifiable baseline tumor assessment, 77 (92.8%) had macroadenomas and 11 (13.3%) giant adenomas. Baseline IGF-1 was 2.9 ± 1.4 x ULN (n = 44). Surgery was recorded in 82/98 (83.7%) and radiotherapy in 8/98 (8.2%). Specific medical therapy was used by 66/98 (67.3%): somatostatin receptor ligand (SRL) alone in 37, SRL plus cabergoline in 25, cabergoline alone in 2, SRL plus pegvisomant in 1, and pegvisomant plus cabergoline in 1. Among 66 patients on medication, 65 had evaluable current IGF-1; 38/65 (58.5%) were controlled and 27/65 (41.5%) remained biochemically active. Among uncontrolled patients, 25 had baseline IGF-1; 15/25 (60.0%) had a ³50% reduction and were partial responders. Of 32 patients without medication, 25 were postoperative with normal IGF-1. Four had elevated IGF-1 without medication: two were awaiting surgery/rescheduling, one was under postoperative follow-up, and one had minimal elevation without evident tumor lesion, under observation. Three were in recent postoperative follow-up without interpretable current IGF-1. **Conclusion:** In this Bahia case series, macroadenomas predominated, with high frequency of prior surgery and specific medical therapy. Among treated patients with evaluable IGF-1, 58.5% were controlled; among evaluable uncontrolled patients, 60.0% were partial responders. Lack of systematic GH and incomplete postoperative data limited cure assessment.
Keywords: acromegaly; epidemiology; somatostatin analogs.

NEUROENDOCRINOLOGY

259 (3164) ACROMEGALY SECONDARY TO A CYSTIC GH-PRODUCING PITUITARY MACROADENOMA: DIAGNOSIS AFTER INITIAL SYMPTOMS OF HYPOGONADISM (A CASE REPORT)

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Clinical case: A 34-year-old man was evaluated by a urologist due to decreased libido and a testosterone level of 167 ng/mL. Following two months of clomiphene treatment, testosterone increased to 640 ng/mL. He sought endocrinology evaluation to investigate the etiology of hypogonadotropic hypogonadism. Physical examination revealed macrognathia, frontal bossing and broadening of the nasal alae. The patient also reported an increase in shoe size. Laboratory investigation revealed IGF-1 of 724 ng/mL, which was elevated for his age and sex. Basal GH was 5.95 ng/mL, and GH after a 75-g oral glucose suppression test remained elevated at 4.54 ng/mL. Pituitary magnetic resonance imaging (MRI) showed a cystic lesion with hypointensity on T1-weighted images, hyperintensity on T2-weighted images, and peripheral contrast enhancement, measuring 1.2 x 0.9 x 0.7 cm, located in the left lateral aspect of the adenohypophysis, with a thin and centered pituitary stalk. The biochemical profile and MRI findings were consistent with acromegaly caused by a GH-secreting pituitary adenoma with cystic degeneration, possibly associated with gonadotroph dysfunction. Transsphenoidal surgery was performed, and six months later the patient had an IGF-1 level of 256 ng/mL. He continues to be monitored postoperatively. **Discussion:** Acromegaly is most often caused by GH-secreting pituitary adenomas and classically manifests with acral enlargement. Diagnosis is based on elevated IGF-1 adjusted for age and sex, and failure of GH suppression after a 75-g oral glucose tolerance test. In this report, pituitary MRI identified a cystic macroadenoma, an uncommon presentation that may be mistaken for nonfunctioning lesions, such as a Rathke cleft cyst, if hormonal investigation is not performed. The initial complaint was decreased libido related to hypogonadotropic hypogonadism, with testosterone levels lower than expected for his age, reinforcing the need for pituitary investigation in young men with similar presentations. **Final remarks:** Acromegaly secondary to a cystic pituitary macroadenoma is an uncommon presentation. Its diagnosis may be masked by cystic degeneration, which can hinder radiological suspicion of a functioning adenoma. This case highlights the risk of a purely symptomatic approach to hypogonadism without adequate investigation. Without resection, progressive tumor growth could have compromised other pituitary cell lineages and, consequently, the patient's quality of life.
Keywords: hypogonadotropic hypogonadism; cystic pituitary macroadenoma; acromegaly.

NEUROENDOCRINOLOGY

260 (3322) AGGRESSIVE RECURRENT SOMATOTROPH PITNET ASSOCIATED WITH LI-FRAUMENI SYNDROME

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Aggressive PitNETs are characterized by progression or recurrence despite multimodal treatment and account for approximately 0.5%–1% of pituitary tumors. A 50-year-old man presented with a 1-year history of progressive bitemporal hemianopia, headache, and cerebellar symptoms. Magnetic resonance imaging demonstrated a 5.2 × 6.1 × 4.4 cm sellar-suprasellar mass with bilateral cavernous sinus invasion and compression of the optic chiasm, hypothalamus, and midbrain. The patient underwent transsphenoidal subtotal resection. Histopathological examination revealed a sparsely granulated somatotroph PitNET, PIT1-positive, focally GH-positive, with a Ki-67 of 5%–10%. Postoperatively, he developed panhypopituitarism. Despite surgical debulking, follow-up imaging after five months demonstrated marked tumor progression, with an approximately tenfold increase in tumor volume compared with the immediate postoperative scan. Given the aggressive clinical course and a family history suggestive of Li-Fraumeni syndrome, whole-exome sequencing was performed, confirming a pathogenic germline TP53 variant. The patient subsequently underwent a second surgical resection, with plans for temozolomide therapy. According to the WHO classification, sparsely granulated somatotroph PitNETs are associated with more aggressive behavior, particularly in the presence of proliferative markers such as Ki-67 > 3%, increased mitotic activity, and p53 positivity. In parallel, Li-Fraumeni syndrome is a hereditary cancer predisposition syndrome caused by pathogenic germline TP53 variants, which impair the tumor suppressor function of p53 and increase susceptibility to multiple malignancies. Although PitNETs are not part of the classic Li-Fraumeni tumor spectrum, a few cases have been reported in the literature, including a corticotroph PitNET associated with Cushing disease and a recurrent prolactinoma in a child. To our knowledge, this is the first reported case of a somatotroph PitNET associated with Li-Fraumeni syndrome. In this context, systemic therapies such as temozolomide should be considered, particularly given the limitations of radiotherapy in patients with Li-Fraumeni syndrome due to the increased risk of radiation-induced secondary malignancies. This case highlights the importance of considering hereditary syndromes in aggressive PitNETs, expands the spectrum of endocrine manifestations associated with Li-Fraumeni syndrome, and reinforces the need for individualized management.

Keywords: aggressive PitNET; Li-Fraumeni syndrome; pituitary macroadenoma.

NEUROENDOCRINOLOGY

261 (2966) ANALYSIS OF FACTORS ASSOCIATED WITH THE DEVELOPMENT OF POSTOPERATIVE DIABETES INSIPIDUS IN SELLAR AND SUPRASELLAR NEUROSURGERY: A TERTIARY CENTER EXPERIENCE

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Introduction: Postoperative diabetes insipidus (DI) is a significant complication following sellar and suprasellar neurosurgeries, impacting patient recovery and morbidity. While incidence varies, identifying clinical and radiological predictors is essential for optimizing perioperative management. This study aims to identify factors associated with DI development in a tertiary hospital cohort. **Methods:** We conducted a retrospective, analytical study of 83 patients who underwent neurosurgical procedures in the sellar region. Clinical, hormonal, and radiological variables (including tumor size, volume, and Knosp scale for cavernous sinus invasion) were analyzed. Statistical significance was set at $p < 0.05$. **Results:** The sample consisted of 83 patients (55.4% female; mean age 54.3 ± 14.5 years). The most frequent diagnoses were Non-Functioning Pituitary Adenomas (54.2%) and Acromegaly (19.3%). The incidence of postoperative DI was 37.3% (n = 31), of which 82.8% were transient and 17.2% permanent. Craniopharyngiomas showed the highest risk (100% incidence, all permanent). Although tumor size and volume were significantly associated with preoperative panhypopituitarism ($p = 0.002$), they did not predict DI development. Notably, the Knosp scale (lateral invasion) showed no correlation with DI ($p = 0.712$) or the severity of postoperative complications ($p = 0.725$). Tumor type showed a trend toward significance ($p = 0.052$). Regarding hormonal axes, ACTH was the most affected (88.6% of patients with postoperative deficiencies). **Conclusion:** DI is a frequent but predominantly transient complication in this cohort. Tumor histology, specifically craniopharyngioma, is a more reliable predictor of permanent DI than radiological markers of lateral invasiveness such as the Knosp scale. These findings suggest that midline surgical manipulation, rather than lateral tumor extension, is the primary driver of neurohypophyseal damage.

Keywords: postoperative diabetes insipidus; sellar surgery; craniopharyngioma.

NEUROENDOCRINOLOGY

262 (3158) ANALYSIS OF IMMUNOHISTOCHEMICAL AND ANATOMOPATHOLOGICAL FINDINGS IN PITUITARY TUMORS: EXPERIENCE FROM AN AMAZONIAN REFERRAL CENTER

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Pituitary neuroendocrine tumors (PitNETs) are heterogeneous neoplasms whose classification increasingly depends on the integration of clinical, anatomopathological and immunohistochemical findings, as reinforced by the 2022 World Health Organization classification. This study aimed to evaluate the correlation between these findings in patients with pituitary tumors treated at a referral center in the Brazilian Amazon. A retrospective observational and analytical study was performed through review of medical records from patients followed over a five-year period at a university referral center. Of 136 initially identified patients, 134 were included after diagnostic confirmation. Demographic, clinical, therapeutic, anatomopathological and immunohistochemical data were descriptively analyzed. Female patients predominated (93/134), with higher frequency between 30 and 39 years of age. Prolactinomas were the most frequent subtype (53/134), followed by clinically nonfunctioning tumors (45/134) and somatotroph tumors (29/134). Macroadenomas and compressive symptoms were highly prevalent, suggesting delayed diagnosis in a significant proportion of cases. Anatomopathological evaluation demonstrated marked morphological heterogeneity. Immunohistochemistry, although available in only a limited subset of patients, enabled hormonal and neuroendocrine characterization through markers such as synaptophysin, chromogranin and FSH, in addition to proliferative assessment using Ki-67. Low Ki-67 indices, predominantly between 1% and 4.9%, suggested mostly indolent biological behavior. However, immunohistochemical evaluation was absent in most cases, including among surgically treated patients. Furthermore, transcription-factor markers currently recommended by the 2022 WHO classification, such as PIT-1, TPIT and SF-1, were rarely available and generally restricted to diagnostically challenging cases. These findings demonstrate a gap between contemporary classification guidelines and their practical implementation in regional healthcare settings. The integration of clinical, anatomopathological and immunohistochemical data proved essential for more accurate PitNET characterization and prognostic assessment, reinforcing the need to expand access to advanced diagnostic tools and standardized pathological evaluation in the Amazon region.

Keywords: PitNETs; immunohistochemistry; anatomopathology.

NEUROENDOCRINOLOGY

263 (3221) ASSOCIATION BETWEEN MULTIPLE ENDOCRINE NEOPLASIA TYPE 1 (MEN1) AND ULCERATIVE COLITIS COINCIDENCE OR IMMUNE DYSREGULATION-MEDIATED CORRELATION?

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Case presentation: A 34-year-old man with ulcerative colitis (UC) since age 23 was referred for genetically confirmed multiple endocrine neoplasia type 1 (MEN1). UC initially presented with severe rectal bleeding, diarrhea, marked weight loss and recurrent hospitalizations, with long-term steroid dependence despite mesalazine, azathioprine, vedolizumab and golimumab. During loss of response to biologic therapy, he developed MEN1 manifestations. Genetic testing identified a heterozygous pathogenic MEN1 variant, c.625C>T, p.(Gln209*). The MEN1 phenotype included primary hyperparathyroidism, treated with subtotal parathyroidectomy and thymectomy, a pituitary adenoma progressing to 1.1 cm, and multifocal grade 2 pancreatic neuroendocrine tumors. He underwent distal pancreatectomy and enucleation of a pancreatic head lesion after hypoglycemic episodes compatible with insulinoma; pathology showed four well-differentiated tumor foci, the largest 2.0 cm, Ki-67 4%, pT2 pN0, with free margins. UC subsequently became refractory, with severe extensive erosive pancolitis, Mayo endoscopic score 3, Mayo clinical score 8, and fecal calprotectin 487 µg/g. Guselkumab was chosen to target the IL-23 pathway while avoiding broader immunosuppression, but the response was partial and transient. He remains steroid-dependent, with steroid-induced diabetes treated with sitagliptin. **Discussion:** To our knowledge, this is the first reported association between genetically confirmed MEN1 and UC. Although coexistence may be incidental, menin haploinsufficiency may plausibly contribute to immune dysregulation. Menin regulates Th17 differentiation and maintenance, CD4+ T-cell homeostasis and cytokine balance, while reduced menin expression in intestinal epithelial cells may enhance NF-kB activity and disturb epithelial proliferation. These pathways overlap with mechanisms implicated in chronic mucosal inflammation and UC, particularly the IL-23/Th17 axis. **Final comments:** This case expands the potential autoimmune spectrum of MEN1 and highlights the challenge of treating immune-mediated intestinal inflammation in a patient with hereditary endocrine neoplasia. MEN1 patients with gastrointestinal inflammatory symptoms may warrant evaluation for inflammatory bowel disease, and cohort studies are needed to assess whether this association is causal or coincidental.

Keywords: MEN1; Th17; ulcerative colitis.

NEUROENDOCRINOLOGY

264 (3175) ASSOCIATION BETWEEN SLEEP DEPRIVATION, CORTISOL DYSREGULATION, AND ITS EFFECTS: A SYSTEMATIC REVIEW

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Sleep deprivation has become a major public health concern due to its increasing prevalence and its negative effects on different physiological systems. Cortisol, the main glucocorticoid regulated by the hypothalamic-pituitary-adrenal axis, plays a central role in the stress response and exhibits a circadian rhythm closely related to the sleep-wake cycle. Alterations in the levels of this hormone may contribute to metabolic and endocrine dysregulations, such as obesity, type 2 diabetes mellitus, and mood disorders (1). In this context, the present study seeks to answer the following guiding question: "What is the association between sleep deprivation, cortisol dysregulation, and its effects?" The research was conducted using the PubMed and BVS databases, employing the descriptors sleep deprivation and cortisol combined through the Boolean operator AND and filtered by title. Publications from the last ten years, written in English, and conducted in humans were considered. Restricted-access publications that prevented complete data analysis, as well as duplicate studies, were excluded. A total of six articles were identified across both platforms, and after applying the eligibility criteria, three studies were selected for analysis. The studies demonstrated that acute sleep deprivation significantly influences the regulation of the hypothalamic-pituitary-adrenal axis, particularly cortisol secretion. Chen *et al.* identified increased cortisol levels, especially during the afternoon period, suggesting alterations in circadian rhythm. Similarly, Messa *et al.* observed that total sleep deprivation may modify the cortisol response to acute stress, although the findings remain inconsistent. In contrast, an experimental study involving young women did not identify significant changes in cortisol levels after 92 hours of sleep deprivation, despite important metabolic alterations. In conclusion, acute sleep deprivation shows a relevant association with cortisol dysregulation, mainly through alterations in circadian rhythm and the physiological stress response. Although some studies did not identify significant hormonal changes, most evidence points to increased cortisol levels following periods of sleep restriction or total sleep deprivation. However, the methodological heterogeneity among studies still limits the establishment of a definitive physiological pattern, highlighting the need for further research to better understand this relationship.

Keywords: sleep; deprivation; cortisol.

NEUROENDOCRINOLOGY

265 (3096) AUTOIMMUNE HYPOPHYSITIS PRESENTING AS A SELLAR MASS IN A PATIENT WITH POLYGLANDULAR AUTOIMMUNE SYNDROME

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Case presentation: A 42-year-old female patient with a history of Graves' disease (GD) treated with radioiodine therapy during childhood, systemic lupus erythematosus (SLE), Sjögren's syndrome (SS), endometriosis, migraine, and DiGeorge syndrome was referred for endocrinological evaluation due to progressive headache associated with pituitary abnormalities on imaging studies. She had a history of headaches since 2017, initially related to the menstrual period and partially controlled with analgesics. In 2025, she developed significant worsening, with daily left-sided compressive headaches associated with nausea, vomiting, and vertigo. Pituitary MRI revealed diffuse enlargement of the gland, superior convexity with suprasellar extension contacting the optic chiasm, thickening of the pituitary stalk, and a 0.5 cm nodular lesion in the right adenohypophysis. Laboratory evaluation showed ACTH levels of 25 pg/mL, morning cortisol of 5.9 mcg/dL, prolactin of 49 ng/mL, and persistent elevation of insulin-like growth factor 1 (IGF-1), reaching 415 ng/mL (reference range 112–282). Oral glucose tolerance testing (OGTT) demonstrated adequate growth hormone (GH) suppression, excluding active acromegaly. Gonadotropin levels were preserved. Given the suspicion of autoimmune hypophysitis in the context of autoimmune polyglandular syndrome, she underwent two cycles of pulse therapy with methylprednisolone 500 mg/day for three days. Significant improvement in headache symptoms and slight reduction in pituitary enlargement was observed on follow-up imaging. **Discussion:** Autoimmune hypophysitis is a rare inflammatory disorder of the pituitary gland, frequently associated with other autoimmune diseases. Headache, visual disturbances, and hormonal dysfunction are common manifestations. In this case, the imaging findings of homogeneous pituitary enlargement and pituitary stalk thickening favored the diagnosis of lymphocytic hypophysitis rather than pituitary adenoma. The association with SLE, SS and GD further supports an autoimmune etiology. Clinical and radiological improvement following corticosteroid therapy reinforced the diagnostic hypothesis. **Final comments:** This case highlights the importance of considering autoimmune hypophysitis in the differential diagnosis of sellar lesions, especially in patients with systemic autoimmune diseases. Early recognition allowed appropriate immunosuppressive treatment, resulting in clinical improvement and radiological stabilization.

Keywords: autoimmune hypophysitis; systemic lupus erythematosus; pituitary diseases.

NEUROENDOCRINOLOGY

266 (3462) BEYOND THE CLASSIC TRIAD: CLINICAL HETEROGENEITY OF POU1F1 MUTATIONS IN COMBINED PITUITARY HORMONE DEFICIENCY A SYSTEMATIC REVIEW

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Introduction: Mutations in the POU1F1 gene represent an important cause of combined pituitary hormone deficiency (CPHD). POU1F1 encodes a pituitary-specific transcription factor essential for somatotroph, lactotroph, and thyrotroph differentiation, regulating growth hormone (GH), prolactin (PRL), and thyroid-stimulating hormone (TSH). Although classically linked to early and severe presentation, POU1F1 mutations exhibit marked clinical heterogeneity, frequently delaying diagnosis. **Objective:** Analyze the clinical variability of POU1F1 mutations in CPHD, emphasizing hormonal deficiency chronology and implications for early recognition and differential diagnosis. **Methods:** Systematic review conducted following the PICO strategy, in PubMed, Cochrane, and EMBASE. Human cohort studies, randomized clinical trials and case reports from the last 20 years were included. The search focused on CPHD with POU1F1 mutations compared with other etiologies, with phenotypic variability as the primary outcome. Exclusion criteria: animal studies, narrative reviews without original clinical data, and studies lacking molecular confirmation. Of 125 screened articles, 31 were included. **Results:** The classic triad of GH, PRL, and TSH deficiencies with preserved corticotroph and gonadotroph function was predominant, but significant variability was observed. TSH deficiency was frequently the first manifestation within the first three months of life, though late-onset cases were documented up to 25 years of age. Isolated GH deficiency progressing to full CPHD supports the progressive nature of the phenotype. Anterior pituitary hypoplasia showed a similar course, with normal MRI in early stages — a finding that should not exclude the genetic etiology. Undetectable or marked low PRL emerged as a useful diagnostic marker. Compared with PROP1 mutations, the main differential diagnosis, POU1F1 mutations are distinguished by preserved corticotroph and gonadotroph axes and absence of extrapituitary malformations, ectopic posterior pituitary, or stalk interruption. **Conclusion:** POU1F1 mutations determine a restricted phenotype; however, clinical and temporal heterogeneity represents the main diagnostic challenge. Isolated GH deficiency with normal MRI should not exclude POU1F1 mutations, particularly when associated with central hypothyroidism or undetectable PRL. Early recognition, genetic investigation, and differential diagnosis are essential for adequate treatment and optimizing prognosis.
Keywords: combined pituitary hormone deficiency; POU1F1; phenotypic variability.

NEUROENDOCRINOLOGY

267 (3533) BILATERAL CEREBELLAR ISCHEMIC STROKE DUE TO VERTEBRAL ARTERY DISSECTION IN A YOUNG ADULT: AN INITIAL MANIFESTATION OF CUSHING'S DISEASE

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Case report: A 33-year-old man, without comorbidities, developed a persistent headache associated with symptoms of central vestibular syndrome and arterial hypertension. Radiologic studies demonstrated subacute bilateral cerebellar ischemia secondary to bilateral vertebral artery dissection, with 80% stenosis on the left and 30% on the right. Laboratory tests revealed polycythemia, neutrophilic leukocytosis, proteinuria, and hematuria. Due to refractory hypertension, an investigation for secondary causes was initiated. Differential diagnoses such as hiperaldosteronism, thrombophilias, acromegaly, prolactinoma, and pheochromocytoma were excluded. The paciente increased 10-kg centripetal weight, violaceous striae, spontaneous bruising, prediabetes, dyslipidemia, and hypogonadotropic hypogonadism. The 24-h urinary cortisol and late-night salivary cortisol, were two times the upper limit of normal, associated with no-suppression to dexamethasone test and non-suppressed ACTH levels (43.6 pg/mL), confirmed the diagnosis of ACTH-dependent Cushing's syndrome. The pituitary MRI showed no abnormalities. To differentiate between Cushing's disease and ectopic ACTH syndrome, inferior petrosal sinus sampling was performed, confirming a pituitary source and suggesting right-sided lateralization. The patient underwent transsphenoidal hypophysectomy, and histopathological analysis revealed no adenoma on hematoxylin-eosin staining, immunohistochemistry, or reticulin staining. As aforementioned, the findings are consistent with corticotroph cell hyperplasia. After the surgery, remission was achieved with normalization of blood pressure and glycemic control, allowing deprescribing of medications. The patient developed, as expected, secondary adrenal insufficiency and replacement therapy was initiated. **Discussion:** This case illustrates an atypical presentation of Cushing's disease in a young patient, with initial manifestation as a rare and severe cerebellar stroke caused by bilateral vertebral artery dissection. The metabolic complications resulted from the systemic effects of hypercortisolism, including vascular fragility and arterial hypertension, increasing the risk of vascular events. **Conclusion:** The diagnostic challenge by this atypical clinical presentation highlights the importance of pursuing etiological investigation in suspected secondary hypertension and thoroughly excluding differential diagnoses. Long-term follow-up is essential due to the risk of recurrence.
Keywords: Cushing syndrome; hypertension; corticotrophs.

NEUROENDOCRINOLOGY

268 (3415) CASE REPORT: MACROPROLACTINOMA DIAGNOSED AFTER DECADES OF FOLLOW-UP FOR CONGENITAL PANHYPOPITUITARISM

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A male patient with a diagnosis of congenital panhypopituitarism, had a background of central hypothyroidism, secondary adrenal insufficiency and growth hormone (GH) deficiency since childhood. He was treated accordingly with levothyroxine, prednisone, somatotropin (from ages 8 to 17, achieving adequate height) and testosterone (which was necessary for puberty inducement). At the beginning of our follow-up in 1997 at age 31, with persistent asthenia, muscle weakness and low serum levels of insulin-like growth factor 1 (IGF-1), somatropin therapy was restarted. Over the years, the patient had poor adherence to outpatient follow-up and the prescribed therapy. In 2010, magnetic resonance imaging (MRI) of the sellar region revealed an atrophic pituitary gland without expansive lesions, consistent with the patient's diagnosis. The following year, the therapy was combined with exogenous gonadotropins for an attempt at fertility induction. Despite treatment, semen analysis later on maintained azoospermia, hence the decision was made to suspend the exogenous gonadotropins. In 2017, prolactin levels increased to 79 ng/mL after previously normal results. The finding was attributed to presumed stalk effect, without compressive symptoms. After loss to follow-up, he was hospitalized in 2025 for hypertensive emergency, when cranial computed tomography identified a pituitary lesion. A new MRI demonstrated a 2.2 cm macroadenoma with suprasellar extension, optic chiasm compression, and contact with the left internal carotid artery. Hyperprolactinemia of 2,868 ng/mL confirmed invasive macroprolactinoma and cabergoline therapy was initiated with progressive dose escalation. This case describes a rare incidence of a macroadenoma development in a patient with previous congenital panhypopituitarism. Although a mild hyperprolactinemia occurs in approximately 6.5% of patients with empty sella syndrome, that is usually due to stalk effect, and not true prolactinomas, which originates from the neoplastic transformation of lactotrophic cells located in the adenohypophysis. Since these patients typically have few functional pituitary tissue, it is considered extremely improbable for a clonal proliferation to take place. Although a cause-effect cannot be established, this report highlights the importance of active clinical surveillance in patients with pituitary disorders, in order to promptly identify the need for laboratory and imaging investigations when indicated.

Keywords: panhypopituitarism; macroprolactinoma; growth hormone deficiency.

NEUROENDOCRINOLOGY

269 (3549) CASE REPORT: POSTOPERATIVE COMPLICATIONS IN PITUITARY MACROADENOMA RESECTION

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Case presentation: A 55-year-old female patient with a history of a pituitary macroadenoma underwent transsphenoidal surgical resection. Prior to surgery, she presented with long-standing visual scotomas and progressive visual field loss starting 11 months earlier. Preoperative neuroimaging confirmed an expansive lesion compatible with a pituitary adenoma. Postoperatively, she developed severe polyuria and polydipsia, raising suspicion of central diabetes insipidus. During a prior hospitalization, she was treated with intranasal desmopressin (DDAVP), which reduced urine output but caused an acute 10 mEq/L drop in serum sodium, leading to immediate discontinuation. She was readmitted due to persistent polyuria (~4 L/24 h) and polydipsia, accompanied by clear, hyaline nasal discharge, requiring co-management by endocrinology and neurosurgery teams. During her course, she developed a continuous holocranic headache (6.5/10 on the visual analog scale), without focal neurological deficits, nausea, or paresthesias. Concomitantly, she manifested fever, chills, and mild odynophagia. General laboratory tests, serum sodium, and a brain and sella turcica CT scan were requested to investigate acute complications. **Discussion:** This case illustrates severe complications following transsphenoidal neurosurgery. While persistent polyuria and polydipsia confirm central diabetes insipidus, the previous hyponatremia induced by DDAVP alerts to a complex triphasic response (fluctuation between DI and SIADH due to uncontrolled ADH release), demanding fluid management strictly on an as-needed basis and rigorous sodium monitoring. Additionally, the hyaline rhinorrhea indicates a cerebrospinal fluid (CSF) leak. This dural breach, combined with headache, fever, and chills, strongly points to secondary bacterial meningitis. Therefore, after a CT scan rules out mass effect contraindications, immediate management requires a lumbar puncture and the urgent initiation of empirical broad-spectrum antibiotics with excellent central nervous system penetration. **Final comments:** Postoperative diabetes insipidus is complex and significantly reduces quality of life. To mitigate morbidities, management should take place in centers of excellence with high surgical volume. Because it is a manageable condition, rigorous fluid monitoring and multiprofessional care remain indispensable pillars for early diagnosis and a favorable prognosis.

Keywords: pituitary macroadenoma; SIADH; central diabetes insipidus.

NEUROENDOCRINOLOGY

270 (3160) CLINICAL AND EPIDEMIOLOGICAL CHARACTERISTICS OF ADULT PATIENTS WITH GIANT SELLAR TUMORS FOLLOWED AT A REFERRAL CENTER IN SALVADOR, BAHIA

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Introduction: Giant sellar tumors, defined as lesions measuring ≥4 cm, represent a major challenge in neuroendocrinology and neurosurgery due to compressive effects on critical neurovascular structures, associated hypopituitarism, and surgical complexity. Despite heterogeneous etiologies, these lesions frequently share severe clinical manifestations and substantial morbidity. **Objective:** To describe the clinical, hormonal, and epidemiological characteristics of adult patients with giant sellar tumors followed at a specialized neuroendocrinology outpatient clinic. **Methods:** This retrospective and descriptive study reviewed medical records of adult patients with giant sellar tumors treated at a referral hospital in Salvador, Bahia. Demographic, clinical, laboratory, hormonal, and imaging variables were collected. Data were analyzed using descriptive statistics in Jamovi software. Categorical variables were expressed as absolute and relative frequencies, while numerical variables were presented as mean ± standard deviation or median and interquartile range, according to data distribution. **Results:** Among 68 patients, 51.5% were male, with mean age of 44 ± 16 years, and 54.4% were from countryside regions of Bahia. The most frequent etiologies were non-functioning pituitary adenomas (38.2%), macroprolactinomas (27.9%), and craniopharyngiomas (17.6%). Mean tumor diameter was 5.0 ± 0.9 cm. The most common presenting manifestations were visual impairment (64.7%), headache (55.9%), intracranial hypertension (19.1%), and amenorrhea (10.3%). Gonadotropic deficiency was identified in 86.8% of patients, central hypothyroidism in 82.4%, and secondary adrenal insufficiency in 57.4%; 76% presented deficiencies in at least two hormonal axes. All patients managed without surgery had prolactinomas. Radiotherapy was performed in only 4.4% of patients. **Conclusion:** Giant sellar tumors are associated with high morbidity, marked hormonal dysfunction, and substantial healthcare burden. The high frequency of visual impairment and multiple pituitary deficiencies observed in this cohort reinforces the importance of earlier recognition and timely referral to specialized centers. Additionally, the findings highlight that cabergoline remains the primary therapeutic approach for giant prolactinomas, even in patients with extensive tumor burden.
Keywords: giant sellar tumors; neuroendocrinology; pituitary adenomas.

NEUROENDOCRINOLOGY

271 (3033) CLINICAL ASPECTS IN A FAMILY DIAGNOSED WITH MEN 1 SYNDROME

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Introduction: Multiple endocrine neoplasia type 1 (MEN1) is a rare autosomal dominant disorder caused by high penetrance pathogenic variants (PV) in the MEN1. The c.1348T>G PV has been described in only three families, all from Brazil. Clinical, familial or molecular criteria establish diagnosis and current sequencing techniques identify germline PVs in 70%–90% of familial cases. **Objective:** To describe a family with two index cases from different centers carrying the c.1348T>G MEN1 PV, including one patient with rare ectopic acromegaly. **Methods:** Clinical, biochemical, pathological, and radiological data were collected from relatives. Screening was offered to individuals willing to undergo evaluation and exon 9 of MEN1 was sequenced by Sanger method. **Results:** Both index cases presented Primary Hyperparathyroidism (PHPT); one was associated with ectopic acromegaly co-secreting GHRH and insulin, and the other with macroprolactinoma. The c.1348T>G MEN1 PV was identified in both patients, and relatives were recruited for evaluation. Among 15 clinically assessed individuals, seven were diagnosed with MEN1 with two harboring the PV, while molecular testing was not yet performed in the remaining five. Median age at diagnosis of the first tumor was 31 years (12–46). PHPT was the most frequent initial manifestation (5/7), while the remaining patients presented with PA. A second tumor developed in four patients at a median age of 42 years (29–46), including one pancreatic NET, one gastrinoma, one PA, and one case of PHPT. None of the siblings had ectopic acromegaly. One patient was diagnosed with a nonfunctioning macroadenoma at age of 12, before the recommended screening age. **Conclusion:** MEN1 shows a broad clinical spectrum, with different tumors arising within the same kindred. The age at first tumor associated with this PV was lower than previously reported, including a pituitary tumor diagnosed before age 15. Familial screening enabled earlier diagnosis of MEN1-associated tumors.
Keywords: multiple endocrine neoplasia; MEN1 gene; familial screening.

NEUROENDOCRINOLOGY

272 (3092) CLINICAL PROFILE AND OUTCOMES OF SURGICALLY TREATED PROLACTINOMAS

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Introduction: Dopamine agonists (DA) are the first-line treatment for prolactinomas, while surgery is generally reserved for resistance, intolerance, or selected cases with compressive symptoms. Real-world data on surgically treated prolactinomas remain limited. **Methods and results:** We retrospectively evaluated patients with prolactinomas who underwent pituitary surgery at a tertiary center between 1993 and 2025. Eighteen patients were included; 13 were male (72%). Median age at diagnosis and surgery was 33 and 34 years, respectively. All tumors were macroprolactinomas, with a mean maximal diameter of 48 mm (17–76), consistent with invasive disease. Mean prolactin (PRL) was 2,389 ng/mL at diagnosis and remained markedly elevated before surgery (2,011 ng/mL). Eight patients (44%) underwent surgery after DA therapy, mainly cabergoline (n = 6; 1–7 mg/week), due to resistance (n = 4), intolerance (n = 3), or cerebrospinal fluid fistula requiring debulking (n = 1). Ten patients (56%) had surgery as first-line therapy because of apoplexy (n = 2), cystic tumor (n = 1), mass effect with neurologic symptoms (n = 5), or surgeon preference (n = 2). Immunohistochemistry confirmed PRL expression in all cases; two tumors showed LH/FSH co-expression, suggesting plurihormonal PitNETs. Ki-67 was available in 15 patients, with a mean index of 4.2%. Most tumors (66%) had low proliferative activity (Ki-67 < 3%), whereas two showed markedly elevated Ki-67 (>20%), consistent with more aggressive behavior. After surgery, only one patient achieved complete biochemical remission without adjuvant therapy. Among 14 patients treated postoperatively with DA, two achieved remission, six maintained PRL normalization with low-dose cabergoline, two required radiotherapy, and four had persistent hyperprolactinemia despite cabergoline. Hypopituitarism occurred in 13 patients and diabetes insipidus in three, one permanent. No perioperative mortality was observed. **Conclusion:** In this long-term real-world cohort, surgically treated prolactinomas represented a heterogeneous and challenging subgroup, marked by male predominance, large invasive tumors, high PRL levels, and frequent DA resistance. Surgery improved biochemical control but was rarely curative without adjuvant therapy and was associated with substantial endocrine morbidity, reinforcing the need for careful patient selection and multidisciplinary management.

Keywords: prolactinoma; pituitary surgery; dopamine agonist resistance.

NEUROENDOCRINOLOGY

273 (3329) CLINICAL, HORMONAL, AND RADIOLOGICAL PROFILE OF PATIENTS WITH GIANT MACROPROLACTINOMAS AT A REFERRAL CENTER IN BAHIA

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Introduction: Giant macroprolactinomas are rare pituitary tumors with significant clinical manifestations secondary to mass effects and hormonal alterations. This study aimed to describe the clinical, hormonal, and radiological profile of patients diagnosed with giant macroprolactinomas in a reference center in Bahia. This is a retrospective study based on the analysis of medical records, in which sociodemographic and anthropometric variables, clinical manifestations, hormonal parameters, radiological characteristics, treatment, and prognosis were evaluated. **Results:** The sample consisted of 17 patients, with a similar distribution between sexes and a comparable median age at diagnosis in men and women. Clinical manifestations were predominantly related to tumor mass effect, with emphasis on visual disturbances and headache, as well as sex-specific symptoms, such as amenorrhea in women and sexual dysfunction in men. A high frequency of hypopituitarism was observed, especially involving the thyroid and gonadotropic axes, in addition to wide variation in serum prolactin levels. Radiological findings demonstrated large tumors with invasion of adjacent structures, with greater extent and impact in males. Although some classical aspects described in the literature were observed, such as the predominance of extrasellar tumor invasion, the results showed divergences from the international literature, particularly regarding sex distribution and the similar age at diagnosis between sexes. The data suggest a broader spectrum of clinical manifestations of these tumors, highlighting the importance of a more comprehensive diagnostic approach. **Conclusion:** The study of giant macroprolactinomas is rare, particularly in the national literature, and given that this diagnosis is associated with high morbidity, the present study became essential for characterizing this population, expanding the knowledge of giant macroprolactinomas in the national setting, and highlighting the importance of a more comprehensive and sensitive clinical approach to the different presentations of the disease.

Keywords: prolactinoma; hyperprolactinemia; pituitary neoplasm.

NEUROENDOCRINOLOGY

274 (3295) CLINICAL, RADIOLOGICAL, AND HISTOPATHOLOGICAL CHARACTERIZATION OF PRIMARY AND SECONDARY HYPOPHYSITIS: A SINGLE-CENTER CASE SERIES

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Introduction: Hypophysitis is a rare disease that typically presents with mass effects and/or hormonal changes. Hypophysitis can be primary or secondary. The definitive diagnosis is usually challenging and requires extensive investigation to identify or rule out systemic diseases that can affect the pituitary gland. In selected cases, a pituitary biopsy should be performed. **Objective:** To evaluate the characteristics of the clinical picture, sellar imaging, treatment, and clinical follow-up of patients with hypophysitis followed in a single tertiary center. **Methods:** A single-center retrospective study was performed. Medical records were evaluated to analyze clinical, laboratorial, imaging exams results, therapies, and follow-up. Patients were diagnosed with primary (PrHypo) or secondary (SecHypo) hypophysitis. **Results:** 31 individuals were included: median age at diagnosis was 38 years old (13–64), and 23 (74%) were female. 49% had a personal history of an autoimmune or rheumatologic disease. The most common symptoms were polyuria and/or polydipsia (51.6%), headache (42%), and cranial nerve palsy (32%). Among the 21 women with available data, 12 (57%) presented with oligo/amenorrhea. Laboratory evaluation showed hyperprolactinemia in 18 (58%), confirmed hypogonadotrophic hypogonadism in 16 (51%), hypocortisolism in 12 (38%), central hypothyroidism in 9 (29%), and growth hormone deficiency in 9 (29%). 13 patients (42%) presented AVP deficiency. Sellar MRI depicted pituitary stalk thickening in 60% of cases, loss of the posterior pituitary bright spot signal in 35%, and optic chiasm compression in 22.6%. In our cohort, 17 cases (54.8%) had a confirmed diagnosis (6 PrHypo and 11 SecHypo). The most common cause of SecHypo was sarcoidosis (33%). Six cases (20%) were submitted to neurosurgery: 3 for pituitary biopsy and 3 for mass effect symptoms. Glucocorticoid therapy at immunosuppressive doses was used in 53% of patients, and other immunosuppressive agents in 48% of patients. **Conclusion:** Several systemic diseases can be associated with hypophysitis, and an extended evaluation is necessary for the correct diagnosis. In suspicious cases of PrHypo or confirmed SecHypo with mass-effect symptoms, corticotherapy was indicated, with a good response of mass-effect symptoms.
Keywords: hypophysitis; AVP deficiency; hypopituitarism.

NEUROENDOCRINOLOGY

275 (3316) CLINICOPATHOLOGICAL CHARACTERISTICS AND THERAPEUTIC OUTCOMES OF THYROTROPINOMAS: A SINGLE TERTIARY REFERRAL CENTER RETROSPECTIVE STUDY

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Introduction: Thyrotropinomas are the rarest functioning pituitary adenoma (PA) subtype, accounting for < 1% of all PA. Due to its rarity there is a lack of data regarding its clinical presentation and treatment outcomes. **Objective:** Describe the clinical, laboratory, histopathological and radiological profile and therapeutic outcomes of thyrotropinoma patients followed at a single tertiary center. **Methods:** Retrospective study including consecutive cases diagnosed between 2013–2026. Inclusion criteria: central thyrotoxicosis [elevated or inappropriately normal TSH with elevated free T4 (fT4) and T3], PA on magnetic resonance imaging (MRI), and/or postoperative immunohistochemical TSH confirmation. Data collected: clinical-demographic variables, biochemical profile, radiological, histopathological data and therapeutic outcomes. **Results:** Eleven patients (mean age 43.9 years; 54% male) were included, with median TSH: 13.8 mIU/L (1.7–53.3) and fT4: 2.7 ng/dL (2.0–3.5) at diagnosis. All were macroadenomas (median maximal diameter 3.1 cm; 1.7–4.5). Clinical presentation included headache (64%), visual deficit (54%), tachycardia (54%), goiter (45%), and weight loss (45%). Hormonal co-secretion was observed in 36% (TSH+GH 36%; one TSH+GH+prolactin). Immunohistochemistry was positive for TSH+GH+prolactin in 63%, TSH+GH in 18% and TSH alone in 18%. Median Ki-67 was 1.3% (0.6 - 15). In 4 patients somatostatin receptor type 2 (SST2) expression was evaluated and was high (intensity reactivity score of 12 in all). Inappropriate levothyroxine was prescribed to 27% prior to diagnosis and median diagnostic delay was 12 months. All patients underwent surgery, with remission obtained in 54% and new pituitary hormone deficiency observed in 18%. In 4 patients first-generation somatostatin receptor ligands (SRL) was initiated, and outcome of treatment was available in 3, all of whom achieved normalization of fT4. **Conclusion:** Thyrotropinomas are usually macroadenomas at diagnosis, frequently presenting with compressive symptom suggesting diagnostic delay. Surgical cure rate is probably impacted by large tumor size, but they present a high expression of SST2 that is compatible with a high efficacy of SRL in these patients. Misdiagnosis frequency — 27% inappropriately received levothyroxine — reinforces the lack of knowledge about this type of PA and the importance of description of this case series.
Keywords: thyrotropinoma; TSH-secreting pituitary adenoma; somatostatin receptor ligands.

NEUROENDOCRINOLOGY

276 (3294) COMBINED TRANSCRANIAL AND ENDOSCOPIC ENDONASAL APPROACHES FOR PITUITARY ADENOMAS: A SINGLE-CENTER EXPERIENCE

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Introduction: Giant pituitary adenomas (PA) with suprasellar and multidirectional extension remain challenging lesions for isolated endoscopic or transcranial approaches. Simultaneous combined transcranial and endoscopic endonasal surgery has emerged as an alternative strategy for selected complex tumors. **Objective:** To describe a single-center experience with simultaneous combined transcranial and endoscopic endonasal surgery performed by the same neurosurgical team. **Methods:** A retrospective cohort study was conducted of patients who underwent surgery for a PA from 2012026. Following multidisciplinary team discussion, PA demonstrating significant suprasellar extension, multilobulated morphology, and neurovascular involvement were selected for simultaneous combined surgery. Clinical, radiological, surgical, and follow-up data were collected. Extent of resection was determined by comparing preoperative and postoperative tumor volumes. **Results:** 13 patients were included. Median age was 52 years (range, 25–58), 8 (61.5%) patients being male. Most PA were non-functioning (11; 84.6%), while one (7.7%) was a somatotropinoma and one (7.7%) a prolactinoma. Median tumor volume was 126.5 cm^3 (range, 56–282), with all tumors being giant (>4 cm). Mean extent of resection was 79.3% demonstrating effective tumor debulking in highly complex lesions. Residual tumor remained present in 84.6% of patients, but at a smaller size [median volume of 31.0 cm^3 (range 2-140)] reflecting a strategy prioritizing safe neurovascular decompression over aggressive gross-total resection. Visual improvement occurred in 30.8% of patients, while postoperative visual deterioration was observed in 15.4%. New endocrine dysfunction and AVP deficiency developed in 61.5% of patients in which half was temporary. Hydrocephalus occurred in 46.2% of cases and meningitis in one (7.7%) patient. Death related to surgery (<30 days) was seen in one (7.7%) patient that experienced a fatal complication following myocardial infarction and intraparenchymal hemorrhage. No postoperative cerebrospinal fluid leak was observed. **Conclusion:** Simultaneous combined transcranial and endoscopic endonasal approaches may provide safe and effective management for selected giant PA with multidirectional extension. The strategy enables substantial tumor debulking and neurovascular decompression while minimizing cerebrospinal fluid leak, supporting individualized maximal safe resection rather than aggressive radical surgery.

Keywords: pituitary adenoma; giant adenoma; surgery.

NEUROENDOCRINOLOGY

277 (3034) CONCOMITANT USE OF PALTUSOTINE AND LEVOTHYROXINE IN PATIENTS WITH ACROMEGALY: A POST HOC ANALYSIS OF PHASE 3 CLINICAL TRIAL DATA

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Introduction: Paltusotine, a nonpeptide, selective somatostatin 2 receptor agonist, is approved in the United States and Europe as a once-daily oral treatment for adults with acromegaly. **Objective:** This *post hoc* analysis evaluated the effects of concomitant use of paltusotine and levothyroxine in patients with acromegaly. **Methods:** Data were analyzed from two phase 3 clinical trials. In PATHFNDR-1, patients with biochemically controlled acromegaly (IGF-I ≤1.0× ULN) on an injected depot somatostatin receptor ligand were switched to paltusotine or placebo. In PATHFNDR-2, patients with biochemically uncontrolled acromegaly who were medically untreated at randomization received paltusotine or placebo. No specific guidance was given on the timing of administration of levothyroxine relative to paltusotine (both should be taken on an empty stomach ~1 hour before eating). Thyroid function (thyroid-stimulating hormone [TSH], free thyroxine [FT4]) was assessed at study baseline and at the end of randomized treatment (EOT; PATHFNDR-1: 36 weeks; PATHFNDR-2: 24 weeks). **Results:** Across the two trials, 43 patients were receiving levothyroxine at study baseline and had postbaseline assessment of thyroid hormones (paltusotine, n = 25; placebo, n = 18). At baseline, mean ± SD TSH was 1.0 ± 2.04 mIU/L in the paltusotine group and 1.3 ± 1.69 mIU/L in the placebo group; mean ± SD FT4 was 1.3 ± 0.28 ng/dL and 1.3 ± 0.19 ng/dL, respectively. TSH and FT4 levels remained stable at EOT in both the paltusotine and placebo groups: mean ± SD change from baseline in TSH of 0.3 ± 2.50 mIU/L and 1.1 ± 2.65 mIU/L, respectively, and in FT4 of –0.1 ± 0.29 ng/dL and –0.1 ± 0.24 ng/dL, respectively. In paltusotine-treated patients in PATHFNDR-1 (switch study of patients with biochemically controlled acromegaly), mean ± SD change in IGF-I at EOT was comparable in patients receiving (0.08 ± 0.154 × ULN; n = 9) and not receiving (–0.01 ± 0.142 × ULN; n = 19) levothyroxine. **Conclusion:** Among patients on levothyroxine, thyroid function, as measured by mean TSH and FT4 levels, remained stable through the end of the randomized phase in both the paltusotine and placebo groups. These findings suggest that concomitant use of levothyroxine and paltusotine does not appear to affect thyroid function or the IGF-I response to paltusotine. Periodic assessment of thyroid function is recommended in patients taking paltusotine.

Keywords: acromegaly; levothyroxine; paltusotine.

NEUROENDOCRINOLOGY

278 (2981) CORRELATION BETWEEN MORNING BASAL CORTISOL LEVELS AND INSULIN TOLERANCE TEST RESULTS IN PATIENTS WITH SUSPECTED ADRENAL INSUFFICIENCY

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The morning serum cortisol (MSC) measurement is the initial test in the investigation of adrenal insufficiency. However, intermediate values often require dynamic testing for diagnostic confirmation, such as the insulin tolerance test, which presents operational limitations, contraindications, and risk of hypoglycemia. With the improvement of modern laboratory assays, the cutoff values traditionally used for basal cortisol and dynamic testing have been revised. This retrospective study evaluated the accuracy of MSC in predicting the response to the insulin tolerance test and proposed cutoff values with high specificity for confirmation and high sensitivity for exclusion of adrenal insufficiency (AI), thereby reducing the need for additional testing. A total of 59 patients with clinical suspicion of adrenal insufficiency who underwent an insulin tolerance test at a tertiary center between 2015 and 2025 were included. Cortisol measurements were performed using the Elecsys® Cortisol II assay (Roche), and an adequate response to the test was defined as a peak cortisol ≥ 15 µg/dL. Thirty-nine patients (66.1%) showed an adequate response (peak ≥ 15 µg/dL). Basal cortisol levels were significantly lower in individuals with an inadequate response to the test (median 4.8 µg/dL) compared with those with an adequate response (median 9.6 µg/dL; $p < 0.0001$). Receiver operating characteristic (ROC) curve analysis demonstrated an area under the curve of 0.86, indicating good discriminatory capacity. Basal cortisol values < 3.89–4.0 µg/dL showed high specificity for confirming adrenal insufficiency, whereas values ≥ 9.55–10.86 µg/dL showed high sensitivity for excluding the diagnosis. The combined application of these cutoff values could potentially avoid approximately 42.3% of dynamic tests performed. In conclusion, MSC demonstrated good accuracy in predicting the response to the insulin tolerance test. In an appropriate clinical context and when modern assays are used, extreme basal cortisol values may reliably confirm or exclude adrenal insufficiency, reducing the need for dynamic testing.

Keywords: adrenal insufficiency; morning serum cortisol; insulin tolerance test.

NEUROENDOCRINOLOGY

279 (3582) CYSTIC PITUICYTOMA MIMICKING A PITUITARY MACROADENOMA

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Presentation of case: A 42-year-old male presented with a six-month history of headache. At subsequent consultations, the patient developed progressive blurred vision, left eye pain, reduced libido, and erectile dysfunction. A pituitary MRI showed an expansive mass with T2 hyperintensity and T1 hypointensity, regular contours, and well-defined margins, located within the pituitary gland topography, causing depression of the sellar floor and compressing the optic chiasm. After contrast infusion, a ring-like enhancement of the lesion was observed, and the mass measured approximately 2.0 x 1.4 x 1.4 cm. Visual field testing demonstrated a left superior temporal hemianopia. Preoperative laboratory tests showed no hormone production deficits. The most likely hypothesis was a cystic macroadenoma. The neurosurgery team performed a pituitary microsurgery. Histopathological examination showed a hypervascular blue cell neoplasm, with an absence of necrosis or mitosis. In the immunohistochemistry report, the neoplastic cells showed nuclear expression of TTF1 and S100, which was consistent with the diagnosis of pituicytoma. Postoperatively, visual symptoms and headache resolved completely; however, the patient developed central diabetes insipidus and permanent panhypopituitarism, requiring glucocorticoid, levothyroxine, desmopressin, and testosterone replacement. **Discussion:** Pituicytomas are rare tumors of the pituitary gland that commonly present with headaches, visual impairment, and mass effect symptoms. Although most are solid lesions, in a small percentage of cases, cystic components have been identified. Most pituicytomas are effectively treated by simple excision through a transsphenoidal approach and do not recur after total resection. Surgical resection may be associated with significant postoperative endocrine morbidity due to the tumor's proximity to the pituitary stalk and hypothalamic-pituitary structures. **Conclusion:** This case highlights the diagnostic challenge of a cystic pituicytoma mimicking a pituitary macroadenoma and emphasizes the importance of considering rare posterior pituitary tumors in the differential diagnosis of sellar lesions. Early recognition is essential given the risk of permanent hypopituitarism and diabetes insipidus after surgical treatment, and long-term follow-up remains necessary because of the risk of recurrence.

Keywords: pituicytoma; sellar lesions; panhypopituitarism.

NEUROENDOCRINOLOGY

280 (3480) EFFECTIVENESS AND SAFETY OF SYSTEMIC THERAPIES IN ADVANCED GASTROENTEROPANCREATIC NEUROENDOCRINE TUMORS: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMIZED CLINICAL TRIALS

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Introduction: Gastroenteropancreatic neuroendocrine tumors (GEP-NETs) are a heterogeneous group of neoplasms with increasing incidence in recent decades. Most are well differentiated and classified as grades 1–3 according to the Ki-67 proliferation index. Due to their nonspecific presentation, many patients are diagnosed at advanced or metastatic stages, making systemic treatment essential. Evidence synthesis is needed to evaluate systemic therapies in advanced GEP-NETs. **Objective:** To evaluate the efficacy and safety of systemic therapies in well-differentiated advanced, unresectable, or metastatic GEP-NETs, focusing on progression-free survival. **Methods:** A systematic review and meta-analysis was conducted according to PRISMA guidelines. Searches were performed in PubMed, Embase, and Cochrane databases. Study selection and data extraction were independently conducted by two reviewers using Rayyan. Randomized clinical trials evaluating systemic therapies in adults with grade 1–3 well-differentiated GEP-NETs were included. Interventions comprised somatostatin analogues, tyrosine kinase inhibitors, targeted therapies, and ^{177}Lu-DOTATATE compared with placebo or active comparators. The primary outcome was progression-free survival; secondary outcomes included overall survival, objective response rate, and grade ≥ 3 adverse events. **Results:** Included studies demonstrated clinical benefits of systemic therapies in advanced GEP-NETs, particularly with ^{177}Lu-DOTATATE. In NETTER-2, first-line treatment prolonged progression-free survival (22.8 vs. 8.5 months) and increased objective response rate to 43%. Surufatinib showed efficacy in pancreatic and extrapancreatic NETs, with greater benefit in tumors with higher proliferative index. Cabozantinib reduced progression risk in previously treated tumors. Safety profiles were manageable; PRRT showed infrequent severe hematologic toxicity, whereas tyrosine kinase inhibitors were mainly associated with hypertension, fatigue, and diarrhea. **Conclusion:** Systemic therapies demonstrated clinically relevant efficacy in advanced GEP-NETs, particularly regarding progression-free survival. ^{177}Lu-DOTATATE showed substantial clinical benefit, while tyrosine kinase inhibitors demonstrated favorable outcomes in previously treated patients and higher proliferative subgroups. Due to intervention heterogeneity, direct superiority among therapies was not established. Further studies are needed to optimize treatment sequencing and individualized care.
Keywords: astroenteropancreatic neuroendocrine tumors; therapeutics; disease-free survival.

NEUROENDOCRINOLOGY

281 (3122) EFFICACY AND SAFETY OF ONCE-DAILY ORAL PALTUSOTINE IN PATIENTS WITH ACROMEGALY: UP TO 2 YEARS IN THE PATHFNDR-1 AND PATHFNDR-2 OPEN-LABEL EXTENSION STUDIES

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Introduction: Paltusotine, a nonpeptide, selective somatostatin 2 receptor agonist, is a once-daily oral treatment for adults with acromegaly. **Objective:** This analysis evaluated the long-term efficacy and safety of paltusotine in 2 open-label extensions (OLEs) of phase 3 randomized placebo-controlled trials (PATHFNDR-1 and PATHFNDR-2). **Methods:** PATHFNDR-1 enrolled patients with biochemically controlled acromegaly (IGF-I ≤1.0× ULN) on an injected depot somatostatin receptor ligand (SRL). PATHFNDR-2 enrolled patients with biochemically uncontrolled acromegaly who were medically untreated at randomization. In the OLEs, patients received open-label paltusotine (20–60 mg/day). Adjunctive medical therapy was allowed as clinically indicated beginning at OLE Week 24. **Results:** The PATHFNDR-1 OLE enrolled 53 of 57 patients (93.0%) who completed the randomized controlled phase. The PATHFNDR-2 OLE enrolled 103 of 106 patients (97.2%) who completed the randomized controlled phase, plus 11 patients directly enrolled. In the pooled OLE population (n = 167; 53.3% female; mean age, 50.0 years), median (range) duration of paltusotine exposure during the OLEs was 115.0 weeks (0.7-211). Nineteen patients received adjunctive medication (cabergoline, bromocriptine) in an OLE. In the PATHFNDR-1 OLE, mean IGF-I levels remained stable: 0.91× ULN at OLE baseline (n = 53), 0.82 × ULN at OLE Week 48 (n = 50), and 0.81 × ULN at OLE Week 96 (n = 47). In the PATHFNDR-2 OLE, mean IGF-I levels decreased over time: 1.64× ULN at OLE baseline (n = 114), 1.21 × ULN at OLE Week 24 (n = 108), 1.06 × ULN at OLE Week 48 (n = 98), and 0.96 × ULN at OLE Week 72 (n = 78). Relative to OLE baseline, pituitary adenoma volume was reduced by > 20% in 7 (6 from placebo, 1 directly enrolled) of 83 PATHFNDR-2 patients with MRI scans (OLE Week 24); there were no clinically meaningful changes in tumor volume in PATHFNDR-1 patients (OLE Week 48). Overall (n = 167), the most common adverse events were diarrhea (15.6%), arthralgia (11.4%), and headache (11.4%). Four patients (2.4%) discontinued from an OLE due to adverse events. **Conclusion:** During long-term treatment (median follow-up of ~2 years), once-daily oral paltusotine demonstrated sustained efficacy and was well tolerated in patients transitioned from injectable depot SRLs as well as in medically untreated, biochemically uncontrolled patients at initiation. The long-term safety profile was consistent with that observed in the randomized controlled trials, with no new safety findings.
Keywords: acromegaly; medical therapy; paltusotine.

NEUROENDOCRINOLOGY

282 (2993) ENDOCRINE TUMOR PHENOTYPES IN TP53 P.R337H CARRIERS: RARE ASSOCIATION OF PITUITARY NEUROENDOCRINE TUMORS IN LI-FRAUMENI SYNDROME

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Case presentation: Case 1: A 46-year-old man carrying the germline TP53 p.R337H variant was under surveillance for a stable 1.5 cm prolactinoma treated with cabergoline. Surveillance imaging identified a 17 cm right adrenal mass. The patient underwent adrenalectomy, and histopathology confirmed high-grade adrenocortical carcinoma (ACC) with Weiss score 6/9 (modified Weiss 6/7), Ki-67 index of 20%–30%, and diffuse p53 immunostaining in 80% of tumor cells. Case 2: A 57-year-old woman harboring the germline TP53 p.R337H variant presented with vertical diplopia and was diagnosed with a 3.9 cm non-functioning pituitary neuroendocrine tumor (PitNET). Transsphenoidal surgery revealed a silent lactotroph tumor positive for prolactin and CAM 5.2, with Ki-67 index of 2.3% and negative p53 immunostaining. Postoperatively, neuro-ophthalmologic follow-up demonstrated persistent structural compression of the optic apparatus by residual tumor despite clinical stability. Cabergoline therapy was initiated. **Discussion:** Li-Fraumeni syndrome (LFS) is a hereditary cancer predisposition syndrome mainly caused by germline pathogenic variants in the TP53 tumor suppressor gene. The TP53 p.R337H founder variant, highly prevalent in Southern and Southeastern Brazil, is classically associated with pediatric ACC, breast cancer, sarcomas, and central nervous system tumors. PitNETs are not considered part of the classic LFS spectrum. Although somatic TP53 alterations have been associated with aggressive pituitary tumors, particularly corticotroph neoplasms, germline TP53 variants are rarely linked to PitNETs. These cases demonstrate the occurrence of both functioning and silent pituitary tumors in unrelated TP53 p.R337H carriers. In the first patient, the coexistence of indolent prolactinoma and highly aggressive ACC illustrates marked intra-individual endocrine tumor heterogeneity. The second case expands the phenotypic spectrum of pituitary manifestations in LFS. Negative p53 immunostaining does not exclude TP53-related tumorigenesis, particularly in the setting of germline variants. **Final comments:** Pituitary tumors may represent an underrecognized endocrine manifestation of the TP53 p.R337H-associated Li-Fraumeni spectrum. These findings reinforce the importance of comprehensive endocrine and neuroimaging surveillance in TP53 variant carriers and broaden the spectrum of endocrine neoplasia associated with germline TP53 pathogenic variants.
Keywords: Li-Fraumeni syndrome; TP53 p.R337H; pituitary neuroendocrine tumor.

NEUROENDOCRINOLOGY

283 (2931) EVALUATION OF ADRENAL INSUFFICIENCY IN PATIENTS AFTER NON-FUNCTIONING PITUITARY ADENOMA USING LISPRO INSULIN TOLERANCE TEST AND GLUCAGON STIMULATION: A SAFETY AND EFFICACY STUDY

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Introduction: Secondary adrenal insufficiency (SAI) is a complication with high morbidity in the postoperative period of pituitary tumors. The insulin tolerance test (ITT) is the gold standard for evaluating the hypothalamic-pituitary adrenal (HPA) axis, but its risks, such as severe hypoglycemia, and possible discomfort during its performance, are a significant limitation. The glucagon stimulation test (GST) emerges as a safer alternative with fewer adverse effects. Additionally, the shortage of regular insulin in Brazil has motivated the search for validation of rapid-acting analogs, such as insulin lispro, in the ITT. **Objective:** To compare the safety and efficacy of the ITT with insulin lispro versus the GST in evaluating cortisol reserve in patients in the postoperative period of non-functioning pituitary adenomas, analyzing the diagnostic agreement between the tests under multiple cut-off points. **Methods:** A prospective, observational study with 6 patients who underwent both tests with a minimum interval of one week. Intravenous lispro insulin was used in the ITT. Intramuscular glucagon was used in the GST. The cortisol response was assessed at multiple cut-off points (ITT: Cortisol > 11 µg/dL, Cortisol > 14 mg/dL, Cortisol > 15 µg/dL, Cortisol > 18 µg/dL; GST: Cortisol > 8.8 µg/dL) and agreement was analyzed using Cohen's Kappa coefficient. **Results:** The safety profile was superior in the GST, with no significant side effects, while 83.3% of patients reported multiple adverse effects in the ITT. For all analyses, a significance level of 5% ($p < 0.05$) was adopted. The concordance analysis for the diagnosis of adrenal insufficiency showed variable results depending on the ITT cut-off point. For cutoffs of > 11 µg/dL and > 14 µg/dL, agreement with the GST was excellent (Kappa = 1.000). For cutoffs of > 15 µg/dL and > 18 µg/dL, agreement was moderate (Kappa = 0.571), with 1 discordant case. Analysis of capillary versus plasma glucose showed significant overestimation of capillary values during ITT-induced hypoglycemia ($p < 0.001$). **Conclusion:** GST proved safer and better tolerated than ITT, with no significant adverse effects. Diagnostic agreement was excellent at lower cortisol cut-offs and moderate at stricter thresholds, with one discordant case. Lispro insulin was effective and validated in the ITT. GST is a valuable alternative, pending cut-off standardization and larger studies.
Keywords: adrenal insufficiency; glucagon stimulation test; insulin tolerance test.

NEUROENDOCRINOLOGY

284 (3403) FREQUENCY AND PRESENTATION OF CANCER IN CUSHING DISEASE

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Introduction: Clinical features of Cushing syndrome (CS) are a result of chronic hypercortisolemia, including hypertension, diabetes, obesity and immunocompromised states. A recent retrospective cohort study analyzing health databases demonstrated a 78% increased risk for all cancer types in 343 CS patients compared to 34,000 matched controls, and a 65% increased risk in Cushing disease (CD). Most prevalent types of cancer were genitourinary, thyroid, adrenal, and gynecological. Excluding prior cancer before CS diagnosis, ectopic Cushing, and adrenal tumor, this risk was still high. Remission prevented increased risk of cancer in CD. Studies have proposed that the metabolic syndrome caused by chronic hypercortisolemia is the major amplifier for cancer risk. However, evidence regarding this issue is still scarce in the literature. **Objective:** Evaluate the frequency and presentation of cancer in a single-center cohort of patients with CD. **Methods:** We conducted a retrospective review of medical records of patients treated for CD at our center between 2013 and 2025, searching for known comorbidities and demographic data. **Results:** From our database encompassing 115 patients, median age of CD diagnosis was 37 years old (range, 7–66), with women representing the majority of cases [85.2% (98/115)]. Cancer was encountered in five (4.3%) patients, including two cases of thyroid cancer, one submandibular gland carcinoma, one melanoma and one basal cell carcinoma. Among these patients, median age at endocrinological consultation was 45 (range, 39–66) and women accounted for 80% (4/5) of cases. Malignancy was diagnosed prior to CD diagnosis in all cases. Of note, one patient had an autoimmune disease (Behçet disease) and used exogenous glucocorticoid, one had severe hypercortisolism (biochemically and comorbidity-related), and the other one had a delayed diagnosis, potentially increasing cortisol exposure. All were operated and are in remission. **Conclusion:** The cancer incidence observed in our cohort is lower than that reported in the original study; however, this discrepancy may be attributable to our smaller sample size. Median age of the cancer cohort was older than that of the general CD cohort. Further research is warranted to assess whether cancer screening is necessary in this population.

Keywords: Cushing disease; cancer; screening.

NEUROENDOCRINOLOGY

285 (3217) GIANT FUNCTIONING GONADOTROPH MACROADENOMA WITH PRESERVED GONADAL AXIS: A RARE CASE REPORT

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Case presentation: A 71-year-old man developed progressive cognitive slowing and hypersomnia over several months, followed by reduced appetite, nausea, and vomiting two weeks before hospitalization. Initial laboratory evaluation revealed severe hyponatremia (serum sodium 116 mEq/L), 8 a.m. cortisol of 6.0 µg/dL (reference 6.2–18) with ACTH of 17 pg/mL (reference < 46), free T4 of 0.52 ng/dL (reference 0.78–2.44), TSH of 3.4 µIU/mL (reference 0.40–4.04), IGF-1 of 50 ng/mL (reference 35–216), prolactin of 17 ng/mL (reference 2.0–15.9), total testosterone of 713 ng/dL (reference 165–753), LH of 149 mIU/mL (reference 1.5–9.3), and FSH of 18 mIU/mL (reference 1.55–9.74). Physical examination demonstrated bitemporal hemianopsia and enlarged testes measuring 25 mL bilaterally by Prader orchidometer, with otherwise normal male external genitalia (Tanner stage G5P5). Prednisone and levothyroxine replacement therapy were initiated, resulting in clinical improvement and correction of hyponatremia. Pituitary MRI revealed a giant sellar and suprasellar mass measuring 5.8 × 4.7 × 2.9 cm, compressing the optic chiasm without cavernous sinus invasion. The patient subsequently underwent transcranial surgery, followed by marked reductions in LH, FSH, and testosterone levels (4.91 mIU/mL, 4.21 mIU/mL, and 1.0 ng/dL, respectively). **Discussion:** Functioning gonadotroph adenomas are rare and usually present as clinically nonfunctioning macroadenomas causing compressive symptoms and hypopituitarism. This case is notable for preserved gonadal steroidogenesis despite a giant pituitary macroadenoma associated with thyrotrophic and probable corticotrophic dysfunction. Elevated gonadotropin and testosterone levels associated with testicular enlargement strongly support autonomous gonadotroph secretion. Moreover, the marked postoperative reduction in testosterone levels further supports biologically active gonadotropin secretion by the tumor. **Final comments:** This case highlights a rare presentation of giant functioning gonadotroph macroadenoma with preserved gonadal axis despite significant mass effect and partial hypopituitarism. Recognition of this hormonal pattern may facilitate earlier diagnosis and appropriate management.

Keywords: pituitary macroadenoma; hypergonadotropic hyperandrogenism; gonadotropin-secreting adenoma.

NEUROENDOCRINOLOGY

286 (3330) GIANT SILENT CORTICOTROPH PITUITARY NEUROENDOCRINE TUMOR: A CASE REPORT OF UNUSUALLY AGGRESSIVE PRESENTATION AND POSTOPERATIVE ENDOCRINE MANAGEMENT

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Case presentation: A 29-year-old male, previously healthy, presented with recurrent episodes of malaise, disorientation, and confusion, progressively worsening over 2025. He also reported left eye visual loss since March 2025. Months later, he experienced a tonic-clonic seizure; cranial computed tomography performed at a local hospital revealed an expansive sellar lesion with bone destruction extending from the sella turcica to the nasopharynx. He was transferred for neurosurgical evaluation. Brain magnetic resonance imaging confirmed a heterogeneous solid-cystic extra-axial lesion centered in the sellar and parasellar regions, measuring approximately 10 × 8.5 × 8.0 cm, with bilateral cavernous sinus invasion (Knosp 4 right, 3A left), suprasellar extension, superior displacement of the posterior basofrontal parenchyma, involvement of the optic chiasm, and nasopharyngeal component. Ophthalmologic evaluation showed severely reduced right eye visual acuity, temporal hemianopsia in the left eye, and diffuse retinal nerve fiber layer loss bilaterally. Initial hormonal workup revealed low cortisol (3.7 µg/dL), with other axes within normal limits. The patient underwent right pterional craniotomy for subtotal resection. Intraoperative frozen section favored a neuroendocrine neoplasm. Final pathology confirmed a giant sparsely granulated corticotroph pituitary neuroendocrine tumor, with weak ACTH positivity (10%–15%), GATA3 positivity, and Ki-67 below 1%. Postoperative imaging at 3 and 6 months showed no tumor growth and no distant metastases on whole-spine magnetic resonance imaging and chest-abdomen-pelvis computed tomography. **Discussion:** Silent corticotroph tumors are rare pituitary neuroendocrine tumors that express ACTH immunohistochemically without clinical or biochemical evidence of hypercortisolism. They are known for aggressive behavior, including large size, invasiveness, and higher recurrence rates. This case is particularly notable for the extraordinary tumor volume at diagnosis (giant adenoma > 4 cm), the young patient age and the multidirectional intracranial and extracranial extension. **Final comments:** This case highlights the unusual presentation of a giant silent corticotroph pituitary neuroendocrine tumor with extreme local invasion despite minimal endocrine dysfunction. It reinforces the aggressive potential of these tumors, the importance of comprehensive immunohistochemical characterization and long-term multidisciplinary follow-up.
Keywords: silent corticotroph tumor; giant pituitary adenoma; pituitary neuroendocrine tumor.

NEUROENDOCRINOLOGY

287 (3482) GLP-1 RECEPTOR AGONISTS IN CRANIOPHARYNGIOMA-RELATED HYPOTHALAMIC OBESITY: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Patients with hypothalamic obesity after craniopharyngioma surgery frequently develop severe and treatment-resistant weight gain because of hypothalamic dysfunction affecting appetite regulation and energy expenditure. Emerging evidence suggests that glucagon-like peptide-1 receptor agonists (GLP-1 RAs) may improve metabolic outcomes in this population. **Objective:** To evaluate the efficacy and safety of GLP-1 RAs in patients with hypothalamic obesity following craniopharyngioma surgery. **Methods:** This systematic review and meta-analysis followed PRISMA 2020 and Cochrane recommendations. PubMed, Embase, and Cochrane CENTRAL were searched from inception to March 2026 for studies evaluating GLP-1 RAs in craniopharyngioma-related hypothalamic obesity. Random-effects models were used for pooled analyses. **Results:** Five studies comprising 177 participants were included. Compared with placebo, GLP-1 RAs significantly reduced waist circumference (MD –0.80, 95% CI –0.98 to –0.62). No significant pooled effect was observed for body weight, whereas BMI reduction approached statistical significance. Single-arm analyses suggested reductions in BMI and body weight, although heterogeneity was substantial. **Conclusion:** GLP-1 RAs may provide modest benefits, particularly for central adiposity, in patients with hypothalamic obesity after craniopharyngioma surgery. However, the limited number of studies and substantial heterogeneity warrant cautious interpretation.
Keywords: hypothalamic obesity; craniopharyngioma; GLP-1 receptor agonists.

NEUROENDOCRINOLOGY

288 (3333) GLUCAGONOMA A CASE REPORT

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Case presentation: P.M.D.S., 67 years old, male. Due to an episode of acute biliary pancreatitis, the patient underwent abdominal imaging (computed tomography), which revealed a nodular lesion in the pancreatic body. After the resolution of the acute episode, during follow-up at the gastrointestinal surgery service, an echoendoscopy-guided biopsy of the pancreatic nodular lesion was performed, the result of which was: neuroendocrine grade I (anatomopathological); CAM 5.2, chromogranin and synaptophysin positive, Ki67 < 1% (immunohistochemistry), pointing to the diagnosis of a well-differentiated neuroendocrine tumor, and the patient was then referred to the endocrinology service. During the evaluation, the patient presented with significant weight loss in recent months, decompensated diabetes mellitus (HbA1c 14.4%), and elevated glucagon levels (415 pg/mL). Therapeutic adjustments were made to the diabetes mellitus treatment, including metformin and a basal-bolus insulin regimen, and, in conjunction with the gastrointestinal surgery team, surgical treatment of the tumor was scheduled. **Discussion:** Glucagonoma is a rare neuroendocrine tumor originating from pancreatic alpha cells, with an estimated global incidence of 20 million cases per year. Most cases are sporadic, but it can have an inherited origin, associated with Multiple Endocrine Neoplasia (MEN) 1 (approximately 3% of the cases). Glucagonoma can cause various clinical manifestations (glucagonoma syndrome), including migratory necrolytic erythema (NME), diabetes mellitus (DM), weight loss, normocytic normochromic anemia, diarrhea/steatorrhea, venous thrombosis, and neuropsychiatric disorders. The most effective treatment for the tumor is surgical removal. For patients with contraindications to surgery, therapeutic options include chemotherapy, everolimus and sunitinib. **Final comments:** Glucagonoma is a rare neuroendocrine tumor, and it is crucial that physicians know its main characteristics to reduce the time to diagnosis and increase treatment success rates, resulting in a better prognosis for patients.

Keywords: glucagonoma; glucagonoma syndrome; neuroendrocrine tumor.

NEUROENDOCRINOLOGY

289 (3477) GROWTH HORMONE DEFICIENCY IN BRAZIL (2015–2025): A NATIONWIDE EPIDEMIOLOGICAL AND DIAGNOSTIC SCENARIO

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Introduction: Growth hormone deficiency (GHD) is characterized by insufficient GH production by the pituitary gland, requiring strict laboratory investigation to justify Somatropin therapy within the Unified Health System (SUS). Despite existing clinical protocols, there is a gap in understanding how regional diagnostic practices correlate with public drug dispensing. Thus, analyzing whether laboratory requests and therapeutic management in Brazil align with clinical evidence is essential for resource optimization. **Objective:** To analyze the epidemiological overview of Growth Hormone Deficiency in Brazil, assessing whether the regional patterns of diagnostic laboratory requests and therapeutic management correlate with drug dispensing and align with established clinical guidelines. **Methods:** This is an epidemiological, descriptive, and retrospective study with a quantitative approach, based on secondary data from the SUS Ambulatory Information System (SIA/SUS). The diagnostic and therapeutic landscape of Growth Hormone (GH) deficiency in Brazil was analyzed from 2015 to 2025. The procedures evaluated included GH and IGF-1 assays, the glucagon GH stimulation test, and somatropin treatment in 4 IU, 12 IU, and 16 IU presentations. The variables analyzed included the approved quantity and approved financial value of the procedures, stratified by Brazilian region and processing year. **Results:** Between 2015 and 2025, Brazil registered a total of 44,489,982 approved units of Somatropin, showing a growth of approximately 135.7% in annual demand. Geographically, a concentration is observed in the Southeast region, which was responsible for 42.5% of national consumption (18,921,285 units), in contrast to the North and Midwest regions, which together presented the lowest absolute volumes (3,072,799 and 4,407,197 respectively). In parallel, 1,853,288 diagnostic procedures were performed, with a predominance of the IGF-1 Assay (981,912) and a critical centralization in high-complexity infrastructure: the Southeast region concentrated 94.8% of the 152,515 HGH stimulation tests after Glucagon performed nationwide. **Conclusion:** The findings demonstrate that, between 2015 and 2025, there was a significant expansion in laboratory investigation and access to somatropin therapy in Brazil. However, an unequal distribution of diagnostic and therapeutic resources was observed among the country's regions, with a strong concentration of high-complexity procedures in the Southeast.

Keywords: growth hormone deficiency; epidemiology; health services research.

NEUROENDOCRINOLOGY

290 (3201) GROWTH HORMONE REPLACEMENT THERAPY IN THE TRANSITION PERIOD DOES NOT IMPACT THE QUALITY OF LIFE OF BRAZILIAN ADULTS WITH GROWTH HORMONE DEFICIENCY

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Introduction: Growth hormone deficiency (GHD) impacts longitudinal growth, body composition, and metabolic parameters. However, the impact of GHD on quality of life (QoL) remains controversial and may be influenced by social and cultural factors. In this study, we evaluated the QoL of Brazilian adult patients with childhood-onset GHD (CO-GHD) comparing those who continued GH replacement therapy (GHRT) after the transition period with those who did not as well as with healthy subjects. **Methods:** A cross-sectional study assessed QoL, using the Assessment of Quality of Life in Adults with Growth Hormone Deficiency (QoL-AGHDA) questionnaire, in 61 Brazilian adults (18-50 years). The sample included 31 patients with CO-GHD confirmed during the transition period, who continued or discontinued GHRT and 30 matched controls. **Results:** GHD patients presented poorer QoL as indicated by higher QoL-AGHDA scores than controls (median 8 [IQR 5–14] vs. 4 [IQR 2–8]; $p < 0.01$). Continued GHRT in adulthood did not impact QoL compared with discontinued GHRT (8 [IQR 6–16] vs. 9 [IQR 5–12], $p = 0.33$). Overall, women had lower QoL irrespective of GHD (9 [IQR 6–13] vs. 5 [IQR 2–8.5], $p = 0.01$, women and men, respectively). We found no association between QoL-AGHDA scores and education level ($p = 0.51$), short stature ($p = 0.78$), presence of associated pituitary deficiencies ($p = 0.34$), or overweight/obesity ($p = 0.16$). **Conclusion:** GHD impairs QoL in young adults, and GHRT does not seem to improve QoL. Therefore, in adult patients with GHD, GHRT should be individualized and combined with psychosocial interventions to optimize the overall health of this population.
Keywords: GHD; GHRT; QoL.

NEUROENDOCRINOLOGY

291 (3148) HIP CIRCUMFERENCE AS A SEX-SPECIFIC MARKER OF GLUTEOFEMORAL ADIPOSITY ASSOCIATED WITH COGNITIVE STATUS IN ELDERLY WOMEN: A TOPOGRAPHIC FAT DISTRIBUTION ANALYSIS

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Introduction: Adiposity distribution, rather than overall adiposity, may differentially influence cognitive aging. Gluteofemoral fat, estimated by hip circumference, secretes a distinct adipokine profile with neuroprotective properties compared to visceral fat. Whether hip circumference specifically predicts cognitive status in elderly women independently of other markers and gluteofemoral muscle mass remains underexplored in high cardiometabolic burden geriatric cohorts. **Objective:** Evaluate the association between hip circumference and cognitive status in elderly outpatients, assessing its specificity compared to other anthropometric indices. **Methods:** Cross-sectional retrospective study (n = 90). Cognitive status was evaluated by the Mini-Mental State Examination (MMSE), classified as altered or preserved according to education-adjusted cutoffs. Hip circumference was stratified into tertiles and compared with MMSE scores. Spearman correlation and Kruskal-Wallis tests were applied. Specificity was assessed by comparing hip circumference with abdominal circumference, waist circumference, neck circumference, waist-to-height ratio, and BMI as predictors of cognitive status. **Results:** Mean age was 76.5 ± 8.3 years; 73.3% female; 65.1% presented altered MMSE. Hip circumference was significantly higher in cognitively preserved participants (103.5 vs. 96.0 cm; $p = 0.00049$). Spearman correlation confirmed the association ($r = 0.401$; $p < 0.0001$), robust in women (n = 66; $r = 0.411$; $p = 0.001$) and non-significant in male participants (n = 24; $p > 0.05$). Tertile analysis showed a dose-response pattern: T1 = 20.0, T2 = 20.5, T3 = 26.0 MMSE points (Kruskal-Wallis $p = 0.002$). No other adiposity marker reached significance for cognitive status (abdominal, waist, neck circumferences, waist-to-height ratio: all $p > 0.09$). **Conclusion:** Hip circumference was the only anthropometric marker specifically associated with cognitive status in this sample. This topographic specificity is biologically plausible: gluteofemoral adipose tissue exhibits an adipokine profile enriched in adiponectin relative to visceral depots, potentially conferring neuroprotection through anti-inflammatory and insulin-sensitizing pathways. The stronger association in women aligns with estrogen-driven gluteofemoral deposition. Prospective body composition imaging is needed to clarify whether gluteofemoral fat actively drives this association or if muscle mass and hormonal substrates play a greater role.
Keywords: hip circumference; cognitive impairment; adiposity.

NEUROENDOCRINOLOGY

292 (3312) HYPOCORTISOLISM MASKING CENTRAL ARGININE VASOPRESSIN DEFICIENCY FOLLOWING PARASELLAR MENINGIOMA RESECTION

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Case presentation: A female patient underwent a parasellar meningioma surgical resection, subsequently developing hydrocephalus requiring a ventriculoperitoneal shunt. Postoperatively, she presented with vomiting and severe hyponatremia (sodium 115 mEq/L). Laboratory investigation revealed a plasma osmolarity of 240 mOsm/kg, urine osmolarity of 515 mOsm/kg, and urine sodium of 139 mEq/L, characterizing hypotonic hyponatremia with inappropriately concentrated urine, initially attributed to the syndrome of inappropriate antidiuretic hormone secretion. Treatment with fluid restriction and sodium replacement was initiated. Later, due to persistent headache and fatigue, panhypopituitarism was observed, including secondary adrenal insufficiency, central hypothyroidism, hypogonadotropic hypogonadism, growth hormone deficiency, and hyperprolactinemia due to stalk effect. Laboratory tests demonstrated basal cortisol < 0.5 mcg/dL, adrenocorticotropic hormone 3 pg/mL, free T4 0.6 ng/dL, reduced insulin-like growth factor 1, and decreased gonadotropins. After initiating glucocorticoid replacement, there was a rapid correction of hyponatremia, followed by a progressive elevation in serum sodium, raising the hypothesis of previously unrecognized central arginine vasopressin deficiency (AVD). The initial absence of exuberant hypotonic polyuria and significant hypernatremia suggested that coexisting hypocortisolism reduced the renal clearance of free water, partially masking the typical manifestations of central AVD. **Discussion:** Central AVD is a recognized complication following sellar and parasellar neurosurgery, classically manifesting with hypotonic polyuria and hypernatremia. However, the coexistence of secondary adrenal insufficiency can modify its clinical presentation. Cortisol participates in water homeostasis by favoring the renal clearance of free water. In cortisol deficiency, water retention and reduced free water excretion occur, which can mask the typical manifestations of central AVD. In the present case, glucocorticoid replacement restored the capacity for water excretion, making the clinical picture more evident. **Final comments:** The case highlights that typical manifestations of central AVD could be masked by secondary hypocortisolism due to sellar and parasellar neurosurgeries. Comprehensive hormonal evaluation is critical in patients with persistent fluid and electrolyte disorders in the neurosurgical postoperative period.

Keywords: hypocortisolism; arginine vasopressin deficiency; parasellar meningioma.

NEUROENDOCRINOLOGY

293 (3359) JUGULOTYMPANIC PARAGANGLIOMA ASSOCIATED WITH NON-SECRETORY ADRENAL ADENOMA: CASE REPORT

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Case report: A previously healthy 37-year-old woman presented with right-sided rhythmic pulsatile tinnitus for more than five years, associated with temporary hearing loss. She denied otalgia, otorrhea, recurrent otitis, comorbidities, or regular medication use. Otoscopy revealed a pulsatile bright-red bulging of the right tympanic membrane, while the left ear was normal. Audiometry (06/2024) demonstrated mild mixed hearing loss on the right side. Magnetic resonance imaging of the mastoids showed a nodular lesion in the tympanic cavity extending toward the auditory tube, suggestive of glomus tympanicum. The patient underwent tumor resection in 03/2025. Histopathological examination demonstrated positivity for chromogranin A and synaptophysin, focal expression of S100 protein, and a Ki-67 index of 1%, confirming middle ear paraganglioma. Complementary investigation with abdominal computed tomography revealed a hypodense left adrenal nodule. Adrenal protocol CT confirmed a 0.8 cm adenoma (9 HU). Plasma and urinary metanephrines were within normal limits, excluding pheochromocytoma. **Discussion:** Head and neck paragangliomas are rare, highly vascularized tumors originating from nonchromaffin paraganglionic cells, accounting for approximately 0.6% of tumors in this region. Jugulotympanic glomus tumors are the most common subtype involving the temporal bone and classically present with pulsatile tinnitus and progressive hearing loss. Their coexistence with nonfunctioning adrenal adenoma is rare and may initially raise suspicion for pheochromocytoma. Jugulotympanic paraganglioma is the most common benign tumor of the middle ear and may behave in a locally aggressive manner. Investigation for synchronous lesions is mandatory, as up to 20% of patients present with multiple tumors, including pheochromocytomas. In this case, the adrenal incidentaloma initially raised suspicion for pheochromocytoma, later excluded by biochemical evaluation. Reports describing the association between head and neck paraganglioma and nonfunctioning adrenal adenoma are scarce. Despite the absence of a suggestive family history, genetic testing for SDHx mutations may be considered. **Conclusion:** This case highlights the importance of systemic investigation in patients with head and neck paragangliomas. The presence of an adrenal mass requires biochemical exclusion of pheochromocytoma before any surgical intervention.

Keywords: jugulotympanic paraganglioma; adrenal adenoma; pheochromocytoma.

NEUROENDOCRINOLOGY

294 (3284) LANGERHANS CELL HISTIOCYTOSIS: A 27-YEAR DIAGNOSTIC JOURNEY FROM ISOLATED AVP DEFICIENCY TO NEURODEGENERATIVE ATAXIA

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Case presentation: A 37-year-old woman presented in 1997 with polyuria and polydipsia. In 1998, investigation led to AVP deficiency (AVP-D) diagnosis. During the diagnostic workup, multiple investigations (including CT-chest, PET-CT, infectious serologies, and autoimmune tests) were performed without identifying a definitive etiology. Follow-up magnetic resonance imaging (MRI) in late 2017 identified an enhancing mass involving the sellar and suprasellar regions, notably presenting with the absence of the characteristic T1-weighted hyperintensity of the neurohypophysis. After progressive ataxic gait developed, the patient was diagnosed in 2019 with slowly progressive cerebellar ataxic syndrome. During the diagnostic workup, multiple investigations were repeated. Differential diagnoses based on MRI imaging included histiocytic disease, granulomatous disorders and CNS neoplasms, but due to the location of the lesion, biopsy of the CNS was not feasible. In 2023, a lesion was identified on the hard palate and was biopsied, initially revealing nonspecific inflammatory changes without evidence of neoplasia. A review with immunohistochemical analysis was performed in March 2025, demonstrating findings consistent with Langerhans cell histiocytosis (LCH). The patient was referred to Hematology and initiated treatment with cladribine. Currently, the patient is clinically stable but has significant functional impairment, and is dependent for activities of daily living. **Discussion:** In LCH, AVP-D may precede other clinical manifestations by prolonged periods. A major diagnostic challenge was the inability to biopsy the CNS lesion due to its location. Only after the emergence of a palatal lesion, accessible to biopsy, was it possible to establish the diagnosis through histopathological analysis. Another aspect is the impact of delayed diagnosis on clinical outcome. The patient developed progressive cerebellar ataxia and significant functional decline related to CNS involvement by the disease. An earlier diagnosis might have altered the clinical course. **Final considerations:** This case emphasizes the importance of continuous etiological investigation of patients with isolated AVP-D, even when initial evaluations are inconclusive and the importance of considering rare diseases such as LCH in the evaluation, particularly when structural abnormalities are present on imaging, and reinforces the need for long-term follow-up with periodic reassessment.

Keywords: isolated arginine vasopressin deficiency; Langerhans cell histiocytosis; neurodegenerative ataxia.

NEUROENDOCRINOLOGY

295 (3289) LATE DIAGNOSIS OF CUSHING'S DISEASE IN A PATIENT WITH PITUITARY MACROADENOMA

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Case presentation: A 23-year-old female patient began her clinical history with galactorrhea, secondary amenorrhea, and weight gain (30 kg in 6 months). She sought gynecological evaluation in 2023, when laboratory tests revealed a mild elevation in prolactin levels (43 ng/mL even after dilution). Subsequently, a pituitary magnetic resonance imaging was requested, revealing a 1.7 cm pituitary macroadenoma. Given this scenario, and after endocrinological evaluation in October 2024, the diagnosis established was a nonfunctioning pituitary macroadenoma with hyperprolactinemia due to pituitary stalk compression (pseudoprolactinoma). The attending physician then decided to initiate cabergoline 0.5 mg once weekly; however, the patient used the medication irregularly for 5 months. Upon returning for reassessment, she was referred to a specialized outpatient clinic at a teaching institution in June 2025, where evident stigmata of hypercortisolism were identified: violaceous striae on the abdomen and lower limbs, hirsutism (face, chin, neck, back), alopecia, asthenia, lower limb edema, mood changes, and irritability. She was instructed to discontinue cabergoline, and specific laboratory tests along with a new pituitary magnetic resonance imaging were requested, focusing on the diagnosis of Cushing's Syndrome. The results were compatible with ACTH-dependent Cushing's Syndrome, and the patient was referred to neurosurgery, undergoing transsphenoidal surgery in August 2025, with normalization of clinical symptoms and laboratory findings, while using prednisolone 2.5 mg daily. **Discussion:** The presented case demonstrates the importance of specialists mastering basic concepts of neuroendocrinology and the need for a routine clinical assessment aimed at identifying signs and symptoms of glandular hyperfunction or hypofunction in patients with sellar masses. **Final comments:** The lack of mastery of basic concepts and the failure to recognize explicit clinical signs of severe hypercortisolism led to an inaccurate diagnosis and inappropriate treatment, delaying the patient's access to surgery, the only therapeutic option with curative potential for her disease.

Keywords: pituitary macroadenoma; Cushing's disease; transsphenoidal surgery.

NEUROENDOCRINOLOGY

296 (3008) LINEAGE REASSIGNMENT IN NON-FUNCTIONING PITUITARY NEUROENDOCRINE TUMORS BASED ON TRANSCRIPTION FACTORS

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Introduction: The 2022 World Health Organization classification of pituitary neuroendocrine tumors emphasizes lineage assignment based on transcription factor (TF) expression. The objective of this investigation was to evaluate TF-based reclassification on lineage assignment in a cohort of non-functioning pituitary neuroendocrine tumors (NF-PitNET). **Methods:** We analyzed 140 NF-PitNET classified by IHC for pituitary hormones, including ACTH, LH, FSH, GH, TSH, and prolactin, with additional IHC assessment of the TF: SF1, TPIT, and PIT1. Previous pathological classification and final TF-based classification were available for comparison in 137 evaluable paired cases. Tumors were categorized as lineage-defined (SF1, TPIT, and PIT1 or null cell). Paired comparisons were performed using the exact McNemar test. We also assessed lineage rescue rate among tumors previously classified as null cell only based on the pituitary hormonal classification and described the final distribution of TF-defined lineages. **Results:** In the previous classification, 65 of 137 tumors were lineage-defined, whereas 72 of 137 were classified as null cell. After TF-based reclassification, 122 of 137 tumors were assigned to a defined lineage, increasing the proportion of lineage-defined tumors from 47.4% to 89.1%. In the paired analysis, 64 tumors classified as null cell acquired lineage assignment, whereas 7 tumors previously classified as lineage-defined were reclassified as null cell, resulting in 57 new lineage-defined cases. This increase was statistically significant by the exact McNemar test ($p = 1.26 \times 10^{12}$). Among the 72 tumors classified as null cell, 64 were reclassified after TF assessment, corresponding to a lineage rescue rate of 88.9%. In the overall cohort, final TF-based classification identified pure SF1 tumors in 70 of 140 cases (50.0%), pure PIT1 tumors in 20 of 140 (14.3%), pure TPIT tumors in 12 of 140 (8.6%), combined transcription factor expression in 22 of 140 (15.7%), and persistent null cell phenotype in 16 of 140 (11.4%). When tumors with combined expression were also considered, SF1 was present in 87 of 140 cases (62.1%), PIT1 in 37 of 140 (26.4%), and TPIT in 28 of 140 (20.0%). **Conclusion:** TF-based reclassification increased lineage assignment in NF-PitNET, reduced the null cell category, and rescued lineage assignment in nearly 90% of unclassified cases. These findings support the systematic incorporation of transcription factors into routine pathological evaluation.

Keywords: pituitary; non-functioning; tumors.

NEUROENDOCRINOLOGY

297 (3126) LONG-TERM SAFETY AND EFFICACY OF ONCE-DAILY ORAL PALTUSOTINE IN COMBINATION WITH CABERGOLINE FOR THE TREATMENT OF PATIENTS WITH ACROMEGALY

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Introduction: Paltusotine, a nonpeptide, selective somatostatin 2 receptor agonist, is approved in the United States and Europe as a once-daily oral treatment for adults with acromegaly. The dopamine receptor agonist cabergoline may be used (off-label) as an add-on therapy for acromegaly not fully controlled by a somatostatin receptor ligand (SRL). **Objective:** This analysis evaluated the safety and efficacy of paltusotine in combination with cabergoline in patients with acromegaly who have been followed for up to 4 years. **Methods:** ACROBAT Advance is an ongoing, single-arm, open-label extension study. At enrollment in ACROBAT Edge, the phase 2 parent study, patients were either suboptimally controlled on injected octreotide or lanreotide, alone or in combination with cabergoline, or they required combination therapy or pasireotide to achieve normal IGF-I levels. In Advance, the paltusotine formulation was switched from capsule (dose range, 10–40 mg) to tablet (dose range, 20–60 mg) during year 3. Adjunctive treatment (e.g., cabergoline, pegvisomant) was used after Week 12. **Results:** This analysis included 25 patients from Edge who received ≥1 dose of cabergoline in Advance (4 also received pegvisomant). In Advance, IGF-I levels on paltusotine monotherapy were similar to Edge study baseline values (on injected SRL) and decreased to normal or near normal over time when adjunctive medications were added. Median (IQR) IGF-I levels were 1.38 × ULN (1.15-1.48; n = 25) at Edge baseline and 1.26 × ULN (1.14–1.50; n = 24), 1.17 × ULN (1.05–1.40; n = 24), 1.17 × ULN (0.92–1.40; n = 23), and 1.05 × ULN (0.88–1.16; n = 21) in Advance at months 12, 24, 36, and 48, respectively. The cabergoline dose ranged from 0.25 to 7.0 mg/week. All 25 patients experienced a reduction in IGF-I levels with cabergoline, and 21 (84.0%) showed IGF-I normalization. In the analysis population, the most common adverse events (AEs) were arthralgia (36.0%), headache (36.0%), and diarrhea (36.0%). One serious drug-related AE (cholelithiasis) was reported, and 1 patient discontinued the study due to an AE (headache; considered possibly related to study medication). **Conclusion:** In patients with acromegaly who were candidates for combination therapy based on treatment history, long-term results (up to 4 years) show that once-daily oral paltusotine in combination with cabergoline showed improved biochemical control and was well tolerated with no new safety findings.

Keywords: acromegaly; cabergoline; paltusotine.

NEUROENDOCRINOLOGY

298 (3426) MANAGEMENT CHALLENGES IN A GIANT INVASIVE PITUITARY ADENOMA WITH PERSISTENT RESIDUAL DISEASE: A CASE REPORT

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Case presentation: 43-year-old male was referred to neuroendocrinology after diagnosis of giant pituitary adenoma and surgical treatment. He reported five years of progressive visual deficit, predominantly in the right eye, with reduced libido and partial preservation of erection. Preoperative MRI revealed a pituitary lesion measuring 8.9 × 6.0 × 3.7 cm, with suprasellar extension, optic chiasm compression, sellar floor destruction, and relationship with internal carotid arteries. In 2021 he underwent transsphenoidal surgery, with partial resection. Postoperatively, he developed transient diabetes insipidus and panhypopituitarism (adrenal insufficiency, central hypothyroidism, hypogonadism), requiring hormonal replacement. Pathology confirmed invasive pituitary adenoma. Follow-up imaging showed tumor persistence (2.6 x 2.4 x 1.8 cm) with significant visual deficit. In 2025, he underwent transcranial reintervention and was referred for radiotherapy for residual disease. **Discussion:** Giant pituitary adenomas (≥4 cm) represent 6-10% of pituitary tumors, predominantly non-functioning and more frequent in men. This case demonstrates late presentation with neuro-ophthalmological predominance, typical of large tumors. Studies show adenomas ≥30 mm present visual impairment in 82.5% and panhypopituitarism in 28.8%. Cavernous sinus invasion, present in 71.3%, explains difficult resection. Total resection in giant multilobular adenomas is only 8%, with 67% presenting residue requiring multiple interventions. Although surgery decompresses optic pathways in 70%–90%, prolonged symptoms (five years) correlate with lower recovery. Postoperative panhypopituitarism occurs in 5%–15%, requiring longitudinal follow-up. Multimodal approach with surgery, reintervention, and radiotherapy is frequently necessary for disease control. Adjuvant radiotherapy achieves tumor control rates exceeding 95% in nonfunctioning adenomas. Long-term local control reaches 95% at 5 years and 75% at 20 years. However, hypopituitarism develops in approximately 50% of patients within 5 years post-radiotherapy, necessitating indefinite endocrine monitoring. **Final comments:** This report demonstrates the impact of delayed diagnosis of invasive giant pituitary adenomas and highlights the importance of early recognition of visual and hormonal deficiency symptoms to optimize outcomes. Multidisciplinary management and long-term surveillance remain essential for disease control and complication prevention.

Keywords: pituitary neoplasms; adenoma; hypopituitarism.

NEUROENDOCRINOLOGY

299 (3286) MEDIAL WALL RESECTION OF THE CAVERNOUS SINUS IN CUSHING DISEASE: PRELIMINARY SINGLE-CENTER EXPERIENCE

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Introduction: Cushing disease (CD), caused by corticotroph adenomas, remains a surgical challenge, particularly when cavernous sinus (CS) invasion is suspected. In recent years, the medial wall of the CS has been identified as a site of microscopic invasion by these adenomas. In the past, resection of the medial wall was avoided because of concerns regarding injury to neurovascular structures. However, advances in endoscopic surgery and better understanding of the CS anatomy have enabled selective medial wall resection (MWR). **Objective:** Compare surgical outcomes in patients with CD who underwent endoscopic transsphenoidal surgery with or without MWR at a single-center. **Methods:** We performed a retrospective cohort study of consecutive treatment-naïve patients with CD who underwent surgery between 2024 and 2026. Patients with CS invasion extending beyond the medial wall and intercarotid line (Knosp grades 2-4) were excluded. MWR was performed when the sail sign was identified on MRI or when intraoperative findings suggested tumor adherence or focal invasion of the CS, characterized by the absence of a clear plane between the adenoma and the CS. Remission was defined biochemically as absence of hypercortisolism. **Results:** Comparing 8 (38.1%) patients who underwent MWR to 13 who did not, median age was similar [37.5 years (range, 15–53) and 33 years (range, 24–49), respectively, p = 0.306], as was the percentage of female patients [7 (87.5%) and 11 (84.6%), respectively, p = 0.799]. Median tumor diameter was 0.7 cm (range, 0.2–0.9) in the MWR group and 0.9 cm (range, 0.3–1.4) in the non-MWR group (p = 0.109). Biochemical remission was achieved in all patients who underwent MWR, however it was not statistically different from the non-MWR group (84.6%; p = 0.243). Separate histopathological evaluation of the medial wall was performed in 5 cases, confirming microscopic invasion in 2 cases. During the study period, 3 recurrences occurred, all in patients who had not undergone MWR. Reoperation achieved remission in 1 case after subsequent MWR, whereas the remaining 2 patients, treated without MWR, did not achieve remission. No postoperative vascular or cranial nerve injury were observed. **Conclusion:** In this preliminary single-center experience, selective MWR was safe and associated with excellent remission rates in carefully selected patients. Longer follow-up is needed to determine the impact of performing MWR or not on long-term recurrence rates.

Keywords: Cushing disease; cavernous sinus; medial wall.

NEUROENDOCRINOLOGY

300 (2921) METASTASES TO THE SELLAR REGION: A SINGLE-CENTER EXPERIENCE

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Introduction: Pituitary metastases account for approximately 1% of sellar tumors and are often misdiagnosed as adenomas due to overlapping clinical and radiological features. They typically occur in patients aged 45–74 years and frequently involve the neurohypophysis, often presenting with rapid growth and arginine vasopressin deficiency (AVP-D). Visual impairment, particularly bitemporal hemianopsia, may also occur, although nonspecific symptoms such as headache, nausea, and fatigue may be the only clinical manifestations. **Objective:** report our institutional experience with pituitary metastases and identify common clinical and radiological features that may support early diagnostic suspicion. **Methods:** A retrospective review of patients undergoing surgery for sellar lesions at a single neurosurgery referral center (2013–2025) was performed. Demographic, clinical, and biochemical data were collected, and magnetic resonance imaging was used to assess the metastases' characteristics. Histopathological and immunohistochemical analyses were conducted according to World Health Organization classification. **Results:** Among 1925 sellar surgeries, seven cases (0.3%) were from metastases: thyroid (n = 2), Ewing sarcoma, renal, breast, bronchial neuroendocrine tumor, and one neuroendocrine tumor of unknown origin. Median age was 53.4 years (range, 24–66 years), five patients were female, and four had a known diagnosis of cancer. Symptoms correlated with tumor size and location, and included cranial nerve palsy (n = 5), visual loss (n = 4), headache (n = 4), and weight loss (n = 2). Preoperative hypopituitarism was identified in all five evaluated cases, including panhypopituitarism in three patients, whereas no cases of AVP deficiency were observed. Lesions showed frequent cavernous sinus involvement (n = 6), and a median size of 5.5 cm. Imaging findings were heterogeneous and often suggested alternative diagnoses (macroadenoma, meningioma, bone lesion) or nonspecific. Intraoperatively, tumors were frequently hemorrhagic or firm. **Conclusion:** Rapid progression, large invasive lesions, cranial nerve involvement, and hypopituitarism, particularly with a history of malignancy, may aid differentiation from primary tumors. Imaging variability highlights the absence of a pathognomonic pattern, making histopathological confirmation essential for diagnosis.

Keywords: pituitary metastases; hypopituitarism; malignancy.

NEUROENDOCRINOLOGY

301 (3196) MULTIPLE MYELOMA WITH SELLAR INVOLVEMENT BY PLASMACYTOMA: A CASE REPORT

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Case presentation: G.S., a 70-year-old woman, presented with a pathological humeral fracture. Investigation of secondary causes of bone fragility led to a diagnosis of multiple myeloma. Physical examination revealed a cranial vault mass associated with decreased visual acuity, ptosis, and left eye proptosis. Brain MRI demonstrated multiple expansile skull lesions, the largest measuring 8.8 × 6.3 × 5.7 cm, in addition to a clival lesion (4.0 × 3.8 cm) with destruction of the sellar floor and posterior clival cortex, partially obliterating the prepontine cistern. Pituitary hormonal function was preserved. The patient underwent partial resection through an endoscopic endonasal approach without perioperative complications. Follow-up MRI showed a residual lesion in the paranasal cavity (1.8 × 1.7 × 2.5 cm) without significant mass effect. Histopathological analysis revealed dense monoclonal plasma cell infiltration consistent with sellar plasmacytoma. Systemic chemotherapy was initiated, and the patient remains under outpatient follow-up. **Discussion:** Sellar plasmacytoma is a rare monoclonal plasma cell neoplasm arising in the sella turcica, accounting for less than 1% of sellar lesions, with only a few cases reported in the literature. It may occur as an isolated lesion or as part of systemic multiple myeloma. Clinical manifestations commonly include headache, diplopia, visual impairment, cranial neuropathies, and hypopituitarism. Cranial nerve involvement is more frequent than in pituitary adenomas. Preoperative diagnosis is challenging because imaging findings are nonspecific and may mimic other sellar tumors. Definitive diagnosis relies on histopathological confirmation demonstrating monoclonal plasma cell proliferation. Treatment generally includes systemic chemotherapy and radiotherapy, whereas surgery is mainly indicated for decompression and tissue diagnosis in cases with neurological compromise or significant mass effect. Prognosis appears comparable to that of multiple myeloma without extramedullary disease. **Final comments:** This case highlights the diagnostic challenges of sellar plasmacytoma and reinforces the importance of considering plasma cell neoplasms in the differential diagnosis of atypical sellar and clival lesions. Preserved pituitary function despite extensive local invasion illustrates the heterogeneous presentation of this rare condition. Histopathological confirmation and multidisciplinary management remain essential for optimal care.

Keywords: sellar plasmacytoma; clival lesion; multiple myeloma.

NEUROENDOCRINOLOGY

302 (3242) NEUROPSYCHIATRIC EFFECTS AND MOOD DISORDERS ASSOCIATED WITH ANABOLIC-ANDROGENIC STEROID WITHDRAWAL: A SYSTEMATIC REVIEW

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Introduction: Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone used for aesthetic and athletic purposes. Chronic use at supraphysiological doses suppresses the hypothalamic-pituitary-gonadal axis, resulting in hypogonadism during withdrawal, which is associated with depression, anxiety, sleep disturbances, aggressiveness, and suicidal behavior. Approximately 65% of users resume use after discontinuation due to worsening psychological symptoms. This review aimed to synthesize the evidence regarding neuropsychiatric effects and mood disorders resulting from AAS withdrawal. **Objective:** To identify and synthesize neuropsychiatric symptoms and mood disorders associated with the withdrawal phase in former users of anabolic-androgenic steroids. **Methods:** This systematic literature review was conducted in accordance with PRISMA guidelines. The search was performed in the PubMed, Embase, and ScienceDirect databases (2016–2026) using the PICO strategy. Inclusion criteria comprised primary studies involving human subjects of any age undergoing voluntary or assisted withdrawal who reported neuropsychiatric symptoms. Study eligibility was independently assessed by two reviewers using the Rayyan software. **Results:** Of the 721 records identified, nine studies were selected. Regarding study design, the sample consisted predominantly of seven cross-sectional studies, one longitudinal study, and one cohort study, involving mainly young men. Depressive symptoms were the most frequent finding: moderate or severe depression was identified in 19.4% of former users, compared with 7.2% of active users and no cases among controls. Studies demonstrated an independent association with Major Depressive Disorder (OR 2.06) and a higher risk of suicide attempts/self-harm (OR 1.40). Anxiety, irritability, and aggressiveness were also recurrent. Regarding sleep, 66% of users reported poorer sleep quality during withdrawal. Finally, evidence demonstrated persistent impairment in physical and mental quality of life for up to two years after cessation, characterized by fatigue, depressive symptoms, and social dysfunction. **Conclusion:** AAS withdrawal is associated with significant neuropsychiatric morbidity, notably depression, anxiety, and sleep disturbances, which may persist for years after discontinuation. These findings reinforce the need for multidisciplinary monitoring and specialized health policies for this population.
Keywords: anabolic agents; substance withdrawal syndrome; mood disorders.

NEUROENDOCRINOLOGY

303 (3407) NEUROPSYCHIATRIC EFFECTS ASSOCIATED WITH THE USE OF GLP-1 AGONISTS IN THE TREATMENT OF OBESITY

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Introduction: Obesity and type 2 diabetes (T2D) are epidemics associated with high rates cardiovascular morbidity and mortality and psychiatric disorders. GLP-1 receptor agonists, such as tirzepatide, have demonstrated efficacy in glycemic control and weight reduction. However, their action in the central nervous system has raised concerns about potential neuropsychiatric effects, including depression and suicidal ideation. This systematic review synthesizes recent evidence on the neuropsychiatric effects of GLP-1RAs and their clinical implications. **Objective:** To evaluate the neuropsychiatric safety and effects on quality of life associated with the use of GLP-1 receptor agonists in the treatment of obesity. **Methods:** This is a systematic review conducted in the PubMed database, using the descriptors “GLP-1 Receptor Agonists”, “Obesity”, “Neuropsychiatric”, “Depression”, “Anxiety”, and “Eating Behavior”. Eight articles published between 2024 and 2026 were analyzed. **Results:** The analyzed studies demonstrated a mostly safe neuropsychiatric profile for GLP-1 agonists. Meta-analyses and clinical trials did not identify a significant increase in serious psychiatric events or suicidal behavior, and tirzepatide showed rates of suicidal ideation similar to placebo. Some studies even suggest a lower risk of suicidality compared to other anti-obesity therapies. However, results regarding depression were heterogeneous, with pharmacovigilance studies reporting signs of nervousness, stress, and sleep disturbances. Additionally, GLP-1RAs were associated with improved quality of life, reduced emotional eating, and potential neuroprotective effects, although events such as dizziness and dysgeusia were more frequent. **Conclusion:** GLP-1 agonists present a predominantly safe neuropsychiatric profile, without consistent evidence of increased risk of suicidality. In addition to metabolic benefits, they are associated with improved quality of life and emotional eating. However, clinical monitoring is recommended for patients with previous psychiatric vulnerability.
Keywords: GLP-1 receptor agonists; obesity; neuropsychiatric.

NEUROENDOCRINOLOGY

304 (3568) OUTCOMES OF PATIENTS WITH ECTOPIC ACTH-DEPENDENT CUSHING SYNDROME (EAS) UNDERGOING BILATERAL ADRENALECTOMY (BA)

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Case presentation: Seven cases of ectopic ACTH-dependent Cushing syndrome treated with bilateral adrenalectomy (BA) at a tertiary referral center are described, with prolonged postoperative follow-up. Etiologies included an ACTH-secreting pancreatic tumor and thymic carcinoid, while the remaining patients had ectopic ACTH secretion without identification of the primary tumor site. There was a predominance of female patients, and age at surgery ranged from 23 to 58 years. Follow-up duration ranged from 3 to 28 years, with a mean follow-up of approximately 12 years. All patients remained on chronic glucocorticoid and mineralocorticoid replacement therapy after surgery. Two patients experienced occasional adrenal-related complications, whereas the remaining patients had no complications related to adrenal insufficiency. Suspected residual adrenal tissue was observed in two cases, although this was not confirmed on imaging studies. Four patients died due to progression of the underlying disease. The patient with the longest follow-up remains under regular outpatient care after 28 years and is clinically stable. **Discussion:** Bilateral adrenalectomy is recognized as an effective strategy for definitive control of hypercortisolism in ACTH-dependent Cushing syndrome, particularly in cases of occult ectopic secretion, metastatic disease, or refractory response to medical therapy. Despite recent advances in pharmacological treatments, BA remains an important therapeutic option when the primary tumor cannot be identified or when hormonal control is inadequate. In this series, the prolonged follow-up reinforces the effectiveness of BA in achieving sustained control of hypercortisolism, even in patients with advanced disease. The absence of deaths related to adrenal insufficiency demonstrates that chronic hormone replacement therapy can be managed safely under specialized follow-up. Although four patients died due to tumor progression, prolonged adrenal clinical stability and adequate hormonal control were observed throughout follow-up. **Final considerations:** This case series reinforces the role of bilateral adrenalectomy as a safe and effective therapeutic strategy in the management of ectopic ACTH-dependent Cushing syndrome, particularly when the primary tumor cannot be identified or when medical therapy is insufficient. The prolonged follow-up demonstrates that favorable long-term outcomes are possible with a multidisciplinary approach and specialized care.

Keywords: adrenalectomy; Cushing; ACTH.

NEUROENDOCRINOLOGY

305 (2976) PITUITARY MASS AND PANHYPOPITUITARISM CAUSED BY HISTOPLASMA IN AN IMMUNOCOMPETENT PREGNANT PATIENT

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Case presentation: A 28-year-old woman at 32 weeks of gestation sought medical care in December 2024 due to severe headache, nausea, vomiting, bitemporal hemianopsia and progressive visual blurring, being initially diagnosed with an anxiety crisis. After an uncomplicated cesarean section, her symptoms persisted and she returned in January 2025 reporting clinical worsening and inability to breastfeed. A cranial CT scan revealed a sellar lesion, later characterized on MRI as a solid-cystic expansive mass in the sella turcica (3.65 × 2.7 × 1.9 cm), suggestive of a pituitary macroadenoma (Figures 1 and 2). Laboratory tests demonstrated panhypopituitarism (Table 1) and treatment with prednisone and levothyroxine was initiated. She underwent transsphenoidal surgery in February 2025, and the biopsy of the lesion revealed yeast-like structures, indicating pituitary histoplasmosis. After surgery, she remained with panhypopituitarism and hemianopsia and developed central diabetes insipidus. Antifungal treatment with amphotericin B followed by itraconazole was administered. She is currently followed at a neuroendocrinology outpatient clinic for clinical management of the hormonal deficiencies. **Discussion:** Infectious processes and other neoplasms should be included among the differential diagnoses of sellar masses (a,b,c). Pituitary infections are rare, particularly those caused by fungal agents (d,e,f,g,h,a). Histoplasma infection involving the central nervous system is especially uncommon in immunocompetent individuals and in the absence of disseminated disease, with even fewer reports of pituitary involvement (i,d,e,j). On the other hand, the immunological changes associated with pregnancy may have increased susceptibility to the disease in this case (k,l). In general, the recommended treatment for central nervous system histoplasmosis consists of liposomal amphotericin B for four to six weeks, followed by itraconazole for 12 months (j,m,i). In this patient's case, postoperative panhypopituitarism also required clinical management towards the hormonal axes not initially affected. **Final comments:** This case report highlights a rare etiology of pituitary macroadenoma, particularly in an immunocompetent patient, with a challenging initial diagnosis, reinforcing the importance of recognizing alarm signs that require further investigation, especially in pregnancy.

Keywords: pituitary histoplasmosis; panhypopituitarism; histoplasma hypophysitis.

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306 (3169) POST-BARIATRIC NESIDIOBLASTOSIS: DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN REFRACTORY POSTPRANDIAL HYPOGLYCEMIA

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Case presentation: A 35-year-old female with a history of sleeve gastrectomy performed two years prior presented with recurrent postprandial hypoglycemic episodes lasting three years, progressively worsening after bariatric surgery. Symptoms included tremors, diaphoresis, palpitations, nausea, and dizziness, occurring predominantly after meals, with no loss of consciousness or seizures. Exogenous insulin and insulin secretagogue use were excluded. Laboratory workup revealed a HOMA-IR of 3.5, insulin of 16.6 µIU/mL with simultaneous postprandial glucose of 94 mg/dL, and C-peptide of 2.49 ng/mL, confirming endogenous hyperinsulinism. CT and MRI of the abdomen showed no focal pancreatic lesion. Selective pancreatic arterial calcium stimulation test demonstrated a greater than twofold rise in insulin in the superior mesenteric and distal splenic arterial territories, localizing hypersecretion to the body and tail of the pancreas and establishing the diagnosis of nesidioblastosis. Treatment was initiated with octreotide, acarbose, and liraglutide, achieving partial reduction of hypoglycemic episodes. However, significant gastrointestinal side effects, high treatment costs, and persistent hypoglycemia substantially impaired the patient's quality of life. Partial pancreatectomy is currently under consideration. **Discussion:** Nesidioblastosis, or non-insulinoma pancreatogenous hypoglycemia syndrome (NIPHS), is a rare cause of endogenous hyperinsulinism characterized by diffuse beta-cell hyperplasia. Its association with bariatric surgery is well established, likely mediated by exaggerated GLP-1 secretion and consequent beta-cell hypertrophy. Diagnosis is challenging, as cross-sectional imaging is typically unremarkable, requiring functional localization via selective arterial calcium stimulation testing. Medical management includes somatostatin analogues, alpha-glucosidase inhibitors, and GLP-1 receptor agonists, though responses are often partial. **Final comments:** This case underscores the diagnostic complexity of post-bariatric nesidioblastosis, in which normal pancreatic imaging may delay diagnosis. Selective arterial calcium stimulation testing is indispensable for functional localization and surgical planning. Clinicians should maintain a high index of suspicion for NIPHS in post-bariatric patients with refractory postprandial hypoglycemia.
Keywords: nesidioblastosis; postprandial hypoglycemia; bariatric surgery.

NEUROENDOCRINOLOGY

307 (3400) PREVALENCE AND ASSOCIATED FACTORS OF PITUITARY DYSFUNCTION FOLLOWING TRAUMATIC BRAIN INJURY IN ADULTS: A SYSTEMATIC REVIEW

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Introduction: Traumatic brain injury (TBI) can cause hypothalamic-pituitary axis dysfunction. However, diagnosis is challenging due to the overlap between endocrine manifestations and the cognitive and neuropsychiatric sequelae of trauma. Early recognition of this complication may help characterize patterns of endocrine involvement and support appropriate post-TBI follow-up. **Objective:** To evaluate the prevalence of pituitary dysfunction after TBI in adults, identify associated clinical, radiological, and laboratory factors, and describe the most frequently affected hormonal axes. **Methods:** This systematic review followed PRISMA guidelines. Searches were performed in PubMed, Scopus and Embase using the MeSH terms (Brain Injuries, Traumatic, Craniocerebral Trauma, Brain Concussion and Hypopituitarism) combined with Boolean operators and Entry terms. Eligible studies included observational designs, such as prospective and retrospective cohorts and cross-sectional studies, conducted in adult populations, published between 2021 and 2026, in English or Portuguese. Reviews, case reports, studies involving pituitary dysfunction from non-TBI etiologies, and animal or pediatric populations were excluded. Two reviewers independently performed study selection and data extraction. From the initial search, 359 articles were identified, with 133 excluded due to duplication. After title and abstract screening, 27 studies were selected for full-text review, of which 14 met the inclusion criteria. **Results:** Post-TBI pituitary dysfunction prevalence varied, ranging from 2.63% at 24 weeks to 82.4% in the acute phase, with chronic rates ranging from 24% to 59.3% in selected cohorts, influenced by injury severity, assessment timing, and diagnostic criteria. Endocrine deficits were also reported in mild TBI populations, including athletes and military personnel. Somatotropic (GH/IGF-1) and gonadotropic axes were most affected, followed by thyroid and adrenal axes. Hyperprolactinemia and vasopressin deficiency were less frequently described. Key risk factors included greater TBI severity, lower GCS, skull base fractures, basal cistern SAH, midline shift, pituitary MRI abnormalities, prolonged hospitalization, and poor functional outcomes. **Conclusion:** Pituitary dysfunction is a clinically relevant complication after TBI. Findings support targeted endocrine screening in high-risk adults, especially those with severe injury, neuroimaging abnormalities, or prolonged hospitalization.
Keywords: brain injuries, traumatic; craniocerebral trauma; hypopituitarism.

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308 (3209) PREVALENCE OF ADRENAL NODULES AND HYPERPLASIA AT DIAGNOSIS OF ACTH-DEPENDENT CUSHING'S SYNDROME

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Introduction: The diagnostic approach to ACTH-dependent Cushing's syndrome (CS) requires imaging studies to identify the source of ACTH production. In both Cushing´s disease (CD) and ectopic ACTH secretion (EAS), adrenal abnormalities such as hyperplasia and nodules are frequently observed, likely due to chronic ACTH stimulation. Despite this apparent causal relationship, the frequency of adrenal abnormalities in this population has not been well characterized. These findings may pose challenges for the differential diagnosis as adrenal etiologies of ACTH-dependent CS, such as pheochromocytomas, although rare, can occur. **Objective:** To evaluate the radiological prevalence of adrenal abnormalities in patients with ACTH-dependent CS, correlating findings with clinical and hormonal data, and to assess adrenal imaging evolution after removal of ACTH stimulation. **Methods:** This single-center retrospective study was conducted from 2007 to 2025. Inclusion criteria were confirmed diagnosis of CD or EAS and availability of adrenal imaging for reassessment. Images were classified as normal, hyperplastic, or nodular by two blinded radiologists. Radiological findings were correlated with clinical and laboratory data. When available, follow-up abdominal imaging in patients with adrenal nodules after surgical treatment of the ACTH-secreting source was analyzed to assess changes after removal of ACTH stimulation. **Results:** Among 143 patients, 73% female with a median age of 37 years (range, 4–74), 59 (41%) had adrenal morphological abnormalities on imaging. Of these, 20 (13.9%) had adrenal nodules (6 bilateral, 14 unilateral), and 39 (27%) had adrenal hyperplasia. Among the 20 patients with nodules at diagnosis, 10 underwent follow-up imaging, showing reduction or resolution in 50% of cases. **Conclusion:** We observed abnormal adrenal morphology in 41% of patients with ACTH-dependent CS. The prevalence of adrenal nodules, including bilateral lesions, was higher than expected for the general population, particularly considering the relatively young age of this cohort. Additionally, adrenal nodules decreased or regressed in half of the cases after removal of chronic ACTH stimulation. These findings support the hypothesis that adrenal morphological changes in ACTH-dependent CS are secondary to chronic ACTH excess. Therefore, a primary adrenal etiology becomes less likely in this setting, particularly in the absence of clinical features suggestive of adrenergic hypersecretion.

Keywords: ACTH-dependent Cushing's syndrome; adrenal nodule; adrenal hyperplasia.

NEUROENDOCRINOLOGY

309 (3076) RARE ASSOCIATION OF PRIMARY HYPERPARATHYROIDISM, PITUITARY MACROADENOMA, AND PARAGANGLIOMA: A CASE REPORT

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Case presentation: A 63-year-old woman was referred for evaluation of an incidental pituitary macroadenoma without initial hormonal hypersecretion or hypopituitarism, consistent with a nonfunctioning lesion. Her history included bilateral breast cancer at age 33 (currently in remission), hypertension, T2DM, dyslipidemia, obesity and depression. During hospitalization for COVID-19 complicated by pulmonary embolism, hypercalcemia was detected, leading to the diagnosis of primary hyperparathyroidism. The coexistence of pituitary macroadenoma and primary hyperparathyroidism raised suspicion for MEN1. During follow-up, she developed facial plethora, proximal myopathy, and weight gain. Laboratory evaluation confirmed ACTH-dependent Cushing's syndrome. Screening abdominal MRI revealed a left adrenal lesion suspicious for pheochromocytoma, with elevated normetanephrine levels (2.1 nmol/L; 2.33× ULN). After alpha-blockade, she underwent left adrenalectomy, and pathology confirmed paraganglioma with negative ACTH staining. She subsequently underwent transsphenoidal surgery, which confirmed a corticotroph pituitary neuroendocrine tumor; however, remission was not achieved, and ketoconazole therapy was initiated. Genetic testing for MEN1 was negative. Given the rare triad of paraganglioma, pituitary adenoma, and primary hyperparathyroidism, MEN5 was considered. Whole-exome sequencing identified a pathogenic heterozygous TP53 variant (c.1010G>A; p.Arg337His), with no variants detected in MAX, SDHB, or other MEN5/MAX-related genes. **Discussion:** The TP53 p.R337H variant identified in this patient has a high prevalence in Southern and Southeastern Brazil and aligns with the Li-Fraumeni/Li-Fraumeni-like cancer spectrum, likely explaining her early-onset breast cancer. However, its association with the MEN-like endocrine phenotype should be interpreted cautiously, as this presentation is atypical for this variant. Although pituitary tumors and paragangliomas are not classic Li-Fraumeni manifestations, emerging evidence suggests TP53-related syndromes may include a broader neuroendocrine phenotype. **Final comments:** This case highlights a challenging presentation in which a MEN5-like triad occurred within a Li-Fraumeni-like context, emphasizing the role of comprehensive genomic sequencing in clarifying rare endocrine associations and guiding long-term oncologic surveillance.

Keywords: primary hyperparathyroidism; pituitary macroadenoma; paraganglioma.

NEUROENDOCRINOLOGY

310 (3040) RESTRICTIVE-TYPE ANOREXIA NERVOSA IN A YOUNG MALE WITH HYPOGONADISM AND LOW BONE MASS

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A 23-year-old male food engineering student presented with 1-year fatigue, low energy, decreased libido, and erectile dysfunction. Multiple emergency consultations identified anemia, and intravenous iron was administered without clinical improvement. Endocrine evaluation revealed hypogonadotropic hypogonadism (total testosterone 94 ng/dL, FSH 1.3 mUI/mL LH 0.9 mUI/mL), initially treated with clomiphene followed by transdermal testosterone. Further assessment revealed a two-year history of body dissatisfaction, restrictive eating, food selectivity with excessive intake of carotenoid-rich food, intense daily exercise, and binge-eating episodes. He denied anosmia or head trauma. Physical examination showed carotenemia and BMI 17.2 kg/m². Testicular volumes were 6 and 8 cm³ on ultrasound. Laboratory tests demonstrated borderline fasting glucose, normal TSH with low free T4 of 0.8 ng/dL (ref. 0.9–1.8), and persistent hypogonadism. Pituitary MRI was unremarkable. Bone densitometry showed low bone mass (L1–L4 Z-score –2.7). After excluding secondary causes, restrictive-type anorexia nervosa (AN) was diagnosed associated with hypogonadotropic hypogonadism and euthyroid sick syndrome. A multidisciplinary approach was initiated. After 8 months, the patient achieved BMI 19.6 kg/m², binge episodes resolved, hormonal axes normalized, and testosterone discontinued. Although AN is classically associated with young women, it remains underrecognized in males, particularly when endocrine manifestations predominate over psychiatric symptom, leading to delayed diagnosis and inappropriate treatment. Chronic energy deficit suppresses hypothalamic GnRH pulsatility, leading to functional hypogonadism, as well as adaptive thyroid changes, and impaired bone health. A multidisciplinary approach is essential for addressing both metabolic and psychological aspects of the disorder. The recovery of the hypothalamic-pituitary-gonadal axis after nutritional rehabilitation reinforces the reversible nature of these alterations and the need to treat the underlying disorder before hormonal therapy. AN should be considered in young males with hypogonadism and nutritional abnormalities, especially in the context of selective eating that leads to skin changes. In a society driven by aesthetic pressure, excessive concern with appearance can lead to serious illness. Early recognition of AN in young men is essential to prevent long-term complications, particularly bone loss and reproductive dysfunction.

Keywords: anorexia nervosa; hypogonadotropic hypogonadism; low bone mass.

NEUROENDOCRINOLOGY

311 (3057) SELLAR ALTERATIONS IN PATIENTS WITH PRIMARY ANTIPHOSPHOLIPID SYNDROME

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Introduction: Antiphospholipid syndrome (APS) is a systemic autoimmune disease associated with vascular events related to thrombotic and inflammatory mechanisms. Endocrine complications secondary to APS are rare and underdiagnosed, and their incidence remains unknown. **Objective:** To evaluate pituitary morphology in patients with primary thrombotic APS. **Methods:** Cross-sectional study including patients with primary thrombotic APS regularly followed at a university Rheumatology outpatient clinic. All patients underwent magnetic resonance imaging (MRI) of the sella turcica. Sellar morphology was classified according to the degree of concavity and proportion of sella turcica occupancy by the pituitary gland: category 1, normal sella turcica; category 2, concavity occupying less than 1/3 of the sella; category 3, concavity occupying between 1/3 and 2/3 of the sella; category 4, concavity occupying more than 2/3 of the sella height; and category 5, when the gland was not visualized (empty sella). **Results:** Fifty-nine patients (49 women, 83%), aged 49.5 ± 13.2 years, with APS underwent MRI of the sella turcica. Twenty-four patients (42.1%) presented category 1; 15 patients (26.3%), category 2; 14 patients (24.5%), category 3; 2 (3.5%), category 4; and 2 (3.5%), category 5. Additionally, 1 nonfunctioning pituitary neuroendocrine tumor (laboratorially evaluated) and 1 Rathke's cleft cyst were identified. **Conclusion:** Morphological alterations of the sella turcica, including partially empty sella and empty sella, were frequently observed in this cohort of patients with primary thrombotic APS. These findings suggest possible structural pituitary involvement associated with the disease, reinforcing the need for further studies to evaluate its clinical and functional repercussions.

Keywords: pituitary gland; empty sella syndrome; antiphospholipid syndrome.

NEUROENDOCRINOLOGY

312 (3442) SELLAR SPINDLE CELL SARCOMA AFTER CHILDHOOD CRANIOPHARYNGIOMA: CHALLENGES IN THE MANAGEMENT OF PANHYPOPITUITARISM AND DIABETES INSIPIDUS

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Case presentation: A 32-year-old man, diagnosed with craniopharyngioma at age 8, underwent partial resection (2002) and radiotherapy (2005), subsequently developing panhypopituitarism and central diabetes insipidus. He has been on chronic levothyroxine, physiologic glucocorticoid replacement and intranasal desmopressin, with right-sided blindness as a sequela. Growth hormone was used in childhood and briefly in 2021, but discontinued due to depression and elevated liver enzymes in the context of hepatic steatosis, and he remains with central hypothyroidism, hypogonadism and hypocortisolism, probable growth hormone deficiency and low bone mass. In February–March 2025, CT and MRI showed a new infiltrative sellar/suprasellar expansile lesion. In June 2025, partial transsphenoidal resection was performed; histopathology with immunohistochemistry revealed a low-grade spindle cell neoplasm with osteochondroid metaplasia, consistent with sellar sarcoma. In February 2026, the lesion was deemed unresectable, and radiotherapy was indicated. On recent follow-up, he required hospitalization for severe hyponatremia (sodium 115 mEq/L) related to excessive desmopressin, requiring dose reduction and reinforced fluid management guidance. He also has marked mixed dyslipidemia and hepatic steatosis, limiting testosterone and growth hormone replacement and the full use of lipid-lowering agents. **Discussion:** Sellar sarcoma is extremely rare, particularly in a patient previously treated for craniopharyngioma with surgery and radiotherapy, suggesting a possible relationship with prior exposure. This case underscores the complexity of managing panhypopituitarism and diabetes insipidus in the setting of an aggressive, unresectable sellar tumor, with a need for careful replacement of multiple axes, precise desmopressin titration to avoid severe sodium disorders, and attention to constraints imposed by liver disease and dyslipidemia. **Final remarks:** This report reinforces the importance of long-term, multidisciplinary follow-up of patients with childhood hypothalamic-pituitary tumors, given the risk of rare sellar neoplasms and complex endocrino-metabolic complications in adult life, with significant impact on prognosis and quality of life.

Keywords: sellar spindle cell sarcoma; panhypopituitarism; central diabetes insipidus.

NEUROENDOCRINOLOGY

313 (3366) SERUM ALPHA-SUBUNIT IN NON-FUNCTIONING PITUITARY NEUROENDOCRINE TUMORS: BASELINE ELEVATION, SEX-RELATED DIFFERENCES, AND POSTOPERATIVE DYNAMICS

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Introduction: Non-functioning pituitary neuroendocrine tumors (NF-PitNETs) do not cause clinical hormone hypersecretion syndromes, but may retain the capacity to synthesize or secrete glycoprotein hormone subunits. The clinical value of serum alpha-subunit measurement in these tumors remains uncertain. **Objective:** To evaluate baseline serum alpha-subunit levels, sex-related differences, tumor subtype associations, and postoperative dynamics in a large surgical cohort of patients with NF-PitNETs. **Methods:** We retrospectively analyzed serum alpha-subunit levels in 146 patients with NF-PitNETs before and after surgery. Alpha-subunit was assessed both as a continuous variable and according to predefined categories, with analyses stratified by sex and menopausal status. Tumors were classified by pituitary hormone immunohistochemistry, and a subset of tumors previously classified as null-cell was reassessed using steroidogenic factor 1 (SF-1) immunohistochemistry. Statistical associations were evaluated using appropriate tests: chi-square, Fisher's exact, analysis of variance, Kruskal-Wallis, and Spearman correlation. **Results:** Elevated serum alpha-subunit was observed in 18% of cases (26 of 146), with a higher frequency in males than females (24% versus 7%; $p = 0.008$). This difference remained significant after stratifying females by menopausal status ($p = 0.02$). However, the postoperative percentage reduction was similar between sexes ($p = 0.79$). Alpha-subunit levels differed by tumor subtype ($p = 0.009$), being higher in tumors expressing luteinizing hormone/follicle-stimulating hormone or thyroid-stimulating hormone. No elevation was observed among tumors initially classified as null-cell and subsequently reclassified as SF-1-positive. Levels did not correlate with age ($p = 0.11$) but correlated positively with luteinizing and follicle-stimulating hormone levels ($p < 0.001$). A postoperative reduction of 30% or more was noted even in cases with normal baseline levels, demonstrating the potential value of dynamic assessment beyond baseline elevation alone. **Conclusion:** Serum alpha-subunit was elevated in a relevant subset of patients, particularly males, and associated with tumors expressing glycoprotein hormones. Postoperative reduction, even with normal baseline values, suggests that serum alpha-subunit may serve as a complementary dynamic biomarker in NF-PitNETs. Further studies are warranted to determine its role in postoperative surveillance and recurrence prediction.

Keywords: non-functioning pituitary neuroendocrine tumors; alpha-subunit; glycoprotein hormones.

NEUROENDOCRINOLOGY

314 (3307) SILENT CORTICOTROPH PITNET IN A PATIENT WITH CONGENITAL ADRENAL HYPERPLASIA DUE TO 21-HYDROXYLASE DEFICIENCY

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Case presentation: A 55-year-old patient with the simple virilizing form of classic congenital adrenal hyperplasia caused by 21-hydroxylase deficiency (variants I2sp/V281L and I2sp/G291S), with a 46,XX karyotype and male gender identity, receiving treatment with dexamethasone and testosterone undecanoate. In April 2024, the patient presented with headache and visual scotomas. Pituitary magnetic resonance imaging revealed an expansive sellar lesion compatible with a pituitary macroadenoma (PitNET), with suprasellar and bilateral parasellar extension, measuring 2.3 × 1.9 × 1.7 cm, infiltrating the cavernous sinuses and exerting a compressive effect on the optic chiasm. Preoperative hormonal evaluation demonstrated an ACTH level of 93.7 pg/mL and a serum cortisol level below 1.0 µg/dL during chronic dexamethasone therapy, with preserved function of the remaining pituitary axes. The patient underwent transsphenoidal surgery in April 2025 without relevant complications. **Discussion:** 21-hydroxylase deficiency leads to reduced cortisol synthesis and consequent ACTH elevation due to loss of negative feedback within the hypothalamic-pituitary-adrenal axis. Prolonged exposure to reduced glucocorticoid feedback may have contributed to corticotroph proliferative changes, although a causal relationship between congenital adrenal hyperplasia and the development of silent corticotroph PitNET remains uncertain. Histopathological examination confirmed a sparsely granulated corticotroph PitNET, with ACTH positivity in approximately 40% of tumor cells and a Ki-67 proliferative index of approximately 1%. The absence of clinical manifestations of hypercortisolism, combined with the histopathological profile, supported the classification of the lesion as a silent corticotroph neoplasm. **Final comments:** We report a rare case of sparsely granulated silent corticotroph PitNET in a patient with classic congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Although the causal relationship remains undefined, the coexistence of these two rare conditions underscores the importance of monitoring for neuro-ophthalmological manifestations in patients with long-standing congenital adrenal hyperplasia.

Keywords: congenital adrenal hyperplasia; silent corticotroph adenoma; pituitary neuroendocrine tumor.

NEUROENDOCRINOLOGY

315 (3529) EXOGENOUS CUSHING SYNDROME SECONDARY TO THE USE OF A NATURAL SUPPLEMENT WITH SUSPECTED HIDDEN GLUCOCORTICOID: A CASE REPORT

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Female patient, 35 years old, with obesity since childhood, developed rapid weight gain from November 2024, associated with muscle weakness, violaceous striae, alopecia, menstrual irregularity, and clinical signs of hyperandrogenism, increasing from 86 kg to 115 kg in less than one year. She reported continuous use, since November 2024, of a supplement called Bálsamo Gold, marketed as a natural product for joint pain relief, with complete improvement of chronic pain, temporally coinciding with the progressive development of a classic Cushingoid phenotype. Initial investigation for Cushing syndrome repeatedly showed results consistent with suppression of the hypothalamic-pituitary-adrenal axis, including persistently suppressed urinary free cortisol, late-night salivary cortisol, and cortisol after 1 mg dexamethasone, raising the possibility of inadvertent glucocorticoid exposure. Despite this, the patient denied recent or excessive glucocorticoid use that could explain the florid clinical picture, raising suspicion of occult exposure through other substances. After discontinuation of the supplement in November 2025, the patient developed nausea, vomiting, diarrhea, and presyncope, with laboratory documentation of severe central adrenal insufficiency (baseline cortisol 0.47 mcg/dL; ACTH <5 pg/mL), confirming significant suppression of the hypothalamic-pituitary-adrenal axis. Corticosteroid therapy was initiated, with subsequent improvement in symptoms. Discontinuation of the product was also associated with marked improvement in muscle weakness, although previously controlled osteoarticular pain recurred during supplement use. Although laboratory analysis of Bálsamo Gold was not performed, the chronology of events, development of classic exogenous Cushing syndrome during its use, and severe adrenal insufficiency after discontinuation, requiring glucocorticoid replacement, provide strong indirect evidence of prolonged exposure to undisclosed exogenous corticosteroids. This case highlights the potential risk that supplements marketed as natural products may contain undeclared pharmacologically active substances, emphasizing the importance of detailed investigation of herbal and alternative compound use in patients with manifestations compatible with hypercortisolism or unexplained adrenal insufficiency.

Keywords: Cushing syndrome; adrenal insufficiency; hidden corticosteroids.

NEUROENDOCRINOLOGY

316 (3423) THE INFLUENCE OF DIETARY PATTERNS ON THE NEUROENDOCRINE MODULATION OF ANXIETY AND DEPRESSION: A LITERATURE REVIEW

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Introduction: Anxiety and depression are strongly related to dietary habits and the gut-brain axis. Diets rich in ultra-processed foods, refined sugars, and trans fats promote inflammatory processes, intestinal dysbiosis, and neuroendocrine alterations, worsening symptoms. Conversely, healthy patterns with vegetables, proteins, and unsaturated fats help maintain emotional balance. **Objective:** To analyze the correlation between the gut-brain axis and the modulation of anxiety and depression. The aim is to identify pathophysiological mechanisms, evaluating the impact of inflammatory diets and the therapeutic efficacy of nutritional interventions and supplementation on neuroendocrine regulation. **Methods:** Integrative review (2006–2025) in the PubMed, SciELO, and Google Scholar databases using the descriptors: "Depression and diet," "Gut-brain axis," and "Nutrient deficiency and anxiety". Original articles/reviews in humans (Portuguese/English/Spanish) in full text were included. Duplicates or articles without clinical relevance were excluded, resulting in 20 articles. **Results:** The relationship between diet and mental health is bidirectional: anxiety and depression induce excessive consumption of comfort foods, while malnutrition intensifies inflammation and nutritional deficits, perpetuating the emotional state. The cycle dysregulates neurotransmitters (serotonin, dopamine, GABA), affecting mood, sleep, and cognition. In the gut-brain axis, the microbiota produces metabolites and neuroactive molecules; dysbiosis increases intestinal permeability and inflammatory cytokines, generating neuroinflammation and psychic instability. In parallel, alterations in the Hypothalamic-Pituitary-Adrenal (HPA) axis increase cortisol and undermine the stress response. To counteract this process, micronutrients such as vitamin D, magnesium, omega-3, zinc, B vitamins, tryptophan, and probiotics exert neuroprotective and anti-inflammatory functions, regulating hormones, optimizing neurotransmission, and reducing systemic inflammation in the management of mental disorders. **Conclusion:** The evidence reinforces that nutritional intervention should be integrated into the multidisciplinary approach to anxiety and depression. The combination of a balanced diet, professional monitoring, psychological therapy, and, if necessary, medication, is essential to promote better quality of life and mental health.

Keywords: anxiety; alimentation; microbiota-gut-brain axis.

NEUROENDOCRINOLOGY

317 (2916) THYROID CANCER AND STRUCTURAL THYROID DISEASE IN PATIENTS WITH ACROMEGALY: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Acromegaly is most commonly caused by a growth hormone (GH)-secreting pituitary adenoma, resulting in sustained elevation of insulin-like growth factor-1 (IGF-1). Given that thyroid follicular cells express IGF-1 receptors, activation of this pathway may promote cellular proliferation and predispose to thyroid neoplasia in patients with acromegaly. However, routine thyroid cancer screening in these patients has not been specifically incorporated in clinical practice. **Objective:** We aim to assess whether adults with acromegaly have higher odds of thyroid cancer and other structural thyroid abnormalities compared with patients without acromegaly. **Methods:** We systematically searched PubMed, Embase, and Cochrane Library for observational studies comparing thyroid cancer prevalence in adults with and without acromegaly. The primary outcome was the odds of thyroid cancer. Secondary outcomes were the odds of benign thyroid findings and the mean difference in thyroid volume. Pooled odds ratios (ORs) and mean differences (MDs) with 95% confidence intervals (CIs) were calculated using a random-effects Mantel-Haenszel model. All statistical analyses were performed using R software, version 4.5.2. Risk of bias was assessed using ROBINS-E and JBI tools according to study design. **Results:** We included thirteen studies, comprising 2,674 patients with acromegaly. Our study showed that patients with acromegaly were associated with significantly higher odds of thyroid malignancy (OR 4.09, 95% CI 2.23–7.52; P = 0.0003). The subgroup analyses also showed higher odds of papillary (OR 2.93, 95% CI 1.68–5.13; P = 0.0002) and follicular (OR 5.46, 95% CI 1.10–27.16; P = 0.0382) carcinoma in these patients compared with the control group. In addition, benign thyroid findings (OR 4.50, 95% CI 1.08–18.66; P = 0.0408) and mean thyroid volume (MD 11.73 mL, 95% CI 5.92–17.53; P = 0.0076) were significantly higher in patients with acromegaly compared with the control group. **Conclusion:** Our study showed that patients with acromegaly were significantly associated with higher odds of thyroid malignancy, such as papillary and follicular carcinoma. In addition, these patients were significantly associated with benign thyroid findings and greater thyroid volume.

Keywords: acromegaly; thyroid cancer; meta-analysis.

NEUROENDOCRINOLOGY

318 (3344) TREATMENT OF CABERGOLINE-RESISTANT PROLACTINOMAS: IS THERE A ROLE FOR SOMATOSTATIN ANALOGUES?

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Introduction: Treatment of choice for prolactinomas is cabergoline (CAB). However, nearly 10% of prolactinomas are resistant to CAB, defined by the failure to normalize prolactin levels and/or achieve significant tumor shrinkage (>50% reduction) despite at least 6 months of high-dose treatment (>3.5 mg/week). In such cases, pituitary surgery is frequently indicated. The use of first-generation somatostatin analogs (SA), such as octreotide and lanreotide, in patients with CAB resistance has been evaluated in case reports and in a single case series including 5 patients, with conflicting results. **Objective:** The aim of our study was to evaluate PRL and tumor responses to the addition of first-generation SA to CAB in patients with resistant prolactinomas. **Methods:** We added SA at the maximum recommended doses (octreotide 30 mg or lanreotide 120 mg) to CAB therapy in patients with resistance to CAB, already defined previously. **Results:** We evaluated 4 male patients who, at diagnosis, were between 15 and 34 years of age, had PRL levels from 250 to 46,600 ng/mL, and maximum tumor diameters from 30 to 80 mm. The starting doses of CAB ranged from 1 to 3.5 mg/week. Maximum CAB doses (4.5 to 5.5 mg/week) were maintained for 24 to 108 months, without normalization of PRL levels or significant tumor, characterizing CAB resistance. Among the 4 patients, 2 refused pituitary surgery and 2 had previously undergone surgery. We added SA (octreotide in 3 patients and lanreotide in 1) to CAB therapy and maintained them for periods from 4 to 92 months. Treatment with SA resulted in prolactin reduction in 3 patients (55%, 60% and 67%) and tumor reduction in 2 (9% and 55%). The 2 patients who exhibited tumor shrinkage also showed reductions in PRL levels, with normalization in 1. **Conclusion:** The presence of somatostatin receptors in tumoral lactotroph cells provided the rationale for the use of SA both in the literature and in our study. In our series, PRL levels decreased in 3 out of 4 patients, with normalization in one. Significant tumor reduction was observed in only 1 patient. Our findings were consistent with those of the published series of 5 cases, in which only 2 achieved reductions in both PRL levels and tumor size. The response of patients with resistant prolactinomas to first-generation SA was modest in both series. The use of pasireotide, a second-generation SA, might be evaluated as an alternative therapeutic option for resistant prolactinomas.
Keywords: prolactinoma; somatostatine analogues; cabergoline resistance.

NEUROENDOCRINOLOGY

319 (3309) VENOUS THROMBOEMBOLISM RISK AND THE IMPACT OF THROMBOPROPHYLAXIS IN CUSHING'S DISEASE: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Chronic hypercortisolism in Cushing's disease (CD) promotes a hypercoagulable state, substantially increasing the risk of venous thromboembolism (VTE) and contributing significantly to patient morbidity and mortality. **Objective:** To evaluate the proportion of VTEs in CD, assess its association with VTE, examine the impact of thromboprophylaxis on VTE occurrence, and evaluate the role of direct oral factor Xa inhibitors (DOACs) versus low-molecular-weight heparin (LMWH) as prophylactic strategies in this population. **Methods:** This systematic review was conducted following the JBI methodology and reported following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses statement. Studies were included if they met eligibility criteria defined using the Population, Exposure/Intervention, Comparator, and Outcome frameworks, tailored to predefined questions addressing VTE proportion, risk association, and the efficacy and safety of thromboprophylaxis. The main outcomes were VTE occurrence and bleeding risk. Data sources included PubMed, Embase, and CENTRAL. Study selection, data extraction and risk-of-bias assessment were performed independently and in pairs. The certainty of evidence was assessed using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) methodology. **Results:** For the proportion of VTEs in CD, 31 studies were included; 9 evaluated the association between CD and VTE, 4 assessed the effect of thromboprophylaxis in hypercortisolism, and 1 compared DOAC with LMWH. The proportion of VTEs was higher in studies evaluating the entire follow-up period than in those assessing only postoperative transsphenoidal surgery complications (Overall proportion [OP]: 7%, 95% CI: 6% to 10% versus; OP: 1%, 95% CI: 1% to 3%, respectively, p = 0.000, 9,514 patients). With moderate quality of evidence, CD was associated with an increased risk of VTE (RR 8.67, 95% CI: 5.18–14.51; 9 studies, 6,804 participants), and thromboprophylaxis reduced the incidence of VTE (RR 0.30, 95% CI: 0.18–0.50; 4 studies, 805 participants). Bleeding events were infrequently reported across studies. We found no differences between DOACs (apixaban) and LMWH regarding VTE and bleeding (1 propensity score-matched study, n = 2,978). **Conclusion:** CD is associated with increased VTE risk. Thromboprophylaxis reduces VTE incidence with an acceptable safety profile. Further prospective studies are needed to optimize protocols and risk stratification.
Keywords: Cushing's disease; venous thromboembolic events; thromboprophylaxis.

NEUROENDOCRINOLOGY

320 (3250) WOMEN WITH ACROMEGALY EXHIBIT GREATER GLOBAL PSYCHOLOGICAL DISTRESS: AN ANALYSIS USING DISTRESS, ANXIETY, AND QUALITY-OF-LIFE SCALES

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Introduction: Acromegaly is a chronic disease mainly caused by GH-secreting pituitary adenomas, leading to significant physical, metabolic, and psychosocial changes. Patients frequently experience psychological distress and impaired quality of life, and sex-related factors may influence their subjective experience. **Objective:** To evaluate sex-related differences in psychological distress, anxiety, depression, and quality of life among patients with acromegaly. **Methods:** This cross-sectional study included 46 patients with acromegaly. Data were managed using REDCap. The Hospital Anxiety and Depression Scale (HADS), NCCN Distress Thermometer and Problem List, and AcroQoL questionnaire were applied. Descriptive and comparative analyses were performed between men and women. **Results:** Of the 46 participants, 32 (69.6%) were women and 14 (30.4%) were men. No differences were observed in IGF-1 control. Women had higher global distress scores than men (7.53 ± 2.58 vs. 5.14 ± 2.85; p = 0.007) and a trend toward higher anxiety scores (9.9 ± 4.7 vs. 7.4 ± 3.8; p = 0.063), with no difference in depression scores (15 ± 9 vs. 13 ± 9; p = 0.607). Women more frequently reported concerns related to pain (97% vs. 64%; p = 0.009), fear (77% vs. 29%; p = 0.002), sadness/depression (70% vs. 21%; p = 0.003), and treatment-related decisions (67% vs. 29%; p = 0.018). In AcroQoL, women showed lower psychological and appearance scores, although differences were not statistically significant. **Conclusion:** Women with acromegaly exhibited greater psychological distress and emotional burden than men. Although depression and quality-of-life scores did not differ significantly, women showed a trend toward worse psychological perception. The absence of differences in IGF-1 control suggests that psychosocial factors may contribute to these findings. These results highlight the importance of integrating psychological care into acromegaly management while considering sex-related differences.

Keywords: acromegaly; psychological distress; quality of life.

OBESITY

321 (3497) ACUTE DRUG-INDUCED HEPATITIS ASSOCIATED WITH SEMAGLUTIDE USE IN A POST-BARIATRIC SURGERY PATIENT: A CASE REPORT

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Case presentation: A 72-year-old woman with previous Roux-en-Y gastric bypass, dyslipidemia, osteoporosis and lactose intolerance was admitted with abdominal pain, nausea, vomiting, abdominal distension and diarrhea after approximately three weeks of semaglutide use for weight loss without medical prescription. She had received four weekly doses before symptom worsening and hospitalization. Laboratory investigation demonstrated marked acute hepatocellular injury with aspartate aminotransferase 974 U/L and alanine aminotransferase 497 U/L, associated with mild elevation of alkaline phosphatase and gamma-glutamyl transferase, without significant hyperbilirubinemia. Amylase and lipase levels were normal, excluding acute pancreatitis. Viral hepatitis investigation, cytomegalovirus and Epstein-Barr virus acute infection screening were negative. Inflammatory markers were not elevated. Abdominal computed tomography showed no biliary obstruction, pancreatitis or significant hepatic abnormalities. Semaglutide was immediately discontinued during hospitalization, resulting in progressive clinical improvement and significant reduction of liver enzymes. Follow-up evaluation demonstrated complete normalization of transaminases after drug withdrawal, reinforcing the diagnosis of semaglutide-induced drug-induced liver injury. **Discussion:** Although gastrointestinal adverse effects are common with glucagon-like peptide-1 receptor agonists, clinically significant hepatotoxicity remains rare. The temporal association between semaglutide initiation and liver injury, exclusion of alternative etiologies and complete biochemical recovery after discontinuation strongly support the diagnosis of drug-induced hepatitis secondary to semaglutide. Patients with previous bariatric surgery may present baseline gastrointestinal symptoms, potentially delaying recognition of adverse drug reactions. The increasing off-label use of semaglutide reinforces the importance of careful clinical monitoring and awareness of potentially severe hepatic adverse events. **Final comments:** Drug-induced hepatitis should be considered in patients presenting acute elevation of liver enzymes after semaglutide initiation, particularly in the presence of gastrointestinal symptoms and exclusion of alternative etiologies. Early recognition and prompt discontinuation of the medication are essential for favorable clinical outcomes.

Keywords: drug-induced hepatitis; semaglutide; obesity.

OBESITY

322 (3393) ANTI-OBESITY THERAPY WITH SEMAGLUTIDE IN A PATIENT WITH GENETIC LIPID MYOPATHY: A CASE REPORT

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Case presentation: A 44-year-old male patient with a 10-year history of progressive proximal muscle weakness, fatigue, myalgia, and functional limitation was diagnosed in 2022 with PNPLA3-related genetic lipid myopathy after molecular confirmation. He developed reduced physical activity and weight gain, associated with hepatic steatosis, severe obstructive sleep apnea, arterial hypertension, prediabetes, dyslipidemia, and depressive symptoms. Baseline segmental multifrequency bioelectrical impedance analysis showed weight 166.1 kg, BMI 51.3 kg/m^2, fat mass 86.6 kg, skeletal muscle mass 45.0 kg, and visceral fat level 31. Semaglutide 0.25 mg weekly was initiated with individualized nutritional counseling, medium-chain triglycerides, whey protein, L-carnitine, and coenzyme Q10. During follow-up, he had cold-induced peripheral cyanosis, with normal capillary glucose and oxygen saturation and no dyspnea. He initially had weight loss and reduced visceral fat, but also lost 2.1 kg of skeletal muscle mass, leading to HMB 3 g/day and reinforcement of protein intake. After escalation to semaglutide 0.5 mg weekly, he developed diarrhea, nausea, and vomiting, possibly infectious, requiring temporary discontinuation and reintroduction at 0.25 mg. One month later, he showed improvement in energy, mobility, myalgia, sleep apnea, and emotional status, with weight reduction to 160.9 kg, fat mass to 80.0 kg, visceral fat level to 26, and recovery of skeletal muscle mass to 45.6 kg. A new escalation to 0.5 mg occurred without complications. **Discussion:** PNPLA3-related lipid myopathy is rare and progressive, characterized by intramuscular lipid accumulation and high clinical variability, with limited data on metabolic management. This case suggests potential metabolic and functional benefit of semaglutide in obesity with muscular vulnerability, with reductions in fat mass and visceral fat and functional improvement. The initial skeletal muscle mass loss highlights the need for caution with anti-obesity therapies in myopathies and reinforces serial body composition monitoring, nutritional support, and muscle preservation strategies. **Final comments:** Semaglutide, nutritional support, and targeted supplementation were associated with metabolic, functional, and body composition improvement. This case suggests potential benefit of anti-obesity therapy in genetic myopathies associated with severe obesity, when accompanied by rigorous monitoring and muscle preservation strategies.

Keywords: lipid myopathy; semaglutide; severe obesity.

OBESITY

323 (3340) ARTIFICIAL INTELLIGENCE FOR EARLY PREDICTION OF CARDIOMETABOLIC ALTERATIONS IN PATIENTS WITH METABOLIC SYNDROME USING CLINICAL AND ELECTROCARDIOGRAPHIC VARIABLES

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Introduction: Metabolic syndrome is an important risk factor for cardiovascular morbidity and mortality and is characterized by the association of abdominal obesity, arterial hypertension, glycemic abnormalities, and dyslipidemia. Traditional methods have limitations in early cardiovascular risk stratification. In this context, artificial intelligence (AI) may integrate clinical, laboratory, and electrocardiographic variables to improve the identification of high-risk individuals. **Methods:** A retrospective observational study was conducted using a framework based on data compatible with NHANES III. A total of 2,184 adult individuals meeting criteria for metabolic syndrome were evaluated. Variables analyzed included age, sex, body mass index, waist circumference, blood glucose, glycated hemoglobin (HbA1c), total cholesterol, LDL, HDL, triglycerides, heart rate, and electrocardiographic parameters (PR interval, QRS duration, QT interval, and corrected QT [QTc]). An XGBoost model was employed with an 80/20 training/testing split to predict cardiovascular mortality. **Results:** The mean age was 58.4 ± 12.7 years, with 52.1% being male. The prevalence of hypertension was 71.3%, obesity 63.8%, and diabetes mellitus 34.5%. Cardiovascular mortality was identified in 11.7% of participants. The model achieved an accuracy of 91.2%, sensitivity of 88.5%, specificity of 89.8%, F1-score of 0.89, and an AUC of 0.93. The most relevant variables were HbA1c, age, triglycerides, QTc interval, body mass index, and systolic blood pressure. **Conclusion:** The AI-based model demonstrated high predictive performance for cardiovascular mortality in individuals with metabolic syndrome, showing potential applications in cardiometabolic risk stratification and precision endocrinology.
Keywords: artificial intelligence; metabolic syndrome; endocrinology.

OBESITY

324 (3419) ASSOCIATION BETWEEN TIRZEPATIDE USE AND GREATER REDUCTION OF VISCERAL FAT IN ADULTS WITH OBESITY: A SYSTEMATIC REVIEW

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Introduction: Obesity is a chronic disease associated with insulin resistance, metabolic syndrome, and cardiovascular risk. Visceral adiposity contributes significantly to metabolic dysfunction and inflammation. Tirzepatide, a dual glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1 receptor agonist, has demonstrated important effects on weight loss and body composition. **Objective:** To evaluate whether tirzepatide is associated with greater visceral fat reduction in adults with obesity compared with semaglutide or placebo. **Methods:** A systematic review was conducted according to the PICO strategy. Searches were performed in PubMed, SciELO, and Cochrane Library using the descriptors “Obesity,” “Tirzepatide,” “Semaglutide,” “Visceral Fat,” “Body Composition,” and “Weight Loss.” Randomized clinical trials, prospective studies, and systematic reviews published between 2020 and 2026 in English, Portuguese, and Spanish were included. Eligible studies evaluated adults with obesity treated with tirzepatide compared with semaglutide or placebo and assessed visceral adiposity through imaging or body composition analysis. **Results:** Eighteen studies were included. Tirzepatide promoted greater visceral fat reduction compared with placebo and showed superior outcomes to semaglutide in several studies. SURMOUNT trials demonstrated mean body weight reductions ranging from 15% to 22.5%, accompanied by marked decreases in visceral adiposity assessed by magnetic resonance imaging and dual-energy X-ray absorptiometry. Comparative studies also identified greater reductions in waist circumference and waist-to-hip ratio in tirzepatide-treated groups ($p < 0.05$). Proposed mechanisms include synergistic activation of glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1 receptors, resulting in enhanced satiety, delayed gastric emptying, improved insulin sensitivity, and increased ectopic fat mobilization. The most common adverse effects were mild to moderate gastrointestinal symptoms, including nausea, vomiting, and diarrhea. **Conclusion:** Tirzepatide was associated with greater visceral fat reduction and improved cardiometabolic parameters in adults with obesity compared with placebo and semaglutide, supporting its therapeutic potential in obesity management.
Keywords: obesity; tirzepatide; visceral fat.

OBESITY

325 (2990) ASSOCIATION OF PATERNAL OBESITY WITH NUTRITIONAL STATUS OF OFFSPRING BORN TO MOTHERS WITH GESTATIONAL DIABETES

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Introduction: Gestational diabetes mellitus (GDM) and maternal obesity are associated with obesity in offspring, but the impact of father's obesity on the metabolic health of offspring needs more studies. **Objective:** To evaluate the association of obesity in offspring exposed to GDM in utero with pre-gestational maternal and paternal obesity (BMI ≥ 30 kg/m²). **Methods:** This retrospective cohort study involved 148 children aged 2-14 years, born to mothers with GDM. Data was collected during routine antenatal care and an evaluation of the children were performed 2 to 14 years later. Excessive weight was defined according to WHO criteria. For children ≥ 5 years (n = 88): BMI-Z score ≥+2 = obesity; ≥+1 and <+ 2 = overweight, and for children < 5 years (n = 60): BMI-Z score >+ 3 = obesity; ≥+1 and <+ 3 = overweight. Offspring's overweight/obesity was compared by presence of parental pre-gestational obesity. **Results:** Prevalence of pre-gestational obesity was 49.3% among mothers and 25.7% among fathers. In children aged 2-to-4yrs, overall prevalence of overweight/obesity was 20.0%; being 7.7% when neither parent had obesity, 20.0% when only the father had obesity, 16.7% when only the mother had the condition and 54.5% when both parents were affected (p = 0.013). Among children aged 5-to-14 yrs, overall prevalence of obesity was 33.0%; 21.2% when neither parent had obesity; 36.4% when only the father had obesity; 33.3% when only the mother had the condition; and 63.6% when both parents were affected (p = 0.291). In logistic regression analysis parental obesity (both parents) was associated with obesity in the offspring (OR 6.5 95% CI 2.0 to 21.1, p = 0.002) after adjustment for child sex, parental education level, and the child's diet and screen time. **Conclusion:** There is a gradual increase in the prevalence of overweight/obesity among offspring when one or both parents are affected with pre-gestational obesity. Father's obesity had an influence on children's overweight/obesity, independent of mother's characteristics. These results emphasize the significance of addressing pregestational paternal obesity as a potential modifiable risk factor to reduce cardiometabolic risk in offspring exposed to gestational diabetes in uterus.
Keywords: obesity; gestational diabetes mellitus; risk factors.

OBESITY

326 (3365) BINGE EATING DISORDER IN PATIENTS WITH OBESITY WITHIN THE UNIFIED HEALTH SYSTEM: THE IMPORTANCE OF CLINICAL SCREENING

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Introduction: According to 2023 national data, the prevalence of adult obesity in Brazil reached 24.3%. In weight management, binge eating disorder stands out due to its high prevalence, underdiagnosis, and risk of weight regain. It is clinically defined by the consumption of large amounts of food in a short period of time, accompanied by a sense of loss of control and marked distress, in the absence of compensatory behaviors. A study pointed to an adjusted sample prevalence of 1.4% for the diagnosis of this disorder in the general Brazilian population, a rate that rises significantly in subgroups with severe obesity. Identifying these alterations through screening within the Unified Health System is crucial for multidisciplinary therapeutic guidance. **Objective:** To investigate the prevalence of positive screening for binge eating disorder among patients with obesity in an outpatient endocrinology clinic of the Unified Health System. **Methods:** This is a preliminary cross-sectional, observational, and descriptive study with prospective data collection conducted through face-to-face interviews. The Portuguese-validated version of the Questionnaire on Eating and Weight Patterns-5 was utilized. Positive screening followed the instrument's criteria based on the Diagnostic and Statistical Manual of Mental Disorders, requiring weekly episodes and the absence of compensatory behaviors. **Results:** The final sample consisted of 61 participants, with a predominance of 52 women (85.2%) compared to 9 men (14.8%). Regarding the time since disease diagnosis, 40 individuals presented with more than 10 years of obesity (65.6%). Screening for binge eating disorder identified four positive cases (6.55%), all of whom were female. Among the participants with positive screening, three presented a Body Mass Index greater than 40 (75.0%) and one presented a Body Mass Index of 38 (25.0%), associated with distress classified as very or extremely intense. There were no cases of bulimia nervosa. **Conclusion:** Preliminary analysis evidenced a 6.55% prevalence of positive screening for the disorder within the context of chronic and severe obesity inside the Unified Health System. The continuity of data collection with a consequent sample increase will allow the establishment of more robust statistical associations. In any case, the preliminary results ground the need to implement systematic screening protocols in outpatient practice as a tool for comprehensive care and prevention of weight regain.
Keywords: obesity; binge eating disorder; Unified Health System.

OBESITY

327 (3385) BODY COMPOSITION CHANGES DURING INCRETIN-BASED OBESITY THERAPY: A SYSTEMATIC REVIEW

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Introduction: Incretin-based therapies have substantially changed obesity treatment by producing levels of weight reduction previously observed mainly after bariatric surgery. However, total body weight loss alone incompletely reflects changes in body composition, visceral adiposity, and functional adaptation during treatment. **Objective:** To evaluate body composition changes during incretin-based obesity therapy, with emphasis on adipose tissue reduction, lean mass remodeling, visceral adiposity, and functional outcomes. **Methods:** A systematic review was conducted according to PRISMA recommendations, including randomized and prospective studies evaluating dual-energy X-ray absorptiometry-derived body composition outcomes during incretin-based therapy in adults with obesity, overweight, or type 2 diabetes mellitus. Studies assessing exercise-associated functional outcomes were also analyzed. **Results:** Four studies were included in the primary synthesis: STEP 1 supplementary DXA analysis, SURMOUNT-1 DXA substudy, SUSTAIN 8 DXA substudy, and S-LiTE. Across studies, weight reduction was predominantly attributable to adipose tissue reduction. In the SURMOUNT-1 DXA substudy, tirzepatide reduced body weight by 21.3%, with approximately 74% of total weight loss attributable to fat mass and 26% to lean mass reduction, in addition to a 40.1% reduction in visceral adipose tissue. STEP 1 supplementary DXA analysis demonstrated greater reductions in fat mass than lean mass during semaglutide therapy, with increased relative lean mass proportion despite reductions in absolute lean mass. Similar body composition changes were observed in SUSTAIN 8. In S-LiTE, exercise combined with liraglutide was associated with improved cardiorespiratory fitness and greater long-term weight-loss maintenance compared with isolated interventions. **Conclusion:** Available evidence suggests that incretin-based obesity therapies induce predominantly adipose-driven weight reduction, while lean mass remodeling represents a proportionally smaller component of total weight loss. These findings suggest that body composition and functional parameters may provide clinically relevant complementary information beyond total body weight reduction alone.
Keywords: obesity; body composition; incretin therapy.

OBESITY

328 (3005) BODY COMPOSITION CHANGES DURING TIRZEPATIDE-INDUCED WEIGHT LOSS: A SYSTEMATIC REVIEW WITH DESCRIPTIVE QUANTITATIVE SYNTHESIS

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Introduction: Tirzepatide promotes substantial weight reduction in individuals with obesity and type 2 diabetes mellitus. However, beyond the magnitude of body weight reduction, understanding its effects on body composition has become clinically relevant, particularly regarding fat mass, lean mass and visceral adiposity. **Objective:** To systematically evaluate the effects of tirzepatide on body composition in adults with obesity, overweight and/or type 2 diabetes mellitus. **Methods:** A systematic review was conducted according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 recommendations. Searches were performed in PubMed/MEDLINE, Embase, Scopus, Web of Science, Cochrane CENTRAL and ClinicalTrials.gov until March 2026. Randomized controlled trials, substudies and post-hoc analyses derived from randomized controlled trials evaluating tirzepatide and body composition were included. Due to heterogeneity among assessment methods used in the included studies, including dual-energy X-ray absorptiometry, magnetic resonance imaging and BOD POD, a descriptive quantitative synthesis of body composition outcomes was performed. **Results:** Among 1,379 identified records, six randomized controlled trials met eligibility criteria. The included studies demonstrated consistent reductions in body weight, fat mass, visceral adiposity and waist circumference with tirzepatide treatment. In the SURMOUNT-1 dual-energy X-ray absorptiometry substudy involving 160 participants, treatment with tirzepatide at doses of 5, 10 and 15 mg over 72 weeks resulted in reductions of 21.3% in body weight, 33.9% in fat mass and 10.9% in fat-free mass, compared with reductions of 5.3%, 8.2% and 2.6% observed with placebo, respectively. Approximately 74% of total weight reduction was attributed to adiposity loss. **Conclusion:** Tirzepatide promotes favorable body composition changes primarily through reductions in total and visceral adiposity. Although concomitant reductions in fat-free mass occur, current evidence suggests that weight reduction is predominantly driven by adiposity loss.
Keywords: tirzepatide; body composition; obesity.

OBESITY

329 (3432) CHARACTERIZATION OF THE MOST PREVALENT EATING BEHAVIOR PHENOTYPES IN PATIENTS WITH OBESITY

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Introduction: Obesity is a chronic and multifactorial disease resulting from the interaction of genetic, environmental, and behavioral factors. Tools capable of identifying specific eating behavior patterns have gained relevance in clinical practice, including the Eating Behavior Phenotype Scale (EBPS), used to characterize eating profiles. Classification into emotional, compulsive (C), hedonic (H), disorganized (D), and hyperphagic (HF) phenotypes allows a broader understanding of obesity and supports individualized nutritional, psychological, and pharmacological interventions. **Objective:** To characterize the most common eating behavior phenotypes in patients with obesity. **Methods:** This observational, retrospective, cross-sectional, and descriptive study included adults with obesity (BMI > 30 kg/m²) followed at an endocrinology outpatient clinic who signed informed consent forms. The new Eating Behavior Phenotype Scale (EBPS 2025) was applied, and data were tabulated in Excel spreadsheets and analyzed using IBM SPSS Statistics version 29. **Results:** The sample consisted of 64 patients, 92.2% women and 7.8% men, with a mean age of 53 ± 14 years. Regarding ethnicity, 39.1% self-identified as white, 14.1% as black, and 45.3% as mixed-race. Most participants were non-smokers (71.9%), while 26.6% were former smokers. Alcohol consumption was reported by 39.7%. Duration of obesity diagnosis was <10 years in 46.9%, 10–20 years in 24.2%, and >20 years in 29.0%. Regarding eating behavior phenotypes, 36.0% were classified as D phenotype, 30.6% as E, 16.2% as C, 11.7% as H, and 5.4% as HF. Concerning obesity severity, among individuals with C phenotype, 55.5% had grade III obesity; among E phenotype, 46.6% had grade III obesity; among D phenotype, 48.6% had grade III obesity; among H phenotype, 41.6% had grade I obesity; and among HF phenotype, 40% had grade II obesity and 40% had grade III obesity. **Conclusion:** The findings demonstrated heterogeneity in eating behavior patterns among patients with obesity, with predominance of D and E phenotypes. These results reinforce the importance of phenotypic characterization as a complementary tool in obesity assessment, enabling more individualized therapeutic approaches. The EBPS proved applicable in outpatient practice, although further studies are needed to evaluate associations between eating phenotypes and long-term clinical outcomes.
Keywords: obesity; phenotype; eating behavior.

OBESITY

330 (3358) CORRELATION OF THE TRIGLYCERIDE-GLUCOSE INDEX (TYG) WITH BIOCHEMICAL AND ANTHROPOMETRIC MARKERS IN CHILDREN: PIPA KIDS STUDY

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Introduction: Childhood represents a period of increased biological vulnerability, during which early metabolic alterations may have lifelong repercussions. In this context, the Triglyceride-Glucose (TyG) index has emerged as a promising marker of metabolic risk in children. **Objective:** To evaluate the correlation between the TyG index and biochemical and anthropometric markers in children 4, 5, and 6 years. **Methods:** This was a cross-sectional study conducted with preschool children aged 4 to 6 years, of both sexes, approved by the Research Ethics Committee (Approval: 8.072.978; CAAE: 93796225.3.0000.5147). Data collection included anthropometric variables and biochemical parameters. Weight and height were measured to calculate BMI-for-age and classify nutritional status according to the WHO growth curves. Biochemical parameters were obtained through fasting blood samples. Statistical analyses were performed using SPSS, including normality test and descriptive analysis of the sample. Comparison of mean TyG index values was performed using the Mann–Whitney test, considering the median value of the studied population (8) as the cutoff point. Spearman's correlation analysis was used to assess the relationship between variables, adopting a significance level of $p \leq 0.05$. **Results:** A total of 129 children were included, with a median age of 5 years, and most participants were female (54.3%). The mean BMI percentile was 46.56 ± 30.83, with 21.7% of the participants classified as overweight. In the comparison of TyG index means, the group with higher TyG values presented higher glucose levels (78.44 vs. 82.18; $p = 0.015$), triglycerides (59.41 vs. 110.29; $p < 0.001$), and VLDL-c (11.88 vs. 22.06; $p < 0.001$), as well as lower HDL-c concentrations (48.72 vs. 46.50; $p = 0.039$), compared with the group with lower TyG values. Correlation analysis demonstrated significant relations between the TyG index and glucose ($r = 0.221$; $p = 0.012$), HDL-c ($r = –0.181$; $p = 0.041$), triglycerides ($r = 0.964$; $p < 0.001$), and VLDL-c ($r = 0.964$; $p < 0.001$). No significant correlation was observed between the TyG index and BMI. **Conclusion:** The findings demonstrated correlation between the TyG index and glycemic alterations, as well as an unfavorable lipid profile in preschool children, evidenced by higher triglyceride and VLDL-c concentrations and lower HDL-c levels in the group with higher TyG values. These results reinforce the importance of early monitoring of metabolic risk factors during childhood.
Keywords: child; insulin resistance; lipid metabolism.

OBESITY

331 (3065) EATING BEHAVIOR PHENOTYPES AS PREDICTORS OF WEIGHT LOSS RESPONSE TO SIBUTRAMINE AND TOPIRAMATE IN ADULTS WITH OBESITY

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Introduction: Despite the expansion of pharmacological options for obesity treatment, therapeutic response remains highly variable. Therapeutic selection guided by eating behavior phenotypes has been proposed as a strategy to individualize treatment. However, evidence from real-world clinical settings remains limited. **Objective:** To evaluate the effectiveness of therapeutic selection based on the hyperphagic eating phenotype, identified using the Eating Behavior Phenotypes Scale (EFCA), in adults with obesity treated with sibutramine alone or in combination with topiramate. **Methods:** This retrospective longitudinal observational study was based on the analysis of medical records of adults with obesity and moderate or high hyperphagic phenotype according to the EFCA scale (score ³ 6; BMI > 27 kg/m²). Patients received sibutramine as monotherapy or combined with topiramate and were followed for a minimum period of 12 months. The primary outcome was achievement of ³5% body weight loss. Secondary outcomes included identification of predictors of therapeutic response and evaluation of changes in eating behavior phenotypes throughout follow-up. Statistical analysis included Wilcoxon, Mann-Whitney U, Fisher's exact, and Spearman correlation tests. **Results:** Sixteen patients were included (mean age 34.8 ± 14.1 years; 87.5% female; mean BMI 38.4 ± 5.5 kg/m²). Mean overall percentage weight loss was 5.34% ± 5.91, with statistically significant weight reduction during follow-up (p = 0.0013). The overall therapeutic success rate (³5% weight loss) was 37.5%. Patients with a high hyperphagic phenotype showed a trend toward a higher success rate compared with those with a moderate phenotype (75% versus 25%; p = 0.077). Reduction in total EFCA score was significantly correlated with percentage weight loss (rho = 0.602; p = 0.0135). The hyperphagic domain showed the greatest magnitude of behavioral improvement (Cohen's d = 1.72), with significant reductions also observed in the emotional (p = 0.024) and hedonic (p = 0.038) domains. **Conclusion:** Therapeutic selection guided by the hyperphagic eating phenotype was associated with significant weight reduction and improvement in eating behavior patterns. Higher scores in the hyperphagic component of the EFCA may identify a subgroup potentially more responsive to sibutramine-based therapy, a hypothesis that requires confirmation in prospective studies with larger sample sizes.
Keywords: obesity; sibutramine; topiramate.

OBESITY

332 (3409) EFFICACY AND SAFETY OF SEMAGLUTIDE IN THE TREATMENT OF OBESITY IN ADOLESCENTS: A SYSTEMATIC LITERATURE REVIEW

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Introduction: Pediatric obesity is a chronic and relapsing disease associated with metabolic, cardiovascular, and psychosocial complications. Although lifestyle modification remains the first-line therapy, sustained results are often limited. In this context, glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have emerged as promising therapeutic options, with semaglutide standing out due to its high efficacy in reducing body mass index (BMI) and improving cardiometabolic outcomes in adolescents with obesity. **Objective:** To synthesize current evidence regarding the efficacy, metabolic benefits, and safety of semaglutide in the treatment of obesity in adolescents aged 12 to 18 years. **Methods:** A systematic review was conducted using PubMed, MEDLINE, and Cochrane Library databases. Randomized clinical trials, systematic reviews, and meta-analyses evaluating the use of semaglutide in adolescents with obesity were included. Outcomes related to weight reduction, BMI reduction, and adverse events were analyzed. **Results:** Semaglutide 2.4 mg promoted a mean BMI reduction of up to 16.1% after 68 weeks, compared with 0.6% in the placebo group. Pharmacokinetic studies demonstrated similar efficacy and safety between the 1.7 mg and 2.4 mg doses in this population. Meta-analyses showed semaglutide superiority over other GLP-1 receptor agonists regarding weight loss and BMI z-score reduction. Improvements were also observed in waist circumference, glycated hemoglobin, and lipid profile. Gastrointestinal adverse events occurred in approximately 62% of patients, generally mild to moderate, while cholelithiasis was reported in nearly 4% of cases. Discontinuation studies suggested possible weight regain after treatment withdrawal. **Conclusion:** Semaglutide demonstrates high efficacy in treating adolescent obesity, promoting significant BMI reduction and improvement of metabolic parameters. However, its use is associated with gastrointestinal adverse events and possible risk of cholelithiasis, in addition to evidence of weight regain after treatment discontinuation. Despite promising results, long-term studies are still needed to evaluate safety and potential effects on growth and pubertal development.
Keywords: adolescent; obesity; semaglutide.

OBESITY

333 (3177) ENDOTHELIAL DYSFUNCTION AND VISCERAL FAT: THE ROLE OF BODY COMPOSITION DENSITOMETRY

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Introduction: Obesity is a chronic multisystemic disease with a high prevalence worldwide and constitutes an important risk factor for several diseases, not only cardiovascular diseases. Furthermore, it results in a reduction in life expectancy. Visceral fat is associated with type 2 diabetes, metabolic dysfunction-associated fatty liver disease (MAFLD), and certain malignant neoplasms **Objective:** This study evaluated endothelial function and visceral adipose tissue (VAT), in addition to other obesity parameters, aiming to identify the relationship between body composition studied by DXA and its relationship with endothelial function, investigated by venous occlusion plethysmography (VOP). Low-grade inflammation was also assessed by measuring high-sensitivity C-reactive protein (hs-CRP). **Materials and methods:** Cross-sectional study of 101 adults of both sexes, aged between 18 and 50 years, divided into four body mass index groups: group 1 (BMI, 20–24.9, n = 30), group 2 (BMI, 25–29.9, n = 22), group 3 (BMI, 30–34.9, n = 27) and group 4 (BMI, 35–39.9, n = 22). Endothelial function was assessed using the Hokanson AI6 plethysmograph through analysis of forearm blood flow (FBF) and calculated in ml¹. Body composition was performed using Lunar iDXA DXA, highlighting adiposity parameters, especially VAT, with the Corescan tool. Regarding statistical analysis, SPSS version 25 software was used. Depending on whether the data were parametric or not, comparison tests between groups were performed using ANOVA or Kruskal-Wallis, and correlation tests were performed using Pearson or Spearman. For categorical variables, the c^2 test was used. **Results:** Regarding the comparative aspect between the groups, a decline in flow related to the progression of BMI and adiposity parameters, mainly VAT, was identified. An inverse correlation was observed between arterial blood flow in VOP and TFT (total fat mass), % RFM (regional fat mass), FMI (fat mass index), and VAT (visceral adipose tissue) in DXA. hsCRP values showed a direct correlation with the progression of adiposity and VAT between the groups. **Conclusion:** The decline in endothelial function was associated with the progression of VAT, as well as an increase in inflammation. Body composition DXA demonstrated, in this study, potential for early identification of individuals already at increased cardiovascular risk.
Keywords: DXA; visceral fat; obesity.

OBESITY

334 (3050) EUGLYCEMIC KETOACIDOSIS ASSOCIATED WITH TIRZEPATIDE IN NON-DIABETIC PATIENTS: A SYSTEMATIC REVIEW OF PUBLISHED CASES

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Introduction: Tirzepatide, a dual GIP/GLP-1 receptor agonist, has demonstrated substantial metabolic benefits in obesity and type 2 diabetes. However, emerging reports of euglycemic ketoacidosis in non-diabetic individuals raise concern for a potentially underrecognized metabolic complication. **Objective:** To systematically review published cases of tirzepatide-associated euglycemic ketoacidosis in non-diabetic patients and explore potential pathophysiological mechanisms and clinical warning signs. **Methods:** A systematic review was conducted according to PRISMA guidelines through a structured PubMed database search. The search strategy included the descriptors “tirzepatide,” “euglycemic ketoacidosis,” and related terms. Case reports and case series involving non-diabetic patients with ketoacidosis temporally associated with tirzepatide were included according to predefined eligibility criteria. Studies involving diabetic patients, duplicate reports, and non-original publications were excluded. Eight eligible reports were qualitatively analyzed regarding clinical presentation, precipitating factors, metabolic context, management, and outcomes. **Results:** Eight published reports identified recurrent precipitating factors, particularly marked caloric restriction, gastrointestinal intolerance, dehydration, and rapid weight loss. The findings suggest that pharmacologically induced negative energy balance may contribute to enhanced lipolysis and ketogenesis despite preserved endogenous insulin production. Most patients presented with euglycemia or mild hyperglycemia, potentially delaying recognition. Management included intravenous fluids, carbohydrate replacement, insulin in selected cases, and drug discontinuation, with favorable recovery in most reports. **Conclusion:** Euglycemic ketoacidosis appears to be a rare but clinically significant adverse event associated with tirzepatide use in non-diabetic individuals. Clinicians should maintain a high index of suspicion in patients presenting with gastrointestinal intolerance, rapid weight loss, reduced oral intake, or unexplained metabolic symptoms during therapy
Keywords: tirzepatide; euglycemic ketoacidosis; GLP-1 receptor agonists.

OBESITY

335 (3395) FAMILIAL PARTIAL LIPODYSTROPHY TYPE 2: CAN SEGMENTAL BIOELECTRICAL IMPEDANCE ANALYSIS IDENTIFY ALTERATIONS IN FAT DISTRIBUTION?

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Introduction: familial partial lipodystrophy (FPLD) is a rare heterogeneous condition characterized by partial loss of adipose tissue. FPLD type 2 (FPLD2), also known as Dunnigan syndrome, is the most common monogenic subtype but remains underdiagnosed. Anthropometric assessment, including skinfold measurements and dual-energy X-ray absorptiometry (DXA), constitutes an essential component of diagnosis. However, despite advances in bioelectrical impedance analysis (BIA) technology, no studies have evaluated the discriminatory performance of segmental body fat analysis using BIA as a screening tool for FPLD. **Objective:** To evaluate the discriminatory performance of segmental body fat analysis using BIA in identifying women with FPLD2. **Methods:** Cross-sectional study at a reference center for lipodystrophy care, tertiary hospital, Ceará, Brazil. Weight, height, and segmental body fat analysis using multifrequency bioelectrical impedance analysis (MF-BIA) were assessed in adult women (18–60 years) with genetically confirmed FPLD2 and healthy controls matched by age and BMI. Fat percentages in the arms, legs, and trunk, as well as trunk-to-leg fat ratio, were analyzed. Trunk-to-leg fat ratio (kg) and a derived central-peripheral fat excess index were calculated from segmental MF-BIA parameters. Mann–Whitney test compared groups ($p < 0.05$). ROC curve analyses were performed to assess discriminatory performance. **Results:** Sixteen women with FPLD2 and 34 healthy controls were evaluated. The mean age of the FPLD2 group was 45 ± 13 years, and the mean BMI was 25.2 ± 3.8 kg/m². No significant differences were observed in isolated segmental body fat percentages between groups. ROC curve analyses for trunk-to-leg fat ratio (kg) showed an AUC of 0.606, with sensitivity of 6.3% and specificity of 100% for identifying FPLD2. The derived central-peripheral fat excess index showed an AUC of 0.550, with sensitivity of 12.5% and specificity of 97.1%. **Conclusion:** Segmental MF-BIA demonstrated limited discriminatory performance for identifying women with FPLD2. Nevertheless, this study is the first to investigate MF-BIA-derived segmental fat distribution parameters as a potential screening approach for FPLD2.

Keywords: familial partial lipodystrophy, type 2; body fat distribution; electric impedance.

OBESITY

336 (3193) FROM HYPOGONADISM TO EARLY CARDIOMETABOLIC DISEASE: A CASE REPORT OF A YOUNG ADULT WITH MORBID OBESITY

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Introduction: The association between metabolic disorders, gonadal dysfunction, and hepatic alterations in young patients represents a significant clinical challenge, particularly due to the risk of early cardiovascular disease and reproductive implications. This case report aims to describe a young adult with multiple metabolic comorbidities, unfavorable laboratory progression, and progressive sexual dysfunction, highlighting the risk of early cardiovascular disease and the relevance of male obesity-associated secondary hypogonadism (MOSH) as a marker of cardiometabolic vulnerability. **Case description:** G.G.M., a 23-year-old man (1.84 m, 190 kg) with morbid obesity since childhood, was under cardiology follow-up. He reported insidious onset erectile dysfunction, with progression over the last 12 months. He had stage III systemic arterial hypertension, type 2 diabetes mellitus (DM), mixed dyslipidemia, and hepatic steatosis. Initial evaluation showed glycated hemoglobin (HbA1c) of 10.4%, fasting plasma glucose of 332 mg/dL, and hyperinsulinemia of 95 mU/L, consistent with uncontrolled DM. The lipid profile revealed total cholesterol of 223 mg/dL, triglycerides of 749 mg/dL, and HDL cholesterol of 34 mg/dL. Hormonal evaluation demonstrated borderline low total testosterone of 381 ng/dL, with free testosterone of 76.6 pmol/L. Physical examination revealed acanthosis nigricans and violaceous striae. Semen analysis demonstrated complete azoospermia. Secondary hypogonadism associated with metabolic syndrome was diagnosed. Treatment was initiated with topical testosterone and semaglutide, along with lifestyle modifications. **Discussion:** Obesity expands adipose tissue, which sequesters testosterone and increases aromatase activity, converting it into estrogens. This suppresses LH secretion, impairing spermatogenesis and worsening MOSH. This mechanism, along with advanced type 2 diabetes, creates a bidirectional relationship that amplifies endothelial dysfunction and heightens early cardiovascular risk. **Conclusion:** This case demonstrates that metabolic syndrome acts as a marker of high cardiometabolic vulnerability, reflecting endothelial dysfunction, severe insulin resistance, and early cardiovascular risk. Identification of metabolic syndrome enables early and integrated intervention, reducing cardiovascular risk and reproductive impairment.

Keywords: metabolic syndrome; hypogonadism; cardiovascular risk.

OBESITY

337 (3191) FUNCTIONAL HYPOGONADISM ASSOCIATED WITH OBSTRUCTIVE SLEEP APNEA AND OBESITY: AN INTEGRATED APPROACH WITH CPAP AND TIRZEPATIDE

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Case report: Functional hypogonadism is frequently associated with reversible conditions, such as visceral obesity and obstructive sleep apnea OSA, mediated by chronic inflammation, insulin resistance, and sleep fragmentation and requires a therapeutic approach focused on treating the underlying causes before initiating testosterone replacement. This report describes the case of a 54-year-old man with obesity (BMI 31.3 kg/m²), increased abdominal circumference, dyslipidemia, fatigue, daytime sleepiness, and snoring. Polysomnography confirmed severe OSA, and laboratory tests showed persistently low total testosterone levels (71–115 ng/dL) with low gonadotropins, consistent with functional hypogonadotropic hypogonadism, while pituitary magnetic resonance imaging was normal. The patient also presented with prediabetes (HbA1c 6.2%) and hepatic steatosis. Treatment included continuous positive airway pressure (CPAP) and initiation of tirzepatide therapy with interdisciplinary follow-up, focusing on weight reduction (initial target ≥7%–10%), control of insulin resistance, and dietary adjustment. **Discussion:** Functional hypogonadotropic hypogonadism in the context of obesity and OSA is a reversible condition driven by metabolic and neuroendocrine dysregulation. Visceral adiposity promotes increased aromatization and inflammatory signaling, suppressing hypothalamic GnRH secretion, while OSA contributes via intermittent hypoxia and sleep fragmentation, which trigger oxidative stress through reactive oxygen species (ROS), induce Leydig cell dysfunction, and blunt nocturnal testosterone peaks. Management should prioritize correction of underlying factors. CPAP may improve sleep architecture and partially restore hormonal balance, whereas tirzepatide, a dual agonist of GIP/GLP-1 receptors, induces significant weight loss and metabolic improvement, potentially impacting the hypothalamic–pituitary–gonadal axis. This integrated approach targeting underlying conditions may enable recovery of endogenous testosterone production, thereby avoiding unnecessary testosterone replacement and its associated risks. **Conclusion:** This case highlights the importance of prioritizing weight loss and treatment of OSA in the management of functional hypogonadism. The combination of metabolic optimization with tirzepatide, weight loss, and CPAP therapy may restore endogenous testosterone production, thereby avoiding the need for hormone replacement therapy.
Keywords: hypogonadism; obstructive sleep apnea; obesity.

OBESITY

338 (2995) GLP-1 RECEPTOR AGONISTS AND THEIR EFFECTS ON SARCOPENIA, MUSCLE FUNCTION, AND BODY COMPOSITION: A SYSTEMATIC REVIEW

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Introduction: GLP-1 receptor agonists (GLP-1RAs) and multi-receptor agonists have significantly changed obesity management by promoting substantial weight reduction. However, concerns have emerged regarding the quality of weight loss, particularly the preservation of lean mass and the potential risk of sarcopenia and frailty, especially in older adults. **Objective:** To evaluate the effects of GLP-1RAs on sarcopenia, muscle functionality, and body composition in preclinical and clinical studies. **Methods:** A systematic search was conducted in August 2025 using PubMed, Web of Science, and Embase. A total of 880 original studies were identified. After removing 55 duplicates and 14 non-English publications, 811 records underwent screening. Following title and abstract evaluation, 262 studies were excluded for irrelevance. Full-text assessment excluded 450 reviews, 3 articles without full text, and 76 conference papers, letters, or book chapters, resulting in 20 eligible studies (12 preclinical and 8 clinical). **Results:** In animal models, GLP-1RAs demonstrated dose-dependent effects on body weight, with lower doses maintaining or increasing weight and higher doses inducing weight loss. Improvements in grip strength and muscle fiber cross-sectional area were observed through AMPK/SIRT1 signaling, associated with reduced proteolysis and oxidative stress. In clinical studies, lean mass accounted for approximately 20% to 40% of total weight loss despite marked reductions in visceral adiposity. Real-world evidence suggested a greater decline in lean mass with tirzepatide compared with semaglutide, although accompanied by greater fat loss. Despite reductions in absolute muscle mass, muscle functionality and muscle quality frequently improved due to reduced intramuscular fat accumulation and systemic inflammation. Strategies such as resistance exercise and higher protein intake were consistently associated with better preservation of lean mass. **Conclusion:** GLP-1RAs are highly effective for reducing adiposity and improving metabolic health; however, the associated decline in lean mass requires careful monitoring. Combined nutritional and exercise-based strategies appear essential to preserve muscle functionality and minimize the risk of sarcopenia during treatment.
Keywords: glucagon-like peptide 1 receptor agonists; sarcopenia; body composition.

OBESITY

339 (3331) GLUCAGON-LIKE PEPTIDE-1 RECEPTOR AGONISTS AND RISK OF ARRHYTHMIAS: A NETWORK META-ANALYSIS

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Introduction: Arrhythmias are often associated with comorbidities such as diabetes and obesity, which significantly influence their onset and progression. In this context, glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have garnered attention for their potential benefits, with emerging evidence indicating that GLP-1 receptor activation might offer antiarrhythmic effects. **Objective:** This study aimed to evaluate the impact of all GLP-1 RAs and co-agonists on the incidence of new-onset arrhythmias, particularly atrial fibrillation (AF). **Methods:** A comprehensive search of databases was conducted from inception to December 2025. Eligible studies included randomized controlled trials (RCTs) focusing on patients with diabetes or obesity receiving GLP-1 RAs or co-agonists. Risk ratios (RR) with 95% confidence intervals (CIs) were estimated. Interventions were ranked using the p-score cumulative rank, ranging from 0 (worst) to 1 (best). **Results:** A total of 94 RCTs involving 155,989 patients were included, comparing 12 GLP-1 RAs and co-agonists. Subcutaneous semaglutide emerged as the most effective intervention, significantly reducing AF incidence compared with placebo (RR 0.82; 95% CI: 0.68 to 0.99), and compared with tirzepatide (RR 0.57; 95% CI: 0.37 to 0.88). In contrast, dulaglutide was associated with an increased risk of AF, while lixisenatide was associated with a decreased risk in atrioventricular blocks. For AF, semaglutide presented a p-score of 0.7885, while tirzepatide and dulaglutide scored 0.1830 and 0.1750, respectively. **Conclusion:** Among GLP-1 RAs and co-agonists, subcutaneous semaglutide is highlighted as the most effective intervention for reducing the risk of AF. Further studies focusing on arrhythmic outcomes are needed to confirm these findings.
Keywords: atrial fibrillation; GLP-1; obesity.

OBESITY

340 (2944) HIDDEN OBESITY BURDEN: CENTRAL ADIPOSITY REVEALS PROFOUND UNDERDIAGNOSIS AND UNDERTREATMENT BEYOND BMI IN A BRAZILIAN COMMUNITY SCREENING INITIATIVE

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Introduction: Obesity remains substantially underdiagnosed and undertreated despite its growing burden. Reliance on body mass index (BMI) alone may fail to identify people with central adiposity and high metabolic risk. We evaluated obesity recognition, treatment access, and the prevalence of hidden central adiposity in a Brazilian community screening. **Methods:** Cross-sectional study conducted during an obesity awareness campaign in São Paulo, Brazil. Anthropometric and metabolic data were collected from 619 participants. Obesity was defined as BMI ≥ 30 kg/m². Central adiposity was defined by waist-to-height ratio > 0.5. Participants were evaluated regarding previous obesity diagnosis, anti-obesity medication (AOM) use, bariatric surgery, willingness to discuss obesity, and cardiometabolic comorbidities. Logistic regression models were used to identify factors associated with obesity diagnosis and willingness to discuss weight. **Results:** Mean age was 50.9 ± 28.5 years, 67.1% were female, and mean BMI was 29.7 ± 9.4 kg/m². Obesity prevalence reached 43.5%, while central adiposity was identified in 83.8% of participants. Among people with obesity, only 6.7% reported a previous medical diagnosis of obesity, 6.3% reported current or previous AOM use, and only 0.4% had undergone bariatric surgery. Despite the high burden, only 23.4% of individuals with obesity expressed willingness to discuss their weight. Cardiometabolic comorbidities were frequent among participants with obesity, including diabetes/prediabetes (26.8%), hypertension (39.4%), and dyslipidemia (21.2%). Higher BMI was independently associated with previous obesity diagnosis (OR 1.02 per kg/m², p = 0.042), but sex and age were not. Willingness to discuss obesity was associated with younger age (p = 0.003), diabetes (p = 0.042), dyslipidemia (p = 0.010), and especially central adiposity (OR 10.3, p = 0.002), while BMI itself was not significant. 72% of individuals without obesity by BMI criteria still presented central adiposity, suggesting a substantial hidden metabolic risk not captured by BMI alone. **Conclusion:** This screening revealed profound underdiagnosis and undertreatment of obesity in a high-risk Brazilian population. Central adiposity was markedly more prevalent than BMI-defined obesity and strongly associated with willingness to discuss weight, highlighting the limitations of BMI as the sole screening tool. These findings support broader use of waist-based metrics into obesity screening and health strategies.
Keywords: obesity; community screening; central adiposity.

OBESITY

341 (3550) HIGH PREVALENCE OF POSSIBLE SARCOPENIA IN ELDERLY OUTPATIENTS WITH CARDIOMETABOLIC COMORBIDITIES: A CROSS-SECTIONAL STUDY USING EWGSOP2 CRITERIA

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Introduction: Sarcopenic obesity — the coexistence of excess adiposity and reduced muscle function — was formally recognized as an independent clinical entity by the ESPEN/EASO 2022 consensus. In older adults, age-related body composition changes promote progressive loss of muscle mass and strength alongside increased adiposity, contributing to frailty, disability, and cardiometabolic complications. In elderly individuals with high cardiometabolic burden, reduced handgrip strength is often underrecognized, making BMI-based screening insufficient for early sarcopenia detection. **Objective:** To estimate the prevalence of possible and confirmed sarcopenia in elderly outpatients with high cardiometabolic burden and to identify clinical factors associated with the possible sarcopenia phenotype. **Methods:** Cross-sectional retrospective study (n = 90). Skeletal muscle mass index (SMI) was estimated by the Lee equation. EWGSOP2 criteria were applied: low handgrip force (women <16 kg; men < 27 kg); low SMI (women < 5.5 kg/m²; men < 7.0 kg/m²). Possible sarcopenia was defined as low force with preserved SMI; confirmed sarcopenia required both. BMI differences between groups were assessed by Mann-Whitney U test with effect size r. Sex, age, HbA1c, and vitamin D were also compared between phenotypes. **Results:** Mean age was 76.5 ± 8.3 years; 73.3% female. Cardiometabolic burden was elevated: hypertension 88.9%, dyslipidemia 76.7%, diabetes mellitus 52.2%. Low handgrip force was detected in 77.8% (70/90). Possible sarcopenia was identified in 74.4% (67/90) and confirmed sarcopenia in 3.3% (3/90), indicating that most muscle dysfunction occurs before structural mass loss. Patients with possible sarcopenia had significantly lower BMI than those without sarcopenia (26.3 [IQR 23.7–30.4] vs. 32.4 [IQR 27.0–35.7] kg/m²; U = 361; p = 0.002; r = 0.33), suggesting that lower body weight characterizes the at-risk profile. Sex, age, HbA1c, and vitamin D were not independently associated with the sarcopenia phenotype. **Conclusion:** Nearly three-quarters of elderly outpatients with high cardiometabolic burden presented possible sarcopenia by EWGSOP2 criteria. The predominance of the possible over the confirmed phenotype points to a clinically actionable window for intervention. Lower BMI emerged as the associated marker, underscoring that handgrip force assessment is essential in geriatric metabolic care and cannot be replaced by anthropometric indices alone.

Keywords: sarcopenic obesity; handgrip strength; aged.

OBESITY

342 (3380) HISTOLOGICAL EFFECTS OF GLP-1-BASED THERAPIES IN MASLD/MASH: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH) are increasingly recognized as important causes of progressive liver fibrosis and cirrhosis. Fibrosis stage is one of the main predictors of liver-related outcomes, making histological endpoints particularly relevant. GLP-1-based therapies have demonstrated important metabolic and weight-loss benefits, although their effects on steatohepatitis resolution and fibrosis improvement remain incompletely defined. **Objective:** To evaluate the histological effects of GLP-1-based therapies in adults with biopsy-confirmed MASLD/MASH. **Methods:** A systematic review and meta-analysis of randomized controlled trials was conducted according to PRISMA 2020 recommendations. PubMed/MEDLINE, Embase and Cochrane CENTRAL were searched through May 2026. Eligible studies evaluated GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists in adults with biopsy-confirmed MASLD/MASH or formerly classified NAFLD/NASH. The primary outcomes were resolution of steatohepatitis without worsening fibrosis and improvement of at least one fibrosis stage without worsening steatohepatitis. Random-effects models were used to calculate pooled risk ratios (RRs) and 95% confidence intervals (95% CIs). **Results:** Four randomized controlled trials were included: LEAN, semaglutide phase 2, SYNERGY-NASH and ESSENCE. These studies evaluated liraglutide, semaglutide and tirzepatide, with treatment duration ranging from 48 to 72 weeks. GLP-1-based therapies were associated with higher rates of steatohepatitis resolution without worsening fibrosis (RR 3.19; 95% CI 1.85–5.50; I^2 = 65%). Fibrosis improvement without worsening steatohepatitis was also more frequent among treated participants (RR 1.70; 95% CI 1.37–2.11; I^2 = %). Gastrointestinal adverse events, mainly nausea, vomiting and diarrhea, were the most commonly reported side effects. **Conclusion:** GLP-1-based therapies were associated with improved histological findings in patients with MASLD/MASH. The pooled effect was larger for steatohepatitis resolution than for fibrosis improvement. Longer follow-up studies are needed to determine whether these findings translate into lower rates of cirrhosis progression and liver-related complications.

Keywords: MASLD; GLP-1 receptor agonists; MASH.

OBESITY

343 (3447) HOSPITAL COST ANALYSIS, AVERAGE LENGTH OF STAY, AND ADMISSIONS FOR OBESITY IN PARÁ: AN EPIDEMIOLOGICAL STUDY FROM 2015 TO 2025

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Introduction: Obesity is a chronic disease characterized by the abnormal or excessive accumulation of body fat. According to the World Health Organization, obesity is defined as a BMI equal to or greater than 30 kg/m². The condition is associated with several comorbidities, including cardiometabolic disorders and certain types of neoplasms. In Brazil, its prevalence has been growing rapidly in recent decades, but regional impacts are still poorly explored, especially in the North Region and the state of Pará. **Objective:** To analyze hospital admissions, hospital costs, and average length of stay for obesity across Brazilian regions between 2015 and 2025. **Methods:** This is a retrospective ecological study conducted using data from the SUS Hospital Information System (SIH/SUS), available via Datasus. The number of admissions, hospital costs, average length of hospital stay, and mortality related to obesity were evaluated according to the International Classification of Diseases (ICD-10: E66), stratified by sex and age (between 20 and 79 years) in the northern region of Brazil. **Results:** It was observed that 1,510 people were hospitalized, with total costs amounting to R$ 8,978,038.65. The average value per Hospital Admission Authorization (AIH) was R$ 5,762.54, with an average stay of 3 days. In 2020, the state showed a small number of admissions (56 records), which evolved to over 600 annual cases between 2024 and 2025. This increase of more than 900% in patient volume was accompanied by a drastic rise in hospital costs, which grew from approximately R$ 300,000 annually at the beginning of the decade to over R$ 3.5 million in recent years, totaling an accumulated investment of nearly R$ 9 million during the analyzed period. From a demographic perspective, hospital obesity in Pará is predominantly female, representing approximately 89% of total admissions. Although fewer in number, male patients present indicators of greater severity or complexity, evidenced by a higher average cost per admission and a slightly longer hospital stay compared to women. **Conclusion:** The increase in obesity-related hospitalizations highlights gaps in the management of the pathology within primary care. While the prevalence of the disease is higher among females, the male clinical profile presents a worse prognosis, resulting in longer hospital stays and elevated hospital costs.

Keywords: obesity; hospitalization; health expenditures.

OBESITY

344 (3514) HOSPITALIZATIONS FOR OBESITY IN BRAZIL IN 2025: AN EPIDEMIOLOGICAL ANALYSIS

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Introduction: Obesity is characterized by the excessive accumulation of body fat. More than 20% of the Brazilian adult population was obese in 2019, and it is associated with more than 168,000 annual deaths in the country attributable to excess weight, significantly impacting the health system. **Objective:** To analyze the epidemiological profile of hospitalizations due to obesity and their total costs in Brazil in 2025. **Methods:** This is an observational, cross-sectional, descriptive, and retrospective study with a quantitative approach, using secondary data collected from the SUS Hospital Information System (SIH/SUS). **Results:** 18,799 hospitalizations were recorded, with a rate of 8.80 hospitalizations per 100,000 inhabitants. The Southeast Region presented the highest prevalence, with 7,756 cases (41.3%), followed by the South Region, with 5,901 cases (31.4%). The state of São Paulo stood out with the highest number of cases, totaling 4,006 hospitalizations (21.3%), followed by Paraná, with 2,741 cases (14.6%), Santa Catarina, with 1,966 cases (10.5%), and Minas Gerais, with 1,588 cases (8.4%), while Rondônia presented the lowest incidence, with 54 cases (0.3%). Elective hospitalizations predominated, corresponding to 17,804 cases (94.7%), while emergency hospitalizations totaled 1,087 cases (5.8%). The type of hospitalization was unknown in all records. Individuals aged 40 to 49 were the most affected, with 6,213 hospitalizations (33.0%), followed by the 30 to 39 age group, with 5,836 cases (31.0%), 50 to 59 years, with 3,492 cases (18.6%), and 20 to 29 years, with 2,201 cases (11.7%). A significant predominance of females was observed, with 16,577 hospitalizations (88.2%), while males accounted for 2,222 cases (11.8%) ($p < 0.05$). White individuals were the most affected, with 9,120 cases (48.5%), followed by mixed-race individuals, with 8,278 cases (44%), Black individuals, with 1,140 cases (6.1%), and Asian individuals, with 261 cases (1.4%). Hospitalizations totaled R$ 66,867,297.21 in hospital services and R$ 122,733,681.24 in health services, corresponding to 23.25% and 31.84%, respectively, of the total expenditure on hospitalizations for endocrine, nutritional, and metabolic diseases. **Conclusion:** Hospitalizations for obesity in Brazil predominated in the Southeast Region, among women aged 40 to 49 years. A significant economic impact on the health system was observed, evidenced by the high costs related to hospital and health services.

Keywords: obesity; hospitalization; Brazil.

OBESITY

345 (3189) HYPOGLYCEMIA DURING PREPARATION FOR BARIATRIC SURGERY: INSULINOMA DIAGNOSIS MASKED BY A RESTRICTIVE DIET

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Case presentation: A 44-year-old female patient with obesity, preparing for bariatric surgery and undergoing a restrictive hypocaloric diet, presented with a severe hypoglycemic episode in February 2024, requiring hospital support. She reported episodes of malaise associated with physical activity and alcohol consumption, without classic autonomic symptoms or previous severe hypoglycemic crises. Subsequently, she experienced weight gain. Laboratory investigation demonstrated endogenous hyperinsulinism, characterized by fasting hypoglycemia (45 mg/dL) associated with elevated insulin (48.7 mIU/L) and C-peptide (6.45 ng/mL) levels. Imaging evaluation, including abdominal ultrasound, showed a 1.4 cm hypoechoic lesion at the body-tail transition of the pancreas, suggestive of an insulinoma, in addition to moderate hepatic steatosis. Abdominal magnetic resonance imaging did not clearly demonstrate pancreatic lesions. However, an endoscopic ultrasound confirmed the presence of a 1.4 cm solid pancreatic nodule in the same location. **Discussion:** Insulinoma is a rare cause of hypoglycemia, with an estimated incidence of 1 to 4 cases per million inhabitants/year. The diagnosis is based on Whipple's triad and the presence of hypoglycemia associated with elevated insulin and C-peptide levels. In this case, the restrictive diet in pre-bariatric preparation constitutes an important confounding factor, which may lead to underestimation of hypoglycemia and delay in diagnosis. The absence of exuberant autonomic symptoms suggests adaptation to recurrent hypoglycemia. The discrepant findings in imaging exams reinforce the greater sensitivity of endoscopic ultrasound for the detection of small pancreatic lesions. Furthermore, this case also emphasizes the need to differentiate hypoglycemia related to caloric restriction from organic causes, especially when laboratory findings are incompatible with simple fasting. **Final comments:** Hypoglycemia in patients preparing for bariatric surgery should not be automatically attributed to dietary restriction. The presence of endogenous hyperinsulinism should raise suspicion of insulinoma, even in seemingly explainable clinical scenarios. Early recognition is essential to avoid diagnostic delays and complications.

Keywords: hypoglycemia; insulinoma; bariatric surgery.

OBESITY

346 (2948) IDENTIFICATION OF ERK-INHIBITORS TRIGGERS THERMOGENIC DIFFERENTIATION IN HUMAN WHITE PROGENITOR CELLS

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Introduction: In recent years, the activation of adipose tissue has been regarded as one of the major innovative and promising channels for treating and ameliorating obesity. However, there are limited and fewer commitments to this process in pharmacological approaches in human cells, which have proven to be problematic. **Objective:** This research evaluated the effects of refametinib on thermogenesis in adipose tissue via the ERK signaling axis to attenuate obesity. **Methods:** Using a reporter assay-based screening approach, several small molecules were screened to identify thermogenic function, and among the screened compounds was an identified phytochemical, Refametinib, which can promote thermogenic differentiation. **Results:** The result shows that the pre-treated refametinib caused no cytotoxic effect on the human cells with potential activation of thermogenic functions, and mitochondrial genes as well as enhance functions during differentiation process. We observed decreased in p-ERK signaling axis and activation of ERK via Sphingosine-1-phosphate reverse the UCP1 induction. Furthermore, the thermogenic effect of the Refametinib-mediated signaling axis was investigated, and a computational approach was used to identify the regulatory proteins involved in the refametinib-mediated signaling axis. Refametinib efficiently promoted Ucp1-dependent thermogenesis by stimulating the ERK-MAPK pathway. Furthermore, molecular docking analysis confirmed the direct molecular target of Refametinib. **Conclusion:** These findings suggest that refametinib could be a promising lead compound in the pursuit of a pharmacological approach to promote energy expenditure in the treatment of obesity.

Keywords: refametinib; pharmacological; thermogenic.

OBESITY

347 (3434) IMPACT OF BARIATRIC SURGERY ON MUSCLE FUNCTION IN POSTMENOPAUSAL WOMEN

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Introduction: Menopause and obesity compound muscle and bone loss through estrogen deficiency, insulin resistance, and chronic inflammation, increasing sarcopenia risk. Bariatric surgery promotes sustained weight loss but also reduces skeletal muscle mass, raising concerns about functional decline in this vulnerable population. **Objective:** To compare muscle strength and physical performance between postmenopausal women with obesity who underwent bariatric surgery and those who did not. **Methods:** Analytical cross-sectional study with 108 postmenopausal women: 55 post-bariatric (surgery over one year prior) and 53 non-surgery (body mass index at or above 30 kilograms per square meter). Outcomes included handgrip strength by dynamometry, six-meter gait speed, thirty-second chair-stand test, appendicular skeletal muscle mass and the Baumgartner index (appendicular skeletal muscle mass divided by height squared) by dual-energy X-ray absorptiometry, and sarcopenia screening scores. Significance set at 5%. **Results:** Groups were similar in age (54.80 ± 5.70 versus 56.98 ± 6.07 years; p = 0.133). The post-bariatric group had lower body mass index (30.77 ± 5.72 versus 38.92 ± 5.99 kilograms per square meter; p = 0.001). Right handgrip strength was higher in the post-bariatric group (20.72 ± 4.30 versus 18.80 ± 6.41 kilograms; p = 0.038), as was left handgrip strength (19.85 ± 4.52 versus 17.38 ± 5.72 kilograms; p = 0.004). Gait speed was greater (0.75 ± 0.19 versus 0.67 ± 0.18 meters per second; p = 0.029) and chair-stand repetitions were higher (8.92 ± 2.22 versus 7.51 ± 2.58; p = 0.003) in post-bariatric group, compared non bariatric group. Conversely, appendicular skeletal muscle mass was lower in the post-bariatric group (16.88 ± 2.37 versus 19.32 ± 2.98 kilograms; p = 0.001), as was the Baumgartner index (6.94 ± 1.02 versus 7.86 ± 1.14 kilograms per square meter; p = 0.001). Sarcopenia screening scores did not differ between groups (p = 0.142 and p = 0.642). **Conclusion:** Despite lower appendicular muscle mass and Baumgartner index, post-bariatric women showed superior strength and functional performance. Metabolic improvement and reduced body load likely explain better function with less mass, reinforcing that functional assessment must complement body composition monitoring in this population.
Keywords: bariatric surgery; postmenopausal; sarcopenia.

OBESITY

348 (3222) IMPACT OF THE OPERA SERGIPE PROGRAM ON EXPANDING ACCESS TO BARIATRIC SURGERY IN THE BRAZILIAN PUBLIC HEALTH SYSTEM (SUS): A TEMPORAL ANALYSIS BETWEEN 2015 AND 2025

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Introduction: Obesity is a chronic disease with high global prevalence; in Brazil, the 2024 Vigitel survey estimated a prevalence of 25.7%, representing a major public health impact. Bariatric surgery stands out as an effective strategy for treating obesity and its comorbidities. However, within the Unified Health System (SUS) access to this procedure has historically been marked by low availability and long waiting lists. In 2025, the state of Sergipe implemented the Opera Sergipe program to expand access to elective surgeries, including bariatric procedures. **Objective:** To analyze the temporal evolution of hospitalizations for bariatric surgery in Sergipe between 2015 and 2025 and its association with the implementation of the Opera Sergipe program and improved access to bariatric surgery via SUS. **Methods:** This ecological, retrospective, and quantitative epidemiological study used data from the SUS Hospital Information System (SIH/SUS), available through the Datasus TabNet platform. Hospitalizations for bariatric surgery in Sergipe between January 2015 and December 2025 were analyzed. The annual number of hospitalizations and procedure rate per 100,000 inhabitants were evaluated using population estimates from the Brazilian Institute of Geography and Statistics (IBGE). Bariatric surgeries related to gastric bypass and vertical gastrectomy (sleeve) techniques were included. **Results:** A total of 600 hospitalizations for bariatric surgery were recorded in Sergipe. Of these, 20.8% (n = 125) occurred during the pre-implementation period of the state program (2015–2024), which was characterized by a low annual procedural frequency, with an average of 12,5 surgeries per year and no sustained growth trend. After the incorporation of bariatric surgery into the program in March 2025, a marked increase in procedures was observed, with 475 surgeries performed in that year alone, corresponding to 79.2% of the cumulative total. The annual rate of bariatric surgeries per 100,000 inhabitants rose from values below 1/100,000 in the initial years of the historical series to approximately 20.6/100,000 in 2025. **Conclusion:** The findings suggest an important change in the historical pattern of access to bariatric surgery in Sergipe, demonstrating a strong temporal association between the expansion of a structured public health policy and increased access to surgical obesity treatment via SUS, with potential implications for reducing healthcare inequalities.
Keywords: obesity; bariatric surgery; epidemiology.

OBESITY

349 (3562) LEPTIN RECEPTOR GENE VARIANTS AND METABOLIC PROFILE IN CHILDREN WITH EARLY-ONSET SEVERE OBESITY: A SYSTEMATIC REVIEW

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Introduction: Early-onset severe obesity is strongly associated with metabolic alterations and genetic factors involved in the regulation of appetite and energy balance. The leptin receptor gene has been widely investigated because of its role in the hypothalamic-leptin-melanocortin pathway, which regulates satiety. **Objective:** To evaluate the association between leptin receptor gene variants and metabolic disorders in children with severe obesity. **Methods:** A systematic review was conducted according to PRISMA guidelines, based on the Population, Exposure, Comparison, and Outcome (PECO) strategy, considering obese children, leptin receptor gene variants, individuals without relevant genetic variants, and metabolic alterations. Searches were performed in the PubMed, SciELO, and Virtual Health Library databases, including studies published between 2012 and 2026, using the descriptors "childhood obesity," "LEPR," "leptin receptor," and "metabolic syndrome," combined using the Boolean operators AND and OR. Observational studies involving early-onset obesity and molecular analysis of the leptin receptor gene were included. Anthropometric parameters, blood pressure, fasting glucose, insulin, leptin, and lipid profile were evaluated in the selected studies. **Results:** The studies revealed a high prevalence of metabolic disorders in pediatric populations with severe obesity, including insulin resistance, hypertension, low levels of high-density lipoprotein cholesterol, and hypertriglyceridemia. Regarding molecular findings, the evaluated literature, including Brazilian pediatric cohorts, did not consistently identify variants associated with metabolic profile in the analyzed populations. Six common variants were described (rs1137100, rs1137101, rs1805134, rs8179183, rs1805096, and an insertion/deletion in the 3' untranslated region). In most studies, no statistically significant association was observed between these variants and metabolic profile or degree of adiposity. **Conclusion:** Evidence indicates that pathogenic mutations in the leptin receptor gene are rare causes of obesity in children. The frequency of polymorphisms is similar to that observed in the general population, suggesting that such variations do not determine metabolic comorbidities, which appear to be more closely associated with the degree of excess body fat than with specific genetic defects in the leptin receptor.
Keywords: childhood obesity; leptin receptor; genetic polymorphism.

OBESITY

350 (3115) METABOLIC DISORDERS AND HEPATIC INFLAMMATION ARE ATTENUATED BY TIME-RESTRICTED FEEDING AND COMBINED EXERCISE TRAINING IN MICE SUBJECTED TO SLEEP RESTRICTION AND A HIGH-FAT DIET

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Obesity is a multifactorial disease with a growing global prevalence, closely associated with insulin resistance and metabolic dysfunction-associated steatotic liver disease (MASLD). Factors such as chronodisruption, a high-fat diet (HFD), and sedentary behavior contribute to metabolic dysregulation, systemic inflammation, and ectopic lipid accumulation in the liver. In this study, we investigated the effects of time-restricted feeding (TRF), either isolated or combined with exercise training, on obesity, glycemic homeostasis, and hepatic steatosis in mice subjected to sleep restriction and an HFD. Ten-week-old male C57BL/6J mice (CEUA: 6414-1/2024) were distributed into the following groups: Control (CTL), fed a standard diet; Diet-Induced Obesity (DIO), fed an HFD; Shiftwork (SW), subjected to sleep restriction and an HFD; SW+TRF, subjected to sleep restriction, an HFD, and TRF; and SW+TRF+EX, subjected to sleep restriction, an HFD, TRF, and combined exercise training (CET). Sleep restriction was performed for 12 hours/day during the light cycle, while TRF occurred for 12 hours/day during the dark cycle. The physical exercise protocol included aerobic treadmill training and resistance ladder-climbing exercise, both at moderate intensity. Interventions were conducted five times a week for 10 weeks. Metabolic, physiological, functional, mitochondrial, and molecular parameters were evaluated. The results demonstrated that TRF, either alone or associated with exercise training, attenuated the deleterious effects of an HFD and chronodisruption, reducing body weight gain, adiposity, and both fasting and postprandial glycemia, in addition to improving insulin sensitivity and mitochondrial function. Additionally, a significant reduction in the expression of hepatic inflammatory genes was observed, accompanied by lower progression of hepatic steatosis. The most pronounced effects were observed in the combination of TRF and physical exercise, indicating a synergistic interaction capable of promoting metabolic and hepatic protection against the impacts of sleep restriction and a high-fat diet.
Keywords: MASLD; chronodisruption; mitochondria.

OBESITY

351 (3036) METFORMIN SELECTIVELY MODULATES HEDONIC EATING PHENOTYPES INDEPENDENT OF WEIGHT LOSS: A RETROSPECTIVE EFCA-BASED STUDY

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Introduction: Obesity is a chronic disease in which eating phenotypes may respond differently to pharmacological interventions. Metformin has been shown to modulate neuroendocrine pathways involved in appetite regulation, potentially affecting food reward processing beyond its metabolic effects. However, its impact on specific eating behavior phenotypes independent of weight loss remains unclear. The *Escala de Fenótipos do Comportamento Alimentar* (EFCA), a validated Brazilian Portuguese multidimensional instrument, assesses five eating behavior domains. **Methods:** We conducted a retrospective cohort study in a public obesity outpatient clinic in São Paulo, Brazil, using a structured REDCap database. Adults with obesity (BMI ≥ 30 kg/m²), using Metformin as an add-on therapy for obesity, and complete paired EFCA and anthropometric assessments at baseline and six months were included. EFCA subscales and anthropometric measures were compared between time points using two-sample t-tests. **Results:** 27 patients were included (mean age 48.7 years; 73.6% female; baseline BMI 41.4 kg/m²). No significant changes were observed in body weight (110.9 vs. 107.4 kg; p = 0.57), BMI (p = 0.48), or waist circumference (p = 0.94). Among the five EFCA domains, only the hedonic subscale showed a significant reduction after six months (13.26 vs. 10.76; Δ = –2.50; p = 0.036). A near-significant reduction was observed in compulsive eating (6.12 vs. 4.92; Δ = –1.20; p = 0.064), while emotional, hyperphagic, and disorganized eating domains remained unchanged. **Discussion:** Although statistically significant, the reduction in hedonic eating was small and was not followed by clinically meaningful weight reduction, suggesting that behavioral modulation may have been insufficient in magnitude or duration to produce measurable weight change. Isolated statistical significance among multiple exploratory comparisons raises the possibility of a chance finding. Nonetheless, the selective reduction in reward-driven eating is biologically plausible and may reflect modulation of food reward pathways, potentially involving GDF15 signaling, endogenous incretin activity, or aversive gastrointestinal feedback mechanisms. **Conclusion:** In this exploratory cohort, six months of metformin treatment was associated with a small selective reduction in hedonic eating behavior without significant weight loss. These findings should be considered hypothesis-generating and warrant prospective, adequately powered controlled studies.
Keywords: metformin; obesity; EFCA.

OBESITY

352 (3273) OBESITY AND METABOLIC SYNDROME ARE ASSOCIATED WITH INCREASED SERUM LEVELS OF ENDOCANNABINOIDS AND REDUCED EXPRESSION OF DIACYLGLYCEROL LIPASE (DAGL) AND CANNABINOID RECEPTOR 2 (CB2) IN THE LIVER OF ADULT WOMEN

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The endocannabinoid system (ECS) is a lipid signaling pathway composed of endocannabinoids (eCBs), including anandamide (AEA), 2-arachidonoylglycerol (2-AG), oleoylethanolamide (OEA), and palmitoylethanolamide (PEA) as well as cannabinoid receptors (CB1 and CB2), and metabolizing enzymes such as monoacylglycerol lipase (MAGL) and diacylglycerol lipase (DAGL). ECS is widely distributed in the central nervous system and peripheral tissues, stimulating positive energy balance and weight gain. In adipose tissue and liver, ECS activation promotes lipogenesis and limits fat oxidation. ECS overactivity has been proposed as a contributing factor in obesity and metabolic syndrome (MS). However, studies investigating ECS components in human obesity have shown mixed results. Women (n = 81) and men (n = 7) from Rio de Janeiro (Brazil) presenting severe obesity and undergoing bariatric surgery were recruited as volunteers for this study. Before surgery, we assessed anthropometric measurements, including body mass index (BMI), and waist and neck circumference. On the day of surgery, fasting blood samples were collected for quantification of plasma eCBs by high-performance liquid chromatography — mass spectrometry (HPLC-MS) and hormones (leptin and resistin) by Multiplex. Liver samples were collected for ECS protein evaluation by Western blotting. Clinical data, including hypertension, type 2 diabetes, and serum triglyceride levels were retrieved from medical records to classify participants according to their metabolic profile (obesity with or without MS), and group comparisons were performed using Student's t-test or Mann-Whitney. Linear regression analyses revealed a significant positive association between circulating AEA and leptin levels (p = 0.005), but no association with anthropometric parameters or resistin levels. However, stratification according to metabolic profile revealed higher plasma concentration of AEA (0.73 ± 0.21 ng/mL) and OEA (3.46 ± 0.086 ng/mL) in obese participants with MS compared to those without (0.63 ± 0.20 ng/mL; p = 0.03) and (3.05 ± 0.92 ng/mL; p= 0.04), respectively. Regarding hepatic ECS components, obese women with MS exhibited a lower hepatic content of DAGL and CB2 compared to those without MS– (–30%, p = 0.03 and –35%, p = 0.04, respectively). Together, these findings suggest that alterations in circulating and hepatic ECS components are associated not merely with severe obesity, but rather with the presence of metabolic dysfunction.
Keywords: endocannabinoids; obesity; liver.

OBESITY

353 (3127) OBESITY-RELATED HOSPITALIZATIONS AND BARIATRIC SURGERY IN BRAZIL FROM 2018 TO 2025: A TIME-SERIES ECOLOGICAL STUDY IN THE ERA OF GLP-1 RECEPTOR AGONISTS

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Obesity is a major public health problem associated with increased morbidity, mortality, and healthcare costs. In recent years, glucagon-like peptide-1 (GLP-1) receptor agonists, such as semaglutide and liraglutide, have significantly transformed obesity management by promoting substantial weight loss and metabolic improvement. In Brazil, the dissemination of these therapies intensified after 2021, particularly with the expansion of semaglutide use. This study aimed to analyze temporal trends in obesity-related hospitalizations, bariatric surgeries, and associated hospital expenditures in Brazil between 2018 and 2025. An ecological time-series study was conducted using secondary data from the Hospital Information System of the Brazilian Unified Health System (SIH/SUS). Variables analyzed included obesity-related hospitalizations, number of bariatric surgeries, and approved hospital expenditures for bariatric procedures. Between 2018 and 2019, all indicators increased, with obesity-related hospitalizations rising from 15,051 to 16,474 cases, bariatric surgeries from 12,214 to 13,502 procedures, and hospital expenditures from R$ 51.7 million to R$ 58.2 million. In 2020 and 2021, a marked reduction was observed, coinciding with the COVID-19 pandemic. Hospitalizations decreased to 4,903 in 2020 and 4,432 in 2021, while bariatric surgeries declined to 4,087 and 3,733 procedures, respectively. Hospital expenditures also decreased to R$ 17.2 million in 2020 and R$ 15.4 million in 2021. From 2022 onward, progressive recovery and subsequent growth were observed across all indicators. Obesity-related hospitalizations increased to 18,234 cases in 2025, while bariatric surgeries reached 15,099 procedures. Hospital expenditures also increased substantially, surpassing R$ 100 million in 2024 and 2025. Despite the growing dissemination of GLP-1 receptor agonists after 2021, obesity related hospitalizations and bariatric surgeries continued to increase during the post-pandemic period. These findings suggest that the population level impact of these therapies may remain limited, potentially due to restricted access, elevated treatment costs, and the persistent rise in obesity prevalence nationwide. Additionally, the reduction observed during the COVID-19 pandemic likely reflects the temporary suspension of elective procedures and hospital reorganization.
Keywords: obesity; GLP-1 receptor agonists; bariatric surgery.

OBESITY

354 (3410) OBSTRUCTIVE CHOLESTATIC SYNDROME ASSOCIATED WITH GLP-1 RECEPTOR AGONIST USE IN A YOUNG PATIENT WITH: A CASE REPORT

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Case presentation: A 21-year-old female patient with class III obesity initiated semaglutide at 0.25 mg with progressive dose escalation, achieving a 6-kg weight loss without further reduction. After temporary interruption, the medication was restarted at a 1 mg dose. Approximately three weeks later, she developed recurrent nocturnal right upper quadrant pain radiating to the right shoulder. Eleven days after symptom onset, she presented with choluria, acholic stools, and jaundice. Abdominal ultrasound revealed a distended gallbladder with wall thickening, biliary sludge, microcalculi, and grade I hepatic steatosis, without biliary duct dilatation. Laboratory evaluation demonstrated fluctuating cholestatic liver injury, with total bilirubin ranging from 4.94 mg/dL to 1.84 mg/dL, elevated transaminases, cholestatic enzymes, and C-reactive protein, without elevation of amylase or lipase. Findings were compatible with transient choledocholithiasis likely secondary to spontaneous migration of a microstone. Due to persistent inflammatory findings, the patient underwent laparoscopic cholecystectomy followed by endoscopic retrograde cholangiopancreatography, with subsequent clinical and laboratory improvement. **Discussion:** Although semaglutide prescribing information reports cholelithiasis in approximately 1.6% of patients and progression to cholecystitis in 0.6%, these complications have gained increasing attention because of their potential severity and recurrence. Proposed mechanisms include reduced gallbladder motility, suppression of cholecystokinin release, and bile stasis, favoring microstone formation and migration. These events may result in obstructive biliary disease and severe complications such as acute pancreatitis. Most reported cases occur in women over 45 years of age and are associated with rapid weight loss and higher medication doses. This case is notable because it occurred in a young patient without significant weight reduction and despite appropriate dose escalation, suggesting a possible direct pharmacological effect of semaglutide on biliary dynamics independent of weight loss. **Final comments:** This case highlights the importance of clinical vigilance and nutritional monitoring during GLP-1 receptor agonist therapy, emphasizing early recognition of biliary symptoms and prevention of complications related to cholelithiasis.
Keywords: obesity; GLP-1 receptor agonists; cholelithiasis.

OBESITY

355 (3100) ORFORGLIPRON REDUCED APPETITE AND SUSCEPTIBILITY TO FOOD ENVIRONMENT IN ADULTS WITH OVERWEIGHT AND OBESITY

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Introduction: This analysis evaluated the effect of once-daily orforglipron on appetite and cognitive and emotional responsiveness to food cues in a subset of ATTAIN-1 participants. **Methods:** Adults with BMI ≥ 30 kg/m² or ≥ 27 kg/m² with obesity-related complications and without diabetes, were randomised 3:3:3:4 to orforglipron 6 mg, 12 mg, 36 mg, or placebo, as an adjunct to healthy diet and physical activity up to Week-72. Subjective appetite ratings were assessed using visual analog scale (VAS) and Cognitive and emotional responsiveness to food cues were measured using Power of Food Scale (PFS) with higher scores indicating greater appetite and responsiveness. The VAS and PFS were administered to fasted participants at baseline, Week-24, and Week-72. Results for pooled orforglipron vs. placebo are reported for the efficacy estimand. Model-based estimate differences between orforglipron and placebo were estimated using mixed-model-repeated-measures. **Results:** The analyses included 108 and 102 participants for the VAS and PFS, respectively. Significantly greater decreases in hunger and prospective food consumption were observed for orforglipron vs. placebo at Week-24 ($p < 0.01$), with non-significant differences at Week-72, which may suggest better appetite control with Orforglipron. Orforglipron treatment significantly decreased the PFS overall, food present, and food tasted domain scores vs. placebo at Week-72 ($p < 0.05$), suggesting less responsiveness to proximal foods. **Conclusion:** In adults with obesity, orforglipron significantly improved control of appetite parameters and cognitive and emotional responsiveness to food cues. **Conflict of Interests:** SW has worked with Eli Lilly, Novo Nordisk, Bausch Health Canada, Abbvie, Amgen, Astra Zeneca, Boehringer Ingelheim, Merck, Metsera, NGX, Regeneron, and The Obesity Society. AMC has worked with Boehringer Ingelheim, Eli Lilly and Company, Novo Nordisk, Roche, Roman Health Ventures, Inc, the National Institute of Nursing Research/National Institutes of Health. CH, WY, LG, SK, and NAX are full-time employees and shareholders of Eli Lilly and Company. MX has no competing interests to declare. **Funding:** This study was funded by Eli Lilly and Company.
Keywords: obesity; food noise; overweight.

OBESITY

356 (3262) PREGNANCY AFTER BARIATRIC SURGERY: ASSOCIATION BETWEEN MATERNAL NUTRITIONAL STATUS AND FETAL OUTCOMES IN A TERTIARY HOSPITAL OF THE BRAZILIAN UNIFIED HEALTH SYSTEM (SUS)

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Pregnancy after bariatric surgery has become increasingly frequent among women of reproductive age. Although post-surgical weight loss is associated with important metabolic benefits, potential nutritional deficiencies may impact fetal development. In this context, this study aimed to evaluate the association between maternal nutritional status, endocrinological follow-up, and fetal outcomes in post-bariatric pregnant women followed at a tertiary public hospital within the Brazilian Unified Health System (SUS). A retrospective chart review was performed, including 42 pregnancies after bariatric surgery. Clinical, surgical, laboratory, and ultrasonographic data were collected. Mean maternal age was 34.8 ± 4.9 years, and mean pregestational body mass index was 35.4 ± 9.6 kg/m². Gastric bypass was the most frequent procedure (73.8%). Vitamin D and vitamin B12 deficiencies were highly prevalent at the first consultation (52.4% and 42.9%, respectively) and remained frequent at the end of pregnancy. The prevalence of small-for-gestational-age (SGA) fetuses was 11.9%, whereas large-for-gestational-age (LGA) fetuses accounted for 7.1% of cases. No significant association was observed between micronutrient deficiencies, type of surgery, or interval between surgery and pregnancy and the outcome of fetal growth restriction. On the other hand, maternal weight showed a moderate positive correlation with fetal abdominal circumference ($r = 0.544$; $p = 0.003$), and pregestational weight was also correlated with fetal growth parameters ($r = 0.490$; $p = 0.008$). These findings suggest that, in this population, maternal anthropometric factors may exert greater influence on fetal growth than micronutrient status alone. Endocrinological follow-up remains essential for the nutritional and metabolic monitoring of these patients, and studies with larger sample sizes are needed to better elucidate these associations.
Keywords: bariatric surgery; pregnancy; obesity.

OBESITY

357 (3144) PREVALENCE OF HOSPITALIZATIONS DUE TO ENDOCRINE DISEASES THAT HAD OBESITY AS A MORBIDITY ACROSS THE REGIONS OF BRAZIL 2015–2025

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Introduction: Obesity is a chronic and multifactorial condition that plays an important role in the onset and progression of several endocrine diseases, such as type 2 diabetes mellitus and metabolic syndrome. Its growing prevalence has become a major challenge for healthcare systems worldwide, both because of its direct effects on health and its contribution as a comorbidity that can aggravate other medical conditions. In Brazil, evaluating hospitalization patterns related to obesity in different regions is essential for supporting public health policies and improving the distribution of healthcare resources. **Objective:** This study aims to analyze the prevalence of hospitalizations due to endocrine diseases that had obesity as a comorbidity between 2015 and 2025 across the regions of Brazil. Materials and **Methods:** This epidemiological study adopts a quantitative approach, utilizing data sourced from Datasus. Data were accessed at "Hospital Morbidity of SUS (SIH/SUS)". Filters applied included: "ICD-10: IV, Nutritional and Metabolic Endocrine Diseases"; "ICD-10 morbidity list: Obesity"; from 2015 to 2025. Estimated population data were extracted from IBGE. **Results:** From 2015 to 2025, a total of 131.466 hospital admissions were recorded for complications in endocrine diseases with obesity as a comorbidity in Brazil. The South (55,993) and Southeast (53,199) regions together accounted for 83% of all hospitalizations, while the Northeast (14,460), Midwest (5,012), and North (2,802) represented only 17%. When analyzing rates per 100,000 inhabitants, the South (179) showed a rate six times higher than the Midwest (30), seven times higher than the Northeast (25.3), and twelve times higher than the North (15). Although the Southeast had an absolute number of hospitalizations similar to the South, its much larger population resulted in a lower relative rate (59). **Conclusion:** The findings demonstrate marked regional disparities in hospitalizations due to endocrine diseases associated with obesity in Brazil. After adjusting for population size, the South presented the highest rate, indicating a proportionally greater burden of obesity-related endocrine complications compared with other regions. These results highlight the importance of strengthening regional public health strategies focused on obesity prevention and management of endocrine diseases, in addition to improving healthcare resource allocation according to regional epidemiological demands.
Keywords: endocrine diseases; obesity; morbidity.

OBESITY

358 (3266) REAL-WORLD EFFECTIVENESS AND SAFETY OF LIRAGLUTIDE IN INDIVIDUALS WITH CLASS IV–V OBESITY WITHIN A PUBLIC HEALTHCARE SYSTEM

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Obesity remains one of the most challenging chronic diseases managed in public healthcare systems. Individuals with class IV and V obesity are frequently excluded or underrepresented in randomized clinical trials evaluating anti-obesity pharmacotherapy, leaving a critical gap in evidence for patients with extreme adiposity and severe cardiometabolic disease. This study evaluated the real-world effectiveness and safety of liraglutide 3.0 mg in adults with class IV and V obesity. This retrospective observational study included adults with BMI ≥ 45 kg/m² who initiated liraglutide through an institutional dispensing program at a public hospital in Rio de Janeiro, Brazil, between September 2023 and April 2025. Outcomes were assessed at 6 and 12 months using intention-to-treat (ITT) analysis with mixed models for repeated measures and per-protocol (PP) analysis using paired Wilcoxon tests. The primary outcome was percentage and absolute weight reduction. Secondary outcomes included fasting glucose and glycated hemoglobin (HbA1c). A total of 244 participants were included in the 6-month ITT analysis and 230 in the 12-month ITT analysis; corresponding PP populations included 195 and 126 individuals, respectively. Participants had marked baseline obesity severity, with mean BMI of 53.5 ± 9.1 kg/m² and high prevalence of hypertension (81.1%), dyslipidemia (70.1%), and type 2 diabetes (43.9%). In the ITT analysis, mean weight reduction was 5.1% at 6 months and 5.4% at 12 months (both $p < 0.0001$), corresponding to approximately 7–8 kg loss. In the PP analysis, weight reduction reached 7.2% at 12 months, corresponding to approximately 10 kg loss ($p < 0.0001$). Glycemic parameters improved, particularly among individuals with type 2 diabetes, with HbA1c reductions of about 1 percentage point. Adverse events were predominantly mild gastrointestinal symptoms occurring mainly during the first 6 months. No serious adverse events or deaths were reported. In a highly vulnerable population with obesity class IV and V treated within a public healthcare system, liraglutide 3.0 mg demonstrated clinically meaningful and sustained weight reduction with significant glycemic improvement and a favorable safety profile. Despite the real-world design, weight reduction numerically approached that reported in randomized clinical trials. These findings reinforce the feasibility of pharmacological obesity treatment in patients traditionally underrepresented in clinical trials.
Keywords: liraglutide; class IV obesity; real-world study.

OBESITY

359 (3300) REPRODUCTIVE PLANNING PROFILE OF PREGNANT WOMEN WHO UNDERWENT BARIATRIC SURGERY

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Introduction: Obesity is a global public health challenge that directly contributes to the increasing number of women of reproductive age undergoing bariatric surgery (BS). Although BS improves metabolic conditions, pregnancy after BS requires appropriate preconception care and reproductive planning to mitigate maternal-fetal risks and ensure adequate nutritional status. **Objective:** To evaluate knowledge regarding reproductive planning among pregnant women with a history of BS. **Methods:** This analytical observational study collected data through questionnaires administered to pregnant women followed at a public maternity hospital by a specialized team. **Results:** A total of 21 pregnant women with a history of BS were evaluated. The mean age was 33.6 years, and 52.4% were in the third trimester of pregnancy. Gastric bypass was the most frequently performed surgical technique (66.7%), followed by sleeve gastrectomy (28.6%), while 4.8% of participants were unaware of the procedure performed. The mean interval between BS and pregnancy was 63.9 months; 20% conceived within 12 months after surgery, whereas 65% conceived after 18 months. Overweight was the most frequent nutritional status (36.8%), followed by class I obesity (21.1%), class II obesity (15.8%), and normal weight (26.3%). Unplanned pregnancy was reported by 80% of participants. Contraceptive use after BS was reported by 42.1% of women, including oral contraceptive pills (50%), intrauterine devices (50%), and injectable hormonal contraceptives (37.5%) during the postoperative period. Only 30% reported folic acid supplementation prior to pregnancy. Half of the participants reported endocrinology follow-up before pregnancy; however, only 33.3% stated that the ideal timing for conception after BS had been discussed during medical consultations. **Conclusion:** This study demonstrated that women undergoing BS and receiving care through the Brazilian public healthcare system present high rates of unplanned pregnancies and limited counseling regarding the safest timing for conception after surgery. Additionally, there was a high prevalence of oral contraceptive use, despite the potential impairment of its effectiveness following BS, suggesting insufficient counseling regarding effective contraceptive methods. Therefore, educational strategies aimed at improving preconception counseling and adequate supplementation are necessary to reduce nutritional and obstetric risks in this population.

Keywords: family planning; bariatric surgery; obesity.

OBESITY

360 (3390) RETATRUTIDE: IS THERE EVIDENCE OF THERAPEUTIC SUPERIORITY OVER TIRZEPATIDE? AN INTEGRATIVE REVIEW OF THE CURRENT EVIDENCE

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Introduction: Obesity and cardiovascular-kidney-metabolic (CKM) syndrome represent major public health challenges due to their high morbidity and mortality. Tirzepatide (a dual GLP-1/GIP receptor agonist) and retatrutide (a triple GLP-1/GIP/glucagon receptor agonist) have emerged as promising therapeutic advances in the management of cardiometabolic diseases. **Objective:** To evaluate whether retatrutide offers superior metabolic and cardiorenal benefits compared to tirzepatide in the management of obesity and CKM syndrome. **Methods:** This integrative review was conducted following the PICO framework and included searches in PubMed/MEDLINE, Embase, Scopus, Web of Science, LILACS, and Cochrane Library databases (Jan 2022–Apr 2026). Randomized clinical trials, observational studies, and secondary analyses evaluating metabolic outcomes (weight loss and glycated hemoglobin – HbA1c), cardiovascular, and renal outcomes (estimated glomerular filtration rate — eGFR, albuminuria, and CKD progression) were included. Study selection followed the PRISMA 2020 guidelines, and methodological quality was assessed using the RoB 2 and ROBINS-I tools. **Results:** A total of 14 studies were included. Tirzepatide demonstrated consolidated evidence of clinical efficacy, with weight reduction of up to 22.5% at 72 weeks (SURMOUNT-1), HbA1c reduction ranging from 1.5% to 2.3%, a 38% reduction in HFpEF decompensations (SUMMIT), and non-inferiority for MACE (SURPASS-CVOT). Retatrutide, evaluated in phase 2/3 studies (TRIUMPH-4), achieved superior weight loss (up to 28.7% at 68 weeks) and HbA1c reduction of up to 2.0% in patients with type 2 diabetes, potentially related to the additional metabolic effect of glucagon receptor agonism. Indirect comparisons suggest greater metabolic efficacy of retatrutide; however, with a higher incidence of gastrointestinal adverse events. Long-term cardiovascular and renal outcomes remain limited. **Conclusion:** Retatrutide demonstrates potential metabolic superiority over tirzepatide, with greater magnitude of weight loss and HbA1c reduction of up to 2.0% in patients with type 2 diabetes, possibly related to its additional glucagonergic action. However, the lack of completed head-to-head studies (TRIUMPH-5 ongoing) and limited robust evidence on long-term cardiorenal outcomes prevent definitive confirmation of clinical superiority. Direct comparative trials with extended follow-up are needed to define the therapeutic positioning of these molecules.

Keywords: retatrutide; tirzepatide; cardiometabolic syndrome.

OBESITY

361 (3140) SEMAGLUTIDE FOR REFRACTORY POST-BARIATRIC HYPOGLYCEMIA AFTER ROUX-EN-Y GASTRIC BYPASS: A CASE REPORT

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Case presentation: A 40-year-old female with a history of grade III obesity underwent Roux-en-Y gastric bypass in 2018, achieving significant postoperative weight loss. Approximately four years after surgery, she developed recurrent postprandial hypoglycemic episodes arising within 15 minutes of carbohydrate ingestion. During a subsequent pregnancy, she gained 30 kg, attributed to frequent eating adopted to prevent hypoglycemic episodes. Insulinoma and nesidioblastosis were excluded at a specialized referral center, establishing a diagnosis of functional post-bariatric hypoglycemia. Physical examination revealed height of 165 cm, weight of 83 kg, and body mass index of 30.4 kg/m². Prior therapeutic attempts, including dietary modification with carbohydrate restriction and acarbose, failed to achieve satisfactory glycemic control. **Discussion:** Post-bariatric hypoglycemia is a recognized complication of Roux-en-Y gastric bypass, driven by accelerated gastric emptying and rapid intestinal glucose absorption, triggering exaggerated glucagon-like peptide-1 release and disproportionate postprandial hyperinsulinemia. Management follows a stepwise approach, beginning with dietary modification and progressing to pharmacological agents such as acarbose, diazoxide, and octreotide; however, a substantial proportion of patients remain refractory. Semaglutide, a long-acting glucagon-like peptide-1 receptor agonist, may attenuate the postprandial incretin surge through chronic receptor activation, glucose-dependent insulin modulation, and delayed gastric emptying. Subcutaneous semaglutide was introduced at 0.25 mg per week and progressively titrated to 1 mg per week over one year, in addition to standard post-bariatric supplementation. **Final comments:** After three months of treatment, the patient reported approximately 80% reduction in the frequency and intensity of hypoglycemic episodes. After one year, she was completely asymptomatic with unrestricted dietary intake. Bioimpedance analysis demonstrated improved body composition with increased fat-free mass, supported by regular resistance exercise. This case reinforces emerging evidence that semaglutide may represent an effective therapeutic alternative for refractory post-bariatric hypoglycemia, highlighting the need for prospective studies to consolidate its role in this clinical setting.

Keywords:
post-bariatric hypoglycemia; semaglutide; Roux-en-Y gastric bypass.

OBESITY

362 (3083) STRATEGIES FOR WEIGHT LOSS IN BREAST CANCER SURVIVORS: A SYSTEMATIC REVIEW OF BEHAVIORAL INTERVENTIONS AND INCRETIN-BASED THERAPIES

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Introduction: Obesity is associated with the development and progression of at least 13 types of cancer, including breast cancer. Among breast cancer survivors with obesity, poorer metabolic and functional outcomes have been reported. Therefore, behavioral and pharmacological interventions have been investigated to optimize obesity management in the post-cancer setting. **Objective:** To investigate, in the national and international literature, behavioral and pharmacological strategies for weight loss in breast cancer survivors, emphasizing safety and efficacy. **Methods:** This study is a systematic literature review conducted from January to May 2026, including articles published between 2021 and 2026 from the Virtual Health Library and PubMed databases, in accordance with the recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). The guiding research question was developed using the PICO strategy (population, intervention, comparison, and outcomes), followed by descriptor selection, establishment of eligibility criteria, study screening using Rayyan software, and synthesis of the findings. **Results:** Thirty studies were included after the exclusion of 128 articles due to thematic inadequacy, 58 for not meeting eligibility criteria, and 17 duplicates. Multimodal strategies involving dietary interventions, physical activity, and longitudinal follow-up predominated (46.67%; n = 14). Nutritional interventions, particularly the Mediterranean diet, were identified in 20.00% of the studies (n = 6), whereas incretin-based therapies were evaluated in 30.00% (n = 9). However, only 16.67% (n = 5) specifically assessed these medications in oncology patients or breast cancer survivors. Overall, behavioral interventions were associated with improvements in anthropometric and metabolic parameters, as well as quality of life among breast cancer survivors. In addition, studies with limited evidence demonstrated promising results regarding sustained weight loss and metabolic control with incretin-based therapies, although concerns related to sarcopenia risk were reported. **Conclusion:** Behavioral strategies remain the cornerstone of obesity management in breast cancer survivors. Incretin-based therapies appear promising; however, evidence in oncology populations remains limited. Therefore, prospective studies are needed to further evaluate the applicability, efficacy, and safety of these therapies for weight management after breast cancer.

Keywords: obesity; breast cancer survivors; incretin mimetics.

OBESITY

363 (3199) TRENDS IN HOSPITALIZATIONS FOR LIVER DISEASES IN BRAZIL AND THEIR ASSOCIATION WITH OBESITY: AN EPIDEMIOLOGICAL ANALYSIS FROM 2012 TO 2021

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Introduction: Liver diseases represent an important cause of morbidity and mortality, with an increasing impact on healthcare systems. Obesity has emerged as a relevant factor associated with the development of these conditions. **Objective:** To analyze the temporal trend of hospitalizations for liver diseases in Brazil from 2012 to 2021 and their association with obesity prevalence. **Methods:** This ecological time-trend study used secondary data on hospitalizations for liver diseases (ICD-10) in Brazil obtained from Datasus between 2012 and 2021, stratified by federal units. Monthly records were aggregated annually and converted into rates per 100,000 inhabitants based on IBGE population estimates. Obesity prevalence data were obtained from Vigitel and included adults living in Brazilian state capitals. Descriptive analyses included frequencies, rates, temporal trends, and time-series comparisons. Spearman's correlation coefficient was used to assess the monotonic association between the variables because of the non-parametric distribution of the data. The study period was defined according to the availability of complete and consolidated information. Limitations include possible underreporting, inconsistencies in records, lack of etiological stratification of liver diseases, and ecological bias and limited representativeness due to the use of aggregated data and obesity information restricted to capital cities. **Results:** An increasing trend in hospitalization rates for liver diseases in Brazil was observed between 2012 and 2019, rising from 20.6 to 24.8 hospitalizations per 100,000 inhabitants. In 2020, the rate decreased to 22.3, followed by an increase in 2021 (23.8), although remaining below the pre-pandemic peak. During the same period, obesity prevalence increased continuously, rising from 17.4% in 2012 to 22.5% in 2021, according to Vigitel data. Similar temporal patterns were observed for both variables, with progressive growth interrupted only in 2020. Statistical analysis demonstrated a strong correlation ($r = 0.78$; $p < 0.008$) between obesity prevalence and hospitalization rates for liver diseases, suggesting an association between increasing obesity prevalence and rising hospitalization rates for these conditions. **Conclusion:** The findings suggest an association between increasing obesity prevalence and hospitalizations for liver diseases, reinforcing the importance of obesity control in disease prevention.

Keywords: liver diseases; obesity; epidemiology.

OTHERS

364 (3287) A CASE REPORT: RANOLAZINE AND MEDICATION-INDUCED HYPOGLYCEMIA

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Case presentation: An 82-year-old male patient with a history of hypertension and coronary artery disease, using Vonoprazan, Edoxaban, Losartan and Ranolazine only. presented with recurrent episodes of weakness, dizziness, disorientation, and loss of consciousness, with symptom improvement after ingestion of sugar water. Symptoms began in 2024, with reported hypoglycemic episodes reaching levels as low as 30 mg/dL, consistent with Whipple's triad. In May 2026, he was admitted to the emergency department following a syncopal episode associated with severe hypoglycemia (12 mg/dL) and depressed level of consciousness lasting approximately two hours. During hospitalization, he continued to experience recurrent symptomatic hypoglycemic episodes. Simultaneous fasting glucose, insulin, and C-peptide measurements were obtained during a hypoglycemic episode and prior to glucose administration, revealing glucose of 49 mg/dL, C-peptide of 4.38 ng/mL, and insulin of 23.13 µIU/mL, consistent with endogenous hyperinsulinemia. Etiological investigation included abdominal computed tomography and magnetic resonance imaging, both of which showed no significant abnormalities capable of explaining the condition. During medication review, the use of Ranolazine was considered. The drug had been initiated approximately two years earlier, temporally correlating with symptom onset. Following discontinuation of the medication, a progressive reduction in hypoglycemic episodes was observed, allowing complete withdrawal of continuous glucose infusion without recurrence of hypoglycemia in two days. **Discussion:** Ranolazine is an antianginal agent that acts on cardiomyocyte sodium channels through inhibition of the late sodium current, thereby reducing intracellular calcium overload and myocardial oxygen consumption. By decreasing intracellular calcium overload, it may protect pancreatic beta cells, increase insulin sensitivity, and influence glucose homeostasis. The persistence of hypoglycemic episodes in the absence of associated structural abnormalities, together with clinical improvement after drug withdrawal, reinforces the hypothesis of Ranolazine-induced hypoglycemia. **Final comments:** This case highlights the importance of detailed pharmacological investigation in patients with hyperinsulinemic hypoglycemia without an evident etiology, especially in polymedicated elderly individuals. Furthermore, it indicates a possible association between Ranolazine and symptomatic endogenous hyperinsulinemia.

Keywords: ranolazine; hypoglycemia; hyperinsulinism.

OTHERS

365 (3110) ACRYLAMIDE INTAKE DURING LACTATION AFFECTS METABOLIC AND HORMONAL PARAMETERS IN WISTAR RAT DAMS AND THEIR OFFSPRING

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Acrylamide (AA) is a toxic compound found in many foods prepared at high temperatures, such as French fries, coffee, and breads. This substance promotes oxidative stress and cellular dysfunction, which may be especially deleterious at early life. This is a concern once AA can be found in the breastmilk, and AA sources are widely consumed by breastfeeding women without any recommendation. Therefore, here, we investigated the impact of maternal acrylamide intake during lactation period, in a safe dose, on biometric, biochemical and hormonal parameters of dams and their offspring. For this, lactating Wistar rats received vehicle (drinking water) or acrylamide solution by gavage at dose of 1 or 0.04 mg/kg/day. These are safe doses, corresponding to "no observed adverse effect level" (NOAEL) and "tolerable daily intake" (TDI), respectively. At birth, litters were adjusted to 8 pups by dam. We evaluated body mass, adiposity, and serum cholesterol, triglycerides and hormones. At adulthood, we performed an oral glucose tolerance test (OGTT) and a food preference test. Data were analyzed by one way ANOVA followed by Dunnett's post-test, comparing NOAEL or TDI groups with control group. Protocol approved: 012/2024. At end of lactation period, dams from NOAEL group showed higher brown adipose tissue mass (+31%), higher triiodothyronine levels (+47%) and lower corticosterone levels (-17%), without changes in body mass. On the other hand, dams from TDI group showed higher leptin levels (+159%) and higher triglycerides levels (+50%), without changes in body mass and adiposity. At weaning, although unchanged body mass and serum cholesterol levels in offspring from both sexes, in TDI group, only female offspring showed lower serum triglyceride levels (-29%) while only male offspring showed higher retroperitoneal adipose tissue mass (+52%). Regardless the maternal acrylamide dose, male offspring showed lower adrenal mass (-22% in the NOAEL group and - 27% in the TDI group), without changes in the corticosterone levels. In adulthood, the offspring did not show changes in body mass, oral glucose tolerance test or in the food preference test. Therefore, even in a safe dose, acrylamide is able to affect metabolic and hormonal parameters of lactating dams, impacting their offspring metabolism at weaning. We intend to evaluate the adult offspring to understand the long-term effects of the acrylamide exposure during lactation on the health of the offspring.

Keywords: acrylamide; lactation; metabolism.

OTHERS

366 (3208) ADVERSE EFFECTS OF ORAL SEMAGLUTIDE VERSUS SUBCUTANEOUS GLP-1 AGONISTS: A RETROSPECTIVE STUDY

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Introduction: Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have transformed the treatment of type 2 diabetes mellitus and obesity, promoting glycemic control, weight reduction, and cardiovascular benefits. The introduction of oral semaglutide represented a therapeutic advancement by enabling non-injectable administration through co-formulation with SNAC. However, pharmacokinetic differences compared to subcutaneous formulations may influence the adverse event profile. **Methods:** This study constitutes a retrospective pharmacovigilance analysis based on the FDA Adverse Event Reporting System (FAERS) database, covering 2019 Q4–2025 Q4, comparing the safety profile of oral semaglutide against subcutaneous GLP-1 receptor agonists and non-incretin agents. Signal detection was based on disproportionality analysis through the simultaneous concordance between the Reporting Odds Ratio (ROR), Proportional Reporting Ratio (PRR), and the Information Component (IC). Causal inference triangulation was applied via Propensity Score Matching (PSM), Inverse Probability of Treatment Weighting (IPTW), and Entropy Balancing (Ebal). **Results:** The final cohort comprised 692,874 patients, of whom 13,191 were users of oral semaglutide. The crude disproportionality analysis identified that the oral formulation was associated with a significant increase in the reporting of hepatobiliary, renal, respiratory, and cardiovascular events. The causal triangulation succeeded in matching the cohorts. After adjustment, primary events such as acute kidney injury maintained significance, while other respiratory signals such as dyspnea were attenuated. The volcano plot evaluation differentiated the magnitude of the risk: paraclinical reactions were more pronounced than widely reported symptoms, albeit with a lower causal association. Finally, the global mapping of the classes found that the oral formulation drives a systemic and atypical dispersion of adverse symptoms. **Conclusion:** The results demonstrate that oral semaglutide presents a distinct safety profile from the subcutaneous formulation, with a higher frequency of renal, respiratory, hepatobiliary, and cardiovascular signals, which persist even after causal adjustments and sensitivity analyses. The data reinforces the importance of continuous pharmacovigilance and individualized risk-benefit assessment in the clinical use of Oral Semaglutide.

Keywords: GLP-1 agonists; adverse effects; retrospective.

OTHERS

367 (3071) ASSOCIATION BETWEEN GESTATIONAL METABOLIC ALTERATIONS AND DELAYED LACTOGENESIS II: A SYSTEMATIC REVIEW OF THE LITERATURE

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Introduction: Insulin resistance and chronic metabolic inflammation, which are pathophysiological bases of obesity and gestational diabetes mellitus (GDM), may alter mammary gland function and interfere with lactogenesis II activation, making it relevant to understand the association between GDM, obesity, and delayed lactogenesis. **Objective:** To analyze the association between gestational diabetes mellitus, maternal obesity, and delayed lactogenesis II. **Methods:** Systematic review of the literature based on the PICO strategy. The search was conducted in the PubMed, Medline, and LILACS databases using the descriptors “Gestational Diabetes”, “Obesity”, “Overweight”, “Lactation”, and “Delayed Lactogenesis”. A total of 597 articles were identified. After applying a five-year time filter and excluding reviews, books, randomized clinical trials, meta-analyses, and theoretical analyses, the remaining studies were evaluated regarding thematic relevance and methodological quality. In the end, 24 articles were included. **Results:** Eight of the 24 studies demonstrated an association between gestational diabetes mellitus and a higher risk of delayed lactogenesis II. Another 8 studies identified overweight and maternal obesity as factors related to delayed secretory activation. In addition, 19 studies (79.1%) associated elevated body mass index with lower probability of early breastfeeding initiation, lower rates of exclusive breastfeeding, and poorer maintenance of lactation. **Conclusion:** Gestational diabetes mellitus, overweight, and maternal obesity were associated with delayed lactogenesis II and unfavorable breastfeeding outcomes. These findings reinforce the role of insulin resistance and metabolic inflammation in mammary secretory dysfunction.

Keywords: gestational diabetes mellitus; obesity; lactogenesis.

OTHERS

368 (2918) ASSOCIATION BETWEEN REDUCED LEAN MASS AND HEPATIC FIBROSIS IN INDIVIDUALS WITH METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE

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Introduction: Reduced lean mass has emerged as a potential risk factor for adverse outcomes in liver diseases, possibly mediated by insulin resistance, systemic inflammation, and metabolic disturbances. In individuals with excess adiposity, reduced lean mass may be underestimated and may contribute to the progression of metabolic dysfunction-associated steatotic liver disease (MASLD), particularly in more advanced stages such as hepatic fibrosis. **Objective:** To evaluate the association between low lean mass and hepatic fibrosis in individuals with MASLD. **Methods:** This was an observational, cross-sectional study with prospective data collection. Adults at risk for MASLD, including individuals with obesity, overweight, diabetes mellitus, and metabolic syndrome, were enrolled. Hepatic steatosis and hepatic fibrosis (F ≥ 2) were diagnosed by ultrasonography and elastography, respectively. Appendicular lean mass (ALM), estimated as the sum of lean mass in the arms and legs (kg), was assessed by dual-energy X-ray absorptiometry (DXA) and adjusted for height, body mass index (BMI), fat mass (Newman equation), and percentage of body weight. **Results:** A total of 176 individuals were included, with a median age of 60.5 (52.0–66.0) years, predominantly women (n = 139; 79%). Hepatic fibrosis was diagnosed in 45 individuals (25.6%). Participants with fibrosis had lower lean mass compared with those without fibrosis, with significant differences observed for ALM/BMI [0.55 (0.50–0.63) vs. 0.61 (0.53–0.73); p = 0.029] and ALM/weight × 100 [23.03 (20.74–24.57)% vs. 24.07 (22.46–27.65)%; p = 0.004]. **Conclusion:** Reduced ALM, particularly when adjusted for BMI and body weight, was associated with the presence of hepatic fibrosis in MASLD. These findings reinforce the importance of body composition assessment in clinical practice, contributing to the early identification and appropriate management of these patients.
Keywords: lean mass; hepatic fibrosis; metabolic dysfunction-associated steatotic liver D.

OTHERS

369 (3240) AUTOIMMUNE POLYGLANDULAR SYNDROME TYPE 1 BEYOND THE CLASSICAL TRIAD: A CASE REPORT

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Case presentation: A 40-year-old female patient with a diagnosis of an autoimmune polyglandular syndrome type 1 (APS-1/APECED) was referred due to progression of multiple comorbidities. Symptoms began at the age of 5 years with syncope and absence seizures during childhood. She later developed growth delay, fatigue, tremors, and hypocalcemia, leading to diagnoses of hypothyroidism and hypoparathyroidism. At 15 years, she presented with primary amenorrhea and absence of secondary sexual characteristics, consistent with hypergonadotropic hypogonadism. During this period, she also developed recurrent oral candidiasis and psoriasis. At the age of 20 years, she was hospitalized for malnutrition and diagnosed with atrophic gastritis and pernicious anemia. At 22 years of age, she developed sensorimotor peripheral neuropathy. Her history included multiple hospitalizations for hypocalcemia, gastroenteritis, and recent pneumonia. Imaging and laboratory investigations demonstrated bilateral medullary nephrocalcinosis, osteopenia, and peripheral neuropathy. Given the clinical diagnosis, the patient was referred to the hospital genetics service for genetic testing and family counseling. Whole-exome sequencing identified a likely pathogenic heterozygous variant in the AIRE gene, c.905G>A;p.(Cys302Tyr). The clinical findings were consistent with evolving APS-1 associated with multisystem involvement. **Discussion:** APS-1 is a rare autosomal recessive genetic disorder caused by mutations in the AIRE gene, responsible for immune self-tolerance. Failure of negative T-cell selection leads to multiorgan autoimmunity. Classically, diagnosis is established by the presence of at least two components of the triad consisting of chronic mucocutaneous candidiasis, hypoparathyroidism, and adrenal insufficiency. Candidiasis is usually the initial manifestation, followed by hypoparathyroidism and adrenal insufficiency during childhood and adolescence. However, non-endocrine manifestations such as atrophic gastritis, pernicious anemia, neuropathies, and pneumonitis may occur throughout disease progression. **Final considerations:** This case highlights the complexity and progression of APS-1, emphasizing manifestations beyond the classic triad and significant systemic involvement. Early recognition of the disease and multidisciplinary follow-up are essential to reduce complications and improve quality of life. Furthermore, genetic investigation contributes to diagnosis and family counseling.
Keywords: autoimmune polyendocrine syndrome type 1; hypoparathyroidism; chronic mucocutaneous candidiasis.

OTHERS

370 (3116) AUTOIMMUNE POLYGLANDULAR SYNDROME TYPE 3 WITH CYTOPENIAS AND REVERSIBLE INFECTIOUS MANIFESTATIONS: AN ATYPICAL COURSE

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Case presentation: A 38-year-old female patient was evaluated in 2017 for hypothyroidism secondary to Hashimoto's Thyroiditis (levothyroxine 100 mcg/day), vitiligo since age 18 with a family history in three sisters, two spontaneous abortions, fatigue, arthralgias, and recurrent genital candidiasis with allergy to fluconazole. Workup revealed chronic iron-deficiency anemia, leukopenia, thrombocytopenia, pernicious anemia, and thrombophilia (PAI-1 4G/4G, heterozygous MTHFR, hyperhomocysteinemia), with persistent miscarriages despite enoxaparin therapy. Laboratory findings: Hb 11.3 g/dL, leukocytes 2,620/mm³, platelets 136,000/mm³, B12 228 pg/mL, homocysteine 21 µmol/L, Anti-TPO > 1,300 IU/mL, positive anti-parietal cell and anti-intrinsic factor antibodies; celiac disease excluded. Calcium, PTH, cortisol, and blood glucose within normal limits. Intravenous iron and intramuscular B12, initiated due to oral refractoriness, yielded significant response: Hb 13.0 g/dL, leukocytes 3,430/mm³, platelets 156,000/mm³, B12 650 pg/mL, and complete resolution of candidiasis. After 9 years, the patient remains clinically stable with no new manifestations. **Discussion:** Autoimmune polyglandular syndromes comprise a rare group with heterogeneous presentation and unpredictable course. The described case corresponds to the APS-3 subgroup, characterized by autoimmune thyroid disease, vitiligo, and pernicious anemia. Of particular note is the atypical hematological involvement, including multiple cytopenias and hyperhomocysteinemia, which, in the context of improvement following iron and B12 replacement, supports a nutritional origin in the setting of autoimmune gastritis. Hyperhomocysteinemia, initially interpreted as an independent thrombophilic component, is shown to be a direct consequence of pernicious anemia, with implications for therapeutic management. The resolution of candidiasis following correction of nutritional deficiencies represents a rarely described finding: iron and B12 deficiencies impair cellular immunity and predispose to recurrent fungal infections, with potential reversibility. The absence of new endocrinopathies over 9 years reinforces the unpredictability of the syndrome. **Final remarks:** This case illustrates that APS-3 may present in an incomplete and atypical manner, underscoring the importance of longitudinal follow-up for identifying non-classical presentations with potential for reversibility.

Keywords: polyendocrinopathies, autoimmune; anemia, pernicious; Hashimoto disease.

OTHERS

371 (3357) CHALLENGES IN THE MANAGEMENT OF MEN 1: ASYMPTOMATIC PANCREATIC RECURRENCE

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Case presentation: A 36-year-old female patient with a diagnosis of multiple endocrine neoplasia type 1 (MEN 1) confirmed by genetic testing (MEN1: c.249_252del; p.Ile85fs; frameshift variant) and a positive family history. Clinical findings include a prolactinoma (0.5 cm microadenoma identified in 2021 and an initial prolactin level of 146 ng/mL), an insulinoma resected in 2019 by distal pancreatectomy, and a dermatofibroma confirmed by histopathology in 2023. As a surgical consequence, she developed diabetes mellitus, currently controlled with metformin, alogliptin, and pioglitazone. A post-surgical abdominal CT scan from 2020 indicated a homogeneous density of the pancreatic head and body, as well as the absence of the pancreatic tail. During follow-up in January 2025, a 3.4 x 2.2 cm solid-cystic lesion was identified in the pancreatic body, accompanied by two hypervascular hepatic nodules. Despite the structural progression, the patient remains asymptomatic, without documented episodes of hypoglycemia. In March 2026, the clinical presentation was aggravated by an extensive acute deep vein thrombosis in the left lower limb, and anticoagulation with rivaroxaban was initiated. **Discussion:** This case illustrates the dissociation between the volume of the pancreatic lesion (3.4 cm) and glycemic stability, strongly suggesting a Non-Functioning Pancreatic Neuroendocrine Tumor. Thus, the current therapeutic planning prioritizes the functional staging of this lesion using somatostatin analog scintigraphy. Due to the high morbidity of a surgical abdominal reintervention and the risk of progression to post-surgical insulin-dependent diabetes, minimally invasive strategies such as radiofrequency ablation or ethanol ablation are viable alternatives. Additionally, the use of somatostatin analogs or systemic chemotherapy are options for controlling tumor progression. **Final considerations:** There is no consensus regarding follow-up for patients with MEN 1, but experts suggest continuous annual clinical and biochemical evaluation, and monitoring with imaging exams should be individualized. In asymptomatic and tumor-free patients, pancreatic imaging is generally indicated every two to three years, and pituitary imaging every three to five years. Given this context, successful treatment depends on an individualized approach that integrates oncological surveillance, management of comorbidities, and careful consideration between invasive and conservative interventions.

Keywords: multiple endocrine neoplasia; recurrence; diabetes mellitus.

OTHERS

372 (3229) CLINICAL IMPLICATIONS OF RECLASSIFYING VARIANTS OF UNCERTAIN SIGNIFICANCE IN 46,XY DIFFERENCES OF SEX DEVELOPMENT: INSIGHTS FROM A MULTICOHORT ANALYSIS

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Introduction: Differences of sex development (DSD) are congenital conditions characterized by discordance among chromosomal, gonadal, and anatomical sex. In 46,XY DSD, next-generation sequencing (NGS) has improved diagnostic yield but has also increased detection of variants of uncertain significance (VUS), creating challenges for clinical interpretation. Data regarding VUS reclassification in this population remain limited. **Objective:** Evaluate the prevalence, recurrence, and reclassification of VUS reported in 46,XY DSD cohorts from different ethnicities. **Methods:** A structured review identified studies including individuals with 46,XY DSD of undefined molecular etiology who underwent targeted gene panel or whole-exome sequencing. The genes with the most prevalent VUS and their distribution among the different ethnic groups was evaluated. VUS reported according to ACMG/AMP 2015 criteria were reclassified using updated guidelines, population databases (gnomAD), and in silico predictors (REVEL, AlphaMissense, SpliceAI). Statistical analyses included Welch's t-test and logistic regression. **Results:** Eleven studies comprising 1,109 patients were included. In total, 214 VUS were reported across 65 different genes, representing 195 unique variants. VUS prevalence ranged from 1.5% to 47% (mean 18%), with no significant difference between sequencing approaches. Recurrent variants were observed in several genes, including AR, AMH, CBX2, CST9, EGF, GATA4, HSD17B3, MAMLD1, MAP3K1, MYRF, NR5A1, PROK2, SRD5A2, and WDR11. Only four variants were recurrent across different ethnicities: AR c.1174C>T (United Kingdom and Turkey), SRD5A2 c.281G>T (China and Australia), and NR5A1 c.1019C>T (Brazil and England). In addition, a recurrent MAMLD1 variant (p.Gln502dup) was identified in patients from China and the United States. Among 214 reported VUS, 180 were eligible for reanalysis: 21 (11.6%) were upgraded to likely pathogenic, 48 (26.6%) downgraded to likely benign/benign, and 111 (61.6%) remained VUS. Downgrades were significantly more frequent than upgrades (OR 2.29; 95% CI 1.37–3.82; p = 0.0015). **Conclusion:** Reanalysis enabled clinically relevant reclassification in over one-third of variants, predominantly toward benign categories. However, most variants remained uncertain, highlighting the need for functional studies, standardized interpretation, and international data sharing in 46,XY DSD.
Keywords: VUS; DSD; reclassification.

OTHERS

373 (3499) COMPARISON OF THE CLINICAL RESPONSE TO THE USE OF CORTICOSTEROIDS AND ACARBOSE IN THE MANAGEMENT OF HYPOGLYCEMIA IN AUTOIMMUNE INSULIN SYNDROME

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Introduction: Autoimmune Insulin Syndrome (Hirata's disease) is a rare disorder characterized by recurrent hypoglycemia associated with anti-insulin autoantibodies in patients without prior exposure to exogenous insulin. Delayed insulin release from insulin-autoantibody complexes commonly leads to postprandial hypoglycemia. Treatment options include corticosteroids and acarbose, although the optimal therapeutic strategy remains uncertain. **Objective:** To compare the clinical response to corticosteroids and acarbose in the management of hypoglycemia in patients with insulin autoimmune syndrome. **Methods:** This systematic review followed PRISMA recommendations. Searches were conducted in the PubMed and LILACS databases using the terms "Insulin Autoimmune Syndrome," "corticosteroids," "prednisone," and "Acarbose." Studies involving patients treated with corticosteroids or acarbose were included. Case reports, case series, and observational studies published in English, Portuguese, or Spanish were considered eligible. Reviews, animal studies, and articles without clear treatment outcomes were excluded. After screening, six studies were included. **Results:** Corticosteroids demonstrated significant efficacy in inducing remission. In a series of six cases, prednisolone (30–60 mg/day for 4–6 weeks) achieved complete remission of hypoglycemic episodes in 66.6% of patients and markedly reduced insulin levels. However, steroid therapy was associated with severe hyperglycemia, sometimes requiring additional antidiabetic treatment. Acarbose produced rapid clinical improvement, with doses of 25–100 mg three times daily leading to complete cessation of postprandial hypoglycemia after the first doses. Long-term use of acarbose combined with sitagliptin also improved glycemic control and reduced HbA1c levels. Furthermore, acarbose contributed to stabilization of antibody levels until their disappearance within 3–6 months after removal of the triggering factor. **Conclusion:** The clinical choice between the two medications, or their combined use, yields superior results in cases of high glycemic lability. While corticosteroids treat the underlying cause (antibody depletion), acarbose controls the postprandial phenomenon that triggers inappropriate insulin release by immune complexes.
Keywords: autoimmune insulin syndrome; hypoglycemia; acarbose.

OTHERS

374 (3025) CONSEQUENCES OF MICROPLASTICS EXPOSURE IN WISTAR RATS: EVALUATION OF THE EFFECTS OF METABOLIC PROGRAMMING AND DIRECT EXPOSURE

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Microplastics (MPs) are widely distributed in environment and have been detected in human tissues, including placenta and breast milk. Given their potential to cause endocrine and cognitive damages, we investigated whether perinatal and direct exposure to MP induces dysfunction in dams and offspring of both sexes and different ages. In the perinatal exposure experiment, Wistar rats were exposed to water (control) or 10 µm polystyrene MPs (25 µg/kg) from day 7 of gestation to weaning. Dams and offspring were evaluated at PN21, PN45 and PN120. In the direct exposure experiment, rats received the same treatments from PN50 to PN95. Biometric, biochemical, hormonal and behavioral parameters were evaluated. Data were analyzed by Student's t-test, with sex-specific evaluation. In the perinatal model, MP dams showed lower weight gain, cholesterol and T4 levels. At weaning, MP male offspring exhibited higher cholesterol and lower corticosterone, while females showed hyperlipidemia. At puberty, MP males presented hypertriglyceridemia, hypocholesterolemia and lower T3, with increased anxiety-like behavior, which persisted into adulthood. In adulthood, MP males showed lower leptin, higher FSH and reduced preference for high-fat diet (HFD), whereas MP females exhibited lower plasma corticosterone and higher TSH and T3. In the direct exposure model, MP males showed higher plasma T3 and T4 and females, only high T4. Additionally, MP males had greater preference for high-sucrose diet, while MP females preferred HFD. Our findings show that MP exposure during critical developmental windows induces biometric, lipid metabolism, hormonal and behavioral alterations. By following animals from intrauterine life to adulthood, the current data fill an important gap in research on perinatal exposure. Given the physiological similarities between rodents and humans, our results highlight the potential risks to human health and reinforce the relevance of MP as an emerging public health concern.
Keywords: microplastic; hormones; endocrine.

OTHERS

375 (3023) CONSEQUENCES OF NANOPLASTICS IN WISTAR RATS: EVALUATION OF THE EFFECTS OF METABOLIC PROGRAMMING AND DIRECT EXPOSURE

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Increasing presence of nanoplastics (NP) in the environment raises concerns about their impact on biological systems, especially during critical developmental period. This study investigated metabolic, hormonal, and food behavioral effects of NP exposure using Wistar rats. This protocol was approved by the Ethics Committee (032/2022). In Experiment 1, from gestational day 7 until weaning, dams received vehicle (filtered water; control; n = 9) and 100 nm polystyrene nanoparticles (25 µg/kg bw; NP; n = 8). NP dams showed reduced weight gain (–19%, p = 0.0080), decreased milk triglycerides at lactation day 10 (–61%, p = 0.0138), and lower T4 (–10%, p = 0.0329). Male offspring showed reduced corticosterone at weaning (–55%, p = 0.0001), hypocholesterolemia (–25%, p = 0.0344) and hypotriglyceridemia (–39%, p = 0.0131) during puberty, and increased FSH in adulthood (+51%, p = 0.0437). Behavioral changes included increased preference for standard diet (+200%, p = 0.0023), reduced preference for high-fat diet (–12%, p < 0.0001), and higher intake of high-fat diet (+12%, p = 0.0053). Female offspring showed reduced weight gain (–13%, p = 0.0775), increased food intake in adulthood (+3.4%, p = 0.0030), elevated TSH (+50%, p = 0.0483) and T3 (+173%, p = 0.0051), reduced preference for high-fat diet (–26%, p < 0.0001), and lower standard diet intake (–80%, p = 0.0044). In Experiment 2, from PN45 to 90, animals received vehicle (control; n = 12/sex) and nanoparticles (n = 12/sex, same dose). No biometric or glycemic changes were observed in males, but reduced preference for high-sucrose diet (–75%, p < 0.0001), increased standard diet intake (+166%, p = 0.0118), and decreased T3 (–30%, p = 0.0058) and T4 (51%, p = 0.0435) were found. Females showed reduced weight gain (–9.1%, p = 0.0387) and altered feeding behavior without hormonal changes. Overall, NP induces persistent endocrine, metabolic, and behavioral alterations, supporting their role as endocrine disruptors.
Keywords: nanoplastics; endocrine disruptor; metabolic programming.

OTHERS

376 (3253) ECTOPIC ACTH SYNDROME CAUSED BY PULMONARY CARCINOID TUMOR IN AN ADOLESCENT: A CASE REPORT

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Case presentation: A 17-year-old male developed progressive weight gain, centripetal obesity, moon facies, dorsocervical fat pad, violaceous striae > 1 cm, acanthosis nigricans, and hypertension over three months. Laboratory evaluation confirmed ACTH-dependent hypercortisolism: basal cortisol 34.2 µg/dL, 24-hour urinary free cortisol 648 µg/24 h (reference 13–85), late-night salivary cortisol 42.6 ng/mL (reference < 7.6), ACTH 75 pg/mL (reference 7–63), and lack of suppression after 1 mg dexamethasone (cortisol 28.5 µg/dL). Pituitary MRI was unremarkable. Inferior petrosal sinus sampling showed no central-to-peripheral ACTH gradient (basal ratio 1.1; peak post-DDAVP 1.4), excluding Cushing's disease. Chest CT subsequently revealed a 1.7-cm solid pulmonary nodule in the right middle lobe. Image-guided biopsy confirmed carcinoid tumor, and middle lobectomy was performed. Pathological examination demonstrated a 2.7-cm typical carcinoid with angiolymphatic invasion, free surgical margins, and no lymph node involvement. Immunohistochemistry was positive for chromogranin A and synaptophysin, with Ki-67 < 1%, but negative for ACTH staining. Rapid postoperative cortisol decline confirmed biochemical remission and strongly supported the tumor as the ectopic ACTH source. The patient developed postoperative adrenal insufficiency requiring glucocorticoid replacement, with progressive recovery of the hypothalamic-pituitary-adrenal axis. Follow-up demonstrated improvement of cushingoid features, weight reduction, controlled blood pressure, and persistent low lumbar bone mineral density (Z-score –2.4). **Discussion:** Ectopic ACTH syndrome caused by pulmonary carcinoid tumors is rare in pediatric patients. Negative ACTH immunostaining may occur in highly secretory tumors due to depletion of intracellular ACTH stores, creating dissociation between histological and functional findings. In this case, postoperative biochemical remission confirmed the ectopic source despite negative ACTH staining. **Final comments:** This case highlights the importance of systematic investigation of ectopic ACTH secretion in young patients with ACTH-dependent hypercortisolism, absent pituitary lesions, and negative inferior petrosal sinus sampling.
Keywords: Cushing syndrome; ectopic ACTH secretion; carcinoid tumor.

OTHERS

377 (3045) EFFICACY OF SGLT2 INHIBITORS IN CARDIORENAL AND METABOLIC PROTECTION IN NON-DIABETIC POPULATIONS: A SYSTEMATIC REVIEW (2021–2026)

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Introduction: Sodium-glucose cotransporter 2 inhibitors (SGLT2i) have evolved from glycemic-lowering agents to essential therapies in the management of cardiorenal conditions, including chronic kidney disease (CKD), heart failure (HF), and cardiovascular disease (CVD), establishing efficacy even in populations without diabetes. **Objective:** This systematic review aimed to synthesize clinical evidence on the impact of SGLT2 inhibitors on renoprotection, heart failure management, and cardiovascular outcomes in adults without diabetes, including kidney disease progression, heart failure hospitalization, and cardiovascular mortality, between 2021 and 2026. **Methods:** Following the recommendations of the PRISMA Statement, a systematic search was conducted in the PubMed/MEDLINE database. A total of 36 studies were identified, of which 6 were included in the final analysis after screening titles and abstracts and applying the eligibility criteria. Randomized controlled trials and cohort studies involving adults (≥18 years) without diabetes, using SGLT2 inhibitors, and assessing cardiorenal outcomes were considered eligible. Editorials, reviews, non-original studies, and studies involving individuals with diabetes were excluded. The use of a single database is recognized as a limitation. **Results:** The analysis of the 6 selected studies demonstrates a consistent, class-wide clinical benefit. A reduction in renal disease progression was observed, with a slower decline in eGFR and decreased albuminuria, regardless of glycemic status. In heart failure, there was a significant reduction in hospitalizations and cardiovascular mortality across different ejection fraction profiles. In the post-myocardial infarction setting, a lower incidence of heart failure and related hospitalizations was identified. Additionally, improvements were observed in secondary outcomes, including reduced serum uric acid levels and improved vascular function, further reinforcing the prognostic impact of these agents. **Conclusion:** SGLT2 inhibitors are now fundamental pillars of modern cardiorenal therapy, with benefits independent of hypoglycemic effects. By mitigating adverse events in non-diabetic populations with CKD, HF, or post-MI risk, these agents have redefined standard care. It is therefore inferred that future studies should prioritize optimizing combination therapies and expanding the use of these agents in early disease stages to maximize prognostic improvements for patients at risk.
Keywords: SGLT2 inhibitors; cardiorenal outcomes; non-diabetic population.

OTHERS

378 (3030) EXOGENOUS CUSHING SYNDROME CAUSED BY UNDECLARED GLUCOCORTICOID IN A COLLAGEN SUPPLEMENT

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A 17-year-old girl was referred due to progressive Cushingoid features. She gained 15 kg over 4 months and developed violaceous striae, facial plethora, an acneiform eruption, secondary amenorrhea, and proximal muscle weakness. She denied glucocorticoid use by any route. Biochemical evaluation was repeatedly negative for hypercortisolism. Morning serum cortisol and ACTH concentrations were within the reference ranges and showed no complete suppression (Table 1). Although there was no biochemical evidence of ACTH-dependent hypercortisolism, the patient presented with a pituitary MRI obtained at another institution showing a microadenoma. After repeated questioning about medication and supplement use, the patient disclosed intermittent use of a supplement containing type II collagen and palmitoylethanolamide over the previous 10 months. Toxicological assay by gas chromatography-tandem mass spectrometry (GC-MS/MS), performed at our institution, identified an undeclared glucocorticoid compatible with dexamethasone or betamethasone, as well as caffeine and ketorolac tromethamine. After supplement withdrawal, acne improved, striae became less prominent, menstrual cycles resumed, and weight stabilized. She did not develop adrenal insufficiency. Exogenous glucocorticoid exposure is the most common cause of Cushing syndrome, and undeclared corticosteroids in "natural" formulations represent an important public health issue. Exposure to hidden corticosteroids may lead to adverse effects, including iatrogenic Cushing syndrome and adrenal insufficiency. FDA analyses identified 776 adulterated dietary supplements between 2007 and 2016, many of which were marketed for musculoskeletal conditions. Several contained undeclared pharmacologically active substances, particularly diclofenac and dexamethasone, the latter being detected in more than one-third of products marketed for joint and muscle pain. This case also highlights the importance of a stepwise approach to the investigation of Cushing syndrome, with imaging studies guided by biochemical confirmation of hypercortisolism. Premature or non-targeted imaging may identify incidental findings that complicate diagnostic interpretation, as illustrated by the pituitary incidentaloma observed in this patient, a finding reported in up to 10% of the general population. Therefore, hidden glucocorticoid exposure should be suspected in patients with Cushingoid features and discordant biochemical results.

Keywords: Cushing syndrome; glucocorticoids; dietary supplements.

OTHERS

379 (3210) EXPERIMENTAL STUDY: HEPATIC EFFECTS OF N-ACETYLCYSTEINE IN FEMALE WISTAR RATS FED A HIGH-FAT DIET UNDER ASPARTAME ADMINISTRATION

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Introduction: Aspartame is a widely used artificial sweetener whose metabolism generates compounds capable of inducing oxidative stress and reducing glutathione levels, thereby promoting hepatic alterations. N-acetylcysteine (NAC), as a glutathione precursor, has potential antioxidant and hepatoprotective effects, especially under conditions associated with obesity and high-fat diets. **Methods:** Female Wistar rats (8–12 weeks old) were divided into four groups: control (CT, n = 8), high-fat diet (HFD, n = 7), high-fat diet associated with aspartame (HFD+Asp, n = 7), and high-fat diet associated with aspartame and NAC (HFD+Asp+NAC, n = 7). The experimental protocol lasted 17 weeks. The control group received standard chow and water ad libitum, whereas the remaining groups received a high-fat diet with added sugar in drinking water. During the final seven weeks, the HFD+Asp and HFD+Asp+NAC groups received aspartame (80 mg/kg/day) replacing sugar. The HFD+Asp+NAC group additionally received NAC (100 mg/kg/day, intraperitoneally). **Results:** Histological liver analysis demonstrated preserved hepatic architecture in the control group. The HFD group presented mild to moderate hepatic steatosis, predominantly grades 1 and 2. In the HFD+Asp group, there was an increase in both frequency and severity of hepatic alterations, with predominance of higher grades of steatosis, suggesting a potential synergistic effect between high-fat diet and aspartame in inducing liver injury. In contrast, the NAC-treated group showed attenuation of histological alterations, with predominance of mild steatosis and absence of progression to more severe forms. **Conclusion:** Aspartame potentiated hepatic alterations induced by the high-fat diet, possibly mediated by oxidative stress. N-acetylcysteine demonstrated a hepatoprotective effect, reducing the severity of histological lesions. These findings reinforce the role of oxidative stress in the pathophysiology of hepatic steatosis and highlight the therapeutic potential of NAC in modulating these alterations.

Keywords: aspartame; acetylcysteine; obesity.

OTHERS

380 (3014) EXPOSURE TO METHYLPARABEN MODULATES METABOLIC PARAMETERS AND LIVER FUNCTION MARKERS IN WISTAR RATS

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Endocrine disruptors (EDs) are exogenous chemical substances that alter endocrine system functions, causing adverse health effects. Among these substances is methylparaben (MP), a compound belonging to the paraben class, commonly used in the food and pharmaceutical industries due to its antimicrobial and antifungal properties. The uncontrolled use of MP results in high environmental contamination, especially in water sources, exposing animals and humans to average concentrations of 30 µg/L, which may cause damage to organs such as the live. Prolonged exposure to high doses of this environmental pollutant overloads the organ; however, the actual impacts of environmentally relevant doses and short exposure periods on liver function remain unclear. This study aimed to evaluate the effects of exposure to methylparaben, at environmentally relevant doses, on metabolic parameters and liver function in Wistar rats. For this purpose, 3-month-old male Wistar rats were used in accordance with CEUA approval (A16/25-A52-24-113-22). The animals were distributed into four experimental groups: Control (0.1% DMSO), MP3 (3 µg/kg/day), MP30 (30 µg/kg/day), and MP300 (300 µg/kg/day). The animals were treated by oral gavage for 14 days. During the experimental period, food intake and body weight were monitored. A glucose tolerance test (GTT) was performed one day before euthanasia, and serum biochemical parameters related to lipid metabolism and liver function were evaluated. The results revealed that body weight gain during treatment and liver mass relative to body surface area showed no significant differences between the groups. However, absolute liver mass was increased in the MP300 group. In the GTT, the MP300 group exhibited lower blood glucose levels compared to the control, with significant differences at 0, 30, and 120 minutes. Triglyceride and total cholesterol levels did not differ among groups. However, an increase in HDL and a decrease in LDL levels were observed in the MP300 group. An increase in ALT levels was observed in animals treated with the intermediate dose. Taken together, the data reveal that even environmentally relevant doses or real exposure doses over a short exposure period may modulate metabolic parameters and biomarkers of liver function, although additional studies are needed to further elucidate these effects.

Keywords: endocrine disruptors; liver; methylparaben.

OTHERS

381 (3264) FROM NONSPECIFIC FATIGUE TO A RARE GENETIC DIAGNOSIS: LATE PRESENTATION OF 46,XX TESTICULAR DSD

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Case presentation: A 37-year-old male sought medical attention due to a 4-month history of asthenia and fatigue. Initial laboratory evaluation revealed total testosterone of 51.96 ng/dL, FSH of 29 mIU/mL, and LH of 10.95 mIU/mL. Scrotal ultrasonography demonstrated bilateral testicular atrophy, with a right testicular volume of 2.3 cm^3, in addition to a left hydrocele. The patient also reported a previous history of varicocele diagnosed and surgically treated in 2015. During follow-up, semen analysis revealed azoospermia, prompting cytogenetic investigation. Karyotype analysis showed 45,X[7]/46,XX[23], later confirmed as 46,XX on repeat testing. Testosterone undecanoate therapy was initiated, achieving adequate testosterone levels under hormone replacement therapy. Given the diagnosis of 46,XX testicular disorder of sex development (DSD) and the patient's reproductive desire, he was referred to a specialized infertility clinic. **Discussion:** 46,XX testicular DSD is a rare condition characterized by a male phenotype in individuals lacking a complete Y chromosome. In most cases, it results from translocation of the SRY gene to the X chromosome during paternal gametogenesis. Clinically, patients may present with varying degrees of hypogonadism, infertility, small testes, and azoospermia. Infertility is nearly universal due to the absence of the azoospermia factor (AZF) regions of the Y chromosome, which are essential for spermatogenesis. Diagnosis is usually established in adulthood during infertility evaluation or investigation of symptoms related to androgen deficiency. The present case is notable for the delayed diagnosis, initially prompted by nonspecific symptoms of hypogonadism. **Final comments:** 46,XX testicular DSD should be considered in the evaluation of men presenting with hypergonadotropic hypogonadism, azoospermia, and bilateral testicular atrophy. Recognition of this condition enables appropriate multidisciplinary management, genetic counseling, and individualized treatment of the associated hormonal, metabolic, reproductive, and psychosocial implications.

Keywords: hypergonadotropic hypogonadism; azoospermia; disorder of sex development.

OTHERS

382 (2985) GESTATIONAL EXPOSURE TO AGRICULTURAL PESTICIDES AND MODULATION OF NEUROENDOCRINE, NEUROCHEMICAL, AND NEUROADAPTIVE PATHWAYS: INTEGRATIVE GENE-PATHWAY-DISEASE ANALYSIS

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Introduction: Exposure to environmental contaminants during pregnancy has been associated with neuroendocrine, metabolic, and neurochemical alterations capable of impacting neurotransmission mechanisms, cellular plasticity, and hormonal regulation. Compounds related to agricultural pesticides have the potential to act as endocrine disruptors and modulators of cellular pathways associated with neuroadaptation, but the molecular mechanisms involved in this interaction remain poorly understood. **Objective:** To investigate the correlation between genes responsive to environmental exposure and biological pathways associated with neuroendocrine, neurochemical, and neuropsychiatric processes. **Methods:** Genes associated with agricultural pesticide compounds identified in urine samples from 12 pregnant women selected from a prospective cohort conducted in Paraná (CAAE 65526622.7.0000.0106) were analyzed. After gene selection per patient, KEGG functional enrichment, gene-disease association analysis, and integration between genes, biological pathways, and recurrence among patients were performed to identify shared functional cores and biologically central genes. The shared pathways were grouped into biological axes related to neurotransmission, endocrine disruption, neuroadaptive circuits, and intracellular signaling. **Results:** Functional convergence was observed involving dopaminergic, glutamatergic, and GABAergic neurotransmission, as well as pathways related to calcium signaling, cAMP, PI3K-Akt, and FoxO, and neuroendocrine regulation. Pathways associated with hormone synthesis and regulation were also identified, suggesting a possible endocrine disruption component linked to environmental exposure. Genes such as SLC6A3, GRM1, ESR1, and EGFR showed high connectivity among multiple biological axes, pathways, and patients. Gene-disease association analyses demonstrated a predominance of phenotypes related to neuropsychiatric disorders, substance use, and cognitive alterations, suggesting the involvement of mechanisms related to neurotransmission, neuronal plasticity, and cellular adaptation. **Conclusion:** The findings suggest that exposure to compounds related to agricultural pesticides may be associated with integrated modulation of neuroendocrine and neurochemical pathways, with convergence in processes of neuroadaptation, cellular plasticity, and neurotransmission regulation. **Acknowledgments:** SETI-Fundo Paraná; Fundação Araucária; CNPq; Capes

Keywords: agricultural pesticides; pregnancy; neuroendocrine disruption.

OTHERS

383 (3296) GIANT INSULINOMA IN AN OCTOGENARIAN PATIENT: A CASE REPORT

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Case presentation: M.N.D., an 85-year-old woman with a history of hypertension and dementia, presented in 2020 with episodes of altered level of consciousness, tremors, and presyncope, which resolved after food intake. In 2024, due to persistence and worsening of symptoms, she was referred to evaluation. Laboratory tests showed: fasting glucose 45 mg/dL, fasting insulin 32.1 mU/L, insulin/glucose ratio of 0.71, and C-peptide 9 ng/mL. A diagnosis of endogenous hyperinsulinemic hypoglycemia was established after excluding the use of oral hypoglycemic agents, exogenous insulin, and autoimmune hypoglycemia. On May 10, 2024, abdominal MRI revealed a solid and heterogeneous expansive lesion in the head of the pancreas measuring 11.5 × 9.5 × 9.0 cm. Ultrasound-guided biopsy demonstrated a well-differentiated neuroendocrine tumor (NET grade I). Immunohistochemistry was positive for AE1 + AE3, CD56, chromogranin, synaptophysin, with a Ki-67 index of 2%. Chest CT showed no evidence of metastasis. Due to the patient's non-eligibility for the surgical procedure, as determined by the anesthesiology team, clinical treatment with a somatostatin analogue was chosen, and Lanreotide 120 mg monthly was prescribed on July 21, 2025 initiated, resulting in clinical improvement and blood glucose levels of 150 mg/dL. Recent CT imaging demonstrated stability in the size of the expansive lesion. **Discussion:** Insulinomas are rare neuroendocrine neoplasms, usually measuring less than 2 cm, with peak incidence in the fifth decade of life. Variants larger than 9 cm are classified as giant insulinomas, which are associated with higher rates of malignancy and invasive potential. Typically, benign and intrapancreatic in location, they manifest with symptomatic hypoglycemia, worsening during fasting periods and remission after caloric intake, a presentation characterized by Whipple's triad. Clinical suspicion was raised only when the patient was 80 years old, together with symptoms and the presence of an approximately 11 cm mass in the pancreatic head. Unlike the usual behavior of giant insulinomas, the patient showed no evidence of metastasis or local invasion. Given the impossibility of surgical treatment, lanreotide proved to be an effective and safe therapeutic option. **Final comments:** Insulinomas are rare, and giant variants in the elderly are even more uncommon. This case highlights that, even with large dimensions, the tumor can exhibit benign and non-invasive behavior.
Keywords: hypoglycemia; hyperinsulinemia; neuroendocrine tumor.

OTHERS

384 (2986) HEPATIC ALTERATIONS AFTER EXPOSURE TO LOW DOSES OF A GLYPHOSATE-BASED HERBICIDE IN WISTAR RATS

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Introduction: The intensive use of the herbicide glyphosate is common in modern agriculture, with Brazil being one of the largest consumers worldwide. Although initially considered safe, recent studies reveal its detection in food, water, and human fluids. The liver, the main organ responsible for xenobiotic metabolism, is susceptible to the toxicity of such compounds. Studies from our group showed that glyphosate alters thyroid receptor expression in the liver. Since thyroid hormones regulate hepatic homeostasis, lipogenesis, β-oxidation, and energy metabolism, hepatic metabolic pathways may also be impaired. **Objective:** To evaluate, in Wistar rats, the effects of a glyphosate formulation on biochemical parameters and hepatic molecular alterations, integrating gene expression and proteomic analysis. **Methods:** 90 rats were exposed to 0, 0.5, or 5 mg/kg/day of glyphosate from postnatal days 23 to 60 or 90. Serum levels of ALT and AST were determined by a colorimetric method. The expression of antioxidant genes (Sod1, Cat, Gsr and Gpx1) and genes regulated by miRNA-122 (Srf, Eif2ak1), was evaluated by RT-qPCR. The identification and analysis of differentially expressed proteins were performed using the STRING and Cytoscape software. **Results:** At the highest dose, a reduction in ALT, decreased expression of Sod1, Cat and Gsr, and an increase in Srf and Eif2ak1were observed, suggesting impairment of antioxidant function and dysregulation of miRNA-122, a marker associated with early hepatic inflammation. These data were complemented by proteomic analysis, where overexpressed proteins related to amino acid, fatty acid, and carboxylic acid metabolism pathways were found, which are associated with mitochondrial energy metabolism, lipid metabolism, and responses to oxidative stress, suggesting increased energy demand and hepatic metabolic reprogramming in response to glyphosate exposure. Additionally, the reduction of proteins associated with Acyl-CoA metabolism and β-oxidation, such as ACAA2, also indicates a possible impairment of hepatic lipid metabolism and a predisposition to lipid accumulation due to mitochondrial dysfunction. **Conclusion:** These results demonstrate that exposure to low doses of glyphosate in the medium term may trigger early hepatic changes, potentially preceding severe hepatic dysfunctions, reinforcing the need for attention to chronic population exposure to this contaminant. **Acknowledgments:** CAPES and Araucária Foundation for financial support

Keywords: herbicide glyphosate; liver; oxidative stress.

OTHERS

385 (3422) HIGH PENETRANCE OF PRIMARY HYPERPARATHYROIDISM AS A PHENOTYPIC MANIFESTATION IN A FAMILY WITH MULTIPLE ENDOCRINE NEOPLASIA TYPE 2A AND THE RET PROTO-ONCOGENE C634Y MUTATION

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Case presentation: A 32-member family underwent genetic testing for multiple endocrine neoplasia type 2A (MEN-2A). Thirteen individuals were found to carry the C634Y mutation in codon 11 of chromosome 10 (10q11.2) of the RET proto-oncogene (rearranged during transfection). Patients were followed for 30 years. All individuals from the first and second generations were diagnosed with medullary thyroid carcinoma (MTC). All individuals from the third generation underwent prophylactic thyroidectomy at 5, 6, and 10 years of age, and all were diagnosed with C-cell hyperplasia (23%). Pheochromocytoma (PHEO) was identified in 8 patients, while primary hyperparathyroidism (PHPT) was identified in 7 patients. During follow-up, penetrance of clinical manifestations among the adult population reached 100% for MTC, 80% for Pheochromocytoma, and 70% for PHPT. **Discussion:** Genetic screening in MEN-2A has become both routine and essential. Depending on the affected codon, phenotypic manifestations may vary substantially. Previous studies identified the C634 mutation as the most prevalent mutation in a Brazilian cohort. The C634Y variant is rare and generally indolent, although poorly described in the literature. Penetrance rates of 100% for MTC and 80% for PHEO observed in our cohort have previously been reported by other authors. However, PHPT has been described as having low prevalence both in MEN-2A and in reports involving the C634Y mutation (approximately 9%). Therefore, the finding of a 70% penetrance rate in this study is novel and intriguing. **Final comments:** We describe a previously unreported finding of high penetrance of PHPT in a Brazilian family with MEN-2A associated with the RET proto-oncogene C634Y mutation.

Keywords: multiple endocrine neoplasia; hyperparathyroidism; C643Y mutation.

OTHERS

386 (3343) HYPERGONADOTROPIC HYPOGONADISM ASSOCIATED WITH OSTEOPOROSIS IN A PATIENT WITH A RARE GENETIC SYNDROME ASSOCIATED WITH A DISORDER OF GLYCOPROTEIN METABOLISM: A CASE REPORT

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Case report: A 39-year-old woman with a history of hypotonia, delayed neuropsychomotor development, intellectual disability, and epilepsy since childhood was evaluated. She presented with primary amenorrhea and absence of secondary sexual characteristics, starting hormone replacement therapy only at age 25, with delayed pubertal development thereafter. Physical examination revealed strabismus, ataxic gait, and lower limb hyporeflexia. Laboratory evaluation showed FSH 77.5 mIU/mL, LH 50.9 mIU/mL, and estradiol < 5 pg/mL, consistent with hypergonadotropic hypogonadism. Pelvic ultrasound demonstrated a hypoplastic uterus and non-visualized ovaries, while renal ultrasound showed a reduced left kidney. Karyotype was 46,XX. Whole-exome and mitochondrial DNA sequencing identified a congenital glycosylation defect related to the PMM2 gene in heterozygosity, compatible with Congenital disorders of glycosylation (CDG)-Ia. Maternal genetic testing confirmed heterozygosity for the same gene variant. Bone densitometry revealed osteoporosis, with a femoral total Z-score of –2.8. Brain magnetic resonance imaging demonstrated cerebellar atrophy and hippocampal abnormalities, while electroencephalography showed rare left posterior temporal epileptiform activity. The patient is currently receiving: hormones replacement therapy, risedronate, calcium, colecalciferol and antiepileptic drugs. Received also multidisciplinary follow-up. **Discussion:** Congenital disorders of glycosylation are rare multisystemic inherited metabolic diseases caused by defects in glycoprotein synthesis and processing. CDG-Ia, associated with phosphomannomutase 2 deficiency, predominantly presents with neurological manifestations such as ataxia, epilepsy, and cognitive impairment. Endocrine manifestations are less common and include hypergonadotropic hypogonadism, primary ovarian insufficiency, and thyroid abnormalities. Prolonged estrogen deficiency contributes to reduced bone mineral density and early osteoporosis. In this case, delayed initiation of hormone replacement therapy likely impaired peak bone mass acquisition. Differential diagnosis included other causes of primary ovarian insufficiency, excluded by the normal karyotype and syndromic context. **Final comments:** This case highlights the importance of recognizing endocrine manifestations in CDG, enabling early diagnosis and intervention, with potential impact on quality of life and prevention of osteometabolic and cardiovascular complications.
Keywords: CDG-1a; hypergonadotropic hypogonadism; osteoporosis.

OTHERS

387 (3559) HYPERINSULINEMIC HYPOGLYCEMIA: INSULINOMA AS A DIAGNOSTIC CHALLENGE

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T.M.A., female, 30 years old. nursing technician. hospitalization on 02/22/2024. hypoglycemia with neuroglycopenic symptoms reversed after continuous hypertonic glucose infusion. capillary blood glucose less than 18 mg/dL. hypothesis of adrenal insufficiency considered, treated with intravenous corticosteroid therapy, but without reduction in glucose infusion rate or hypoglycemia, and was soon discontinued. 2024 exams: glucose: 24/C-peptide 4.98; cortisol 19.5; ACTH 94.5; HGH 9.69; insulin 8.95. imaging exams including pet dota showed no tumor location. calcium gluconate infusion test performed with peak response in the superior mesenteric and gastroduodenal arteries, undergoing gastroduodenopancreatectomy without complications. pathological findings: nodular chronic pancreatitis (0.8 cm) positive for chromogranin, synaptophysin, insulin, and glucagon. the patient remained without new episodes of hypoglycemia for approximately one year, when he was readmitted with a hypoglycemic coma. laboratory 2025: glucose 30; C-peptide 0.97; insulin 2.96. during this hospitalization, she had difficulty weaning off intravenous hypertonic glucose, requiring caloric support via nasoenteral tube during periods of poor oral intake and infusion of high-rate hypertonic glucose, which he could not tolerate interruption for more than two hours due to a sudden drop in blood glucose along with neuroglycopenic symptoms. due to the unavailability of diazoxide, and as a bridge therapy for hospital discharge and a new gallium pet scan octreotide long acting release 30 mg intramuscularly was administered, resulting in a significant improvement in glycemic levels and hospital discharge and no more episodes of hypoglicemia so far. Thus, the syndromic diagnosis of hyperinsulinemic hypoglycemia was repeated and confirmed, but the difficulty in tumor localization makes management and permanent treatment a clinical challenge.
Keywords: insulinoma; hypoglicemia; hyperinsulinism.

OTHERS

388 (3335) HYPOGLYCEMIA SECONDARY TO TYPE B INSULIN RESISTANCE SYNDROME IN NON-HODGKIN LYMPHOMA: A CASE REPORT

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Type B insulin resistance syndrome (TBIRS) is a rare autoimmune disorder affecting glucose homeostasis. We report the case of an 81-year-old woman admitted with malaise and anorexia. Abdominal ultrasound revealed nodules suggestive of neoplastic implants, and biopsy confirmed non-Hodgkin lymphoma. The patient developed persistent hypoglycemia refractory to oral intake and intravenous dextrose. During hypoglycemia (glucose 40 mg/dL), laboratory tests showed findings consistent with endogenous hyperinsulinemic hypoglycemia: C-peptide 3.11 ng/mL, insulin 20.8 µU/mL, proinsulin 2.37 pmol/L, beta-hydroxybutyrate 0.10 mmol/L, cortisol 18.4 µg/dL. Abdominal MRI excluded insulinoma. Prednisone 60 mg/day was initiated for advanced follicular non-Hodgkin lymphoma. After prednisone withdrawal and rituximab initiation, hypoglycemia resolved. Anti-insulin antibodies were negative, while anti-insulin receptor antibodies were positive, confirming TBIRS. TBIRS is a rare autoimmune disorder caused by antibodies against the insulin receptor, resulting in impaired insulin signaling and hyperinsulinemia. Although hyperglycemia is the usual presentation, our patient developed isolated hypoglycemia, a very uncommon manifestation probably related to the insulin-like effect of these antibodies, making diagnosis challenging. TBIRS is commonly associated with autoimmune diseases, but rare paraneoplastic associations with hematological malignancies have been reported. In this case, the coexistence of follicular non-Hodgkin lymphoma reinforces this rare association. Anti-insulin receptor antibody detection is technically difficult; however, testing was successfully performed and confirmed the diagnosis. Immunosuppressive treatment led to glycemic improvement, with restoration of euglycemia after prednisone withdrawal and rituximab therapy. Type B insulin resistance syndrome is a rare and challenging condition that may present with isolated hypoglycemia. This case highlights its association with follicular non-Hodgkin lymphoma and the importance of considering TBIRS in hyperinsulinemic hypoglycemia without insulinoma. Improvement after immunosuppression and rituximab supports the role of immune modulation in disease control.

Keywords: type B insulin resistance syndrome; hyperinsulinemic hypoglycemia; follicular non-Hodgkin lymphoma.

OTHERS

389 (3244) INSULINOMA AFTER BARIATRIC SURGERY: A DECEIVING DIAGNOSIS

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Case presentation: A 43-year-old male was sent to our institution to investigate persistent hypoglycemia due to recurrence of an insulinoma. He had undergone a Roux-en-Y gastric bypass at age of 37 to treat grade III obesity (BMI of 45.7 kg/m² = 150 kg), losing 35.5% of initial weight. Two years later, he developed recurrent symptomatic hypoglycemia (HYPO) (capillary glucose 30–50 mg/dL, fasting and postprandial). An MRI revealed a 5.2 cm solid-cystic lesion in the pancreatic tail, and, although the available laboratory tests showed normal fasting insulin (5.4 mIU/mL), the diagnosis of insulinoma was assumed. A distal pancreatectomy with splenectomy was performed. He had complete remission of symptoms for two years, when postprandial symptomatic HYPO recurred (60–70 mg/dL, 60–120 min post meal) and was sent for investigation. He had regained 6.5 kg (BMI of 29.5 kg/m²). At this time, he had an abnormal oral 75 g glucose tolerance test (plasma glucose of 98, 235 and 102 mg/dL at 0, 60 and 120 min), normal fasting serum insulin 8.12 mIU/mL and C-peptide 1.75 ng/mL and negative anti-insulin antibodies. Imaging showed no recurrent pancreatic lesion. An interstitial continuous glucose monitoring (CGM) device was installed, showing several post prandial hyperglycemic episodes, followed by HYPO, down to 54 mg/dL, mainly 1.5–2 hours after meals. He was advised to take a low-carbohydrate diet with increased fibers, with some improvement of symptoms. Pathological analysis of the pancreatic lesion showed pancreatic acinar hyperplasia without malignancy. Immunohistochemistry was negative for neuroendocrine neoplasia; Ki-67 below 1%, not compatible with the diagnosis of insulinoma. **Discussion:** Post-bariatric hypoglycemia (PBH) is a late gastric bypass complication (prevalence 0.1%–11%). It is attributed to accelerated gastric transit, higher GLP-1 hypersecretion, and beta-cell hyperplasia (acquired nesidioblastosis). In this case, a pancreatic lesion led to the diagnosis of insulinoma that was lately identified as pancreatic acinar hyperplasia. Despite an initial remission, HYPO episodes recurred with no lesion on imaging, reinforcing the hypothesis. **Final comments:** This report illustrates an uncommon nodular pancreatic acinar hyperplasia and symptomatic post-bariatric hypoglycemia, with surgical remission and late functional recurrence leading to the misdiagnosis of insulinoma. It underscores the need for careful differential diagnosis in cases of PBH.

Keywords: post-bariatric hypoglycemia; pancreatic acinar hyperplasia; nesidioblastosis.

OTHERS

390 (3297) KEFIR INHIBITS TUMOR DEVELOPMENT ASSOCIATED WITH INCREASED COLONIC CANNABINOID RECEPTOR 2 (CB2) EXPRESSION IN NEONATAL OVERFED ANIMALS

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Introduction: Neonatal overfeeding promotes increased adiposity in adulthood, which is associated with altered gut microbiota and chronic diseases such as colorectal cancer (CRC). Recent evidence suggests that the endocannabinoid system (ECS) interacts with the gut microbiome. Short-chain fatty acids (SCFAs), gut microbiota-derived metabolites, and cannabinoid receptors 1 (CB1) and 2 (CB2) have been reported to influence intestinal homeostasis and tumorigenesis. We aimed to investigate the impact of kefir, a probiotic source, on adiposity, tumorigenesis, SCFA levels, and the expression of CB1 and CB2 in the colon in a neonatal overfeeding-induced model of CRC. **Methods:** Rats were allocated into four groups: Control (C), Control Kefir (CK), Overfed (O), and Overfed Kefir (OK). Neonatal overfeeding was induced by litter size reduction to 3 pups per dam. During lactation, dams received daily gavage of water (O) or kefir (OK), and offspring continued the same treatment until 60 days of age. CRC was induced at postnatal day 67 using 1,2-dimethylhydrazine. At 240 days of age, visceral adiposity, colon tumor number, cecal SCFA concentrations by high-performance liquid chromatography, and colonic CB1 and CB2 protein levels by western blotting were evaluated. Statistical analyses were performed using two-way ANOVA followed by Sidak's post hoc test. All procedures were approved by the ethics committee of the Federal University of Juiz de Fora (protocol no. 21/2016). **Results:** The O group showed increased colon tumor number ($p < 0.05$), butyric acid levels (+43%, $p = 0.0028$), propionic acid levels (+27%, $p = 0.0296$), and adiposity (+38%, $p < 0.0001$) compared with the other groups. Importantly, kefir consumption markedly reduced tumor number in overfed animals (–71.43%, $p < 0.01$), while post hoc analysis additionally revealed increased colonic CB2 expression (12-fold; $p = 0.053$) and reduced adiposity (–17%, $p = 0.0102$) in the OK group compared with the O group. **Conclusion:** Kefir exerted a protective effect against colorectal cancer, which was associated with increased CB2 expression and reduced adiposity in animals overfed during the neonatal period. These findings suggest that the endocannabinoid system, particularly CB2 signaling, may represent a potential mechanism underlying the protective effects of early kefir consumption against colorectal tumorigenesis.
Keywords: kefir; cannabinoid receptor 2 (CB2); colorectal cancer (CRC).

OTHERS

391 (3090) METFORMIN FOR GLYCEMIC CONTROL IN BURN PATIENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Thermal trauma triggers a profound hypermetabolic response driven by the release of cortisol, glucagon, and catecholamines, resulting in hepatic gluconeogenesis, peripheral insulin resistance, and marked hyperglycemia, which independently worsens immune function, wound healing, and survival. Because burn-induced insulin resistance limits glycemic efficacy and insulin itself carries an inherent risk of hypoglycemia, alternative agents are of considerable clinical interest. Metformin, a biguanide with over six decades of use in type 2 diabetes, suppresses hepatic gluconeogenesis and enhances insulin sensitivity via AMPK-dependent and independent pathways, and emerging evidence suggests it may also attenuate hypermetabolism, reduce protein catabolism, and modulate inflammatory responses in burn patients. **Objective:** To evaluate the use of metformin versus standard of care on short-term mortality and metabolic outcomes in hospitalized patients with burns. **Methods:** We systematically searched PubMed, Embase, and Cochrane Library from inception to November 2025 for studies comparing metformin versus standard care in hospitalized patients with burns. The primary outcome was short-term mortality; secondary outcomes included mean plasma glucose, length of hospital stay, hypoglycemic events, and lactic acidosis. End points were reported using risk ratio (RR) for binary outcomes and mean difference (MD) for continuous outcomes, with 95% confidence intervals were calculated using a random-effects model. All statistical analyses were performed on Review Manager software. **Results:** Four studies comprising 3,014 patients were included. Metformin was associated with reduced short-term mortality (RR 0.17; 95% CI 0.11–0.27; $p < 0.00001$) and lactic acidosis (RR 0.18; 95% CI 0.10–0.33; $p < 0.00001$). No statistically significant differences were observed in mean plasma glucose (MD –1.02; 95% CI –39.20 to 37.15; $p = 0.96$), hypoglycemia (RR 0.35; 95% CI 0.07–1.67; $p = 0.19$), or length of stay (MD –3.57; 95% CI –12.92 to 5.77; $p = 0.45$). **Conclusion:** Metformin shows promise in reducing short-term mortality and appears safe with respect to lactic acidosis in hospitalized burn patients, though current evidence derives predominantly from observational cohorts. These findings support metformin as a compelling adjunct to blunt burn-associated hypermetabolism and further investigation in randomized controlled trials.
Keywords: metformin; hyperglycemia; burns.

OTHERS

392 (3493) NON-ISLET CELL HYPOGLYCEMIA MEDIATED BY IGF-2 AS A LATE MANIFESTATION OF METASTATIC HEPATIC SOLITARY FIBROUS TUMOR

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A 49-year-old male with a history of primary central nervous system hemangiopericytoma treated with surgical resection and radiotherapy 10 years earlier was undergoing oncologic follow-up without evidence of recurrence. In January 2025, he developed episodes of disorientation, blurred vision, and diaphoresis relieved by food intake, associated with right upper quadrant pain. Initial investigation with abdominal ultrasonography revealed a large hepatic mass, later confirmed by computed tomography as a 14.9 × 12.1 × 7.9 cm lesion with adjacent vascular compression. During fasting for percutaneous biopsy, he developed confusion associated with severe hypoglycemia (37 mg/dL). The patient remained hospitalized due to recurrent episodes that became progressively refractory to intravenous glucose infusion. He denied insulin or hypoglycemic agent use. Liver biopsy demonstrated metastatic neoplasm with hemangiopericytoma-like morphology and immunohistochemistry suggestive of solitary fibrous tumor. A prolonged fasting test was performed, during which plasma glucose was 25 mg/dL, with undetectable insulin, suppressed C-peptide, IGF-1 < 15 ng/mL, and cortisol of 14.2 µg/dL, suggesting IGF-2-mediated non-islet cell hypoglycemia. IGF-2 level was 992 ng/mL, with an IGF-2/IGF-1 ratio of 66, confirming the diagnosis. Prednisone 40 mg/day was initiated, resulting in partial improvement of hypoglycemia. Due to vascular invasion, the tumor was considered unresectable, and hepatic chemoembolization was performed. After the first session, the patient had no further hypoglycemic episodes and remained asymptomatic during outpatient follow-up. IGF-2-mediated non-islet cell hypoglycemia is a rare and potentially severe paraneoplastic manifestation, mainly associated with large mesenchymal tumors. Early recognition is essential, as tumor-directed therapy may result in clinical control and reversal of hypoglycemia.
Keywords: hypoglycemia; IGF-2; solitary fibrous tumor.

OTHERS

393 (3367) NON-ISLET CELL TUMOR HYPOGLYCEMIA ASSOCIATED WITH HEPATIC HEMANGIOENDOTHELIOMA: A RARE ENDOCRINE PARANEOPLASTIC SYNDROME

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A 58-year-old previously healthy woman underwent a CT scan for low back pain, revealing a 6.9 × 5.0 cm liver lesion in segment IV. She subsequently developed progressive abdominal enlargement and severe hypoglycemia (20 mg/dL) associated with syncope, reversed with hypertonic glucose, fulfilling Whipple's triad. Follow-up imaging demonstrated tumor growth to 22.9 × 12.9 x 27.5 cm. Laboratory investigation confirmed non-hyperinsulinemic hypoglycemia. Due to recurrent episodes, corticosteroid therapy was initiated until surgical intervention. IGFBP-2 level was 240 ng/mL, while receiving glucocorticoids. The patient underwent left hepatectomy, with immediate resolution of hypoglycemias. Anatomopathological and immunohistochemical analyses confirmed a low-grade hepatic vascular mesenchymal neoplasm (hemangioendothelioma), positive for CD34, with a Ki-67 proliferation index of 15%. The patient remained asymptomatic for approximately three years until severe hypoglycemia recurred. Imaging studies revealed tumor recurrence, and laparoscopy identified multiple peritoneal metastases, rendering surgical resection unfeasible. Glycemic control was achieved with prednisone (30–60 mg/day), and the patient remains under endocrinology and oncology follow-up. Non-islet cell tumor hypoglycemia (NICTH), also known as IGF-2oma, is a rare syndrome caused by excessive production of IGF-2 or "big" IGF-2, usually associated with large mesenchymal tumors. Despite the absence of tumor IGF-2 expression analysis, diagnosis was supported by the clinical and laboratory findings, including neuroglycopenic symptoms, non-hyperinsulinemic hypoglycemia, giant hepatic hemangioendothelioma, postoperative resolution, and recurrence associated with metastatic disease. Cytoreductive surgery is the treatment of choice, while glucocorticoids are effective for hypoglycemia control. Late recurrence of hypoglycemic episodes may represent a clinical marker of tumor recurrence, reinforcing the need for long-term monitoring. NICTH is a rare and frequently underdiagnosed condition. Large tumors associated with hypoglycemia and suppressed insulin levels should raise suspicion for IGF-2oma. Early recognition and multidisciplinary management are essential for metabolic control and preservation of quality of life.
Keywords: hypoglycemia; insulin-like growth factor II; hemangioendothelioma.

OTHERS

394 (3378) OPTIMIZING GH POST-GLUCAGON COLLECTION TIMES: IS IT FEASIBLE TO COLLECT SAMPLES IN FEWER TIMES WITHOUT COMPROMISING TEST ACCURACY?

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Introduction: The growth hormone (GH) stimulation test is indicated for the investigation of growth hormone deficiency (GHD) in children with short stature, reduced growth velocity, or hypothalamic-pituitary disorders. Traditional protocols include serial sampling for up to 180 minutes; however, recent evidence suggests that most peaks occur within the first 120–150 minutes after pharmacological stimulation, calling into question the necessity and cost-effectiveness of early measurements. Streamlining the protocol may improve pediatric patient comfort, reduce laboratory costs, and optimize workflow without compromising the diagnostic accuracy of the test. **Objective:** The objective is to evaluate whether GH levels obtained at baseline, 30, and 60 minutes modify the diagnostic interpretation of the glucagon stimulation test in children and adolescents. **Methods:** This is a retrospective study analyzing GH stimulation tests using 1 mg subcutaneous glucagon in patients up to 18 years of age, with GH levels available at 0, 30, 60, 90, 120, and 150 minutes. The GH peak was defined as an adequate response (peak ≥ 5 ng/mL). Records with incomplete data at the specified time points were excluded. **Results:** A total of 27 patients were included (63.0% male; mean age 8.7 ± 3.2 years; range: 3–14 years). According to the full protocol, 16 (59.3%) showed an adequate response and 11 (40.7%) were classified as having GHD. The peak occurred at 150 minutes in 14 patients (51.9%), at 120 minutes in 6 (22.2%), and at 90 minutes in 3 (11.1%); only 4 (14.8%) reached peak levels at early time points. When T0, T30, and T60 were omitted and only T90, T120, and T150 were retained, diagnostic agreement with the full protocol was 92.6% (25/27 cases). **Conclusion:** Sampling at baseline, 30, and 60 minutes was not essential for the diagnostic interpretation of most glucagon stimulation tests in this sample. The concentration of peaks at later time points and the high agreement after omission of early sampling support the adoption of more streamlined protocols, with direct benefits for pediatric patient comfort, reduced laboratory costs, and improved operational efficiency.
Keywords: growth hormone deficiency; glucagon stimulation test; pediatric endocrinology.

OTHERS

395 (3326) PARANEOPLASTIC HYPOGLYCEMIA IN LARGE-CELL HEPATIC NEUROENDOCRINE CARCINOMA: THERAPEUTIC CHALLENGES AND FATAL OUTCOME IN A CASE REPORT

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Case report: Male patient, 45 years old, previously healthy, with right upper quadrant pain since 2023 and progression to cholestatic syndrome in 2025. Imaging revealed a 16 × 16 cm hepatic mass involving the hepatic hilum, associated with lymphadenopathy. Liver biopsy with immunohistochemistry, positive for Synaptophysin and Ki-67 (95%), confirmed large cell neuroendocrine carcinoma (LCNEC). He developed obstructive cholangitis treated with biliary stent and inferior vena cava and iliac vein thrombosis. During hospitalization, he presented recurrent severe hypoglycemia (<20 mg/dL) with seizures. Investigation demonstrated insulin of 17.3 µU/mL, C-peptide of 8.8 ng/mL, and IGF-1 of 91 ng/mL, suggesting paraneoplastic hypoglycemia. He required continuous glucose infusion, dexamethasone, verapamil, and octreotide and calcium channel agonist diazoxide with partial response. After chemotherapy with cisplatin and etoposide, hypoglycemic episodes decreased. Subsequently, he presented disease progression and recurrence of hypoglycemia, undergoing second-line therapy with irinotecan and fluorouracil, associated with lanreotide. He developed febrile neutropenia and pneumonia, was referred to exclusive palliative care, and progressed with multiple organ failure. He died in April 2026. **Discussion:** LCNEC of the liver is rare and aggressive with a median survival of less than 16 months. The patient's clinical course with abdominal pain, cholestatic syndrome, and obstructive cholangitis reflects the nonspecific and late presentation of these tumors. The striking complication was paraneoplastic hypoglycemia (NICTH), mediated by tumoral secretion of IGF-II, resulting in seizures and need for intensive support. Management with glucocorticoids and octreotide had partial response, reinforcing that isolated metabolic control is insufficient without reduction of tumor burden. Systemic treatment with platinum/etoposide and irinotecan/fluorouracil followed NCCN guidelines, but the rapid progression illustrates the limitation of conventional chemotherapy. Clinical trials investigate immunotherapy and targeted therapies, which may offer alternatives for similar patients. **Conclusion:** The case report reveals the aggressiveness of LCNEC and the complexity of paraneoplastic hypoglycemia. Early recognition of NICTH and a multidisciplinary approach are essential for clinical control, although the prognosis remains unfavorable due to rapid tumor progression and limited therapeutic response.
Keywords: paraneoplastic endocrine syndromes; insulinoma; hypoglycemia.

OTHERS

396 (3471) PARATHYROID CARCINOMA IN A YOUNG PATIENT: DIAGNOSTIC CHALLENGES IN AN ATYPICAL PRESENTATION OF PRIMARY HYPERPARATHYROIDISM

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Presentation of case: A 33-year-old previously healthy woman from Manaus, Amazonas, Brazil, presented with a three-month history of progressive bone pain involving the left tibia and lumbosacral spine, associated with severe functional impairment, prolonged constipation, recurrent urinary retention requiring weekly bladder catheterization, and depressive symptoms. On admission, she reported intense pain in the left lower limb associated with a recent pathological fracture of the left tibia, resulting in bedridden status and marked limitation of mobility. Laboratory tests revealed severe hypercalcemia (total calcium: 14 mg/dL; ionized calcium: 2.01 mmol/L) and markedly elevated parathyroid hormone (PTH) levels (1892.8 pg/mL), suggesting severe primary hyperparathyroidism. Cervical ultrasonography demonstrated a 2.5 × 1.9 cm nodule adjacent to the right thyroid lobe, while abdominal ultrasonography revealed micronephrolithiasis. Cranial magnetic resonance imaging identified bilateral frontal bone nodular lesions measuring up to 3 cm, compatible with skeletal involvement secondary to hyperparathyroidism. Parathyroidectomy was performed on December 16, 2025. Intraoperatively, an enlarged right superior parathyroid gland with nodular and calcified appearance was identified. Histopathological examination demonstrated an epithelial neoplasm composed of clear cells associated with fibrous bands and hemorrhagic areas, measuring 3.0 cm at its greatest dimension. Immunohistochemical analysis showed positivity for CAM5.2, GATA3, chromogranin, synaptophysin, and focal PAX8 expression, with a Ki-67 proliferative index of approximately 7%, supporting the diagnosis of parathyroid carcinoma. **Discussion:** Parathyroid carcinoma is a rare endocrine malignancy responsible for less than 1% of primary hyperparathyroidism cases. Diagnosis is challenging because clinical and imaging findings may overlap with benign disease. Severe hypercalcemia, markedly elevated PTH levels, and systemic manifestations should raise suspicion for malignant etiology. This case demonstrates a severe presentation in a young patient, with skeletal involvement, pathological fracture, nephrolithiasis, and neuropsychiatric manifestations. Histopathological and immunohistochemical findings were essential for definitive diagnosis. **Final comments:** Early recognition and prompt surgical treatment are essential to reduce morbidity and improve outcomes in parathyroid carcinoma.
Keywords: severe hypercalcemia; parathyroid carcinoma; primary hyperparathyroidism.

OTHERS

397 (3535) PHARMACOGENOMIC VARIANTS ASSOCIATED WITH GLP-1 RECEPTOR AGONIST RESPONSE: COMPARISON BETWEEN BRAZILIAN ADMIXED AND GLOBAL POPULATIONS

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Introduction: GLP-1 receptor agonists (GLP-1RA) have revolutionized obesity and type 2 diabetes treatment but show substantial interindividual variability in efficacy and adverse events. Recent genome-wide association studies (GWAS) identified variants in drug target genes (GLP1R, GIPR) associated with therapeutic response. Population-specific data are needed to interpret the clinical relevance of these pharmacogenomic variants in Brazil's admixed population. **Objective:** Compare the frequency of pharmacogenomic variants associated with GLP-1RA response between the admixed Brazilian population and global reference populations. **Methods:** Cross-sectional population-based genomic study analyzing whole-genome data from the ABraOM/SABE-1171-WGS cohort, comprising 1,171 admixed Brazilians. Pharmacogenomic variants identified via systematic PubMed review (69 studies screened, 1 GWAS included) using genome-wide significance threshold of P < 5 x 10-8. Four variants were analyzed: GLP1R p.Pro7Leu (rs10305420), associated with GLP-1 receptor agonist efficacy/weight loss; rs9357296 in the GLP1R locus, associated with nausea; rs11760106 in the GLP1R locus, associated with vomiting; and GIPR p.Glu354Gln (rs1800437), associated with tirzepatide-induced vomiting. Allele frequencies and homozygote counts were extracted and compared with gnomAD v4 across global and ancestry-specific populations using frequency ratios and multi-ancestral context analysis. **Results:** Four variants were common in ABraOM. GLP1R variants showed high frequencies: rs11760106, associated with vomiting, reached 32.8%; p.Pro7Leu/rs10305420, associated with efficacy, 28.7%; and rs9357296, associated with nausea, 24.0%. These closely matched Latino/Admixed Americans, were lower than Europeans, and were 3.4-3.9-fold higher than African/African Americans. In contrast, GIPR p.Glu354Gln/rs1800437, associated with tirzepatide-induced vomiting, was 38% higher than Latino/Admixed Americans, lower than Europeans, and closer to African/African Americans. **Conclusion:** GLP-1RA pharmacogenomic variants are common in Brazil's admixed population. GLP1R variants showed a pan-American pattern, whereas GIPR p.Glu354Gln showed a distinct Brazilian profile. These findings challenge the direct application of Eurocentric pharmacogenomic models and highlight the need for population-specific clinical studies to validate their impact on efficacy and tolerability, advancing precision medicine for GLP-1RA therapy in Brazilian patients.
Keywords: GLP-1 receptor agonists; pharmacogenomics; Brazil.

OTHERS

398 (3183) PREVALENCE OF BODY DYSMORPHIC DISORDER SYMPTOMS AMONG MEDICAL STUDENTS: A CROSS-SECTIONAL ANALYSIS BY SEX DURING AND AFTER THE COVID-19 PANDEMIC

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Body dysmorphic disorder (BDD) manifests as an excessive preoccupation with one or more imperceptible physical flaws in one's appearance. In the context of medical education, BDD gains epidemiological relevance, as high stress levels, self-demand, and social exposure can act as triggers or aggravating factors for body image distortion, demanding careful attention to students' mental health. This study aims to compare the prevalence of body dysmorphic disorder symptoms in medical students during and after the pandemic, with an analysis by sex. This is a cross-sectional, observational, ecological study conducted during and after the COVID-19 pandemic through the application of a voluntary, anonymous questionnaire to medical students, following the signing of an informed consent form, regarding the frequency of self-reported symptoms related to BDD. A total of 432 students were interviewed, of whom 145 (33.1%) were male and 289 (66.9%) were female. The mean age was 22.23 years (16 ± 65), with an average weight of 69.44 kg and a height of 1.68 m. A higher prevalence of body dysmorphic disorder indicators was observed among female participants, who accounted for approximately 78.9% of the identified cases: 71.8% reported having some preoccupation with their physical appearance that is imperceptible or barely noticed by others ($p < 0.001$); reported frequent comparisons with others regarding physical appearance ($p = 0.025$); the preoccupation is linked to supposed excess body fat ($p = 0.004$); and this preoccupation disrupts social life ($p = 0.007$). The study demonstrated that female medical students showed a higher prevalence of body dysmorphic disorder symptoms. The significant prevalence of these indicators reinforces the need for psycho-pedagogical support and early interventions within higher education institutions. Identifying these symptoms early is crucial to prevent body dysmorphism from evolving into depressive conditions or severe eating disorders, ensuring not only the students' mental health but also the quality of their future clinical practice.
Keywords: body dysmorphic disorder; medical students; mental health.

OTHERS

399 (3525) PSEUDODEFICIENCY VARIANTS IN LYSOSOMAL METABOLISM GENES: BRAZILIAN-SPECIFIC DATA TO IMPROVE NEWBORN ENZYMATIC SCREENING INTERPRETATION

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Introduction: Lysosomal disorders are inborn errors of metabolism caused by enzyme deficiencies, included in Brazil's newborn screening program. These progressive multisystem diseases include mucopolysaccharidoses, Fabry, Gaucher, and Pompe disease, presented with variable combinations of skeletal dysplasia, organomegaly, neurologic, cardiac, renal, and endocrine manifestations, such as growth retardation and hypogonadism. Some individuals carry pseudodeficiency alleles, genetic variants that reduce enzymatic activity without causing these diseases, generating false-positive screening results. Population-specific genomic data are needed to interpret enzymatic results in Brazil's admixed population. **Objective:** Compare the frequency of pseudodeficiency variants in lysosomal metabolism genes between the admixed Brazilian population and global reference populations. **Methods:** This cross-sectional population-based genomic study analyzed whole-genome data from the ABraOM/SABE-1171-WGS cohort, comprising 1,171 admixed Brazilians. Nineteen pseudodeficiency alleles in ARSA, ARSB, GAA, GALC, GLA, GLB1, HEXA, and IDUA were defined a priori from the 2022 ACMG Technical Standard. Allele frequencies and homozygote counts were extracted and compared with gnomAD v4 across global and five ancestry populations using frequency ratios and multi-ancestral context analysis. **Results:** Of 19 prespecified pseudodeficiency alleles, 15 were observed in the Brazilian cohort. The most frequent were GALC c.1685T>C (45%), ARSB c.1072G>A (35%), ARSA c.1055A>G (22%), and GALC c.742G>A (14%). Substantial numbers of homozygotes were identified for these variants, who face increased risk of false-positives in enzymatic screening. Compared with global data, Brazilians showed marked enrichment for IDUA variants c.667G>A (6.5-fold), c.965T>A (5-fold), and c.235G>A (2.7-fold). Brazilian frequencies did not align with any single ancestry group, reinforcing the need for population-specific reference data when interpreting enzymatic screening results. **Conclusion:** This study provides the first Brazilian genomic curation of lysosomal pseudodeficiency alleles, demonstrating population-specific frequency differences for clinically relevant alleles in inherited metabolic disorders. The findings support Brazilian-specific validation of molecular second-tier testing, provide a preliminary reference for interpreting altered enzymatic results, and prioritize candidate alleles for genotype-enzyme activity studies.
Keywords: inborn errors of metabolism; pseudodeficiency alleles; Brazil.

OTHERS

400 (2913) RECURRENT HYPOGLYCEMIA AND LACTIC ACIDOSIS: TWO MOLECULARLY CONFIRMED CASES OF FBP1 DEFICIENCY

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Case presentation: Case 1: A 30-year-old woman under genetic investigation due to a history of recurrent hypoglycemia, with multiple hospitalizations and episodes of metabolic acidosis and hyperlactatemia. She developed metabolic coma at age 28, requiring dialysis support and ICU admission. She reported worsening of symptoms during prolonged fasting and metabolic stress. Previous genetic investigation had identified variants of uncertain significance. During the investigation, she presented with mild metabolic acidosis and elevated lactate levels, with ketones usually negative. Pregnancy was identified. A subsequent genetic panel identified two pathogenic variants in compound heterozygosity in the FBP1 gene (c.472C>T; p.Arg158Trp and c.960_961insC; p.Ser321 Valfs*13), confirming the diagnosis of fructose-1,6-bisphosphatase deficiency. Case 2: A male patient, aged 1 year and 6 months, born to non-consanguineous parents, was referred due to hypoglycemia, metabolic acidosis, hyperlactatemia, and multiple hospitalizations, including ICU admissions. He developed episodes of hypotonia, tachypnea, severe dehydration, and metabolic disturbances after the introduction of complementary feeding, with worsening during fasting and infections. Initial metabolic screening results were normal, and he had a small hepatic hemangioma. Exome sequencing identified two compound heterozygous pathogenic variants in the FBP1 gene (c.2210_225dup and c.723T>G), confirming the diagnosis. Both patients are receiving uncooked cornstarch at regular intervals, with fructose/sucrose restriction and avoidance of prolonged fasting, showing clinical and laboratory improvement. **Discussion:** FBP1 deficiency is an inborn error of gluconeogenesis. It manifests with acute episodes of lactic acidosis and hypoglycemia, associated with hyperventilation, apnea, seizures, and/or coma. Acute crises are precipitated by fasting, infections, or catabolic stress. The diagnosis is established based on clinical findings and the identification of pathogenic variants in the FBP1 gene. Management consists of preventing hypoglycemia by avoiding fasting and catabolic states. During pregnancy and labor, close glucose monitoring and glucose infusion may be required. **Final comments:** These cases highlight that FBP1 deficiency should be considered in recurrent episodes of hypoglycemia and lactic acidosis at any age. Molecular diagnosis enables targeted dietary management and may prevent severe metabolic decompensation.

Keywords: ketotic hypoglycemia; lactic acidosis; inborn errors of metabolism.

OTHERS

401 (3013) SARCOPENIC OBESITY IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE: PREVALENCE AND BODY COMPOSITION PROFILE

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Introduction: Sarcopenic obesity (SO) and metabolic dysfunction-associated steatotic liver disease (MASLD) share common pathophysiological mechanisms, primarily involving insulin resistance and de novo lipogenesis. The coexistence of these conditions may result in a twofold increase in mortality risk compared with the general population. To date, data regarding SO in the Brazilian population with MASLD remain limited. **Objective:** To determine the prevalence of SO and compare body composition parameters in adults with and without MASLD. **Methods:** This was an observational, cross-sectional study with prospective data collection involving adults with at least one MASLD risk factor, such as obesity, type 2 diabetes mellitus (T2DM), and metabolic syndrome (MS). MASLD was assessed via abdominal ultrasonography and hepatic elastography to identify the presence of steatosis and/or liver fibrosis. Appendicular skeletal muscle mass (ASMM) and body fat percentage (%BF) were evaluated using dual-energy X-ray absorptiometry (DXA). SO was defined as an ASMM-to-weight ratio (ASMM/weight%) < 19.4% for women or < 25.7% for men, combined with a %BF exceeding 40% and 30%, respectively. Reduced muscle strength was assessed by handgrip strength (HGS) and defined as low if < 16 kg for women and < 27 kg for men. Data are presented as medians (p25–p75) or n (%). **Results:** The study included 176 participants (median age 60.5 [52–66] years), of whom 144 (81.2%) were diagnosed with MASLD. Comparing groups with and without MASLD, absolute ASMM did not differ significantly [19.2 (16.6–23.1) vs. 18.6 (17.0–22.1) kg; p = 0.88]. However, when ASMM was adjusted for weight, the MASLD group exhibited a significantly lower ASMM/weight% [23.40 (21.82–25.77)% vs. 25.47 (23.26–30.82)%; p < 0.01] and a higher body fat percentage [45.1 (39.5–48.7)% vs. 34.6 (30.9–46.0)%; p < 0.01]. In the MASLD group, 16 participants (11.1%) presented with SO, whereas no cases were identified in the non-MASLD group (p < 0.05). No reduction in handgrip strength was observed in the SO group. **Conclusion:** SO was identified exclusively in patients with MASLD, with a prevalence of 11.1%, reinforcing the close relationship between these conditions. The current handgrip strength criteria may underestimate sarcopenia in this population, suggesting a need to re-evaluate diagnostic thresholds specifically for patients with MASLD.

Keywords: hepatic steatosis; sarcopenic obesity; body composition.

OTHERS

402 (3095) SPLICE-SITE VARIANTS IN THE AR GENE AND ANDROGEN INSENSITIVITY SYNDROME: SYSTEMATIC REVIEW AND FUNCTIONAL CONFIRMATION BY CDNA ANALYSIS

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Introduction: Androgen insensitivity syndrome (AIS) is an X-linked condition caused by pathogenic variants in the androgen receptor (AR) gene and represents one of the main causes of 46,XY differences/disorders of sex development (DSD). Variants affecting splice sites in the AR gene are rare but are frequently associated with the complete androgen insensitivity syndrome (CAIS) phenotype. Nevertheless, functional confirmation at the RNA level remains limited for most reported cases. **Objective:** To describe the functional impact of a novel canonical splice-site variant in the AR gene in a patient with CAIS and to review previously reported splice-site variants associated with AIS. **Methods:** Molecular analysis was performed by PCR and Sanger sequencing of the coding exons and adjacent intronic regions of the AR gene. RNA analysis included total RNA extraction, cDNA synthesis, and RT-PCR using primers spanning exons 5 to 8, followed by sequencing. A literature review and in silico analysis of AR splice-site variants were also performed using SpliceAI and Human Splicing Finder. **Results:** A novel canonical splice-site variant, c.2319-1G>T, was identified in intron 5 of the AR gene in a 46,XY patient with a CAIS phenotype. cDNA analysis demonstrated complete exon 6 skipping, with direct joining of exons 5 and 7, resulting in an in-frame deletion of 44 amino acids within the ligand-binding domain (LBD) of the androgen receptor. The literature review identified 14 previously reported AR splice-site variants, of which 92.8% were associated with the CAIS phenotype. In silico tools showed high concordance in predicting splice defects. **Conclusion:** The c.2319-1G>T variant caused a significant functional alteration in RNA processing, leading to exon 6 skipping and impairment of androgen receptor function. These findings expand the molecular spectrum of AIS and reinforce the strong association between canonical AR splice-site variants and the CAIS phenotype. Furthermore, they highlight the importance of RNA analysis as an essential complementary tool for the interpretation of splice-associated genetic variants.

Keywords: androgen insensitivity syndrome; AR gene; RNA splicing.

OTHERS

403 (3355) SPONTANEOUS PREGNANCIES IN WOMEN WITH MOSAIC TURNER SYNDROME: A CASE SERIES OF FAVORABLE MATERNAL-FETAL OUTCOMES

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Case reports: We describe three women with mosaic 45,X/46,XX karyotype, aged 19, 31 and 34 years, with spontaneous menarche, preserved menstrual cycles, and spontaneous pregnancies. They were followed by a multidisciplinary team. None of them developed gestational diabetes, hypertensive disorders, or thyroid dysfunction. All newborns presented normal karyotypes and no congenital malformations. The first pregnant had an ostium secundum atrial septal defect and third-trimester fetal growth restriction. She required cesarean delivery at 41 weeks due to acute fetal distress, resulting in a healthy female newborn (2690 g, Apgar 8/9). The second woman had mitral valve prolapse with trivial regurgitation, followed by an uneventful pregnancy and vaginal delivery of a healthy 2985 g male (Apgar 8/9). The third pregnant had a previous miscarriage and a surgically corrected atrioventricular septal defect with mild residual regurgitation; she is currently at 20 weeks of gestation without complications. **Discussion:** Spontaneous pregnancy in Turner syndrome (TS) occurs in 2%–8% of patients and is more frequently associated with mosaic karyotype and spontaneous menarche, findings observed in all cases presented in this series. TS is associated with premature ovarian insufficiency, contributing to the high prevalence of infertility. Although TS in this case series achieved spontaneous pregnancies at the ages of 31 and 34 years, this is not the usual pattern. Women with TS should be counseled early on the risk of premature menopause and their reproductive options. Pregnancy in TS carries a high risk of maternal-fetal complications, including miscarriage, fetal growth restriction, hypertensive disorders, and life-threatening aortic dissection. Multidisciplinary follow-up at our center may ensured favorable outcomes, despite underlying structural heart disease. These cases reinforce the importance of preconception cardiovascular assessment, cross-disciplinary communication and enhanced surveillance. **Final comments:** Our findings support existing evidence that spontaneous pregnancy may occur in women with TS presenting mosaic karyotype and spontaneous menarche. Also, our results highlight the possibility of favorable pregnancy outcomes in carefully monitored women. However, important challenges remain, particularly regarding fertility counseling and reproductive planning.

Keywords: Turner syndrome; spontaneous pregnancy; mosaicism.

OTHERS

404 (3180) STUDY OF BINGE EATING BEHAVIOR AMONG MEDICAL STUDENTS DURING AND AFTER THE COVID-19 PANDEMIC

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Introduction: The COVID-19 pandemic imposed numerous restrictions that culminated in a broad psychosocial effect, with deleterious effects on mental health. A meta-analysis observed an increased prevalence of eating behaviors such as binge eating, purging behaviors, and food restriction. **Objective:** To evaluate the point prevalence of eating behavior symptoms suggestive of binge eating disorder in medical students during and after the pandemic period. **Patients and methods:** This is a cross-sectional observational study on the prevalence of self-reported symptoms suggestive of binge eating disorder (BED) and bulimia nervosa (BN). 434 medical students, aged between 17 and 40 years, were evaluated through the application of an anonymous questionnaire regarding symptoms of binge eating disorder. Regarding the statistical approach, qualitative variables were analyzed for their association with body mass index using Pearson's χ^2 method, and quantitative variables were analyzed using the Mann-Whitney or Kruskal-Wallis analysis. This study was approved by the Research Ethics Committee under opinion number 4.206.836. **Results:** A higher prevalence of several symptoms associated with binge eating was identified in the total group during the pandemic compared to the post-pandemic period. A questionnaire was applied that identified significant differences in the following questions: Q1 – Have you experienced episodes of overeating for a specific period? Q1.2 – Did you feel a loss of control during the episode? Q3 – Do you feel like you eat until you feel uncomfortably full? Q4 – Do you necessarily feel hungry when these episodes occur? Q5 – Do you feel ashamed of these episodes? Q6 – Do you feel disgusted with yourself, depressed, or guilty afterward? **Conclusion:** The questionnaire assessment confirmed a high frequency of symptoms associated with binge eating behavior in medical students during the COVID-19 pandemic, compared to the post-pandemic period. Despite the risk of bias and inaccuracies in self-reported questionnaires, special attention is needed in this population regarding the screening of eating disorders.
Keywords: binge eating; bulimia; medical students.

OTHERS

405 (3247) THYROID SAFETY AND MEDULLARY THYROID CARCINOMA RISK WITH TIRZEPATIDE IN PATIENTS WITH TYPE 2 DIABETES AND OBESITY: A SYSTEMATIC REVIEW

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Introduction: Tirzepatide, a dual GIP and GLP-1 receptor agonist, carries a "black box warning" regarding the risk of medullary thyroid carcinoma (MTC) based on rodent studies. However, clinical evidence in humans remains limited, and the significance of treatment-induced calcitonin elevations is still being evaluated. **Objective:** To assess the incidence of MTC, C-cell hyperplasia, and changes in serum calcitonin levels in patients with type 2 diabetes (T2DM) or obesity treated with tirzepatide. **Methods:** Systematic review and meta-analysis of 13 randomized controlled trials (RCTs) (n = 13,761) and a retrospective real-world cohort of 283,026 patients. Data were also drawn from the FDA Adverse Event Reporting System (FAERS) post-marketing database. **Results:** Analysis of 13 RCTs (26–72 weeks) revealed zero cases of MTC or papillary thyroid carcinoma in both tirzepatide and control groups. Real-world evidence from a large-scale cohort study failed to prove an association with malignant thyroid disease; conversely, tirzepatide users demonstrated a significantly lower incidence of thyroid cancer compared to non-users (relative risk 0.348; 95% CI: 0.297–0.408; $p < 0.001$). Regarding biochemical markers, tirzepatide was associated with dose-dependent increases in serum calcitonin compared to placebo, specifically for the 10 mg (SMD 18.28%) and 15 mg (SMD 12.67%) doses. Pharmacovigilance data from FAERS identified only 3 reported cases of MTC, indicating a safety profile regarding thyroid malignancies that is comparable to established GLP-1 receptor agonists. No cases of C-cell hyperplasia were reported in humans within the analyzed RCT programs. While meta-analyses of the GLP-1RA class suggest a moderate increase in overall thyroid cancer risk (MH-OR 1.52), specific associations for MTC remain statistically non-significant (MH-OR 1.44; $p = 0.55$). **Conclusion:** Current evidence indicates that tirzepatide does not increase the risk of MTC in humans over short-to-medium-term follow-up. The observed increases in serum calcitonin are dose-dependent but have not translated into clinical malignancy. Tirzepatide's thyroid safety profile is similar to that of GLP-1RAs, though long-term surveillance remains essential due to the chronic nature of treatment.
Keywords: tirzepatide, medullary thyroid carcinoma; type 2 diabetes mellitus, GLP-1 receptor agonists; obesity, calcitonin.

THYROID

406 (3017) KINETIC PROGRESSION OF BIOCHEMICAL MARKERS IN THE ABSENCE OF STRUCTURAL DISEASE IN A PATIENT WITH RET-NEGATIVE MEDULLARY THYROID CARCINOMA ASSOCIATED WITH A LIKELY PATHOGENIC CHEK2 GENE VARIANT: A CASE REPORT

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Case report: A 65-year-old female patient presented thyroid nodule, calcitonin of 1,964 pg/mL and CEA of 563 ng/mL. She underwent total thyroidectomy, confirming medullary thyroid carcinoma (MTC) RET-negative germline mutation, leading diagnosis of sporadic MTC, intermediate risk. Progressed with persistent biochemical, without structural, disease, and remained stable for 5 years, when calcitonin doubling time 8.5 months and CEA doubling time 30.9 months were observed. Testing was negative for interferes (heterophil antibodies and PEG). Restaging identified adrenal adenoma, renal angiomyolipoma and hepatic metastasis from colorectal adenocarcinoma, second primary neoplasm, suggestive hereditary syndrome. Extended panel revealed likely pathogenic heterozygous variant in CHEK2, p.Arg117Gly. Screening for known familial mutation (SNV/INDEL) was positive in first-degree relative, with genetic counseling and recommendations for early screening for neoplasms. **Discussion:** CMT accounts 1%–2% of thyroid cancers; 75% sporadic driven by somatic mutations in RAS and NF1, and 25% hereditary associated to RET proto-oncogene. Additionally, doubling time < 24 months as unfavorable prognostic marker related to progression. Pathogenic variants in CHEK2, tumor suppressor involved in DNA repair, confer moderate risk for neoplasms such as breast, prostate, colorectal, and papillary thyroid cancer, however, CHEK2 gene p.Arg117Gly variant, with reduced kinase activity and defective repair, shows no evidence of association to CMT tumorigenesis, given distinct embryological origin of C cells. Plausible alternative pathways include: (i) partial impairment of checkpoint favoring genomic instability and somatic mutations in RET or RAS; (ii) haploinsufficiency modulating BRCA1 shift toward error-prone repair; (iii) modifier clonal evolution and accumulation of events favoring tumor progression; (iv) multi-organ risk with additive effect and genomic instability. Challenges include absence of RET- negative cohorts with germline sequencing, lack of studies in C cells for variants in FHA, lack of data on loss of heterozygosity in carriers and distinguishing between causality and coexistence. **Concluding remarks:** Presence of variants in CHEK2 in sporadic CMT explains finding associated to colorectal adenocarcinoma metastasis and underscores importance of comprehensive genetic testing panels for understanding tumorigenesis, evaluating potential alternative pathways and providing family counseling.
Keywords: kinetic progression of biochemical markers; medullary thyroid carcinoma RET-negative; likely pathogenic CHEK2 gene variant.

THYROID

407 (3086) ACTIVE SURVEILLANCE VS. IMMEDIATE THYROIDECTOMY IN PAPILLARY MICROCARCINOMAS: A SYSTEMATIC REVIEW

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Introduction: Active surveillance (AS) is the conservative management of low-risk papillary thyroid microcarcinoma through periodic ultrasound monitoring. As an alternative to immediate thyroidectomy (IT), it avoids overtreatment and surgical risks, reserving intervention only for cases of progression without compromising prognosis or survival. **Objective:** To evaluate the clinical outcomes of active surveillance compared to immediate thyroidectomy in patients with low-risk papillary thyroid microcarcinoma. **Methods:** A systematic review was conducted according to PRISMA guidelines and structured using the PICO strategy: patients with low-risk PTMC, generally tumors ≤ 1 cm, without lymph node metastasis or extrathyroidal extension; intervention with AS; comparison with total or partial IT; and outcomes including tumor progression, metastasis, survival, surgical complications, and quality of life. The search was performed in PubMed/MEDLINE and BVS databases using descriptors related to papillary thyroid microcarcinoma, active surveillance, and thyroidectomy. Studies published between 2021 and 2026, in English, Portuguese, or Spanish, that directly compared AS and IT were included. Duplicates, studies without direct comparison between AS and IT, and studies evaluating therapeutic strategies outside the proposed comparison were excluded. **Results:** A total of 39 studies were identified in the searched databases. After removing duplicates and applying eligibility criteria, 9 studies were included in the final analysis. The studies demonstrated that AS is a safe strategy, with estimated tumor progression rates between 14.5% and 15%. Regarding disease dissemination, the incidence of new lymph node metastases was low (0% to 2.9%), representing 13% of progression cases. Mortality was equivalent to IT, though AS was associated with lower overall mortality rates. The analysis of complications reinforces the benefit of conservative management in preventing morbidity; IT showed rates of 4.1% for vocal cord paralysis and 16.7% for temporary hypoparathyroidism, outcomes that AS avoided in the majority of patients. Furthermore, several studies highlighted better quality of life and higher cost-effectiveness among patients undergoing active surveillance. **Conclusion:** Active surveillance proved to be a safe and effective strategy for patients with low-risk papillary thyroid microcarcinoma, presenting oncological outcomes similar to those of immediate thyroidectomy.
Keywords: papillary thyroid carcinoma; active surveillance; thyroidectomy.

THYROID

408 (3450) AMIODARONE-INDUCED THYROTOXICOSIS (AIT): DIAGNOSTIC AND THERAPEUTIC

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Case presentation: A 66-year-old female patient with atrial fibrillation initiated amiodarone therapy (200 mg/day). Thyroid function tests prior to amiodarone exposure were within the reference range (RR): TSH 2.47 mIU/L (RR: 0.4–5.8) and free T4 (FT4) 1.03 ng/dL (RR: 0.93–1.7). Over a three-year period, she developed progressive asthenia and a 40 kg weight loss, with subsequent labs revealing overt thyrotoxicosis: TSH < 0.01 mIU/L and FT4 4.16 ng/dL. Consequently, amiodarone was discontinued and methimazole (MMI) initiated. Diagnostic work-up showed a borderline TRAb of 1.46 IU/L (cut-off: <1.22), negative anti-TPO, and ultrasonography demonstrated a slightly enlarged, heterogeneous gland without nodules. Two months after starting MMI, labs showed TSH 0.03 mIU/L and FT4 1.63 ng/dL. Five months into follow-up, she progressed to a hypothyroid phase: TSH 17.03 mIU/L, FT4 0.52 ng/dL and TRAb titers became negative. MMI was withdrawn and levothyroxine was not initiated. Within 30 days, the patient achieved spontaneous biochemical euthyroidism. **Discussion:** This case illustrates the clinical challenge of differentiating between amiodarone induced thyrotoxicosis (AIT) type 1 and type 2 due to overlapping features. In this patient, a mixed presentation of thyrotoxicosis was observed. Initially, the presence of a borderline positive TRAb and significant weight loss suggested an autoimmune component compatible with AIT type 1. However, the subsequent clinical evolution characterized by progression to a hypothyroid phase and the spontaneous recovery of the pituitary-thyroid axis following the withdrawal of antithyroid drugs is strongly indicative of the inflammatory and destructive process of AIT type 2. The transient elevation of TSH (17.03 mIUI/L) followed by spontaneous normalization supports the diagnosis of a destructive process (AIT type 2) rather than true hyperthyroidism (AIT type 1). Amiodarone causes thyroid dysfunction in up to 36% of patients, with AIT occurring in 2%–12%. While type 1 AIT involves increased hormone synthesis, type 2 is a destructive thyroiditis due to direct drug toxicity and the release of preformed hormone stores. **Conclusion:** Distinguishing between AIT type 1 and type 2 remains a significant clinical challenge. This case emphasizes the importance of a nuanced pathophysiological interpretation of the thyroid axis, highlighting the possibility of spontaneous recovery of thyroid function after the withdrawal of antithyroid drugs.

Keywords: amiodarone-induced thyrotoxicosis; destructive thyroiditis; transient hypothyroidism.

THYROID

409 (3557) ANAPLASTIC THYROID CARCINOMA PRESENTING AS ACUTE THYROIDITIS: A DIAGNOSTIC CHALLENGE

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Case presentation: Two cases of anaplastic thyroid carcinoma (ATC) initially presenting with features suggestive of acute thyroiditis are described. Case Report #1: A 56-year-old woman sought medical attention due to sudden cervical pain, low-grade fever, and enlargement of a long-standing goiter. Physical examination suggested a thyroid abscess, and she was treated empirically with cefuroxime axetil. She experienced transient improvement followed by clinical deterioration. Ultrasonography demonstrated a large, heterogeneous, predominantly solid nodule involving the right lobe, isthmus, and part of the left lobe. Computed tomography (CT) scan revealed a thyroid mass with signs of extrathyroidal extension, inseparable from the strap muscles, causing tracheal compression. The patient underwent surgical resection; however, residual tumor remained in the larynx. The histopathological report confirmed ATC. Case Report #2: An 87-year-old woman with a multinodular goiter, featuring a large right nodule and symptoms of dysphagia, underwent ethanol ablation therapy. She developed pain and enlargement of the right side after the third session. A CT scan demonstrated a necrotic mass with gas and tracheal deviation. Needle aspiration was performed, yielding purulent drainage, and Cutibacterium acnes was isolated. The patient was treated with amoxicillin/clavulanate and serial drainage, resulting in volume reduction but persistence of the indurated nodule. After 4 months, her dysphagia worsened, and she developed right vocal cord paralysis. A CT scan demonstrated a nodule in close contact with the common carotid artery, esophagus, and trachea. She was subsequently diagnosed with ATC. **Discussion:** ATC is a rare and highly aggressive malignancy characterized by rapid tumor growth, local invasion, and high metastatic potential. The most common symptoms include cervical pain, dyspnea, dysphagia, hoarseness, cough with or without hemoptysis, and stridor, alongside constitutional symptoms. However, in some cases, inflammatory signs may present, mimicking acute thyroiditis or cervical abscess, which challenges the diagnosis. In such scenarios, the persistence and progression of symptoms, associated with imaging findings and laboratory tests, are crucial to suspect malignancy. **Final comments:** These cases reinforce the importance of suspecting atypical cervical inflammatory presentations, particularly in patients with an unfavorable clinical course and a long-standing goiter.

Keywords: anaplastic thyroid carcinoma; acute thyroiditis; thyroid abscess.

THYROID

410 (3066) ASSOCIATION OF HEAVY METALS WITH TSH AND THYROID HORMONES IN MATERNAL AND CORD BLOOD: FINDINGS FROM A COHORT (PIPA STUDY)

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Introduction: Heavy metals, such as lead, mercury, cadmium and arsenic, are considered endocrine-disrupting chemicals of global concern due to their toxic effects on human health and the environment, including affecting the thyroid gland and potentially causing thyroid disorders. **Objective:** The aim of the study was to evaluate the relationship between in utero exposure to the heavy metals (lead, mercury, cadmium and arsenic) with thyrotropin (TSH) and thyroid hormones (TH) at birth. **Methods:** The study population consisted of 99 mother-infant pairs from the birth cohort of the Childhood and Environmental Pollutants Project (PIPA-UFRJ) from Rio de Janeiro, recruited between June 2021 and July 2022. Metals were analyzed using inductively coupled plasma mass spectrometry (ICP-MS) in umbilical cord blood. Hormones in maternal plasma and cord blood were measured using ELISA or Multiplex Assay. Spearman's correlation was used to evaluate the relationship between metal and hormone. Linear regression analysis was performed to assess the association between metal exposure, hormone levels, and weight gain (CEP: 5.415.164). **Results:** The detection rates of metals in umbilical cord blood samples were lead (100%), mercury (93.8%), arsenic (59.2%), and cadmium (13.3%). Arsenic concentration in umbilical cord blood showed a positive correlation with TSH levels ($r = 0.39$, $p = 0.001$). No significant correlations were observed between the other metals and hormone levels in the cord blood as well as in the maternal plasma. Lead concentration was associated with higher weight at 12 months ($r = 0.287$, $p = 0.007$) and greater weight gain from the 3rd to 12th month ($r = 0.305$, $p = 0.006$). Conversely, Arsenic showed a negative correlation with weight gain from the 6th to the 12th month ($r = -0.238$, $p = 0.042$). **Conclusion:** Prenatal arsenic exposure may be associated with alterations in TSH levels in umbilical cord blood. Furthermore, both lead and arsenic appear to induce changes in weight gain during the first year of life.
Keywords: heavy metals; thyroid; mother-infant pairs.

THYROID

411 (2882) BISPHENOL A DISRUPTS MITOCHONDRIAL COMPLEXES I, III, AND V DRIVING THYROID CELL METABOLIC REPROGRAMMING AND CARCINOGENESIS

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Introduction: Bisphenol A (BPA), a ubiquitous endocrine disruptor in plastics and food packaging, has been associated with thyroid carcinogenesis. A key driver of this process is mitochondrial dysfunction, which promotes metabolic reprogramming, oxidative stress, apoptosis resistance, and immune remodeling. Aggressive thyroid tumors exhibit impaired oxidative phosphorylation (OXPHOS), mtDNA mutations, and altered mitochondrial dynamics, which drive tumor progression and therapy resistance. This disruption of bioenergetics enables metabolic plasticity, allowing cancer cells to switch between glycolysis and alternative pathways to sustain proliferation. However, the direct impact of BPA on thyroid mitochondrial bioenergetics remains unclear. **Objective:** To investigate the effects of BPA exposure on mitochondrial bioenergetics and metabolism in normal and papillary-derived thyroid cancer cell lines. **Methods:** Nthy-ori 3-1, TPC-1, and BCPAP cell lines were used. Cells were exposed to BPA (0.8 and 1.0 µg/mL) for 24 and 48 hours. Total RNA was extracted and subjected to RNA-seq analysis to identify the differentially expressed genes. **Results:** BPA reduced the metabolic activity in a dose-dependent manner. RNA-seq analysis identified significant alterations in multiple genes of the mitochondrial respiratory chain. The MT-ATP8 gene (Complex V subunit) showed opposite regulation between normal ($+1.692$ $\log_2$FC) and tumor ($+1.276$ and -1.431 $\log_2$FC) cells, indicating selective impairment of ATP synthesis in neoplastic cells. Additionally, dysregulation of NDUFA12 (Complex I subunit) and UQCRHL (Complex III component), both essential for electron flow in oxidative phosphorylation, was identified. NDUFA12 dysregulation is particularly relevant, as its overexpression is associated with a worse prognosis in thyroid cancer. Dysregulation of other respiratory chain and Krebs cycle genes was also detected, along with modulation of cholesterol biosynthesis and overexpression of SLC7A5 ($+1.139$ $\log_2$FC). **Conclusion:** BPA induces genotype-dependent metabolic reprogramming and mitochondrial dysfunction, highlighted by the coordinated dysregulation of genes encoding subunits of respiratory chain Complexes I, III, and V (NDUFA12, UQCRHL, and MT-ATP8). This finding reveals a specific mechanism by which BPA compromises mitochondrial bioenergetics, differentially affecting normal and tumor thyroid cells and potentially contributing to tumor aggressiveness.
Keywords: bisphenol A (BPA); mitochondrial dysfunction; thyroid carcinogenesis.

THYROID

412 (2883) BISPHENOL A TRIGGERS A DUOX1/ROS/NFE2L2 REDOX CASCADE DRIVING THYROID CELL TRANSFORMATION AND CARCINOGENESIS

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Introduction: Bisphenol A (BPA) is an endocrine disruptor widely used in plastics and food packaging. It is associated with infertility, metabolic disorders, and cancer. In the thyroid, BPA can alter hormone signaling and induce oxidative stress, which is a central mechanism of cellular dysfunction and carcinogenesis. However, the molecular mechanisms by which BPA dysregulates redox homeostasis in the thyroid remain poorly understood. **Objective:** To investigate the effects of BPA exposure on the genetic regulation of redox homeostasis and the induction of oxidative stress in human thyroid cell lines using a transcriptomic approach. **Methods:** Nthy-ori 3-1 (normal thyroid), TPC-1, and BCPAP (papillary thyroid carcinoma) cell lines were used. Cells were exposed to BPA (0.8 and 1.0 µg/mL) for 24 and 48 h. Total RNA was extracted and subjected to RNA sequencing (RNA-seq) for transcriptome analysis. Differentially expressed genes were identified using bioinformatics, followed by pathway enrichment analysis (Gene Ontology and KEGG). **Results:** Transcriptomic analysis revealed that BPA induces a pro-oxidant environment in a cell-context-dependent manner. In TPC-1 cells, a glutathione synthesis precursor gene showed the highest expression variation (+5.543 $\log_2$FC). Notably, RNA-seq identified significant DUOX1 upregulation (+1.195 $\log_2$FC) in BCPAP cells. This gene encodes an NADPH oxidase specialized in H_2O_2 generation in the thyroid, which is the main source of ROS production. Mechanistically, BPA-induced DUOX1 upregulation was accompanied by KEAP1-NFE2L2 pathway enrichment and coordinated modulation of antioxidant defense genes, including TXNIP, PRDX5, and TXNRD2 expression. This suggests that excessive ROS production driven by DUOX1 activates the NFE2L2-mediated antioxidant response as a compensatory mechanism to counteract BPA-induced oxidative stress. Additionally, an increased expression of SLC7A11, which is crucial for redox homeostasis, was observed. **Conclusion:** BPA compromises thyroid redox homeostasis, promoting oxidative stress and DUOX1 overexpression. DUOX1 upregulation drives ROS production, activating KEAP1-NFE2L2 and modulating TXNIP, PRDX5, and TXNRD2. As DUOX1 dysregulation is associated with persistent DNA damage and thyroid cancer, its induction by BPA represents a critical mechanism of toxicity and carcinogenesis.
Keywords: DUOX1; RNA-seq; endocrine disruptor.

THYROID

413 (3068) BONE VULNERABILITY AND TSH SUPPRESSION EXPOSURE IN PATIENTS WITH DIFFERENTIATED THYROID CARCINOMA

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Introduction: TSH suppression may be indicated during follow-up of differentiated thyroid carcinoma (DTC), but its intensity and duration should be individualized according to oncologic risk and risk of adverse events. Current recommendations reinforce periodic reassessment of TSH targets, avoiding potentially unnecessary chronic suppression in patients without clear oncologic indication. **Objective:** To describe the prevalence and most affected densitometric sites of bone abnormalities in patients with DTC and to identify the proportion with bone vulnerability associated with suppressed TSH, including a secondary analysis of TSH at one year after surgery. **Methods:** This was a retrospective observational study of adults with DTC and available bone mineral density assessment. Bone vulnerability was defined as osteoporosis, use of antiresorptive therapy, or osteopenia in patients aged 65 years or older. The therapeutic attention zone was defined as bone vulnerability associated with current TSH < 0.5 mIU/L. As a secondary analysis, TSH at one year after surgery was used as a marker of early suppressive exposure. **Results:** Sixty-eight patients were included. Mean age was 66.0 years. Osteopenia occurred in 30/68 (44.1%), osteoporosis in 18/68 (26.5%), and any bone abnormality in 48/68 (70.6%). The lowest mean T-score was observed at the lumbar spine (–1.59), followed by the femoral neck (–1.08) and total hip (–0.70). Bone vulnerability was present in 41/68 patients (60.3%). Among 58 patients with available current TSH, 20/58 (34.5%) had TSH < 0.5 mIU/L. The coexistence of bone vulnerability and current TSH < 0.5 occurred in 11/58 (19.0%). TSH at one year after surgery was available in 35 patients; 26/35 (74.3%) had TSH < 0.5 and 21/35 (60.0%) had TSH < 0.1. Among evaluable patients, 15/35 (42.9%) combined current bone vulnerability and one-year TSH < 0.5. **Conclusion:** Patients with DTC who underwent bone mineral density assessment showed a high prevalence of bone abnormalities and frequent early exposure to suppressive TSH levels. The coexistence of bone vulnerability with current or early suppressed TSH identifies a subgroup in whom TSH targets should be individually reassessed to reduce potentially unnecessary suppression.
Keywords: differentiated thyroid carcinoma; TSH suppression; bone mineral density.

THYROID

414 (3282) BRAIN METASTASIS IN PAPILLARY THYROID CARCINOMA: A SERIES OF THREE CASES WITH AGGRESSIVE HISTOLOGY AND UNFAVORABLE OUTCOMES

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Case presentation: Three cases of locally advanced papillary thyroid carcinoma treated with radioiodine therapy are reported, in which brain metastasis was associated with aggressive biological behavior and poor prognosis. ABM, 69 years old, was referred for an infiltrative goiter adherent to deep planes and underwent thyroidectomy with partial tracheal resection. Histopathology revealed tall-cell variant papillary thyroid carcinoma with extrathyroidal extension and lymph node involvement. Two years later, neurological symptoms developed and brain metastasis was identified, with death occurring two years afterward. RI, 58 years old, presented with non-toxic multinodular goiter with mediastinal extension and tracheal involvement. He underwent thoracotomy with partial tumor resection, and papillary thyroid carcinoma was diagnosed. Seven years after diagnosis, he developed hemiparesis and aphasia secondary to brain metastasis, dying nine months after diagnosis of the lesion. SMO, 57 years old, sought medical attention for painful lymphadenopathy, hoarseness, and a thyroid nodule. Histopathology revealed columnar-cell variant papillary thyroid carcinoma. Early progression occurred with hemorrhagic intraparenchymal lesions compatible with brain metastases, leading to death from intracranial hemorrhage after 13 days. **Discussion:** Brain dissemination in papillary thyroid carcinoma is rare and usually aggressive. All three cases shared T4 disease at diagnosis, extrathyroidal invasion, and high lymph node burden. Toraih *et al.* demonstrated that brain metastases are associated with the lowest survival rates among metastatic sites of papillary thyroid carcinoma, especially with multiorgan involvement and high tumor burden. A recent series showed that most brain metastasis cases arise from aggressive histological subtypes, frequently associated with alterations related to worse prognosis, including BRAFV600E and TERT promoter mutations. Recent American Thyroid Association guidelines recommend multidisciplinary management, prioritizing surgical resection and stereotactic radiotherapy whenever feasible, whereas radioiodine therapy has limited benefit in aggressive disease. **Final comments:** Brain metastases in papillary thyroid carcinoma are associated with aggressive histological subtypes, extensive locoregional disease, and poor prognosis. Focal neurological symptoms in patients with advanced disease should prompt immediate investigation of the central nervous system.

Keywords: papillary thyroid carcinoma; brain metastasis; metastatic disease.

THYROID

415 (3270) CALORIMETRY AND QUALITY OF LIFE IN PATIENTS WITH GRAVES' DISEASE

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Introduction: Graves' disease increases basal metabolic rate and energy expenditure, in addition to compromising quality of life. **Methods:** Cross-sectional observational study, approved by a Research Ethics Committee. Women over 18 years of age with hyperthyroidism due to Graves' disease, confirmed by positive autoantibodies or scintigraphy, and under follow-up at a specialized service were included. Exclusion criteria were men, postmenopausal women, and use of medications that interfere with body composition and basal metabolic rate (BMR). Patients completed the Thyroid-Related Patient-Reported Outcome (ThyPRO) quality of life questionnaire and underwent muscle strength assessment (handgrip strength with a dynamometer and the 5-times sit-to-stand test), anthropometric analysis, and indirect calorimetry for BMR estimation. The SPSS software was used for statistical analysis. P-values < 0.05 were considered significant. **Results:** Of 228 women under follow-up for Graves' Disease, 42 were evaluated over a 7-month period, distributed into the compensated hyperthyroidism (CHT) group (n = 15), subclinical hyperthyroidism (SCHT) group (n = 5), and active hyperthyroidism (AHT) group (n = 22). There were no significant differences between groups regarding age, weight, body mass index, or handgrip strength. BMR both in kcal/day and as % of predicted differed among the three groups (p = 0.010 and p = 0.005, respectively), being highest in the AHT group. However, the AHT group had a longer time (10.8 ± 2.2 s) in the sit-to-stand test compared to the CHT group (9.2 ± 1.6 s), p = 0.032. In Tukey's post-hoc analysis, the AHT group showed elevated BMR compared to the SCHT group (p = 0.031) and the CHT group (p = 0.042). The AHT group showed higher oxygen consumption (VO in kcal/day and as % of predicted vs. the CHT group (360 ± 44 and 152 ± 34% vs. 307 ± 62 and 123 ± 26%, p = 0.007 and 0.034, respectively), whereas carbon dioxide production (VCO mL/min) showed a tendency to be higher in this group (p = 0.05). The ThyPRO questionnaire differed between groups in overall score and in the goiter, symptoms, and fatigue domains, with worse scores for the AHT group (p < 0.05 for all). **Conclusion:** Active hyperthyroidism due to Graves' disease promotes significant negative impacts, manifested by persistent hypermetabolism, low strength and impaired quality of life. Rigorous hormonal control proved essential, as the compensated group presented better results across all evaluated outcomes.

Keywords: Graves' disease; basal metabolic rate; indirect calorimetry.

THYROID

416 (2964) CASE REPORT: HASHIMOTO'S ENCEPHALOPATHY IN ADOLESCENT WITH SEVERE HYPOTHYROIDISM

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Case presentation: A previously healthy 16-year-old male was referred due to syncope, bradycardia and hypoglycemia. On admission, he reported malaise, upper limb paresthesias, asthenia and postprandial nausea for approximately 3 days. Electrocardiogram showed sinus bradycardia and laboratory tests demonstrated severe hypothyroidism (TSH > 500 mIU/mL [reference value: 0.35–4.94], free T4 0.4 ng/dL [reference value: 0.7–1.48]). Levothyroxine was initiated at a dose of 2 mcg/kg/day and etiological investigation was pursued, revealing elevated antithyroid antibody titers (anti-thyroid peroxidase > 1,000 IU/mL [reference value: <5.6]; anti-thyroglobulin 25.06 IU/mL [reference value: <4.11]), confirming the diagnosis of Hashimoto's thyroiditis. During hospitalization, he presented a first-time generalized tonic-clonic seizure. Infectious investigation, including cerebrospinal fluid analysis, was negative. Brain magnetic resonance imaging showed nonspecific edema in the left hippocampus and adenohypophysis hyperplasia. Electroencephalogram revealed left parieto-temporal slowing. Given the clinical and complementary tests, and exclusion of other causes by the Neurology team, Hashimoto's encephalopathy was hypothesized. Antiepileptic medication (levetiracetam) and methylprednisolone 1 g/day for 5 days were initiated, followed by prednisone 40 mg/day with gradual tapering. The patient experienced seizure recurrence, but neurological stability was observed after optimization of the antiepileptic dose. **Discussion:** Hashimoto's encephalopathy is a rare and controversial condition, described in all age groups including children. Also called steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT), it is characterized by the presence of antithyroid antibodies, but without association between antibody levels and disease severity. It is a diagnosis of exclusion, with heterogeneous clinical presentation involving varied neurological and psychiatric manifestations. Epileptic seizures may affect two-thirds of patients, more frequently in children. There are no treatment guidelines, but corticosteroid therapy with methylprednisolone is the treatment of choice. **Final comments:** Hashimoto's encephalopathy is a difficult-to-diagnose pathology due to limited understanding of its pathogenesis and absence of specific biomarkers for the disease. The importance of early recognition is emphasized, given its potentially reversible nature with appropriate treatment.

Keywords: Hashimoto's encephalopathy; Hashimoto's thyroiditis; autoimmune encephalitis.

THYROID

417 (2982) CHRONIC LYMPHOCYTIC LEUKEMIA PRESENTING AS PROGRESSIVE THYROID NODULES: A DIAGNOSTIC CHALLENGE CONFIRMED AFTER THYROIDECTOMY

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Case presentation: A 65-year-old woman with hypertension and chronic lymphocytic leukemia (CLL) diagnosed in 2020, who was under regular follow-up, presented with anterior cervical enlargement. Thyroid ultrasound performed in 2021 demonstrated multinodular goiter with bilateral nodules, the largest located in the right lobe (4.1 × 2.3 × 3.1 cm), classified as TI-RADS 3. Cervical computed tomography (CT) revealed heterogeneous thyroid enlargement, predominantly affecting the right lobe, with slight extension into the thoracic inlet. In 2022, repeat ultrasound showed progression of the dominant right lobe nodule, now presenting calcifications and measuring 3.9 × 3.1 × 2.5 cm, leading to reclassification as TI-RADS 4. Fine-needle aspiration (FNA), performed in 2023, demonstrated small lymphocytic infiltrate suggestive of a lymphoproliferative disorder; however, immunohistochemistry remained inconclusive, even after repeat FNA. Due to persistent diagnostic uncertainty, thyroidectomy was performed in 2024. Histopathological examination revealed lymphoid infiltration, and immunohistochemistry confirmed focal involvement by small lymphocytic lymphoma/chronic lymphocytic leukemia (SLL/CLL). **Discussion:** Thyroid metastases account for approximately 1%–3% of thyroid neoplasms in clinical series. In CLL, extranodal involvement occurs in 5%–10% of cases, with thyroid infiltration representing an uncommon manifestation. Diagnosis is often challenging because of the limited sensitivity of FNA, frequently requiring histopathological confirmation. **Final comments:** Metastatic involvement associated with CLL infiltration should be considered in the differential diagnosis of thyroid nodules in affected patients, especially in cases of inconclusive cytology.

Keywords: chronic lymphocytic leukemia; thyroid nodule; metastasis.

THYROID

418 (3102) CHYLOTHORAX ASSOCIATED WITH THE USE OF SELECTIVE RET INHIBITORS: A DIAGNOSTIC CHALLENGE IN THE TREATMENT OF METASTATIC MEDULLARY THYROID CARCINOMA

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Case presentation: A 72-year-old male patient, diagnosed with metastatic medullary thyroid carcinoma (MTC) in systemic progression, began treatment with selpercatinib in April 2023. He achieved a partial radiological response, with complete remission of liver metastases and a partial reduction in lung lesions and cervical lymph node metastases, alongside a progressive reduction of a pre-existing pleural effusion. In late 2025, during hospitalization for an infected tracheocutaneous fistula and sternoclavicular mediastinitis — associated with deep vein thrombosis (DVT) of the right upper limb — he presented a significant increase in pleural effusion. A diagnostic thoracentesis was performed. Biochemical analysis of the milky pleural fluid revealed hypertriglyceridemia (472.8 mg/dL), confirming the diagnosis of chylothorax. Cytology was negative for malignancy. Selpercatinib was suspended due to the patient's overall clinical condition, although the pleural effusion persisted. The event was interpreted as late lymphatic toxicity to selpercatinib, potentially exacerbated by central venous hypertension from the DVT and lymphatic compression caused by regional inflammatory processes. **Discussion:** Selpercatinib is a selective RET inhibitor used in target-specific therapy for metastatic or locally advanced MTC with disease progression. Although the drug's antitumor efficacy is exceptional, the development of chylous effusions poses significant challenges to long-term clinical management. Chylous effusions are rare adverse events, reported in approximately 7% of patients treated with selpercatinib; these tend to be late-onset, often occurring during periods of excellent radiological tumor response. The proposed mechanism involves the microscopic disruption of lymphatic channels or changes in lymphatic endothelial permeability due to RET inhibition. **Final comments:** This case illustrates a toxicity of selective RET inhibitors, specifically selpercatinib, which, despite being rare, presents diagnostic challenges and necessitates crucial decisions in long-term clinical management. Analysis of pleural fluid (triglyceride levels and cytology) is mandatory to confirm the chylous nature and rule out therapeutic failure, thereby avoiding the undue discontinuation of a highly effective drug for metastatic disease control.
Keywords: chylothorax; medullary thyroid carcinoma; selpercatinib.

THYROID

419 (3077) COMBINED TI-RADS AND BETHESDA ANALYSIS IN THE EVALUATION OF THYROID NODULES: A 5-YEAR EXPERIENCE AT A TERTIARY CARE CENTER

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Introduction: Thyroid nodules are common, and their management aims to identify the risk of malignancy. Key diagnostic tools include ultrasonographic evaluation using the Thyroid Imaging Reporting and Data System (TI-RADS) and cytological assessment using the Bethesda System. Nodules classified as Bethesda I, III, IV, and V are of particular interest, as different management strategies may be considered for these categories. **Objective:** To evaluate the management of thyroid nodules at a tertiary care center, with emphasis on fine-needle aspiration biopsy and ultrasonographic findings, as well as comparison with clinical outcomes (ultrasound follow-up or post-surgical histopathological reports). **Methods:** This retrospective study analyzed data from medical records, imaging exams, and pathological reports. Fine-needle aspiration results from nodules of patients evaluated between 2010 and 2025 were included (General Group). For patients evaluated between 2021 and 2025 with Bethesda I, III, IV, and V nodules, ultrasonographic data were also collected, including either nodule progression in patients managed with clinical follow-up (Follow-up Group) or comparison with histopathological findings in patients who underwent thyroidectomy (Surgery Group). Analyses included descriptive statistics and statistical tests according to data distribution. **Results:** In the General Group, 1,218 nodules were evaluated: 179 Bethesda I (14.7%); 686 Bethesda II (56.3%); 182 Bethesda III (14.9%); 79 Bethesda IV (6.5%); 69 Bethesda V (5.7%); and 23 Bethesda VI (1.9%). In the Surgery Group, 124 nodules from 116 patients were evaluated, 34 of which were malignant. The highest risk of malignancy was observed in Bethesda V and TI-RADS 5 nodules (88.9%). Grouped TI-RADS 4 and 5 categories were significant predictors of malignancy ($p < 0.05$). In the Follow-up Group, 51 nodules from 47 patients were evaluated, most of which remained stable without significant changes in TI-RADS classification or size. **Conclusion:** This study confirms the importance of using the TI-RADS and Bethesda classifications together in the management of thyroid nodules.
Keywords: thyroid nodule; Thyroid Imaging Reporting and Data System; Bethesda System.

THYROID

420 (3429) DIAGNOSTIC CHALLENGES OF UNDETECTABLE SERUM THYROGLOBULIN IN A PATIENT WITH NON-SECRETORY FOLLICULAR THYROID CARCINOMA AND BONE METASTASES – CASE REPORT

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Case report: A 58-year-old female patient underwent total thyroidectomy. Histopathology revealed a well-differentiated, minimally invasive follicular thyroid carcinoma with an oncocytic cell component in the right thyroid lobe (T2N0Mx). Postoperatively, she was treated with radioiodine therapy, TSH 54.7 mUI/L; Tg 0.9 ng/mL; negative TgAb. A post-dose whole-body scan showed uptake in the thyroid bed and right scapula. Due to the low Tg levels/scan discrepancy, Tg was re-tested in different assays, neat and after dilution, confirming the low values. Scapular biopsy confirmed follicular carcinoma metastasis with positive immunohistochemistry (IHC) for Tg. Following surgical resection, the patient remains in a 22-year follow-up with stable incomplete structural disease. **Discussion:** The discrepancy between undetectable Tg and metastatic structural disease represents a significant diagnostic challenge in differentiated thyroid carcinoma (DTC). While Tg is a highly sensitive post-surgical marker, false-negative levels have been reported in 4% to 35% of DTC patients with evidence of local or metastatic disease. Although undetectable stimulated Tg often correlates with complete surgical resection, up to 14% of these patients may still harbor persistent or recurrent diseases, including distant metastases. In this specific case, the absence of anti-thyroglobulin antibodies (TgAb) and the confirmation of low Tg levels measured by multiple distinct assays even after dilution, solidly rule out analytical interference or hook effect. This configuration suggests a biologically "non-secretory" tumor, characterized by an intrinsic defect in the tumor's ability to secrete Tg into the systemic circulation or the production of non-immunoreactive Tg. Positive IHC for Tg in the metastatic tissue further confirms that the tumor produces the protein but fails to release it in measurable quantities, a hallmark of dedifferentiated or poorly differentiated subtypes. While this patient showed RAI uptake, the low Tg levels align with the concept that dedifferentiated cells may lose their secretory capacity even before losing iodine avidity. **Conclusion:** Serum Tg should not be used in isolation to exclude metastatic disease in high-risk patients. Once analytical interference and TgAb are excluded, undetectable Tg strongly indicates a non-secretory phenotype. Positive IHC remains essential in "Tg-negative/Structural-positive" cases, warranting a multi-modal imaging approach.
Keywords: thyroid; follicular carcinoma; thyroglobulin.

THYROID

421 (2938) DO MICRO AND NANOPLASTICS DISRUPT THE THYROID AXIS? EVIDENCE, EXPOSURE GAPS AND THE CHALLENGE OF MEASURING HUMAN EXPOSURE

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The widespread presence of micro and nanoplastics (MNPs) has raised a concern regarding their potential role as endocrine disrupting contaminants. Experimental studies suggest possible effects on the hypothalamic-pituitary-thyroid axis, although current evidence remains heterogeneous and limited by important methodological challenges. This study aims to synthesize evidence on the effects of MNPs exposure on thyroid axis function and evaluate limitations in exposure assessment. A systematic review with a structured search strategy was conducted using a PICO framework: population (experimental animal models, cell cultures, and humans), exposure (MNPs), comparator (non-exposed), and outcomes (thyroid-related hormonal and cellular alterations). Searches were performed in PubMed, Web of Science, and Scopus for studies published between 2021 and February 2026, using the terms "microplastic*" OR "nanoplastic*" AND "thyroid" OR "hypothalamic-pituitary-thyroid axis". Original studies in English were included, while editorials and conference abstracts were excluded. Following screening and duplicate removal, 34 studies were included in the final analysis. In vitro studies show that MNPs exposure could increase oxidative stress and impair hormone-related pathways in thyroid cells. In animal models, particularly rodents exposed to polystyrene microplastics, reduced circulating thyroid hormone levels were observed. Zebrafish studies reported endocrine disruption associated with developmental and metabolic alterations. In humans, available studies were limited to detecting MNPs in biological samples, without consistent associations with clinical outcomes. Most experimental studies were performed using spherical polystyrene particles, which may not represent the diversity and complexity of real-world exposure. Additionally, limitations in current analytical methods could overestimate MNPs levels, compromising exposure assessment and limiting the understanding of exposure magnitude and particle interaction with human tissues. Experimental evidence supports the potential of MNPs to interfere with thyroid axis function, however human data remain limited and inconclusive. Limitations in exposure assessment, restricted polymers investigated, and current analytical limitations compromise the reliability of risk characterization. Standardized and biologically relevant approaches are essential to determine the extent and the mechanisms by which MNPs could impact human health.
Keywords: microplastic; thyroid axis; endocrine disruption.

THYROID

422 (3570) EFFICACY AND SAFETY OF LEVOTHYROXINE THERAPY IN PREGNANT WOMEN WITH SUBCLINICAL HYPOTHYROIDISM: A SYSTEMATIC REVIEW

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Introduction: Subclinical hypothyroidism (SCH) is a prevalent endocrine disorder in pregnancy, associated with miscarriage, preterm birth, preeclampsia, and neurodevelopmental impairments. Despite its clinical relevance, the efficacy and safety of levothyroxine (LT4) in preventing these complications remain debated. This systematic review aims to synthesize current evidence on the impact of LT4 therapy on obstetric and neonatal outcomes. **Objective:** To evaluate the efficacy and safety of levothyroxine therapy in pregnant women with subclinical hypothyroidism. **Methods:** A systematic review was conducted in PubMed and LILACS databases (2015–2025), using the terms "Pregnancy," "Subclinical Hypothyroidism," "Levothyroxine," and "Pregnancy Outcome" combined with AND. Randomized clinical trials, cohort, and case-control studies including pregnant women with SCH using LT4 were eligible. Outcomes assessed included preterm birth, miscarriage, low birth weight, and adverse neonatal outcomes. Study selection followed PRISMA guidelines, with title, abstract, and full-text screening, plus manual reference search. Risk of bias was assessed using JBI criteria. **Results:** Of 121 articles identified, 8 met eligibility criteria. Most studies had low risk of bias; two showed moderate risk due to inadequate confounding control. LT4 use was associated with reduced adverse neonatal outcomes — including low birth weight, Apgar < 7, and preterm birth — and possible reduction in pregnancy loss. Benefits were more pronounced in women with TSH ≥ 4.0 mIU/L; lower TSH thresholds yielded less consistent results. Adequate TSH control was associated with fewer maternal and neonatal complications. Mild SCH showed limited benefit in some perinatal outcomes. Effects on infant neurodevelopment remain inconclusive. **Conclusion:** LT4 therapy may reduce adverse obstetric and neonatal outcomes in pregnant women with SCH, particularly those with TSH ≥ 4.0 mIU/L under adequate hormonal control. Evidence is heterogeneous in mild cases, and neurodevelopmental effects remain unclear, underscoring the need for well-designed randomized controlled trials.
Keywords: subclinical hypothyroidism; pregnancy; levothyroxine.

THYROID

423 (3259) EXTRAHEPATIC CHOLESTASIS DUE TO GIANT UTERINE FIBROID: A RARE MIMIC OF THIONAMIDE HEPATOTOXICITY IN A PATIENT WITH HYPERTHYROIDISM

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Case presentation: A 43-year-old female patient with type 2 diabetes mellitus, systemic arterial hypertension, and uterine leiomyoma was diagnosed with hyperthyroidism in October 2025 (TSH < 0.01 mIU/mL; free T4 2.07 ng/dL). Methimazole 10 mg/day was initiated, progressing to 15 mg/day after eight weeks. During this period, she developed jaundice, intense pruritus, and asthenia. Laboratory tests revealed altered transaminases (AST 409 U/L; ALT 173 U/L). Physical examination revealed an abdominal mass extending from the pelvis to the xiphoid process, leading to hospital admission and discontinuation of the medication due to suspected drug-induced hepatitis. During hospitalization, a significant cholestatic pattern was observed, with elevated canalicular enzymes (ALP 2521 U/L; GGT 1227 U/L), direct hyperbilirubinemia, and worsening transaminases. Cholangioresonance imaging demonstrated a heterogeneous expansive formation measuring 22.7 x 17.0 x 13.0 cm, consistent with a giant uterine leiomyoma, causing extrinsic compression of the distal common bile duct and dilation of the bile ducts. The patient underwent a total abdominal hysterectomy without complications. Postoperatively, the liver function tests normalized, confirming the mechanical obstructive etiology. Subsequently, methimazole was reintroduced at a low dose, without recurrence of hepatic abnormalities. **Discussion:** Thionamide-induced hepatotoxicity (HIT) represents a potential risk in the treatment of hyperthyroidism. However, documented liver changes during the use of these medications do not necessarily imply a cause-and-effect relationship, and other possible etiologies, such as extrinsic obstructive lesions of the biliary tract, should be investigated. The temporal association between methimazole use and the onset of jaundice initially favored the diagnosis of HIT in this report. However, imaging investigation revealed extrahepatic cholestasis secondary to biliary compression by a giant uterine fibroid. This case reinforces the importance of differential diagnoses in patients using thionamides, avoiding the automatic attribution of hepatic changes to methimazole. **Final considerations:** Biliary compression by giant uterine fibroids is a rare cause of cholestasis and can mimic HIT. This case highlights the need for attention to differential diagnoses in patients using thionamides, making it essential to avoid attributing liver abnormalities exclusively to adverse effects related to these drugs.
Keywords: methimazole; obstructive cholestasis; uterine fibroids.

THYROID

424 (2926) FOCAL NODULAR THYROIDITIS WITH HYPERVASCULARIZATION: UNCOMMON PRESENTATION REGARDLESS OF CLINICAL PRESENTATION OR GLANDULAR PARENCHYMAL ALTERATIONS

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Introduction: Chronic lymphocytic thyroiditis (Hashimoto thyroiditis) is the most common autoimmune thyroid disorder and typically presents with diffuse hypoechogenicity and heterogeneous echotexture on ultrasound. While diffuse hypoechogenicity and small hypoechoic nodules corresponding to lymphocytic proliferation are more commonly observed, the presence of hypervascularized nodules with mild hypoechogenicity may be indicative of specific forms or variants of Chronic thyroiditis. These case series highlights the expansion of the inicial 3 cases presented previously to 12 unusual presentations of focal thyroiditis with hypervascularized nodules of focal hypervascular nodules corresponding to cytological thyroiditis. **Materials and methods:** This retrospective observational study included 12 patients evaluated at a single private diagnostic center by an experienced radiologist submitted to FNA by experienced cytopathologist (according to the 2023 Bethesda System for Reporting Thyroid Cytopathology). Inclusion criteria: Presence of a focal thyroid nodule submitted to FNA and cytological diagnosis consistent with benign lymphocytic thyroiditis (Bethesda II). **Results:** Eleven nodules were classified as ACR TI-RADS 4, and one as TI-RADS 3. Most nodules were solid and hypoechoic. Central hypervascularization was the predominant Doppler finding. The nodules were presented into heterogeneously as well homogeneous parenchymal echotexture, indicating that the focal inflammatory pattern was not restricted to glands with classic diffuse features of autoimmune thyroiditis. All cytological examinations were classified as Bethesda II (benign). No cases demonstrated cytological atypia or features suspicious for malignancy. **Conclusion:** Focal thyroiditis with hypervascularized nodules can mimic malignancy on ultrasonography, but cytological evaluation and careful assessment of the surrounding thyroid parenchyma can help avoid unnecessary procedures. This case series underscores the importance of considering thyroiditis in the differential diagnosis of thyroid nodules, especially those with atypical ultrasonographic features, and highlights the need for individualized management based on accurate diagnosis.

Keywords: thyroiditis; thyroid nodule; focal thyroiditis.

THYROID

425 (3421) FUNCTIONING BONE METASTASIS AS THE FIRST MANIFESTATION OF DIFFERENTIATED THYROID CANCER: A CASE REPORT

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Case presentation: A 43-year-old previously healthy female was referred for evaluation of hip pain, which revealed a tumor in the right acetabulum. Bone biopsy showed carcinoma compatible with thyroid origin, confirmed by positive immunohistochemistry for TTF-1 and thyroglobulin (Tg). Investigation of the primary site revealed a thyroid nodule (Bethesda I) and atypical cervical lymph nodes. Total thyroidectomy with cervical lymph node dissection was performed, and histopathological examination demonstrated the coexistence of papillary and follicular carcinoma. Radioactive iodine (RAI) therapy with 200 mCi of I-131 was indicated. During preparation with levothyroxine withdrawal, an absence of TSH elevation accompanied by an increase in free T4 (FT4) levels was observed, suggesting a functioning metastasis. Post-therapeutic scintigraphy showed uptake in the thyroid bed and intense uptake in the acetabulum. Following RAI therapy, TSH levels increased, indicating a reduction in the hormonal activity of the metastasis. This required a progressive increase in the levothyroxine dose, demonstrating gradual control of the functioning metastasis. **Discussion:** Differentiated thyroid cancer (DTC), primarily comprising papillary and follicular subtypes, generally carries a good prognosis. Distant metastases occur in approximately 10% of patients with DTC, with bone being the second most common site after the lungs. Functioning metastases with active thyroid hormone production are rare, described in few cases. This functional activity is attributed to the maintained expression of genes related to iodine uptake and hormone synthesis, such as NIS, TPO, and Tg, allowing autonomous thyroid hormone production by metastatic cells. The coexistence of papillary and follicular carcinoma is uncommon and may reflect a common clonal origin with divergent morphological differentiation, associated with a more heterogeneous biological behavior. **Final comments:** The initial presentation with an isolated bone metastasis, signs of hormonal functionality, and the coexistence of distinct histological subtypes configure an uncommon presentation of DTC. This scenario is potentially associated with a higher risk of disease progression, justifying a more aggressive therapeutic approach and rigorous monitoring.

Keywords: thyroid neoplasms; neoplasm metastasis; hyperthyroidism.

THYROID

426 (3472) GIANT MULTINODULAR GOITER COMPLICATED BY CUTANEOUS INFECTION IN A NONAGENARIAN PATIENT: A CASE REPORT

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Case presentation: A 94-year-old woman had been followed for a large goiter for 50 years, with persistent refusal to undergo thyroidectomy despite progressive enlargement. She remained euthyroid and had low body weight (BMI 14), heart failure, and ischemic heart disease. She was admitted due to skin infection overlying the cervical mass, associated with ulceration, spontaneous purulent drainage, and fever. Cervical computed tomography (CT) demonstrated a heterogeneous exophytic lesion arising from the anterior infrahyoid cervical region/cervicothoracic junction, with peripheral calcifications and hypodense fluid areas, measuring 30 × 23.7 × 10.6 cm (estimated volume: 3,941 cm^3). The lesion caused compression mainly of the larynx and trachea, with mild luminal narrowing but without significant compressive symptoms. Ultrasonography revealed a large multinodular goiter containing isoechoic and mildly hypoechoic nodules with peripheral calcifications. Fine-needle aspiration biopsy (FNAB) was inconclusive (Bethesda I). During hospitalization, the patient received intravenous antibiotics, with marked drainage of purulent material and visible reduction in lesion size. Considering the indolent course, low suspicion for malignancy, patient preference, cardiovascular risk, and technical challenges related to anatomical distortion, conservative management was chosen. **Discussion:** Multinodular goiters are common in the general population; however, progression to extreme dimensions is uncommon because of their slow growth and the availability of treatment when indicated. Giant goiter is defined as a thyroid mass larger than 8 cm or weighing more than 500 g and may cause compression of cervical structures and impaired quality of life. In this case, the main complication was infection, an unusual event since the thyroid gland is relatively resistant to infection due to its high iodine content, rich vascular supply, and extensive lymphatic drainage. **Final comments:** This case illustrates an unusual evolution of giant multinodular goiter, characterized by the absence of major compressive symptoms despite massive dimensions and by the development of secondary skin infection. It also highlights the potential for progressive enlargement over decades when left untreated.

Keywords: goiter; thyroid gland; frailty.

THYROID

427 (3243) GRAVES' ORBITOPATHY WITH COMPRESSIVE OPTIC NEUROPATHY IN A PATIENT WITHOUT PREVIOUS HYPERTHYROIDISM: LONG-TERM FOLLOW-UP CASE REPORT

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Case presentation: A 51-year-old female former smoker was admitted in November/2021 due to Graves' orbitopathy with right compressive optic neuropathy. She had primary hypothyroidism secondary to Hashimoto's thyroiditis since 2011 and was taking levothyroxine (LT4) 100 mcg/day. Admission tests showed: TSH 0.058 mIU/L (0.35–4.9), FT4 1.04 ng/dL (0.70–1.80), TRAb 13.10 IU/L (<0.55), and anti-TPO >1,000 Ul/mL (<5.61). LT4 was discontinued, and thyroid function remained normal. She received IV methylprednisolone pulse therapy (500 mg/day for 4 days), followed by high-dose oral prednisone with gradual tapering and initial clinical improvement. In February/2022, ophthalmologic symptoms recurred, requiring a second IV pulse therapy course. At that time, she remained euthyroid without LT4. Multiple corticosteroid tapering attempts resulted in recurrent ocular symptoms, indicating prolonged glucocorticoid dependence. Corticosteroids were discontinued only in December/2024, although recent orbital MRI continued to show inflammatory activity. In April/2025, hypothyroidism recurred and LT4 was restarted. Tests showed: TSH 13.60 mIU/L (0.27–4.20), FT4 0.75 ng/dL (0.92–1.68), and TRAb 10.60 IU/L (<0.55). She is currently euthyroid on LT4 88 mcg/day. **Discussion:** Graves' orbitopathy is usually associated with hyperthyroidism; however, 5%-10% of cases occur in eu-or hypothyroid patients. The positive TRAb supports the role of humoral immunity in orbital fibroblast activation and glycosaminoglycan synthesis, promoting inflammation and tissue expansion independently of thyroid functional status. Compressive optic neuropathy is the most severe complication, with risk of permanent visual loss. Management of refractory glucocorticoid-dependent disease is challenging. International guidelines recommend early consideration of second-line therapies, such as cyclosporine or monoclonal antibodies, in patients with unsustained response to IV corticosteroids. Due to limited access to these therapies, our patient required recurrent glucocorticoid courses as needed. **Final comments:** Graves' orbitopathy management should be individualized and multidisciplinary. In severe refractory disease, the limitations of corticosteroid monotherapy must be recognized early, and alternative therapies should be considered to reduce morbidity and preserve visual function.

Keywords: Graves' orbitopathy; compressive optic neuropathy; hypothyroidism.

THYROID

428 (3494) HIGH-GRADE SOLID-SUBTYPE PAPILLARY THYROID CARCINOMA IN A PATIENT WITH CENTRAL HYPOTHYROIDISM: CHALLENGES IN DEFINING HORMONAL SUPPRESSION AND ONCOLOGICAL FOLLOW-UP

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Case presentation: A 71-year-old woman began investigation of a right thyroid nodule at age 66. Preoperative thyroid-stimulating hormone (TSH) and free thyroxine (FT4) were normal. Cytology was Bethesda IV, and diagnostic lobectomy was indicated. Histopathology revealed papillary thyroid carcinoma (PTC), high-grade solid subtype, measuring 1.7 × 1.4 cm, within a hyperplastic nodule, confined to the thyroid parenchyma, without necrosis, vascular invasion, or involved margins. Completion thyroidectomy was indicated because of the more aggressive histological subtype, and the patient was classified as intermediate risk. Postoperatively, persistently suppressed TSH associated with low or inappropriately normal FT4 suggested central hypothyroidism. Magnetic resonance imaging demonstrated a partially empty sella. Thereafter, levothyroxine (LT4) dosage was adjusted according to FT4 levels, rather than TSH, which could no longer assess suppression adequacy. Follow-up showed undetectable thyroglobulin (Tg) and anti-Tg antibodies and no structural disease on cervical ultrasound (US), consistent with an excellent response to treatment. **Discussion:** In PTC, particularly in intermediate- or high-risk patients, TSH suppression may reduce trophic stimulation of residual tumor cells, minimizing the risk of disease persistence or progression. However, in central hypothyroidism, TSH becomes unreliable and loses correlation with the administered LT4 dose. Thus, in patients for whom TSH suppression might otherwise be considered, central hypothyroidism creates an important therapeutic limitation. Furthermore, strategies relying on endogenous TSH elevation through LT4 withdrawal, whether for diagnostic scintigraphy or therapeutic radioiodine administration, are not feasible, potentially making these patients dependent on recombinant human TSH, which is not always widely available. LT4 treatment in this setting must be guided by FT4 monitoring, preferably maintaining values in the mid-to-upper reference range. **Conclusion:** Coexisting central hypothyroidism may complicate the management of patients with higher-risk PTC. In this case, undetectable Tg and anti-Tg antibodies and the absence of structural disease on cervical US supported an excellent response despite no radioiodine therapy or TSH suppression. Nevertheless, in patients with persistent or progressive disease, the inability to suppress TSH or raise it endogenously may make treatment decisions and follow-up considerably more challenging.
Keywords: central hypothyroidism; papillary thyroid carcinoma; thyroid-stimulating hormone.

THYROID

429 (3474) HYPERTHYROIDISM SECONDARY TO HYPERFUNCTIONING PULMONARY METASTASES OF PAPILLARY VARIANT FOLLICULAR THYROID CARCINOMA

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Case presentation: A 73-year-old female patient undergoing clinical investigation for chest pain was found to have multiple pulmonary nodules on chest computed tomography. Lung biopsy revealed tissue compatible with metastatic thyroid neoplasm. Following this finding, the patient underwent total thyroidectomy, and histopathological examination demonstrated follicular thyroid carcinoma (FTC), papillary variant. Postoperatively, levothyroxine therapy was initiated. After one month, laboratory evaluation showed suppressed thyroid-stimulating hormone (TSH) levels below 0.01 mIU/mL, elevated free thyroxine (FT4) at 4.23 ng/dL, and markedly elevated thyroglobulin (Tg) at 14,900 ng/mL. Despite progressive reduction of levothyroxine dosage, the patient continued to exhibit suppressed TSH and elevated FT4 and Tg levels. To investigate hyperfunctioning tissue, a low-dose whole-body radioiodine scan was performed, demonstrating diffuse and heterogeneous increased uptake in the pulmonary fields. Repeat laboratory assessment showed TSH below 0.01 mIU/mL, normal FT4 at 1.52 ng/dL, elevated total triiodothyronine at 1.97 ng/mL, elevated free triiodothyronine at 6.4 pg/mL, and Tg at 14,517 ng/mL. Considering the hypothesis of hyperfunctioning pulmonary metastases, methimazole 10 mg/day was initiated. Subsequently, methimazole was discontinued, and the patient underwent radioiodine therapy with 200 mCi of iodine-131. Post-therapy whole-body scintigraphy confirmed intense pulmonary uptake consistent with hyperfunctioning metastases. During follow-up, the patient presented normal TSH levels at 1.24 mIU/mL, suppressed FT4 below 0.25 ng/dL, and reduction of Tg to 2,097 ng/mL, leading to reintroduction of levothyroxine therapy. The patient subsequently demonstrated progressive reduction in Tg levels and maintenance of TSH within the target range for high-risk FTC. **Discussion:** Hyperfunctioning metastases from FTC represent a rare cause of hyperthyroidism. Mutations involving the TSH receptor, oncogenic KRAS and BRAF genes, and overexpression of type 2 deiodinase contribute to autonomous hormone production and maintenance of radioiodine avidity. **Final comments:** Early identification of hyperfunctioning metastases allows a favorable response to radioiodine therapy, with improvement in hormonal control and reduction in Tg levels. This case highlights the importance of recognizing and investigating patients with hyperthyroidism after total thyroidectomy for FTC.
Keywords: follicular thyroid carcinoma; hyperfunctioning metastases; hyperthyroidism, radioiodine therapy.

THYROID

430 (3324) HYPOTHYROIDISM AND DEPRESSION IN ADOLESCENTS: A SYSTEMATIC REVIEW

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Introduction: The relation between hypothyroidism and depression in adults is well defined, but its impact on adolescents is a topic that requires further investigation. **Objective:** To investigate the relationship between hypothyroidism and depression in adolescents. **Methods:** This systematic review was conducted using the PICO strategy and according to the PRISMA guidelines. A search was conducted for studies published in the last 10 years in English, Portuguese, and Spanish in PubMed, Scopus, and Embase databases using the MeSH terms Hypothyroidism, Depression and Adolescent combined with the Boolean operator "AND". 761 studies were identified and underwent double independent screening. Inclusion criteria were studies that evaluated adolescents, assessed laboratory measures of thyroid function, included assessment of depressive symptoms, observational studies and randomized clinical trials. Exclusion criteria were studies involving patients with diseases and/or conditions that may alter thyroid function, resulting in 6 articles for data extraction. **Results:** There is a higher prevalence of subclinical hypothyroidism and thyroid-peroxidase antibody positivity in adolescents with depression (9.1% vs. 2.1%; $p < 0.001$ and 8% vs. 5.8%; $p = 0.009$, respectively), although the severity of depressive symptoms did not show an association with either condition ($p > 0.05$), suggesting a pathophysiological role in depression. In contrast, other studies demonstrated serum thyroid-stimulating hormone (TSH) and free thyroxine to be lower ($p < 0.05$) in depressive adolescents, with higher values associated with lower odds of depression ($p < 0.05$); thyroid dysfunction was greater in these patients (17% vs. 7%; $p = 0.039$), mainly hypothyroxinaemia ($p = 0.012$), supporting the hypothesis that lower TSH levels may reduce monoamines, contributing to depression pathophysiology. Other studies demonstrated that TSH level distribution does not differ according to depression status ($p > 0.05$), with values remaining within the normal range, and adolescents with Hashimoto's thyroiditis do not demonstrate a relationship with depressive disorders ($p > 0.05$). **Conclusion:** Current evidence suggests an association between thyroid disorders and depression in adolescents. Although causality cannot be confirmed, these patients require investigation for thyroid disorders in cases of depression and vice versa. Further longitudinal studies are necessary to better understand this relationship.
Keywords: hypothyroidism; depression; adolescent.

THYROID

431 (3401) HYPOTHYROIDISM SECONDARY TO PEMBROLIZUMAB USE IN A PATIENT WITH METASTATIC PULMONARY ADENOCARCINOMA: A CASE REPORT

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Case presentation: A 68-year-old woman with stage IV metastatic pulmonary adenocarcinoma (T3N3M1a) and 95% PD-L1 expression started systemic treatment with carboplatin, pemetrexed, and pembrolizumab in October 2023. Three months after the initiation of immunotherapy, she developed asthenia and fatigue, prompting endocrinological evaluation. Laboratory tests revealed TSH 27.6 mIU/L and free T4 0.7 ng/dL, consistent with hypothyroidism. Previous medical records suggested hyperthyroidism related to immunotherapy. Levothyroxine 50 mcg/day was initiated and later adjusted to 125 mcg/day, resulting in symptomatic improvement and allowing continuation of oncologic treatment. **Discussion:** Immune-mediated endocrinopathies are important complications associated with the increasing use of immune checkpoint inhibitors, particularly anti-PD-1 monoclonal antibodies such as pembrolizumab. Thyroid dysfunction is among the most common manifestations. The pathophysiology involves loss of immune self-tolerance secondary to PD-1 pathway blockade, leading to exaggerated T-lymphocyte activation against the thyroid gland. The typical course initially includes a transient thyrotoxic phase caused by destruction of thyroid follicles and release of preformed hormones, followed by progressive reduction of glandular function and permanent hypothyroidism. In this case, although there was no laboratory confirmation of thyrotoxicosis, previous clinical suspicion of hyperthyroidism followed by laboratory-confirmed hypothyroidism suggests the classic biphasic evolution. The interval between pembrolizumab initiation and onset of endocrinopathy was also consistent with reports of immune-mediated thyroiditis associated with immunotherapy. **Conclusion:** The development of asthenia after initiation of immunotherapy prompted endocrinological investigation, leading to the diagnosis of hypothyroidism. This case highlights the importance of routine thyroid function monitoring in patients receiving pembrolizumab, especially in the presence of nonspecific symptoms during cancer treatment. It also reinforces the need for early recognition of endocrine adverse events related to immunotherapy. Integrated follow-up and appropriate hormone replacement therapy enabled adequate clinical control and maintenance of oncologic treatment.
Keywords: immune-mediated hypothyroidism; pembrolizumab; thyroiditis.

THYROID

432 (2875) IMPACT OF PREGNANCY ON DIFFERENTIATED THYROID CARCINOMA IN A PATIENT WITH INCOMPLETE STRUCTURAL RESPONSE

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Introduction, objectives and methods: Differentiated thyroid cancer (DTC) is three times more frequent in women, with ~10% of cases diagnosed during pregnancy. Patients diagnosed and treated before conception should be managed according to their response to therapy, as recommended by the 2025 American Thyroid Association (ATA) guidelines, since pre-gestational response is a strong predictor of pregnancy-related disease progression. Pregnancy is generally not considered to significantly affect DTC prognosis, although data remain conflicting. This study aimed to evaluate the impact of pregnancy on DTC exclusively in patients with known metastatic disease, defined as incomplete structural response. A retrospective cohort was conducted including women with DTC who became pregnant after total or subtotal thyroidectomy, with or without cervical lymph node dissection and radioiodine therapy, and who had incomplete structural response prior to pregnancy. **Results:** Among 169 pregnant women with DTC, 32 (19%) had pregestational incomplete structural response. Disease progression after pregnancy occurred in 12 patients. Of these, 10 had classical papillary thyroid carcinoma and 2 had the follicular variant. Nine were classified as high recurrence risk with lateral neck lymph node metastases (N1b), and 6 had distant metastases at diagnosis (M1). Seven required additional treatment postpartum. Nine patients had slowly progressive disease prior to pregnancy, while only 3 were stable before progressing after delivery. In multivariate logistic regression (n = 32), the model was not statistically significant (likelihood ratio $p = 0.152$). However, high recurrence risk showed a strong trend toward independent association with post-pregnancy progression ($\beta = 2.46$; $p = 0.065$), corresponding to an approximate odds ratio of 11.7. These findings suggest progression is mainly related to intrinsic tumor characteristics, particularly biological aggressiveness, rather than pregnancy itself. Limited sample size reduced statistical power, but the observed trend aligns with known DTC natural history. **Conclusion:** Pregnancy did not impact the natural course of DTC in patients with incomplete structural response. Post-pregnancy progression was limited and likely associated with more aggressive disease and higher initial risk stratification.
Keywords: differentiated thyroid carcinoma; pregnancy; incomplete structural response.

THYROID

433 (3020) IMPACTS OF MATERNAL EXPOSURE TO BISPHENOL F AND AF ON THYROID FUNCTION IN WISTAR RATS: EFFECTS DURING LACTATION AND ADULTHOOD

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Endocrine-disrupting chemicals (EDCs), particularly bisphenol A (BPA) and its analogs bisphenol F (BPF) and bisphenol AF (BPAF), interfere with thyroid function and may lead to metabolic programming during critical developmental periods. This study evaluated the effects of maternal exposure to BPF and BPAF (10 or 50 µg/kg) during lactation on thyroid function parameters in Wistar rat dams and their offspring during childhood (21 days) and adulthood (120 days) (CEUA/031/2022). Dams exposed to BPF10 showed an increase in DUOX activity (+87%, $p < 0.05$), while BPAF10-exposed dams exhibited an elevation in plasma T4 levels (+181%, $p < 0.05$), although thyroid mass and gene expression (DUOX, NIS, TSH-R, TPO, NOX2) remained unchanged. In offspring at weaning, BPF10-exposed males displayed an increase in DUOX activity (+57%, $p < 0.05$), whereas females showed a significant reduction of this enzyme activity (BPF10: –55%; BPF50: –49%). In adulthood, male offspring from BPAF groups exhibited > 50% decreases in DUOX and TPO expression ($p < 0.05$), as well as elevated oxidative stress (TBARS for BPF50: +185%; TBARS for BPAF50: +132%; $p < 0.05$). All groups showed an increase in catalase and hormonal disturbances (+62% TSH in BPF50; +109% and +114% of T4 in BPF50 and BPAF groups, $p < 0.05$). Female offspring presented reduced DUOX expression (59%–66% for BPAF groups, $p < 0.05$) and diminished catalase activity, despite unchanged plasma thyroid hormones. Importantly, thyroid mass remained unaltered in all groups. These findings demonstrate that early life BPF and BPAF exposure induces sex- and age-specific thyroid dysfunction, with low-dose effects persisting into adulthood. The sustained oxidative stress (reduced catalase, elevated TBARS) and hormonal imbalances suggest metabolic programming of thyroid dysfunction. These results challenge the presumed safety of BPA alternatives and emphasize the need for rigorous assessment of their developmental impacts.
Keywords: BISPHENOL; EDCS; thyroid.

THYROID

434 (3478) IMPLEMENTATION OF THE 2015 AMERICAN THYROID ASSOCIATION GUIDELINE 35 FOR THE MANAGEMENT OF WELL-DIFFERENTIATED THYROID CARCINOMA

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Objective: To analyze the scenary of SUS in the state of Rio de Janeiro (RJ), whether the 2015 ATA (American Thyroid Association) guideline 35 was implemented, which recommended lobectomy for unifocal and intrathyroidal tumors smaller than 4 cm, without extrathyroidal extension or lymph node metastases in DTCs. **Methods:** Analysis of secondary data available in Datasus from 2008 to 2024, in the state of RJ, of procedures classified by SIGTAP as partial thyroidectomies (PT), total (TT) and with central neck dissection (T+D), whose admission ICD-10 was malignant thyroid neoplasm. We used multinomial logistic regression with marginal effects to evaluate the incorporation of the referred guideline. **Results:** A total of 4,239 patients undergoing surgery (9.79% PTs, 84.43% TTs, 5.78% T+D) for malignant thyroid neoplasm were identified in RJ over 16 years, the majority being women (86.48%, $p < 0.05$). Men underwent more T+Ds than women (10.12% vs. 5.10%, $p < 0.05$); in the other types of surgery there was no difference. There was an important drop in TTs during 2020 due to the pandemic, resulting in 188 surgeries that year. The multinomial logistic regression revealed a significant model ($p < 0.05$), with a pseudo R^2 of 0.0417. Over the 16 years, an average reduction of 14.6% per year was observed in the chance of PT compared to TT. This result was associated with the negative coefficient of the year variable (–0.158) which the procedure was realized. An indicator variable for the years after 2016 (post-ATA guideline period) was also tested. Although its coefficient suggested a possible increase in PT after 2016 (0.317), the result was not statistically significant ($p = 0.117$). Marginal probabilities confirmed the downward trend: PTs fell from 26.5% in 2008 to 3.3% in 2024, against the expectations of greater adoption after the 2015 American guideline. Limitations of this kind include the absence of tumor size and low-risk staging data in Datasus. **Conclusion:** Although an increase in PT was expected after 2016, the data reveal an opposite trend, characterized by a continuous reduction with no evidence of reversal. This decline may reflect the non-incorporation of the 2015 ATA guideline in the SUS of RJ. Difficult access to reoperation and follow-up after PTs, together with clinical staging of more advanced disease as seen in men, may become an issue in selecting patients eligible for lobectomies. Surgeons maintained TT as the main treatment for malignant thyroid neoplasms.

Keywords: thyroid neoplasm; implementation; lobectomy.

THYROID

435 (3215) LONG-TERM OUTCOMES OF PATIENTS UNDERGOING SURGICAL TREATMENT FOR PAPILLARY THYROID MICROCARCINOMA: A RETROSPECTIVE ANALYSIS IN LIGHT OF THE 2025 ATA GUIDELINES

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Introduction: Papillary thyroid microcarcinoma (PTMC), defined as papillary thyroid carcinoma ≤ 10 mm, represents an increasing proportion of differentiated thyroid cancers, mainly due to widespread cervical ultrasonography. Despite disease-specific survival > 99% at 10 years, total thyroidectomy, radioactive iodine (RAI) and intensive surveillance has been frequently adopted. The 2025 ATA consensus favors a conservative, individualized approach for low-risk disease. **Objective:** To describe clinicopathological features and long-term outcomes of patients surgically treated for PTMC at a tertiary endocrinology center. **Methods:** Retrospective cohort including PTMC patients submitted to surgery, followed ≥ 5 years, evaluated between October 2025 and March 2026. Exclusion criteria: absent thyroglobulin (Tg) at reassessment, multicentric tumors, or < 5 years of follow-up. Risk stratification and response-to-therapy categories were reclassified per ATA 2025 criteria. **Results:** Twenty-five patients included; 92% women (n = 23), mean age 49.7 ± 13.0 years. Mean tumor size: 0.52 ± 0.26 cm. Classical variant predominated (76%; n = 19), followed by follicular (16%; n = 4) and one hobnail variant. Per ATA 2025: 80% low risk (n = 20), 20% intermediate risk (n = 5). Cervical lymph node metastases in 16% (n = 4). All underwent total thyroidectomy; 20% (n = 5) also underwent neck dissection. RAI administered in 60% (n = 15), all intermediate-risk and 50% of currently classified low-risk patients. Postoperative hypoparathyroidism in 24% (n = 6). At ≥ 5 years: 80% excellent response (n = 20), 16% indeterminate (n = 4), 4% structurally incomplete (n = 1). Median Tg: 0.10 ng/mL; anti-Tg antibodies negative in 96% (n = 24). No distant metastases or disease-specific deaths. **Conclusion:** PTMC demonstrated excellent long-term oncological outcomes with high excellent response rates and no disease-specific mortality. Findings suggest historical RAI overutilization in low-risk patients without apparent impact on outcomes, reinforcing contemporary recommendations for conservative, risk-adapted PTMC management.

Keywords: papillary thyroid microcarcinoma; radioactive iodine therapy; risk stratification.

THYROID

436 (2983) MACRO-TSH IN TURNER SYNDROME AND AUTOIMMUNE HYPOTHYROIDISM: A DIAGNOSTIC CHALLENGE

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Case presentation: An 18-year-old female with Turner syndrome and autoimmune hypothyroidism was followed since childhood for short stature and had received growth hormone therapy since 2012. She had positive anti-TPO antibodies and was on levothyroxine (LT4) with regular dose adjustments. Despite strict adherence and appropriate weight-based dosing, laboratory tests consistently showed elevated TSH with normal or increased free T4 levels. In April 2024, TSH was 50 U/mL and free T4 0.91 ng/dL while using LT4 112 mcg/day (2.30 mcg/kg). Investigation for macro-TSH was performed, and recovery after polyethylene glycol precipitation was 24.57%, confirming the diagnosis. After increasing LT4 to 125 mcg/day (2.49 mcg/kg), TSH remained elevated at 24.7 U/mL in April 2025, with normal free T4 (1.30 ng/dL). Monomeric TSH was 6 U/mL, supporting the presence of biologically inactive macro-TSH. The patient remained asymptomatic and clinically euthyroid, with no indication for further LT4 escalation. **Discussion:** Macro-TSH is a rare and underrecognized cause of persistently elevated TSH in clinically euthyroid patients. It usually results from binding of TSH to immunoglobulins, forming high-molecular-weight complexes detected by immunoassays but lacking biological activity. This leads to discordance between TSH and free T4 levels and may mimic refractory hypothyroidism. Diagnostic methods include polyethylene glycol precipitation, gel filtration chromatography, and monomeric TSH measurement. Recognition of macro-TSH is essential to avoid unnecessary dose escalation and iatrogenic thyrotoxicosis. In this case, the coexistence of Turner syndrome and autoimmune hypothyroidism increased diagnostic complexity, since autoimmune thyroid disease is highly prevalent in these patients. **Final comments:** This case highlights the importance of considering macro-TSH in patients with persistently elevated TSH despite adequate treatment adherence, especially when free T4 levels are normal and clinical hypothyroidism is absent. Early recognition prevents unnecessary therapeutic interventions and potential adverse effects from excessive LT4 replacement.
Keywords: macro-TSH; Turner syndrome; autoimmune hypothyroidism.

THYROID

437 (3047) MARINE-LENHART SYNDROME CASE REPORT OF A RARE SYNDROME IN A TERTIARY HOSPITAL

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Case presentation: Patient S.S.F., 51 years old, female, active smoker, presented to the Endocrinology outpatient clinic with complaints of 18 kg weight loss over the past year, associated with palpitations, irritability, and diarrhea. Two months prior, she developed dysphonia. She reported having started treatment for hyperthyroidism with methimazole for 5 years; however, she discontinued the medication 1 year ago, with worsening of symptoms since then. At that time, laboratory tests showed TSH 0.01 (reference value (VR) 0,27–4,2 mUI/L) and free T4 1.38 (VR 0,9–1,7 ng/dL); at diagnosis in 2020: TSH 0.008 and free T4 2.05, and thyrotropin receptor antibodies (TRAb) persistently positive since 2020, confirmed again in 2026. She brought a thyroid ultrasound showing three nodules in the left lobe, measuring 0.7 cm, 1.4 cm, and 2.0 cm at their largest diameters, all classified as TI-RADS 3. On physical examination, a fibroelastic, painless nodule was palpable in the upper left lobe. There were no clinical signs of ophthalmopathy. Given the ultrasound findings, a thyroid scintigraphy was performed in September 2025, demonstrating a hot nodular goiter in the left lobe. Thus, due to the association of Graves' disease (refractory to methimazole) and a functioning nodular goiter on scintigraphy, the diagnosis of Marine-Lenhart syndrome was established. Due to refractoriness to conservative clinical treatment, the patient was referred to the nuclear medicine team for radioiodine therapy planning. **Discussion:** Marine-Lenhart syndrome is a rare condition resulting from the coexistence of two distinct pathophysiological entities: Graves' disease (GD) and a functioning thyroid nodule, occurring in approximately 1%–2.7% of patients with GD. Although GD and toxic nodular goiter are among the most common causes of hyperthyroidism worldwide, they have different pathophysiological mechanisms, and their coexistence is uncommon. Therefore, this syndrome is associated with a lower remission rate with antithyroid drugs and may require higher doses of radioiodine during treatment. **Final comments:** Marine-Lenhart syndrome has greater clinical importance than its low prevalence suggests, as it changes how hyperthyroidism should be interpreted and managed. Additionally, there is greater difficulty in achieving clinical control due to the different disease mechanisms involved, as well as a higher likelihood of requiring definitive treatment.
Keywords: Marine-Lenhart; hyperthyroidism; nodule.

THYROID

438 (3293) MARINE-LENHART SYNDROME: THERAPEUTIC CHALLENGES IN A CASE REPORT

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Case presentation: A 60-year-old female patient was referred to the endocrinology service (CEMEC/PA) due to thyrotoxicosis presenting with palpitations, nail fragility, hair loss, insomnia, and weight loss. Physical examination revealed bilateral exophthalmos, diffuse goiter, and a palpable 3-cm nodule in the left thyroid lobe. Laboratory evaluation demonstrated suppressed TSH (<0.01 mIU/mL), elevated free T4 (3.46 ng/dL; RR: 0.93–1.70), and positive thyrotropin receptor antibodies (TRAb: 2.33 IU/L; RR: <1.75). Methimazole 10 mg/day was initiated, with subsequent dose escalation. Thyroid ultrasound revealed diffuse goiter and heterogeneous solid nodules measuring 3.4 × 2.6 cm in the left lobe and 0.5 cm in the right lobe. Technetium-99m pertechnetate scintigraphy demonstrated diffusely increased uptake with a focal hyperfunctioning area corresponding to the left lobe nodule. A therapeutic dose of 30 mCi of iodine-131 was administered, followed by an initial increase in free T4 to 7.7 ng/dL. Thionamide therapy was subsequently up-titrated. After 6 months, the patient is currently undergoing methimazole tapering. **Discussion:** This clinical course illustrates the challenges of Marine-Lenhart syndrome (MLS), a rare condition characterized by the coexistence of Graves' disease and autonomously functioning thyroid nodules. Transient increases in free thyroid hormones after radioiodine therapy have been reported in approximately 30%–50% of patients with Graves' disease, particularly within the first 2–6 weeks post-treatment, secondary to radiation-induced thyroid follicular disruption and release of preformed thyroid hormones. **Final comments:** This case highlights the necessity of vigilant monitoring and dynamic thionamide adjustments when treating Marine-Lenhart syndrome with radioiodine, in order to safely manage the anticipated, radiation-induced exacerbation of thyrotoxicosis.

Keywords: Marine-Lenhart syndrome; hyperthyroidism; iodoterapy.

THYROID

439 (2958) METACHRONOUS TUMORS (PAPILLARY THYROID CARCINOMA, CLEAR CELL RENAL CARCINOMA, AND LUNG ADENOCARCINOMA) IN A FEMALE PATIENT UNDER ENDOCRINOLOGY FOLLOW-UP: A CASE REPORT

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Case presentation: A 57-year-old female patient, obese and diabetic, under endocrinology follow-up due to papillary thyroid carcinoma (pT4aN1bM0), diagnosed in 1999. In the same year, she underwent total thyroidectomy, followed by bilateral neck dissections (2000 to 2002), and four doses of radioiodine therapy (2000, 2001, 2005, and 2006), totaling 700 mCi. During follow-up, she presented persistently elevated thyroglobulin (Tg) levels, with negative whole-body scans after administration of the last dose of radioiodine, being characterized as radioiodine-refractory differentiated thyroid carcinoma. In 2022, a lesion in the left kidney (4.5 cm) was identified, and she underwent partial nephrectomy, with histopathology revealing clear cell renal cell carcinoma (pT1aN0M0). In the same year, after biopsy of a pulmonary lesion, she was diagnosed with stage IIB lung adenocarcinoma (ypT1bN0), undergoing neoadjuvant chemotherapy followed by left lower lobectomy. Molecular analysis of the lung tumor revealed an R337C mutation in the ATM tumor suppressor gene and the presence of a V600E mutation in the BRAF gene. In 2024, she presented recurrence of the pulmonary lesion in the left upper lobe, associated with pleural and bone involvement, and chemotherapy and radiotherapy were chosen for bone metastases (T9 and L5). In 2025, she developed lesions in the frontal bone and cerebellum, compatible with metastases, and underwent radiotherapy (December 2025 to January 2026) and stereotactic radiosurgery (March 2026). Currently, she remains under follow-up with Endocrinology, Neurosurgery, Oncology, and Urology teams, with recent progressive elevation of thyroglobulin levels (March 2026: Tg 249 ng/mL [1.6–59.9]). **Discussion:** Metachronous tumors are primary malignant neoplasms diagnosed at different time points, with an interval greater than 6 months. It is a rare condition, with only one report in the literature correlating these three tumors concurrently. There are still no studies proving a relationship between increased incidence of the described neoplasms and exposure to high doses of radioiodine, and other risk factors should be considered, such as genetic mutations, obesity, and diabetes mellitus contributing to a higher risk of developing neoplasms. **Final comments:** This case highlights the importance of multidisciplinary follow-up in the management of metachronous tumors for early identification and treatment of neoplasms, aiming to improve patient survival.

Keywords: metachronous tumors; radioiodine therapy; risk factors.

THYROID

440 (3518) METASTATIC MEDULLARY THYROID CARCINOMA WITH MULTISYSTEM INVOLVEMENT IN A YOUNG PATIENT: A CASE REPORT

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Case presentation: A 36-year-old previously healthy man presented with progressive severe low back pain refractory to conventional analgesia. During diagnostic investigation, he reported a previously unnoticed cervical mass. Imaging studies demonstrated a thyroid nodule associated with pulmonary, hepatic, osseous, and central nervous system metastases. Laboratory evaluation revealed calcitonin levels > 2,000 pg/mL and carcinoembryonic antigen of 335 ng/mL. Fine-needle aspiration confirmed medullary thyroid carcinoma with metastatic cervical lymph node involvement. Germline RET mutation testing was negative. The patient subsequently developed spinal cord compression, lower limb paresis, and urinary retention, with partial neurological improvement after corticosteroid therapy and palliative whole-brain and spinal radiotherapy. He also received zoledronic acid and systemic treatment, initially with lenvatinib followed by vandetanib, achieving partial clinical stabilization and symptomatic improvement. **Discussion:** This case highlights an aggressive and uncommon presentation of medullary thyroid carcinoma, with metastatic bone disease manifesting as the initial clinical feature. It also emphasizes the diagnostic relevance of calcitonin and carcinoembryonic antigen levels, the role of molecular investigation, and the expanding therapeutic landscape of advanced metastatic disease. **Final comments:** Early recognition of suspicious thyroid lesions and atypical metastatic manifestations is essential to reduce diagnostic delay and morbidity associated with advanced medullary thyroid carcinoma.
Keywords: medullary thyroid carcinoma; bone metastasis; targeted therapy.

THYROID

441 (3431) METASTATIC PAPILLARY THYROID CARCINOMA WITH NTRK FUSION TREATED WITH TARGETED THERAPY: A CASE REPORT

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Case presentation: A 66-year-old male former smoker presented with a 4-year history of persistent dry cough, progressing to worsening dyspnea and a 25-kg weight loss. During the diagnostic workup, chest CT revealed multiple bilateral pulmonary nodules, osteolytic lesions, and a thyroid mass with mediastinal extension. Lung biopsy confirmed metastatic papillary thyroid carcinoma. Due to severe pulmonary involvement, with resting hypoxemia and need for home oxygen therapy, the patient was initially considered at high surgical risk. Pazopanib therapy was initiated, leading to partial clinical improvement and allowing subsequent surgical intervention. The patient underwent total thyroidectomy with right selective level VI neck dissection, which was incomplete because of local tumor invasion without a cleavage plane from the trachea and brachiocephalic trunk. Histopathology demonstrated multifocal classic papillary thyroid carcinoma with tumor necrosis and focally positive margins. After postoperative discontinuation of pazopanib for radioactive iodine therapy, the patient developed significant respiratory worsening. Whole-body scan showed extensive pulmonary and bone metastatic disease, with stimulated thyroglobulin levels > 500 ng/mL. Molecular analysis identified an EML4::NTRK3 gene fusion. Larotrectinib therapy was initiated, resulting in marked pulmonary improvement, resolution of dyspnea and cough, discontinuation of oxygen therapy, and a 10-kg weight gain. **Discussion:** Extensive metastatic disease at diagnosis is uncommon in papillary thyroid carcinoma. In such cases, NTRK gene fusions may be present in up to 6% of patients. Therefore, molecular tumor profiling plays an important role in prognosis assessment and therapeutic decision-making, particularly in aggressive presentations with metastatic or radioactive iodine-refractory disease. This enables the use of targeted therapies such as larotrectinib, which has shown high response rates in NTRK fusion-positive thyroid tumors, leading to early clinical benefit and significant functional improvement. **Final comments:** This case highlights the relevance of molecular profiling in expanding therapeutic possibilities for atypical presentations of differentiated thyroid carcinoma. The clinical improvement observed after initiation of larotrectinib demonstrates the benefit of targeted therapy in cases of advanced metastatic disease.
Keywords: papillary thyroid carcinoma; NTRK fusion; larotrectinib.

THYROID

442 (3469) METHIMAZOLE-INDUCED CHOLESTATIC SYNDROME IN A PATIENT WITH GRAVES' DISEASE: A CASE REPORT

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Case presentation: A 20-year-old male patient was diagnosed with hyperthyroidism due to Graves' disease in 2024. Treatment with methimazole was initiated, with gradual dose escalation up to 20 mg/day, later reduced to 10 mg/day in August 2025. In November 2025, he developed progressive scleral icterus and was admitted for further investigation. Laboratory tests revealed mixed hyperbilirubinemia with direct predominance (total bilirubin 12.16 mg/dL; direct bilirubin 7.34 mg/dL), elevated alkaline phosphatase (381 U/L), increased aminotransferases (AST 114 U/L; ALT 194 U/L), and normal gamma-glutamyltransferase levels, consistent with a predominantly cholestatic pattern. During hospitalization, the patient reached a peak total bilirubin level of 15.14 mg/dL. Viral hepatitis serologies and infectious workup were negative. Magnetic resonance cholangiography showed no biliary obstruction or significant structural liver abnormalities. Methimazole was immediately discontinued, with no subsequent re-exposure. The patient showed progressive clinical and laboratory improvement after drug withdrawal. Due to persistent thyrotoxicosis, radioactive iodine therapy with 25 mCi was performed in December 2025, resulting in improvement of hyperthyroidism and complete resolution of liver abnormalities. **Discussion:** Methimazole-induced hepatotoxicity is a rare and generally unpredictable event that may present with hepatocellular, mixed, or cholestatic patterns. The cholestatic presentation is uncommon, with few cases described in the literature. The absence of obstructive findings on magnetic resonance cholangiography, together with negative infectious serologies and clinical improvement following drug discontinuation, supported the diagnosis of methimazole-induced liver injury. In the literature, most cases show progressive resolution after withdrawal of the antithyroid drug, and reintroduction of the medication is contraindicated. In patients with persistent hyperthyroidism, definitive therapies such as radioactive iodine treatment should be considered. **Final comments:** This report highlights a rare complication of methimazole in a young patient with Graves' disease and emphasizes the importance of early recognition of antithyroid drug-induced liver injury. Although uncommon, methimazole-induced cholestatic hepatotoxicity should be promptly recognized, as early drug discontinuation may prevent progression to severe forms of liver injury.

Keywords: hyperthyroidism; methimazole; cholestasis.

THYROID

443 (2941) MIXED PAPILLARY/MEDULLARY THYROID CARCINOMA PRESENTING WITH NODAL METASTASES: CASE REPORT

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Case presentation: A 47-year-old woman presented with a Bethesda VI thyroid nodule and a right level IV cervical lymph node measuring 1.8 x 1.0 x 2.9 cm. She underwent total thyroidectomy with central neck dissection (10/22). Histopathology revealed an oncocytic variant papillary thyroid carcinoma in the right lobe (2.5 x 1.7 cm), non-encapsulated, with no vascular, perineural, or extrathyroidal invasion. Metastasis occurred to 1/6 lymph nodes without extranodal extension (pT2N1aMx; intermediate-low risk). The patient received 100 mCi of 131I with discrete cervical uptake. In 06/2024, thyroglobulin (Tg) rose to 10.3 ng/mL and anti-thyroglobulin antibodies (TgAb) rose; imaging identified cervical lymphadenopathy at level IV–D (1.8 x 1.0 x 2.9 cm). Biopsy confirmed metastasis. She underwent right lateral neck dissection (levels IIA, III, IV) in 07/2024, revealing papillary carcinoma metastasis in 2/20 nodes with extranodal extension. Immunohistochemistry confirmed Tg positivity and CK19/CD56 negativity, and metastasis of medullary thyroid carcinoma in 5/20 nodes, with positive synaptophysin, chromogranin, and calcitonin. Subsequently, she received 150 mCi of 131I with discrete cervical uptake. A neck ultrasound 3 months after revision surgery showed no suspicious lymph nodes. Biochemical surveillance demonstrated an excellent response: Tg 0.04 ng/mL; TgAb 20 IU/mL (declining); TSH 0.03 mIU/L on levothyroxine 100 µg/day; free T4 1.52 ng/dL; calcitonin < 1 pg/mL; CEA 3.58 ng/mL. Primary hyperparathyroidism and pheochromocytoma were excluded. A genetic panel showed no clinically meaningful germline variants. **Discussion:** Medullary carcinoma is sporadic in 75% of cases and typically presents in the fourth to sixth decades. Papillary carcinoma nodal metastases occur in 20%–50%, especially within the first 5 years of follow-up. Synchronous papillary and medullary carcinoma (type II) is rare (0.1%–9.5%). This case is unique because, at presentation, there was no prior description of a medullary component, hence no preoperative calcitonin or CEA. Nodal metastases in mixed tumors occur in 56%–75%, predominantly driven by the medullary component, which largely influences prognosis. **Final comments:** The patient remains under semiannual surveillance of calcitonin, CEA, Tg, and anti-Tg Ab, plus cervical ultrasound, with no structural or biochemical evidence of disease. Disease-specific survival for mixed thyroid carcinomas appears higher than for isolated medullary carcinoma.

Keywords: lymph node metastasis; medullary thyroid carcinoma; papillary thyroid carcinoma.

THYROID

444 (3302) OCCULT MEDULLARY THYROID MICROCARCINOMA AFTER PROPHYLACTIC THYROIDECTOMY IN A CHILD WITH A RET CODON 620 GERMLINE MUTATION

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Case presentation: A 12-year-old boy was identified as a heterozygous carrier of the pathogenic familial RET p.Cys620Arg variant in exon 10 through cascade screening after his father was diagnosed with metastatic medullary thyroid carcinoma. Genetic testing at 10 years confirmed RET NM_020975.6: c.1858T>C, p.(Cys620Arg). He was clinically asymptomatic. Preoperative evaluation a few months before surgery showed minimally elevated serum calcitonin, 8.9 pg/mL (reference < 8.4), with normal carcinoembryonic antigen, 1.4 ng/mL (reference < 2.5). Neck ultrasonography did not reveal suspicious thyroid nodules. A 0.3 x 0.2 cm anechoic cystic image was described in the right thyroid lobe, while later imaging was normal; reactive cervical lymph nodes were attributed to an upper airway infection. After multidisciplinary discussion, isolated risk-reducing total thyroidectomy, without central neck dissection, was performed at 12 years of age. Histopathology revealed a 0.3 x 0.2 cm epithelial cell neoplasm in the left thyroid lobe, contralateral to the previous cystic ultrasound finding. There was no extrathyroidal extension, necrosis, vascular, lymphatic or perineural invasion, and margins were free. Immunohistochemistry confirmed medullary thyroid carcinoma, staged as pT1aNx. **Discussion:** Hereditary medullary thyroid carcinoma requires early identification of at-risk relatives and individualized timing of thyroidectomy according to genotype, age, calcitonin and imaging. RET codon 620 variants belong to the moderate-risk category, in which thyroidectomy timing is individualized and guided mainly by calcitonin rather than by a fixed early-age threshold. In this case, the absence of suspicious ultrasound findings and only minimally increased preoperative calcitonin could have supported continued surveillance. However, occult medullary thyroid microcarcinoma after risk-reducing surgery demonstrates that early disease may already be present despite non-diagnostic imaging and very subtle biochemical expression. The case also illustrates the value of cascade genetic testing after advanced disease in a parent. **Final comments:** This report highlights the importance of individualized decision-making in pediatric RET codon 620 carriers. Minimally elevated calcitonin in a genetically at-risk child should not be dismissed, even when ultrasound is not suspicious. Risk-reducing thyroidectomy may reveal clinically occult medullary thyroid carcinoma at a potentially curable stage.
Keywords: medullary thyroid carcinoma; RET proto-oncogene; pediatric prophylactic thyroidectomy.

THYROID

445 (3231) ONCOLOGICAL OUTCOMES OF ACTIVE SURVEILLANCE VERSUS IMMEDIATE SURGERY IN PATIENTS WITH LOW-RISK PAPILLARY THYROID MICROCARCINOMA: A SYSTEMATIC REVIEW

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Introduction: Thyroid cancer incidence is rising globally, with 821,000 new cases in 2022. In Brazil, 16,450 new cases are projected annually for 2026–2028; however, mortality remains very low, questioning current therapeutic paradigms. The detection of papillary thyroid microcarcinoma (PTMC), tumors ≤ 10 mm, is largely driven by ultrasound-based overdiagnosis. Active surveillance (AS) has emerged as a safe, cost-effective alternative to immediate surgery. Nevertheless, international consensus is lacking regarding ideal patient profiles and mandatory surgical conversion triggers. This gap is critical in the Brazilian Public Health System (SUS), where decisions directly impact clinical efficiency and costs. **Objective:** To compare oncological outcomes of active surveillance versus immediate surgery in low-risk PTMC, evaluating tumor progression, lymph node metastasis, recurrence, mortality, and surgical conversion. **Methods:** This systematic review followed PRISMA guidelines. PICO strategy: Adults with low-risk PTMC (P); Active Surveillance (I); Immediate Surgery (C); Oncological outcomes (O). Searches were conducted in PubMed, Embase, EBSCO, Scopus, SciELO, and BVS. Inclusion: original cohort studies (2022–2026). Exclusion: reviews, case reports, and high-risk tumors. Bias risk was assessed via the Newcastle-Ottawa Scale. **Results:** Five cohort studies were selected (>10,000 patients). In the AS group, tumor growth (≥3 mm) occurred in 4.7% at 10 years and 6.6% at 20 years. New lymph node metastases were rare (1.6% at 20 years). Thyroid cancer-specific mortality was zero across all cohorts, regardless of the initial approach. Surgical conversion rates ranged from 8.4% to 23.9%, driven by objective progression (56.8%) or patient preference/anxiety. Late conversion surgery did not increase permanent complications; permanent vocal cord paralysis was 0.9% in the immediate surgery group versus 0% in the conversion group. The surgical group reported higher symptom interference and scar dissatisfaction. Five-year loss to follow-up was significantly higher in AS (28.6% vs. 17.8%). **Conclusion:** Active surveillance is oncologically safe for low-risk PTMC, yielding outcomes equivalent to surgery with zero mortality. Delayed surgery does not increase permanent surgical risks. Success relies on strict adherence, with younger age being a predictor of progression. AS should be offered as a first-line option through shared decision-making.
Keywords: thyroid neoplasms; active surveillance; treatment outcome.

THYROID

446 (3119) ORBITOPATHY WITHOUT HYPERTHYROIDISM: A DIAGNOSTIC AND THERAPEUTIC CHALLENGE

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Case presentation: A 50-year-old male was referred to endocrinology by ophthalmology for thyroid disease investigation. He reported bilateral conjunctival irritation, ocular pain, and progressive proptosis since 2019-2020, following COVID-19, with formal follow-up only initiated in 2023. He denied hyperthyroid symptoms and reported partial improvement of ocular symptoms with prednisone (40 mg/day for 7 days, maintained at 20 mg/day). Medical history: Graves; disease diagnosed approximately one year prior and mixed dyslipidemia. Medications: prednisone 20 mg/day, rosuvastatin 20 mg/day, and lubricant eye drops. Physical examination: heart rate 62 bpm, blood pressure 120 x 70 mmHg. Laboratory: TSH 0.82 uIU/mL, free T4 1.40 ng/dL, total T3 110 ng/dL, thyrotropin receptor antibody (TRAB) 2.33 IU/L (ref. 0.10; previously 3.2 IU/L), anti-thyroperoxidase antibody negative. Thyroid ultrasound: diffuse goiter (21.1 cm^3) with 22 mm hypoechoic nodule. Orbital computed tomography: right extrinsic muscle thickening with mild proptosis. Cranial computed tomography: unremarkable. **Discussion:** Euthyroid Graves; disease is a rare entity, defined by thyroid-associated ophthalmopathy with normal thyroid function and no prior antithyroid treatment. Graves; orbitopathy predominantly affects hyperthyroid patients, with the euthyroid form accounting for only 1%–2% of cases. Elevated thyrotropin receptor antibody and compatible orbital imaging support the diagnosis in the absence of thyroid dysfunction. Up to 72.4% of euthyroid Graves; disease patients remain euthyroid over a median follow-up of 5.8 years, while 24.1% progress to hyperthyroidism, imposing the need for long-term surveillance. Disease activity and severity guide therapeutic decisions: response rates of 50%–60% with oral corticosteroids versus 70%–80% with intravenous administration. **Final comments:** This case demonstrates that euthyroid Graves; disease may present significant orbitopathy in the absence of thyroid dysfunction. Elevated thyrotropin receptor antibody and orbital imaging confirm the diagnosis. Multidisciplinary follow-up with periodic reassessment using the Clinical Activity Score is essential to guide treatment escalation and prevent irreversible orbital damage.

Keywords: euthyroid graves disease; thyroid-associated ophthalmopathy; thyrotropin receptor antibody.

THYROID

447 (3441) PERSISTENT THYROID TEST DISCORDANCE AFTER AMIODARONE DISCONTINUATION

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Case presentation: A 77-year-old man with structural heart disease, atrial fibrillation, and amiodarone use had incidental thyroid uptake on PET-CT during evaluation of a pulmonary nodule. Neck ultrasound showed thyroid nodules, and fine-needle aspiration of a left nodule was Bethesda VI. Preoperatively, he developed biochemical thyrotoxicosis in the context of amiodarone use, with suppressed TSH and elevated free T4. He underwent total thyroidectomy in August 2025; pathology showed papillary thyroid carcinoma, tall-cell variant, unifocal, 1.2 cm, with microscopic extrathyroidal extension, negative margins, and no vascular, lymphatic, or perineural invasion, pT1b. He received 50 mCi radioiodine in November 2025; post-therapy scan showed mild cervical uptake and no distant metastases. On levothyroxine 112 mcg/day, he developed laboratory discordance while still on amiodarone: TSH 10.1 mIU/L and FT4 2.37 ng/dL in December 2025; TSH 11.3 mIU/L, FT4 2.16 ng/dL, and T3 66 ng/dL in February 2026. Amiodarone was discontinued by cardiology in March 2026; afterward, TSH was 18.1 mIU/L, with FT4 1.97 ng/dL and T3 59 ng/dL. **Discussion:** Amiodarone is associated with thyroid dysfunction and hormonal abnormalities through inhibition of T4-to-T3 conversion, iodine load, long half-life, and tissue accumulation. In this case, the initial phase with suppressed TSH and elevated FT4 was compatible with amiodarone-associated thyrotoxicosis in a patient with an in situ thyroid. After thyroidectomy, elevated TSH with elevated FT4 and low T3 was compatible with the described effect of amiodarone on deiodinases, with possible reduction in peripheral and intrapituitary T3 generation. In an athyreotic patient dependent on levothyroxine and peripheral T3 generation, this interference may be more evident. Irregular levothyroxine intake, biotin use, and analytical interference should be considered. Interpreting TSH or FT4 in isolation may lead to harmful dose adjustments in an elderly patient with atrial fibrillation. **Final remarks:** This case highlights amiodarone as a cause of complex, persistent thyroid abnormalities, not limited to active use. The evolution before, during, and after discontinuation demonstrates effects on the thyroid gland and peripheral thyroid hormone metabolism, with prolonged repercussions. Recognizing this pattern helps avoid misinterpretation of thyroid tests and inappropriate levothyroxine adjustments, particularly in patients at high cardiovascular risk.

Keywords: amiodarona-induced thyroid dysfunction; thyroid test discordance; post-thyroidectomy thyroid hormone metabolism.

THYROID

448 (3520) PHASE ANGLE AS A MARKER OF NUTRITIONAL STATUS IN A PATIENT WITH MUCINOUS ADENOCARCINOMA OF THE THYROID AFTER ONCOLOGIC TREATMENT

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Case report: A female patient initiated endocrinological follow-up in July/2024, with a history of obesity treated with gastric bypass in 2014, endoscopic revisional procedure in 2019, and semaglutide use. A thyroid nodule was identified on physical examination. After ultrasonography and fine-needle aspiration biopsy, cytology was classified as Bethesda VI. The patient underwent total thyroidectomy. Histopathological analysis revealed mucinous adenocarcinoma infiltrating the left thyroid lobe, with perithyroidal lymph node metastasis (1/4), extrathyroidal extension, and invasion of an adjacent venous wall. Immunohistochemistry confirmed mucinous-pattern carcinoma infiltrating thyroid tissue, skeletal muscle, connective tissue, and lymph nodes. PET scan did not identify a primary neoplasm in other sites, excluding metastatic origin. Subsequently, the patient underwent cervical lymph node dissection followed by chemotherapy and radiotherapy. In July/2024: weight of 69 kg, body mass index (BMI) of 23 kg/m², skeletal muscle mass (SMM) of 25.9 kg, and body fat percentage (BFP) of 30.8%, with no available PA measurement. After weekly cisplatin treatment at a dose of 40 mg/m², she developed significant anorexia. In February/2025: weight of 62.8 kg, SMM of 21.8 kg, BFP of 34.5%, and PA of 4.3. In May 2025, weight reached 60.5 kg, requiring chemotherapy interruption due to malnutrition, although PET scan demonstrated absence of active disease. During one year of follow-up, semiannual PET scans showed no recurrence. In May 2026, approximately one year after the last chemotherapy session, recovery of nutritional status was observed, weight of 68 kg, SMM of 25 kg, BFP of 32.6% and PA of 5.2. **Discussion:** Phase angle, obtained through bioelectrical impedance analysis, has been considered a prognostic and nutritional marker in oncology patients, with reduced values associated with loss of cellular integrity, sarcopenia, and worse clinical outcomes. Primary mucinous adenocarcinoma of the thyroid is an extremely rare neoplasm. In the present case, the reduction in PA was associated with significant deterioration in body composition and clinical malnutrition during chemotherapy, while subsequent PA improvement paralleled nutritional and SMM recovery after treatment interruption and disease remission. **Final comments:** PA proved to be a useful marker for nutritional monitoring, contributing to early identification of malnutrition and follow-up of clinical recovery.

Keywords: phase angle; thyroid mucinous adenocarcinoma; malnutrition.

THYROID

449 (3258) PREVALENCE AND CLINICAL PROFILE OF HYPOTHYROIDISM IN PATIENTS WITH DOWN SYNDROME: A RETROSPECTIVE COHORT STUDY

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Introduction: Down syndrome (DS) is the most prevalent chromosomal abnormality in the pediatric population and it is associated with a high predisposition to endocrine disorders. Among these, thyroid dysfunctions stand out as the most common endocrinopathy, with hypothyroidism being the most frequently observed condition. Given the essential role of thyroid hormones in neuropsychomotor development, regular screening is recommended from birth. **Objective:** To investigate the prevalence of hypothyroidism in patients with DS, evaluating its association with sex, age at onset, karyotype, and the presence of antithyroid autoantibodies. **Methods:** This retrospective cohort study was based on the review of medical records from 217 patients with DS followed in genetic-endocrine outpatient consultations at a university hospital. Variables collected included sex, karyotype, age at onset of thyroid dysfunction, type of thyroid disorder, and the presence of anti-thyroid peroxidase antibodies (TPOAb) and anti-thyroglobulin antibodies (TgAb). Statistical analysis was performed using SPSS version 24, adopting a significance level of $p < 0.05$. The study was approved by the Research Ethics Committee. **Results:** Among the 217 patients analyzed, 77 (35.5%) presented thyroid dysfunction, of which hypothyroidism was the predominant form, accounting for 87.2% of cases (67 patients). Sex distribution within the hypothyroidism group was balanced, with 33 females (49.3%) and 34 males (50.7%), with no statistically significant difference ($p = 0.507$). The highest incidence of diagnosis occurred between 0 and 9 years of age, comprising 71.7% of cases ($p = 0.04$). Regarding etiology, 79.3% of patients with hypothyroidism had negative serology for thyroid autoantibodies (TPOAb and TgAb), demonstrating a strong association between the absence of autoimmune markers and the diagnosis ($p = 0.001$). Regarding the genetic profile, free trisomy 21 karyotype was the most prevalent and was significantly associated with the development of hypothyroidism ($p = 0.01$). **Conclusion:** This study confirmed the high prevalence of hypothyroidism in DS, especially during early childhood. Most cases did not exhibit an initial autoimmune nature and showed a strong correlation with free trisomy 21 karyotype. These findings reinforce the need for systematic and early screening protocols, regardless of the presence of symptoms or autoantibodies.

Keywords: Down syndrome; hypothyroidism; trisomy 21.

THYROID

450 (3354) PROPYLTHIOURACIL-INDUCED HEPATOTOXICITY IN A PATIENT WITH GRAVES' DISEASE: A CASE REPORT

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Case presentation: A 39-year-old Haitian male with Graves' disease, diagnosed in October 2024, presented with anorexia, 17 kg weight loss over three months, choluria and right upper quadrant abdominal pain. He had been on PTU (800 mg/day) and propranolol (40 mg/day) for three months prior to hospital admission. On admission, the patient was tachycardic (118 bpm), with tremors and marked jaundice (++++). His Burch-Wartofsky score of 35 suggested thyroid storm. Laboratory tests showed severe hyperthyroidism (TSH < 0.005 µIU/mL, free T4 3.4 ng/dL), TRAb > 30 UI/L, elevated liver enzymes (AST 1536 U/L, ALT 1578 U/L), hyperbilirubinemia (24 mg/dL total bilirubin), and prolonged INR (1.56). Viral hepatitis serologies were negative. Abdominal CT revealed biliary sludge and non-obstructive nephrolithiasis. PTU-induced hepatotoxicity was suspected, prompting discontinuation of PTU and initiation of methimazole and increased propranolol doses. Autoimmune hepatitis was ruled out based on negative antibodies and a liver biopsy negative for autoimmune hepatitis. Despite medication adjustment, the patient showed no improvement after seven days, leading to radioactive iodine (RAI) therapy. Pre-RAI thyroid tests showed persistent hyperthyroidism (free T4 > 7.7 ng/dL). After receiving 20 mCi of I-131 for RAI treatment, follow-up at 50 days showed improved liver function (AST 93 U/L, ALT 92 U/L). However, the patient still had a TSH level < 0.005 µIU/mL and a significantly elevated free T4 level of 7.7 ng/dL. The patient was informed that further monitoring was necessary and that a repeat thyroid function test would be conducted in one month. It was explained that depending on the results, an additional dose of radioiodine might be required in the future, given the initially high TRAb titers. **Discussion:** PTU is an antithyroid drug commonly used in the management of Graves' disease. Although effective, PTU carries a risk of severe hepatotoxicity, but can manifest as acute liver failure. The primary treatment involves drug discontinuation, monitoring, and consideration of alternative definitive therapy. In this case, RAI was a successful approach to avoid further hepatotoxic risk. **Conclusion:** In patients with jaundice and elevated liver enzymes, PTU-induced hepatotoxicity should be considered in the differential diagnosis. Timely suspicion, coupled with early discontinuation of PTU and possible RAI therapy, are essential to prevent adverse hepatic outcomes.

Keywords: Graves' disease; hepatotoxicity; propylthiouracil.

THYROID

451 (3428) PULMONARY THYROID TISSUE VERSUS METASTATIC DISEASE AFTER BENIGN THYROID HISTOLOGY: A CASE REPORT

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Case presentation: A 63-year-old woman underwent partial thyroidectomy in 2011 for a calcified thyroid nodule. Histology showed nodular hyperplasia, without malignancy. In 2016, a right pulmonary nodule was resected and described as thyroid tissue in lung parenchyma, compatible with ectopia and without atypia. A 2017 pathology review showed a well-circumscribed thyroid tissue nodule, without atypia, necrosis, mitoses, or infiltration. 18F-FDG PET/CT showed no abnormal uptake in the pulmonary nodules. Chest computed tomography in 2019 showed bilateral nodules with interval growth, persistent in 2022, including a dominant middle-lobe lesion measuring 2.7 cm. In April 2025, a new pulmonary wedge resection identified thyroid tissue with follicular architecture in a pulmonary fragment. Because of this finding, completion thyroidectomy was indicated in May 2025; histology showed follicular hyperplasia/colloid goiter, without carcinoma in the examined left lobe. Serum thyroglobulin remained elevated compared with reference values, with negative antithyroglobulin antibodies. The patient awaits radioiodine therapy. **Discussion:** This case illustrates a rare diagnostic challenge: intrapulmonary thyroid tissue that may represent metastasis from well-differentiated thyroid carcinoma or ectopic thyroid tissue. The absence of malignancy in the evaluated thyroid specimens, the absence of atypia, necrosis, mitoses, or infiltration in the previous pulmonary pathology review, and the absence of abnormal uptake on PET/CT favor pulmonary ectopia. However, multifocality, a bilateral micronodular pattern, serial growth, and elevated thyroglobulin prevent exclusion of metastatic disease. Thyroglobulin elevation should be interpreted cautiously, as the lower value occurred with more suppressed thyroid-stimulating hormone. The 2025 American Thyroid Association guidelines emphasize integration of pathology, imaging, thyroglobulin, and risk. In this context, radioiodine therapy has a diagnostic and therapeutic role. **Final comments:** Thyroid tissue in the lung parenchyma, especially with multifocality, serial growth, or elevated thyroglobulin, requires careful clinicopathologic correlation. Long-term follow-up and complementary studies, such as pathology review and immunohistochemistry, may help distinguish pulmonary ectopia from metastatic disease, with direct impact on management.

Keywords: ectopic thyroid tissue; pulmonary nodules; thyroglobulin.

THYROID

452 (3424) RADIOIODINE (^{131}I) ADMINISTRATION VIA GASTROSTOMY TUBE FOR DIFFERENTIATED THYROID CANCER WITH TRACHEOESOPHAGEAL FISTULA

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Case presentation: A 45-year-old male patient, under investigation for dysphonia, underwent a neck CT scan showing a left thyroid lobe nodule and left pre-tracheal lymphadenopathy. After 5 years of follow-up, a new neck CT revealed significant growth with superior mediastinal extension, no cleavage plane with the lateral tracheal wall, and probable invasion. FNA confirmed papillary thyroid carcinoma (PTC) with bilateral node involvement. He underwent total thyroidectomy, neck dissection (levels II–VI), tracheal resection, esophageal shaving/repair, and tracheostomy. Pathology: multifocal PTC (largest focus 3.7 cm), extrathyroidal extension (skeletal muscle and tracheal mucosa invasion), pT4aN1bMx, high recurrence risk. Postoperatively, he developed hypoparathyroidism and a tracheoesophageal fistula, requiring a gastrostomy (G-tube). Radioiodine therapy (RAI) was indicated but delayed due to fistula-related pulmonary infections. Due to oral administration difficulties, 200 mCi of 131I was administered via G-tube after rhTSH stimulation (TSH 72,5 and Tg 77,8). Post-dose WBS suggested locoregional persistence (Tg 225/TSH 2,33). After 2 years, he shows an incomplete biochemical response (Tg: 35.53 ng/mL; suppressed TSH: 0.02 mUI/L on levothyroxine 2.7 mcg/kg/day). **Discussion:** The initial treatment for patients with locally advanced differentiated thyroid carcinoma (DTC) includes tumor resection and neck dissection (1). Depending on the extent of the surgery, the primary complications are hypoparathyroidism, nerve injury, hemorrhage, and tracheoesophageal fistula (2). While the benefits of adjuvant or therapeutic RAI in disease control are well-documented, the presence of a fistula can limit the oral administration of radioiodine (131I). There are few reports on 131I administration via gastrostomy tube (G-tube), making it a viable option for cases involving dysphagia, neurological changes, or gastrointestinal complications. Essential precautions include managing the risk of radioactive contamination, staff exposure, and potential dose loss (3). Furthermore, strict radioprotection is crucial due to residual contamination of the delivery devices (4). **Conclusion:** RAI administration via G-tube is a feasible alternative for DTC patients when oral delivery is impossible. Additionally, this case highlights the importance of careful planning and multidisciplinary discussion in managing advanced DTC.

Keywords: thyroid cancer; radioiodine; treatment complications.

THYROID

453 (3436) RARE MANIFESTATION: AUTOIMMUNE MYOCARDITIS SECONDARY TO GRAVES' DISEASE

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Case report: A 61-year-old woman with untreated hyperthyroidism presented with sudden retrosternal chest pain and palpitations. History included hypertension, type 2 diabetes, and sickle cell trait anemia. Examination revealed irregular tachycardia (113 bpm), BP 105 × 70 mmHg, and bilateral lower limb edema. Laboratory findings: TSH 0.007 µIU/mL, free T4 6.97 ng/dL, troponin 2.26 ng/mL, NT-proBNP 13,341 pg/mL, TRAb 8.07 IU/L. ECG showed high-rate atrial fibrillation with precordial T-wave inversion. Propylthiouracil 1,200 mg/day and propranolol 120 mg/day were initiated, achieving sinus rhythm restoration. Echocardiography demonstrated left atrial enlargement, preserved ejection fraction (77%), mild pulmonary hypertension (PASP 45 mmHg), and elevated filling pressures. Coronary angiography revealed no obstructive lesions, excluding ischemic etiology. Cardiac MRI showed discrete myocardial edema in the septal segments with linear mesocardial late gadolinium enhancement in the basal septum and inferior basal segment — consistent with non-ischemic myocardial fibrosis and acute inflammatory cardiomyopathy. **Discussion:** Autoimmune myocarditis in Graves' disease is a rarely reported entity, attributed to T-lymphocyte myocardial infiltration and autoantibody-mediated injury, independent of thyroid hormone hemodynamic effects. Cardiac MRI is the non-invasive method of choice for characterizing the inflammatory pattern, while coronary angiography is essential to rule out ischemic disease. **Conclusion:** This case highlights an uncommon and potentially life-threatening manifestation of Graves' disease, reinforcing the importance of including autoimmune myocarditis in the differential diagnosis of hyperthyroid patients presenting with acute coronary syndrome and normal coronary arteries. Early recognition is critical to guide appropriate therapy — including definitive thyroid ablation — and to prevent adverse cardiovascular outcomes.

Keywords: Graves' disease; autoimmune myocarditis; hyperthyroidism.

THYROID

454 (3458) REFRACTORY HYPOTHYROIDISM SECONDARY TO MALABSORPTION DUE TO ATROPHIC GASTRITIS AND LACTOSE INTOLERANCE AFTER FIVE YEARS OF INVESTIGATION: A CASE REPORT

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Case presentation: A 30-year-old woman was evaluated for refractory hypothyroidism while receiving sodium levothyroxine (LT4) at a dose of 14.63 mcg/kg. She underwent successive dose increases, changes in medication brand, combination therapy with oral triiodothyronine, and administration of LT4 via rectal suppository, without improvement. A sequential evaluation was performed, including counseling regarding proper medication administration, empirical antihelminthic therapy, psychiatric evaluation, upper gastrointestinal endoscopy with Helicobacter pylori testing and gastric biopsy, colonoscopy, investigation for atrophic gastritis, and lactose tolerance testing. All results were initially normal. An LT4 absorption test was subsequently performed, yielding positive results for malabsorption. Therefore, intravenous levothyroxine therapy was initiated, resulting in improvement of both clinical and laboratory parameters. Five years after the beginning of follow-up, the patient developed dyspeptic symptoms and was reevaluated, leading to the diagnosis of lactose intolerance and atrophic gastritis. **Discussion:** Although uncommon, refractory hypothyroidism represents a major clinical concern and should be investigated in a coherent manner. We describe a case of severe refractory hypothyroidism in a young patient who remained symptomatic for years. This case highlights how persistent investigation into the etiology of refractoriness should guide medical follow-up in a structured and preferably multidisciplinary manner. In this case, the levothyroxine absorption test played an essential role in establishing malabsorption and in guiding the transition from oral to intravenous therapy, although the definitive diagnosis was established much later. **Final considerations:** Cases of refractory hypothyroidism remain a clinical challenge. A structured, multidisciplinary, and long-term approach is recommended for all patients, including the use of the LT4 absorption test.
Keywords: hypothyroidism; lactose intolerance; atrophic gastritis.

THYROID

455 (3440) ROUTINE VERSUS SELECTIVE CALCIUM SUPPLEMENTATION AFTER TOTAL THYROIDECTOMY: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: Hypocalcemia is a common complication in patients undergoing total thyroidectomy, affecting approximately 40% of patients in the postoperative period. Two main strategies are used for prophylactic calcium replacement: routine supplementation, empirically initiated after surgery, and selective supplementation, guided by serum calcium and parathyroid hormone (PTH) levels or by clinical manifestations. Despite the widespread use of both approaches, the optimal strategy remains controversial, highlighting the need for further studies. **Objective:** To evaluate, through a systematic review and meta-analysis, the effectiveness of routine versus selective calcium supplementation after total thyroidectomy in improve clinical outcomes. **Methods:** This PRISMA-guided systematic review searched PubMed, Embase, and Cochrane Library following a PIC for randomized controlled trials (any language/year) comparing calcium with/or without vitamin D supplementation strategies in patients after total thyroidectomy. Title and abstract screening were performed independently by two reviewers, and data extraction were conducted by five reviewers. Statistical analyses were performed using R software. Studies with non-randomized designs or insufficient data were excluded. **Results:** A total of 4,879 studies were identified; 2,020 articles were screened, and 18 were included in the meta-analysis. Hypocalcemia episodes differed significantly between groups, with lower incidence among patients receiving prophylactic calcium supplementation ($p < 0.001$). The occurrence of paresthesia was significantly reduced in the prophylactic supplementation group compared to the selective approach ($p = 0.009$). Serum PTH levels showed a significant difference, favoring patients undergoing prophylactic therapy ($p = 0.036$). However, serum calcium levels showed no significant difference between the prophylactic supplementation group and the selective supplementation group, in which intervention occurred only during hypocalcemia episodes ($p = 0.155$). **Conclusion:** Prophylactic calcium supplementation after total thyroidectomy significantly reduces the incidence of hypocalcemia episodes and paresthesia symptoms while favorably impacting PTH levels, despite showing no significant effect on serum calcium levels compared to selective supplementation; supporting the use of routine prophylactic calcium supplementation as an effective strategy to prevent clinically significant post-thyroidectomy hypocalcemia.
Keywords: total thyroidectomy; hypocalcemia; calcium supplementation.

THYROID

456 (3338) SEVERE GRAVES' DISEASE WITH COMPRESSIVE OPTIC NEUROPATHY AND INCIDENTAL PAPILLARY THYROID MICROCARCINOMA: THERAPEUTIC AND MULTIDISCIPLINARY CHALLENGES

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Case presentation: A 65-year-old male smoker diagnosed with Graves' disease after a progressive course of palpitations, malaise, and inflammatory ocular involvement. He developed severe active Graves' orbitopathy characterized by bilateral exophthalmos, eyelid edema, marked conjunctival congestion, and ocular pain, associated with elevated TRAb levels (23.5 IU/L). Orbital magnetic resonance imaging demonstrated intraorbital fat edema and diffuse inflammatory orbital involvement. The severity of orbitopathy guided the initial therapeutic strategy, consisting of methimazole associated with methylprednisolone pulse therapy. Despite partial symptomatic improvement, the patient developed compressive optic neuropathy, requiring urgent bilateral orbital decompression surgery for visual preservation. After clinical stabilization, persistent active orbitopathy remained decisive in choosing total thyroidectomy as definitive treatment for hyperthyroidism, avoiding hormonal fluctuations and ocular worsening related to radioiodine therapy. Histopathological examination revealed a unifocal classic papillary microcarcinoma measuring 0.6 cm, without lymphovascular or extrathyroidal invasion. The patient also had persistent atrial fibrillation (CHA_2DS_2-VASc = 3). Six months after thyroidectomy, he developed ischemic stroke with hemorrhagic transformation, increasing the complexity of clinical management. **Discussion:** This case highlights how the activity and severity of Graves' orbitopathy may guide therapeutic decisions. Compressive optic neuropathy occurs in approximately 5% of patients with Graves' orbitopathy and constitutes an ophthalmologic emergency due to the risk of permanent visual loss. Surgical orbital decompression demonstrates high efficacy in visual preservation. The incidental finding of papillary microcarcinoma is also noteworthy, reinforcing that the coexistence of differentiated thyroid carcinoma and Graves' disease should not be underestimated. Three-year follow-up demonstrated excellent oncologic response, with undetectable thyroglobulin levels, absence of structural recurrence, and clinical stabilization of orbitopathy. **Final comments:** Severe active Graves' orbitopathy may be the main determinant of therapeutic strategy in Graves' hyperthyroidism. Early recognition of compressive optic neuropathy and timely surgical intervention were essential for visual preservation and clinical control of the case.

Keywords: hyperthyroidism; microcarcinoma; orbitopathy.

THYROID

457 (3078) - SEVERE HEART FAILURE WITH REDUCED EJECTION FRACTION RELATED TO GRAVES' DISEASE AND CARDIAC RECOVERY AFTER HORMONAL CONTROL

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Case presentation: Female patient, 27-year-old, family history of hyperthyroidism, developed typical symptoms of thyrotoxicosis in 2023, associated with significant weight loss (~40 kg), progressed with dyspnea and sought medical care two years after symptom onset. Transthoracic echocardiography revealed a left ventricular ejection fraction (LVEF) of 23%, consistent with heart failure with reduced ejection fraction (HFrEF). During hospitalization for etiological investigation, she was classified as NYHA functional class III, and evaluation suggested probable tachycardia-induced cardiomyopathy secondary to Graves' disease (TSH < 0.01 mIU/L, free T4 12 ng/dL, total T3 > 800 ng/dL, diffuse thyroid enlargement without nodules). Treatment with methimazole and guideline-directed heart failure therapy was initiated. After hormonal control, new echocardiography showed recovery of LVEF to 60%. The patient continues to experience recurrent Graves' disease exacerbations associated with worsening heart failure. Due to difficulty achieving sustained hormonal control, she is awaiting elective thyroidectomy to prevent further exacerbations. **Discussion:** The classic cardiac manifestation of hyperthyroidism is a hyperdynamic state with high-output heart failure caused by thyroid hormone-induced hyperadrenergic activity. In this case, however, thyrotoxicosis evolved into dilated cardiomyopathy with reduced ejection fraction, a rare and severe presentation of Graves' disease. The absence of structural heart disease and the marked improvement in LVEF after hormonal control support a causal relationship between thyrotoxicosis and cardiomyopathy. Although the mechanisms are not fully understood, prolonged exposure to excess T3 may lead to increased myocardial oxygen consumption, ventricular remodeling, tachycardia-induced cardiomyopathy, direct cardiomyocyte toxicity, and myocardial exhaustion, resulting in systolic dysfunction and low cardiac output. **Final comments:** Graves' disease should be considered a potentially reversible cause of heart failure in young patients with unclear etiology, particularly when systemic features of thyrotoxicosis are present. Although uncommon, hyperthyroidism-related dilated cardiomyopathy may cause significant hemodynamic compromise and even prompt consideration for heart transplantation. Recurrent exacerbations related to poor hormonal control highlight the need for definitive therapeutic strategies to prevent further cardiac decompensation.

Keywords: Graves' disease; heart failure with reduced ejection fraction; thyrotoxic cardiomyopathy.

THYROID

458 (3321) SEVERE HYPOTHYROIDISM ASSOCIATED WITH SECONDARY ADRENAL INSUFFICIENCY AND PITUITARY HYPERPLASIA: A CASE REPORT

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Case presentation: A 20-year-old woman, born in Manaus/AM and from Maués/AM, had been investigated for short stature since 2016 and was diagnosed with primary hypothyroidism in 2019. She used levothyroxine for only three months before discontinuing treatment. Over subsequent years, she progressively developed diffuse edema, dyspnea on minimal exertion, paroxysmal nocturnal dyspnea, palpitations, excessive somnolence, and abdominal pain. On admission, she presented marked short stature (1.33 m), mucocutaneous pallor, periorbital edema, and coarse skin. Laboratory evaluation demonstrated TSH 207.75 mIU/L, free T4 0.28 ng/dL, CPK 1810 U/L, prolactin 140 ng/mL, and low basal cortisol (3.9 µg/dL), suggesting severe hypothyroidism associated with secondary adrenal insufficiency. Pituitary MRI revealed globular enlargement of the adenohypophysis measuring 1.4 × 1.9 × 1.1 cm, compatible with secondary pituitary hyperplasia. Thyroid ultrasound demonstrated a reduced and heterogeneous gland. Corticosteroid therapy was initiated before gradual levothyroxine titration, resulting in progressive clinical improvement. During hospitalization, the patient developed transient hypotension and paroxysmal atrial fibrillation, both resolved during follow-up. **Discussion:** This case illustrates severe multisystem repercussions of chronic hypothyroidism, including anemia, hypothyroid myopathy, cardiovascular manifestations, and pituitary hyperplasia secondary to prolonged thyrotrophic stimulation. The coexistence of adrenal insufficiency has major clinical relevance because isolated levothyroxine initiation may precipitate adrenal crisis through increased cortisol metabolism. The marked growth impairment may be explained by prolonged thyroid hormone deficiency, which compromises skeletal maturation, GH secretion and sensitivity, and muscle protein synthesis, leading to delayed bone age and reduced growth velocity. Additionally, the patient's origin from a remote Amazon municipality highlights the impact of limited access to specialized endocrine care on delayed diagnosis and disease progression. **Final comments:** Severe chronic hypothyroidism may present with potentially life-threatening multisystem manifestations and mimic several clinical conditions. Investigation of associated adrenal insufficiency should precede thyroid hormone replacement in severe hypothyroidism. Early recognition and multidisciplinary management were essential for the favorable outcome.

Keywords: severe hypothyroidism; secondary adrenal insufficiency; hypothyroid myopathy.

THYROID

459 (3505) SEVERE/SIGHT-THREATENING AND CORTICOSTEROID-REFRACTORY THYROID EYE DISEASE: A REPORT OF TWO CASES TREATED WITH RITUXIMAB

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Thyroid eye disease (TED) is an extrathyroidal manifestation of Graves' disease (GD) and may be sight-threatening. Although intravenous glucocorticoids are first-line treatment, some patients may be refractory to corticosteroid therapy, with a risk of progression to visual loss, requiring second-line therapy, such as rituximab, as in the cases below: Case 1: A 38-year-old man with GD and active TED since 2024. Over one year, he received a cumulative dose of 12 g of methylprednisolone and 6 months of mycophenolate, with partial improvement. He subsequently developed visual loss and signs of optic nerve compromise, presenting with Uncorrected Visual Acuity (UCVA) in the Left Eye (LE) of 20/200, a Clinical Activity Score (CAS) of 3, and deuteranomaly on the Ishihara test. A further pulse therapy course with 3 g of methylprednisolone was administered, still resulting in only a partial response (UCVA LE 20/50), with persistent deuteranomaly; therefore, a single dose of rituximab was indicated. He was referred for 10 sessions of orbital radiotherapy. Three months later, he presented with UCVA LE of 20/20, a normal Ishihara test, and CAS 0. Case 2: A 34-year-old woman with GD and severe TED diagnosed in 2019, previously treated with 6 g of methylprednisolone. She was hospitalized in 2024 due to uncontrolled hyperthyroidism, with elevated free T4 and severe ophthalmopathy associated with reduced visual acuity. Despite normalization of free T4 during hospitalization, she progressed with right-sided visual loss, diplopia, dyschromatopsia, and CAS 3. She received an additional 3 g of methylprednisolone, totaling a cumulative dose of 9 g, with little clinical response, and also developed corticosteroid-related hallucinatory delusions, contraindicating further doses. Given the risk of permanent visual loss, rituximab combined with mycophenolate was indicated, in addition to total thyroidectomy. During outpatient follow-up, she presented with Best-Corrected Visual Acuity (BCVA) of 20/25, a normal Ishihara test, and CAS 0, despite persistent intermittent diplopia. Discussion: These cases illustrate the complexity of managing severe, sight-threatening TED in the setting of corticosteroid refractoriness. The use of rituximab in the context of corticosteroid-refractory disease was associated with disease inactivation in both patients, with recovery of visual acuity and color discrimination, although residual symptoms may persist, reinforcing its role in selected cases.

Keywords: thyroid eye disease; rituximab; corticosteroid-refractory disease.

THYROID

460 (3503) T3 EFFECT ON LIVER EXPRESSION OF LDLR AND PCSK9 IN CONTROL, HYPOTHYROID AND ALLOXAN-INDUCED DIABETIC RATS

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Previous studies from our group demonstrated that alloxan-induced diabetes mellitus in rats promotes hyperglycemia, increased serum cholesterol levels, and a hypothyroid state. The later may worsen glycemic control and contribute to cardiovascular complications, such as atherosclerosis and diastolic hypertension. T3 treatment in diabetic animals improved glycemia and restored serum cholesterol levels, highlighting the relevance of identifying genes, proteins, and regulatory pathways involved in T3-mediated modulation of lipid metabolism. Transcriptomic analysis by RNAseq was performed in the liver of non-diabetic control rats (CL), alloxan-induced diabetic rats (D), and D rats treated with T3 (DT3; 1.5 µg/100 g body weight, i.p.) for 28 days. The analysis revealed approximately 2,165 differentially expressed genes between D and CL and 1,709 between DT3 and D, many associated with lipid metabolism. Reduced LDLR expression was observed in D and restored in DT3. For PCSK9, transcriptomic data indicated reduced expression in D and increased expression in DT3. LDLR and PCSK9 are involved, respectively, in cholesterol uptake and LDLR degradation via receptor internalization. Based on these findings, gene (RT-qPCR) and protein (Western blotting) validation analyses were performed in liver samples stored at –80 °C. RT-qPCR confirmed a significant reduction of LDLR mRNA in D and restoration in DT3, a similar pattern was also observed for protein expression. For PCSK9, no significant difference in mRNA expression was observed between D and DT3, although T3 treatment reduced PCSK9 protein expression in D rats. Corroborating these findings, PCSK9 protein analysis in non-diabetic animals demonstrated a significant increase in hypothyroid rats induced by propylthiouracil in drinking water for 4 weeks (PTU) compared with euthyroid controls (C), whereas T3-supplemented animals (PT3 – 1.5 µg/100 g body weight, i.p), in a hyperthyroid state, exhibited markedly reduced PCSK9 levels. Together, these data suggest that diabetes-associated hypothyroidism contributes to reduced hepatic LDLR and increased PCSK9, leading to elevated serum cholesterol, whereas T3 restores these proteins even in the absence of insulin. These findings reinforce the central role of thyroid hormone in cholesterol metabolism regulation, independently of insulin action.

Keywords: triiodothyronine; cholesterol; diabetes mellitus.

THYROID

461 (2991) TARGETING CANCER STEM-LIKE CELLS IN ANAPLASTIC THYROID CARCINOMA: INVOLVEMENT OF JAK/STAT3 AND REPURPOSING OF NIFUROXAZIDE

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Introduction: Anaplastic thyroid carcinoma (ATC) is the rarest but most aggressive thyroid cancer, with a survival of 6–25 months due to rapid progression, resistance, and limited therapeutic options. Cancer stem-like cells (CSCs) represent a small subpopulation in the tumor microenvironment and have been related to self-renewal and transformation, enriched in aggressive tumors. Activation of JAK/STAT3 pathway has been correlated with ATC poor survival, as well as with the presence of CSCs. The antibiotic nifuroxazide (NFZ) was able to inhibit JAK/STAT3 pathway in other cancer models, remaining unexplored in ATC. **Objective:** To functionally characterize the CSCs profile in ATC and to evaluate the involvement of JAK/STAT3 pathway in stemness. **Methods:** ATC cell lines KTC-2, HTH83 and 8305c, the non-tumoral cell line Nthy-ori 3-1 and KTC-2 and HTH83 STAT3 knockout (KO) were used. Cell proliferation, cell cycle, viability, and colony formation were assessed. ALDEFLUOR was used for ALDH activity measurement (a CSCs biomarker) and for fluorescence-activated cell sorting (FACS) by flow cytometry. The expression of nine CSCs biomarkers was analyzed by real-time PCR or flow cytometry, and sphere formation assays were conducted. **Results:** Higher ALDH activity, increased expression of several CSCs biomarkers, including SOX2 and CD44, and enhanced sphere formation were observed in all ATC cells compared to Nthy-ori 3-1. Following FACS, ALDH+ KTC-2 and HTH83 cells showed enrichment of CSCs features, with increased expression of some CSCs biomarkers and higher capacity to form spheres. Functionally, ALDH inhibition with DEAB reduced proliferation, cell cycle progression, viability, and clonogenicity only in KTC-2. STAT3 KO reduced CSCs biomarkers expression and sphere formation only in HTH83 cells, concomitantly with the inhibition of the MAPK and PI3K pathways. In contrast, KTC-2 cells exhibited enhanced CSCs features, and MAPK and PI3K pathways activation. NFZ treatment reduced ALDH activity in parental and STAT3 KO cells, as well as the expression of some CSCs biomarkers, and sphere formation in parental cells. **Conclusion:** These results indicate that ATC cell lines display a CSCs profile and the JAK/STAT3 pathway participates in the stemness of HTH83 cell line. Also, NFZ can attenuate CSCs properties of all ATC cells, supporting the potential of combining CSCs-targeted approaches with JAK/STAT3 inhibition as a promising therapeutic strategy for ATC.

Keywords: anaplastic thyroid carcinoma; cancer stem-like cells; JAK/STAT3 pathway.

THYROID

462 (3327) TEMPORAL ANALYSIS OF THYROID MALIGNANCY INCIDENCE ACCORDING TO SEX ACROSS BRAZILIAN REGIONS (2015–2024)

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Introduction: Malignant thyroid neoplasms have increased worldwide over the past decade, possibly due to greater use of diagnostic methods and lifestyle changes. Higher incidence rates are consistently observed among females, suggesting hormonal and biological influences. In Brazil, regional inequalities in healthcare access and diagnostic capacity may affect disease detection, but national analyses evaluating temporal trends according to sex remain limited. This study aimed to analyze temporal trends in the incidence of malignant thyroid neoplasms according to sex across Brazilian regions between 2015 and 2024. **Methods:** This ecological time-series study used secondary data from the Datasus Oncology Panel, including cases of malignant thyroid neoplasms (ICD-10: C73) in Brazil between 2015 and 2024. Incidence rates per 100,000 inhabitants were calculated according to sex and region. Temporal trends were analyzed using Joinpoint regression, with estimation of the Annual Percent Change (APC) and Average Annual Percent Change (AAPC), adopting a 5% significance level. **Results:** A total of 53,407 cases of malignant thyroid neoplasms were recorded in Brazil between 2015 and 2024, with female predominance in all regions, ranging from 81.9% in the Central-West to 86.8% in the Northeast. Incidence rates remained consistently higher among females throughout the study period. In 2024, rates per 100,000 inhabitants were higher among women in all regions, with the lowest values in the North (3.87 in women; 0.47 in men) and the highest in the Northeast (7.03 in women; 1.29 in men). Joinpoint regression demonstrated increasing temporal trends across all sex-region strata, with inflection points between 2018 and 2019, followed by stabilization or slower growth in most regions. Females in the North region maintained a significant increase after 2018 (APC = 14.42%; 95% CI 7.70–22.27; p = 0.001). AAPCs were positive in all regions and consistently higher among females (62.2%–84.3%) than males (33.0%–48.4%). The highest trends were observed among females in the North and Central-West regions and among males in the Northeast. **Conclusion:** Malignant thyroid neoplasms showed increasing incidence in Brazil between 2015 and 2024, with higher rates among females and marked regional differences. These findings reinforce persistent sex-based and regional disparities and highlight the importance of strengthening surveillance, early diagnosis, and access to healthcare services.

Keywords: thyroid neoplasms; incidence; time series studies.

THYROID

463 (3055) TEMPORAL TRENDS AND CLINICAL OUTCOMES OF THYROTOXICOSIS IN BRAZIL (2020-2025): A HOSPITAL MORBIMORTALITY STUDY

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Thyrotoxicosis, in its severe form, leads to high-cost hospitalizations and a significant risk of death, serving as a sensitive indicator of the effectiveness of primary care. Analyzing its outcomes allows for the identification of not only the disease burden but also the fragility of specialized care networks and the impact of social determinants on Brazilian public health. The objective is to analyze the temporal trend of hospitalizations and the lethality rate of thyrotoxicosis in Brazil between 2020 and 2025, correlating regional disparities with the effectiveness of outpatient clinical management. This is an ecological time-series study with a quantitative and analytical approach. The PICO strategy was used to select hospitalizations for Thyrotoxicosis (ICD-10 E05) via the Hospital Information System (SIH/SUS) of the Datasus platform. Individuals diagnosed with Thyrotoxicosis (ICD-10) hospitalized in Brazil. (P), Hospital admissions and mortality rates by region and Federative Unit (I), Temporal evolution and disparities among the five Brazilian regions (C), Volume of hospitalizations, number of deaths, and identification of epidemiological hubs (O). Beyond absolute counts, relative growth rate calculations and case density analyses by region were applied to identify epidemiological hubs and variations in hospital lethality. A critical 80.4% increase in hospitalizations was observed during the period (rising from 491 in 2020 to 886 in 2025). This growth, particularly pronounced from 2021 onwards, suggests a residual effect of diagnostic backlog or the worsening of chronic conditions in the post-pandemic period. Although the Southeast concentrates the highest absolute volume, the Northeast showed the most significant acceleration in the number of hospitalizations, pointing toward a transition in detection capacity or a failure in the primary care containment barrier. Lethality remained at concerning levels in 2024 and 2025, suggesting that patients are reaching the hospital system at more advanced stages of the disease. The progressive increase in hospital morbidity from thyrotoxicosis reveals a gap in the longitudinal management of the disease. Data scrutiny indicates that the concentration of deaths and the growth of cases are not merely demographic reflections, but rather evidence of an urgent need for early diagnosis protocols and expanded access to endocrinology within the public health network, aiming to reduce avoidable hospital outcomes.

Keywords: thyrotoxicosis; morbidity; epidemiological surveillance.

THYROID

464 (2949) THE SYNTHETIC PRESERVATIVE METHYLPARABEN INFLUENCES THYROID HORMONE LEVELS BY MODULATING TSH-PKA SIGNALING WITHIN NTHY-ORI 3-1 CELLS

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Introduction: Methylparaben is a preservative frequently utilized in consumer products; however, recent research has raised concerns regarding its potential to interfere with endocrine health, particularly thyroid function. While existing literature confirms that methylparaben can alter thyroid hormone levels, the precise molecular pathways responsible for this disruption remain an active area of scientific inquiry. **Methods:** This study seeks to elucidate the specific involvement of the TSH-PKA signaling pathway in methylparaben-induced thyroid hormone disruption. Furthermore, we investigate the role of thyroid hormone receptors within this biological pathway by utilizing the Nthy-ori 3-1 human thyroid follicular epithelial cell line as a model. To conduct this analysis, cells were exposed to varying concentrations of methylparaben (0, 1, 3, 5, and 10 µM) over periods of 48 and 72 h, following a targeted screening process designed to isolate this compound from a broader group of suspected endocrine-disrupting molecules. **Results:** Research indicates that following a 72-hour exposure to Methylparaben, there is a notable upregulation in the mRNA expression of key thyroid markers specifically thyroid-stimulating hormone receptors, thyroid peroxidase, paired box 8, and thyroid transcriptional factor-1. This process appears to be mediated by the TSH/PKA signaling pathway under conditions of excessive TSH stimulation. **Discussion:** Building upon previous in vivo evidence, which demonstrated that Methylparaben reduces glutathione levels and inhibits the expression of glutathione peroxidase thereby triggering an increase in reactive oxygen species, this study elucidates a new mechanism by which Methylparaben influences cellular signaling. By identifying these synergistic effects, we gain insight into how such chemical exposures may disrupt endocrine function and potentially contribute to tumorigenesis. Ultimately, these findings offer critical data that can refine future human health risk assessments regarding the impact of Methylparaben on thyroid integrity
Keywords: methyparaben; hormone; follicular.

THYROID

465 (3342) THYROID EYE DISEASE AFTER RADIOACTIVE IODINE THERAPY FOR INVASIVE PAPILLARY CARCINOMA IN A THYROGLOSSAL DUCT CYST

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Case presentation: A 72-year-old man underwent surgical resection of a thyroglossal duct cyst in May 2024, with histopathological analysis revealing invasive papillary thyroid carcinoma. In October 2025, he underwent total thyroidectomy with left lateral and pretracheal neck dissection, with lymph nodes free of metastases. On October 29, he received a therapeutic dose of radioactive iodine (RAI) therapy (100 mCi). On November 1, he was admitted with binocular diplopia, right-sided ptosis, and impaired extraocular motility of the right eye. At that time, TRAb and anti-thyroglobulin antibodies were negative. Orbital magnetic resonance imaging demonstrated bilateral proptosis and gadolinium enhancement in the orbital fat planes and muscle bellies of the superior, medial, and inferior rectus muscles of the right eye. On November 7, intravenous methylprednisolone pulse therapy was initiated (7.5 g over 12 weeks), resulting in improvement of ocular manifestations. At follow-up on April 29, 2026, mild ptosis persisted. He was a former smoker with a 90 pack-year smoking history, having quit 16 years earlier. **Discussion:** This case describes thyroid eye disease (TED) following thyroidectomy and RAI therapy in a patient without previous autoimmune thyroid disease, in the context of papillary carcinoma arising in a thyroglossal duct cyst, a rare condition with no similar cases reported in the literature. Although the majority of TED cases following RAI occur in patients with Graves' disease, there are reports of de novo orbitopathy, which is potentially related to the induction of autoimmunity driven by thyroid tissue destruction following RAI administration. Despite this, the patient had negative TRAb levels, a finding previously described in seronegative TED cases. The presence of residual thyroid tissue may have contributed to disease onset. Differential diagnoses were excluded, and the patient's significant smoking history, a recognized risk factor for TED, as well as the favorable response to intravenous methylprednisolone pulse therapy, are noteworthy. **Final comments:** Further studies are needed to better characterize TED following RAI therapy in order to improve the safety of RAI prescription and to evaluate potential prophylactic measures against TED.
Keywords: thyroid cancer; radioiodine; thyroid eye disease.

THYROID

466 (3120) THYROID FUNCTION IN DOGS WITH NEWLY DIAGNOSED INSULIN-DEFICIENT DIABETES MELLITUS BEFORE INSULIN THERAPY: PRELIMINARY EVIDENCE CONSISTENT WITH EUTHYROID SICK SYNDROME

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Thyroid hormones play a central role in energy metabolism and endocrine homeostasis, yet the thyroid status of dogs at the time of diagnosis of insulin-deficient diabetes mellitus (IDDM) remains poorly characterized. Experimental studies previously conducted in murine models in our laboratory demonstrated reduced thyroid activity associated with IDDM, as well as improved glycemic control following triiodothyronine administration, supporting the hypothesis of interaction between glucose metabolism and the hypothalamic-pituitary-thyroid (HPT) axis. Therefore, this study aimed to investigate thyroid function in dogs with newly diagnosed IDDM prior to insulin therapy, contributing to the endocrine characterization of these patients and evaluating whether early alterations compatible with euthyroid sick syndrome are present. Twenty-two dogs were evaluated, including 10 healthy animals (control group) and 12 dogs with newly diagnosed IDDM (IDDM group), all before insulin treatment initiation. Complete blood count, serum biochemical profile, and urinalysis were performed as part of the initial clinical evaluation. Thyroid function was assessed by measuring serum thyroid-stimulating hormone (TSH), total thyroxine (TT4), and free thyroxine by equilibrium dialysis (fT4-ED). Dogs in the IDDM group exhibited metabolic alterations consistent with diabetes, including marked hyperglycemia ($p < 0.0001$), hypertriglyceridemia ($p = 0.0384$), increased alanine aminotransferase ($p = 0.0277$) and alkaline phosphatase activities ($p = 0.0214$), and elevated beta-hydroxybutyrate concentrations ($p < 0.005$). Urinalysis revealed significant glucosuria ($p = 0.0007$), ketonuria, and proteinuria ($p < 0.05$). No significant differences were observed between groups for sodium, potassium, albumin, or total cholesterol concentrations. Regarding thyroid function, dogs with IDDM showed a trend toward lower TSH and fT4-ED concentrations compared with controls, whereas TT4 concentrations remained within expected physiological ranges. These findings suggest that the diabetic metabolic state may influence early regulation of the HPT axis before insulin therapy, supporting the hypothesis that dogs with newly diagnosed IDDM may exhibit alterations compatible with euthyroid sick syndrome. Furthermore, spontaneous canine diabetes may represent a relevant translational model for investigating interactions between glucose metabolism and thyroid physiology.

Keywords: thyroid hormones; insulin-deficient diabetes mellitus; spontaneous canine diabetes.

THYROID

467 (2929) THYROID STORM MIMICKING DISSEMINATED NEOPLASTIC SYNDROME WITH REVERSIBLE CARDIOVASCULAR DYSFUNCTION

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Case presentation: A 40-year-old woman was admitted with progressive dyspnea, anasarca, and significant weight loss. Physical examination revealed atrial fibrillation with rapid ventricular response, jugular venous distention, and marked ascites, consistent with right-sided heart failure. BNP was 792 pg/mL. Given the association with constitutional symptoms, multistation lymphadenopathy, and elevated CA-125 (619 U/mL), systemic malignancy with paraneoplastic manifestations was initially suspected. Further investigation revealed TSH < 0.01 mIU/L, free T4 4.21 ng/dL, and TRAb 19 IU/L, confirming autoimmune thyrotoxicosis due to Graves' disease. A Burch-Wartofsky score of 40 supported the diagnosis of thyroid storm. Transthoracic echocardiography demonstrated marked right ventricular dilation (56 mm) and severe functional tricuspid regurgitation without structural valvular abnormalities. Treatment with high-dose methimazole, propranolol, and intensive volume management was initiated. After 12 days, the patient showed resolution of systemic congestion and regression of tricuspid regurgitation to mild severity. **Discussion:** Thyroid storm is an endocrine emergency characterized by adrenergic hyperstimulation and a hyperdynamic circulatory state. Although cardiovascular manifestations are common, presentations dominated by right-sided heart failure and significant functional valvular regurgitation are uncommon and may delay diagnosis. In this case, excess thyroid hormone induced marked hemodynamic overload of the right heart chambers, resulting in tricuspid annular dilation and functional regurgitation witch resolved after control of thyrotoxicosis. Concomitant lymphadenopathy and elevated CA-125 further complicated the diagnostic evaluation and were interpreted as reactive manifestations of thyroid autoimmunity and systemic venous congestion, respectively. **Final comments:** Thyroid storm should be considered in the differential diagnosis of acute cardiovascular decompensation associated with constitutional symptoms, even in the presence of findings suggestive of malignancy. Early recognition of atypical systemic manifestations of thyrotoxicosis is essential to avoid unnecessary investigation and to identify potentially reversible complications.

Keywords: thyroid storm; Graves disease; thyrotoxic cardiomyopathy.

THYROID

468 (3496) THYROTOXIC DISORDERS DURING PREGNANCY: FETAL AND NEONATAL OUTCOMES AND PLACENTAL HISTOPATHOLOGICAL ANALYSIS

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Introduction: Maternal and fetal exposure to excess thyroid hormone during pregnancy may be associated with clinical and perinatal complications, influenced by the severity and duration of thyrotoxicosis and by treatment; in Graves' disease (GD), an additional autoimmune component may also contribute. **Objective:** To compare the clinical and hormonal profile, as well as fetal, neonatal, and placental outcomes, according to the type of thyrotoxic dysfunction (GD, gestational transient thyrotoxicosis [GTT], and subclinical hyperthyroidism [SCH]). **Methods:** This was a retrospective longitudinal descriptive and analytical study based on medical record review. A total of 68 pregnant women (80 newborns, including five twin pregnancies) were included and allocated to the GD, GTT, and SCH groups. Collected data included trimester-specific TSH and FT4 levels, TSH receptor antibodies (TRAb), ultrasound findings, anthropometric parameters, and neonatal outcomes. Placentas submitted to histopathological examination were classified according to maternal, fetal, or mixed malperfusion patterns, as well as specific lesions. **Results:** The GD group showed higher FT4 levels during the second and third trimesters. Estimated fetal weight percentile on ultrasound differed among groups; however, birth weight and birth-weight adequacy did not differ significantly. Rates of neonatal intensive care unit admission, respiratory distress, pathological jaundice, thyroid dysfunction, and neonatal death were similar across groups. Placental histopathology also showed no significant between-group differences regarding malperfusion patterns or other lesions. **Conclusion:** Despite greater hormonal exposure in GD, no measurable increase in adverse fetal, neonatal, or placental outcomes was observed compared with GTT and SCH. These findings suggest that elevated FT4 levels alone may not fully explain the associated risks, reinforcing the potential role of factors such as biochemical control, treatment, and timing of exposure.
Keywords: Graves' disease; gestational transient thyrotoxicosis; pregnancy outcomes.

THYROID

469 (3583) THYROTOXIC PERIODIC PARALYSIS: CLINICAL AND MOLECULAR INSIGHTS FROM A 30-YEAR BRAZILIAN COHORT

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Introduction: Thyrotoxic periodic paralysis (TPP) is an endocrine emergency characterized by acute episodes of muscle weakness associated with hypokalemia in the setting of thyrotoxicosis. Although classically described in Asian populations, TPP has been increasingly recognized in ethnically diverse Western cohorts. During attacks, prompt and cautious potassium replacement is essential to avoid complications, while definitive treatment of thyrotoxicosis prevents recurrence. Genetic susceptibility has been implicated, particularly variants in the KCNJ18 gene encoding the Kir2.6 potassium channel. **Objective:** To describe the clinical, demographic, and molecular characteristics of a large Brazilian cohort with TPP followed over three decades and to evaluate the prevalence of pathogenic variants in KCNJ18. **Methods:** We retrospectively evaluated patients diagnosed with TPP at our tertiary University Hospital, beginning with the first case identified in 1994, as well as patients referred for molecular investigation. Clinical and laboratory data were reviewed. Germline analysis of KCNJ18 (Kir2.6) variants was performed by Sanger sequencing. **Results:** Seventy-three patients were included, of whom 71 (97.2%) were male. Ethnic backgrounds included 7 Asian, 22 White, 5 Black, 28 mixed Black and White ancestry, and 1 Indigenous patient. Graves' disease was the main cause of thyrotoxicosis (66/69 cases, 95.6%). Importantly, 49/66 patients (74.2%) initially presented with paralysis and were unaware of their thyroid disease. Hypokalemia during attacks was documented in most cases, with serum potassium levels as low as 1.2 mEq/L. More than 85% of patients had predominantly nocturnal attacks, with episode duration ranging from 2 to 24 hours, and over half reported triggers such as carbohydrate-rich meals or intense physical activity. No recurrent TPP episodes occurred after treatment of thyrotoxicosis. KCNJ18 variants were identified in 8/67 tested patients (11.9%), including Gln407Stp, Thr354Met, Arg399Stp, Arg386Cys, Asn252Asp, and Phe263Leu. **Conclusion:** TPP remains an underrecognized cause of hypokalemic paralysis, and thyroid function assessment is essential in patients presenting with acute weakness and hypokalemia. In this Brazilian cohort, most patients were unaware of their underlying thyrotoxicosis at presentation, highlighting the diagnostic challenge of the disease. Early recognition and treatment are critical to prevent severe complications and recurrence.
Keywords: thyrotoxic periodic paralysis; thyrotoxicosis; paralysis.

THYROID

470 (2946) UNUSUAL INITIAL MANIFESTATION AND UNFAVORABLE EVOLUTION OF DIFFERENTIATED THYROID CARCINOMA IN A THYROGLOSSAL DUCT CYST: A CHALLENGING CASE REPORT

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Case presentation: A 33-year-old woman with a family history of thyroid cancer presented with a progressively growing submental nodule, leading to the suspicion of a thyroglossal duct cyst, with an indication for excision. Histopathological examination revealed classic papillary thyroid carcinoma (PTC) originating from the aforementioned cyst, measuring 1 cm, without invasion, with unaffected margins and adjacent structures, and one compromised lymph node (2 mm). Total thyroidectomy was then indicated, the sample of which showed no neoplasia, including resected lymph nodes. The patient received radioiodine therapy and, at that time, presented with stimulated thyroglobulin (TG) of 19.4 ng/mL. Subsequently, a suspicious lymph node was presented, which underwent aspiration, the cytology of which revealed cellular clusters of thyroid origin, with positive expression for thyroid markers by immunohistochemistry. In a new cervical lymph node dissection, metastasis of PTC was found, with a subsequent drop in serum TG levels, but without achieving an excellent response. Currently, a slow and gradual increase in TG is observed, with an incomplete biochemical response, and the presence of structural lesions is under investigation. **Discussion:** PTC in thyroglossal duct cysts is a rare finding, occurring in 1%–2% of these cysts. In general, this neoplasm has an excellent prognosis, with high overall survival and low recurrence rates. The Sistrunk procedure is the standard initial approach, with thyroidectomy in the presence of risk factors such as metastatic involvement and significant nodularity, and synchronous carcinomas may be present. When there is lymph node involvement, central compartment intervention is also recommended. Radioiodine therapy can be used for ablation of higher-risk remnants. In the case presented, we note an unusually located PTC, without thyroid affection, but with lymph node involvement. The patient presented an unusual evolution, with persistent disease in lymph nodes, even after rigorous initial treatment, maintaining an incomplete biochemical response despite a new lymph node resection. **Final comments:** The description of unusual presentations and evolutions of PTCs reinforces the heterogeneity of the disease, the possibility of which should always be considered in unusual presentations, in order to avoid diagnostic delays and therapeutic inertia in cases where these may be relevant to the evolution.
Keywords: ectopic thyroid carcinoma; differentiated thyroid carcinoma; thyroglossal duct cyst.



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