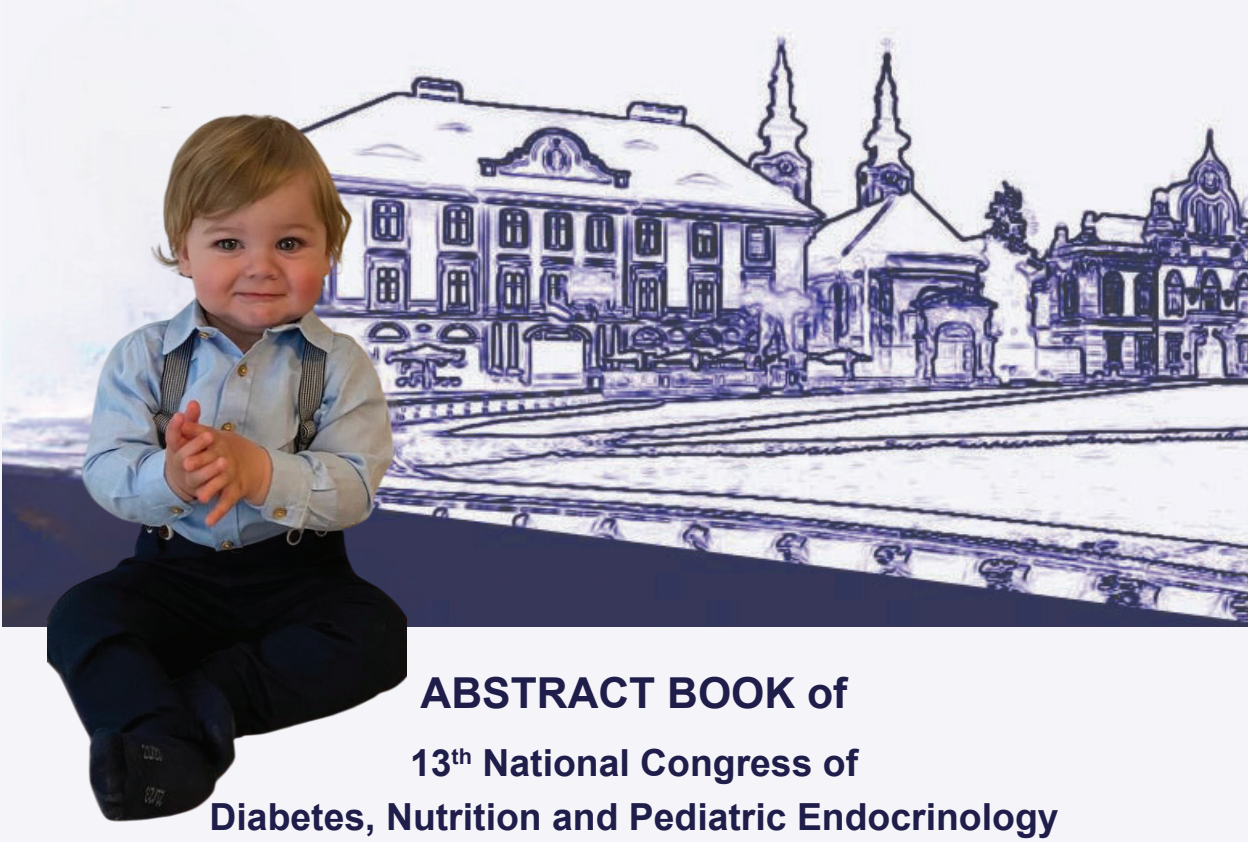

ABSTRACT BOOK of

13th National Congress of Diabetes, Nutrition and Pediatric Endocrinology

– with international participation –

April 22 – 25, 2026, TIMIȘOARA, ROMANIA

VOLUM DE REZUMATE

Al 13-lea Congres Național de Diabet, Nutriție și Endocrinologie Pediatrică

– cu participare internațională –

22 – 25 Aprilie, 2026, TIMIȘOARA, ROMANIA

EDITURA MIRTON

ROMÂNIA

OSIM

OFICIUL DE STAT PENTRU INVENȚII ȘI MĂRCI

Certificat de înregistrare a mărcii

Nr. 200614

Acordat în temeiul Legii nr 84/1998 privind mărcile și indicațiile geografice (republicată), pentru marca:

ENDOPED

SOCIETATEA ROMÂNĂ DE DIABET NUTRIȚIE ȘI ENDOCRINOLOGIE PEDIATRICĂ

Durata de protecție a mărcii este de zece ani cu începere de la data de **07.11.2023**, cu posibilitatea de reînnoire.

Marca este înregistrată pentru clasele de produse/servicii înscrise în fila din Registrul Mărcilor.

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OFICIUL DE STAT PENTRU INVENȚII ȘI MĂRCI • ROMÂNIA •

Tipărit la ...

ABSTRACT BOOK of
The 13th National Congress of
Diabetes, Nutrition and Pediatric Endocrinology
- with international participation -
April 22 – 25, 2026, TIMIȘOARA, ROMANIA

VOLUM DE REZUMATE
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Diabet, Nutriție și Endocrinologie pediatrică
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Stimați colegi, dragi prieteni

Avem onoarea de a vă invita la cea de-a 13-a ediție a Congresului Național ENDOPED®, eveniment de referință care va reuni comunitatea de diabet, nutriție și endocrinologie pediatrică la Timișoara, între 22 și 25 aprilie 2026.

Într-o eră a schimbărilor tehnologice accelerate, medicina pediatrică evoluează constant. Ediția 2026 a congresului nostru își propune să fie un hub de inovație, punând accent pe noile protocoale terapeutice, integrarea inteligenței artificiale în monitorizarea diabetului zaharat și abordările multidisciplinare în bolile endocrine.

Evenimentul va reuni experți de renume național și internațional, oferind o platformă de dialog esențială pentru schimbul de bune practici și prezentarea celor mai recente cercetări din domeniu.

Obiectivul nostru comun rămâne același: îmbunătățirea calității vieții micilor noștri pacienți prin excelență clinică și empatie.

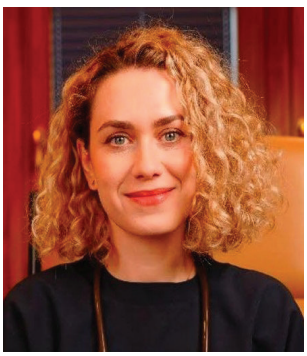
Vă încurajăm să vă alăturați acestei comunități de profesioniști pentru a împărtăși experiențe, a dezbate cazuri clinice complexe și a consolida parteneriatele profesionale care ne definesc.

Așteptăm cu nerăbdare să vă primim din nou în orașul istoric și fermecător care este Timișoara.

Cu prietenie și respect,

Prof. dr. Iulian Velea
Președinte „ENDOPED

Prof. dr. Corina Paul
Președinte CONGRES



As. univ. dr. Simina Mihuța
Secretar Științific

Dear colleagues, dear friends

We have the honor to invite you to the 13th edition of the ENDOPED® National Congress, a landmark event that will bring together the diabetes, nutrition and pediatric endocrinology community in Timișoara, between April 22 and 25, 2026.

In an era of accelerated technological change, pediatric medicine is constantly evolving. The 2026 edition of our congress aims to be an innovation hub, emphasizing new therapeutic protocols, the integration of artificial intelligence in diabetes monitoring and multidisciplinary approaches in endocrine diseases.

The event will bring together nationally and internationally renowned experts, providing an essential dialogue platform for the exchange of best practices and the presentation of the latest research in the field.

Our common objective remains the same: improving the quality of life of our young patients through clinical excellence and empathy.

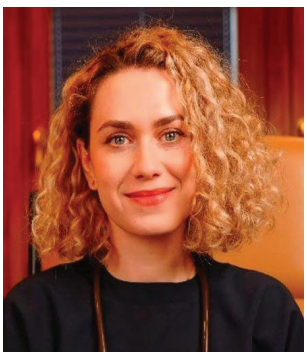
We encourage you to join this community of professionals to share experiences, discuss complex clinical cases, and strengthen the professional partnerships that define us.

We look forward to welcoming you again to the historic and charming city of Timișoara.

With friendship and respect,

Iulian Velea MD, PhD
President of „ENDOPED"

Corina Paul MD, PhD
CONGRESS President



Simina Mihuța MD, PhD
Scientific Secretary

CONTENTS / CUPRINS

INFORMAȚII GENERALE

Locul de desfășurare
”*Timișoara Convention Center*”
Str. Mărășești 1-3, Timișoara

Website-ul oficial:
www. https://endoped.ro/evenimente-viitoare/

Limba oficială: limba română și limba engleză

Acreditarea științifică:
Acest eveniment este creditat EMC de Colegiul Medicilor din România, precum și de de Colegiul Dieteticienilor și Nutriționiștilor din România.

Management congres: Event-Consulitng https://event-consulting.ro/contact/
Cristina Negrean: cristina.negrean@event-consulting.ro

Secretariatul congresului amplasat la etajul 1 al centrului de conferințe ”*Timișoara Convention Center*” va funcționa după următorul program:

- Miercuri 22 aprilie: 12.00 – 19.30
- Joi 23 aprilie și Vineri 24 aprilie: 08.15 – 18.00
- Sâmbătă 25 aprilie: 08.30 – 12.30

ECUSONUL
Se eliberează la scretariatul congresului pentru toti participanții înscriși.
Accesul in sălile congresului se va face pe baza ecusonului.

CERTIFICATELE DE PARTICIPARE
Se eliberează electronic, în termen de maxim 30 de zile de la finalizarea evenimentului pe adresa de e-mail furnizată de participant la înscriere.

TELEFOANELE MOBILE
Se vor pune pe mod silențios în sala de conferințe.

INSTRUCȚIUNI PENTRU COMUNICATORI
Detaliile au fost transmise prin email de către colegii noștri din comitetul de organizare. Prezentările pot fi predate și la secretariat cu cel puțin 24 ore înainte de prezentare.
Comunicatorii sunt rugați respectuos să se încadreze în timpul alocat prezentarii. În caz contrar moderatorii vor fi nevoiți să întrerupă prezentarea.
În sala în care se vor derula lucrările congresului va fi afișat un timer (cronometru) cu timpul rămas pînă la finalul prezentării.

E-postere (Posterele electronice) vor rula pe toată durata congresului pe ecrane multimedia amplasate în holul secretariatului.

EXPO MEDICA - Va funcționa pe toată durata congresului

GENERAL INFORMATION

VENUE
”TIMIȘOARA CONVENTION CENTER”
1-3, Mărășești str, TIMIȘOARA

Official website:
www. https://endoped.ro/evenimente-viitoare/

Official language: Romanian and English

Scientific accreditation:
This event is accredited by the Romanian College of Physicians, as well as the Romanian College of Dietitians and Nutritionists.

Congress management: Event-Consulitng https://event-consulting.ro/contact/
Cristina Negrean: cristina.negrean@event-consulting.ro

The congress secretariat located on the 1st floor of the „Timișoara Convention Center” will operate according to the following schedule:

- Wednesday, April 22: 12.30 – 19.30
- Thursday, April 23 and Friday, April 24: 08.15 – 18.00
- Saturday, April 25: 08.00 – 12.30

THE BADGE
Issued at the congress secretariat for all registered participants.
Access to the congress rooms will be based on the badge.

PARTICIPATION CERTIFICATES
Issued electronically, within a maximum of 30 days from the completion of the event, to the email address provided by the participant upon registration.

MOBILE PHONES
Will be put on silent in the conference room.

INSTRUCTIONS FOR COMMUNICATORS
The details have been sent by email by our colleagues from the organizing committee. Presentations can also be submitted to the secretariat at least 24 hours before the presentation. Communicators are respectfully asked to adhere to the time allotted for the presentation. Otherwise, the moderators will have to interrupt the presentation.
A timer will be displayed in the room where the congress will take place with the time remaining until the end of the presentation.

E-posters (Electronic Posters) will run throughout the congress on multimedia screens located in the secretariat lobby.

EXPO MEDICA - Will operate throughout the congress.

STEERING COMMITTEE

Romanian Society of Diabetes Nutrition and Pediatric Endocrinology

President
Iulian Velea

Vice President
Corina Paul
Diana Miclea

Scientific Secretary
Monica Simina Mihuța

Members:
Alina Grama
Cristina Mihai
Carmen Oltean

Treasurer
Anișoara Răduțu

SCIENTIFIC COMMITTEE OF THE CONGRESS

Dan Alexa (*Timișoara / Romania*)
Dana Anton – Păduraru *(Iași / Romania)*
Corin Badiu *(Bucharest / Romania)*
Carmen Barbu *(Bucharest / Romania)*
Simona Bogdan *(Timișoara / Romania)*
Joanne Claire Blair *(Liverpool / UK)*
Adela Chiriță-Emandi *(Timișoara / Romania)*
Alex R. Constantinescu *(Hollywood/USA)*
Anda Dumitrașcu *(Bucharest / Romania)*
Viviana Elian *(Bucharest / Romania)*
Claudia Jurca *(Oradea/Romania)*
Diana Miclea *(Cluj Napoca/Romania)*
Cristina Mihai *(Constanța / Romania)*
Nastascia di IOrgi *(Genoa / Italy)*
Philippe Lysy (*Bruxelles / Belgium*)
Gheorghe Mihalaș *(Timișoara / Romania)*
Cristian Minulescu *(Bucharest / Romania)*
Simina Mihuța *(Timișoara / Romania)*
Carmen Oltean *(Iași / Romania)*
Corina Paul *(Timișoara / Romania)*
Raluca Pop *(Tg. Mures / Romana)*
Cristina Slăvescu *(Cluj-Napoca Romania)*
Dana Stoian *(Timișoara / Romania)*
Bogdan Timar *(Timișoara / Romania)*
Christina Ungureanu *(Iași / Romania)*
Iulian Velea *(Timișoara / Romania)*
Adrian Vlad *(Timișoara / Romania)*
Mihaela Vlad *(Timișoara / Romania)*
Mihaela Vlăiculescu *(Bucharest Romania)*
Simone von Sengbusch (*Lübeck / Germany*)

INVITED SPEAKERS

Joanne Clare BLAIR

Honorary Professor, Institute of Translational Medicine, University of Liverpool, UK.
Consultant Endocrinologist, Alder Hey Children's NHS Foundation Trust
April 2021 to present: Professional Co-Lead, Health and Care Across the Life Course Theme, National Institute of Health Research, Applied Research Collaboration, North West Coast.
May 2021 to present: Member, Steering Committee, Winter School, European Society for Pediatric Endocrinology.
September 2022 to present: Co-Director, NIHR Clinical Research Facility, Alder Hey Children's Hospital.
She practice in all areas of pediatric endocrinology and have a special interest in neuroendocrinology, adrenal insufficiency and inherited endocrine tumors.
She jointly chaired the Genetic Endocrine Tumors theme of the Endocrine European Reference Network (Endo-ERN) from its inception to the time at which the United Kingdom left the European Union in 2021. Work in general and specialist outpatient clinics, including oncology late effects, gynaecology, inflammatory bowel disease and outreach clinics in North Wales. She care inpatients and give expert opinions for patients with endocrine morbidity in a range of specialties.
Throughout the career, she have taught to students at all levels of higher education including medical students, doctors in training and those studying for a higher degree (PhD, MD, MPhil and MRes).
She published many aspects of pediatric endocrinology in peer reviewed journals and text books.
She have led or been a co-applicant on successful applications for research funding totaling more than £15M. This funding has supported research in the fields of childhood diabetes, adrenal insufficiency, asthma and pharmacogenomics, and research infrastructure.

Alex. R. CONSTANTINESCU

Memorial Healthcare System/Joe DiMaggio Children's Hospital, United States of America

University Appointment:

- Clinical Professor, Affiliated, Pediatrics – College of Allopathic Medicine, NSU, Ft. Lauderdale, FL – 2017-Present
- Clinical Professor of Pediatrics – Nova Southeastern University, Ft. Lauderdale, FL – April 2011 – Present
- Clinical Affiliate Professor, Integrated Medical Sciences – Charles E. Schmidt College of Medicine, FAU, Boca Raton, FL – August 2021 – Present

Hospital Appointments:

- Chief, Pediatric Nephrology and Hypertension – Joe DiMaggio Children's Hospital (JDCH), Hollywood, FL – 1/2003 – present
- Medical Director, Pediatric Dialysis Unit JDCH – 2004 - present
- Chair, Pediatric Clinical Pathways Governance Board – Jan 2020 - present
- Chair, Department of Medicine, Joe DiMaggio Children's Hospital – 2021– 2025

Research Activities and Grant Support: Over 20 research projects/grants.

Society Memberships:

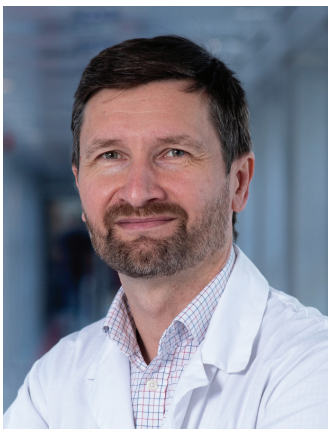
- International Pediatric Nephrology Association - 1992 – present
- American Society of Nephrology - 1993 – present
- American Society of Pediatric Nephrology - 1996 – present
- American Society of Hypertension - 1999 – present
- Member Scientific Council of Romanian Journal of Pediatric Nephrology and Urology – 2002 – 2005
- Florida Society of Nephrology – 2005 – 2024
- American Board of Quality Assurance and Utilization Review Physicians (ABQAURP) – 2019 – 2024

Awards and Honors:

- Young Investigator's Award – NKF of NY/NJ – 1998;
- Listed in "Best Doctors in America" (since 1998), "Top Pediatricians", "Florida Super Doctors", "Leading Physicians of the World", Top Pediatrician in Gold Coast Magazine, FL Top Doc;
- Excellence in Leadership – Nov 2014 – MHS Physician Leadership Development Program – 2013-2014;
- FAU 2016 Outstanding Clinical Teacher in a Subspecialty – February 7, 2017;
- "Spirit of Healing" Award – Memorial and JDCH Foundations – May 17, 2019;

Natascia Di IORGI

Natascia Di Iorgi has been Associate Professor of Pediatrics at the University of Genoa, Italy since 2010. She has 18 years' experience in endocrinology, including one year (2007) working as a fellow at the Center of Imaging and Bone Research, Radiology Department, Children's Hospital, Los Angeles, where she learned densitometric techniques (DXA and QCT). Her specific expertise is in the pediatric hypothalamus and pituitary gland and the metabolism of bones.
Since 2019, Professor Di Iorgi has been responsible of the Center for the Diagnosis and Treatment of Osteoporosis and Metabolic Bone Diseases at the Giannina Gaslini Institute in Genoa, Italy. Her main research interests include the etiology, investigation and management of growth and bone disorders, and she has published more than 40 papers addressing growth and more than 12 on bone health in children from infancy to transition.
Her clinical practice covers the spectrum of pediatric endocrinology, including all forms of growth hormone deficiency.
She has extensive experience in the diagnosis and management of the long-term endocrine outcomes of pediatric cancer survivors and primary and secondary osteoporosis.



Philippe LYSY

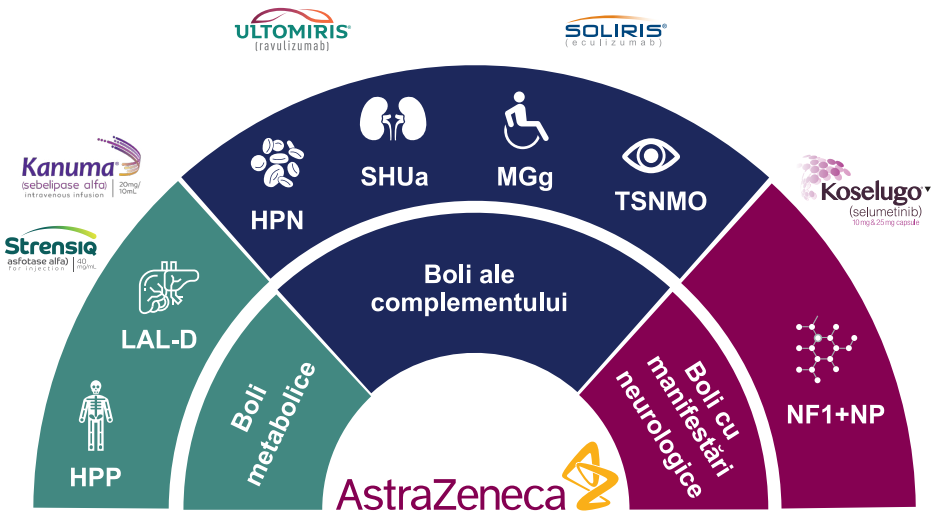
Professor Philippe Lysy is Pediatric Endocrinologist and Diabetologist at Cliniques Universitaires Saint-Luc, Brussels, Belgium
Prof. Philippe Lysy is a highly esteemed pediatric endocrinologist and diabetologist with extensive expertise in clinical care, research, and academic leadership.
Currently serving as Head of Specialized Pediatrics Service and Pediatric Endocrinologist at Cliniques Universitaires Saint-Luc, Brussels, Belgium.
Dr. Lysy's distinguished academic journey includes multiple honors and degrees from UCL Brussels, complemented by post-doctoral training at Harvard Medical School.
He has published prolifically and presented internationally on topics such as beta cell differentiation and early programming's impact on the endocrine system.

Areas of expertise: Type 1 diabetes mellitus, Growth disorders (such as growth hormone deficiencies), Thyroid disorders (including thyroid nodules and thyroid cancer), Disorders of sexual development (DSD), Precocious puberty, Disorders related to insulin resistance (e.g., polycystic ovary syndrome in adolescents), Adrenal disorders (e.g., congenital adrenal hyperplasia), Pituitary disorders, Metabolic bone diseases (e.g., osteoporosis in children), Obesity and its related metabolic complications in children and adolescents.

Simone von SENGBUSCH

Dr. von Sengbusch is primary pediatrician at the University Hospital for Pediatrics and Adolescent Medicine in Lübeck.
Areas of expertise: Pediatrics, pediatric endocrinology and diabetology.
He holds a master's degree in public health and serves on numerous diabetes committees.
He holds the title of Privatdozent (Private Lecturer) at the University of Lübeck. He works in the Pediatrics and Adolescent Medicine Clinic of UKSH, being responsible for the Pediatric Diabetology section. Her work focuses on the diagnosis and treatment of type 1 diabetes in children. She is the coordinator of the mobile diabetes education program in Schleswig-Holstein (MDSH). It stood out for promoting telemedicine and digital solutions, such as the "ViDiKi" project (Virtual Diabetes Ambulance for Children), which facilitates the remote monitoring of young patients.
He has overseen the successful pilot project "Mobile Diabetes Education Schleswig-Holstein" (MDSH) since its inception in 1999. Together with teams from different pediatric clinics, she organizes 24 five-day diabetes group training sessions for children, adolescents and young adults and their parents in eight different pediatric clinics in Germany's northernmost state.
He received the Federal Cross of Merit. Federal President Christian Wulff presented her with the award on 4 October 2011 in Berlin in recognition of her long-term commitment to the care of children and adolescents with diabetes in Schleswig-Holstein.
He is the author of numerous scientific studies published in international journals, focused on the management of type 1 diabetes in children and adolescents.

Angajamentul AstraZeneca în Bolile Rare



Abrevieri:
HPN = hemoglobinuria paroxistică nocturnă; SHUa = sindromul hemolitic uremic atipic; MGg = miastenia gravis generalizată; LAL-D = deficit de lipază acidă lizozomală; HPP = hipofosfatazia; TSNMO = tulburare din spectrul neuromielitei optice; NF1+NP = neurofibromatoza de tip 1 + neurofibrom plexiform.

Pentru informații suplimentare, vă rugăm să consultați versiunea actualizată a Rezumatului Caracteristicilor Produsului **KANUMA**, scanând codul QR.

Pentru informații suplimentare, vă rugăm să consultați versiunea actualizată a Rezumatului Caracteristicilor Produsului **ULTOMIRIS**, scanând codul QR.

Pentru informații suplimentare, vă rugăm să consultați versiunea actualizată a Rezumatului Caracteristicilor Produsului **SOLIRIS**, scanând codul QR.

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Acest material este destinat profesioniștilor din domeniul sănătății.
Evenimentele adverse pot fi raportate online https://contactazmedical.astrazeneca.com, sau la adresa farmacovigilenta@astrazeneca.com.
Medicament eliberat pe bază de prescripție medicală.

RO-27534/09.2025

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013714, sector 1, București, România
Tel.: +40 21 317 60 41; Email: office.romania@astrazeneca.com

Scientific Programme

Program Științific

Wednesday, April 22, 2026 / Miercuri, 22 Aprilie 2026

12.30	Deschiderea secretariatului – Înregistrarea participanților
	Scientific session I / Sesiunea științifică I **"Junior Doctors"** ***Chairs / moderatori: Mihaela Vlad, Corina Paul***
16.00 16.15	**JD 01** The diagnosis of psychogenic polydipsia (potomania) – good news or a burden for the patient? / *Diagnosticul de polidipsie psihogenă (potomania) – veste bună sau povară?* Petronela Cristean, Oana Popa, Mihaela Vlad *(Timișoara / Romania)*
16.15 16.30	**JD 02** Type II autoimmune polyglandular syndrome in a child - case report / *Sindromul poliglandular autoimun de tip II la copil - prezentare de caz.* Andreea-Minodora Nistoran, Anisia Pop, Ramona Albulescu, Corina Pienar, Monica Simina Mihuța, Corina Paul (*Timișoara / Romania*)
16.30 16.45	**JD 03** GATA6 Syndrome: permanent neonatal diabetes associated with pancreatic agenesis and congenital heart disease / *Sindromul GATA6: diabet neonatal permanent asociat cu agenezie pancreatică și cardiopatie congenitală* Eșanu Valeriu, Chiriac Andrian, Eșanu Veronica, Palii Ina (*Chișinău / Rep. Moldova*)

16.45 17.00	**JD 04** Rahitismul hipofosfatemic X-linkat la un copil mic cu manifestări scheletice și dentare severe: prezentare de caz / *X-Linked Hypophosphatemic Rickets in a Young Child With Severe Skeletal and Dental Manifestations: A Case Report* Lorena-Alexandra Andriș, Monica Simina Mihuța, Lavinia Stretcu, Mălina Popa, Cristina Ilieș, Ana-Maria Pani, Corina Paul *(Timișoara / Romania)*
17.00 17.15	**JD 05** Early-onset hyperphagic obesity as a clue to monogenic disease: a case associated with a heterozygous NTRK2 splice -site variant / *Obezitatea hiperfagică cu debut precoce ca indiciu al bolii monogenice: un caz asociat cu o variantă heterozigotă a situsului de îmbinare NTRK2.* Bogdan Mihai Pascu, Roxana Ionela Enăchescu *(București / Romania)*
17.15 17.30	**JD 06** Congenital Adrenal Hyperplasia in childhood and adolescence: closing gaps and facing challenges / *Hiperplazia suprarenală congenitală în copilărie și adolescență: eliminarea lacunelor și confruntarea cu provocări* Alina-Elena Șchiopu, Maria-Christina Ungureanu, Cristina Preda *(Iași / Romania)*
17.30 17.45	**JD 07** Therapeutic complications in congenital hyperinsulinism / *Complicații terapeutice în hiperinsulinismul congenital* Ioana Vasiliu, Delia Andreia Bizim, Carmen Oltean, Ionela Butnariu, Monica Cristina Pinzaru, Oana Roșca, Elena-Lia Spoială, Laura Mihaela Trandafir *(Iași / Romania)*

Scientific session II / Sesiunea științifică II
"NextGen Endo: How AI is changing clinical practice"
"NextGen Endo: Cum schimbă AI practica clinică"
Chair / Moderator: Gheorghe Mihalaș

18.00 18.20	Introduction. Artificial intelligence as a catalyst for precision medicine / *Introducere. Inteligența artificială catalizator al medicinei de precizie.* Ghorghe Mihalaș (*Timișoara / Romania)*
18.20 19.10	Smart algorithms and precision medicine: transforming the management of pediatric diabetes / *Algoritmi inteligenți și transformarea managementului diabetului zaharat pediatric* *Dan Alexa (Eta-2U Timișoara / Romania)*
19.10 19.25	Closed Loop Systems a new paradigm in the safety and quality of life of the child with DM / *Sistemele de Buclă Închisă o nouă paradigmă în siguranța și calitatea vieții copilului cu diabet zaharat tip 1* *Iulian Velea (Timișoara / Romania)*
19.30	**OPENING CEREMONY / CEREMONIA DE DESCHIDERE**
20.00	**WELCOME COCKTAIL / COCKTAIL DE BUN VENIT**

Thursday, April 23, 2026 / Joi 23 aprilie 2026

Scientific session III / Sesiunea științifică III
Chairs / moderatori: Carmen Barbu, Diana Miclea

08.30 08.50	**P.L. 01** Presentation of the International guideline on genetic testing of children with short stature / *Prezentarea ghidului internațional privind testarea genetică a copiilor cu statură mică* Adela Chiriță-Emandi *(Timișoara / Romania)*
08.50 09.10	**P.L. 02** Genetic short stature - diagnostic and treatment / *Talia mică de cauză genetică - diagnostic și tratament* Diana Miclea, Camelia Alkhzouz (*Cluj-Napoca / Romania*)
09.10 09.20	SANDOZ Symposium / Simpozion susținut de SANDOZ Quality of life, disparities and inequities in short stature / *Calitatea vieții, discrepanțe și inechități în statura mică* *Moderator:* Corina PAUL *(Timișoara / România)* *Lector*: Raluca Pop *(Târgu Mureș / Romania*)
09.25 09.45	**P.L. 03** Sports that promote growth. Myth or reality? / *Sporturi ce promovează creșterea. Mit sau realitate ?* Carmen Barbu *(București / Romania)*
09.45 10.05	**P.L. 04** Long-Acting GH Therapy: Efficacy, Adherence and Beyond / *Terapia cu GH cu acțiune prelungită: eficacitate, aderență și dincolo de aceasta* Nastascia di Iorgi *(Genoa / Italy)*
10.05 10.10	**DISCUȚII**
10.10 10.40	MERCK symposium / Simpozion susținut de MERCK Connected to growth: benefits for somatropin therapy in childhood *Conectat la creștere: beneficii pentru terapia cu somatropină la copii* Corina Paul *(Timișoara / Romania)*
10.40 11.00	Coffee break / Pauza de cafea

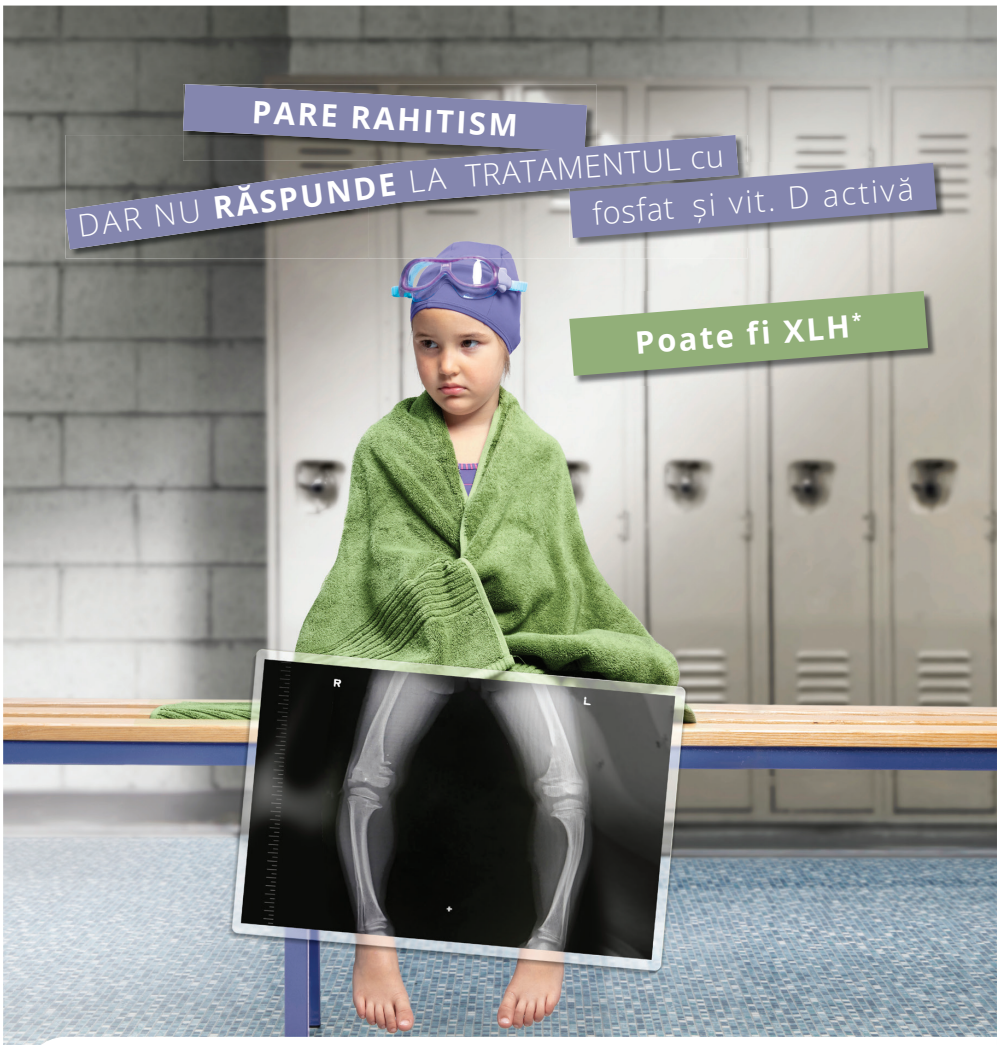
Scientific session IV / Sesiunea științifică IV
Chairs / moderatori: Dana Stoian, Maria-Christina Ungureanu

11.00 11.20	**P.L. 05** Tall stature in children: when should a genetic etiology be suspected. *Statura înaltă la copii: când ar trebui suspectată o etiologie genetică.* Maria Claudia Jurca, Aurora Alexandra Jurca *(Oradea / Romania)*
11.20 11.40	**P.L. 06** Beyond the Mirror: Hirsutism in Adolescence - Clinical Approach / *Dincolo de oglindă: Hirsutismul în adolescență - o abordare clinică* Maria-Christina Ungureanu, Ioana Armașu *(Iași / Romania)*
11.40 12.00	**P.L. 07** Precocious puberty in the context of gender specific pediatrics / *Pubertatea precoce în contextul pediatriei specifice genului* Nastascia di Iorgi *(Genoa / Italy)*
12.00 12.20	**P.L. 08** An approach to the tall child / *Abordarea copilului cu talie înaltă* Jo Blair (*Liverpool / UK*)
12.20 12.40	**P.L. 09** Clinical approach to thyroid nodules in children: an overview / *Abordarea clinică a nodulilor tiroidieni la copii: prezentare generală* Philippe Lysy *(Brussels / Belgium)*
12.40 13.10	**P.L. 10** Meet the expert session Ultrasound diagnosis of breast abnormalities in children and adolescents / *Diagnosticul ecografic al anomaliilor mamare la copii și adolescenți.* Dana Stoian *(Timișoara / Romania)*
13.10 13.30	**P.L. 11** Management of pediatric Graves' disease / *Managementul bolii Graves la copil* Corina Paul (*Timișoara / Romania*)
13.35 14.50	Lunch Break / Pauză de prânz

Scientific session V / Sesiunea științifică V
Chairs / moderatori: Mihaela Vlad, Raluca Pop

15.00 15.20	**O.P. 01** Factors associated with low alkaline phosphatase and its role in detecting hypophosphatasia in 15.031 Romanian children / *Factori asociați cu fosfataza alcalină scăzută și rolul acesteia în detectarea hipofosfataziei la 15.031 de copii din România* Adela Chiriță-Emandi, Oana Aburel, Florin Horhat *(Timișoara / Romania)*
15.20 16.00	Astra Zeneca Symposium / Simpozion susținut de Astra Zeneca How does hypophosphatasia (HPP) manifest? Persistently low alkaline phosphatase activity and genetic testing - their role in the diagnosis of HPP / *Cum se manifestă hipofosfatazia (HPP)? Activitatea persistent scăzută a fosfatazei alcaline și testarea genetică - rolul lor în diagnosticul HPP* Corina Paul *(Timișoara / Romania)*
15.40 16.00	The first patient in Romania with >10 years of treatment with Strensiq / *Primul pacient în România, cu durată >10 ani de tratament cu Strensiq* Ioana Vasiliu (*Iași / Romania*)
16.00 16.20	**P.L. 12** Prolactinoma in children and adolescents / *Prolactinomul la copii și adolescenți* Jo Blair *(Liverpool / UK)*
16.20 16.40	**P.L. 13** Disorders of Sodium and Water Homeostasis: From Differential Diagnosis to Targeted Management / *Tulburări ale homeostaziei sodiului și apei: de la diagnosticul diferențial la tratamentul țintit* Raluca Pop, Ionela Pașcanu *(Târgu Mureș / Romania)*
16.40 17.00	**P.L. 14** Peculiarities in the biochemical investigation of adrenocortical function in children / *Particularități în investigarea biochimică a funcției adrenocorticale la copil.* Corin Badiu *(București / Romania)*

17.00 17.20	Swixx Biopharma Symposium / Simpozion Swixx Biopharma X linked hypophosphatemia from diagnosis to management / *Hipofosfatemia X-linkată, de la diagnostic la managementul bolii.* Corina Paul *(Timișoara / Romania)*
17.20 17.40	**P.L. 15** Transition from Pediatric to Adult Endocrine Care in Patients with Pituitary Disorders / *Tranziția de la îngrijirea endocrinologică pediatrică la cea pentru adulți, la pacienții cu afecțiuni hipofizare* Mihaela Vlad, Carmen D. Dorogi *(Timișoara / Romania)*
17.40 18.00	**O.P. 2** Hyperandrogenemia in a teenager: is it only polycystic ovary syndrome? / *Hiperandrogenemia la o adolescentă: este vorba doar de sindromul ovarelor polichistice?* Anda Dumitrașcu, Dana Terzea, Oana-Claudia Sima, Mihai Costachescu, Ana-Maria Gheorghe, Sorina-Violeta Schipor, Augustin Dima, Mara Carsote *(București / Romania)*



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Link-ul spre platforma Swixx Academy XLH

Acest material este destinat profesioniștilor din domeniul sănătății. * - hipofosfatemie X-linkată; Referințe: **1.** Haffner D et al. Nat Rev Nephrol 2019;15:435–455. **2.** Beck-Nielsen SS et al. Orphanet J Rare Dis 2019;14:58. 3. Skrinar A et al. J Endocr Soc 2019;3:1321–1334. **4.** Lo SH et al. Qual Life Res 2020;29:1883–1893. **5.** Carpenter TO et al. J Bone Miner Res 2011;26:1381–1388.

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1. Wharton S, Freitas P, Hjelmesæth J, et al. Once-weekly semaglutide 7.2 mg in adults with obesity (STEP UP): a randomised, controlled, phase 3b trial. Lancet Diabetes Endocrinol. 2025;S2213-8587(25)00226-8. Online ahead of print. 2. Lincoff AM, Brown-Frandsen K, Colhoun HM, et al. Semaglutide and cardiovascular outcomes in obesity without diabetes. N Engl J Med. 2023;389(24):2221-2232. 3. Rezumatul Caracteristicilor Produsului Wegovy 4. Wilson L, Zhao Z, Divino V, Bassan M, Hartaigh BO, Ozer K. Semaglutide is associated with a lower risk of cardiovascular events compared with tirzepatide in patients with overweight or obesity and ASCVD and without diabetes in routine clinical practice. Results from the STEER study. Presented at: ESC Congress 2025; August 29- September 1, 2025; Madrid, Spain. 5. Hjelmesæth J, Bhat S, Garvey WT, et al. Effect of semaglutide on body composition and proximal muscle strength: the STEP UP trial. Presented at: The 61st European Association for the Study of Diabetes (EASD) Annual Meeting; September 15-19, 2025; Vienna, Austria.

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Friday, April 24, 2026 / Vineri 24 aprilie 2026

Scientific session VI / Sesiunea științifică VI

Chairs / moderatori: Iulian Velea, Dana Anton-Păduraru

08.30 08.50	**P.L. 16** Bias in pediatric obesity - from definition to mitigation / *Prejudecăți în obezitatea pediatrică - de la definiție la metode de combatere* Raluca Pop *(Târgu Mureș / Romania)*
08.50 09.10	**P.L. 17** Obesity in children and adolescent with type 1 diabetes / *Obezitatea la copiii și adolescenții cu diabet zaharat de tip 1.* Dana-Teodora Anton-Păduraru, Carmen Oltean *(Iași / Romania)*
09.10 09.30	**P.L. 18** Obesity: from biology to behavior / *Obezitatea: de la biologie la comportament* Bogdan Timar *(Timișoara / Romania)*
09.30 09.45	NovoNordisk Symposium / Simpozion susținut de NovoNordisk Together for an easier life with Wegovy® - Beyond weight loss: Step UP, safety and „food noise" control / *Impreună pentru o viață trăită mai ușor cu Wegovy® - Dincolo de scăderea în greutate: Step UP, siguranță și controlul "food noise".* Bogdan Timar *(Timișoara / Romania)*
09.45 10.00	**P.L. 19** GLP-1 Receptor Agonists in Pediatric Hypothalamic Obesity: Clinical Utility and Limitations / *Agoniștii GLP-1 în obezitatea hipotalamică infantilă: între utilitatea clinică și limitări* Monica Simina Mihuța, Iulian Velea, Dana Stoian, Corina Paul *(Timișoara / Romania)*
10.00 10.20	**P.L. 20** Novel lipid-lowering therapies for children and adolescents – efficacy and safety / *Terapii hipolipemiante de ultimă generație la copil și adolescent - eficacitate și siguranță* Viviana Elian *(București / Romania)*

10.20 10.40	AstraZeneca Symposium / Simpozion susținut de Astra Zeneca Challenges in the diagnosis and management of the patient with LAL-D (Lysosomal acid lipase deficiency) / *Provocări în diagnosticul și managementul pacientului cu LAL-D (Deficitul lipazei acide lizozomale)* Tudor Pop *(Cluj Napoca / Romania)*
10.40	Coffee break / Pauza de cafea

Scientific session VII / Sesiunea științifică VII

Chairs / moderatori: Cristina Mihai, Mihaela Vlăiculescu

11.00 11.20	**P.L. 21** Early detection of type 1 diabetes – a window of opportunity in patients with associated autoimmune disease / *Detectarea timpurie a DZ tip 1 – o fereastră de oportunitate la pacienții cu boli autoimune asociate* Cristina Maria Mihai, Tatiana Chișnoiu *(Constanța / Romania)*
11.20 11.40	**P.L. 22** Balancing between the stages of type 1 diabetes in children / *Armonizarea tranziției între stadiile diabetului zaharat tip 1 la copil* Tatiana Chișnoiu, Cristina Maria Mihai *(Constanța / Romania)*
11.40 12.00	**P.L. 23** Etiological diagnosis of pediatric patients with atypical diabetes mellitus / *Diagnosticul etiologic al pacienților pediatrici cu diabet zaharat atipic* Philippe Lysy *(Bruxelles / Belgia)*
12.00 12.20	**P.L. 24** Acute Kidney Injury (AKI) as a presentation of Insulin-Dependent Diabetes Mellitus (IDDM) – a diagnostic dilemma / *Insuficiența renală acută (IRA) ca manifestare a diabetului zaharat insulinodependent (DZ tip 1) – o dilemă diagnostică* Alex R. Constantinescu *(Hollywood / USA)*
12.20 12.40	**P.L. 25** Video Consultations for children with diabetes: from a pilot study to standard care in northern Germany / *Consultații video pentru copiii cu diabet: de la un studiu pilot la îngrijirea standard în nordul Germaniei.* Simone von Sengbusch *(Lubeck / Germany)*

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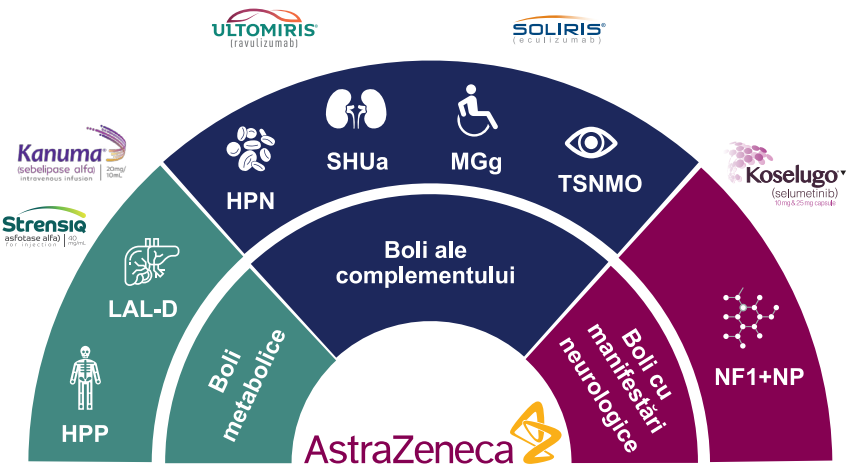

Abrevieri:
HPN = hemoglobinuria paroxistică nocturnă; SHUa = sindromul hemolitic uremic atipic; MGg = miastenia gravis generalizată; LAL-D = deficit de lipază acidă lizozomală; HPP = hipofosfatazia; TSNMO = tulburare din spectrul neuromielitei optice; NF1+NP = neurofibromatoza de tip 1 + neurofibrom plexiform.

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12.40 13.10	Medtronic Symposium / Simpozion susținut de Medtronic Use of the MiniMed 780G System in Children Ages 2-6 / *Utilizarea sistemului MiniMed 780G la copii cu vârste între 2-6 ani.* Iulian Velea *(Timișoara / Romania)*
13.15	Lunch Break / Pauza de prânz

Scientific session VIII / Sesiunea științifică VIII

Chairs / moderatori: Adrian Vlad, Viviana Elian

15.00 15.20	**O.P. 03** Particularities of insulin pump therapy in young children with type 1 diabetes / *Particularități ale terapiei cu pompa de insulină la copiii mici cu diabet zaharat tip 1* Mihaela Vlăiculescu *(București / Romania)*
15.20 15.40	**P.L. 26** Mesangial cells in diabetic kidney disease / *Celulele mezangiale în nefropatia diabetică.* Alex R. Constantinescu (*Hollywood / USA*)
15.40 16.00	**P.L. 27** Similarities and differences in the outpatient care of children with type 1 and type 2 diabetes / *Asemănări și diferențe în îngrijirea în ambulatoriu a copiilor cu diabet de tip 1 și tip 2* Simone von Sengbusch (*Lubeck / Germania*)
16.00 16.20	**P.L. 28** SGLT2 inhibitors in the therapy of childhood type 2 diabetes / *Inhibitorii SGLT2 în tratamentul diabetului zaharat tip 2 la copii.* Adrian Vlad *(Timișoara / Romania)*
16.20 16.40	**O.P. 04** Prevalence of metabolic syndrome in childhood brain tumor survivors *Prevalența sindromului metabolic la supraviețuitorii tumorilor cerebrale din copilărie* Luminița N. Cima, Jyan Al-Ghallie, Marina Iliescu, Anca Coliță, Cristina Jercan, Carmen G. Barbu, Simona Fica *(București / Romania)*

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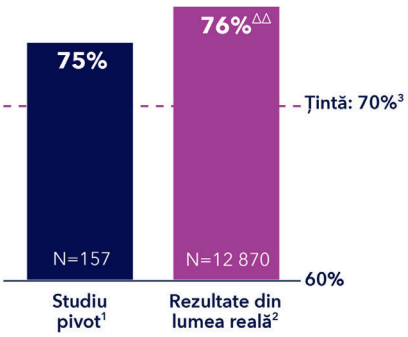

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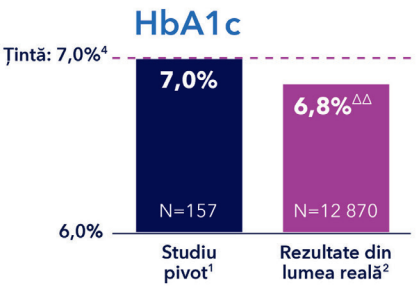

Referințe bibliografice

* Este necesară o citire a glicemiei atunci când se utilizează funcția SmartGuard™. Dacă alertele privind glucoza și valorile CGM nu corespund cu simptomele, se recomandă utilizarea unui glucometru pentru a lua decizii privind tratamentul diabetului. Consultați ghidul de utilizare al sistemului – funcția SmartGuard™. Este necesară o anumită interacțiune cu utilizatorul.
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1. Carlson AL, et al. Diab Tech and Therap. 2021;DOI 10.1089/dia.2021.0319.
2. Medtronic data on file: MiniMed™ 780G data uploaded voluntarily by 12 870 patients in EMEA to CareLink™ Personal, from 27 August 2020 to 22 July 2021.
3. Battelino T, et al. Clinical Targets for Continuous Glucose Monitoring Data Interpretation: Recommendations From the International Consensus on Time in Range. Diabetes Care 2019; 42(8): 1593-1603.
4. ADA Guidelines https://www.diabetes.org/a1c
5. Collyns OJ, et al Diabetes Care. 2021;44(4):969-975

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16.40 17.00	**O.P. 05** The challenging journey from severe neonatal hypoglycemia to stable glycemic control: a case of congenital hyperinsulinism / *Călătoria dificilă de la hipoglicemia neonatală severă la controlul glicemic stabil: un caz de hiperinsulinism congenital* Elena-Lia Spoială, Delia Andreia Bizim, Carmen Oltean, Ioana Vasiliu, Lăcrămioara Ionela Butnariu, Oana Roșca, Gabriela Păduraru, Laura Mihaela Trandafir *(Iași / Romania)*

Saturday, April 25, 2026 / Sâmbătă 25 aprilie 2026

Scientific session IX / Sesiunea științifică IX
(Sesiune comună cu Colegiul Dieteticienilor din România)
Chairs / moderatori: Slăvescu Kinga Cristina, Simona Bogdan

08.30 08.50	**O.P. 06** Ketogenic diet in epilepsy refractory to treatment / *Dieta Cetogenică în epilepsiile refractare la tratament* Sorina Adam (*Cluj - Napoca / Romania)*
08.50 09.10	**O.P. 07** Excessive cow`s milk intake as a cause of severe iron deficiency anemia and protein-losing enteropathy in toddlers: two case reports and diagnostic challenges / *Consumul excesiv de lapte de vacă – cauză a anemiei feriprive severe și a enteropatiei cu pierdere de proteine la copilul mic: două cazuri clinice și dificultăți de diagnostic.* Slăvescu Kinga Cristina *(Cluj-Napoca / Romania)*
09.10 09.30	**O.P. 08** Impact of hypocaloric versus isocaloric diets with differential macronutrient distribution in children with Obesity / *Impactul dietelor hipocalorice versus izocalorice cu distribuție diferențiată a macronutrienților, la copiii cu exces ponderal* Denisa Pescari, Simina Mihuța, Dana Stoian *(Timișoara / Romania)*
09.30 09.50	**O.P. 09** Medical management of a newborn with suspected inherent metabolic disease / *Managementul medical al nou-născutului cu suspiciune de boală metabolică congenitală* Cristian Minulescu *(București / Romania)*
09.50 10.10	**O.P. 10** Nutritional intervention as a central element in therapy with GLP-1 agonists in children and adolescents / *Intervenția nutrițională ca element central în terapia cu agoniști de GLP-1 la copii și adolescenți* Ema Claudia Mărginean, Bogdan Pascu, Anca Bălănescu *(București / Romania)*

10.10 10.30	**O.P. 11** Pediatric Nutrition–Inflammation Crosstalk: A Network Medicine Perspective / *Interacțiunile nutriție–inflamație în pediatrie: o perspectivă a medicinei de rețea* Ana Dragomir *(Timișoara / Romania)*
10.30 10.50	**O.P. 12** Catch-up Growth in Pediatric Practice: The Role of Nutritional Intervention / *Recuperarea creșterii în practica pediatrică: rolul intervenției nutriționale.* Anelise Capotescu, Corina Pienar, Corina Paul, Laura Savu, Simina Mihuța, Liviu Pop *(Timișoara / Romania)*
10.50 11.00	InBody Symposium / Simpozion susținut de InBody InBody analysis: the foundation for personalized childhood obesity management / *Analiza corporală InBody: fundamentul unui management personalizat al obezității la copil.* Ema Claudia Mărginean *(București / Romania)*
11.00 11.20	**O.P. 13** The role of lifestyle interventions in the post-treatment care of children with oncological disease / *Rolul intervențiilor în stilul de viață în îngrijirea post-tratament a copiilor cu boală oncologică.* Contiu Lascu Liana, Boroghină Steluța, Albu Horațiu *(București / Romania)*
11.20 11.40	**O.P. 14** Vegetarian and vegan diets in children: growth, micronutrient status, and bone health outcomes / *Dietele vegetariene și vegane la copii: creșterea, starea micronutrienților și rezultatele sănătății osoase* Maior Raluca *(Tg. Mureș / Romania)*
11.40 12.00	**O.P. 15** The role of the dietitian in step-by-step weight management in children / *Rolul dieteticianului în gestionarea pas cu pas a greutății la copii.* Sânpălean Ioana Monica *(Târgu Mureș / Romania)*
12.20 12.30	CLOSING CEREMONY / CEREMONIA DE INCHIDERE

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Referințe:
1. Alkindi® Rezumatul Caracteristicilor Produsului; **2.** Coope H, et al., Expert Opinion on Orphan Drugs 2021;9:3:67-76; **3.** Neumann U, et al., JCEM 2021;106(3):e1433–e40;

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Data pregătirii: Martie 2026

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SCIENTIFIC WORKS
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PLENARY LECTURES

ORAL PRESENTATION

ORAL PRESENTATION „JUNIOR DOCTORS"

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ABSTRACTS

PLENARY LECTURES:

P.L. 01
PRESENTATION OF THE INTERNATIONAL GUIDELINE ON GENETIC TESTING OF CHILDREN WITH SHORT STATURE

Adela Chiriță-Emandi[1,2]

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Background and Aims: Advances in genomic technologies have transformed the diagnostic approach to short stature, yet international consensus on indications, testing strategies, and interpretation have been lacking. To address this gap, the Clinical Practice Committee of the European Society of Paediatric Endocrinology (ESPE) initiated the development of a comprehensive international guideline on genetic testing for short stature. This work was carried out by the newly formed International Growth Genetics Guideline Consortium (IGGGC), a global collaborative network of experts in pediatric endocrinology and clinical genetics.

Methods: A steering committee was first established to define the guideline framework, recruit an experienced methodologist, and propose expert contributors. Pediatric endocrinologists with extensive experience in genetic evaluation of short stature were invited from ESPE and other regional pediatric endocrine societies. Presidents of the European Society of Human Genetics (ESHG) and the American College of Medical Genetics (ACMG) were informed of the initiative and invited to participate. Clinical geneticists with recognized expertise in growth disorders across three continents were subsequently added. The consortium includes 32 members: 21 pediatric endocrinologists, 9 clinical geneticists, 1 laboratory scientist, and 1 clinical epidemiologist/adult endocrinologist. ESPE served as the sole sponsor, covering all project-related costs.

Results: The IGGGC assembled a geographically diverse, multidisciplinary group of experts to ensure a balanced, evidence-based, and globally applicable guideline. The consortium includes clinical geneticists from Brazil, Germany, Sweden, Spain, Italy, Belgium, the Czech Republic, the UK, and the USA. Collaborative work has involved structured evidence review, consensus-building processes, and harmonization of diagnostic approaches across regions with differing healthcare resources and genetic testing availability.

Conclusion: The International Growth Genetics Guideline Consortium represents an global collaboration aimed at standardizing the genetic evaluation of children with short stature. The resulting guideline is expected to enhance diagnostic accuracy, support equitable access to genetic testing, and provide clinicians worldwide with a unified framework for evaluation and management. This presentation will summarize the development process, global engagement, and anticipated clinical impact of this forthcoming international guideline.

Keywords: *Short stature, Genetic testing, International guideline, Pediatric endocrinology, Growth disorders*

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P.L. 02
GENETIC SHORT STATURE – DIAGNOSTIC AND TREATMENT

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Short stature is defined by a height that is less than two standard deviations below average for general population of the same age and sex. The contribution of genetic factors in height development is up to 90%, the studies indicating thousands of variants in more than 600 genes involved. The majority of these variants have a lower effect, the combinations of them giving a better or lower height prognosis. However, there are also variants with a more detrimental effect on gene functions which leads to an important effect on height, influencing different mechanisms, usually at the level of bone cartillage (primary short stature) or acting on endocrine and metabolic functions (secondary short stature). These disorders are usually diagnosed thorough clinical, radiological (bone age), and laboratory evaluation, followed by genetic testing (karyotype, gene panels or exome/genome sequencing). Main genetic etiologies include: *SHOX, FGFR3, NPR2, IHH, PTPN11, COMP, ACAN, BMP2, COL2A1, NPPC, PAPPA2, GH1, IGF1R, GHRHR, PROP1*, and other genes. Treatment often involves rhGH, but there also new options like vosoritide indicated in achondroplasia (also promising for other genes alterations) and other specific treatments for bone metabolic and endocrine disorders.

Conclusions: Knowing a precise genetic diagnosis in short stature will support in some situations selecting the adequate option for treatment, often available in our region or in some situations avoiding a classical treatment due to possible adverse effects or due to lack of potential effects on growth.

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P.L. 03

SPORTS THAT PROMOTE GROWTH. MYTH OR REALITY?

Carmen Barbu
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No abstract

P.L. 04

LONG-ACTING GH THERAPY: EFFICACY, ADHERENCE AND BEYOND

Nastascia di Iorgi
Genoa / Italia

No abstract

P.L. 05

TALL STATURE IN CHILDREN: WHEN SHOULD A GENETIC ETIOLOGY BE SUSPECTED

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Background: Tall stature is clinically defined as height exceeding +2 standard deviations above the mean for age and sex. Although most children present with familial or constitutional tall stature, recognizing cases with an underlying genetic basis is essential for appropriate diagnostic orientation and patient counseling.

Materials and Methods: This study proposes a structured clinical approach to the evaluation of children with tall stature, focused on identifying features suggestive of a genetic etiology. Auxological parameters, body proportions, and dysmorphic features were assessed and correlated with growth trajectory. Based on these criteria, cases were classified into three categories: monogenic disorders, chromosomal abnormalities, and overgrowth syndromes. Genetic testing was guided by clinical suspicion.

Results: Monogenic disorders frequently showed abnormalities of body proportions or connective tissue features. Chromosomal abnormalities were associated with complex phenotypes and subtle dysmorphic signs. Overgrowth syndromes were suggested by the association of tall stature with macrocephaly, asymmetry, or minor malformations. Careful clinical evaluation enabled differentiation between benign variants and syndromic forms and allowed targeted genetic testing.
Conclusions: Systematic clinical examination remains essential in the assessment of children with tall stature, allowing identification of cases requiring genetic evaluation. Early recognition of syndromic forms supports appropriate follow-up and genetic counseling.
Keywords: *tall stature, monogenic disorders, chromosomal abnormalities, overgrowth syndromes*
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P.L. 06
BEYOND THE MIRROR: HIRSUTISM IN ADOLESCENCE - CLINICAL APPROACH
Maria-Christina Ungureanu, Ioana Armașu
Iași / Romania

No abstract

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PRECOCIOUS PUBERTY IN THE CONTEXT OF GENDER SPECIFIC PEDIATRICS
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No abstract

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AN APPROACH TO THE TALL CHILD

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Background

Children are considered to be of tall stature when their height is > 2 standard deviations (SD) above the population mean for age, sex and ethnicity. Occasionally children of normal stature, >2 SD above their mid-parental height (MPH) range (MPHR), also warrant assessment. Most children have genetic or constitutional tall stature and require no intervention, although a number of rare and serious diagnoses should not be overlooked (Table 1).

Table 1: Causes of tall stature

Familial tall stature
Constitutional tall stature
Obesity
Endocrine causes
- Precocious or delayed puberty
- Thyrotoxicosis
- Growth hormone excess
- Familial glucocorticoid deficiency
- Aromatase deficiency
- Mutations of the oestrogen receptor α gene
- Androgen insensitivity
Syndromic causes
- Beckwith–Wiedemann
- Homocystinuria
- Marfan syndrome
- Klinefelter (XXY) or other sex chromosome aneuploidy associated with additional X or Y chromosome
- Simpson-Golabi-Behmel
- Sotos

Clinical assessment

Clinical assessment starts with a careful history, including a description of the child's pattern of growth. Have the parent noticed signs of precocious puberty or symptoms of thyrotoxicosis? Are there concerns regarding development or behaviour? Is there a family history of tall stature or features suggestive of a genetic endocrine tumour syndrome? In the adolescent, is puberty delayed? Is there a history suggestive of glucocorticoid deficiency?

A detailed auxological assessment includes height, sitting height, arm span, weight, pubertal phase and where possible, height velocity. Measurement of parents and determination of the MPH and MPHR are also essential. Healthy tall children of tall parents are most likely to have inherited tall stature. Children with simple obesity have a height towards or just above the upper limit of the mid-parental height range, a tendency to exaggerated adrenarche, early puberty and modestly advanced bone age. Final adult height is not excessive.

Endocrine causes of tall stature should be explored with examination for clinical features of thyrotoxicosis, premature puberty in girls age < 8 years and boys aged < 9 years. Café au Lait skin lesions may be an of indication McCune Albright Syndrome associated with precocious

puberty, growth hormone or thyroid hormone excess. Delayed puberty may result in tall stature in the adolescent due to delayed epiphyseal fusion. Very rare diagnoses associated with delayed epiphyseal fusion include mutations of the oestrogen α receptor and aromatase deficiency.
Consideration should be given to syndromic diagnoses in children with disproportionate tall stature (e.g. Marfan or Klinefelter syndrome, homocystinuria, oestrogen deficiency or resistance) or dysmorphic features and learning difficulties (e.g. homocystinuria, Sotos syndrome, Weaver syndrome).

Recommended baseline investigations are summarised in Table 2.

Table 2: Baseline investigations for the child with tall stature

- Karyotype
- Thyroid function tests
- IGF-I, interpreted for pubertal phase and bone age
- Bone age
- For children with premature or delayed puberty:
- LH, FSH, oestradiol or testosterone
- Pelvic ultrasound in girls
- If Marfanoid features, consider plasma homocysteine
- Consider DNA storage for children who may have syndromic tall stature

Management

Treatment of thyrotoxicosis, precocious puberty and growth hormone excess result in slowing of growth and in most cases, normalisation of height. Historically, high dose sex steroids have been used to accelerate epiphyseal fusion in children with tall stature of other aetiologies.
However, treatment is associated with significant side effects including reduced fertility in girls, the degree to which final adult height is reduced by these interventions is not well documented. Epiphysiodesis (surgical destruction of the epiphyses of the distal femur, proximal tibia and fibula) may be offered to selected patients.

Summary

Careful clinical assessment excludes serious pathology requiring intervention in most tall children. However, there are a number of rare but important diagnoses that should not be overlooked. Medical interventions, other than treatment of endocrine causes of tall stature, are no longer recommended, but epiphysiodesis may be considered in selected cases.

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CLINICAL APPROACH OF THYROID NODULES IN CHILDREN: AN OVERVIEW

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This presentation reviews the clinical approach to thyroid nodules in children, focusing on diagnosis, risk assessment, and management. Thyroid nodules are relatively uncommon but carry a higher risk of malignancy in children than in adults (≈25–27%), which justifies careful evaluation.

Assessment begins with clinical history and physical examination, looking for risk factors such as radiation exposure or hereditary syndromes. Thyroid ultrasound is the key diagnostic tool, allowing characterization of nodules and risk stratification (e.g., using TIRADS). Suspicious nodules—typically ≥1 cm or with concerning ultrasound features—require fine-needle aspiration (FNA) for cytological analysis.

The most frequent pediatric thyroid cancers are papillary and follicular carcinomas, which may present with lymph node metastases but generally have an excellent prognosis (≈98% survival). Treatment mainly involves surgery (usually total thyroidectomy), sometimes followed by radioiodine therapy and long-term follow-up.

Overall, early and structured evaluation allows accurate diagnosis while avoiding unnecessary surgery for benign nodules.

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ULTRASOUND DIAGNOSIS OF BBREAST ABNORMALITIES IN CHILDREN AND ADOLESCENTS

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Breast ultrasound (US) is the imaging modality of choice for evaluating breast abnormalities in children and adolescents. Owing to the high density of the developing breast and the well-established need to avoid ionizing radiation in young patients, mammography has no routine role in this age group. High-frequency linear transducers (12–18 MHz) provide excellent spatial resolution, enabling detailed visualization of the superficial glandular structures that characterize the hormonally active breast during puberty. Ultrasound is therefore the first-line, and often the only required, imaging examination in pediatric and adolescent breast assessment.

The clinical indications for breast ultrasound in this population include palpable masses, focal breast pain, asymmetry, nipple discharge, inflammatory signs, trauma, and evaluation of gynecomastia in adolescent boys. The overwhelming majority of lesions encountered are benign. Fibroadenoma represents the most frequent solid tumor in adolescent girls and typically appears on ultrasound as a well-circumscribed, oval, homogeneously hypoechoic mass with parallel orientation and minimal posterior acoustic features. Ultrasound allows accurate characterization of margins, echotexture, posterior features, and internal vascularity, facilitating confident diagnosis and reducing unnecessary invasive procedures.

Cystic lesions are also common and are readily identified by their anechoic content, posterior acoustic enhancement, and thin walls. Complicated cysts, galactoceles, and post-traumatic

hematomas can be differentiated based on internal echoes and clinical context. Inflammatory conditions such as mastitis and breast abscess are particularly well assessed by ultrasound, which demonstrates skin thickening, increased echogenicity of surrounding tissues, hyperemia on Doppler imaging, and, when present, fluid collections amenable to image-guided drainage. The real-time capability of ultrasound makes it ideal for guiding minimally invasive interventions while preserving cosmetic integrity—an important consideration in the developing breast.

Although primary breast malignancy is rare in children and adolescents, ultrasound plays a critical role in identifying suspicious features that warrant biopsy. Red flags include irregular or spiculated margins, non-parallel orientation, heterogeneous internal architecture, posterior acoustic shadowing, and increased internal vascularity. Associated axillary lymphadenopathy further raises concern. When indicated, ultrasound-guided core needle biopsy provides tissue diagnosis with high accuracy and minimal morbidity. Secondary breast involvement from systemic malignancies, though uncommon, may also be detected through targeted ultrasound evaluation.

Breast ultrasound is equally important in longitudinal follow-up. Many fibroadenomas are managed conservatively with periodic imaging to document stability. Serial examinations allow objective measurement of lesion dimensions, growth rate, and structural changes. Rapid enlargement, particularly in giant juvenile fibroadenomas, can be accurately monitored and used to inform surgical timing. Post-procedural follow-up after biopsy or surgical excision relies on ultrasound to detect hematoma, seroma, infection, or recurrence.

Advanced techniques further enhance diagnostic performance. Color and power Doppler imaging assess vascular patterns, supporting differentiation between inflammatory, benign proliferative, and suspicious lesions. Shear-wave elastography provides quantitative assessment of tissue stiffness, potentially improving specificity in selected cases and reducing unnecessary biopsies. Standardized reporting systems adapted to the pediatric context promote consistency and multidisciplinary communication.

In **conclusion**, breast ultrasound is the cornerstone of diagnostic evaluation and follow-up in children and adolescents with breast concerns. Its radiation-free nature, high-resolution imaging, real-time interventional capability, and adaptability to advanced techniques make it ideally suited to the unique anatomical, physiological, and psychosocial aspects of pediatric breast care. As technology and operator expertise continue to evolve, ultrasound will remain central to safe, accurate, and patient-centered management in this vulnerable population.

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MANAGEMENT OF PEDIATRIC GRAVES' DISEASE

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No abstract

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PROLACTINOMA IN CHILDREN AND ADOLESCENTS

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Background.

Prolactin is a polypeptide hormone which has many roles, of which the most important relates to induction and maintenance of lactation. Prolactin is synthesised and secreted spontaneously by lactotroph cells in the anterior pituitary and inhibited by hypothalamic dopamine.

The prevalence of pituitary adenomas in children and young people (CYP) aged 10-19 years is 0.5 and 3/100,000 in boys and girls respectively,[1] with prolactinomas accounting for approximately 50% of pituitary adenomas in this age group. As in adults, prolactinomas affect females much more commonly than males.

Clinical presentation.

Patients may present with clinical features of prolactin excess including galactorrhoea, gynecomastia, delayed or arrested puberty, primary or secondary amenorrhoea. Prolactin promotes food intake during pregnancy, and weight gain is common at the presentation of prolactinoma. Patients may also present with mass effects including loss of other anterior pituitary hormones, visual field loss and headache.

Prolactin concentrations and adenoma size are related to age and sex, with boys and younger children having higher prolactin concentrations at diagnosis than girls or older CYP.[2] Macroprolactinomas (diameter >1cm) are reported much more commonly in CYP than in adults, 90% of males and 50% of females. [3]

Investigation.

Causes of secondary hyperprolactinaemia should be excluded before a diagnosis of prolactinoma is made (*Table 1*).

Macroprolactin, a polymeric form of prolactin, is of lower biological activity than monomeric prolactin, High concentrations of macroprolactin may not require treatment and the concentration of macroprolactin should be determined before treatment is recommended. Extremely high concentrations of prolactin saturate binding sites in two-

site immunoradiometric assays resulting in an underestimate of prolactin concentrations. As tumour size and prolactin concentrations are directly related, blood samples in patients with large prolactinomas but only modestly increased prolactin concentrations should be repeated following dilution.
Patients should be offered genetic testing for inherited conditions associated with prolactinoma (*Table 2*), as screening is required for other manifestations of these syndromes and genetic testing can be offered to first degree relatives.

Table 1: Causes of secondary hyperprolactinaemia

Condition	Affected gene	Other clinical features
Multiple endocrine neoplasia type 1	MEN1	Parathyroid hyperplasia, other functional pituitary adenomas, pancreatic neuroendocrine tumours, other endocrine tumours (e.g. adrenocortical tumours), non-endocrine tumours (e.g. facial angiofibroma, meningioma)
Multiple endocrine neoplasia type 4	CDKN1B	Parathyroid hyperplasia, other functional pituitary adenomas, pancreatic neuroendocrine tumours, adrenocortical tumours
Familial isolated pituitary adenoma	AIP	Other functional pituitary adenomas
Carney complex	CDKN1B	Cardiac myxoma, cutaneous and mucosal myxoma, breast myxoma, thyroid tumours, primary pigmented nodular adrenocortical disease, large cell calcifying Sertoli cell tumours, other functional pituitary adenomas, psammomatous melanotic schwannoma, skin pigment abnormalities

Table 2: Syndromes associated with prolactinoma

Hypothyroidism
Medications associated with hyperprolactinaemia including
- Antipsychotics (first and second generation)
- Antidepressants (tricyclic and serotonin reuptake inhibitors)
- Neuroleptic – like medications
- Anti-emetics
- Antihypertensives
- Oestrogens

Renal impairment
Hepatic impairment
Polycystic ovarian syndrome
Pituitary stalk interruption syndrome

Treatment
Dopamine agonists (DA) are effective treatment for prolactinoma in CYP. Cabergoline is more effective and has fewer side effects than bromocriptine and is the first choice DA. The risk of valvopathies is very low in adults treated with DA for prolactinoma. However, the cumulative

dose of DA may be high in CYP treated for prolonged periods and a baseline echocardiogram is recommended with ongoing monitoring in patients thought to be at increased risk of developing heart valve abnormalities.[4] Treatment is recommended for at least two years. Consideration can then be given to withdrawal of treatment if prolactin concentrations are normal and the prolactinoma has reduced in size by >50% on MRI.[4]

Outcomes

The likelihood of achieving remission with DA is inversely related to adenoma size and prolactin concentration at diagnosis, and invasion of the cavernous sinus invasion. Surgery is recommended for patients unable to tolerate DA and those resistant to DA therapy.[4] However, given the limitations of medical therapy and good outcomes from transsphenoidal surgery, there is some debate about the place of earlier surgical intervention.[5]

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P.L. 13

DISORDERS OF SODIUM AND WATER HOMEOSTASIS: FROM DIFFERENTIAL DIAGNOSIS TO TARGETED MANAGEMENT

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Disorders of sodium and water homeostasis are among the most time-critical electrolyte problems because even modest shifts in effective plasma tonicity can rapidly produce cerebral edema (hyponatremia) or cellular dehydration with intracranial injury (hypernatremia).

Serum sodium should be interpreted as a marker of water balance, shaped by vasopressin (AVP), thirst, and renal sodium handling via the renin-angiotensin-aldosterone system.

Significant fluid losses from outside the kidneys frequently increase the risk of electrolyte imbalances. Because sodium is the primary driver of serum osmolality, fluctuations in its

concentration can cause water to move between the intracellular and extracellular spaces[1]. Children are particularly vulnerable because total body water is proportionally higher and cranial compliance is limited[2].

The diagnostic algorithm for electrolyte disturbances is not always applicable in practice, which leads sometimes to empirical treatment[3]. Long term management of electrolyte disturbances should focus on establishing etiology with a great degree of certainty and balancing the benefit/risk ratio of overtreatment [3].

Congenital adrenal hyperplasia, pseudo-hypoaldosteronism, syndrome of inappropriate antidiuresis (SIAD), renal salt wasting are all part of the differential diagnosis in case of hyponatremia, while AVP deficiency, resistance[4], fluid loss and salt poisoning are among the culprits for hypernatremia.

A thorough history and physical examination might be the cornerstones in establishing diagnosis, paired with a focused paraclinical assessment.

Use of isotonic fluids has moderately decreased the incidence of hyponatraemia, but it still is present in 20% of hospitalized children [5], but not in outpatient evaluations, which emphasizes the need to individualize indications for electrolyte assessments in pediatric patients.

Conclusion: Sodium and water homeostasis are of particular interest for pediatric endocrinology, as their diagnosis and management is often a difficult and dynamic process with impact on mortality and morbidity later in life.

Keywords: *hypernatremia, hyponatremia, pseudo-hypoaldosteronism, SIAD*

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P.L. 14

PECULIARITIES IN THE BIOCHEMICAL INVESTIGATION OF ADRENOCORTICAL FUNCTION IN CHILDREN.

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No abstract

P.L. 15

TRANSITION FROM PEDIATRIC TO ADULT ENDOCRINE CARE IN PATIENTS WITH PITUITARY DISORDERS

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Introduction: The transition age is the period between childhood and adulthood; it refers to a broad set of physical, cognitive, and sociocultural modifications, arbitrarily defined as starting in late puberty and ending with full adult maturation—from mid to late teenage years until 6-7 years after reaching final height. This is a critical period for adolescents and young adults with chronic endocrine disorders, especially those with hypopituitarism and childhood-onset growth hormone deficiency. This phase is associated with an increased risk of loss to endocrinological follow-up, treatment discontinuation, metabolic disorders, and impaired bone and muscle mass development.

Method: For this paper, we reviewed guidelines, studies, and other publications addressing transition models, hormonal reassessment strategies, GH management during transition, and the actual outcomes of following these cases.

Results and discussions: Transition is not merely transfer of care but a structured, purposeful process that includes preparation, handover, and post-transfer integration into adult-centred healthcare systems. During this period, patients are characterized by the development of executive function, identity formation, changing family roles, and increased risk-taking behaviours, all of which influence disease self-management and direct involvement in medical care. At this stage, patients usually have other priorities such as educational goals, employment, relocation, and social integration, which can reduce adherence to monitoring, which is important and even vital, especially in the case of complex endocrine disorders that require lifelong hormone replacement.

Hypopituitarism presents unique challenges during the transition period due to the potential dynamic evolution of pituitary deficiencies and the need for hypothalamic-pituitary reassessment. Retesting of growth hormone (GH) secretion after completion of height growth reveals that a substantial proportion of patients with idiopathic isolated GH deficiency show normalization of GH secretion, while subjects with GH deficiency of genetic origin, or with hypothalamic-pituitary structural alterations, or associated with other pituitary hormone deficiencies, more frequently show persistence of GH-deficiency into adulthood. Appropriate biochemical reassessment during the transition period is therefore essential to avoid both overtreatment and undertreatment.

Discontinuation of GH therapy in patients with persistent deficiency after reaching final height is associated with reduced bone mineral density, a body composition characterized by increased fat mass and reduced lean mass, and a potential future increase of cardiometabolic risk. Clinical studies support the need to continue GH replacement therapy in adolescents

and young adults with confirmed persistent GH deficiency, using intermediate doses between pediatric and adult regimens, with dose adjustment based on IGF-1.
Studies conducted during the transition period report a loss to follow-up rate ranging from 12% to over 30% for all endocrine conditions. Structured transition programs-which include early preparation, readiness assessment, joint pediatric - adult clinics, multidisciplinary collaboration, and individualized case management - are associated with significantly higher case retention, often exceeding 80-90%.
Psychiatric comorbidities, such as anxiety, depression, and eating disorders, can complicate the management of endocrine diseases during transition and contribute to discontinuation of care.
Conclusions: The transition period for patients with pituitary disorders requires the integration of medical reassessment, individualized hormone replacement strategies, and structured organizational models. For patients with persistent GHD or complex hypopituitarism, continuation of GH therapy during the transition period is essential for optimal skeletal maturation and prevention of metabolic disorders.
Keywords: *transition, hypopituitarism, growth hormone, therapy*
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P.L. 16
BIAS IN PEDIATRIC OBESITY - FROM DEFINITION TO MITIGATION

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The prevalence of pediatric obesity has been rising across Western societies[1] and has increased markedly in Romania over recent years[2]. In addition, concerns about future trends remain substantial, as current projections provide little evidence that the epidemic is likely to abate in the near term [3].

Numerous clinical guidelines outline approaches to the management of childhood overweight and obesity[4]. However, these consistently underscore the primacy of prevention, given that available treatment strategies frequently yield suboptimal outcomes, even though multicomponent interventions may confer measurable benefits[5].

Children with excess weight are frequently exposed to weight-related stigma, which is associated with adverse psychosocial outcomes, including diminished self-esteem, elevated symptoms of depression and anxiety, and poorer body image. Empirical studies further indicate that children living with obesity who experience negative peer attitudes exhibit higher rates of suicidal ideation and suicidal behaviors compared with their non-stigmatized counterparts. Parents are the source for the most negative stigma faced by children with obesity[2], but healthcare providers are also among the culprits.[6] Click or tap here to enter text. There is insufficient available data regarding the subject, and most professionals acknowledge lack of training in communication skills especially regarding weight management [6].

Conclusion: Addressing bias and stigma is particularly important when devising management strategies for gaining and maintaining a healthy weight in a child and more emphasis should be put on training healthcare professionals in this regard.

Keywords: *bias, children, obesity, stigma*

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P.L. 17
OBESITY IN CHILDREN AND ADOLESCENTS WITH TYPE 1 DIABETES

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Background and Aims: With the increase in the prevalence of overweight and obesity in the general population, the prevalence of obesity in children and adolescents with type 1 diabetes

(T1D) has also increased, complicating the differential diagnosis of the type of diabetes at this age. Obesity can contribute to the development of T1D, but it can also be its result. The aim of our presentation was to present the actual knowledge about the aetiology of obesity in children with T1D, the complications and the modalities of treatment.

Material and Methods: To create the presentation, we selected articles from the last 5 years that appeared in various databases.

Results: Although patients with T1D are usually thin, in recent years the number of cases with obesity has increased (35%), diabesity (the presence of both diabetes and obesity in an individual) being an increasing problem all over the world. Numerous factors are cited in the prevalence of obesity in T1D: dietary mistakes, excessive carbohydrate intake in order to prevent hypoglycemia induced by insulin administration, sedentary lifestyle, different insulin therapy regimens, psychosocial factors. Insulin intensive therapy is considered a key factor that contribute to the appearance of obesity in T1D. The mechanism by which insulin therapy causes weight gain is not well known, but several hypotheses have been proposed: switching from multiple daily injections to continuous subcutaneous insulin infusion, overeating to prevent the risk of hypoglycemia secondary to intensive insulin therapy, insulin penetration into the systemic circulation after administration, with an effect on adipose tissue. The presence of obesity in patients with T1D favors the occurrence of complications (cardiovascular and renal, metabolic dysfunction-associated fatty liver disease, metabolic syndrome, polycystic ovary syndrome), as well as increased morbidity and mortality, also being a T1D progression predictor. The management of obesity in children and adolescents with T1D is challenging, involving both non-pharmacological and pharmacological interventions: dietary interventions (low calorie diet based on the principles of Mediteranean diet), physical activity (a combination of aerobic and strength exercises minimum 60 minutes daily, 3 times/ week), adjunctive pharmacological therapies (Metformin).

Conclusions: The coexistence of obesity in children and adolescents with T1D affects the evolution and prognosis of diabetes. Insulin can contribute to weight gain in patients with T1D, but despite this, it remains the main therapeutic option that must be associated with a healthy lifestyle and a structured education. Interventions on modifiable lifestyle risk factors for obesity in children and adolescents with T1D are important for the prevention and management of obesity in these patients. Future studies are needed on the coexistence of obesity in children with T1D, as well as the discovery of new adjunctive drugs applicable to children.

Keywords: *adolescents, children, diabetes, obesity.*

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P.L. 18

OBESITY: FROM BIOLOGY TO BEHAVIOR

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No abstract

P.L. 19

GLP-1 RECEPTOR AGONISTS IN PEDIATRIC HYPOTHALAMIC OBESITY: CLINICAL UTILITY AND LIMITATIONS

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Hypothalamic obesity represents one of the most challenging forms of pediatric obesity, characterized by severe hyperphagia, reduced energy expenditure, and dysregulation of autonomic and neuroendocrine control. While advances in identifying genetic and epigenetic contributors to hypothalamic obesity have improved our understanding of body-weight regulation, most patients develop the condition secondary to acquired hypothalamic damage.[1] Central nervous system tumours represent the leading acquired cause of hypothalamic obesity, accounting for approximately 50–60% of cases. In pediatric populations, CNS tumours are the second most frequent malignancy, comprising about one quarter of all childhood cancers. Among these, 5–16% involve the suprasellar region, with adamantinomatous craniopharyngiomas responsible for up to 80% of such lesions. Genetic alterations, including mutations in genes such as *BRAF* and *CTNNB1*, which participate in the β-catenin signaling pathway, have been described in craniopharyngiomas; however, their role as definitive driver mutations remains uncertain.[1]

In contrast to acquired forms, hypothalamic obesity may also arise from genetic disorders, most notably Prader–Willi syndrome. PWS results from the loss of paternally expressed genes on chromosome 15q11–q13 and is characterized by early hypotonia, developmental

delay, hyperphagia, and progressive severe obesity. Dysregulation of hypothalamic appetite pathways, altered ghrelin secretion, and reduced energy expenditure contribute to the distinctive metabolic phenotype observed in these patients.

In recent years, GLP-1 receptor agonists have emerged as promising therapeutic options due to their effects on satiety, gastric motility, and glycemic regulation. Available evidence suggests that these agents may reduce food intake and improve metabolic parameters in some patients with hypothalamic obesity, including those with genetic syndromes or acquired hypothalamic injury. However, therapeutic responses are heterogeneous, and weight loss is often modest compared with common obesity, reflecting the complex central mechanisms underlying the condition.[2,3,4]

Limitations of therapy include variability in individual response, the need for long-term treatment, gastrointestinal tolerability issues, and high costs. Moreover, pediatric data remain limited, and optimal patient selection criteria are not yet clearly defined.[4, 5]

GLP-1 receptor agonists are unlikely to represent a standalone solution for hypothalamic obesity, but they may serve as a valuable component of a multidisciplinary strategy that integrates nutritional, hormonal, and behavioral interventions. Identifying responder phenotypes and incorporating therapy early within personalized management pathways represent important directions for future research.

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P.L. 20

NOVEL LIPID-LOWERING THERAPIES FOR CHILDREN AND ADOLESCENTS – EFFICACY AND SAFETY

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Background:
Dyslipidemia in children and adolescents is a major modifiable risk factor for premature cardiovascular disease (CVD). Familial hypercholesterolemia (FH) affects 1 in 200–500 individuals in its heterozygous form (HeFH) and 1 in 160,000 in its homozygous form (HoFH). Atherosclerosis begins in early childhood as Bengalusa Study proved. Despite first-line therapies (statins, ezetimibe), only 20% of HeFH patients achieve LDL-C <100 mg/dL and HoFH patients are largely unresponsive to conventional treatments, with LDL-C often exceeding 500 mg/dL. These unmet needs have driven the clinical research of novel lipid-lowering agents in the pediatric population.
Methods:
A narrative review was conducted using PubMed/MEDLINE, ClinicalTrials.gov, and EMA/FDA databases. Articles from January 2019 to October 2024 were included. Selection criteria prioritized randomized controlled trials (RCTs), phase 2–3 clinical studies, systematic reviews, meta-analyses, and current guidelines.
Results:
Statins remain the cornerstone of therapy (LDL-C reduction 30–50%; approved ≥8–10 years), with ezetimibe adding an additional 15–25% reduction. Among novel agents, evolocumab (≥10 years) reduced LDL-C by 38.3% versus placebo in children aged 10–17 with HeFH; alirocumab (≥8 years) achieved reductions of 43.3% (Q2W) and 33.8% (Q4W) at 24 weeks, with 77.3% of patients reaching LDL-C <130 mg/dL. Pooled analysis confirmed a weighted mean difference of −37.92% (95% CI: −43.06 to −32.78%), with no excess adverse events versus placebo. In HoFH, evinacumab reduced LDL-C by 48% from a baseline of 302 mg/dL in children aged 5–11, with sustained reductions of 40.7% at 72 weeks. Inclisiran is under evaluation in adolescents aged 12–17 in ORION-13 (HoFH) and ORION-16 (HeFH), with results expected in 2025.
Conclusions:
Novel lipid-lowering therapies represent a paradigm shift in severe pediatric dyslipidemia management. PCSK9 monoclonal antibodies provide significant, sustained LDL-C reductions with an excellent safety profile in HeFH. Evinacumab represents the most important advance for HoFH, the first therapy effective from ≥5 years. Early, guideline-directed, multidisciplinary care (cardiology, genetics, dietetics, diabetology) combined with novel agents reduce the cardiovascular burden of familial hypercholesterolemia. Pending results of ORION-16 and ongoing CRISPR-based gene therapy trials will further define the evolving treatment landscape.
Keywords: *pediatric dyslipidemia; familial hypercholesterolemia; PCSK9 inhibitors*
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P.L. 21

EARLY DETECTION OF TYPE 1 DIABETES – A WINDOW OF OPPORTUNITY IN PATIENTS WITH ASSOCIATED AUTOIMMUNE DIASEASE

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Background and aims

Type 1 diabetes (T1D) is a chronic autoimmune disorder characterized by immune-mediated destruction of pancreatic β-cells, resulting in lifelong insulin dependence[1]. The disease develops through a prolonged preclinical phase, during which pancreatic autoantibodies can be detected years before the onset of symptomatic hyperglycemia. Individuals with other autoimmune diseases represent a particularly vulnerable group due to shared genetic susceptibility, immune dysregulation, and common environmental triggers[2]. Conditions such as autoimmune thyroid disease, celiac disease, autoimmune gastritis, and other immune-mediated disorders frequently coexist with T1D[3]. Early identification of individuals at risk offers the possibility of closer monitoring, prevention of severe metabolic decompensation at diagnosis, and participation in emerging disease-modifying interventions[4,5]. The aim of this plenary lecture is to emphasize the importance of early detection of T1D in patients with associated autoimmune diseases and to discuss the clinical implications and opportunities offered by screening strategies.in children presenting with severe new-onset DKA, illustrated by two representative cases.

Material and methods

This presentation is based on a narrative review of current literature and recent evidence from clinical studies, screening programs, and international recommendations regarding the staging and early identification of T1D. Particular focus is placed on populations with associated autoimmune diseases, evaluating the prevalence of pancreatic islet autoantibodies (including GAD, IA-2, ZnT8, and insulin autoantibodies), the rate of progression to clinical diabetes, and the benefits of systematic screening. Data from longitudinal cohort studies and prevention trials were analyzed to highlight the role of early detection in clinical practice.

Results

Multiple studies demonstrate that individuals with pre-existing autoimmune diseases have a higher prevalence of pancreatic autoantibodies compared with the general population. The presence of two or more autoantibodies is associated with a markedly increased risk of progression to clinical T1D. Screening programs conducted in high-risk populations have shown that identification of individuals in presymptomatic stages allows early metabolic surveillance and significantly reduces the incidence of diabetic ketoacidosis at diagnosis. Furthermore, early detection facilitates patient education, timely clinical follow-up, and potential enrollment in immunomodulatory or preventive clinical trials aimed at delaying disease progression.

Conclusions

Patients with associated autoimmune diseases represent an important target group for early T1D screening. Recognition of presymptomatic stages provides a critical window of opportunity for improved disease monitoring, prevention of acute complications, and possible implementation of disease-modifying strategies. Integrating pancreatic autoantibody screening into the routine management of patients with autoimmune conditions may enhance early diagnosis and improve long-term outcomes, supporting a more proactive and personalized approach to the management of type 1 diabetes.

Keywords: *Type 1 diabetes, Early detection, Pancreatic autoantibodies*

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P.L. 22

BALANCING BETWEEN THE STAGES OF TYPE 1 DIABETES IN CHILDREN

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Background and aims
Type 1 diabetes (T1D) in children is a chronic autoimmune condition characterized by progressive destruction of pancreatic β-cells[1]. Advances in the understanding of T1D pathogenesis have led to the recognition of distinct stages of the disease, ranging from the presymptomatic phase with islet autoimmunity to the clinical stage with overt hyperglycemia and metabolic instability[2]. This staging model provides a valuable framework for early identification, monitoring, and potential intervention before the onset of symptomatic disease[3]. In pediatric populations, timely recognition of the transition between stages is particularly important to reduce the risk of severe metabolic decompensation at diagnosis and to improve long-term outcomes[4,5]. The aim of this plenary lecture is to highlight the clinical significance of the different stages of T1D in children, to discuss the challenges of balancing surveillance and intervention across these stages, and to emphasize opportunities for early detection and prevention.
Material and methods
This presentation is based on a narrative review of recent literature, including longitudinal cohort studies, pediatric screening programs, and international guidelines addressing the staging and management of T1D in children. Evidence from observational studies and clinical trials evaluating the natural history of islet autoimmunity, progression rates from stage 1 and stage 2 to clinical diabetes, and the impact of early monitoring strategies were analyzed. Particular attention was given to the role of pancreatic autoantibody testing, metabolic markers, and risk stratification tools used in pediatric populations.
Results
Current evidence demonstrates that T1D evolves through three recognized stages. Stage 1 is characterized by the presence of two or more islet autoantibodies with normoglycemia, while stage 2 includes persistent autoimmunity accompanied by dysglycemia without overt symptoms. Stage 3 corresponds to clinically manifest diabetes with hyperglycemia and typical symptoms such as polyuria, polydipsia, weight loss, and potential diabetic ketoacidosis. Longitudinal studies have shown that children with multiple autoantibodies have a high lifetime risk of progressing to clinical diabetes. Early identification through screening programs allows for structured monitoring of glycemic changes, family education, and prevention of severe metabolic complications at diagnosis. Moreover, emerging immunomodulatory therapies and prevention trials highlight the potential to delay disease progression when interventions are implemented during the presymptomatic stages. These developments underscore the importance of continuous risk assessment and individualized follow-up in children identified at early stages of T1D.
Conclusions
Understanding and recognizing the stages of type 1 diabetes in children provides a critical opportunity for earlier diagnosis, improved monitoring, and potential preventive interventions. Balancing surveillance, clinical management, and emerging therapeutic strategies across the different stages of the disease may reduce the incidence of diabetic ketoacidosis at onset and improve long-term outcomes. Integrating staging concepts into pediatric clinical practice

supports a proactive and personalized approach to the management of T1D and opens new perspectives for disease-modifying strategies in children at risk.

***Key words**: Type 1 diabetes; Disease staging; Islet autoimmunity*

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PL 23

ETIOLOGICAL DIAGNOSIS OF PEDIATRIC PATIENTS WITH ATYPICAL DIABETES MELLITUS

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This presentation addresses the difficulty of diagnosing atypical diabetes in children, a heterogeneous group often misclassified as type 1 or type 2 diabetes. Some of these patients actually have monogenic forms of diabetes (e.g., MODY), which represent about 2–3% of diabetes cases and require different management.
The GENEPEDIAB study combines clinical phenotyping and genetic testing to improve diagnosis. A diagnostic tool called the DIAMODIA score was developed using markers such as absence of autoantibodies, persistent C-peptide secretion, and specific glycemic indices. This score helps identify patients with profiles suggestive of monogenic diabetes who should undergo genetic screening.
Overall, the approach aims to enable etiology-based and personalized diagnosis of pediatric diabetes, improving treatment selection and patient care.

P.L. 24

ACUTE KIDNEY INJURY (AKI) AS A PRESENTATION OF INSULIN-DEPENDENT DIABETES MELLITUS (IDDM) – A DIAGNOSTIC DILEMMA

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Acute Kidney Injury (AKI), a serious complication of Diabetes Mellitus (DM), can also be the presentation of IDDM, especially in DKA. The overlapping of acute nephritic processes may pose a dilemma in diagnosis. The incidence of AKI in children hospitalized for diabetic ketoacidosis (DKA) has been reported to be as high as 64.2%. It can often be triggered by dehydration, contrast media, infections, or worsening diabetic control, and even if not recurrent, it can increase the risk for chronic kidney disease (CKD), need for dialysis, and death. Thus, it requires close monitoring and adequate glycemic control to ensure kidney health. In the last decade, we have gained invaluable insights into the mechanisms of injury, repair, and the use of biomarkers to understand the progression of AKI.
Several common triggers for AKI in patients with DM:

1. Dehydration: mostly during infections with vomiting or diarrhea (e.g., severe Diabetic Ketoacidosis - DKA).
2. Contrast Agents: Used in imaging studies (CT scans, angiograms).
3. Medications: some antibiotics (like aminoglycosides) or antihypertensive drugs.
4. Sepsis/Infection: Major infections are more dangerous for diabetic kidneys.
5. Surgery: Higher risk after major surgeries.

Several factors seen in DM that increase the risk of AKI:

1. Vascular Damage: Hyperglycemia causes inflammation of endothelium in renal vasculature, reducing blood flow.
2. Inflammation & Atherosclerosis: Diabetes leads to atherosclerosis, further compromising kidney perfusion.
3. Impaired Repair: Metabolic changes in DM hinder the kidney's ability to self-repair after injury.

4. Comorbidities: Common diabetic issues like hypertension, obesity, and heart failure worsen kidney vulnerability.

Long-Term Consequences of AKI in IDDM

1. Faster Progression: AKI accelerates the development of CKD and End-Stage Renal Disease (ESRD).
2. Increased Mortality: Higher risk of death, mainly from cardiovascular issues.
3. Recurrence: these patients are prone to repeated episodes of AKI.

Principles of Management & Prevention – crucial in this population to prevent long-term complications

1. Strict Glucose Control: Keeping HbA1c at least below 7%.
2. Manage Blood Pressure: Control hypertension – attention to latest guidelines
3. Careful Medication Use: avoid nephrotoxic agents.
4. Stay Hydrated: especially during acute illnesses.
5. Monitor Kidney Function: Regular monitoring of GFR and urine microalbumin-to-creatinine ratio.

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P.L.25
VIDEO CONSULTATIONS FOR CHILDREN WITH DIABETES: FROM A PILOT STUDY TO STANDARD CARE IN NORTHERN GERMANY

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Background:
The introduction of continuous glucose monitoring and the transfer of this data to medical clouds has created the basis for telemedical consultation in diabetology. Statistical pre-evaluation and graphical representation form the basis for consultation in both diabetes outpatient clinics and virtual settings.
Material and Methods:
As part of a randomized, controlled long-term study called VIDIKI (virtual diabetes care for children with diabetes), qualitative and quantitative research methods were used to investigate the effect of monthly video consultations in 240 children from northern Germany. With the onset of the COVID pandemic, the study was extended by one year. More than 5,000 video consultation appointments were conducted from 2017-2021.
Results:
Video consultations proved to be a form of counseling that was very well received by families and young people. A significant positive effect on metabolic status could only be demonstrated after one year, but parents' satisfaction with the therapy increased significantly and the burden on mothers decreased significantly in the comparative study phase.
Conclusions:
The results of the VIDIKI studies have been incorporated into guidelines in Germany. In phases with a high demand for counseling, telemedical forms of counseling should also be offered to families. Since 2023, video consultations for children have been available as an alternative care model to regular care for those insured by certain statutory health insurance companies.
Keywords: *children, diabetes care, outpatient clinic, telemedicine, video consultation*
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P.L. 26
MESANGIAL CELLS IN DIABETIC KIDNEY DISEASE

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Mesangial cells (MCs) injury is a significant pathogenic mechanism in the development of diabetic kidney disease (DKD), via oxidative stress, mitochondrial dysfunction, dysregulated lipid metabolism and epigenetic modifications.

MCs form a supportive network that holds the glomerular capillaries in place, preventing them from collapsing or moving freely within the glomerulus. Along with endothelial cells and podocytes, MCs are essential for the formation and integrity of the glomerular tuft. As modified smooth muscle cells, they contain actin and myosin. Exposure of MCs to AngII leads to their contraction, decrease of the GBM surface area and a lower glomerular filtration rate (GFR). MCs are also helping regulate blood flow through the glomerulus, similar to how vascular smooth muscle cells control flow in arterioles. Zimmerman, in 1933 described the mesangium with cells and extracellular matrix, and in 1962 Palade's group described the phagocytic properties of these cells. They can take up macromolecules and immune complexes from the glomerular space, contributing to cellular immune responses within the glomerulus. Also, MCs produce and remodel the extracellular matrix (ECM) components that form the mesangium, such as proteins like collagen IV, fibronectin, and laminin. When stressed, MCs secrete various cytokines, growth factors, and inflammatory mediators that play a role in glomerular homeostasis and are involved in inflammation and fibrosis during disease states. One of the most important roles of these MCs, is that of a signaling hub within the glomerulus, communicating with other glomerular cells to maintain overall glomerular health and function. However, these functions can be altered in glomerular injury, leading to MC hypertrophy, proliferation and ECM expansion, as well as "pathogenic cross-talk". The stressors identified are a) metabolic, i.e., hyperglycemia and advanced glycosylated end-products (AGEs); b) immunologic; c) hemodynamic; d) inflammatory, oxidative stress being the common pathway for amplifying injury and inflammatory signaling.

In hyperglycemia, MCs exhibit a rise in *Fyn* (gene encoding for Fyn kinase, with subsequent decrease of catalase expression), a decrease in Nrf2 (known transcription factor/protein, Nuclear factor E2-related factor; thus leading to a decrease in antioxidant gene expression), and inhibition of FoxO1 (another transcription factor/protein, Forkhead box O1; leading to inhibition of autophagy). ECM expansion leads to hypoxia, rise in HIF (hypoxia-induced factor)-1α, resulting in cell proliferation. The MCs can defend themselves against these stressors through autophagy, modulation of survival signaling pathways, and secretion of protective cytokines and growth factors, via several intracellular signaling pathways. Many studies have shown the multifaceted role of the JAK/STAT (Janus Kinase/Signal Transducer and Activator of Transcription) signaling pathway and the necessity of combined multi-drug and multi-mode treatment in DKD. Also, the cross-talk mediated by exosomes produced by MCs exposed to hyperglycemia, can be another therapeutic target in prevention of DN. In addition, diabetic nephropathy (DN) being a complication mediated by the "pathogenic cross-talk" between JAK/STAT and transforming growth factor (TGF)-β1/Smad family of transducer

proteins, targeting downstream TGF-β/Smad signaling (i.e., by inhibiting STAT3 acetylation, or by overexpressing Smad7-dependent fibrosis), may represent a novel and effective strategy for the treatment of DN.

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P.L. 27

SIMILARITIES AND DIFFERENCES IN THE OUTPATIENT CARE OF CHILDREN WITH TYPE 1 AND TYPE 2 DIABETES

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Background:

In Germany, the number of children with type 1 diabetes is increasing steadily, as is the number of children with type 2 diabetes. A particularly sharp increase in the incidence of type 2 diabetes was observed during the coronavirus pandemic, with more girls affected. The guidelines provide clear recommendations for the treatment of onset of type 1 and type 2 diabetes.

However, long-term outpatient care for both diseases differs in terms of prevention strategies, the basic treatment concept, the necessary specialist staff, and medication strategies, and, in the case of type 2 diabetes, surgical procedures if necessary.

Preventive strategies and various courses of type 2 diabetes are presented and discussed using case studies.

Keywords: *children, diabetes care, prevention, type 2 diabetes, type 1 diabetes*

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P.L. 28

SGLT2 INHIBITORS FOR THE THERAPY OF TYPE 2 DIABETES MELLITUS IN CHILDREN AND ADOLESCENTS

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Type 1 diabetes mellitus is the most prevalent chronic diseases diagnosed in children. Type 2 diabetes, once rare at a young age, has been increasing in frequency in parallel with childhood obesity. It typically manifests after puberty, with the highest rate between age 15 and 19 years. Though common in some ethnic groups, it is still quite rare in Caucasians. However, its incidence is expected to increase, together with the raise in the number of children with obesity.

Being known that cardiovascular diseases determine the death of more than two thirds of the elderly with Type 2 diabetes, current guidelines recommend an early and aggressive control of all the risk factors for atherosclerosis, including hyperglycemia. The prescription of the antihyperglycemic drugs that have proven to be cardio- and nephroprotective in clinical trials (i.e., SGLT2 inhibitors and GLP-1 receptor agonists) is recommended by the international guidelines for the adults and is more and more prevalent.

SGLT2 inhibitors are efficient in controlling blood glucose, have a low risk of hypoglycemia, and in adults proved to significantly reduce renal events and the hospitalizations for heart failure. In patients with known cardiovascular disease, they significantly reduce mortality, as well. In other words, the drugs from this class increase the life expectancy of the adults with type 2 diabetes and improve its quality.

There is less experience regarding the therapy of type 2 diabetes in children, as compared to the adults. Several classes of antihyperglycemic drugs are approved for use in these cases,

with emphasis on metformin, insulin, SGLT2 inhibitors and GLP-1 receptor agonists.
Clinical trials with SGLT2 inhibitors have been performed in children, as well. However, due to the rarity of the disease in this age group, the number of patients included is much lower than in adult trials. They have proven some beneficial effects: good glycemic control, low risk of hypoglycemia, good safety profile, and ease of use. However, long-term influence on major adverse cardiovascular and renal events is still to be proven. Real-life studies are expected to provide some more insights. However, till additional data is gathered, SGLT2 inhibitors are not considered by the regulatory boards to have benefits regarding nephro- and cardioprotection in children.

Keywords: *children; protection; SGLT2 inhibitors; type 2 diabetes mellitus*

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ORAL PRESENTATION:

O.P. 01

FACTORS ASSOCIATED WITH LOW ALKALINE PHOSPHATASE AND ITS ROLE IN DETECTING HYPOPHOSPHATASIA IN 15.031 ROMANIAN CHILDREN

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Background and Aims: Generally, elevated alkaline phosphatase (ALP) is regarded as a red flag for bone disease, whereas low values tend to be underrecognized. Nevertheless, reduced ALP is linked to several conditions that affect bone turnover, including hypothyroidism, anemia, cancer, arthritis, malnutrition, and hypophosphatasia. Our objective was to investigate factors associated with low alkaline phosphatase in children and to assess its usefulness in identifying hypophosphatasia. In addition, we examined factors associated with elevated ALP.
Material and methods: A large cohort of Romanian children had serum alkaline phosphatase levels assessed in the laboratory of a tertiary Children's Hospital in Timișoara, Romania, between 2014–2025. Age- and sex-specific cut-offs were applied to define low and high ALP values (<1st percentile and >99th percentile, respectively). ALP category (low/normal/high) was retrospectively analyzed in relation to age, sex, environment, diagnosis at the time of ALP testing, and fracture history.
Results: A total of 15,031 individuals (27,310 ALP measurements) were included, predominantly from western Romania. The median age was 6.61 years (IQR 1.64–12.8). Low ALP occurred in 595 children (1,010 measurements), 28 of whom had fractures. After a detailed review of medical records, one child remained without a plausible explanation for low ALP; however, this individual did not meet the clinical criteria for hypophosphatasia.
Conclusion: Low ALP was found in 3.9% of children (3.6% of measurements), while elevated ALP was observed in 4.2% of measurements. Factors associated with low ALP included age under 12 years, male sex, and a history of cancer. Among children with both low ALP and fractures, none fulfilled criteria for hypophosphatasia.
Keywords: *low alkaline phosphatase, hypophosphatasia, children, age, gender*

O.P. 02

HYPERANDROGENEMIA IN A TEENAGER: IS IT ONLY POLYCYSTIC OVARY SYNDROME?

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Background

Hyperandrogenic status and menses disturbances are typically associated with polycystic ovary syndrome or congenital adrenal hyperplasia (late-onset) in most non-tumour-related cases that are firstly identified at teenagers and young adults. In exceptional circumstances, hyperandrogenaemia is related to adrenal causes involving a hormonally active tumour such as an adrenal cancer. [1-5]

Aim

Our objective was to introduce a complex case that was diagnosed as polycystic ovary syndrome during teenager years and then confirmed with an androgen-producing adrenal tumour with malignant profile.

Material and Methods

This is a case report. Retrospective imaging, endocrine and surgical data are recorded amid a multidisciplinary practice in a tertiary centre of endocrinology (university hospital).

Results

This is a 17-year-old female who experienced menstrual disturbances (a menstrual delay from 2 months to 6 months) since the age of 16 years. She associated acne, tendency to alopecia, and obesity (a body mass index between 30 and 37 kg/sqm, despite restrictive diet and intense physical exercise). She was identified with a high total testosterone of 5.42 nmol/L (normal level below 1.67 nmol/L), increased dehydroepiandrosterone-sulphate (DHEA-S) of 604 μg/dL (normal ranges between 148 and 407 μg/dL), and normal 17-hydroxyprogesterone. The menses resumed under oral contraceptives, which she stopped after two years. She was also identified with insulin resistance based on an increased value of Homeostatic Model of Insulin Resistance (HOMA-IR) of 4 (insulin resistance is defined at a value above 2 to 2.5). While she experienced amenorrhea for another few months and declined further medication, she suffered an acute abdominal pain that was confirmed as biliary crisis (without gallbladder stones). At that point, she was investigated

as an emergency (via abdominal ultrasound), and a right adrenal mass was identified. Magnetic resonance imaging was later performed and showed an adrenal tumour of 7.1 cm maximum diameter (with a well-defined shape and small cystic areas within the tumour). She underwent an open right adrenalectomy with a rapid post-surgery recovery and a short hospital stay. Pathological report pointed out a tumour of 8.2 cm, which was confirmed as being an adrenocortical carcinoma of oncocytic type (with malignant behaviour based on Lin-Weiss-Bisceglia score with two major criteria). Post-operatory, the circulating androgens normalized, but the menses did not resume. Computed tomography was performed one month after surgery, and showed no disease relapse. Further mitotane is planned to the initiated with a close surveillance.

Conclusions

In this case, the hyperandrogenaemia that was considered as part of the traditional landscape in the polycystic ovary syndrome might have been caused by the adrenal tumour with non-syndromic presentation. This particular histological type of adrenal malignancy is very rare and the prognosis varies. The diagnosis at this young age is exceptionally reported. The tumour is distinct from oncocytoma, which displays a clearly benign behaviour.

Keywords: *adrenal, computed tomography, magnetic resonance imaging, mitotane, ovary*

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O.P. 03
PARTICULARITIES OF INSULIN PUMP THERAPY IN YOUNG CHILDREN WITH TYPE 1 DIABETES

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Background and Aims:

Type 1 diabetes incidence at young ages is increasing. The condition requires lifelong insulin treatment, comprehensive education and intensive effort in order to control glycemia. Rigid dosage on injection devices, psychoemotional pain-related stress, inflexible meal schedules make insulin pump therapy a suitable and growing alternative [1,2,3,4] Early pump initiation is associated with sustained glucose control.[5]

Material and Methods:
Data from files of 58 children aged 1-7 years old treated in clinic were collected. Treatment type, motive of choice, insulin dosage, metabolic parameters were analyzed.
Results:
Children had an average age of 4.9 years (SD 1.9), mean diabetes duration was 2.3 years (SD 1.6), 69% were using a pump. Mean diabetes duration at pump initiation was 1.6 years (SD 0.6). Main reasons to start CSII were injection related stress, hypoglicemia, flexibility in dosing insulin and HbA1c. The most used model was Omnipod Dash, thighs and buttocks were the most common site for pump insertion. Mean pump use duration (at data collection point) was 2.51 years (SD=1.4). Mean HbA1c on pump was 6.34% versus 6.89% on pen. Best results were obtained at age 0-4 yo (mean HbA1c 6.32%) and first 2 years since diagnosis (mean HbA1c 6.12%). Pump therapy performed better on mean time in range (71.9%) and hypoglicemia (2.8%) compared to multiple daily injections. Normal growth, height and weight, was maintained in both children with pump and MDI.
Conclusions:
In young children glucose control can be achieved more effectively and safely using insulin pumps. Greater flexibility in dosing, better glucose control, fewer hypoglicemia events are main benefits of this therapy. The need for skilled health professionals and easy ketosis are limits of this therapy.
Keywords: *children, insulin, ketones, pump therapy*
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O.P. 04

PREVALENCE OF METABOLIC SYNDROME IN CHILDHOOD BRAIN TUMOR SURVIVORS

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Background
Childhood brain tumor survivors (CBTS) are at increased risk of developing late metabolic complications secondary to multimodal therapy. Metabolic syndrome (MetS) represents a clinically relevant cluster of cardiometabolic risk factors in this population. Cranial radiotherapy (CRT) can disrupt hypothalamic–pituitary regulation, predisposing survivors to dyslipidemia, obesity, and insulin resistance.
Objective
This study aimed to determine the prevalence of MetS in long-term CBTS compared with healthy controls and to evaluate treatment- and dose-related predictors of metabolic alterations.
Material and method: A retrospective case–control study included 52 CBTS and 41 age- and sex-matched controls. Anthropometric, biochemical, and treatment data were extracted from medical records. MetS was defined according to International Diabetes Federation (IDF) criteria. Between-group comparisons assessed metabolic and anthropometric parameters, and associations between treatment factors and metabolic outcomes (TG/HDL ratio, BMI z-score, HOMA-IR) were examined using multiple linear and logistic regression. Radiation dose–response relationships and correlations were evaluated using continuous dose models and Spearman coefficients.
Results: MetS prevalence was 9.6% in CBTS (95% CI 2.7–23.1) and 0% in controls. Among children <10 years, 69% exhibited clustering of ≥2 MetS components. CBTS demonstrated significantly higher TG/HDL ratios than controls (median 1.48 [0.93–3.20] vs. 1.10 [0.80–1.75], p = 0.0405). CRT was independently associated with elevated TG/HDL (β = 1.07, 95% CI 0.25–1.89; p = 0.011), and each additional Gy of cranial dose increased TG/HDL by 0.03 units (β = 0.030, 95% CI 0.014–0.046; p < 0.001). No treatment or dose effect was observed for BMI z-score or HOMA-IR.
Conclusion: MetS and related dyslipidemia are more prevalent in CBTS than in healthy peers. Cranial irradiation exerts a dose-dependent and selective effect on lipid metabolism, highlighting the need for early lipid screening and tailored metabolic follow-up in survivors exposed to CRT. These findings are consistent with previous studies.
Keywords: *Childhood brain tumor survivors; metabolic syndrome; survivorship.*
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O.P. 05

THE CHALLENGING JOURNEY FROM SEVERE NEONATAL HYPOGLYCEMIA TO STABLE GLYCEMIC CONTROL: A CASE OF CONGENITAL HYPERINSULINISM

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Background and Aims

Neonatal hypoglycemia is a potentially life-threatening condition that requires rapid recognition and prompt initiation of targeted treatment in order to prevent acute complications and long-term neurological impairment. Among the various etiologies, congenital hyperinsulinism represents the most common cause of persistent and recurrent hypoglycemia in neonates and young infants, resulting from inappropriate insulin secretion despite low plasma glucose levels.

Material and Methods

We present a complex case of severe neonatal hypoglycemia due to congenital hyperinsulinism, emphasizing diagnostic challenges, extensive genetic exclusion, and favorable evolution under diazoxide therapy.

Results

We report the case of a male infant aged 4 months and 3 weeks, admitted for clinical and biological reevaluation. The first hospitalization occurred at 15 days of life for severe hypoglycemia and convulsive syndrome. He was born at 39 weeks of gestation by cesarean section, birth weight 2900 g, from a monitored pregnancy. At 5 days of life, the patient presented with feeding refusal, lethargy, perioronasal cyanosis, severe hypoglycemia (<5 mg/dl), metabolic acidosis, and hyponatremia, requiring neonatal intensive care. Two tonic-clonic seizures were documented, with subsequent favorable neurological evolution.

Diagnostic work-up included hormonal assays during hypoglycemia, metabolic and endocrine evaluation, and extensive genetic testing. Whole exome sequencing and targeted next-generation sequencing excluded pathogenic variants in genes associated with congenital hyperinsulinism and related syndromes, including ABCC8, KCNJ11, AKT2, CACNA1C, CACNA1D, CREBBP, EP300, FOXA2, GCK, GLUD1, HADH, HNF1A, HNF4A, INSR, KDM6A, KMT2D, MAFA, MAGEL2, NSD1, PHOX2B, PMM2, SLC16A1, TRMT10A, as well as the non-coding regulatory region of HK1.

Biochemical evaluation revealed detectable insulin levels during hypoglycemia (2.5 µUI/ml at plasma glucose 33 mg/dl and 4 µUI/ml at 48 mg/dl) and elevated C-peptide levels, consistent

with congenital hyperinsulinism according to international guidelines. Associated adrenal insufficiency was documented (high ACTH 115.8 ng/ml, low cortisol 2.1 µg/dl), prompting initiation of oral hydrocortisone therapy (8 mg/m² body surface). Diazoxide therapy was initiated at 5 mg/kg/day and gradually titrated up to 10.4 mg/kg/day, resulting in effective glycemic control. At 4 months of age, continuous glucose monitoring demonstrated that 98% of glucose values were within the target range (70–180 mg/dl) over the preceding 30 days, with no episodes of hypoglycemia <70 mg/dl while on Diazoxide 4.2 mg/kg/day orally. Hydrocortisone therapy was gradually discontinued as serum cortisol increased to 7.7 µg/dl, with stable glycemic control. Growth and neurodevelopment were age-appropriate, and no hypoglycemic events occurred.

Conclusions

This case highlights the diagnostic complexity of neonatal hypoglycemia with congenital hyperinsulinism in the absence of identifiable genetic mutations. Comprehensive genetic exclusion, individualized diazoxide therapy, and continuous glucose monitoring are essential for achieving optimal metabolic control and favorable neurological outcomes. Although glycemic control is currently stable, the ultimate classification as transient or permanent will be determined over time, requiring ongoing monitoring and follow-up.

Keywords: *congenital hyperinsulinism; diazoxide; genetic testing; neonatal hypoglycemia*

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O.P.06

KETOGENIC DIET FOR DRUG-RESISTANT EPILEPSY

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Epilepsy is a neurological disorder affecting both children and adults. The effects of the ketogenic diet on seizure control have been observed since the 1920s; however, over the past three decades it has increasingly been adopted as a therapeutic option for drug-resistant epilepsy. In 2018, clinical

guidelines was elaborated under the supervision of Dr. Eric Kossoff, clearly outlining the indications and contraindications for the use of this dietary therapy in drug-resistant epilepsy management. The ketogenic diet is characterized by high fat intake, low carbohydrate consumption, and moderate protein content, designed to induce a state of ketosis. During ketosis, ketone bodies are utilized as the primary energy source instead of glucose, a metabolic shift shown to contribute to improved seizure control. Currently, four main variants of the ketogenic diet are in use. The selection of a specific approach should be tailored to the patient's nutritional requirements and dietary habits in order to achieve optimal therapeutic outcomes.

Although the ketogenic diet is an effective therapeutic option for certain forms of epilepsy, limitations such as variable patient response, potential adverse effects, and adherence challenges remain. Ongoing monitoring and individualized adjustments are therefore required, and a multidisciplinary team - including neurologists, dietitians, and nurses - is essential to optimize clinical outcomes.

Keywords: *dietitian, drug-resistant epilepsy, ketogenic diet*

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O.P. 07

EXCESSIVE COW'S MILK INTAKE AS A CAUSE OF SEVERE IRON DEFICIENCY ANEMIA AND PROTEIN-LOSING ENTEROPATHY IN TODDLERS: TWO CASE REPORTS AND DIAGNOSTIC CHALLENGES

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Background and Aims

Protein-losing enteropathy (PLE) is not a single disease but a clinical syndrome characterized by excessive loss of serum proteins through the gastrointestinal tract, leading to hypoproteinemia and hypoalbuminemia. In toddlers, excessive cow's milk intake has been increasingly recognized as a potential trigger of severe iron deficiency anemia (IDA), food protein-induced enteropathy, and secondary PLE. Periorbital or peripheral edema may be the presenting sign, often mimicking nephrotic syndrome. This presentation aims to highlight the association between excessive cow's milk consumption, severe IDA, and exudative enteropathy in young children, emphasizing diagnostic challenges and therapeutic outcomes.

Material and Methods

We present two pediatric cases (female, aged 1 year 3 months and 1 year 9 months) admitted for pallor and/or edema. Detailed dietary histories revealed high intake of cow's milk (300–450 mL/day in the first case; >1 L/day in the second). Clinical evaluation included anthropometric assessment and examination for edema and nutritional status. Laboratory investigations comprised complete blood count, ferritin, serum protein and albumin, inflammatory markers, metabolic profile, renal function, urine protein/creatinine ratio, stool examination, fecal alpha-1 antitrypsin, total and specific IgE to cow's milk proteins, immunoglobulin levels, thyroid function tests, and cardiac ultrasound (in case 2). Differential diagnoses included malabsorption, chronic blood loss, nephrotic syndrome, and impaired hepatic synthesis. Management consisted of albumin infusion (case 1), diuretics, elimination of cow's milk proteins, introduction of amino acid–based or extensively hydrolyzed formula, and oral iron supplementation with vitamin support. Patients were followed clinically and biologically at 1 week, 1 month, and 3–6 months.

Results

Both patients presented with severe microcytic hypochromic anemia and hypoproteinemia; the first case also had significant hypoalbuminemia with palpebral and lower limb edema. Inflammatory markers were negative, and renal protein loss was excluded (normal urine protein/creatinine ratio). Specific IgE to cow's milk proteins was positive in case 1. Partial immunoglobulin deficiencies were identified in both patients. Cardiac evaluation in case 2 revealed left ventricular hypertrophy, likely secondary to chronic severe anemia. Following elimination of cow's milk proteins and initiation of iron therapy, both children showed rapid clinical improvement, resolution of edema (case 1), progressive correction of anemia and serum protein levels, and normalization of cardiac findings at follow-up. No recurrence of edema occurred after strict dietary exclusion.

Conclusions

Excessive cow's milk intake in toddlers may lead to severe iron deficiency anemia and secondary protein-losing enteropathy, sometimes presenting with edema and mimicking nephrotic syndrome. Also, children with severe IDA should always be evaluated for PLE even in the absence of clinical signs of hypoalbuminemia because this condition might be more frequent than diagnosed. Early recognition and management—elimination of cow's milk proteins and appropriate iron supplementation—result in favorable clinical and biological outcomes. Increased awareness among pediatricians can prevent delayed diagnosis and avoid unnecessary investigations.

Keywords: *Cow's milk allergy; Excessive cow's milk intake; Hypoproteinemia; Iron deficiency anemia; Protein-losing enteropathy;*

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O.P. 08

IMPACT OF HYPOCALORIC VERSUS ISOCALORIC DIETS WITH DIFFERENTIAL MACRONUTRIENT DISTRIBUTION IN CHILDREN WITH OBESITY

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Dietary strategies for the management of overweight and obesity in both adults and children broadly include calorie-restricted diets, which induce an explicit energy deficit, and isocaloric diets, where total energy intake is maintained but macronutrient distribution is modified to influence metabolic responses and satiety.[1] While caloric restriction has traditionally been considered the cornerstone of weight management, growing evidence suggests that macronutrient composition may independently affect insulin sensitivity, lipid metabolism, appetite regulation, and long-term adherence.[2] In adult populations, randomized controlled trials indicate that when energy intake is reduced, weight loss is largely determined by the degree of caloric deficit and behavioral adherence, with relatively small long-term differences between low-fat, low-carbohydrate, or high-protein dietary patterns.[3] Pragmatic trials comparing isocaloric or near–isocaloric diets differing in macronutrient distribution have demonstrated comparable weight loss when diet quality is emphasized, suggesting that food composition and sustainability may be as relevant as macronutrient ratios alone.[4,5]. Meta-analyses report modest short-term advantages of low-carbohydrate diets for weight reduction and triglyceride levels, although these effects tend to attenuate over time, with heterogeneous impacts on LDL cholesterol depending on dietary fat quality and baseline cardiometabolic risk.[6] In pediatric populations, nutritional interventions require special consideration due to ongoing growth, neurodevelopment, and the risk of disordered

eating behaviors.[7] Current evidence supports structured, family-centered dietary approaches that prioritize nutritional adequacy and behavioral change rather than aggressive caloric restriction.[7] Nonetheless, controlled trials in adolescents with obesity have shown that carbohydrate-restricted diets, even when relatively isocaloric, can lead to short-term weight loss comparable to or greater than hypocaloric low-fat diets, when implemented under close medical supervision.[8,9] Studies examining macronutrient quality, such as low–glycemic index diets, suggest potential metabolic benefits in children, although results remain inconsistent and highly dependent on adherence and study design.[10] Overall, existing literature indicates that caloric restriction remains a key driver of weight loss across age groups, while isocaloric dietary strategies with tailored macronutrient distribution may offer metabolic advantages and improved adherence in selected individuals. In children and adolescents, dietary modulation should prioritize safety, developmental needs, and long-term behavior change, whereas in adults, macronutrient distribution may be strategically applied to optimize cardiometabolic outcomes within an energy-balanced or mildly hypocaloric framework. [1,7]

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O.P. 09
MEDICAL MANAGEMENT OF A NEWBORN WITH SUSPECTED INHERENT METABOLIC DISEASE

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Objectives
Through this paper, we aim to specify the correct medical attitude in the case of a newborn with suspected congenital metabolic disease

Material and method
The neonatal clinical picture of an inborn metabolic disease falls into a clinical-paraclinical entity called neonatal neurological distress.
This includes five categories. Each category has a specific clinical picture and a clear etiology. Correct identification of the type of neonatal neurological distress allows the rapid

Types	Clinical tyoe	Most usual diagnosis
I	Neurological deterioration, « intoxication » type abnormal movements hypertonia	MSUD
II	Neurological deterioration, « intoxication » type dehydration Neurological deterioration, « energy deficiency » type with liver or cardiac symptoms	Organic aciduria, Ketolysis defects FAO & ketogenesis defects
III	Neurological deterioration, « energy deficiency » type , polypnea, hypotonia, lactic acidosis	Congenital lactic acidosis
IVa	Neurological deterioration, « intoxication » type , moderate hepatocellular disturbances, hypotonia, seizures, coma	Urea cycle defects, HHH syndrome, FAO defects, PA, MMA, TVA
TVb	Neurological deterioration, Seizures, myoclonic jerks, severe hypotonia	
Va Vb Vc Vd	Recurrent hypoglycemia with hepatomegaly Hepatomegaly, liver failure, joundice Hepatomegaly, cholestatic jaundice Hepatosplenomegaly, storage signs	Glycogenosis Galactosemia, tyrosinemia I Inborn errors of bile acids Lysosomal disease

Saudubray, van den Berghe, Walter. Inborn Metabolic Diseases : Diagnostic and Treatment, Fifth Edition, 2011

establishment of the etiological diagnosis, thus leading to the initiation of specific treatment and the prevention of sequelae.

Results

Currently, five types of neonatal neurological distress are known.

Each of them has a specific clinical picture. Establishing the type is done by urgently performing three investigations, namely

- blood gas and lactate
- glycemia
- ammonemia

Depending on the results, the type of neonatal neurological distress is established and the specific investigation plan is initiated.

Conclusions

Since the therapeutic approach is different depending on the distress, and the chances of recovery decrease significantly if the diagnosis is not established quickly, the correct classification of the form that allows the initiation of the correct treatment is essential.

Keywords*-inborn metabolic disease, neonatal neurological distress,*

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O.P. 10

NUTRITIONAL INTERVENTION AS A CENTRAL COMPONENT OF GLP-1 RECEPTOR AGONIST THERAPY IN CHILDREN AND ADOLESCENTS

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Background:

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are increasingly used in pediatric obesity management. However, appetite suppression during critical growth periods may

predispose children and adolescents to lean mass loss, suboptimal bone mass accrual, and functional micronutrient deficiencies. A structured nutritional strategy is therefore essential to ensure treatment safety and optimize long-term outcomes.

Methods:

At YUNO Clinic – EASO COMS Center, we implemented a standardized multidisciplinary protocol for children and adolescents aged ≥6 years with persistent obesity (BMI ≥ +2 SDS) after unsuccessful lifestyle intervention. The evaluation team includes pediatric endocrinology, pediatrics, dietetics (central coordinating role), pediatric cardiology, and psychology.

Baseline assessment comprises growth trajectory analysis, pubertal staging, metabolic screening, and systematic evaluation of micronutrient status (vitamin D, iron, vitamin B12, folate, calcium, zinc), along with eating behavior assessment and implementation of a hunger–satiety awareness scale.

Nutritional intervention precedes GLP-1 RA initiation and includes a protein intake of 1.6–1.8 g/kg ideal body weight or lean body mass, balanced protein distribution (20–30 g/meal), structured meal timing (3 meals and 1–2 snacks/day), prioritization of complex carbohydrates, adequate fiber intake, and hydration. Ongoing monitoring targets preservation of lean mass and bone health, ensuring calcium intake of 1000–1300 mg/day, vitamin D supplementation (1000–2000 IU/day), and regular weight-bearing exercise.

Expected Results:

A reduction in BMI SDS ≥0.25 over 6–12 months, maintenance or increase of lean body mass, improved metabolic parameters, normal pubertal progression, and development of sustainable eating behaviors.

Conclusions:

In pediatric patients, GLP-1 RA therapy should not be considered solely a pharmacologic weight-loss intervention. Its safety and efficacy depend on early, structured, and continuous nutritional integration within a coordinated multidisciplinary framework, with active monitoring of muscle and bone health.

O.P. 11

PEDIATRIC NUTRITION – INFLAMMATION CROSSTALK: A NETWORK MEDICINE PERSPECTIVE

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Background and aims:

Pediatric overweight and obesity constitute a heterogeneous condition. While some children maintain a metabolically healthy phenotype (MHO), others manifest metabolically unhealthy (MUO) traits characterized by low-grade inflammatory activation. Increasing evidence suggests that this reflects in a coordinated dysregulation of immune-metabolic signaling networks rather than isolated biomarker abnormalities. Conventional analyses frequently focus on individual inflammatory markers and fail to capture the systems-level organization of these pathways.

Materials and Methods:
A previously reported 40-gene transcriptomic signature differentiating MUO from MHO children with overweight/obesity identified by Limma analysis was retrieved from the published data. Because one gene was reannotated, this resulted in a 39- gene set used for network reconstructions.
A functional interaction (FI) network was constructed using Reactome FIViz in Cytoscape (v.3.10). The network was analyzed as undirected using Network Analyzer. Community detection was performed via Reactome FI clustering, and pathway enrichment analysis was conducted with false discovery rate (FDR) correction.
In the inflammation-enriched module, centrality metrics (Degree and Betweenness Centrality) were calculated to identify high-centrality bridge regulators. Tissue-level protein expression plausibility of prioritized genes was assessed using the Human Protein Atlas (HPA). Nutritional model integration was performed at the pathway level by mapping established roles of vitamin D signaling and choline-related metabolism onto enriched inflammatory pathways.
Results:
The reconstructed interactome comprised 67 nodes and 127 edges, forming a single connected component with modular organization (modularity= 0.4594). One module (Module1; N= 20 nodes) showed significant enrichment for innated immune pathways, including Toll-like receptor (TLR) cascades (FDR= 0.0077), inflammasome-mediated interleukin-1 family signaling (FDR= 0.0076), and TNFR2 non-canonical NF-kB signaling (FDR= 0.0098).
Topological prioritization in this inflammation core identified five high-centrality bridge regulators: UBA52 (Degree= 16; Betweenness = 0.23), UBC (14; 10.16), TP53 (10; 0.09), TOMM20 (9; 0.04) and CUL3 (9; 0.03). These genes serve as structural connectors between inflammatory signaling and broader cellular processes.
HPA data confirmed protein expression of these genes supporting their biological plausibility. Enriched pathways converged on NF-kB-related and cytokine signaling cascades. Vitamin D signaling and choline-related metabolic pathways intersect with these axes at the pathway level, suggesting potential nutritional interaction points within the identified inflammation module.
Conclusions:
Metabolically unhealthy pediatric obesity is characterized by a structured immune module centered on high-centrality regulatory bridges. Network reconstruction provides a system-level view of immune pathways in children with adverse metabolic profiles.
In silico modeling of these central bridge genes helps prioritize specific immune pathways for future mechanistic and clinical studies. Mapping nutritional pathways onto these network structures supports the hypothesis that targeted nutritional modulation could be explored as part of precision nutrition approaches in pediatric metabolic risk management.
Keywords: *Inflammasome, interactome, metainflammation, network medicine, precision nutrition.*
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O.P. 12

CATCH-UP GROWTH IN PEDIATRIC PRACTICE: THE ROLE OF NUTRITIONAL INTERVENTION

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Faltering growth refers to the inability to maintain an expected growth trajectory and represents a common clinical challenge in pediatric practice. It is identified through serial assessment of weight, weight-for-length or height, and body mass index using z-scores. A z-score between −2 and < −3 standard deviations (SD) defines moderate malnutrition, while values < −3 SD indicate severe malnutrition. Importantly, faltering growth may be detected before these thresholds are reached, as it reflects deviation from an individual growth trajectory rather than a static anthropometric deficit. Its etiology is multifactorial, including inadequate intake, increased metabolic demands, nutrient losses, organic disease, and psychosocial factors. Early identification of underlying causes enables timely and targeted intervention.

The primary objective of nutritional management is to achieve catch-up growth, defined as growth velocity exceeding age- and sex-specific medians after a period of faltering, facilitating recovery toward genetic growth potential. Clinically, this corresponds to weight gain approximately two to three times the expected rate for age, with progressive improvement in anthropometric indices.

To promote catch-up growth, energy and protein intake should exceed standard age-based recommendations and be tailored to severity and etiology. Energy requirements must be individualized according to the desired rate of weight gain and clinical status. Nutritional rehabilitation should be initiated gradually and adjusted based on inflammatory status and response. Adequate protein intake is essential to ensure positive nitrogen balance and appropriate

lean body mass accretion, supported by a protein-to-energy ratio of approximately 8.9–12%.
Optimization of oral nutrition is the first-line approach in mild-to-moderate cases, focusing on increased dietary energy density and oral nutritional supplements. When oral intake is insufficient, enteral nutrition should be initiated, while parenteral nutrition is reserved for cases in which enteral feeding is contraindicated or not feasible. Correction of micronutrient deficiencies is integral to therapy. Continuous monitoring of intake, tolerance, biochemical parameters, and growth response is necessary to guide adjustments and ensure safe recovery.
In severe malnutrition or prolonged inadequate intake, rehabilitation must begin cautiously at approximately 40–50% of estimated energy requirements, with gradual increases. Close electrolyte monitoring is mandatory to prevent refeeding syndrome, a potentially life-threatening condition characterized by hypophosphatemia, hypokalemia, hypomagnesemia, and thiamine deficiency, reported in up to 7.4% of pediatric intensive care cases.
Catch-up growth is a key indicator of short-term therapeutic efficacy and long-term metabolic and developmental outcomes, underscoring the importance of early, individualized, and carefully monitored nutritional strategies.

Keywords: *catch-up growth, faltering growth, malnutrition, nutritional intervention*

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O.P. 13

THE ROLE OF LIFESTYLE INTERVENTIONS IN THE POST-TREATMENT CARE OF CHILDREN WITH ONCOLOGICAL DISEASES

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Background and aims

Therapeutic advances in recent decades have led to a significant increase in survival rates in pediatric cancer, currently exceeding 75% in most European countries. In the European Union, approximately 13,800 children and adolescents aged 0–19 years are diagnosed with cancer each year, while in Romania more than 400 new cases are reported annually. The growing number of survivors is associated with long-term complications, among which obesity and metabolic disorders are frequently observed. Studies show that the prevalence of overweight and obesity in children in remission after oncologic treatment may reach 40–50%, influenced by multiple factors such as corticosteroid therapy, cranial radiotherapy, endocrine dysfunction, decreased physical activity, and changes in eating behavior. The aim of this paper is to highlight the role of lifestyle interventions, particularly nutritional intervention and family education, in the post-treatment care of children with oncological diseases and in the prevention of long-term metabolic complications.

Material and methods

This paper represents a narrative analysis based on data from the scientific literature and clinical experience in nutritional counseling of children with a history of oncological diseases. The mechanisms involved in the development of post-treatment obesity and metabolic disorders were analyzed, as well as the effectiveness of individualized nutritional interventions, dietary education, and multidisciplinary management. The importance of involving both the child and the family in the nutritional intervention was evaluated, together with the role of the registered dietitian in screening, assessment, monitoring, and long-term guidance. In addition, the need for standardized educational materials was analyzed, including the development of a guide dedicated to parents of children with oncological diseases, in order to facilitate access to accurate, clear, and evidence-based information.

Results

The analyzed data show that overweight and obesity are frequently encountered during remission and are associated with insulin resistance, decreased muscle mass, and endocrine changes secondary to oncologic treatment. Early and individualized nutritional intervention contributes to reducing the risk of metabolic complications and improves long-term adherence. Active involvement of the child in the nutritional intervention process reduces maladaptive eating behaviors and increases compliance with recommendations. Parental education proved essential, as misinformation may compromise the nutritional intervention. The use of scientifically validated educational materials and the development of a guide for parents of children with oncological diseases facilitate the implementation of recommendations and support effective collaboration between the family and the medical team.

Conclusions

Lifestyle interventions represent an essential component of long-term care in childhood cancer survivors. Early nutritional intervention, continuous monitoring, and proper family education can positively influence metabolic outcomes and quality of life. The registered dietitian plays an important role in preventing complications and in developing healthy eating behaviors. The development of educational guidelines for parents and the promotion of evidence-based information ar necessary to combat misinformation and to optimize the long-term management of these patients.

Keywords: *pediatric cancer, lifestyle interventions, nutrition, obesity, survivorship*

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O.P. 14

VEGETARIAN AND VEGAN DIETS IN CHILDREN: GROWTH, MICRONUTRIENT STATUS, AND BONE HEALTH OUTCOMES

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Background and Aims:

Plant-based diets are increasingly adopted in pediatric populations for ethical, environmental, and health reasons. This study aimed to synthesize recent evidence (2021-2026) on growth outcomes, micronutrient deficiencies, metabolic benefits, and bone health in children following vegetarian and vegan diets compared to omnivorous diets.

Material and Methods:

We conducted a comprehensive systematic review of recent systematic reviews and meta-analyses published between 2021 and 2026. Electronic databases (PubMed, Google Scholar, SciSpace) were searched using terms including "vegetarian diet," "vegan diet," "children," "growth," "micronutrients," "bone health," and "systematic review." Studies were included if they reported on pediatric populations (ages 0-18 years) following plant-based diets. Primary outcomes included anthropometric measures (height, weight, BMI), micronutrient biomarkers (vitamin B12, ferritin, 25(OH)D), metabolic parameters (lipid profiles), and bone mineral density. Quality assessment used GRADE criteria as reported in included systematic reviews

Results:

Thirteen systematic reviews encompassing 131 unique studies were analyzed. Lacto-ovo vegetarian diets generally supported normal growth parameters comparable to omnivorous diets, while vegan diets showed increased risk of underweight status and stunting. Meta-analysis demonstrated vegan children consumed protein at 3.54% energy lower than omnivores (95% CI: -5.08 to -2.00). Vitamin B12 deficiency was common in unsupplemented vegetarian and vegan children but corrected with supplementation. Despite higher reported dietary iron intake

in vegans (+8.01 g/1000 kcal), ferritin levels were consistently lower, indicating reduced iron stores. Lower 25(OH)D levels were observed in unsupplemented plant-based children. Favorable metabolic effects included lower total cholesterol and LDL-cholesterol in vegan children compared to omnivores. However, bone mineral content and density were consistently lower in vegetarian and especially vegan children, associated with reduced calcium and vitamin D intake. Evidence certainty was rated low to very low across most outcomes due to small sample sizes, cross-sectional designs, and heterogeneity in dietary patterns and supplementation practices.

Conclusions:

Plant-based diets can support adequate growth and development in children when carefully planned with appropriate supplementation, particularly for vitamin B12, vitamin D, and consideration of iron, zinc, and calcium. Vegan diets require more intensive monitoring due to higher risk of growth faltering and micronutrient deficiencies. Bone health concerns necessitate ensuring adequate calcium and vitamin D intake through fortified foods or supplementation. Pediatric endocrinologists should implement regular growth monitoring, annual laboratory screening (complete blood count, ferritin, vitamin B12, 25(OH)D), and referral to pediatric dietitians for individualized dietary planning. Future long-term prospective cohort studies are needed to assess fracture risk, peak bone mass attainment, and neurocognitive outcomes in children following plant-based diets.

Keywords: *bone health, children, micronutrients, vegan diet, vegetarian diet*

Table 1. Comparison of Key Nutrient Intakes and Biomarkers: Vegetarian/Vegan vs. Omnivore Children

Nutrient	Vegetarian/Vegan	Omnivore	Clinical Significance
Protein	↓ 3.54% energy	Reference	Monitor growth velocity
Iron (intake)	↑ Higher	Reference	↓ Ferritin despite ↑ intake
Vitamin B12	↓ 97 pmol/L	Reference	Supplementation required
Calcium	↓ Lower	Reference	Bone health concern ↓ 25(OH)D, ↓ BMC/BMD
Fiber	↑ Higher +8.01 (6.96 to 9.06)	Reference	Not assessed

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O.P. 15

THE ROLE OF THE DIETITIAN IN STEP-BY-STEP WEIGHT MANAGEMENT IN CHILDREN

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Background and aims
Childhood obesity represents a major public health concern worldwide and is associated with increased risk of metabolic, cardiovascular, and psychosocial complications. Evidence shows that nutritional and behavioral interventions implemented during childhood can reduce body mass index (BMI) and improve eating behaviors, especially when family involvement and multidisciplinary management are included. The prevalence of pediatric obesity continues to increase, and effective management requires structured interventions adapted to the child's age and family environment. The dietitian plays a key role in nutritional assessment, intervention planning, education, and long-term monitoring. The aim of this paper is to highlight the step-by-step role of the dietitian in the weight-loss process in children and to emphasize the importance of family education and interdisciplinary collaboration in achieving sustainable results.
Material and methods
This paper represents a narrative analysis based on scientific literature and clinical experience in nutritional counseling of children with overweight and obesity. The stages of dietetic intervention were analyzed, including the first interaction with parents, initial assessment, goal setting, gradual implementation of dietary changes, and follow-up monitoring. Studies show that interventions combining nutrition education, behavioral strategies, and parental involvement are more effective than dietary advice alone. The importance of communication adapted to the child's age, the use of practical recommendations, and the role of multidisciplinary collaboration were also evaluated.
Results
Clinical studies indicate that individualized nutritional interventions combined with behavioral counseling and family-based approaches can lead to significant reductions in BMI and improvements in eating habits in children. Initial assessment performed in a non-stigmatizing manner helps build trust and improves adherence to the intervention. Setting realistic short- and long-term goals allows gradual implementation of lifestyle changes adapted to the child and family. Parental education plays a crucial role, as misinformation may negatively influence the effectiveness of the intervention. Practical recommendations focused on balance rather than restriction increase acceptance and compliance. Regular monitoring and interdisciplinary collaboration support long-term success and reduce the risk of relapse.
Conclusions
The dietitian has a central role in the management of childhood obesity through proper assessment, nutrition education, and step-by-step lifestyle intervention. Family involvement,

correct parental guidance, and multidisciplinary cooperation are essential for successful weight management in children. Strategies based on flexibility, education, and sustainable behavioral change contribute to the development of healthy eating habits and long-term weight control.

Keywords: *childhood obesity, dietitian, nutritional intervention, family education, weight management*

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ORAL PRESENTATION ”JUNIOR DOCTORS”

J.D. 01

THE DIAGNOSIS OF PSYCHOGENIC POLYDIPSIA: RELIEF OR BURDEN? - CASE PRESENTATION

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Background and Aims:

Psychogenic polydipsia is a type of primary polydipsia that determines increased fluid intake due to disruptions of non-homeostatic influences in water balance. The main long-term complication is dilutional hyponatremia, which can lead to: nausea, vomiting, seizures, delirium, cognitive deficits, ataxia and falls, rhabdomyolysis and stress cardiomyopathy, increased risk of osteoporotic fractures and, in severe cases, osmotic demyelination syndrome.

Material and Methods:

We present the case of an 18-year-old adolescent, admitted to the Endocrinology Department with suspicion of Diabetes Insipidus, presenting polyuria and polydipsia of approximately 13L/day, with the onset of symptoms one month ago, with no electrolyte imbalances. A water deprivation

test was performed, with plasma osmolarity maintained within normal limits during the test and a discrete progressive increase in urinary osmolarity, followed by the administration of vasopressin, without significant modification of urinary density or volume. On psychiatric evaluation, emotional detachment with overlapping negative affect was noted, without anxious, depressive or psychotic elements and MRI did not reveal hypothalamo-pituitary lesions. From his personal history, we found that the patient was going through a demanding period with important exams, in a conflictive family setting. Fluid intake diminished following psychotherapy, with complete remission of symptoms after completion of high school graduation exams.

Tables no. 1: Differential Diagnosis of Psychogenic polydipsia:

	PRIMARY POLIDIPSIA			
DIABETUS INSIPIDUS	polydipsia in patients with **neurodevelopmental disorders** (autism, intellectual disabilities) and **chronic psychotic disorders** (schizophrenia, schizoaffective disorder, bipolar disorder and psychotic depression)	psychogenic polydipsia or compulsive water drinking (in obsessive-compulsive disorder, alcoholism, anxiety disorder, depression, and anorexia nervosa)	dipsogenic polydipsia (excessive thirst due to hypothalamic lesions caused by trauma, vascular or infiltrative diseases or patients with habitual polydipsia)	iatrogenic polydipsia, (medical advice or act that leads to increased fluid intake)
Polyuria due to impaired AVP secretion (central DI) or AVP resistance in the kidneys (nephrogenic DI)	Disruption of normal anterior hippocampus restraint of stress impacts lateral hypothalamic and nucleus accumbens, non-homeostatic modulators of water intake and motivated behaviors (relieve dysphoria)	Disruption of non-homeostatic influences on water intake	Disruption of homeostatic influences on thirst	Believe drinking in excess is healthy, promotes cognition and performance

Results:

Psychological stress is a risk factor for primary polydipsia in adolescents without psychiatric comorbidities, which may lead to disruption of non-homeostatic influences on water intake. Adolescents can often be overwhelmed by intensely conflicting emotions and needs, especially in environments with low psycho-emotional support, being exposed to the risk of developing dysphoria, that can progress to the development of dissociative symptoms and somatization. The remaining functional part tends to avoid stimuli that produce dysphoria, thus appearing emotional numbness, which in time leads to the maintenance of the emotional regulation disorder, affecting personal and social functioning. Drinking might reduce aversive states of arousal and function as 'primitive' coping behavior. Studies made on psychiatric patients with polydipsia show that resting-state functional connectivity of anterior hippocampus-hypothalamic circuit, involved

in the impact of psychological stress on neuroendocrine function and behavior is disrupted in polydipsic patients, in proportion to the severity of the social deficits of the polydipsic patients that present diminished oxytocin secretion, but for non-psychiatric patients there are poor data.
Conclusions: Although psychogenic polydipsia does not benefit from a specific treatment like diabetes insipidus, this condition can indicate emotional regulation disorders, providing the opportunity for their psychotherapeutic treatment and resolution, with a positive impact on the somatic, psycho-emotional and social development of adolescents.
Keywords: *adolescents, diabetes insipidus, primary polydipsia, psychological stress, water deprivation test.*
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J.D. 02

TYPE II AUTOIMMUNE POLYGLANDULAR SYNDROME IN A CHILD - CASE REPORT

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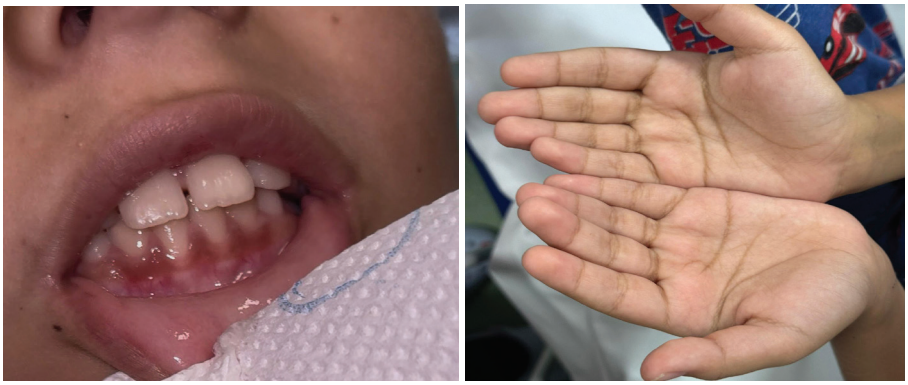
Background
Addison's disease is a rare disorder characterized by adrenocortical insufficiency resulting from dysfunction or destruction of the adrenal glands. Clinical manifestations are usually non-specific and subtle, often leading to delayed diagnosis and increased morbidity and mortality. Symptoms may develop gradually or acutely, as in an Addisonian crisis-a life-threatening condition associated with hemodynamic instability and electrolyte disturbances.[1,2,3] When Addison's disease occurs in association with other autoimmune conditions, autoimmune polyglandular syndromes (APS) should be considered. Type II APS is defined by primary adrenal insufficiency combined with

autoimmune thyroid disease and/or type 1 diabetes mellitus. Other associated autoimmune disorders include celiac disease, vitiligo, hypogonadism, and pernicious anemia.[4]

Case report

We present the case of a 9-year-old boy admitted for persistent anorexia, lethargy, myalgia, and nausea, which began two weeks prior to admission following SARS-CoV-2 infection. Clinical examination revealed poor general condition, thready pulse, cold extremities, persistent skin tenting, discolored and dehydrated mucous membranes, and cutaneous hyperpigmentation, more pronounced in flexural areas, gums, and post-traumatic scars.



Laboratory evaluation showed severe hyponatremia (109 mmol/L), normal potassium, hypoglycemia (40 mg/dL), and metabolic acidosis (pH 7.19, BE −14.3). Given the persistence of hyponatremia despite correction, together with hypoglycemia and characteristic hyperpigmentation, an endocrine etiology was suspected. Hormonal evaluation revealed markedly decreased serum cortisol (<0.5 µg/dL), elevated ACTH, and low aldosterone levels. The Synacthen test confirmed primary adrenal insufficiency (absence of cortisol response).

An autoimmune etiology was supported by positive anti-21-hydroxylase antibodies. In the context of growth deficit, hypochromic microcytic anemia, and mild hypertransaminasemia, celiac disease was also suspected. Intestinal biopsy confirmed the diagnosis, revealing lesions consistent with Marsh IIIB–IIIC classification. Given the coexistence of autoimmune adrenal insufficiency and celiac disease, autoimmune polyglandular syndrome was considered. Thyroid function tests showed elevated TSH (8.24 mIU/L) with low fT4 (12.39 pmol/L); however, thyroid autoantibodies were negative, so autoimmune thyroid disease could not be confirmed, although it remained suspected.

Treatment was initiated intravenously with hydrocortisone hemisuccinate, followed by oral hydrocortisone acetate, fludrocortisone, and levothyroxine. A gluten-free diet was also introduced.

Results. This case illustrates the concomitant presentation of primary adrenal insufficiency, celiac disease, and thyroid dysfunction without confirmed thyroid autoimmunity. Although the absence of thyroid autoantibodies precludes a definitive diagnosis of type II autoimmune polyglandular syndrome at this stage, the coexistence of autoimmune adrenalitis and celiac disease warrants careful long-term surveillance, as patients remain at risk of developing additional autoimmune endocrinopathies within the APS spectrum.

Conclusion

Addison's disease in children may present with non-specific or atypical symptoms. Maintaining a high index of clinical suspicion is essential to ensure timely diagnosis and prevent life-threatening complications.[5]

Key words: *Addison's disease, adrenal insufficiency, autoimmune, celiac disease, treatment*

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J.D.03

GATA6 SYNDROME: PERMANENT NEONATAL DIABETES ASSOCIATED WITH PANCREATIC AGENESIS AND CONGENITAL HEART DISEASE

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Background and Aims. Neonatal diabetes mellitus (NDM) is a rare metabolic disorder presenting within the first six months of life, with an estimated incidence of 1:90,000–1:160,000 live births. Approximately 80% of diabetes diagnosed before 6 months is monogenic in origin. Among the implicated genes, GATA6 mutations account for nearly 3% of permanent NDM cases and up to 50% of pancreatic agenesis cases. GATA6 encodes a zinc-finger transcription factor critical for endodermal and mesodermal organogenesis, including pancreatic and cardiac development.

Pathogenic variants are associated with permanent insulin-dependent diabetes, exocrine pancreatic insufficiency, congenital heart defects (CHD), and multisystem anomalies.

We aimed to describe a complex case of permanent neonatal diabetes due to a de novo GATA6 mutation, emphasizing diagnostic strategy and multidisciplinary management.

Material and Methods. Clinical, biochemical, imaging, and genetic data were retrospectively analyzed from birth to 5 years of age. Investigations included serial glycemic profiles, HbA1c,

C-peptide, fecal elastase, lipid profile, abdominal ultrasound and computed tomography (CT), echocardiography, electrocardiography, and next-generation sequencing for monogenic diabetes.

Results

A female infant born at 35–36 weeks' gestation (birth weight 1560 g) developed persistent hyperglycemia (17–28 mmol/L) on day 5 of life, accompanied by poor weight gain. C-peptide was markedly reduced (0.3 ng/mL), indicating severe insulin deficiency.

Fecal elastase <15 µg/g confirmed profound exocrine pancreatic insufficiency. Abdominal CT demonstrated complete pancreatic agenesis. Cardiac evaluation revealed atrial septal defect (ASD), patent ductus arteriosus (PDA), and moderate pulmonary hypertension (40 mmHg). Surgical correction was performed at 12 months. Genetic testing identified a heterozygous de novo pathogenic deletion in GATA6. Insulin therapy was initiated early using a basal-bolus regimen, later transitioned to continuous subcutaneous insulin infusion, improving metabolic stability. Pancreatic enzyme replacement therapy ensured digestive improvement. HbA1c ranged between 6.1% and 11.5%. No severe ketoacidosis episodes were recorded. Growth delay and protein-calorie malnutrition were documented. At 5 years, celiac disease was diagnosed.

Conclusions

Persistent hyperglycemia within the first weeks of life should prompt evaluation for monogenic diabetes.

The coexistence of endocrine and exocrine pancreatic insufficiency with congenital heart defects strongly suggests GATA6-related disease.

Early insulin replacement and pancreatic enzyme therapy are essential.

Genetic confirmation enables precision medicine, tailored follow-up, and appropriate family counseling.

Multidisciplinary management remains fundamental due to the broad phenotypic spectrum.

***Keywords**: Congenital heart disease; GATA6; Neonatal diabetes; Pancreatic agenesis; Precision medicine*

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J.D. 04

X-LINKED HYPOPHOSPHATEMIC RICKETS IN A YOUNG CHILD WITH SEVERE SKELETAL AND DENTAL MANIFESTATIONS: A CASE REPORT

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X-linked hypophosphatemic rickets (XLH) is the most common inherited form of rickets, caused by pathogenic variants in the *PHEX* gene leading to excess FGF23 activity, renal phosphate wasting, and impaired bone mineralization. Early diagnosis is essential to prevent progressive skeletal deformities, growth impairment, and dental complications.[1,2]

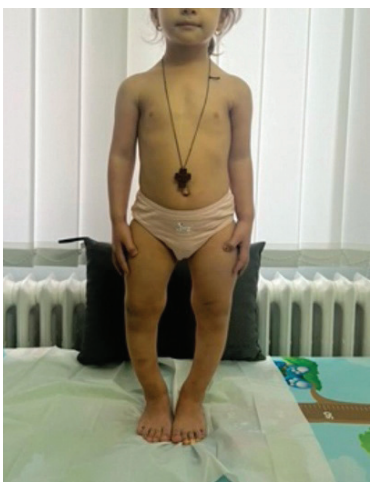
Case presentation

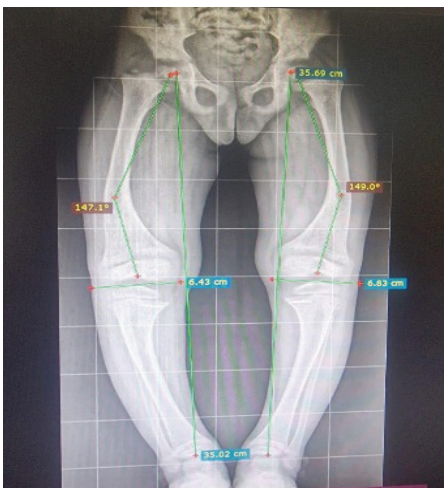
We report the case of a 5-year-old girl evaluated for progressive lower limb deformities, short stature, and recurrent dental abscesses. She presented with bilateral genu varum developing after the onset of walking and progressively worsening. Family history was highly suggestive: her father had short stature, full loss of dentition by his thirties, and multiple lower limb deformity surgeries without further investigations in childhood; he now presents with genu valgum and severe lower limb pain, consistent with a rickets phenotype.

Anthropometric evaluation showed significant growth impairment (height SDS −3.6) with preserved BMI. Physical examination revealed lower limb bowing (7 cm intercondylar distance), wrist enlargement, multiple dental lesions, and an acute dental abscess.

Full-length lower limb radiography confirmed metaphyseal widening and irregularity typical of rickets. The rickets severity score was 4.[3] During the 6-minute walk test, the patient walked 237 m, reporting mild lower limb pain at minute 4:35 but completing the test without interruption.

Biochemical assessment showed persistent hypophosphatemia (serum phosphate 2.1 mg/dL), mild corrected hypocalcemia (8.2 mg/dL), markedly elevated alkaline phosphatase (783 U/L), normal PTH levels, and normal renal function. FGF23 was elevated for age (81.7 pg/mL), supporting an FGF23-mediated disorder, while 1,25-$(OH)_2$ vitamin D was normal (78.2 pg/mL). Twenty-four-hour





urine testing did not demonstrate phosphate wasting, and TRP and TmP/GFR were within normal ranges, although these findings do not exclude the diagnosis. Renal ultrasound revealed mild right hydronephrosis; other systemic evaluations were unremarkable.

Genetic testing (Centogene CentoXome®) identified a heterozygous nonsense likely pathogenic variant in the *PHEX* gene (c.264G>A; p.Trp88*), confirming X-linked hypophosphatemic rickets. Because phosphate supplements are not available in Romania and the family could not obtain them abroad, we initiated temporary calcium carbonate and cholecalciferol supplementation to correct hypocalcemia. Dentistry treatment was initiated promptly. Genetic testing was requested during the week of first presentation, and the result was obtained within one month. Specific therapy with burosumab 10 mg every two weeks was started promptly after genetic confirmation.[4,5]

A notable aspect of this case is that the child had multiple contacts with the healthcare system due to a ventricular septal defect surgically corrected at one year of age, yet she was never referred to endocrinology or orthopedics after lower limb bowing became evident. Moreover, her father who is now 39 years old, has never been reffered to a rickets specialist before.

Conclusion

This case highlights the importance of early recognition of clinical and biochemical features suggestive of XLH, particularly in the presence of family history and dental involvement. Prompt genetic confirmation enables accurate diagnosis, family screening and management, and timely initiation of disease-specific therapy, improving growth outcomes and reducing long-term skeletal complications.

Keywords: *Burosumab, FGF23, genu varum, hypophosfatemia, XLH*

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J.D. 05

EARLY-ONSET HYPERPHAGIC OBESITY AS A CLUE TO MONOGENIC DISEASE: A CASE ASSOCIATED WITH A HETEROZYGOUS NTRK2 SPLICE-SITE VARIANT

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Background and Aims:
Early-onset hyperphagia with severe obesity is a key clinical clue for monogenic obesity. The BDNF–TrkB (NTRK2) pathway plays a central role in hypothalamic appetite regulation. Rare NTRK2 variants have been associated with neurodevelopmental disorders accompanied by hyperphagia and obesity.

Material and Methods:
A female patient was referred at 4 years of age for tall stature and accelerated growth, with caregiver-reported marked hyperphagia, hyperactivity, and stereotyped behaviours. Neuropsychological assessment confirmed attention deficit and cognitive and language delay. Growth velocity was markedly increased (up to 12 cm/year), with intermittently elevated IGF-1.

Severe early-onset obesity was present from early childhood (BMI >99th percentile), predominantly central (waist-to-height ratio 0.53), complicated by arterial hypertension. Pubertal development corresponded to Tanner stage 2 at 9 years.

Pituitary MRI showed normal morphology. Hormonal evaluation revealed intermittently elevated morning ACTH and cortisol, with normal late-night salivary cortisol and 24-hour urinary free cortisol. Hyperprolactinaemia normalised after polyethylene glycol precipitation, consistent with macroprolactinaemia. Subsequent IGF-1 values normalised. Metabolic evaluation demonstrated impaired fasting glucose, hyperinsulinaemia, dyslipidaemia and hyperuricaemia. Whole-exome sequencing identified a heterozygous canonical splice-site variant in NTRK2 (NM_006180.4: c.1765-2A>G), currently classified as a variant of uncertain significance.

Results:
A GLP-1 receptor agonist (liraglutide) was initiated as symptomatic therapy targeting hyperphagia and appetite dysregulation, alongside structured nutritional intervention and multidisciplinary follow-up including endocrinology, genetics and mental health services.

Conclusions:
This case highlights early-onset hyperphagia with neurodevelopmental abnormalities and central obesity as important diagnostic signals for monogenic obesity. Although the identified NTRK2 splice-site variant remains classified as a VUS, the phenotype is consistent with impaired TrkB signalling. Phenotype-driven management, including GLP-1 receptor agonist therapy, may represent a rational approach while awaiting further functional or genetic evidence.

Keywords: *early-onset hyperphagia; GLP-1 receptor agonist; monogenic obesity; neurodevelopmental disorder; NTRK2; TrkB signalling.*

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J.D. 06

CONGENITAL ADRENAL HYPERPLASIA IN CHILDHOOD AND ADOLESCENCE: CLOSING GAPS AND FACING CHALLENGES

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Background and Aims

Pubertal transition and early activation of the hypothalamic–pituitary–gonadal axis represent particularly challenging phases in congenital adrenal hyperplasia (CAH) due to 21-hydroxylase deficiency, as adequate suppression of adrenal androgen excess must be balanced against the risks of glucocorticoid-related impairment of growth and metabolic health. Although neonatal screening and standardized replacement therapy have improved early outcomes, achieving optimal disease control during childhood and adolescence remains difficult in routine clinical practice. Physiological changes associated with pubertal activation, including increased cortisol clearance and rising adrenal androgen production, frequently result in discrepancies between biochemical markers and clinical evolution.

During these periods, higher glucocorticoid exposure may be required to control hyperandrogenism and prevent accelerated skeletal maturation; however, supraphysiological dosing may adversely affect growth velocity, final height, body composition, and metabolic risk. In parallel, increasing autonomy and the psychological burden of chronic disease may impair treatment adherence and further destabilize disease control.

This report aims to illustrate real-life therapeutic challenges encountered during childhood and adolescence in patients with classical CAH, emphasizing the clinical difficulty of balancing androgen suppression with preservation of growth and metabolic health and highlighting the importance of integrating psychological evaluation into routine multidisciplinary care.

Case Report

We present two pediatric patients with classical CAH followed in a tertiary pediatric endocrinology center: a 10-year-old girl with central precocious puberty under treatment and

a 16-year-old boy undergoing late pubertal transition. Both patients received conventional glucocorticoid and mineralocorticoid replacement therapy and underwent regular clinical, auxological, and biochemical evaluation during follow-up.

In both cases, clinical evolution during periods of pubertal activation or progression was characterized by difficulties in maintaining stable disease control during phases of rapid growth and hormonal change. Episodes requiring intensification of glucocorticoid therapy were associated with signs of accelerated skeletal maturation and reduction in growth velocity, illustrating the narrow therapeutic window characteristic of these developmental stages.

Management consistently required balancing adequate androgen suppression against the risks associated with glucocorticoid overexposure. Clinical challenges included weight changes, the need for close monitoring of blood pressure and electrolyte balance, and variability in mineralocorticoid requirements. Periods of reduced treatment adherence, particularly during adolescence, further contributed to instability of disease control and highlighted the importance of psychological evaluation and multidisciplinary follow-up.

Conclusions

This two-case report highlights periods of pubertal activation and transition as phases in which the therapeutic dilemma of CAH management becomes most evident, with competing priorities between androgen control, growth preservation, and metabolic safety. Biochemical targets alone may not adequately reflect disease control during these developmental stages, emphasizing the need to integrate clinical, auxological, hormonal, and psychosocial factors in therapeutic decision-making. A proactive, multidisciplinary approach that anticipates physiological change and adapts therapy accordingly is essential to navigate this narrow therapeutic window and to improve long-term growth, metabolic outcomes, and quality of life in patients with CAH.

Keywords: *congenital adrenal hyperplasia, 21 -hydroxylase deficiency, growth impairment, pubertal transition, treatment challenges*

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J.D. 07
THERAPEUTIC COMPLICATIONS IN CONGENITAL HYPERINSULINISM

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Background and Aims:
Therapeutic complications in congenital hyperinsulinism (CHI) arise from both pharmacological management and surgical interventions, often causing significant morbidity despite efforts to control severe hypoglycaemia. Major complications include severe side effects from medications like diazoxide (hypertrichosis, fluid retention, pulmonary hypertension, gastrointestinal issues) and somatostatin analogues (necrotizing enterocolitis, biliary Issues, endocrine suppression, tachyphylaxis).

Material and Methods:
We present the case of a male infant with severe hypoglycaemia from the first day of life biochemically diagnosed with hyperinsulinism. Under specific treatment with Diazoxide, later with Sandostatin developed severe therapeutic complications (ppulmonary hypertension with heart failure and acute hepatitis).

Results:
The 16 days old infant had at a glycaemic value of 47 mg/ dl an insulin of 1,5 uUI/ ml repeated at a glycemia of 44 mg/ dl- the insulin came 5,8 uUI/ ml with a C peptide of 0,54 ng/ml (our normal values been 1,1-4,4 ng/ ml). We concluded that is a high probability of congenital hyperinsulinism and we started treatment with Diazoxide to whom he was responsive. At the start of therapy, he also showed intense jaundice with indirect bilirubin (IB = 16 mg/ dl at 3 weeks of life) which improved over time. An extensive genetic test was recommended at Blue Print Genetics – WES – it came negative. At a dose of 8 mg/ kg/ day diazoxide the general condition deteriorated, heart failure with pulmonary hypertension been detected – initial PAPs 50. We considered highly probable to be an adverse effect to Diazoxide, we started treatment for heart failure and changed the treatment to Sandostatin. After 11 days on Sandostatin 10,5 mcg/ kg/ day (Octreotide) he presented an increase of transaminases (AST= 409 U/ L, ALT =351 UI, GGT= 438 U/ L). We decrease the dose of Octreotide and stop the medication because we could not exclude another side effect. We investigated a possible another cause (CMV, EBV, Hep B, Hep C, Toxoplasma, inflammatory markers and CBC were normal) and he remained on oral feeding on request and a continuous IV glucose (aprox 4 mg/ kg/hour glucose IV) through central venous line without experiencing hypoglycemia.

Conclusions:
Despite the fact that the patient was responsive to Diazoxide and Sandostatin, in case of development of severe adverse reactions, therapeutic alternatives are limited.

Keywords: *complications, congenital hyperinsulinism, Diazoxid, Sandostatin*

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ELECTRONIC POSTERS:

P.P. 01

SEVERE HYPERTENSION IN AN ADOLESCENT LIVING WITH SEVERE OBESITY: A DIAGNOSTIC CHALLENGE

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Background:

Severe hypertension in children requires systematic evaluation for secondary causes, particularly endocrine disorders. However, the growing prevalence of severe pediatric obesity has led to increasingly complex cases in which obesity-related mechanisms may clinically mimic endocrine hypertension.[1,2]

Case presentation

A 13-year-old boy with class III obesity (BMI 41 kg/m², Z-score +3.43; weight 109 kg, height 163 cm) was admitted for evaluation of severe arterial hypertension. His medical history included metabolic dysfunction–associated steatotic liver disease (MASLD) with moderate fibrosis (F2/F3), severe obstructive sleep apnea (AHI 18/h) with very poor adherence to CPAP therapy, and echocardiography-documented concentric left ventricular hypertrophy.

On clinical examination, the patient presented severe acanthosis nigricans, generalized white striae, and complete pubertal development with hyperpilosity and normal genitalia. No significant difference in blood pressure values between upper and lower limbs was observed. Twenty-four-hour ambulatory blood pressure monitoring confirmed severe non-dipping hypertension (mean BP 170/97 mmHg; maximum BP 234/138 mmHg). Given the severity and evidence of early target-organ damage, an extensive workup for secondary causes was undertaken. Abdominal ultrasound was unremarkable except for severe hepatic steatosis. Cardiac ultrasound demonstrated a normal aortic arch. Renal and hepatic function were normal. Mild hyperuricemia was detected. HOMA-IR was 5.3, with normal HbA1c and OGTT results. Mild albuminuria was noted without overt proteinuria. Serum sodium and potassium were persistently normal. Thyroid function tests were normal. Morning serum cortisol, 24-hour urinary free cortisol, and ACTH were within normal ranges. Plasma renin, aldosterone, and their ratio were normal. Twenty-four-hour urinary metanephrines and normetanephrines were within the reference range. Given Tanner stage V at age 13, congenital adrenal hyperplasia due to 11β- or 17α-hydroxylase deficiency was considered; however, 11-deoxycorticosterone levels were normal. Angio-MRI demonstrated normal renal and iliac arteries with preserved renal perfusion.

Management

Treatment with the ACE inhibitor enalapril (20 mg daily) was initiated, resulting in normalization of blood pressure while maintaining normal serum creatinine levels. [3] Omega-3 fatty acids and cholecalciferol supplementation were added. Lifestyle interventions aimed at weight reduction were initiated, although adherence has been poor. Semaglutide therapy was recommended but is currently not accessible due to financial constraints.

Discussion

This case illustrates that severe obesity in adolescents may present with a phenotype mimicking endocrine hypertension. However, extreme blood pressure values and early target-organ damage warrant thorough exclusion of secondary causes, including renal, endocrine (Cushing syndrome, primary hyperaldosteronism, pheochromocytoma/paraganglioma, hyperthyroidism, and CAH due to 11β- or 17α-hydroxylase deficiency), and vascular causes (renal artery stenosis, coarctation, or aortic arch hypoplasia). Monogenic hypertension syndromes such as Liddle or Gordon syndrome would be considered if potassium abnormalities were present or if blood pressure had failed to respond to ACE inhibitor therapy. [4,5]

Conclusion

Obesity-related hypertension remains the most plausible cause in this case, likely driven and aggravated by untreated obstructive sleep apnea. The favorable response to ACE inhibition supports a renin–angiotensin–mediated metabolic mechanism. Severe hypertension in adolescents with obesity should not be underestimated and requires a multidisciplinary approach.

Keywords: *endocrine evaluation, pediatric hypertension, secondary hypertension, severe obesity.*

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P.P. 02

SINGLE-CENTER RETROSPECTIVE ANALYSIS OF CHILDHOOD-ONSET TYPE 1 DIABETES

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Introduction: Type 1 diabetes is a disease with major impact on quality of life but also is an important public health problem. The incidence has increased significantly in last decade, reaching around 20 per 100.000 children annually.[1] Although there have been important steps for early diagnosis, the onset in children is frequently with ketoacidosis, with the rates that vary greatly around the world (between 15-70%). [2]

Methods: We conducted a retrospective study of children diagnosed with type 1 diabetes over a period of 5 years (2020-2025). We analysed the demographic profile type of onset and risk factors present at that moment.
Results: There were 61 patients diagnosed with T1D in the period analysed. The mean age of diagnosis was 10.27 years old. Boys accounted for 54% of the patients. Regarding type of onset, the majority of children presented in ketoacidosis with 36% mild, 20% moderate and 18% severe forms. The rest, 26% were patients presented with hyperglycemia without ketoacidosis. An important association at admission was infection, which was observed in 36% of the patients. The most frequent infections identified were respiratory and urinary. Only 5% of the patients were already diagnosed with other autoimmune disease. Family history of autoimmunity was identified in 22% of patients (8% T1D, 10% other autoimmune disease, 4% more than one autoimmune diseases). From our group, 43 children had vitamin D level measured at admission and 76% had insufficient serum levels.
Conclusions: The prevalence of ketoacidosis at onset in our group is very high, emphasizing the need for faster diagnosis and education programs. The most important risk factors identified were vitamin D deficiency and infections. These findings are in accordance with the literature, and highlights the importance of investigating them in all patients at onset of T1D.
Key words: *type 1 diabetes, ketoacidosis, vitamin D deficiency, infection*
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P.P. 03
BEYOND ADRENAL INSUFFICIENCY: TRANSIENT SECONDARY PSEUDOHYPOALDOSTERONISM IN AN INFANT WITH URETEROHYDRONEPHROSIS

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Background and aims
Pseudo-hypoaldosteronism (PHA) is a rare disorder characterized by resistance to the action of aldosterone at the renal tubular level, leading to electrolyte imbalance despite elevated

renin and aldosterone levels[1]. In infancy, secondary or transient PHA may occur in association with urinary tract infections (UTI) or congenital anomalies of the urinary tract[2,3]. This condition can mimic other endocrine or metabolic disorders and may present with severe electrolyte disturbances[3]. Early recognition is essential because the condition is reversible once the underlying infection or obstruction is treated[4,5]. The aim of this report is to present the clinical and biochemical features of transient secondary pseudo-hypoaldosteronism in an infant with acute pyelonephritis and urinary tract malformation.

Material and methods

We report the case of a 6-month-old infant admitted to the Pediatric Clinic of County Clinical Emergency Hospital of Constanta for evaluation of previously detected hypercalcemia and failure to thrive. Clinical history, laboratory findings, and imaging studies were reviewed. Growth parameters and previous medical history were analyzed, including febrile episodes and laboratory investigations performed before admission.

Results

The infant had a history of recurrent febrile episodes beginning at the age of two months, associated with inflammatory laboratory markers. At admission (6 months), the patient presented with severe growth failure (weight 6020 g; Z-score −3.38 SD) and significant biochemical abnormalities. Laboratory evaluation revealed lymphocytosis with neutropenia, marked thrombocytosis (791 $\times 10^3/\mu L$), metabolic acidosis, severe hyponatremia (124.5 mmol/L), and hyperkalemia (6.01 mmol/L). Lipid profile showed mixed dyslipidemia with hypertriglyceridemia (836 mg/dL). Mild hypercalcemia persisted (11.68 mg/dL) with suppressed parathyroid hormone levels. Hormonal evaluation demonstrated markedly elevated plasma renin (>1000) and very high aldosterone levels (2456 ng/dL), suggesting mineralocorticoid resistance.

Urinalysis and urine culture confirmed urinary tract infection with *Escherichia coli*. Renal ultrasound revealed left-sided uretero-hydronephrosis (grade III) with hydroureter, suggesting a congenital urinary tract anomaly such as ureteral duplication or vesical malformation.

The association of severe electrolyte imbalance (hyponatremia, hyperkalemia, metabolic acidosis), markedly elevated renin and aldosterone levels, urinary infection, and structural urinary tract abnormality strongly suggested transient secondary pseudo-hypoaldosteronism due to acute pyelonephritis. The pathophysiology involves temporary resistance to aldosterone at the distal renal tubule caused by inflammation and tubular dysfunction during infection or obstructive uropathy.

Conclusions

Transient secondary pseudo-hypoaldosteronism should be considered in infants presenting with hyponatremia, hyperkalemia, and metabolic acidosis in the context of urinary tract infection. The condition may mimic primary adrenal insufficiency or congenital adrenal disorders, but it is a reversible functional disturbance. Recognition of this entity is crucial to avoid unnecessary endocrine investigations and to ensure prompt treatment of the underlying infection and urinary tract abnormality. Early diagnosis and appropriate management lead to normalization of electrolyte balance and favorable clinical outcomes.

Keywords: *Transient pseudo-hypoaldosteronism; Infant; Pyelonephritis; Urinary tract malformation*

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P.P. 04

BEYOND METABOLIC ACIDOSIS: PANCREATIC INVOLVEMENT IN SEVERE NEW-ONSET PEDIATRIC DIABETIC KETOACIDOSIS

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Background and aims

Severe diabetic ketoacidosis (DKA) remains a major pediatric metabolic emergency, particularly at the onset of type 1 diabetes mellitus (T1DM)[1,2]. Gastrointestinal symptoms such as abdominal pain, nausea, and vomiting frequently overlap with those of DKA, potentially obscuring concomitant pancreatic injury[3]. Although elevations in amylase and lipase are commonly observed during DKA, true acute pancreatitis is considerably less frequent but clinically significant[4]. This study aimed to explore the pathophysiological mechanisms, clinical features, diagnostic challenges, and management strategies related to pancreatic involvement in children presenting with severe new-onset DKA, illustrated by two representative cases.

Material and methods

We performed a narrative review of recent literature addressing pancreatic abnormalities in pediatric DKA and present two cases of severe new-onset DKA with biochemical evidence of pancreatic involvement. Clinical presentation, laboratory findings, imaging results, therapeutic approaches, and short-term outcomes were analyzed.

Results

Case 1 describes an 11-year-old boy admitted with severe new-onset DKA (pH 6.84, HCO_3^- 3.1 mmol/L, glucose 514 mg/dL), dehydration, Kussmaul respiration, and abdominal pain. Persistent gastrointestinal symptoms during metabolic correction raised suspicion of pancreatic involvement. Literature data indicate that hyperamylasemia occurs in approximately 20–40% of pediatric DKA cases, whereas confirmed acute pancreatitis is reported in only 2–5%, typically defined by lipase levels exceeding three times the upper limit of normal together with supportive imaging findings.

Case 2 involves an 8-year-old girl with severe new-onset DKA associated with complicated influenza and pneumonia. Abdominal ultrasound revealed hepatic steatosis with fibrosis markers (7 kPa), nondilated bile ducts, and a diffusely thickened gallbladder wall containing biliary sludge and a 27 × 10 mm hyperechoic conglomerate with posterior acoustic shadowing. On the third hospital day, serum amylase and lipase rose significantly; however, CT and ultrasound showed no imaging criteria for acute pancreatitis. The enzyme elevation was interpreted as secondary pancreatic involvement related to severe metabolic imbalance, systemic inflammation, dehydration, transient pancreatic hypoperfusion, and biliary pathology.

The literature suggests multiple contributing mechanisms, including profound dehydration, impaired pancreatic perfusion, metabolic acidosis, hypertriglyceridemia, and systemic inflammatory response. Importantly, elevated pancreatic enzymes may occur in the absence of radiologic evidence of pancreatitis, complicating differential diagnosis during acute DKA[5].

Management in both cases consisted primarily of standard DKA therapy (fluid resuscitation, continuous intravenous insulin infusion, and careful electrolyte correction), anti-infective treatment when indicated, and supportive gastrointestinal care. No invasive pancreatic intervention was required, and both clinical and biochemical parameters improved progressively with metabolic stabilization.

Conclusions

Pancreatic involvement in severe pediatric DKA is likely underrecognized because of overlapping abdominal symptoms. Elevated amylase and lipase levels do not necessarily indicate true acute pancreatitis; therefore, imaging confirmation and clinical correlation are essential. Persistent abdominal pain or sustained enzyme elevation should prompt systematic evaluation. A multidisciplinary approach that combines optimal DKA management with targeted assessment of pancreatic and biliary pathology may enhance diagnostic accuracy and improve patient outcomes.

Keywords: *diabetic ketoacidosis; type 1 diabetes mellitus; pancreatitis*

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P.P. 05

INHERITED RICKETS REVEALED BY SEVERE COMPLICATED PNEUMONIA: A CASE REPORT

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Background and aims:

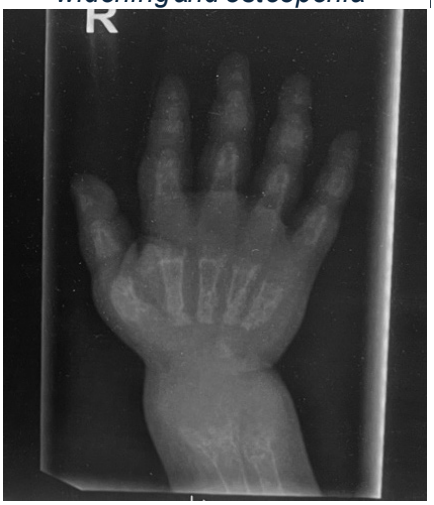
Vitamin D–dependent rickets type 1 (VDDR1A) is a genetic cause of hypocalcemic rickets with autosomal recessive inheritance. It affects a key enzyme (1-alpha-hydroxylase) responsible for the final step in the formation of calcitriol.[1,2]

Case description:

A 2-year-old girl from a socioeconomically deprived environment presented to the emergency department with breathing difficulties and cough. Clinically, she exhibited growth and motor delay, frontal bossing, a large anterior fontanelle for her age, micrognathia, rachitic rosary, funnel chest, and Harrison's groove. The patient required oxygen therapy to maintain adequate oxygen saturation and presented typical signs of pneumonia, including crackles at the right lung base.

Laboratory findings revealed hypocalcemia, hypophosphatemia, a significant increase in alkaline phosphatase, elevated parathyroid hormone (PTH), and normal levels of calcifediol (25-OH vitamin D). Leukocytosis with monocytosis and lymphocytosis was also noted. Radiographic findings showed fraying of the wrist metaphysis and osteoporosis. Chest X-ray demonstrated severe osteopenia and bilateral areas of consolidation.

Figure 1. Hand X-Ray: Metaphyseal widening and osteopenia



Sequential antibiotic therapy was initiated and continued for a total duration of 56 days due to highly resistant pneumonia. To determine the underlying cause of the multidrug-resistant pneumonia, other related conditions were ruled out (cystic fibrosis, immunodeficiency, etc.).

During hospitalization, the patient received daily supplementation with calciferol; however, serum alkaline phosphatase levels remained elevated, with no significant improvement in calcium–phosphate metabolism. A significant decrease in 1,25-OH vitamin D was observed, despite normal 25-OH vitamin D levels and supplementation with 25-OH vitamin D. These biochemical modifications were suggestive of a genetic cause of vitamin D deficiency.

Whole-exome sequencing was performed to identify a genetic cause of rickets and revealed a homozygous CYP27B1 variant (c.335C>T, p.Pro112Leu), consistent with vitamin D–dependent rickets type 1.

The patient then received treatment with alfacalcidiol and adequate dietary calcium intake, with slow normalization of calcium–phosphate metabolism, but with a persistent elevated alkaline phosphatase.

Treatment with high-dose active vitamin D or its analogues, along with calcium supplementation, quickly resolves the biochemical abnormalities. However, there is a risk of nephrocalcinosis due to hypercalcemia and hypercalciuria, which requires constant monitoring of calcium and phosphorus metabolism.[3,4]

Conclusion: This case illustrates the complex interplay between a rare genetic cause of vitamin D deficiency and severe acute respiratory infections.[5]

Keywords: *CYP27B1, calcium, pneumonia, rickets, vitamin D.*

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P.P. 06

CASE SERIES OF SILVER–RUSSELL SYNDROME WITH DISTINCT MOLECULAR ETIOLOGIES IN PEDIATRIC PATIENTS

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Background and aims

Silver–Russell syndrome (SRS) is a rare imprinting disorder characterized by prenatal growth restriction, persistent postnatal short stature, relative macrocephaly, feeding difficulties, and variable endocrine, metabolic, and neurodevelopmental complications[1,2]. Molecular confirmation is achieved in approximately two-thirds of patients, most frequently due to loss of methylation (LOM) at 11p15 or maternal uniparental disomy of chromosome 7 (matUPD7)[3].

Epigenetic alterations involving the H19/IGF2 domain further contribute to phenotypic variability[4]. Early recognition and molecular characterization are essential for prognosis and individualized follow-up[5]. We present a pediatric case series highlighting distinct molecular mechanisms and heterogeneous clinical evolution.

Material and methods

This retrospective observational case series includes three pediatric patients diagnosed with SRS in County Clinical Emergency Hospital of Constanta, Pediatric Department. Clinical diagnosis was established using phenotypic criteria and supported by the Netchine–Harbison Clinical Scoring System (NH-CSS). Molecular testing included methylation analysis of the 11p15 region and assessment for maternal uniparental disomy of chromosome 7. Longitudinal clinical data were collected, including growth parameters, bone age, endocrine profile, metabolic status, and neurodevelopmental outcomes.

Results

All three patients presented with prenatal growth restriction and persistent postnatal growth failure but differed in age at diagnosis, molecular findings, and associated complications.

Patient 1 was diagnosed during the first year of life and demonstrated loss of methylation (LOM) at 11p15. Early feeding difficulties and failure to thrive prompted genetic testing. During follow-up, this patient developed insulin resistance in late childhood, highlighting the metabolic risk associated with 11p15-related epigenetic defects. Neurodevelopment was otherwise appropriate for age.

Patient 2 was diagnosed at 6 months of age and had maternal uniparental disomy of chromosome 7 (matUPD7). Growth restriction was evident in infancy, and early molecular confirmation enabled prompt nutritional and endocrine monitoring. The clinical course was comparatively milder, with transient feeding difficulties and mild neurodevelopmental delay. No insulin resistance or significant metabolic abnormalities were identified during follow-up.

Patient 3 represented the most severe phenotype and was diagnosed at 4 years of age, with hypomethylation of the H19/IC1 locus within 11p15. This patient exhibited the most pronounced growth failure, significant speech delay, and delayed bone age at presentation. Despite marked auxological impairment, no insulin resistance was detected. The later diagnosis correlated with greater growth deficit and developmental delay.

Growth hormone therapy was initiated according to international recommendations, resulting in improved growth velocity, particularly when started early. Bone age delay was most evident in Patient 3, consistent with severe stature impairment.

Conclusions

This case series underscores the phenotypic and molecular heterogeneity of Silver–Russell syndrome. Early molecular diagnosis facilitates timely intervention and may mitigate growth and developmental deficits. Patients with 11p15-related defects may be at increased risk for metabolic complications such as insulin resistance, requiring long-term surveillance. SRS should be approached as a multisystem disorder requiring coordinated endocrine, metabolic, and developmental follow-up tailored to molecular subtype.

Keywords: *Silver–Russell syndrome; 11p15 loss of methylation; maternal uniparental disomy chromosome 7; H19/IC1 hypomethylation; insulin resistance; growth failure.*

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P.P. 07

ENDOCRINE-DISRUPTING CHEMICALS AND GENETIC RISK IN AUTISM SPECTRUM DISORDERS

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Background and Aims

Human brain development depends on tightly regulated genetic and endocrine processes. Autism spectrum disorder (ASD) is a multifactorial neurodevelopmental condition with a strong genetic basis, including rare pathogenic variants, copy number variations, and polygenic risk factors. However, genetic predisposition alone does not fully account for ASD occurrence, highlighting the importance of environmental contributors. Endocrine-disrupting chemicals (EDCs) interfere with hormonal pathways critical for brain maturation and may interact with genetic susceptibility. This work aims to summarize current evidence linking EDC exposure, genetic background, and ASD-related neurodevelopmental outcomes.

Material and Methods

This paper provides a focused narrative synthesis of selected studies addressing genetic susceptibility in ASD and its potential interaction with endocrine-disrupting chemicals. Evidence from molecular genetics, epigenetic research, and experimental models was considered to explore how EDC exposure may influence gene expression, hormonal signaling pathways, and neurodevelopmental processes. Representative epidemiological and experimental findings were integrated to illustrate possible gene–environment interactions.

Results

Genetic research has identified multiple ASD-associated variants affecting synaptic function, neuronal signaling, and brain development. Environmental exposure to EDCs—including bisphenols, phthalates, polychlorinated biphenyls, polybrominated diphenyl

ethers, and per- and polyfluoroalkyl substances—has been associated with ASD traits and behavioral alterations. Experimental evidence indicates that EDCs can disrupt thyroid and sex hormone signaling, alter neuronal proliferation and synaptic organization, and modify gene expression through epigenetic mechanisms. These biological effects may be particularly relevant in genetically susceptible individuals, suggesting that EDC exposure could modulate neurodevelopmental risk pathways involved in ASD.

Conclusions

ASD likely arises from complex interactions between genetic vulnerability and environmental exposures. Current evidence supports a contributory role of endocrine-disrupting chemicals in neurodevelopmental alterations, potentially through hormone-mediated and epigenetic mechanisms. Integrative research approaches are necessary to better understand gene–environment interplay and to inform preventive strategies aimed at reducing exposure during critical developmental periods.

Keywords: *autism spectrum disorder; endocrine-disrupting chemicals; gene–environment interaction; neurodevelopment; thyroid hormones*

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P.P. 08

BUROSUMAB TREATMENT IN X-LINKED HYPOPHOSPHATEMIC RICKETS DURING THE TRANSITION PERIOD: CLINICAL AND BIOCHEMICAL OUTCOMES

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Background:

X-linked hypophosphatemic rickets (XLH) is a rare genetic disorder caused by mutations in the *PHEX* gene, characterized by elevated levels of FGF-23, leading to hypophosphatemia, hyperphosphaturia, decreased 1,25$(OH)_2$ vitamin D levels, and impaired bone mineralization.

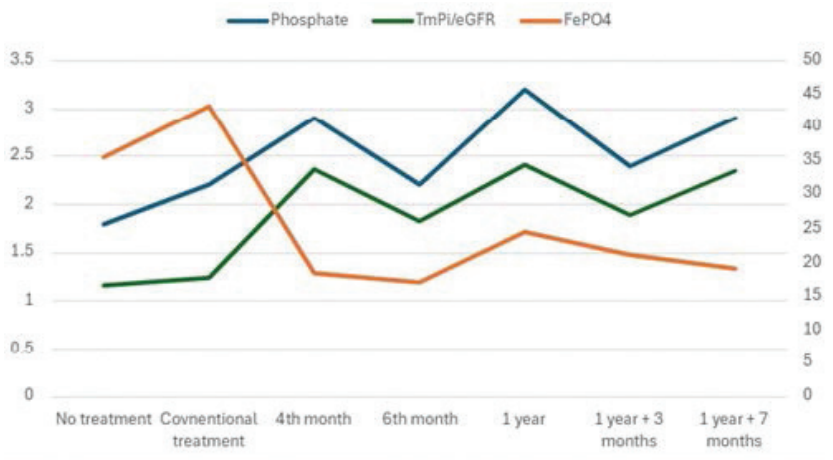
Clinical manifestations include short stature, skeletal deformities, chronic musculoskeletal pain, waddling gait, dental abscesses, and a significant reduction in quality of life.
The transition period from childhood to adulthood represents a particularly vulnerable stage in the course of XLH, during which delayed diagnosis and suboptimal management may exacerbate skeletal complications and further impair quality of life.
Burosumab is a humanized monoclonal anti-FGF-23 antibody that acts pathogenically by increasing renal phosphate reabsorption and serum $1,25(OH)_2$ vitamin D levels.

Material and Methods:

We present the case of a patient diagnosed in childhood with vitamin D–resistant hypophosphatemic rickets, who was re-evaluated at the age of 20 years for chronic musculoskeletal pain, skeletal deformities, and a history of growth retardation, after a family member was diagnosed with XLH.
Clinical examination revealed disproportionate short stature, waddling gait, and lower limb deformities with genu varum (intercondylar distance = 10 cm).
Laboratory investigations showed persistent hypophosphatemia, bone-specific alkaline phosphatase at the upper limit of normal, increased urinary phosphate excretion, reduced tubular phosphate reabsorption (TmPi/eGFR), normocalcemia, and low–normal levels of $1,25(OH)_2$ vitamin D, along with the presence of pseudofractures on skeletal radiographs. The diagnosis was confirmed by genetic testing, which identified a pathogenic mutation in the *PHEX* gene.

Results:

Initially, the patient received conventional therapy with phosphate salts and active vitamin D analogues, with poor clinical and biochemical response.
Given the persistence of symptoms and biochemical abnormalities, treatment with Burosumab was started.
Under therapy, rapid normalization of serum phosphate levels and TmPi/eGFR was observed, along with an increase in $1,25(OH)_2$ vitamin D levels, a decrease in bone-specific alkaline phosphatase, healing of pseudofractures, and prevention of their recurrence. No adverse effects, such as hypercalcemia or secondary hyperparathyroidism, were recorded. Clinically, the patient experienced a significant improvement in pain scores and quality of life.



Conclusions: Treatment with burosumab during the transition period led to rapid normalization of biochemical parameters and significant improvement in quality of life, without major adverse reactions, demonstrating the efficacy and safety of targeted therapy at this critical stage of disease progression.

Keywords: *hypophosphatemia, rickets, transition, treatment*

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P.P. 09

INFLUENZA-TRIGGERED FULMINANT DIABETIC KETOACIDOSIS WITH COMA AT THE ONSET OF TYPE 1 DIABETES MELLITUS IN A CHILD

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Background and aims

Influenza is a highly contagious respiratory viral infection responsible for annual epidemics and significant pediatric morbidity, according to the World Health Organization[1]. Viral infections are well-recognized triggers of metabolic decompensation in children with diabetes[2].

Type 1 diabetes mellitus (T1DM) affects approximately 1.8 million children worldwide, and diabetic ketoacidosis (DKA) is present at diagnosis in 30–40% of cases, as reported by the International Diabetes Federation[3]. Severe DKA, defined by the International Society for Pediatric and Adolescent Diabetes as pH <7.1 or bicarbonate <5 mmol/L, may lead to life-threatening complications, including cerebral edema[4,5].

We report a case of influenza-triggered severe DKA with coma at the onset of T1DM in a child, highlighting diagnostic urgency during epidemic periods.

Material and methods

An 8-year-old girl presented with a two-day history of fever, sore throat, progressive somnolence, and altered mental status in the context of confirmed influenza A infection. The

family reported a six-month history of polyuria, polydipsia, nocturia, and weight loss.
On admission, the patient was comatose (Glasgow Coma Scale 5/15). Laboratory findings revealed severe DKA: blood glucose 560 mg/dL, venous pH 6.7, bicarbonate 2.7 mmol/L, base excess −31.5 mmol/L, and ketonuria. BMI was at the 40th percentile, and there was no prior diagnosis of diabetes, supporting the diagnosis of new-onset T1DM complicated by severe DKA precipitated by influenza A.

Results

The patient received immediate fluid resuscitation, intravenous bicarbonate, and continuous intravenous insulin infusion and was transferred to the Pediatric Intensive Care Unit. Antiviral therapy (oseltamivir) was initiated. Continuous clinical and biochemical monitoring was performed.
Early complications included bladder distension; urinary catheterization was delayed due to a suspected urinary tract malformation. Approximately three hours after admission, hypokalemia developed, requiring careful electrolyte correction.
After 12 hours of treatment, neurological status improved modestly (GCS 9/15), although glycemic control was slower compared to other severe DKA cases. At 36 hours, GCS improved to 13/15; however, bilious vomiting occurred. Abdominal ultrasound revealed pancreatic inflammatory reaction and gallbladder microlithiasis without signs of cholecystitis. Conservative management included bile acids for five days and dietary measures. Enteral feeding was delayed for seven days due to persistent gastrointestinal symptoms.
Transition to subcutaneous rapid-acting and long-acting insulin was achieved once metabolic stability was maintained. The patient was discharged after 15 days, following comprehensive diabetes education for the patient and family.

Conclusions

Influenza infection may precipitate fulminant severe DKA with coma at the onset of T1DM. During influenza epidemics, clinicians should maintain a high index of suspicion for hyperglycemia and DKA in febrile children presenting with altered mental status or suggestive symptoms of diabetes.
Influenza vaccination in high-risk pediatric populations, including children with known or suspected diabetes, may reduce infection-triggered metabolic decompensation and DKA incidence.

***Key words**: influenza, coma, diabetic ketoacidosis, children*

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SEVERE RESPIRATORY INFECTION IN A PATIENT WITH AUTOIMMUNE POLYGLANDULAR SYNDROME- A CASE PRESENTATION FROM BRASOV, ROMANIA

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Background

Autoimmune polyglandular syndrome represents a rare association of endocrine manifestations. Type 1 APS presents with hypoparathyroidism, adrenal insufficiency, and chronic candidiasis.[1] Those who suffer from it are more susceptible to respiratory infections,[2] as their immune systems may be dysregulated (modified type I interferon cytokine pathways)[3], combined with long-term cortisone administration.[4]

Case Presentation

We present the case of an 11-year-old girl who was admitted to our hospital for respiratory distress and high fever. The rapid antigen test from nasal secretions came back positive for type A influenza. Her medical history revealed that the girl was diagnosed with autoimmune polyglandular syndrome type 1 at 2 years old, and she has been on cortisone and fludrocortisone supplementation ever since (12.5 mg of hemisuccinate hydrocortisone orally per day and 0.05 mg of fludrocortisone orally per day).

After admission, she started treatment with Oseltamivir and received high-flow oxygen therapy.

Laboratory tests showed highly elevated levels of C-reactive protein (10.5 mg/dl, NV=<1 mg/dl). Moderate anemia was found on her WBC-8.7 g/dl (NV=11.5-14.5 g/dl), and RT-PCR from nasal swabs determined the presence of type A influenza and Streptococcus pneumoniae. Her potassium and calcium levels were carefully monitored during hospital admission, with calcium supplements added to the complex treatment.

Her general state rapidly deteriorated, and the thoracic X-ray showed bilateral, diffuse infiltrates that were consistent with the onset of acute respiratory distress syndrome, secondary to influenza-related pneumonia and pneumococcal superinfection. An important part of the medication regimen included the dosage increase of cortisone (she received methylprednisolone intravenously, 1 mg/kg/day, in a singular dose), the administration of antibiotics (meropenem and vancomycin), and immunoglobulins. Her hemoglobin dropped to 7.2 g/dl, and blood transfusion was administered in order to raise her hemoglobin level.

The patient's general state started to improve afterwards, with the normalization of oxygen saturation level and of the chest X-ray aspect. She was discharged after 9 days of treatment.

Learning Points/Discussion

We consider this rather rare case to be a learning point for clinicians, as these children's clinical condition may rapidly deteriorate after a viral infection. Strong collaboration with endocrinologists is necessary, as these patients need a cortisone dosage increase, secondary to their innate hormonal deficits.[5] We strongly advise forming a multidisciplinary team when dealing with these patients.

Keywords: *acute respiratory distress syndrome, APS type 1, influenza.*

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BEYOND ALGORITHMS IN DIAGNOSING NEPHROGENIC SYNDROME OF INAPPROPRIATE ANTIDIURESIS

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Background and Aims:
Nephrogenic syndrome of inappropriate antidiuresis (NSIAD) is a rare genetic disease with a prevalence of <1/1000000, first described in 2005. It is caused by an activating mutation in the arginine vasopressin receptor-2 (AVPR2) gene. Affected patients excrete concentrated urine despite very low AVP levels and consequently develop euvolemic hyponatremia. Due to its low frequency, patients may be misdiagnosed and treated incorrectly.

Material and Methods:
We present the case of a male infant with clinical and biochemical features of syndrome of inappropriate antidiuretic hormone secretion (SIADH) with no apparent cause. Fluid restriction increased his serum sodium despite the plasma AVP level being low. *AVPR2* gene analysis revealed a known mutation (c.409C>T; p.R137C) and confirmed the diagnosis of Nephrogenic syndrome of inappropriate antidiuresis (NSIAD).

Results:
The 7month old male, apparently healthy with normal physical exam findings presented with persistent severe hyponatremia, low plasma osmolality and low plasma renin-aldosterone levels. These clinical and laboratory findings were compatible with SIADH without apparent cause. Consequently, fludrocortisone was initiated with a presumptive diagnosis of hyporeninemic hypoaldosteronism (HH). The low AVP levels and the activating mutation of

AVPR2 (c.409C>T; p.R137C) confirmed the diagnosis of NSIAD. The therapeutic approach was changed to fluid restriction with correction of hyponatremia.

Conclusions:

Due to its low frequency, it is not usually considered in the differential diagnosis of euvolemic hyponatremia. Therefore, lack of awareness of this rare disease may cause delay in determining the etiology of hyponatremia and even misdiagnosis.

NSIAD should be considered as a diagnosis in patients presenting with unexplained hyponatremia and low plasma osmolality.

As a first step in the investigation, plasma AVP levels should be measured. In patients with undetectable or low AVP levels, genetic testing of AVPR2 can simply confirm diagnosis.

It should be noted that if NSIAD is not considered, the plasma renin-aldosterone profile can be confused with HH, especially in infants.

***Keywords**: AVPR2 gene, hyponatremia, inappropriate antidiuretic hormone secretion*

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PRIMARY OVARIAN INSUFFICIENCY AND BILATERAL HEARING LOSS SUGGESTING PERRAULT SYNDROME: A CASE REPORT

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Background and Aims:

Perrault syndrome is a rare autosomal recessive disorder characterized by sensorineural hearing loss and primary ovarian insufficiency (POI) in females, with variable additional

features, including neurological abnormalities and dysmorphic features.[1,2]
However, atypical presentations may pose diagnostic challenges. Early recognition is crucial for genetic counseling and multidisciplinary management.
We present a case of an adolescent with clinical features that raise suspicion for Perrault syndrome.

Material and Methods:

A 17-year-old female was evaluated for secondary amenorrhea and bilateral neurosensorial hypoacusis. Clinical assessment included detailed medical and family history, physical examination, hormonal evaluation, pelvic ultrasound, abdomino-pelvic MRI, and karyotype analysis.

Results:

Menarche occurred at 13.5 years, with progressive menstrual irregularity (2-3 cycles/year). Family history included autoimmune thyroiditis in the mother and maternal grandmother, irregular menses in the mother, and hypoacusis in a 19-year-old brother. Physical examination showed normal anthropometry with dysmorphic features (epicanthus, micrognathia, long neck, small ears, downslanting palpebral fissures), dental anomalies (absent lateral incisors, one canine, and premolars), and café-au-lait spots. Sexual development was Tanner stage IV for breast development, with absent axillary and pubic hair and normal external genitalia. Laboratory results confirmed POI with markedly elevated FSH (99 mUI/ml) and LH (42.5 mUI/ml), profoundly suppressed estradiol (<9 pg/ml), undetectable AMH (<0.01 ng/ml), and low progesterone and testosterone. Anti-ovarian antibodies were slightly elevated, while anti-adrenal antibodies were negative. Thyroid function was euthyroid with positive anti-thyroid antibodies. Imaging revealed a small tubular uterus with a thin endometrium and poorly visualized ovaries. Karyotype was normal, 46,XX. WES/WGS focusing on Perrault syndrome genes (CLPP, ERAL1, HARS2, HSD17B4, LARS2, TWNK) and syndromic hearing loss genes (SLC26A4) were recommended to confirm the suspected diagnosis. Estrogen-progesterone hormone replacement therapy was initiated, and cardiac and neurological evaluations were recommended.

Conclusions:

Perrault syndrome should be considered in adolescent females with POI and sensorineural hearing loss, even in the absence of classic features.[1,2] Additional features, including dysmorphic traits, dental anomalies, dissociated pubertal development, and autoimmune markers, may indicate atypical phenotypes. Molecular genetic confirmation through WES/WGS is essential for definitive diagnosis, genetic counseling, and multidisciplinary management.[3,4] This case emphasizes comprehensive evaluation in POI patients to identify syndromic causes and initiate appropriate hormone replacement therapy.[5]

Keywords: *Adolescent, Hearing loss, Perrault syndrome, Primary ovarian insufficiency, Sensorineural hypoacusis*

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P.P. 13

THE TRANSITION PERIOD AND GH THERAPY: DISCUSSIONS ON A CLINICAL CASE

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Background and Aims:

The main role of growth hormone (GH) treatment in children with GH deficiency is to ensure growth. This effect of GH substitution therapy ends with the closure of the growth cartilages. Adults with GH deficiency develop an increase of adipose tissue and a decrease in muscle mass, decreased bone mineral density (BMD) with an increased fracture rate, earlier onset of cardiovascular disease, reduced quality of life, and increased mortality.

The long-term benefits of GH treatment in adults, on body composition and metabolism, are well known and recognized. These effects of GH, unrelated to growth, fully justify the long-term administration of substitution therapy in GH-deficient adults. The adherence to GH substitution in adulthood can be as low as 70%, thus it remains of paramount importance to outline the benefits of GH substitution.[1]

Materials and Methods:

B.M., a 27-year-old patient, was diagnosed at the age of 2 with thyrotropin, corticotropin and somatotropin deficiency after severe episodes of hypoglycemia.

Replacement therapy was promptly initiated with oral levothyroxine and hydrocortisone, and later, at the age of 8, with growth hormone. At the age of 16, replacement therapy with sex hormones was also initiated, due to gonadotropic deficiency. At the age of 18, she was transferred for monitoring to our Clinic. The patient was of normal height according to predicted final height and had mature secondary sexual characteristics.

Somatotropin replacement therapy was discontinued for 2 months, and IGF-1 value was measured, with an IGF-1 SDS of -3.2.[2] Based on the IGF-1 value and the presence of three other pituitary insufficiency, the patient was diagnosed with ongoing GH deficiency. GH therapy was resumed at a dose of 0.3 mg/day. MRI examination revealed hypoplastic adenohypophysis, thin pituitary stalk and ectopic neurohypophysis. Currently, the patient is continuing replacement therapy with somatotropin, levothyroxine, prednisone and combined estrogenic-progestogen replacement therapy, with a favourable outcome.

Results:
The benefits of GH substitution therapy during transition period and adult life were established and are not related to growth.[3] In such condition, nowadays it is recommended to evaluate the status of GH secretion during transition period and, if GH deficiency is confirmed, somatotropin substitution may continue during this period and adult life, too. The substitution dose should be closely monitored and adjusted based on IGF-1 values and taking into consideration the age-group specific dose[4,5].

Conclusion:
In patients with GH deficiency who are in the transition period to adult life, it is important and useful to consider the possibility that this deficiency may continue. In such cases, GH therapy has continued, being recognized as a safe and useful treatment, nowadays. Moreover, the positive metabolic effects of GH treatment are demonstrated till an elder age.

Keywords: *growth hormone, pediatrics, transition-period*

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ADULT LIFE DIAGNOSIS OF TURNER SYNDROME AND THE TREATMENT CHALLENGES ASSOCIATED WITH IT

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Background and Aims:
Turner syndrome is commonly associated with primary or secondary amenorrhea, due to gonadal dysgenesis, therefore, most patients will need hormonal replacement therapy

(HRT). Although HRT (preferably transdermal estradiol combined with 10 days of progestin monthly) is essential for maintaining secondary sexual characteristics, bone mineral density, cardiovascular health, cognitive and psychological well-being, adverse effects and contraindications should be considered.

Material and Methods, case presentation:

A 26-year-old woman, presented to the Endocrinology Clinic with 5 months of secondary amenorrhea, complaining of asthenia, fatigue, and weight gain. She was admitted to the Cardiology department in 2023 for multiple cardiovascular pathologies and in June 2025, stenting of the aortic isthmus was performed for coarctation of the aortic isthmus. The patient presented late spontaneous menarche at 18 years of age, with periods of several months of secondary amenorrhea, developed secondary sexual characteristics, and she was referred to Gynecology where estroprogestative therapy was initiated, which she followed for 3 months, but discontinued by the patient due to weight gain (reaching 69 kg). Due to the clinical picture (short stature 144 cm, facies with hypertelorism, short neck with low hair insertion, pigmented nevi) and the patient's history (periods of amenorrhea, cardiovascular disease), Turner syndrome was suspected. Blood pressure was 154/100 mmHg, heart rate 90 bpm. The evaluation of the hypothalamic-pituitary-gonadal axis confirmed hypergonadotropic hypogonadism (elevated FSH, LH, low estradiol, normal prolactin). Pelvic ultrasonography revealed normal appearance of the uterus (6.65/1.95 cm), linear endometrium, non-visualized ovaries. The karyotype analysis confirmed the diagnosis of Turner syndrome (45,X0). The laboratory showed LDL-cholesterol of 180 mg/dL, vitamin D 8.5 ng/dL, normal thyroid tests. The recommended treatment included ramipril, aspirin, clopidogrel, pantoprazole, and high doses of vitamin D.

Discussion:

Congenital heart disease occurs in about 75% of fetuses and 25–45% of live-born girls with Turner syndrome. The most common abnormalities are bicuspid aortic valve (16%) and coarctation of the aorta (11%). Aortic dissection occurs in 1-2% of the cases, relatively early in life at a median age of 35 years. One of the major contributing risk factors for cardiovascular events is hypertension, which affects up to 25% of adolescents and 40-60% of adults with Turner syndrome. In patients with this syndrome and associated cardiovascular abnormalities, particularly coarctation of the aorta, even after stenting, HRT requires careful individualization. Estrogen therapy, especially oral formulations, may increase thromboembolic risk and influence vascular function, which is of particular concern in the presence of hypertension and underlying aortic pathology. Turner syndrome is intrinsically associated with an increased risk of aortic complications, including dissection, and this risk may be further exacerbated by exogenous estrogen exposure.

Conclusions:

In this context, the indication for estrogen–progestin therapy should balance its well-established benefits on bone and metabolic health against potential cardiovascular risks. Although transdermal estrogen is generally considered safer due to a lower impact on coagulation pathways, evidence in Turner patients with complex cardiovascular disease remains limited. Therefore, therapeutic decisions should be made cautiously within a multidisciplinary team.

Key words: *Turner syndrome, amenorrhea, aortic coarctation, estroprogestatives, karyotype*

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AUTOIMMUNE DISEASES AND TYPE 1 DIABETES

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Background and Aims:
Type 1 diabetes mellitus (T1DM) is characterized by an autoimmune etiology, which render affected patients at increased risk for the development of additional autoimmune diseases. The most prevalent of these conditions are autoimmune thyroiditis and celiac disease.
Autoimmune thyroiditis is characterized by the presence of anti-thyroperoxidase antibodies and antithyroglobulin antibodies and may be associated with normofunction, hyperthyroidism or, most commonly, hypothyroidism. Serological screening plays an important role in the early identification of celiac disease in children with T1DM, in whom the condition is frequently asymptomatic.
Material and Methods:
We present 2 clinical cases of type 1 diabetes mellitus that have been associated with other autoimmune diseases since the time of diagnosis.
Results:
The first case involves a female patient who was diagnosed with T1DM at the age of 1 year and 10 months. At the time of diagnosis, anti-tissue transglutaminase IgA antibody levels were found to be tenfold higher than the upper limit of normal.
The second case concerns an 8 years old girl at the time of diagnosis of T1DM. Concomitantly, anti-tissue transglutaminase Ig A antibodies were detected at levels suggestive of celiac disease, along with the presence of anti-thyroid peroxidase antibodies associated with hypothyroidism.

Conclusions:
The presence of one or more additional autoimmune diseases in the same patient significantly impacts quality of life, potentially altering glucose metabolism and complicating diabetes management. Integrated treatment of coexisting autoimmune conditions can contribute to more favorable clinical outcomes.

Keywords: *autoimmune diseases, celiac disease, autoimmune thyroiditis, type 1 diabetes*

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THE REMISSION PHASE IN TYPE 1 DIABETES MELLITUS

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Background and Aims:
Remission in type 1 diabetes mellitus (T1DM) is characterized by a reduction in insulin requirements and stable glycemic control occurring shortly after the initiation of insulin therapy. Remission may be either partial or complete.

Material and Methods:
We report the case of a 13-year-old boy diagnosed with T1DM at the age of 11. Two months after disease onset, the glycemic profile allowed for complete discontinuation of insulin therapy, a condition that persisted for a period of two years.

Results:
The patient was diagnosed with T1DM at the age of 11, confirmed by elevated glycated hemoglobin, reduced C-peptide levels and positive anti-GAD antibodies. Basal bolus insulin

therapy was initiated. He benefited from a continuous glucose monitoring system, which facilitated glycemic control.

Two months after diagnosis, based on the glycemic profile, the patient completely discontinued insulin therapy, including rapid-acting and long-acting insulin. One year after the onset of the remission phase, immunological investigations were repeated, revealing positive titers of anti-islet cell antibodies and anti-IA2 antibodies. At present, the patient's glycated hemoglobin level is 5,8%, while receiving basal insulin therapy with occasional correction doses of rapid-acting insulin.

Conclusions:

Remission appears to be a beneficial prognostic factor in the subsequent course of T1DM, as preservation of beta-cell function may reduce the risk of vascular complications and severe hypoglycemia. A remission period persisting for more than three years may indicate possible misclassification and warrant consideration of other forms of diabetes mellitus.

Keywords: *complete remission, glycated hemoglobin, remission phase, type 1 diabetes*

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PAPILLARY THYROID CARCINOMA PRESENTING WITH DELAYED PUBERTY AND GROWTH IMPAIRMENT – CASE REPORT

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Background and Aims:

Papillary thyroid carcinoma (PTC) is the most common thyroid malignancy in children is frequently characterized by an indolent clinical course and subtle or absent local symptoms,

even in the presence of advanced locoregional disease. In pediatric patients, diagnosis may therefore be delayed, particularly when clinical attention is directed toward other endocrine concerns rather than targeted thyroid evaluation.Growth impairment and delayed pubertal development are among the most frequent reasons for referral to pediatric endocrinology services. While these conditions are usually investigated in the context of hypothalamic–pituitary or gonadal disorders, they may also represent indirect or competing clinical presentations that obscure the presence of clinically silent thyroid pathology. The coexistence of delayed puberty and short stature as the initial context in which PTC is detected is exceptionally rare and sparsely documented in the literature.

This report aims to describe an unusual presentation of pediatric papillary thyroid carcinoma in an adolescent male evaluated primarily for delayed puberty and borderline short stature, and to emphasize the importance of systematic thyroid assessment as part of the endocrine evaluation of adolescents presenting with growth or pubertal abnormalities.

Material and Methods:

We present the clinical case of a 14-year-old boy referred to a tertiary pediatric endocrinology centre for evaluation of delayed puberty and growth impairment. The diagnostic work-up included auxological assessment, pubertal staging, biochemical evaluation of thyroid function, and cervical ultrasonography. Surgical management, histopathological examination, radioactive iodine ablation, and postoperative follow-up were performed according to current pediatric thyroid cancer guidelines. Clinical, laboratory, and imaging data were analysed descriptively and interpreted in the context of published literature.

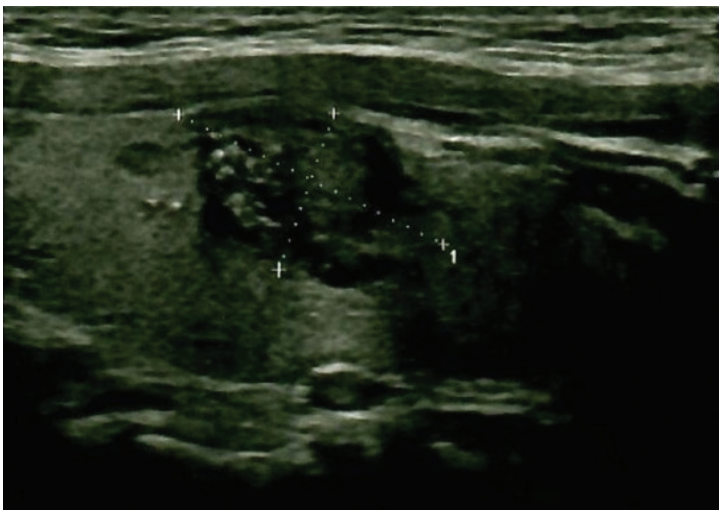

Fig 1. Greyscale ultrasonographic image of the left thyroid lobe showing a hypoechoic, irregular, micro lobulated solid–cystic nodule (15.4 × 8.2 mm) with punctate echogenic foci consistent with microcalcifications. According to ACR-TIRADS, this nodule was highly suspicious.

Results:

At presentation, the patient exhibited delayed puberty (Tanner stage I) and borderline short stature (–1.73 SDS), while thyroid function tests were within normal limits, and no

cervical compressive symptoms were reported. Physical examination did not reveal clinically significant thyroid enlargement. Cervical ultrasonography, performed as part of the endocrine evaluation, identified a solitary high-suspicion thyroid nodule with irregular margins and microcalcifications, classified as TI-RADS 5 (fig. 1). No distant metastatic lesions were detected at initial imaging.

Surgical intervention revealed an unencapsulated papillary thyroid carcinoma with mixed histological variants, associated with regional lymph node metastases. Based on histopathological findings and risk stratification, completion thyroidectomy with central neck lymph node dissection followed by adjuvant radioactive iodine ablation was performed in accordance with pediatric thyroid cancer guidelines. Post-therapeutic assessment demonstrated biochemical and imaging features suggestive of persistent disease. However, under TSH-suppressive levothyroxine therapy, serial follow-up showed a progressive and marked decline in serum thyroglobulin levels, reflecting a favorable early biochemical response and partial remission. Clinical follow-up did not reveal new local symptoms, and endocrine monitoring demonstrated stable thyroid hormone levels under suppressive treatment.

Conclusions:

Papillary thyroid carcinoma in adolescents may present atypically with growth impairment and delayed puberty, even in the absence of thyroid dysfunction or local symptoms. Such presentations may delay diagnosis if thyroid evaluation is not systematically included in the assessment of endocrine growth or pubertal disorders. A low threshold for thyroid ultrasonography should be considered in adolescents with unexplained growth failure or delayed puberty, allowing timely diagnosis and appropriate management of clinically silent but potentially advanced thyroid malignancy.

Keywords: *Delayed puberty; Growth impairment; Papillary thyroid carcinoma; Thyroid ultrasound.*

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PEDIATRIC OBESITY BETWEEN EXCESS WEIGHT AND VITAMIN D AND IRON DEFICIENCY

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Background and Aims:
Excess weight in childhood is one of the most pressing concerns in current medical practice, given the exponential increase in the number of cases of childhood obesity. Vitamin D and iron are micronutrients involved in the inflammatory process, current research showing the existence of minimal chronic inflammation in obesity. On the other hand, an inadequate diet also causes deficiencies in the organism. The aim of the study was to investigate the levels of these two micronutrients in normal-weight children and obese children.

Material and Methods:
The study was retrospective and included 64 children aged between 10 and 14 years, 20 of normal weight and 44 overweight, whose serum iron and 25-OH-vitamin D levels were investigated. The data collected included age, sex, background, serum iron levels, and 25-OH vitamin D levels. Patients were selected and evaluated during 2025. The inclusion criteria for the study were obesity (BMI ≥95th percentile, in accordance with WHO criteria for sex and age) for the study group, and for the control group, the inclusion criterion was normal weight (BMI ≤5th percentile but ≤85th percentile).The exclusion criteria were as follows: presence of autoimmune or inflammatory diseases, presence of endocrine or hereditary disorders, presence of other chronic conditions or liver diseases. Patients with iron deficiency anemia, malformations, infectious pathology in the last 3 months, immunocompromised patients, or those who had been treated with antibiotics or iron supplements in the last three months were also excluded.The analysis of clinical and epidemiological data was performed in an integrated manner, using the chi-square ($\chi 2$) comparison test in the SPSS10 program.

Results:
The clinical and epidemiological analysis of the group included 64 patients, 68.7% (n=44) obese, and 31.2% (n=20) control group. The group of obese patients had a mean age of 11.6±1.3 years, BMI 29.5±2.7, serum iron 17.3±6.7 µg/dl, vitamin D 19.6±9. 46 ng/mL. 81.1% of these patients had iron deficiency, and 88.6% had vitamin D deficiency, with the majority coming from urban areas (56.8%) and being male (59.1%). The group of normal-weight patients had a mean age of 11.35±0.9 years, BMI 17.8±0.7, serum iron 37.6±15.1 µg/dl, vitamin D 51.6±17.03 ng/mL. Twenty percent of these patients had iron deficiency, and 15% had vitamin D deficiency, with the majority coming from rural areas (75%) and being male (85%). Statistical analysis demonstrated a clear association between excess weight and both iron and vitamin D deficiency ($p<0.001$), as well as an association between the two deficiencies ($p<0.001$).

	Parameters % (no.cases)	Normal Weight	Obesity	P value (X^2 test)
Age		11.35±0.9	11.6±1.3	
Gender	Female	15% (3)	40.9% (18)	P=0.04
	Male	85% (17)	59.1% (26)	
Environment	Urban	25% (5)	56.8% (25)	P=0.01
	Rural	75% (15)	43.2% (19)	
BMI		17.8±0.7	29.5±2.7	
Serum Fe	µg/dl	37.6±15.1	17.3±6.7	P<0.001
	Deficit	20% (4)	81.8% (36)	
Vitamin D	ng/mL	51.6±17.03	19.6±9.46	
	Deficit	15%(3)	88.6%(39)	

Conclusions:

Research suggests that being overweight is linked to mineral deficiencies, including iron and vitamin D. Our study agrees with this idea, highlighting the importance of a healthy lifestyle not only to combat excess weight but also to maintain adequate levels of nutrients necessary for a child's growing body.

Keywords*: iron, pediatric obesity, vitamin D*

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FROM PITUITARY DYSFUNCTION TO SKELETAL DYSPLASIA: A CHALLENGING CASE OF SHORT STATURE

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Background:

Short stature has a broad differential diagnosis, encompassing growth hormone (GH) deficiency, chronic systemic disorders, malnutrition, and genetic syndromes.

Establishing the underlying cause may be particularly challenging in adults with delayed diagnosis, as GH levels in previously deficient individuals may overlap with those of healthy subjects.[1]

Genetic etiologies include syndromic conditions such as Prader–Willi syndrome, Turner syndrome, and Laurence–Moon syndrome, as well as skeletal dysplasia including chondrodysplasia and osteogenesis imperfecta.[2]

Osteogenesis imperfecta is an inherited connective tissue disorder characterized by increased bone fragility, recurrent fractures, short stature, and skeletal deformities.[3,4] Although its severity ranges from mild to perinatal lethal forms, the clinical presentation may be either overt or subtle, complicating timely recognition.

Methods:

We report a 55-year-old male presenting with disproportionate short stature, grayish-blue sclerae, multiple fractures, long bone deformities, joint hypermobility and atrophic scarring.

Results:

A 55-year-old male presented with a left femoral neck fracture following minimal trauma. *Clinical evaluation* revealed short stature (155 cm vs. mid-parental height 171.5 cm), disproportionate body segments, grayish-blue sclerae, sparse body hair and multiple atrophic scars. History was notable for childhood-onset partial hypopituitarism with poor adherence to hormone replacement, delayed puberty, congenital hip and radial head dislocation, delayed motor development, recurrent low-impact fractures, and adolescent joint hypermobility.

The patient reported being consistently shorter than his peers throughout childhood and adolescence and denied experiencing a significant pubertal growth spurt.

Hormonal assessment demonstrated central adrenal insufficiency, hypogonadotropic hypogonadism, normal growth hormone and IGF-1, and central hypothyroidism with positive anti-thyroid peroxidase antibodies.

Imaging revealed Wormian bones, codfish vertebrae, rib fractures and bilateral radial head dislocation.

Audiometry showed bilateral neurosensorial hearing loss. Given the constellation of skeletal fragility, hypermobility, and extra-skeletal features, a connective tissue disorder was suspected. Next-generation sequencing identified a likely pathogenic COL1A1 missense variant (c.940G>A; p.Gly314Arg), consistent with a COL1-related overlap disorder of osteogenesis imperfecta and Ehlers-Danlos syndrome.

Conclusions:
Skeletal dysplasia should be considered in the differential diagnosis of short stature, particularly in patients with low-impact fractures or joint hypermobility. In complex cases with coexisting endocrine deficiencies, hormonal and genetic evaluation is crucial to avoid misattribution and to ensure accurate diagnosis and early multidisciplinary management.
Keywords*: connective tissue disorder; hypopituitarism; osteogenesis imperfecta.*
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PREVALENCE AND AGE-RELATED VARIATIONS OF DYSLIPIDEMIA AMONG HOSPITALIZED CHILDREN IN A TERTIARY PEDIATRIC CENTER: A 5-YEAR RETROSPECTIVE STUDY

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Background and aims.
Dyslipidemia in childhood represents an early cardiovascular risk factor and may be influenced by growth, endocrine modulation, and developmental stage. Hospitalized pediatric populations reflect real-world clinical heterogeneity and may reveal important

patterns of lipid variability. The aim of this study was to evaluate the prevalence and age-related distribution of lipid parameters in children hospitalized in a tertiary referral pediatric center over a five-year period.

Material and Methods.

A retrospective cross-sectional study was conducted at the IMSP Mother and Child Institute, a tertiary pediatric referral center, including children hospitalized between 2020 and 2026.

All patients aged 0–18 years who had a lipid profile performed during admission were included. Data from institutional databases were merged and analyzed as a single cohort.

Collected variables included age, sex, total cholesterol (CT), LDL-cholesterol, and HDL-cholesterol. Age categories were defined as 0–5, 6–10, 11–15, and 16–18 years.

Statistical analysis included descriptive statistics, independent t-test for sex comparisons, ANOVA with Tukey post-hoc testing for age differences, chi-square testing for hypercholesterolemia prevalence (CT ≥5.2 mmol/L), and linear and quadratic regression models to assess age-related trends. Statistical significance was set at $p < 0.05$.

Results.

A total of 418 hospitalized children met inclusion criteria (52% boys, 48% girls). Mean lipid values were: CT 5.63 mmol/L, LDL 3.82 mmol/L, and HDL 1.45 mmol/L. Notably, hypercholesterolemia (CT ≥5.2 mmol/L) was identified in approximately 70% of patients. No statistically significant sex differences were observed for CT (p=0.12), LDL (p=0.22), or HDL (p=0.17), although girls showed slightly higher mean CT and LDL values. ANOVA demonstrated significant age-related differences for CT (p=0.0006) and HDL (p=0.0005), but not for LDL (p=0.107). Post-hoc analysis revealed significantly higher CT values in children aged 0–5 years compared to adolescent groups. Linear regression showed a modest but significant decline of CT with age ($\beta = -0.051$ mmol/L/year, p=0.023). HDL also decreased with age ($\beta = -0.026$ mmol/L/year, p=0.007). A quadratic model better described HDL dynamics (R^2=0.179), indicating a curvilinear developmental pattern. Despite numerical variation, hypercholesterolemia prevalence did not differ significantly between age groups (χ^2, p=0.21).

Conclusion.

In this 6-year retrospective analysis from a tertiary pediatric center, a high prevalence of hypercholesterolemia (≈70%) was observed among hospitalized children. Lipid parameters demonstrated significant age-related variability but no significant sex differences. Total cholesterol was highest in early childhood and declined with age, while HDL exhibited a curvilinear developmental pattern. These findings emphasize the importance of systematic lipid assessment and age-adapted interpretation in hospitalized pediatric populations.

Keywords: *age, dyslipidemia, hospitalized children, lipid profile, tertiary center*

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CLINICAL ASPECTS AND RESULTS OF GROWTH PROMOTING THERAPY IN TWO CHILDREN WITH ACAN GENE VARIANTS

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Background

Heterozygous ACAN pathogenic variants are an important monogenic cause of short stature, often associated with advanced bone age and familial inheritance,[1,2] with a broad phenotype. ACAN encodes aggrecan, essential for growth plate structure.[3] Patients may respond to recombinant human growth hormone therapy [2,4,5]

Case 1

The patient was referred for evaluation of short stature in the context of a positive maternal family history of short stature. Initial examination revealed a -3.3 height SDS, relative microcephaly, frontal bossing, and shortening of the limbs (diagnosed with campomelic dwarfism). Skeletal radiographs excluded achondroplasia while bone age (BA) was advanced compared to chronological age (CA). Further evaluation in the pediatric endocrinology department showed normal IGF-1 level (Z-score −1.02) and the dynamic tests excluded growth hormone deficiency. Molecular analysis was performed and identified a heterozygous pathogenic nonsense variant in the ACAN gene (c.1551C>G; p.Tyr517*). This variant is

predicted to introduce a premature stop codon resulting in truncated or absent aggrecan protein and was not reported in population databases. Considering the severe short stature, rhGH therapy was initiated. The patient demonstrated a favorable growth response, with a correction of heigh deficit of 1 DS in the first two years of treatment.

Case 2

A 10-year-4-month-old girl was admitted to our department for evaluation and therapy. The patient who resided in Sweden until the age of 7 y, was born small for gestational age (BW=1670 g at 37 weeks of gestation). Pregnancy was reported as physiologic, although fetal growth restriction was identified from 20 weeks' gestation, prompting amniocentesis with normal results. She was evaluated for short stature at age 4, without satisfactory catch-up growth (CUG). She presented dysmorphic features including short neck, hypertelorism and low-set inward-rotated ears. Growth hormone deficit was confirmed and genetic testing identified a de novo heterozygous mutation in the ACAN gene corresponding to variant c.757+2T> C. Considering the history of SGA without CUG, the presence of ACAN mutation and the GHD, rhGH treatment was initiated. One year later, she developed signs of early pubertal onset. The wrist radiography demonstrated advanced BA compared to the CA. The basal and stimulated levels of LH, Estradiol (including a GnRH stimulation test) confirmed the diagnosis of central precocious puberty. Treatment with a GnRH analogue (triptorelin) was initiated to delay pubertal progression and also, to allow correction of height deficit. The rhGH treatment ceased last year (for unclear reasons), but the GnRH treatment continued. At presentation in our clinic, the patient presented with short stature (−1.8 SDS relative to parental height SDS), dysmorphic features, pubertal stage I/II and 1 year advanced BA. Continuation of combined rhGH and GnRH analogue therapy was recommended.

Conclusions

These cases highlight the phenotypic variability associated with heterozygous ACAN pathogenic variants and underscore the importance of genetic testing in children with growth disorders. ACAN variants are recognized monogenic causes of short stature, typically associated with impaired linear growth and advanced bone age, sometimes accompanied by precocious puberty [1,2]. Treatment with rhGH has shown satisfactory outcomes.

Key words: *ACAN variants, advanced bone age, pediatric short stature, precocious puberty, rhGH therapy*

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JUVENILE HEMORRHAGIC METROPATHY IN A PATIENT WITH TURNER's SYNDROME - FROM ROUTINE TO DIAGNOSTIC CHALLENGE

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Introduction.

Turner's syndrome is defined by short stature, ovarian dysgenesis and absence of pubertal progression.

We report the rare case of a completely feminized patient (adult-type Tanner pubertal stage), including the onset of menstrual cycles at the age of 13.

Material and methods.

A 16-year-old patient was admitted in hemorrhagic shock after prolonged and heavy metrorrhagia. The lab results confirmed severe secondary regenerative anemia (Hb 4.4 g/dl, CHEM 31g/dl, VEM 71fL, reticulocytes 53$^{o}/_{oo}$). The extended hormonal balance provided normal values (FSH, LH, estradiol, progesterone, TSH, FT4, TPO Ab, IGF1). The banded karyotype confirmed the diagnosis of Turner's syndrome by mosaicism 45,XO/46,X,r(X)(p22.2-q28).

Conclusions.

Although most patients with Turner's syndrome are diagnosed with primary amenorrhea, 15-20% of them have been reported to have spontaneous menarche, a clinical aspect related to the particularity of the genetically detected structural anomaly. Even if our patient falls into this restricted category of patients with Turner syndrome, the mesomelic short stature present from birth and subsequently throughout the growth period, should have attracted the attention of the attending physicians before the onset of menstrual abnormalities.

Keywords*: juvenile hemorrhagic metropathy, mesomelic short stature, ring X chromosome, Turner syndrome.*

P.P. 23
TRANSITION FROM SEVERE INSULIN RESISTANCE TO AUTOIMMUNE INSULIN DEFICIENCY IN A CHILD WITH POLYGLANDULAR AUTOIMMUNITY: A METABOLIC–AUTOIMMUNE ENDOTYPE

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Background and Aims

The coexistence of insulin resistance and pancreatic autoimmunity in children represents an emerging and still poorly defined metabolic–immune endotype[1,2].

While type 1 diabetes is traditionally characterized by autoimmune β-cell destruction and type 2 diabetes by insulin resistance, some pediatric patients present with a hybrid phenotype involving both mechanisms[3,4].

We report a complex case demonstrating progression from severe insulin resistance with hyperinsulinemia to overt autoimmune insulin deficiency in the setting of familial polyautoimmunity[5].

Material and Methods

We conducted a longitudinal analysis of clinical, metabolic, immunologic, and familial data in a boy diagnosed with autoimmune thyroiditis at age 11 and treated with levothyroxine. Serial evaluations included fasting glucose, HbA1c, insulin, C-peptide, HOMA-IR, pancreatic autoantibodies (anti-GAD, anti-insulin, IA-2), thyroid antibodies, lipid profile, renal parameters, and vitamin D levels. A four-generation pedigree was constructed to assess familial clustering of metabolic and autoimmune disorders.

Results

At 12 years and 9 months, the patient presented with obesity (BMI 27.7 kg/m², >95th percentile), impaired fasting glucose (103 mg/dL), markedly elevated fasting insulin (97.5 µU/mL), and profound insulin resistance, consistent with severe compensatory hyperinsulinemia without overt diabetes (HbA1c 5.7%).

Over the subsequent 18 months, fasting glucose progressively increased to 237 mg/dL, and HbA1c rose to 9.3%, indicating poorly controlled diabetes. Initially elevated C-peptide (11 ng/mL) declined to 0.75 ng/mL, reflecting transition from hyperinsulinemia to significant β-cell dysfunction.

Autoimmune testing showed strongly positive anti-GAD (>2000 UI/mL) and anti-insulin antibodies (>40 U/mL), with negative IA-2. The combination of marked hyperinsulinemia, high anti-insulin titers, and glycemic variability suggested an autoimmune insulin syndrome–like presentation overlapping with evolving autoimmune diabetes. Thyroid autoimmunity persisted (anti-TPO and anti-TG positive), confirming polyglandular autoimmunity. Persistent vitamin D deficiency was noted throughout follow-up.

Current therapy consists of a basal–bolus insulin regimen (rapid-acting insulin 16-16-16 U and basal insulin 27 U daily), with suboptimal metabolic control (HbA1c 8.9–9.3%).

Family history demonstrated strong aggregation of diabetes (both adult-onset and type 1), autoimmune thyroid disease, Graves disease, ankylosing spondylitis (HLA-B27 positive), and autosomal dominant polycystic kidney disease, supporting a genetic susceptibility to immune dysregulation.

Conclusions

This case highlights a dynamic metabolic–autoimmune trajectory from severe insulin resistance with compensatory hyperinsulinemia to progressive autoimmune β-cell failure. The coexistence of high anti-GAD and anti-insulin antibodies suggests an intermediate pre-insulinopenic autoimmune phase, potentially resembling autoimmune insulin syndrome prior to overt insulin dependence.

Recognition of this hybrid endotype has important implications for early screening of first-degree relatives, genetic counseling, and integrated immune–metabolic monitoring. Such cases may challenge the traditional T1D/T2D dichotomy and support the development of personalized preventive strategies in families with polyautoimmunity.

Keywords: *autoimmune diabetes, insulin resistance, anti-GAD antibodies, anti-insulin antibodies, polyglandular autoimmunity*

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P.P. 24

WHEN AUTOIMMUNITY STRIKES TWICE: RECURRENT ACALCULOUS CHOLECYSTITIS IN TYPE 1 DIABETES AND THYROIDITIS

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Background and Aims

Autoimmune polyendocrine syndromes encompass a spectrum of immune dysregulation characterized by the coexistence of multiple autoimmune disorders[1]. Type 1 diabetes mellitus (T1DM) and autoimmune thyroiditis frequently cluster and may precede additional endocrinopathies, including adrenal insufficiency[2,3]. Recurrent systemic inflammatory episodes accompanied by cytopenias, hepatic dysfunction, and acute acalculous cholecystitis (AAC) raise suspicion of broader immune dysregulation beyond classical polyglandular autoimmunity[4,5]. We report the case of a patient with T1DM and autoimmune thyroiditis who developed recurrent inflammatory episodes with hepatic and biliary involvement, aiming to explore potential underlying mechanisms.

Material and Methods

We conducted a retrospective clinical analysis of a female patient with known T1DM and autoimmune thyroiditis who experienced recurrent inflammatory episodes between 2013 and 2024. Clinical, laboratory, imaging, endocrine, immunological, and genetic data were reviewed.

Results

Between 2013 and 2024, the patient experienced nine recurrent episodes characterized initially by alimentary vomiting progressing to bilious vomiting, fever, and abdominal pain.

Laboratory findings during episodes showed normocytic hypochromic anemia, leukopenia with persistent lymphopenia (or normal leukocyte counts with lymphopenia), and elevated inflammatory markers (CRP, ESR, fibrinogen, ferritin). Hepatic involvement was consistent, with evidence of cytolysis and cholestasis. Intermittent hematuria was documented outside menstrual periods.

Abdominal imaging during several episodes demonstrated acute acalculous cholecystitis without gallstones, hepatomegaly, and transiently increased liver stiffness (FibroScan F1), which subsequently normalized (F0).

Two positive sweat chloride tests (77 and 87 mmol/L) were recorded; however, cystic fibrosis was excluded based on negative CFTR gene sequencing. The coexistence of T1DM and autoimmune thyroiditis places the patient within the spectrum of Autoimmune Polyglandular Syndrome type II, with possible evolving adrenal insufficiency.

Given the persistent lymphopenia and recurrent systemic inflammation, the differential diagnosis includes seronegative autoimmune hepatitis, immune-mediated cholangiopathy, common variable immunodeficiency (CVID), and an attenuated IPEX-like syndrome. The positive sweat tests in the absence of CFTR mutations raise the possibility of hypoaldosteronism contributing to electrolyte dysregulation.

Conclusions

Recurrent acute acalculous cholecystitis associated with hepatic cytolysis and systemic inflammation in a patient with established polyautoimmunity suggests an underlying immune dysregulation rather than isolated biliary disease. Autoimmune Polyglandular Syndrome type II with potential latent adrenal insufficiency remains the leading hypothesis; however, concomitant immune dysfunction or primary immunodeficiency should be carefully excluded. A multidisciplinary diagnostic approach is essential to clarify the pathophysiology and guide management.

Keywords: *Autoimmune polyglandular syndrome type II; acute acalculous cholecystitis; type 1 diabetes mellitus.*

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