17—20 September 2026 Vodice, Croatia

CROATIAN SUMMER SCHOOL OF ENDOCRINOLOGY

Hrvatsko endokrinološko društvo
Croatian Endocrine Society

Cleveland Clinic

IMPRESSUM

Croatian Summer School of Endocrinology

Vodice, 17—20 September 2026

Organisers:

Croatian Endocrine Society

Cleveland Clinic

J. J. Strossmayer University of Osijek, Faculty of Medicine

Croatian Endocrine Society

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Tamara Turk Wensveen (HR)
Felix Wensveen (HR)
Przemysław Witek (PL)
Divya Yogi-Morren (US)

WELCOME LETTER

Dear Colleagues and Participants,

It is our great pleasure to welcome you to the **Croatian Summer School of Endocrinology 2026**, an international postgraduate educational event jointly organised by the **Cleveland Clinic and the Croatian Endocrine Society**, taking place at Hotel Olympia Sky in Vodice, Croatia, from 17 to 20 September 2026.

Over the years, the Croatian Summer School of Endocrinology has become one of the most recognised regional scientific meetings in endocrinology and metabolic medicine, bringing together clinicians, researchers, trainees, and healthcare professionals from across Europe. Following the success of previous editions, this year's meeting will continue to foster excellence in medical education, interdisciplinary collaboration, and international professional exchange. The scientific programme will feature distinguished faculty members from **Cleveland Clinic, Ohio, USA**, **Cleveland Clinic London, UK**, and leading European university hospitals and clinical centres. Through interactive lectures, workshops, and clinically oriented **Endo Case Lab** sessions, participants will exchange cutting-edge knowledge and practical experience relevant to contemporary endocrine practice.

The Summer School also continues its commitment to supporting young physicians and future leaders in endocrinology through educational initiatives and international fellowship opportunities, including **two one-month preceptorship awards at Cleveland Clinic, Ohio, USA**.

Beyond the scientific programme, we hope the Summer School will provide an inspiring environment for professional exchange, new collaborations, and stronger connections among colleagues from Croatia and abroad.

We are delighted to welcome you to the **Croatian Summer School of Endocrinology 2026** and look forward to several stimulating and rewarding days of education, discussion, and collegial exchange in Vodice.

Welcome to Vodice, and we wish you an enjoyable and successful Summer School!

Silvija Canecki-Varžić, MD, PhD

President of Croatian Endocrine Society of the Croatian Medical Association

Associate Professor, J. J. Strossmayer University of Osijek Faculty of Medicine

Croatia

Mario Škugor, MD, FACE

Director of Education, Endocrine and Metabolic Institute, Cleveland Clinic

Clinical Assistant Professor, Cleveland Clinic Lerner College of Medicine, CWRU

United States

PROGRAM

THURSDAY 17.09.

17:00–17:30 WELCOME RECEPTION AND REGISTRATION

17:30–17:45 OPENING CEREMONY | **Tomislav Mihaljevic, Mario Skugor, Silvija Canecki-Varžić**

Session 1: BONE
Moderators: **Daniela Fabris Vitković** (HR), **Mladen Krnić** (HR)

17:45–18:10 Beyond Postmenopausal Bone Loss: The Endocrine Spectrum of Secondary Osteoporosis | **Mario Skugor** (US)

18:15–18:40 Unraveling Calcium Disorders: Real Cases, Real Insights | **Mario Skugor** (US)

18:40–19:00 Q&A | **Daniela Fabris Vitković** (HR), **Mladen Krnić** (HR)

FRIDAY 18.09.

Session 2: REPRODUCTIVE & WOMEN'S HEALTH — PART 1
Moderators: **Mojca Jensterle Sever** (SLO), **Suhaniya Samarasinghe** (UK), **Ivan Jandrić** (HR)

08:30–08:55 Obesity and Infertility: Endocrine Mechanisms and Clinical Implications | **Tommaso Falcone** (US)

09:00–09:25 From Excess to Deficiency: The Spectrum of Female Reproductive Endocrine Disorders | **Suhaniya Samarasinghe** (UK)

09:30–09:55 Fertility Preservation in Cancer Patients | **Tommaso Falcone** (US)

Session 3: ENDOCRINE CARDIOLOGY
Moderators: **Ines Bilić-Ćurčić** (HR)

10:00–10:25 When Hormones Damage the Heart: Cardiomyopathies in Endocrine Disease | **Elmir Omerovic** (SWE)

10:30–11:00 COFFEE BREAK

Session 4: PITUITARY & RELATED DISORDERS — PART 1
Moderators: **Dubravka Jurišić-Eržen** (HR), **Gorana Mirošević** (HR)

11:00–11:25 Growth Hormone Replacement During Lifespan — Efficacy and Safety | **Mirjana Doknić** (SRB)

11:30–11:55 Cushing's Disease: Beyond Cortisol Control — Optimizing Treatment with Osilodrostat | **Przemysław Witek** (POL)

Session 5: THYROID
Moderators: **Tomislav Jukić** (HR)

12:00–12:25 Challenging Thyroid Cases: Insights from Clinical Practice | **Mario Skugor** (US)

ENDO CASE LAB: PITUITARY, THYROID & BONE
Moderators: **Przemysław Witek** (POL), **Mirjana Doknić** (SRB), **Mario Skugor** (US)

12:30–12:40 Osilodrostat for Cushing's Disease in a Patient with Type 1 Diabetes | **Andrijana Koceva** (SI)

12:40–12:50 A Case of Acromegaly with Plurihormonal Secreting Adenoma | **Silvia Vandeva** (BG)

12:50–13:00 A Pituitary Mass with a Twist: Hypophysitis Mimicking a Macroadenoma | **Kristina Steiner** (HR)

13:00–13:10 When Hashimoto's Turns into Graves': A Rare Autoimmune Thyroid Transition | **Jakov Matas** (HR)

13:10–13:20 Hormonal Profiles in Females with Allergic Diseases: An Integrated Analysis of Thyroid Hormones, Androgens, Familial Atopy, and Aeroallergen Sensitization | **Violeta Lokaj-Berisha** (KS)

13:20–13:30 Thyroid — What Lurks in the Dark? | **Dubravka Vrljić Borojević** (HR)

13:30–13:35 MODERATOR SYNTHESIS | **Przemysław Witek** (POL), **Mirjana Doknić** (SRB), **Mario Skugor** (US)

13:35–14:30 LUNCH BREAK

Session 6: ADRENAL
Moderators: **Tamara Turk Wensveen** (HR), **Marina Gradišer** (HR)

14:30–14:55 Pheochromocytoma and PGLs: A Changing Perspective and Current Concepts | **Ricardo Correa** (US)

15:00–15:25 Benign or Malignant? Navigating the Differential Diagnosis of Adrenal Lesions | **Ricardo Correa** (US)

ENDO CASE LAB: ADRENAL & ELECTROLYTES
Moderators: **Ricardo Correa** (US), **Tamara Turk Wensveen** (HR), **Marina Gradišer** (HR)

15:30–15:40 Ectopic ACTH Syndrome: Personalizing Treatment in a Complex Clinical Setting | **Ana Uroš** (HR)

15:40–15:50 Severe Hyponatremia Associated With Hypophysitis | **Marina Andrešić** (HR)

15:50–16:00 Bilateral Adrenal Infarction in Pregnancy: A Rare and Life-Threatening Clinical Challenge | **Ivana Irma Lerotić** (HR)

16:00–16:10 Incidence and Clinical Characteristics of Mild Autonomous Cortisol Secretion (MACS) | **Klara Džojić** (HR)

16:10–16:20 From Pheochromocytoma to Macroprolactinoma | **Ema Schönberger** (HR)

16:20–16:30 MODERATOR SYNTHESIS Ricardo Correa (US), **Tamara Turk Wensveen** (HR), **Marina Gradišer** (HR)

16:30–17:00 COFFEE BREAK

Session 7: Translational Endocrinology

Moderators: **Elmir Omerovic** (SWE), **Felix Wensveen** (HR)

17:00–17:30 Bridging Basic and Clinical Research: Methodological Principles in Translational Endocrinology | **Tamara Turk Wensveen** (HR), **Felix Wensveen** (HR)

17:35–18:00 Meta-Analysis in Clinical Medicine: Core Concepts and Interpretation | **Eduard Oštarijaš** (HR)

SATURDAY 19.09.

Session 8: OBESITY & CARDIOMETABOLIC

Moderators: **Barbara McGowan** (UK), **Maja Čavlović** (HR), **Sanja Klobučar** (HR)

08:30–08:55 Obesity as an Immunometabolic Disease | **Barbara McGowan** (UK)

09:00–09:25 GLP-1-Based Therapies Across the Female Lifespan | **Mojca Jensterle Sever** (SLO)

09:30–09:55 GLP-1 Receptor Agonists: Effects Beyond Obesity and Type 2 Diabetes | **Barbara McGowan** (UK)

10:00–10:25 CVOTs in 2026: From Evidence to Organ Protection | **Dipesh Patel** (UK)

10:30–11:00 COFFEE BREAK

Session 9: ANDROLOGY & MEN'S HEALTH

Moderators: **Jelena Vučak Lončar** (HR), **Kristina Kljajić** (HR)

11:00–11:25 Pathological vs Functional Male Hypogonadism: Compare and Contrast | **Richard Quinton** (UK)

11:30–11:55 Obesity and Men's Health | **Dipesh Patel** (UK)

12:00–12:25 Androgen Deficiency in CKD: Time to Treat? | **Richard Quinton** (UK)

ENDO CASE LAB: REPRODUCTION & METABOLISM

Moderators: **Richard Quinton** (UK), **Dipesh Patel** (UK)

12:30–12:40 Early Medical Complications During Stroke Recovery: GLP-1 RA versus SGLT-2 Inhibitors | **Marina Gradišer** (HR)

12:40–12:50 Labor as a Trigger of Chromogranin A Processing: Implications for Metabolic and Neuroendocrine Adaptations in Gestational Diabetes | **Ivka Djaković** (CH)

12:50–13:00 Metformin-Associated Lactic Acidosis in Critically Ill Patients: A Single-Centre Experience | **Nikolina Marić** (HR)

13:00–13:10 Normospermia Despite Testosterone Replacement Therapy: An Unexpected Clinical Finding | **Božo Smajić** (HR)

13:10–13:20 Secondary Amenorrhea in a 20-Year-Old Female with Active Systemic Lupus Erythematosus: A Therapeutic Challenge | **Maja Mizdrak** (HR)

13:20–13:30 Metabolic Syndrome & Pregnancy | **Filip Paukoviić** (HR)

13:30–13:35 MODERATOR SYNTHESIS | **Richard Quinton** (UK), **Dipesh Patel** (UK)

13:35–14:30 LUNCH BREAK

Session 10: REPRODUCTIVE & WOMEN'S HEALTH — PART 2

Moderators: **Ivan Jandrić** (HR)

14:30–14:55 Reframing Menopause: The Role of Tailored Hormone Replacement Therapy | **Umasuthan Srirangalingam** (UK)

Session 11: ELECTROLYTES

Moderators: **Dipesh Patel** (UK)

15:00–15:25 Between Thirst and Balance: Polyuria, Polydipsia, and Sodium Disorders | **Umasuthan Srirangalingam** (UK)

Session 12: PITUITARY & RELATED DISORDERS — PART 2

Moderators: **Przemysław Witek** (POL), **Mirjana Doknić** (SRB)

15:30–15:55 An Update on Hypophysitis | **Divya Yogi-Morren** (US)

16:00–16:25 Incidentalomas Reconsidered: Are We Managing Pituitary Lesions Differently in 2026? | **Divya Yogi-Morren** (US)

Session 13: NEUROENDOCRINE TUMORS

Moderators: **Umasuthan Srirangalingam** (UK)

16:30–17:00 Neuroendocrine Tumours, Diagnosis and Treatment: Where Are We Now? **Ashley B. Grossman** (UK)

17:00–17:30 COFFEE BREAK

ENDO CASE LAB: CALCIUM METABOLISM & NET

Moderators: **Umasuthan Srirangalingam** (UK), **Mario Skugor** (US)

17:30–17:40 When Fragile Bones Lead to the Pituitary: A Tale of Two Osteoporosis Cases | **Karla Lauš** (HR)

17:40–17:50 Primary Hyperparathyroidism Presenting with a Full Spectrum of Complications and Hypertrophic Cardiomyopathy | **Barbara Themel** (HR)

17:50–18:00 Lithium-Induced Hyperparathyroidism | **Kristijan Lipovac** (HR)

18:00–18:10 Restoring Physiological Calcium Balance: Patient Journeys with Palopegteriparatide | **Manuela Ferjančić** (HR)

18:10–18:20 Iatrogenic Hypercalcemia in a Patient with Type 2 Diabetes Treated with a GLP-1 Receptor Agonist and Chronic Kidney Disease | **Maja Mačković** (HR)

18:20–18:30 From Non-Functioning to Functioning: Hyperinsulinemic Hypoglycemia in Metastatic Pancreatic NET | **Matea Majetić** (HR)

18:30–18:40 MODERATOR SYNTHESIS | **Umasuthan Srirangalingam** (UK), **Mario Skugor** (US)

SUNDAY 20.09.

Session 14: DIABETES & METABOLISM

Moderators: **Maja Bakula** (HR), **Nataša Moser** (HR), **Tina Tičinović Kurir** (HR), **Tomas Matić** (HR)

08:30–08:55 Rare Lipid Disorders: Modern Management of HoFH, FCS, and Beyond | **Ivan Pećin** (HR)

09:00–09:25 Advances in Continuous Ketone Monitoring: Clinical Applications and Future Perspectives | **Ines Bilić-Ćurčić** (HR)

09:30–09:55 Before It Begins: Transforming Type 1 Diabetes Through Early Screening and Intervention | **Mladen Grgurević** (HR)

10:00–10:25 Metformin-Associated Lactic Acidosis: Clinical Reality or Persistent Myth? | **Mladen Krnić** (HR)

10:30–10:55 Next-Generation Anti-Obesity Therapies: Science, Innovation, and Clinical Impact | **Silvija Canecki-Varžić** (HR)

11:00–11:15 CLOSING REMARKS
Preceptorship Award Announcement
Mario Skugor, Silvija Canecki-Varžić

11:30–12:30 FAREWELL LUNCH

BIOGRAPHIES

INES BILIĆ-ĆURČIĆ

Ines Bilić-Ćurčić, MD, PhD, is an Associate Professor, Vice Dean for Postgraduate studies at the Faculty of Medicine in Osijek, and attending physician at University Hospital Osijek in the Department of Endocrinology. She graduated from the Faculty of Medicine in Osijek, the University of Zagreb in 2003 with an M.D. From the Postgraduate study in biomedicine, Faculty of Medicine, the University of Osijek with a Ph.D. in 2006.

From 2003 to 2005 she was a postdoctoral fellow at the Department of Genetics and Developmental Biology, University of Connecticut Health Center, the USA acquiring the experience and skills necessary for research and lab work. Since 2005 she has also been working at the University of Osijek, Faculty of Medicine, in various positions ranging from associate Junior Assistant to part-time Associate Professor. In May 2021 she was appointed the Head of the Department of Pharmacology and in October 2021 she was appointed Vice Dean for Postgraduate studies. Currently, she is a lecturer in Pharmacology courses for undergraduate medical and biomedical laboratory diagnostics students and Ph.D. students.

Her field of interest is basic and clinical endocrinology, specifically bone and metabolic disorders and the prevention of complications and is the principal investigator of several institutional research projects. She participates as an invited lecturer at international and domestic scientific meetings. In June 2023. she was elected 1st vice-president of the Croatian Endocrinological Society. From 2022 she is the mentor of the student section for endocrinology and diabetology ENDOS. She also participates as a reviewer of numerous international scientific journals and international scientific projects, actively participates in the popularization of science and has proven herself as a successful mentor by supervising graduate and doctoral theses. Scientific work has so far resulted in the publication of over 70 papers cited in JCR/WoSCC, SJR/Scopus and Medline.

SILVIJA CANECKI-VARŽIĆ

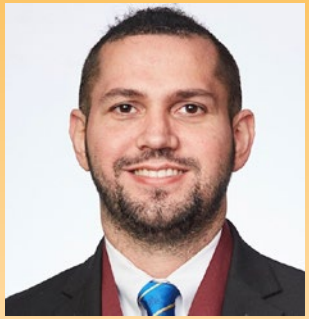
Professor Silvija Canecki-Varžić, MD, PhD, is a senior academic clinician whose work spans endocrinology, diabetology, obesity, osteoporosis and cardiometabolic medicine. She combines clinical leadership, undergraduate and postgraduate teaching, guideline development, research mentorship and extensive experience in international clinical trials.

She graduated from the University of Zagreb School of Medicine in 1996, completed specialist training in internal medicine in 2002 and subspecialist training in endocrinology and diabetology in 2007. She earned her PhD at the Faculty of Medicine Osijek in 2013 with a dissertation on cytokine gene polymorphisms in type 2 diabetes. In 2022, she was appointed Senior Research Associate in the field of biomedicine and health sciences. Her research focuses on diabetes technologies and glycaemic variability, obesity and novel pharmacotherapies, cardio-kidney-metabolic risk, adrenal and pituitary disorders, osteoporosis and metabolic bone disease. She has authored or co-authored more numerous scientific papers indexed in international databases and has contributed to more than 50 phase II-IV clinical trials including major cardiovascular and diabetes outcome trials as principal investigator and national coordinator.

Professor Canecki-Varzic is a chief mentor for specialist training in internal medicine and in endocrinology and diabetology. She supervises doctoral and graduate research and teaches in undergraduate, specialist and postgraduate programmes. She has led and co-led international postgraduate programmes, including the Croatian Summer School of Endocrinology and courses developed in collaboration with Cleveland Clinic. She is an editor and lead author of national guidelines, consensus statements and clinical handbooks, including Croatian guidance on acromegaly, osteoporosis and obesity care. Her work is dedicated to translating contemporary endocrine science into high-quality clinical practice, multidisciplinary education and improved patient outcomes.

Current Appointments:

- Head, Department of Internal Medicine, University Hospital Centre Osijek, Croatia
- Head, Division and Regional Centre for Endocrinology, Diabetes and Metabolic Diseases, University Hospital Centre Osijek
- Associate Professor, Josip Juraj Strossmayer University of Osijek
- President, Croatian Endocrine Society
- Second Vice-President, Croatian Society for Diabetes and Metabolic Disorders



RICARDO CORREA

Ricardo Correa, MD, EdD, FACP, FACE, FACMQ, FAPCR, is a Clinical Professor of Medicine, the Director for Diversity, equity and Inclusion, for mentoring and Belonging and the Director for Longitudinal Curriculum at The Lerner College of Medicine and the School of Medicine of Case Western Reserve University. He is the Program Director for the Endocrinology, diabetes and metabolism fellowship and the Director for health equity and Inclusive Initiatives at the Cleveland Clinic in Cleveland, Ohio. In addition he was the health equity fellowship director for Creighton School of Medicine Phoenix and battalion surgeon for the 492 civil affair unit of the army reserve. He is an editor and peer reviewer of multiple journals in endocrine, research and medical education. He has more than 100 article published in peer-review journals.

He has been awarded by the Presidential leadership scholarship, SGIM Herbert Nicken award, ASP Eric Nielsen award, Arizona humanitarian award, AACE and Medscape rising star award, NMQF 40 under 40 award among many others.

MIRJANA DOKNIĆ

Mirjana Doknić, MD, PhD, is a full Professor of Internal Medicine and Endocrinology at the Faculty of Medicine, University of Belgrade, Serbia. She graduated and obtained PhD degree at the same institution. Dr. Doknić has been employed in the Department of Neuroendocrinology at Clinic of Endocrinology, Diabetes and Metabolic Diseases, University Clinical Center of Serbia. The center where she has been working belongs to the group of pituitary tumor centers of excellence (PTCOE).

Her main fields of interest are pituitary tumors, the medical treatment of acromegaly and Cushing syndrome, hypopituitarism, long-term endocrine and metabolic consequences of childhood cancer survivors in adults. She is a pioneer in the formation of the so-called "transition units" in the Balkan region to facilitate the transfer of patients with pituitary diseases from pediatrician to adult endocrinologist. Professor Doknić has been actively involved in several domestic and international projects Professor Doknić is actively involved in several international projects dealing with neuroendocrinology diseases: 1) Project of mapping the genetic basis of congenital hypopituitarism in the region; 2) Study of adherence to growth hormone therapy during the transition period in cooperation with the neuroendocrine units of 15 faculties in Italy; 3) Hypopituitarism ENEA - Rare Observational Study (HEROS study); 4) ENEA Resistant Prolactinoma Study; 5) Therapeutic options and risk factors for the onset and progression of Graves' orbitopathy, endorsed by the Ministry of Science, Technological Development and Innovation of the Republic of Serbia.

Professor Doknić has published more than 80 peer-reviewed articles in the field of neuroendocrinology, endocrine and metabolic disorders and hypothalamic/pituitary pathologies. She was the principal investigator in several projects related to acromegaly and growth hormone deficiency, sponsored by Crinetics, Ascendis, Pfizer, Camurus. Professor Doknić has been a reviewer in the following journals: Pituitary, European Journal of Endocrinology, Cancer, Journal of Endocrinological Investigation, Journal of Clinical Medicine, Endocrine, Expert Review of Endocrinology and Metabolism, American Association Clinical Endocrinology Case Report (AACE), Growth Hormone & IGF Research and a member of the Review Editorial board of the journal "Frontiers in Endocrinology".

TOMMASO FALCONE

Tommaso Falcone, MD, FRCS(C), FACOG, is Executive Vice President and President of International and Emerging Markets. In this role, Dr. Falcone leads and executes Cleveland Clinic's strategic initiatives in established and emerging international markets. This includes enterprise-wide Global Patient Services, International Operations, Cleveland Canada and Cleveland Clinic Nevada. He also works closely with leaders at Cleveland Clinic London and ClevelandClinic Abu Dhabi.

Previously, Dr. Falcone was Chief of Staff at Cleveland Clinic London before becoming its interim president in June 2023, where he led the transition from design phase to operational hospital. Dr. Falcone graduated from McGill University School of Medicine in Montreal, Quebec. He then completed a residency in obstetrics and gynecology and a fellowship in reproductive endocrinology at McGill. He is certified by the American Board of Obstetrics and Gynecology and by the Royal College of Physicians and Surgeons of Canada and was appointed Fellow (ad eundem) of the Royal College of Obstetricians and Gynaecologists of the United Kingdom.

Dr. Falcone has published more than 500 original manuscripts, abstracts, and book chapters. He is co-editor of 8 books and has served on the editorial board of several journals including Obstetrics & Gynecology. He is past President of the Society for Reproductive Surgeons and Past Chair of the Endometriosis special interest group of the American Society of Reproductive Medicine. He has served on review committees for the National Institute of Child Health and Human Development and MRC. Dr. Falcone has been the recipient of many awards for laparoscopic and robotic surgery at different societies.

Dr. Falcone continues to serve as Professor of Obstetrics, Gynecology and Reproductive Biology at Cleveland Clinic Lerner College of Medicine of Case Western Reserve University. Dr. Falcone is the senior author on the first publication in the world on gynecologic robotic surgery and is a member of the team that performed the first uterus transplant in the U.S.

MLADEN GRGUREVIĆ

Mladen Grgurević, MD, PhD, completed his medical degree at the University of Zagreb in 2005. He is a specialist of internal medicine, endocrinologist and diabetologist.

He completed PhD degree focusing on depression and glycaemic control among people with diabetes in 2020. He is the author and co-author of several scientific and professional papers and conference abstracts in the field of diabetology and is also a member of the Croatian Society for Diabetes and Metabolic Diseases, Croatian Endocrine Society and the European Association for the Study of Diabetes (EASD).

He is working at the University Clinic Vuk Vrhovac, Merkur Clinical Hospital, Zagreb with a special interest in intensive insulin treatment, the use of insulin pumps and sensors for continuous glucose measurement (CSII, CGM, AHCL), from which he was in professional and clinical training at several foreign institution (General University Hospital In Prague, 3rd Internal Clinic, Prague, Czech Republic; Center for Diabetes and Metabolism – Fachklinik – Bad Heilbrunn, Germany).

ASHLEY GROSSMAN

Professor Ashley Grossman FMedSci was originally at St Catharine's College, Cambridge, then completed a degree in Psychology and Social Anthropology at the University of London, and then started a medical degree at University College London where he received a First-Class degree in Anatomy, and the University Gold Medal. He became Professor of Neuroendocrinology at St. Bartholomew's Hospital in 1993, then Professor of Endocrinology at the University of Oxford, and Senior Research Fellow of Green-Templeton College. He is currently Consultant in Neuroendocrine Tumours at the Royal Free Hospital, London, and is also Professor of Endocrinology at Barts and the London School of Medicine.

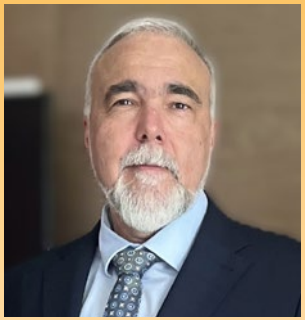
He has received many international awards, and has published more than 580 peer-reviewed papers and over 470 chapters and reviews, most recently on hypothalamic and pituitary tumours and endocrine oncology. He has been President of the ENEA, the Society for Endocrinology (2012-2014), Chairman of UKINETS (2010-2012), President of the Pituitary Society (2015-2016), and is President of Endotext. He has received a number of awards including the Geoffrey Harris Award in Neuroendocrinology, European Society for Endocrinology (2014), the Iain MacIntyre Prize, Barts and the London School of Medicine (2017), Outstanding Mentor Laureate, American Endocrine Society (2020), Rolf Gaillard Award, ENEA (2024), Society for Endocrinology Jubilee Medal (2025), Endocrine Society/ESE Transatlantic Alliance Award (2025) and the ENETS Lifetime Achievement Award (2026).

MOJCA JENSTERLE SEVER

Prof. Mojca Jensterle, MD, PhD, is a consultant endocrinologist at the University Medical Centre Ljubljana, Head of the Endocrine Outpatient Clinics, and Professor of Internal Medicine at the University of Ljubljana. She completed her postdoctoral training in Endocrinology and Metabolism at the Academic Medical Center, University of Amsterdam.

Her research focuses on the pharmacological treatment of obesity and polyendocrine metabolic ovarian syndrome (PMOS), with particular expertise in incretin-based therapies. She leads and participates in national and international clinical trials and research projects. Over the past decade, she and her team have conducted clinical trials investigating the effects of incretin-based therapies on gastric emptying, taste perception, reproductive and metabolic outcomes, brown adipose tissue activation, muscle function, and bone health. She co-led the development of the Slovenian national guidelines for the pharmacological treatment of obesity in adults.

Prof. Jensterle has published more than 120 SCI-indexed papers and has a ResearchGate h-index of 38, with over 4,400 citations in the past decade. She is an active member of the ESE and AE-PCOS Society and has represented Slovenia in UEMS Endocrinology.



MLADEN KRNIĆ

Mladen Krnić, MD, PhD, is Associate Professor at the University of Split School of Medicine and a specialist in internal medicine and endocrinology and diabetology at the University Hospital of Split, Croatia. His main clinical and academic interests include diabetes, obesity and metabolic syndrome, cardiometabolic medicine, glucose monitoring and diabetes technology, and metabolic bone disease. He is actively involved in undergraduate and postgraduate medical education, clinical research, and international clinical trials. His academic work includes scientific publications, national clinical guidelines, international expert recommendations and professional consensus statements, with a particular interest in translating scientific evidence into everyday clinical practice. He serves as President of the Croatian Society for Diabetes and Metabolic Disorders and as a Board Member of the Croatian Endocrine Society. He is a regular invited speaker at national and international scientific and professional meetings.

BARBARA MCGOWAN

Professor Barbara McGowan is a Consultant and Professor of Endocrinology and Diabetes at King's College London. She also leads the Obesity, Diabetes and Endocrinology service at the Cleveland Clinic London. She is the Chief Medical Officer for Reset Health, which hosts the Roczen platform, a NICE approved digital weight management service.

Professor McGowan is an investigator for several clinical trials in gut hormone therapies for obesity and was awarded an NIHR/RCP/CRN prize in recognition of outstanding research within the NHS. She is a member of the Finance Committee for the International Society for Endocrinology and co-chairs EASO's Obesity Task Force. She is the recipient of the 2024 KCL Faculty of Life Sciences and Medicine Research Impact on Society Award and the 2025 Outstanding Clinical Practitioner Award by the Society for Endocrinology.

ELMIR OMEROVIC

Elmir Omerovic, MD, PhD, FESC, FHFA, is a senior consultant cardiologist, Chair Professor of Cardiology at the University of Gothenburg, and, since 2007, head of the Heart Group at the Wallenberg Laboratory for Cardiovascular Research, Sahlgrenska Academy. He is an experienced clinical trialist and principal investigator of several large randomized trials and has served on the steering committees of multicentre national and international trials. He is the author of over 330 scientific publications.

He is a translational researcher in Takotsubo syndrome, heart failure, and myocardial ischemia, combining experimental models with registry-based randomized and observational studies within the SWEDEHEART registry. He is a trained interventional cardiologist who performs diagnostic coronary angiography and percutaneous coronary interventions at Sahlgrenska University Hospital."

EDUARD OŠTARIJAŠ

Eduard Oštarijaš, MD, PhD, MPH, is a physician and researcher in endocrinology and evidence-based medicine. He is assistant professor at the Catholic University of Croatia and is completing his medical residency in endocrinology and diabetology at University Hospital Centre Osijek. He previously worked in research methodology and medical statistics at the University of Pécs and at Semmelweis University in Budapest.

He holds a PhD from the University of Pécs, a master's degree in public health from the University of Zagreb School of Medicine and a medical degree from the University of Rijeka. He currently serves as Secretary and Board Member of the Croatian Endocrine Society. His research interests include endocrinology, metabolism and evidence-based medicine, with a focus on systematic reviews, meta-analyses and evidence synthesis in medical research.

DIPESH C. PATEL

Dr Dipesh C. Patel, BSc, MBBS, PhD, FRCP is a consultant physician in diabetes, endocrinology, and acute medicine at the Royal Free Hospital and Cleveland Clinic London, and Honorary Clinical Associate Professor at University College London. He has held multiple national and international leadership positions, including Chair and Co-Director of the UK Association of British Clinical Diabetologists (2017–2023), Vice Chair of the Diabetes Care Trust (2024–), UK representative on the European Union Diabetes Forum (2022–), and steering committee lead for ABCD international collaborations (2023–). He has contributed to stakeholder work with NICE, the Royal College of Physicians, and the London Strategic Clinical Network.

His main clinical and research interests are in diabetes, obesity, lipidology, and cardiorenal disease, with a focus on the evaluation of novel therapies and models of care. He was awarded his PhD at Imperial College London for work on glycaemic influences on cholesterol transport (ABCA-1), and has since served as principal investigator in several international Phase III trials.

His contributions have been recognised with national and institutional teaching awards and with clinical prizes from the Society for Endocrinology and University of London. He is Associate Editor of the British Journal of Diabetes and reviewer for several international journals.

Dr. Patel has authored or co-authored numerous peer-reviewed publications spanning diabetes, cardiovascular and renal outcomes, obesity management, and COVID-19. His current work combines clinical practice, education and translational research.

IVAN PEĆIN

Associate Professor Ivan Pećin, MD, PhD, is one of Croatia's leading lipidologists and a specialist in internal medicine with a subspecialty in endocrinology and diabetology. He is Head of the Division of Metabolic Diseases at University Hospital Centre Zagreb, Associate Professor of Internal Medicine at the University of Zagreb School of Medicine, and Head of the Croatian Ministry of Health Reference Centre for Rare and Metabolic Diseases.

He serves as Vice President of the Croatian Atherosclerosis Society, the Croatian Society for Rare Diseases and the Croatian Hypertension League, and is a Board Member of the Croatian Endocrine Society.

His clinical and scientific work focuses on dyslipidaemias, familial hypercholesterolaemia, preventive cardiology and rare metabolic diseases. He is actively engaged in international collaborations with the International Atherosclerosis Society (IAS), European Atherosclerosis Society (EAS), European Alliance for Cardiovascular Health (EACH), European Society of Cardiology (ESC), International Lipid Expert Panel (ILEP) and FH Europe Foundation.

Professor Pećin also leads the public-health initiatives "Hunting the Silent Killer" and "Know Your Numbers?", bringing cardiovascular prevention, screening and health education directly into communities. His scientific work has achieved an H-index of 27 and more than 27,000 citations.

RICHARD QUINTON

Dr Richard Quinton MB BChir MA MD FRCP(E) is Consultant Endocrinologist in Newcastle-upon-Tyne since 1999 and honorary Reader in Reproductive Endocrinology at Imperial College London since 2024.

Graduating from Cambridge University in 1988, he trained in Internal Medicine at St Bart's London and Erasmus University Hospital Rotterdam, followed by Endocrinology training at UCL Hospitals, London.

He has been active in research for >30 years, with a particular interest in Hypogonadotropic Hypogonadism and >200 publications. In 2000, he was awarded the Ralph Noble Prize by Cambridge University and, in 2023, the Outstanding Clinical Practitioner award by the Society for Endocrinology (SfE).

He co-led SfE guidelines groups on male (2022) and female (2024) hypogonadism; EU-COST consortium on the Biology of GnRH deficiency (2013–18), and NIHR-funded TestES consortium on cardiovascular safety of testosterone in men (2018–20).

SUHANIYA SAMARASINGHE

Dr Suhaniya Samarasinghe is a Clinician Scientist and Honorary Consultant in Endocrinology and Diabetes at Imperial College London and Imperial College Healthcare NHS Trust. She completed her PhD in Obesity and Female Fertility at Imperial College London, during which she served as Lead Clinical Research Fellow for the BAMBINI randomised controlled trial, comparing bariatric surgery with standard medical care in women living with polycystic ovary syndrome (PCOS) and obesity. The trial's findings, published in The Lancet (2024), have since informed international clinical guidelines for the management of PMOS.

Dr Samarasinghe's research and clinical practice focus on the interplay between metabolic dysfunction and women's reproductive health, including the effects of obesity, insulin resistance and metabolic therapies on fertility outcomes. She currently holds an NIHR Imperial BRC Postdoctoral Fellowship and combines academic research with specialist clinical practice in obesity medicine and female reproductive endocrinology.

UMASUTHAN SRIRANGALINGAM

Dr Umasuthan Srirangalingam is a Consultant Endocrinologist with a practice based at University College London Hospitals (UCLH) and Cleveland Clinic London. He graduated from the University of Edinburgh in 1999 and completed specialist training at St Bartholomew's Hospital.

In 2012 he was awarded a PhD for work on splicing therapeutics and went on to gain an NIHR Clinical Lectureship developing an interest in adrenal disease and the familial paraganglioma syndromes. At UCLH he leads the clinical service for congenital adrenal hyperplasia (CAH) and male hypogonadism/fertility induction. He is currently the PI on a number of early phase clinical trials for new therapeutics targeting the HPA axis, CAH and Cushing syndrome.

MARIO SKUGOR

Professor Mario Skugor, M.D., FACE, is a distinguished medical professional with an extensive career in the field of endocrinology. He completed his medical degree in 1986, followed by a two-year research associate stint at the Institute for Nuclear Medicine in Zagreb. After serving in the Croatian army from 1991 – 1992, he concluded his Nuclear Medicine Residency in 1993, and joined Ohio State University as a researcher in the Bone and Mineral Metabolism Laboratory. His Internal Medicine residency was completed at St. Luke's Medical Center, Cleveland in 2000, with his final year as chief resident.

From 2000 – 2002, he underwent an Endocrinology fellowship at Cleveland Clinic, post which he joined the staff, specializing in diabetes, obesity, thyroid disorders and calcium metabolism disorders. His tenure at Cleveland Clinic involved being:

— the Associate Director of Endocrinology Fellowship Program (2009 – 2011)
— Director of Center for Thyroid Disorders (2008 – 2012)
— Vice-Chairman (2009 – 2013),
— Chairman (2013 – 2015) of Global Patient Services.

Dr. Skugor served on the Board of Directors for the Ohio River Valley Chapter of the American Association of Clinical Endocrinologists and presided over it from 2007 – 2008. Presently, Dr. Skugor holds the title of Clinical Associate Professor of Medicine at Cleveland Clinic Lerner College of Medicine of CWRU, holds the Catherine and Edward Lozick Chair in Endocrinology and Metabolism, and serves as Director for Education
for Endocrine and Metabolic Institute.

TAMARA TURK WENSVEEN

Tamara Turk Wensveen, MD, PhD, is an associate professor of internal medicine and a specialist in internal medicine with a subspecialty in endocrinology and diabetology. She heads the Center for Diabetes, Endocrinology and Cardiometabolism at the Clinic for the Treatment, Rehabilitation and Prevention of Cardiovascular Diseases, Thalassotherapia Opatija. She also works at the Department of Endocrinology, Diabetes and Metabolic Diseases, Clinic for Internal Medicine, University Hospital Centre Rijeka. Since 2005, she has been actively involved in teaching and research at the Department of Internal Medicine, Faculty of Medicine, University of Rijeka. Her early research focused on diabetic nephropathy, including advanced training at University Medical Center Utrecht in the Netherlands from 2007 to 2008. She obtained her PhD in 2010 for research into the role of bone morphogenetic protein signalling pathways in diabetic nephropathy and completed her specialization in internal medicine in 2012. In 2013, she undertook further clinical training in diabetology and endocrinology at UMC Utrecht. Since 2014, her research has expanded into immunometabolism in diabetes, resulting in publications in leading journals, including Nature Immunology, Immunity, and Diabetes Care.

Professor Turk Wensveen has completed postgraduate studies in biomedicine, internal medicine, and endocrinology and diabetology. She has presented her clinical and scientific work at numerous national and international conferences and has received several distinctions, including the Faculty of Medicine, University of Rijeka award for the best young researcher in clinical sciences. She has received two European grants from ERA-EDTA and has collaborated on several European research projects supported by the UKF, EASD, and EFSD. She currently leads the Croatian Science Foundation project, "The Impact of Type 2 Diabetes Mellitus and Its Therapy on Lymphocyte Function."

Her current work has a strong cardiometabolic focus, integrating the management of diabetes, obesity, renal disease, and cardiovascular risk. She was recently appointed President of the Cardiovascular–Renal–Metabolic (CRM) Task Force of the Croatian Cardiac Society, through which she promotes interdisciplinary collaboration and the advancement of integrated cardiometabolic care in Croatia. Professor Turk Wensveen is the author or co-author of eight book chapters and more than 30 scientific articles. She is a member of numerous Croatian and international professional societies.

FELIX WENSVEEN

Felix Wensveen is full professor and group leader at the University of Rijeka, Faculty of Medicine. Prof. Wensveen received his PhD at the University of Amsterdam, studying fundamental CD8 T cell biology. In 2010 he moved to the University of Rijeka where he obtained a postdoctoral position in the group of Prof. Bojan Polić. In 2015, he started his own group at this institute, investigating the role of the immune system in metabolic disease. In recent years, the team of Prof. Wensveen has focused on investigating how the immune system impacts systemic metabolism both in context of viral infection and in metabolic dysfunction. The work of his team has led to breakthrough findings in the field of meta-inflammation, published in world-leading journals. They identified a pivotal role of metabolic stress sensing and inflammation in the development of Type 2 diabetes and metabolic dysfunction-associated steatotic liver disease (Wensveen Nature Immunol. 2015, Marinović Science Immunol. 2023). Moreover, his team showed at a molecular level how viral infection can contribute to the development of T2D and metabolic dysfunction (Šestan Immunity 2018, Šestan Nature Immunol. 2024, Krapić Nature Metabolism 2025).

Finally, his team has made important contributions to the field of meta-inflammation in context of type 2 diabetes (Turk Wensveen Diabetes Care 2020, Kavazović Diabetes 2022, Gašparini Diab Res Clin. Pract. 2024, Troskot Cuculić Frontiers in endocrinol. 2026).

Prof. Wensveen has published 60 papers and has an H-factor of 29. He has received numerous international grants and various national and international awards.

PRZEMYSŁAW WITEK

Professor Przemysław Witek, MD, PhD, is Head of the Department of Internal Medicine, Endocrinology and Diabetes; Faculty of Medicine; Medical university of Warsaw, Poland.

He is a Member of the Executive Board of the Polish Society of Endocrinology; the Author of many original papers in the field of pituitary disorders, especially Cushing's disease, acromegaly and prolactinoma; participant in the clinical trials on pasireotide LAR in acromegaly and Cushing's disease, levoketoconazole in Cushing syndrome and in LINC 4 trial – osilodrostat in Cushing's disease.

The co-author of registration papers in JCEM and Lancet regarding pasireotide LAR, levoketoonazole and osilodrostat in Cushing's syndrome.

DIVYA YOGI-MORREN

Dr. Divya Yogi-Morren, M.D., FACE is Medical Director of the Cleveland Clinic Pituitary Center and Staff Physician in the Department of Endocrinology, Diabetes and Metabolism at Cleveland Clinic, where she also serves as Clinical Assistant Professor of Medicine at the Cleveland Clinic Lerner College of Medicine. A valedictorian graduate of the University of the West Indies and recipient of a national full-merit scholarship, she completed Internal Medicine residency at Cleveland Clinic Florida (2012) and Endocrinology fellowship at Cleveland Clinic, where she was Chief Endocrine Fellow.

Her clinical and research focus is neuroendocrine pituitary disease, including Cushing's disease, acromegaly, prolactinomas, and complex sellar/parasellar disorders. Since 2019 she has led the Pituitary Center's multidisciplinary program with neurosurgery, neuro-oncology, neuro-ophthalmology, and neuroradiology, developing subspecialty pathways for diagnosis, peri-operative care, and long-term outcomes.

Dr. Yogi-Morren has been principal investigator for multicenter clinical trials of novel therapies, including levoketoconazole for Cushing's syndrome, oral octreotide for acromegaly, and antisense inhibition of the growth-hormone receptor, and has published on pituitary imaging, thromboprophylaxis in Cushing's disease, giant prolactinomas, empty sella, and IPSS interpretation.

She is active in education and program leadership: Associate Program Director for the Endocrinology Fellowship (2019–2022), founder/director of the fellowship's Medical Education Track, core faculty and Clinical Competency Committee member, and co-director of the Endocrine and Reproductive Biology course at CCLCM. Her professional development includes a Medical Education Fellowship and leadership training through the Mandel Global Leadership & Learning Institute. An invited advisor to multiple industry scientific boards (Camurus, Chiesi, Crinetics, Corcept, Pfizer, Chiasma), she also serves as a media resource on pituitary and endocrine topics for national outlets. Her career combines clinical leadership of a high-volume pituitary program with translational research and education aimed at improving outcomes in complex hormonal disorders.

ABSTRACTS

ENDO CASE LAB: PITUITARY, THYROID & BONE

OSILODROSTAT FOR CUSHING'S DISEASE IN A PATIENT WITH TYPE 1 DIABETES

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Background: Cortisol excess increases insulin resistance and insulin requirements. In people with type 1 diabetes using an advanced hybrid closed-loop (AHCL) system, automated insulin delivery may partly compensate for this change, potentially delaying recognition of evolving hypercortisolism. Longitudinal pump and continuous glucose monitoring (CGM) data may therefore provide an additional clinical signal of evolving hypercortisolism.

Case presentation: A 35-year-old woman with type 1 diabetes and psoriasis presented with mild cushingoid facial features, behavioral change, anxiety and a new fear of hypoglycemia that was not supported by CGM. In parallel, her total daily insulin dose progressively increased, while glycemic control remained suboptimal despite use of an AHCL system with fast-acting insulin aspart. An initial dexamethasone suppression test showed inadequate cortisol suppression and non-suppressed ACTH, and repeat testing after discontinuation of an estrogen-containing oral contraceptive confirmed ACTH-dependent hypercortisolism. Pituitary magnetic resonance imaging (MRI) showed a 7-mm lesion, and serum cortisol decreased by more than 50% during a high-dose dexamethasone suppression test, supporting a pituitary source. The patient declined transsphenoidal surgery and was later started on osilodrostat. Within four weeks, total daily insulin dose decreased by 36%, TIR increased to 86% and time below range (TBR) remained low. Within two months, urinary free cortisol (UFC) had normalized, alongside improvements in blood pressure and body weight, and facial rounding diminished. Subsequent biochemical variability required cautious dose adjustments and changes to pump settings.

Conclusion: Coexisting ACTH-dependent hypercortisolism and type 1 diabetes is a distinct diagnostic and therapeutic challenge. This case illustrates the bidirectional link between hypercortisolism and glycemic control. Rising insulin requirements and loss of glycemic stability during advanced hybrid closed-loop therapy may provide an early metabolic clue to hypercortisolism when interpreted alongside new physical and neuropsychiatric changes. Low-dose initiation and individualized titration of osilodrostat, combined with close glucose and safety monitoring, enabled rapid reduction of cortisol exposure and insulin requirements while maintaining low hypoglycemic exposure.

A CASE OF ACROMEGALY WITH PLURIHORMONAL SECRETING ADENOMA

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Introduction: Acromegaly is a rare chronic disorder due to growth hormone (GH) hypersecretion and lack of control is related to increased mortality. Approximately 20% of patients have co-secretion of growth hormone and prolactin. Simultaneous production of GH and adrenocorticotropic hormone (ACTH) is extremely rare and only about 20 cases have been described in the literature.

Clinical case: We present a patient with acromegaly, diagnosed in 2013 at 28 years of age, due to headache, hyperhydrosis and typical facial changes. After visualization of pituitary macroadenoma transsphenoidal surgery was performed, followed by short-term treatment with dopamine agonist (cabergoline). The same year the patient underwent telegammatherapy. After the irradiation he was admitted for the first time at our department. Active acromegaly was confirmed and medical treatment with first-generation long-acting somatostatin analogue (Octreotide LAR) was initiated. Besides high GH and IGF1 leveles, high ACTH was also found, with lack of supression of serum cortisol after 1 mg midnight Dexamethasone supression test. He showed no typical symptoms of hypercortisolism. Immunohistochemistry of pituitary adenoma proved the co-expression of GH and ACTH. In February 2014 a transcranial readenomectomy was performed due to worsening of the visual fields. Subsequently, the highly selective GH-receptor antagonist Pegvisomant was added to maximal dose of Octreotide LAR and Cabergoline 2 mg per week. The former had a good effect, but was eventually stopped in one year due to severe local side effects. In 2016 the patient underwent repeat radiotherapy, after which normal ACTH was documented. Biochemical control of acromegaly was achieved in 2018 after initiating treatment with second-generation long-acting somatostatin analogue (Pariseotide LAR). During the regular follow-ups secondary hypogonadism, hypothyroidism and latent hypocorticism were registered.

Conclusion: Young age of occurence of pituitary adenoma and its plurihormonal secretion are probably related to more aggressive disease course. Achievement of remission required multimodal treatment and multidisciplinary approach. The co-secretion of GH and ACTH may explain the better effect of Pasireotide LAR, which, unlike Octreotide LAR, binds to all types of somatostatin receptors (SSR) and has a high affinity for SSR type 5 (mainly expressed by pituitary adenomas, secreting ACTH).

A PITUITARY MASS WITH A TWIST: HYPOPHYSITIS MIMICKING A MACROADENOMA

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Introduction: Hypophysitis is an uncommon inflammatory disorder of the pituitary gland that can present as a sellar or suprasellar mass and closely mimic a pituitary macroadenoma. The clinical and radiological overlap between these entities may pose a significant diagnostic challenge. On magnetic resonance imaging (MRI), hypophysitis may demonstrate pituitary enlargement, homogeneous contrast enhancement, suprasellar extension, and stalk thickening—findings that can be indistinguishable from those of a macroadenoma. Differentiating these conditions is essential, as their management differs substantially. We present a case of hypophysitis radiologically mimicking a pituitary macroadenoma, highlighting the importance of integrating clinical, laboratory and MRI findings in establishing the diagnosis.

Case report: A 29-year-old man was initially hospitalized following generalized tonic–clonic seizures. Neuroradiological evaluation revealed a sellar mass measuring up to 15 mm, appearing hypointense and morphologically suggestive of a pituitary macroadenoma. The lesion displaced the infundibulum to the left and caused mild superior elevation of the optic chiasm. At that time, the patient declined further evaluation for personal reasons. Four years later, he presented to the endocrinology clinic with visual disturbances, frontal headaches, and erectile dysfunction. Follow-up pituitary MRI demonstrated significant progression of the lesion, measuring 2.0 × 1.4 × 1.7 cm, with suprasellar extension and elevation of the optic chiasm. The mass was inseparable from the terminal segments of both internal carotid arteries, both middle cerebral arteries, and the left anterior cerebral artery. Additionally, the normal T1-hyperintense signal of the neurohypophysis was absent. Endocrinological evaluation revealed panhypopituitarism, and hormone replacement therapy was initiated, primarily with hydrocortisone. Neuro-ophthalmological examination showed no visual field deficits. The case was subsequently discussed by a multidisciplinary team. In view of the clinical, endocrine, and radiological findings, hypophysitis was considered the most likely diagnosis. An immunological workup was unremarkable, and the patient did

not meet diagnostic criteria for an underlying systemic autoimmune disease. Hydrocortisone was subsequently switched to prednisone 40 mg daily, followed by a gradual taper. Endocrinological reassessment is planned after 16 weeks of corticosteroid therapy.

Conclusion: Hypophysitis should be considered in the differential diagnosis of an enlarging sellar or suprasellar mass, even when the radiological appearance initially suggests a pituitary macroadenoma. This case highlights the substantial overlap between hypophysitis and macroadenoma on MRI and the potential for delayed diagnosis. The presence of pituitary stalk involvement, loss of the posterior pituitary bright spot, and panhypopituitarism may provide important diagnostic clues when interpreted in the appropriate clinical context. A comprehensive assessment integrating clinical, endocrinological, radiological, and multidisciplinary findings is essential for accurate diagnosis and appropriate management, potentially avoiding unnecessary surgical intervention.

Key words: hypophysitis; hypopituitarism; magnetic resonance imaging; pituitary macroadenoma; sellar mass.

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WHEN HYPOTHYROIDISM TURNS INTO HYPERTHYROIDISM: A CASE OF GRAVES' DISEASE EMERGING IN LONG-STANDING HASHIMOTO'S THYROIDITIS

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Background: Hashimoto's thyroiditis and Graves' disease represent different phenotypes within the spectrum of autoimmune thyroid disease. Although transition from Graves' hyperthyroidism to hypothyroidism is well recognized, the reverse transition from long-standing Hashimoto's hypothyroidism to Graves' hyperthyroidism is uncommon. We present a case of an elderly patient with established autoimmune hypothyroidism who developed endogenous thyrotoxicosis associated with positive TSH-receptor antibodies.

Case presentation: A 71-year-old woman with long-standing autoimmune hypothyroidism treated with levothyroxine, vitiligo and pre-existing atrial fibrillation presented with progressive weakness, palpitations, tremor, frequent bowel movements and a 5-kg unintentional weight loss. Before admission, she was receiving levothyroxine 50 µg three days per week and 25 µg four days per week. Laboratory evaluation on May 20, 2026, demonstrated marked thyrotoxicosis with TSH <0.005 mIU/L, free T4 42.6 pmol/L and free T3 15.3 pmol/L, with anti-TPO >1000 kIU/L and anti-Tg 98 kIU/L.

Levothyroxine was discontinued on hospital admission on May 22. Despite withdrawal of replacement therapy, thyrotoxicosis persisted, and treatment with methimazole and bisoprolol was initiated. On May 27, free T4 remained elevated at 31.7 pmol/L, and on June 1, ten days after levothyroxine withdrawal, TSH remained suppressed at 0.008 mIU/L with free T4 22.6 pmol/L and free T3 5.1 pmol/L. Thyroid ultrasound demonstrated a moderately enlarged, heterogeneous and hypoechoic gland without nodules. Doppler vascularity assessment and thyroid scintigraphy were not performed.

The course was complicated by worsening cardiovascular status in the setting of pre-existing atrial fibrillation, with NT-proBNP 2449 pg/mL and echocardiographic LVEF of 40%, biatrial enlargement and severe tricuspid regurgitation.

During methimazole treatment, thyroid hormone levels declined rapidly, and by June 23 the patient developed biochemical hypothyroidism (TSH 5.555 mIU/L, free T4 2.9 pmol/L), prompting substantial dose reduction. TSH-receptor antibodies

were subsequently positive at 2.6 IU/L, supporting Graves' disease developing on the background of pre-existing Hashimoto's thyroiditis. By late July, thyroid function had approached euthyroidism on low-dose methimazole.

Conclusion: This case illustrates an uncommon autoimmune switch from long-standing Hashimoto's hypothyroidism to Graves' hyperthyroidism. Persistent thyrotoxicosis after discontinuation of a relatively low levothyroxine dose, together with positive TSH-receptor antibodies, supported endogenous hyperthyroidism rather than exogenous hormone excess. Hashimoto's thyroiditis and Graves' disease should be viewed as overlapping phenotypes of autoimmune thyroid disease, in which changes in the balance of stimulatory and blocking TSH-receptor antibodies may alter the functional phenotype over time. In older patients, thyrotoxicosis may additionally exacerbate pre-existing cardiovascular disease and contribute to clinical decompensation.

Keywords: Hashimoto's thyroiditis; Graves' disease; autoimmune switch; TSH-receptor antibodies; thyrotoxicosis.

HORMONAL PROFILES IN FEMALES WITH ALLERGIC DISEASES: AN INTEGRATED ANALYSIS OF THYROID HORMONES, ANDROGENS, FAMILIAL ATOPY, AND AEROALLERGEN SENSITIZATION

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Background: Sex-specific endocrine mechanisms may contribute to the development and expression of allergic diseases. Previous studies have reported higher thyroxine (T4) concentrations among patients with respiratory allergic disease; however, it remains unclear whether this association reflects alterations in thyroid function itself or is secondary to interactions with adrenal, reproductive, or androgenic hormonal pathways. This study aimed to investigate hormonal profiles in females with allergic diseases and to determine whether other endocrine factors could explain elevated T4 concentrations.

Methods: A cross-sectional analysis was conducted among females categorized as healthy controls, respiratory allergies, and urticaria. Serum concentrations of T4, T3, thyroid-stimulating hormone (TSH), cortisol (morning and evening), prolactin, estrogen, progesterone, testosterone, androstenedione, DHEA-SO4, and total IgE were evaluated. Group comparisons, correlation analyses, ANCOVA adjusted for age and body mass index (BMI), and multivariable regression analyses were performed. Familial atopy and aeroallergen sensitization patterns were also assessed.

Results: Females with respiratory allergic disease exhibited significantly higher T4 and IgE concentrations compared with controls. These associations remained significant after adjustment for age and BMI. Most participants had T4 concentrations within the reference ranges; however, allergic participants showed a shift toward higher-normal T4 values. T4 correlated positively with follicular progesterone and prolactin, and negatively with testosterone, DHEA-SO4, follicular FSH, and selected estrogen-related variables. Morning and evening cortisol concentrations were not significantly associated with T4. House dust mite sensitization and positive family history of atopy were more frequent among allergic participants. Importantly, the association between allergic disease and elevated T4 persisted after consideration of adrenal, reproductive, and androgenic hormonal variables.

Conclusion: Females with allergic diseases demonstrate a distinct endocrine profile characterized by higher-normal T4 concentrations and elevated IgE levels. Although T4 is associated with multiple endocrine pathways, including reproductive and androgenic hormones, these relationships do not fully explain the observed differences between allergic participants and controls. The persistence of the T4 signal after adjustment for cortisol, reproductive hormones, and androgens suggests that T4 may represent an independent endocrine feature of allergic disease in females, potentially reflecting broader endocrine–immune interactions rather than overt thyroid dysfunction.

Keywords: allergy, thyroid hormones, T4, females, IgE, cortisol, progesterone, androgens, endocrine – immune interactions, allergic sensitization

THYROID — WHAT LURKS IN THE DARK?

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Introduction: Primary thyroid lymphoma (PTL) is a rare malignancy, accounting for approximately 1–5% of thyroid malignancies. Diffuse large B-cell lymphoma (DLBCL) is the most common histological subtype. Hashimoto's thyroiditis is strongly associated with an increased risk of PTL and may pose a diagnostic challenge due to overlapping clinical and ultrasonographic features.

Case presentation: A 69-year-old woman was referred for severe hypothyroidism and an enlarged thyroid. Laboratory tests showed TSH 100.98 mIU/L, fT4 6.4 pmol/L, and anti-TPO >1300 kIU/L, supporting the diagnosis of Hashimoto's thyroiditis. Levothyroxine replacement therapy was initiated. Despite longstanding mild hoarseness, rapidly progressive dysphonia developed over three weeks. Ultrasound revealed an enlarged, markedly hypoechoic, heterogeneous thyroid gland with large hypoechoic areas and poorly delineated confluent nodules, with suspected tracheal infiltration. Urgent CT demonstrated a 6.5 × 5.8 × 5.6 cm hypovascular thyroid mass circumferentially involving the trachea, with suspected invasion of the trachea, laryngeal structures and major cervical vessels. Cytology suggested non-Hodgkin lymphoma. Biopsy confirmed aggressive DLBCL with a germinal-center B-cell phenotype. Tumor cells were CD20, BCL2, and BCL6 positive; CD10, MUM1, and CD5 negative. C-MYC expression was present in 80% of tumor cells and Ki-67 was approximately 95%. FISH detected no BCL2, BCL6, or C-MYC rearrangements. Six cycles of R-CHOP followed by two additional rituximab administrations resulted in marked tumor regression and complete remission.

Conclusion: Primary thyroid lymphoma should be considered in patients with Hashimoto's thyroiditis who develop new or rapidly progressive compressive symptoms, even without an obviously enlarging gland. Prompt imaging and histopathological diagnosis are essential, as timely immunochemotherapy may achieve complete remission even in locally advanced disease.

ENDO CASE LAB: ADRENAL & ELECTROLYTES

ECTOPIC ACTH SYNDROME: PERSONALIZING TREATMENT IN A COMPLEX CLINICAL SETTING

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Introduction: Ectopic adrenocorticotropic hormone (ACTH) syndrome is a rare cause of Cushing's syndrome caused by excessive ACTH production from nonpituitary tumors. While the majority of ectopic ACTH cases originate from intrathoracic tumors, pancreatic origin is considered rare. Timely diagnosis and treatment are essential to achieve clinical stabilization and to ensure continuation of oncological therapy.

Case report: A 56-year-old male with recently diagnosed metastatic pancreatic adenocarcinoma was hospitalized for first cycle of chemotherapy. Due to severe hypokalemia (2,7 mmol/L) accompanied by hypertension, an endocrinology consult was requested. Further work-up revealed significantly elevated levels of ACTH (74.1 pmol/L) and serum cortisol (1463 nmol/L), with suppressed aldosterone and renin levels. Spironolactone was initiated to manage the hypokalemia, while treatment with the steroidogenesis inhibitor metyrapone was started due to suspected ectopic ACTH syndrome. The patient was subsequently transferred to the Department of Endocrinology for further evaluation and therapy titration. Although the clinical history and markedly elevated ACTH levels strongly pointed toward an ectopic source, a pituitary MRI was performed to rule out a pituitary etiology, revealing an incidental lesion suspected to be a Rathke's cleft cyst. Following the initiation of metyrapone and spironolactone therapy, cortisol levels initially decreased, potassium levels normalized, and the patient was started on systemic antineoplastic therapy. During endocrinologic follow-up visits, the dose of metyrapone was gradually increased due to rising cortisol levels. Additionally, due to the risk of treatment-induced adrenal insufficiency, the patient was adviced to take supplemental hydrocortisone during episodes of acute illness. A follow-up CT scan after three cycles of chemotherapy revealed progression of the underlying malignancy, with bilateral adrenal gland changes consistent with secondary involvement. Due to these findings, the patient was advised to start regular daily hydrocortisone as part of a block-and-replace regimen. Also, due to abdominal pain associated with high-dose metyrapone and the high pill burden, metyrapone was replaced with osilodrostat. With osilodrostat, biochemical control of the Cushing's

syndrome was achieved. Unfortunately, the patient eventually passed away to the progression of his primary disease,

Conclusion: Ectopic ACTH syndrome resulting from a pancreatic adenocarcinoma is a rare entity. The challenge in treating these patients is timely diagnosis and stabilization of clinical condition as both are important for starting or continuation of oncological treatment. For tumors that are not resectable, the therapy of choice are steroidogenesis inhibitors. In this case metyrapone was chosen as the initial medical therapy due to its availability and fast onset of action. Later, due to its short half-life, the high pill burden and gastrointestinal side effects the therapy was switched to osilodrostat due to its efficacy and longer half-life, which allows for more practical dosing and increases quality of life. Due to the risk of adrenal insufficiency with the use of steroidogenesis inhibitors, some prefer the block-and-replace strategy that reduces this risk. This strategy allows for faster titration of the steroidogenesis inhibitor dose and consequently leads to faster normalization of cortisol levels. Additionally, in this case, the patient had suprarenal metastases, which further increase the risk of adrenal insufficiency. In summary, an individualized choice of therapy and proactive management of the risk of adrenal insufficiency are key to achieving biochemical control and improving outcomes in pancreatic ectopic ACTH syndrome.

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SEVERE HYPONATREMIA ASSOCIATED WITH HYPOPHYSITIS

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Introduction: Hypophysitis is a rare inflammatory disorder of the pituitary gland, typically characterised by pituitary enlargement and varying degrees of hypopituitarism. It can be classified as primary or secondary. Primary hypophysitis encompasses predominantly lymphocytic, granulomatous, and xanthomatous forms, whereas secondary hypophysitis occurs in association with systemic inflammatory or infiltrative diseases, immune checkpoint inhibitor therapy, or other sellar lesions. (1)

Case report: A 58-year-old woman initially presented to the emergency department, where she was evaluated by a neurologist for dizziness and gait instability accompanied by nausea and vomiting. Initial laboratory investigations demonstrated severe hyponatremia and hypokalemia. Brain CT was unremarkable. To help differentiate the underlying etiology of severe hyponatremia, urinary sodium concentration was measured and the result was indicative of possible syndrome of inappropriate antidiuretic hormone secretion (SIADH). Given the presence of severe symptomatic hyponatremia, correction with hypertonic saline was initiated in the emergency department.

It is important to note that the patient had depressive syndrome that was treated with escitalopram that can also cause hyponatremia. A psychiatrist was consulted for medication adjustment, escitalopram was discontinued and oxazepam was initiated. Patient was admitted in Intensive Care Unit. With the administration of hypertonic fluids, partial improvement in serum sodium levels was observed, accompanied by mild clinical improvement. Consequently the patient was transferred to Department of Endocrinology. After the transfer, hormonal evaluation revealed secondary adrenal insufficiency, gonadotropin levels were inappropriately low for the patient's age, low levels of IGF-1, and TSH was inappropriately low relative to the peripheral thyroid hormone levels. The patient was started on hydrocortisone. Levothyroxine was added to the treatment regimen several days after initiation of hydrocortisone therapy to avoid precipitating an adrenal crisis. Pituitary MRI revealed signs of possible hypophysitis. An etiological workup for possible hypophysitis was unremarkable, and no underlying cause was identified. Following the initiation of specific hormone replacement therapy, a significant

clinical improvement was observed, along with complete normalization of serum sodium levels. Follow-up assessments continue to demonstrate persistent hypopituitarism, while the patient remains clinically stable on hydrocortisone and levothyroxine replacement therapy.

Conclusion: Hyponatremia in hypophysitis is frequently a clinical consequence of unrecognized secondary adrenal insufficiency, often manifesting as an early, life-threatening indicator of pituitary dysfunction. (2) Clinicians should maintain a high index of suspicion for hypocorticism in patients with recurrent or refractory hyponatremia accompanied by nonspecific constitutional symptoms. Early recognition and hydrocortisone administration can prevent fatal complications. Levothyroxine replacement should be introduced only after glucocorticoid coverage has been established to mitigate the risk of precipitating an acute adrenal crisis.

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BILATERAL ADRENAL INFARCTION IN PREGNANCY: A RARE AND LIFE-THREATENING CLINICAL CHALLENGE

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Introduction: Adrenal infarction is a rare but potentially life-threatening cause of acute abdominal pain in pregnancy, and its recognition may be challenging because of its nonspecific clinical presentation. The unique vascular anatomy of the adrenal gland, with a rich arterial supply and limited venous drainage, makes it particularly susceptible to infarction in hypercoagulable states such as antiphospholipid syndrome and pregnancy. Pregnancy is a physiological prothrombotic state, while increased adrenal stimulation and perfusion and reduced venous drainage due to compression by the gravid uterus may further increase the risk, particularly during the third trimester. Bilateral adrenal infarction is especially rare and clinically significant because adrenal insufficiency may develop.

Case Presentation: A 20-year-old primigravida with no history of thromboembolic events or previous hormonal therapy was hospitalized at 29 weeks and 2 days of gestation because of sudden-onset, severe, continuous pain in the left upper quadrant accompanied by vomiting. Her medical history was notable for obesity (body weight 118 kg, height 175 cm, BMI 40.5 kg/m²) and smoking prior to pregnancy. Her family history was notable for the death of her paternal grandfather from thrombotic complications at the age of 57 years. Until that time, the pregnancy had been monitored because of suspected oligohydramnios, with a normal oral glucose tolerance test (OGTT).

At 15 weeks of gestation, the patient had already been hospitalized because of abdominal colicky pain and elevated inflammatory markers (CRP 113 mg/L). Because of suspected appendicitis, abdominal magnetic resonance imaging (MRI) was performed. Compared with a CT scan obtained in February 2025, MRI demonstrated a newly developed right adrenal lesion with a cystic component measuring up to 4.2 cm, accompanied by a small amount of surrounding free fluid. An acute surgical condition was excluded, and the patient was treated with antibiotics and symptomatic therapy.

Upon readmission for severe abdominal pain at 29+2 weeks of gestation, the patient was afebrile and hemodynamically stable (blood pressure 128/80 mmHg), with leukocytosis of 18.7 × 109/L, neutrophilia of 15.24 × 109/L, and a CRP level of

109 mg/L, which increased to 165 mg/L the following day. Blood and urine cultures were sterile, and the D-dimer level was 2.14 mg/L. Abdominal MRI performed on 10 July 2026 demonstrated diffuse enlargement of the left adrenal gland, with morphological features consistent with acute left adrenal infarction, while the right adrenal gland was normal in size and morphology at that time. Retrospective reevaluation of the MRI obtained at 15 weeks of gestation, in consultation between the endocrinologist and radiologist, raised the suspicion that the previously described cystic lesion of the right adrenal gland represented a consequence of an earlier infarction that had completely resolved radiologically by the time of the current examination. Based on the clinical and radiological findings, the diagnosis was acute left adrenal infarction with a probable previous right adrenal infarction. Given the probable sequential bilateral adrenal involvement, adrenocortical function was assessed. During the acute phase, serum cortisol was 270 nmol/L, with a sodium level of 136 mmol/L and potassium level of 3.9 mmol/L. Although the patient showed no electrolyte abnormalities or hemodynamic signs of overt adrenal crisis, the total cortisol concentration was considered inappropriately low in the context of acute physiological stress and the expected physiological increase in cortisol during pregnancy. Because adrenal insufficiency was suspected, glucocorticoid replacement therapy with hydrocortisone was initiated, initially at 100 mg intravenously, followed by 50 mg twice daily for two days. Treatment was subsequently continued with oral hydrocortisone at a dose of 20 mg in the morning and 10 mg in the afternoon. During follow-up, the patient remained normotensive, with normal glucose and electrolyte levels. At endocrinological follow-up, glucocorticoid replacement was considered adequate, with a plan to reevaluate the hypothalamic–pituitary–adrenal axis after completion of the pregnancy.

Given the presumed thrombotic etiology, therapeutic-dose low-molecular-weight heparin was initiated twice daily, and an extensive hypercoagulability evaluation was performed. Protein C activity (1.26), antithrombin activity (1.04), and free protein S antigen (0.82) showed no deficiency. Lupus anticoagulant was negative, as were anticardiolipin antibodies (aCL-IgM 0.8 MPL/mL; aCL-IgG 0.0 GPL/mL) and anti-β2-glycoprotein I antibodies (IgM 0.2 AU/mL; IgG 0.0 AU/mL), providing no laboratory evidence of antiphospholipid syndrome. Fibrinogen (8.2 g/L) and von Willebrand factor activity (3.15) were elevated and interpreted by the hematologist in the context of pregnancy, obesity, and glucocorticoid therapy. Final hematological evaluation revealed no inherited thrombophilia.

Therapeutic-dose low-molecular-weight heparin and hydrocortisone were continued with multidisciplinary endocrinological, hematological, and obstetric follow-up.

Conclusion: Adrenal infarction should be considered in the differential diagnosis of sudden-onset severe abdominal or lumbar pain during pregnancy, particularly when initial evaluation identifies no more common obstetric, surgical, or urological cause. This case is noteworthy for probable sequential bilateral adrenal involvement, with possible right adrenal infarction during the second trimester followed by radiologically confirmed acute left adrenal infarction during the third trimester, accompanied by suspected consequent adrenal insufficiency.

Despite extensive hematological and molecular evaluation, no high-risk inherited thrombophilia was identified, and antiphospholipid antibody testing was negative. In the absence of another clearly established thrombophilic disorder, the physiological hypercoagulability of pregnancy, potentiated by severe obesity and other individual risk factors, may have played an important role in the development of adrenal infarction.

Early recognition of this rare complication and a multidisciplinary approach are essential for diagnosis, adrenal function assessment, and timely initiation of anticoagulant therapy and, when indicated, glucocorticoid replacement therapy.

INCIDENCE AND CLINICAL CHARACTERISTICS OF MILD AUTONOMOUS CORTISOL SECRETION (MACS)

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Introduction: Mild autonomous cortisol secretion (MACS) is a frequent functional abnormality in patients with adrenal incidentalomas and may be associated with cardiometabolic comorbidities despite the absence of overt Cushing syndrome. This study aimed to characterize patients with adrenal incidentalomas, with particular focus on the prevalence and clinical, biochemical, and radiological features associated with MACS and adrenal hormone secretion.

Methods: We retrospectively analyzed 150 patients with adrenal lesions. Demographic characteristics, comorbidities, biochemical and hormonal parameters, radiological findings, and treatment were evaluated. MACS was assessed using the 1-mg dexamethasone suppression test. Associations between hormonal secretion and clinical and radiological characteristics were analyzed, and multivariable logistic regression was performed to identify independent predictors of secretory tumors and operative treatment.

Results: The median age was 66 years (IQR 60–73), and 66% of patients were women. Hypertension (72%), dyslipidemia (59.3%), metabolic syndrome (37.3%), and diabetes (24.7%) were common comorbidities. MACS was diagnosed in 21 patients (14%), with an additional 3 patients (2%) classified as suspected MACS. Overall, 34 patients (22.7%) had hormonally secretory adrenal tumors. Most adrenal lesions were ≤4 cm (91%), had unenhanced attenuation ≤10 HU (81%), and were unilateral (69.3%); adenoma was the predominant radiological diagnosis (80%). No significant association was found between HU category and MACS or overall secretory status. Compared with patients with non-secretory tumors, those with secretory tumors had a higher prevalence of hypertension (85% vs. 68%, P=0.04), diabetes (44% vs. 19%, P=0.003), and higher HbA1c levels (median 6.45% vs. 5.8%, P=0.01). Secretory tumors were more frequently >4 cm (24% vs. 5%, P=0.002) and bilateral (50% vs. 25%, P=0.005). Multivariable analysis identified age, tumor size >4 cm (OR 11.9), and bilateral lesions (OR 6.1) as independent predictors of secretion.

Conclusion: MACS represented the most prominent functional phenotype among adrenal incidentalomas in this cohort. Its presence within a population characterized by a high burden of cardiometabolic comorbidities underlines the clinical importance of systematic cortisol assessment. Although the metabolic associations observed in this study concerned the overall secretory group, the findings support careful endocrine evaluation of adrenal incidentalomas, particularly in older patients and those with larger or bilateral lesions.

FROM PHEOCHROMOCYTOMA TO MACROPROLACTINOMA

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Introduction: Pheochromocytomas are rare catecholamine-producing neuroendocrine tumors, with an estimated annual incidence of 2–8 cases per million individuals. Pituitary adenomas are considerably more common, but their coexistence with pheochromocytoma is rare and may suggest an underlying hereditary predisposition. Such cases are included within the 3P association, comprising pituitary adenomas with pheochromocytoma and/or paraganglioma, and have been reported particularly in patients with germline SDHx variants. We present a patient with histologically confirmed pheochromocytoma and a large prolactin-secreting pituitary macroadenoma undergoing genetic evaluation.

Case presentation: A 51-year-old man was evaluated for secondary hypertension following central retinal vein occlusion. Biochemical testing revealed more than fourfold elevated metanephrine and normetanephrine concentrations. Abdominal computed tomography demonstrated a 3.5 × 3.1 cm solid left adrenal lesion with an unenhanced attenuation of 48 HU, consistent with pheochromocytoma. Further endocrine evaluation revealed normal parathyroid hormone and calcitonin concentrations and markedly elevated prolactin (>42,000 mIU/L), accompanied by hypogonadotropic hypogonadism. Pituitary MRI demonstrated a 33 × 23 × 19 mm macroadenoma with small intratumoral hemorrhagic areas, cavernous sinus extension and elevation of the optic chiasm. Ophthalmological examination demonstrated bitemporal hemianopia. ^{18}F-DOPA PET/CT was performed to assess disease extent and showed increased tracer uptake confined to the left adrenal lesion, without additional PPGL lesions.

Following preoperative α-adrenergic blockade with phenoxybenzamine, laparoscopic left adrenalectomy was performed in May 2026. Histopathological examination confirmed pheochromocytoma. Postoperatively, plasma metanephrine (0.05 nmol/L) and normetanephrine (0.16 nmol/L) concentrations normalized. Cabergoline was gradually increased to 3 mg/week (0.5 mg six days per week), resulting in a decrease in prolactin from >42,000 to 3,514 mIU/L. Despite the marked biochemical response, follow-up pituitary MRI showed only mild tumor regression, to approximately 30 × 21 × 15 mm, with persistent cavernous sinus involvement and optic chiasm displacement. Due to the limited radiological response and persistent mass effect, neurosurgical treatment is planned. Genetic testing for an underlying hereditary predisposition is ongoing.

Conclusion: The coexistence of pheochromocytoma and pituitary macroadenoma is rare and may represent either a sporadic occurrence or an underlying hereditary predisposition. Recognition of this uncommon tumor combination should prompt genetic evaluation, particularly in view of the reported association with SDHx-related disease. Identification of a pathogenic variant may have important implications for long-term surveillance of the patient and screening of at-risk relatives.

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ENDO CASE LAB: REPRODUCTION & METABOLISM

EARLY MEDICAL COMPLICATIONS DURING STROKE RECOVERY IN PATIENTS WITH TYPE 2 DIABETES TREATED WITH GLP-1 RECEPTOR AGONISTS VERSUS SGLT-2 INHIBITORS

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Background: Stroke survivors commonly experience medical complications during early recovery that interrupt rehabilitation participation and delay functional recovery. Patients with type 2 diabetes mellitus (T2DM) are at increased risk for cardiometabolic and infectious complications following stroke. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have demonstrated cardiovascular, renal, and anti-inflammatory effects that may influence post-stroke medical stability. This study compared early medical complications during stroke recovery in patients with T2DM treated with GLP-1 RAs versus sodium-glucose cotransporter-2 inhibitors (SGLT2i).

Design: Retrospective cohort study.
Setting: TriNetX; a multi-institutional electronic health record network
Participants: Adults with T2DM and ischemic stroke treated with either a GLP-1 RA or an SGLT2i. After propensity score matching for demographics, comorbidities, and concurrent medications, 34,272 patients were included in each cohort.
Interventions: N/A
Main Outcome Measures: Early post-stroke medical complications measured at 30 and 90 days, including heart failure, acute kidney injury (AKI), pneumonia, and sepsis.

Results: After propensity score matching, GLP-1 RA therapy was associated with lower risk of several early post-stroke medical complications compared with SGLT2 inhibitor therapy. At 90 days, heart failure occurred in 2.7% of GLP-1 RA users versus 5.4% of SGLT2i users (risk ratio 0.51; hazard ratio 0.50; $p < 0.001$). Acute kidney injury occurred in 3.1% versus 4.1% ($p < 0.001$). Pneumonia occurred in 1.8% versus 2.2% ($p < 0.05$), and sepsis in 1.5% versus 1.8% ($p < 0.05$). Similar, statistically significant differences were seen at 30 days.

Conclusions: In patients with T2DM following ischemic stroke, GLP-1 receptor agonist therapy was associated with lower risk of early post-stroke medical complications compared with SGLT2 inhibitors, particularly heart failure and acute kidney injury, with additional reductions in infectious complications. These findings suggest medication class may influence medical stability during stroke recovery, with potential implications for rehabilitation participation and post-acute care management.

LABOR AS A TRIGGER OF CHROMOGRANIN A PROCESSING: IMPLICATIONS FOR METABOLIC AND NEUROENDOCRINE ADAPTATION IN GESTATIONAL DIABETES

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Introduction: During pregnancy, activation of the neuroendocrine and sympathoadrenal systems may increase chromogranin A (CgA) release from neuroendocrine and placental tissue. Proteolytic processing of CgA produces bioactive peptides, including pancreastatin, involved in glucose metabolism and insulin secretion, and may be enhanced in gestational diabetes mellitus (GDM). Labor, as a strong physiological stressor, may further affect CgA processing and the balance between its precursor and derivatives.

Materials and Methods: This prospective study included pregnant women at the Department of Obstetrics, Sestre milosrdnice University Hospital Centre. Only multiparous women with singleton pregnancies and planned vaginal delivery without epidural analgesia were included. Participants were divided into a GDM group (n=15) and healthy controls (n=14), with all GDM cases diet-controlled. Primiparity, labor at admission, chronic inflammatory or autoimmune, drug abuse, and antidiabetic therapy were exclusion criteria. Blood samples were collected before labor, during fetal expulsion, and two hours postpartum for measurement of serum CgA and pancreastatin. Statistical analysis included appropriate parametric/non-parametric tests, with repeated measures ANOVA applied for normally distributed data, and Pearson or Spearman correlations ($p<0.05$).

Results: Maternal and neonatal characteristics did not differ between groups, confirming comparability. ANOVA revealed distinct and opposing patterns of CgA and pancreastatin during labor, suggesting differential regulation and processing under physiological stress. Overall, findings indicate a complex dynamic of CgA

and its derivatives in pregnancy and labor, particularly in GDM, reflecting secretion, proteolysis, and peptide generation.

Conclusion: Labor may represent a state of enhanced CgA proteolytic processing, shifting the balance between precursor and active peptides.

METFORMIN-ASSOCIATED LACTIC ACIDOSIS IN CRITICALLY ILL PATIENTS: A SINGLE-CENTRE EXPERIENCE

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Background: Metformin is one of the most widely prescribed medications for type 2 diabetes mellitus. Its use is restricted in patients with impaired kidney function, and discontinuation is recommended when the eGFR falls below 30 mL/min/1.73 m². Metformin-associated lactic acidosis (MALA) is a rare but potentially life-threatening complication, commonly defined by an arterial blood lactate concentration >5 mmol/L and a pH <7.35 in the setting of recent metformin exposure. Measurement of the blood metformin concentration is not required for diagnosis. The pathophysiology of MALA is complex and remains incompletely understood. This study aimed to describe the characteristics, treatment modalities, and outcomes of patients with MALA admitted to a single medical intensive care unit (MICU).

Methods: We conducted a retrospective, single-centre observational study of patients with MALA treated in a MICU between 2017 and 2025. We assessed demographic, clinical, and laboratory characteristics; the need for vasoactive support; the use and modality of renal replacement therapy (RRT); and in-hospital mortality.

Results: Among 4,307 MICU admissions, 58 patients (1.35%) were diagnosed with MALA. The median age was 74 years, 65.5% of patients had de novo acute kidney injury while 34.5% had worsening of preexisting chronic kidney disease. 70.7% patients presented with shock. The median lactate concentration was 13.17 mmol/L, the median pH was 6.97, and the median serum creatinine concentration was 672.5 µmol/L. RRT was used in 94.8% of patients, most commonly as continuous RRT (69.0%). In-hospital mortality was 27.6%. Non-survivors had higher APACHE II scores than survivors (median, 34.5 vs 27.5; P = 0.006), and shock was more frequent among non-survivors (93.8% vs 61.9%; P = 0.023). The number of vasopressors administered was associated with mortality (P = 0.009). The APACHE II score showed significant discrimination for in-hospital mortality (AUC, 0.734; 95% CI, 0.601–0.868; P = 0.006), whereas admission lactate concentration, pH, and daily metformin dose did not.

Conclusions: In critically ill patients with MALA, overall acute illness severity and haemodynamic instability were more strongly associated with mortality than isolated markers of metabolic acidosis or prescribed metformin dose.

NORMOSPERMIA DESPITE LONG-TERM TESTOSTERONE REPLACEMENT THERAPY: AN UNEXPECTED CLINICAL FINDING

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Background: Male hypogonadism is characterized by inadequate testosterone production and/or impaired spermatogenesis. Testosterone replacement therapy (TRT) suppresses the hypothalamic-pituitary-gonadal axis through negative feedback, reducing LH and FSH secretion and consequently spermatogenesis. Therefore, exogenous testosterone is generally contraindicated in men seeking to preserve fertility, for whom gonadotropin therapy is preferred.

Case presentation: We present a man born in 1995 who had been treated for hypogonadotropic hypogonadism with long-term testosterone undecanoate (Nebido, 1 g every 3 months) since 2011. During follow-up, hormonal evaluation unexpectedly demonstrated measurable gonadotropins despite ongoing TRT: FSH 4.2 IU/L, LH 2.7 IU/L, prolactin 140 mIU/L, total testosterone 14.4 nmol/L, SHBG 21 nmol/L, and free testosterone 0.37 nmol/L. Thyroid function was normal, with no evidence of secondary erythrocytosis.

Because of the unexpectedly preserved gonadotropin secretion, additional evaluation of the hypothalamic-pituitary-gonadal axis was performed, including assessment of FSH, LH, testosterone, free testosterone, estradiol, prolactin, and other pituitary hormones. Pituitary MRI in January 2026 showed normal pituitary size and morphology, without evidence of adenoma or other focal lesions. At follow-up in August 2026, gonadotropin levels remained measurable and semen analysis demonstrated normospermia despite continued exogenous testosterone therapy.

Discussion: Long-term testosterone undecanoate therapy is expected to cause substantial suppression of gonadotropin secretion. However, interpretation of LH and FSH during TRT depends on the interval since the last injection, as testosterone concentrations and hypothalamic-pituitary suppression may fluctuate. Residual LH secretion may reflect incomplete suppression of the HPG axis and potentially preserve a degree of Leydig cell activity and intratesticular testosterone production.

Conclusion: Although exogenous testosterone generally suppresses gonadotropin secretion and spermatogenesis, suppression of the HPG axis is not invariably complete. This case demonstrates an unusual phenotype of persistent gonadotropin secretion and preserved spermatogenesis despite long-term testosterone undecanoate therapy, highlighting individual variability in the response to TRT.

SECONDARY AMENORRHEA IN A 20-YEAR-OLD FEMALE WITH ACTIVE SYSTEMIC LUPUS ERYTHEMATOSUS: A THERAPEUTICAL CHALLENGE

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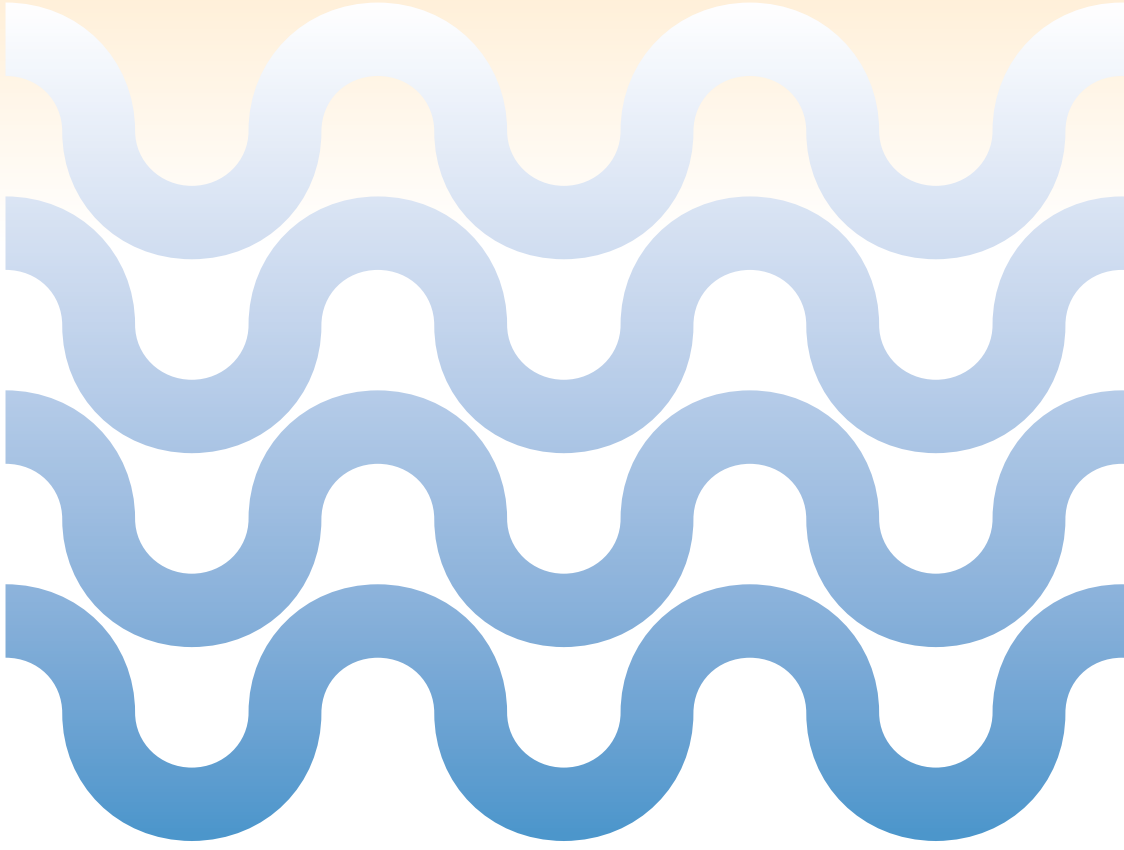
Case presentation: We present the case of a 20-year-old female patient who presented with a severe, acute onset of Systemic Lupus Erythematosus presenting with arthritis, malar rash, profound fatigue, and significant fluid retention. Laboratory tests revealed highly elevated autoantibodies with ANA > 1:640 and anti-dsDNA at 2,344 IU/mL. Severe complement consumption was noted with C3 at 0.376 g/L and C4 at 0.022 g/L. Anti-SSA, anti-Ro52, anti-Sm, and anti-RNP antibodies were all positive. The patient developed nephrotic syndrome with massive proteinuria, gross hematuria, and severe hypoalbuminemia. A renal biopsy confirmed diffuse proliferative lupus nephritis, class IV with high activity and no chronicity. The patient also experienced hypertensive crises complicated by secondary encephalopathy and generalized tonic-clonic seizures. Brain MRI was unremarkable, but EEG showed diffuse dysrhythmia. Concurrently, she developed a severe adjustment disorder with mixed anxiety, depression, and transient suicidal ideation. To suppress life-threatening organ involvement, an aggressive induction regimen was initiated:
1. High-Dose Glucocorticoids: Intravenous pulse methylprednisolone followed by high-dose oral maintenance.
2. Cyclophosphamide: The patient received two pulse doses of Endoxan.
3. Maintenance & Biological Therapy: Following immediate stabilization, the regimen was switched to the biological agent Belimumab combined with mycophenolate mofetil, CellCept. This combination was deliberately chosen by the rheumatology team due to her young age to avoid further gonadotoxic cyclophosphamide exposure.

However, this patient developed secondary amenorrhea immediately following the onset of severe disease and initial cyclophosphamide therapy. Her last regular menstrual period was recorded on the exact date of her first hospital admission. Over the subsequent months of high-dose steroid therapy, she developed a prominent cushingoid phenotype and lost 12 kg. An endocrine and gynecological consultation evaluated the persistent amenorrhea: Estradiol: <18.4 pmol/L, FSH and LH: 2.94 IU/L and 0.689 IU/L respectively and AMH 40 pmol/L. Gynecological examination was normal. The clinical consensus confirmed a diagnosis of central

amenorrhea. The cessation of menses is attributed to severe systemic inflammatory stress, high-dose corticosteroid feedback, and the initial gonadotoxic insult of cyclophosphamide. Because her ovarian follicular reserve remains biologically intact, hormone replacement therapy was deemed unnecessary and also harmful.

ENDO CASE LAB: CALCIUM METABOLISM & NET



WHEN FRAGILE BONES LEAD TO THE PITUITARY: A TALE OF TWO OSTEOPOROSIS CASES

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Introduction: Osteoporosis in men is often associated with an underlying secondary cause, with endocrine disorders representing an important and potentially treatable group of conditions. Because hormonal disturbances may remain unrecognized until their systemic consequences become apparent, evaluation of secondary osteoporosis may provide an opportunity to identify an otherwise unsuspected endocrine disease. We present two male patients in whom investigation of osteoporosis ultimately led to the diagnosis of pituitary disorders.

Case presentation: The first patient was initially referred for evaluation of a left adrenal incidentaloma. As part of his endocrine assessment, dual-energy X-ray absorptiometry revealed osteoporosis of the lumbar spine, prompting further investigation for secondary causes. This unexpectedly demonstrated an insulin-like growth factor 1 (IGF-1) concentration exceeding 1.3 times the upper limit of normal (ULN), shifting the diagnostic focus toward the somatotropic axis. Further testing showed elevated growth hormone (GH), while a 75-g oral glucose tolerance test (OGTT) demonstrated inadequate GH suppression, establishing the biochemical diagnosis of acromegaly. Other evaluated pituitary axes showed no significant abnormalities. Despite the biochemical findings, the patient had not previously noticed typical acral changes, including an increase in shoe size or evident soft-tissue enlargement. Pituitary magnetic resonance imaging (MRI), performed under sedation because of severe claustrophobia, subsequently revealed a 5 × 5 mm microadenoma in the right adenohypophysis, providing a structural correlate for the hormonal abnormality.

The second patient had a longer history of skeletal disease. Osteoporosis had previously been diagnosed and treated with bisphosphonates since 2022; nevertheless, osteoporotic bone mineral density persisted at individual lumbar vertebrae and the right femoral neck. Reassessment for an ongoing secondary cause revealed marked hyperprolactinemia, with repeated prolactin concentrations ranging from 1,870 to 1,924 mIU/L. The elevation persisted after precipitation, with monomeric prolactin concentrations of 1,412–1,516 mIU/L, confirming true hyperprolactinemia. Reduced testosterone with inappropriately low luteinizing hormone and follicle-stimulating hormone levels was consistent with hypogonadotropic hypogonadism, providing a plausible contributor to impaired bone health. Pituitary MRI demonstrated an 8-mm lesion adjacent to the left adenohypophysis, clinically considered consistent with a microprolactinoma.

The diagnostic assessment was further complicated by repeatedly elevated IGF-1 concentrations. Two measurements exceeded 1.3 × ULN, reaching 45.2 nmol/L (1.97 × ULN) and 33.9 nmol/L (1.48 × ULN). On targeted questioning, the patient reported that his wedding ring no longer fitted and that his shoe size had increased by one to two sizes, suggesting acral enlargement. A subsequent IGF-1 measurement decreased to 28.5 nmol/L (1.24 × ULN). During a 75-g OGTT, GH reached a nadir of 0.35 μg/L. Because the patient had a BMI >25 kg/m², this represented inadequate suppression according to the proposed BMI-specific cutoff of <0.2 μg/L. Thus, two IGF-1 measurements above 1.3 × ULN, compatible acral changes and inadequate GH suppression strongly supported concomitant mild acromegaly. However, because of variability in serial IGF-1 concentrations, repeated assessment using the same validated assay and continued evaluation of the GH/IGF-1 axis were planned.

Conclusion: These two cases highlight the importance of considering secondary endocrine causes in men with osteoporosis. In the first patient, evaluation of low bone mineral density led to the diagnosis of previously unrecognized acromegaly associated with a pituitary microadenoma. In the second, persistent osteoporosis uncovered true hyperprolactinemia, hypogonadotropic hypogonadism and an 8-mm pituitary lesion consistent with a microprolactinoma. Repeated IGF-1 elevation above 1.3 × ULN, suggestive acral changes and inadequate BMI-adjusted GH suppression additionally provided strong evidence of concomitant mild acromegaly, although serial IGF-1 variability warranted continued biochemical reassessment. Together, these cases demonstrate that osteoporosis may be the presenting manifestation of an underlying pituitary disorder and that careful endocrine evaluation can reveal clinically important, potentially overlapping pituitary hormone abnormalities.

PRIMARY HYPERPARATHYROIDISM PRESENTING WITH A FULL SPECTRUM OF COMPLICATIONS AND HYPERTROPHIC CARDIOMYOPATHY: A CASE REPORT

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Introduction: Primary hyperparathyroidism (PHPT) is an endocrine disorder characterized by excessive secretion of parathyroid hormone and hypercalcemia that typically presents with renal and skeletal complications. Cardiovascular involvement, particularly hypertrophic cardiomyopathy (HCM), is a rare but critical manifestation that may be driven by hypercalcemia. We hypothesize that the HCM in this patient is secondary to chronic hypercalcemia. In about 80% cases of PHPT the cause is an adenoma of one parathyroid gland which can be treated surgically.

Case report: We presented a 49- year old male patient who first came to our attention with laboratory findings suggestive of PHPT with high serum calcium 3.69 mmol/L and PTH 42.5 pmol/L, phosphorus 0.62 mmol/L, creatinine 132 umol/L, eGFR CKD 54 mL/min/1,73m2, Ca (dU) 5.2 mmol/dU. Asymptomatic, complaining only about chronic stress from work and sometimes back pain. He is a smoker with BMI 29 kg/m2, BP 140/80 mmHg. Three years earlier he has been diagnosed with arterial hypertension, hyperlipidemia, hypertrophic cardiomyopathy (HCM), monovascular coronary artery disease of the LAD without stenosis, dilated aortic bulb and portion of the ascending aorta, mild aortic, mitral and tricuspid regurgitation. On ECG: LVH with strain pattern. ECHO: EFLK 55%, MAPSE 11, GLS -16%. The results of genetic testing for aritmogenic HC are being processed. Our evaluation included ultrasound of the neck that showed a mass in the lower part of the right thyroid lobe 42x33x23 mm, on DXA scan osteoporosis of the lumbar spine, on abdominal ultrasound bilateral multiple nephrolithiasis. SPECT/CT Parathyroid scan confirmed a hyperfunctioning parathyroid tissue - 38mm x 38mm x 28 mm. After a succesful parathyroidectomy an immediate drop of PTH has been noted - from 62.1 pmol/L before surgery to 7.6 pmol/L after surgery.

Conclusion: PHPT can be treated successfully if diagnosed before the development of chronic complications. In patients with renal stones, osteoporosis and cardiovascular disease before the age of 50, we should consider investigating for an underlying cause. This case also underlines the importance of cardiovascular screening in patients with severe PHPT.

In this patient we anticipate a potential regression of the myocardial hypertrophy following the biochemical resolution of hypercalcemia after parathyroidectomy.

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LITHIUM-INDUCED HYPERPARATHYROIDISM

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Introduction with Objective: Parathyroid hormone (PTH) is a key regulator of calcium and phosphate homeostasis. Under physiological conditions, the secretion of parathyroid hormone is regulated by serum calcium levels. In primary hyperparathyroidism, there is an inappropriate secretion of PTH, leading to elevated serum calcium levels. The condition is most often asymptomatic, although it can manifest with subtle or nonspecific symptoms such as fatigue, bone pain, or nephrolithiasis. The aim of this paper is to present a case of primary hyperparathyroidism in a patient treated with lithium for depression.

Case Presentation: A 58-year-old female patient was hospitalized for further evaluation due to repeatedly elevated total calcium levels of about 2.70 mmol/L, ionized calcium about 1.50 mmol/L, and borderline elevated PTH (7.45 pmol/L), measured in outpatient settings. Her medical history included paroxysmal atrial fibrillation, lumbosacral syndrome, depression, and recurrent urinary infections. Additionally, a prior bone densitometry result indicated osteoporosis of the spine. Her medications included: lithium carbonate 300 mg 1+1/2+1 tablet, fluvoxamine 100 mg 1 tablet in the evening, flurazepam 1 tablet as needed, bisoprolol 2.5 mg 1 tablet in the morning, cholecalciferol 25,000 IU every 15 days (2 ampoules), and folate 5 mg 1 tablet in the morning. Physical examination was unremarkable. Laboratory tests showed elevated total (2.76 mmol/L) and ionized (1.41 mmol/L) calcium, as well as PTH (9.6 pmol/L), with vitamin D at 81.3 nmol/L. A 24-hour urine sample was collected and analysed for calcium, phosphate, total proteins, and albumin, all of which were normal. Sestamibi SPECT scan of the neck and chest revealed intense tracer accumulation at the lower pole of the left thyroid lobe, as well as accumulation in both thyroid lobes. Thyroid ultrasound showed an enlarged thyroid with diffusely altered echo structure, and a hypoechoic zone (6 x 4.5 mm) at the lower pole of the left lobe, which was punctured for PTH and cytological analysis. The cytological report described peripheral blood and homogeneous material, without evidence of parathyroid enlargment. The bone densitometry results indicated osteopenia. A chest and lumbosacral spine X-ray showed scoliosis with degenerative changes, without signs of compressive fractures. The patient declined switching from lithium to another antidepressant and was discharged with recommendations to continue her current therapy, with a corrected dose of cholecalciferol 25,000 IU 1 ampoule every 15 days and the initiation of cinacalcet 30 mg 2x1 tablets daily to reduce calcium levels. The patient was further educated on the proper use of cinacalcet (it must be taken with food to increase bioavailability). Subsequent follow-up visits showed normalization of calcium levels allowing dose reduction to 30 mg once daily. At the last follow-up, total calcium was 2.35 mmol/L, and PTH was 8.5 pmol/L.

Discussion: The aetiology of primary hyperparathyroidism is complex, with many conditions, medications, and gene mutations associated with the development of this disease. In this patient, chronic lithium therapy was the most likely cause of hyperparathyroidism, as lithium is known to increase both PTH and calcium levels. The underlying pathological substrate is most often parathyroid gland hyperplasia, rather than a solitary adenoma.

Conclusion: This case illustrates the diagnostic complexity of determining the aetiology of primary hyperparathyroidism and demonstrates the effectiveness of cinacalcet when surgical treatment is not feasible or when hyperparathyroidism is medication-induced. Untreated hyperparathyroidism can lead to osteoporosis, nephrolithiasis, and cardiovascular disease. Pharmacological approach in this patient, which included treatment with cinacalcet, effectively stabilised calcium and PTH levels, preventing further complications. This case also serves as a reminder of the complex interaction between medications and the endocrine system, particularly in patients using psychotropic drugs.

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RESTORING PHYSIOLOGICAL CALCIUM BALANCE: PATIENT JOURNEYS WITH PALOPEGTERIPARATIDE

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Introduction: Hypoparathyroidism after thyroidectomy often requires high doses of calcium and active vitamin D, and in more severe cases, managing mineral status can be extremely challenging. Croatia has recently made palopegteriparatide, a long-acting parathyroid hormone analogue, available in Croatia, but experiences with its use are still limited.

Objective: To present initial experiences and preliminary data on the efficacy and safety of palopegteriparatide in the treatment of severe postoperative hypoparathyroidism in Croatian endocrinology centers.

Methods: Retrospective data were collected on three patients with severe postoperative hypoparathyroidism following total thyroidectomy from three Croatian endocrinology centers. Demographic characteristics, previous therapy, doses of palopegteriparatide, laboratory findings (serum and ionised calcium, phosphate, calcium in 24-hour urine, PTH, and 25-OH vitamin D), and clinical outcomes were analyzed.

Results: All three patients had severe, refractory hypocalcemia after total thyroidectomy, and all three patients received high doses of active vitamin D, while two of the three (P-01 and P-03) also received teriparatide before the introduction of palopegteriparatide—P-01 for 2 months and P-03 for 43 months. After the introduction of palopegteriparatide (18–21 mcg daily), two patients (P-02, P-03) continued treatment with a reduction or maintenance of calcium dosage. Patient P-03, who had previously received teriparatide for the longest duration, showed the best outcome: stabilization of serum calcium (2.28→2.37 mmol/L), normalization of ionized calcium (1.15→1.27 mmol/L), no hypocalcemic episodes, and an improvement in quality of life. Patient P-02, despite the increase in the dose of palopegteriparatide (21→27 mcg), had persistently low ionized calcium (0.98 mmol/L) and an increase in phosphate. In patient P-01, the drug was discontinued after 17 months because of episodes of hypocalcemia and hypercalcemia and the reintroduction of active vitamin D.

Conclusion: Palopegteriparatide shows promising results in selected patients with severe postoperative hypoparathyroidism, with variable results. A more comprehensive evaluation of the efficacy and safety would be possible if data on all patients in Croatia were collected and could serve as a basis for including the drug on the list of particularly expensive medications.

IATROGENIC HYPERCALCEMIA IN A PATIENT WITH TYPE 2 DIABETES TREATED WITH GLP-1 RECEPTOR AGONIST AND CHRONIC KIDNEY DISEASE

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[3] Division of Endocrinology, Diabetes and Metabolic Diseases, Department of Internal Medicine, Sveti Duh University Hospital, Zagreb, Croatia

Background: The introduction of glucagon-like peptide-1 receptor agonists (GLP-1 RA) and non-steroidal mineralocorticoid receptor antagonists (nsMRA) has significantly improved outcomes in patients with type 2 diabetes, obesity and chronic kidney disease (CKD). However, the increasing use of combination therapies may result in previously unrecognized adverse drug interactions.

Case report: We report the case of a 59-year-old man with obesity, type 2 diabetes, CKD stage 3b, albuminuria and established cardiovascular disease receiving optimized treatment with renin–angiotensin–aldosterone system blockade, sodium-glucose cotransporter-2 inhibition, semaglutide and finerenone. Mild finerenone-associated hyperkalemia was treated with calcium polystyrene sulfonate. Several weeks later, the patient developed symptomatic severe non-parathyroid hormone-mediated hypercalcemia accompanied by acute kidney injury, fatigue, muscle weakness, constipation, polyuria and polydipsia. Extensive investigations excluded solid and hematological malignancy, granulomatous disease and endocrine disorders. Hypercalcemia was attributed to increased calcium absorption from calcium polystyrene sulfonate, likely facilitated by prolonged gastrointestinal transit secondary to semaglutide-induced constipation. Treatment consisted of withdrawal of the offending medications, aggressive intravenous hydration, loop diuretics and a reduced dose of intravenous zoledronic acid, resulting in normalization of serum calcium and recovery to baseline kidney function. Semaglutide and finerenone were subsequently reintroduced without recurrence of hyperkalemia or hypercalcemia, eliminating the need for potassium binders.

Conclusion: This case highlights a previously unreported mechanism of iatrogenic hypercalcemia resulting from the interaction between contemporary cardio-renal-metabolic therapies and underscores the importance of careful monitoring for unexpected adverse effects when combining newer agents with established treatments.

FROM NON-FUNCTIONING TO FUNCTIONING: HYPERINSULINEMIC HYPOGLYCEMIA IN METASTATIC PANCREATIC NEUROENDOCRINE TUMOR

Matea Majetić[1,2], Klara Džojić[1,2], Ema Schönberger[1,2], Romana Marušić[2,3], Kristina Steiner[1], Karla Lauš[1,2], Silvija Canecki-Varžić[1,2], Ines Bilić-Ćurčić[1,2]

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Introduction: A 68-year-old woman with type 2 diabetes mellitus was diagnosed approximately two years earlier with a well-differentiated grade 2 pNET (5 mitoses/2 mm², Ki-67 3%). Initial imaging demonstrated a 43 × 32 × 44 mm tumor of the pancreatic head. She underwent pancreatic surgery with lymphadenectomy. Subsequent somatostatin receptor imaging demonstrated receptor-positive disease involving the pancreatic region and liver, and long-acting octreotide therapy was initiated. During follow-up, radiological progression with hepatic metastatic disease was documented. Approximately 17 months after the initial diagnosis, the patient developed recurrent and progressively more frequent hypoglycemia. Although she had pre-existing type 2 diabetes, hypoglycemia persisted despite complete withdrawal of glucose-lowering therapy. During spontaneous hypoglycemia (plasma glucose 2.3 mmol/L), insulin was markedly elevated (89.47 mIU/L), with a C-peptide concentration of 3.23 nmol/L, confirming endogenous hyperinsulinemic hypoglycemia. In the context of progressive metastatic pNET, these findings raised suspicion of acquired insulin secretion and evolution toward a clinically functioning phenotype. CGM demonstrated a substantial hypoglycemic burden. During a 28-day assessment, 36 low-glucose events were recorded, with a mean duration of 85 minutes. Following multidisciplinary evaluation, long-acting octreotide was temporarily discontinued because of its potential to aggravate hypoglycemia. Due to persistent clinically significant hypoglycemia, diazoxide was initiated at a dose of 50 mg three times daily, with dose adjustment according to the glucose profile. During the available 14-day CGM period spanning treatment initiation, 18 low-glucose events were recorded, with a mean duration of 71 minutes. These findings suggested early improvement in both the frequency and duration of hypoglycemic episodes, although residual hypoglycemia persisted.

Discussion: The emergence of endogenous hyperinsulinemic hypoglycemia during progression of a previously clinically non-functioning pNET suggests acquired

functional activity, a rare but clinically important phenomenon. Persistence of hypoglycemia after withdrawal of antidiabetic therapy, together with simultaneous measurement of glucose, insulin, and C-peptide during hypoglycemia, was crucial for diagnosis. CGM objectively demonstrated the frequency and duration of hypoglycemia and supported assessment of the early therapeutic response. Diazoxide, through inhibition of insulin secretion, remains an important pharmacological option when definitive tumor-directed treatment is not immediately feasible.

Conclusion: New-onset hypoglycemia in patients with metastatic pNET should prompt evaluation for acquired hormone secretion, even in those with pre-existing type 2 diabetes. Biochemical assessment combined with CGM facilitates diagnosis and monitoring, while diazoxide may provide effective symptomatic control while further oncological management is pursued.

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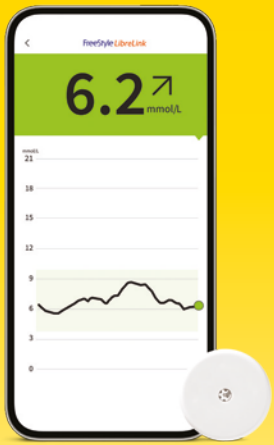

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1. Fokkert, M. BMJ Open Diabetes Research & Care (2019). https://doi.org/10.1136/bmjdrc-2019-000809. **2.** Haak, T. Diabetes Therapy (2017). https://doi.org/10.1007/s13300-016-0223-6. **3.** Unger, J. Postgraduate Medicine (2020). https://doi.org/10.1080/00325481.2020.1744393. **4.** Podaci u datoteci. Abbott Diabetes Care, Inc. Na temelju broja korisnika diljem svijeta za FreeStyle Libre portfelj u usporedbi s brojem korisnika za druge vodeće sustave za praćenje glukoze temeljene na senzorima za osobnu upotrebu.

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Broj odobrenja za stavljanje lijeka u promet: EU/1/25/1946/001

Ovaj promotivni materijal sadrži bitne podatke o lijeku istovjetne onima iz Sažetka opisa svojstava lijeka (SPC), sukladno članku 15. Pravilnika o načinu oglašavanja o lijekovima (Narodne novine br. 43/15). Prije propisivanja i/ili davanja lijeka molimo pročitajte cjeloviti odobreni SPC/Uputu o lijeku dostupne na internetskoj stranici Agencije za lijekove i medicinske proizvode RH (www.halmed.hr).

Terapijske indikacije: Liječenje osteoporoze u žena u postmenopauzi i u muškaraca s povećanim rizikom od fraktura. Liječenje gubitka koštane mase povezano s hormonskom ablacijom u muškaraca s rakom prostate koji imaju povećani rizik od fraktura i povezano s dugotrajnom sistemskom terapijom glukokortikoidima u odraslih bolesnika koji imaju povećani rizik od fraktura. **Doziranje:** Preporučena doza je 60 mg - jedna supkutana injekcija u bedro, trbuh ili nadlakticu jednom u 6 mjeseci. Bolesnici moraju biti opskrbljeni kalcijem i vitaminom D i mora im se dati uputu o lijeku i karticu s podsjetnikom za bolesnika. Optimalno trajanje terapije nije utvrđeno. Potreba za kontinuiranom terapijom treba se individualno periodički procjenjivati, posebno nakon 5 ili više godina primjene. *Starije osobe (dob ≥ 65)* i *Oštećenje funkcije bubrega* - Nije potrebna prilagodba doze. *Oštećenje funkcije jetre* - Djelotvornost i sigurnost nisu ispitivani u ovih bolesnika. *Pedijatrijska populacija* - ne smije se primjenjivati u djece < 18 godina. **Način primjene:** Supkutano, od strane osobe primjereno naučene za davanje injekcija. **Kontraindikacija**: Preosjetljivost na djelatnu tvar ili neku od pomoćnih tvari. **Posebna upozorenja i mjere opreza pri uporabi:** Radi sljedivosti bioloških lijekova, naziv i broj serije potrebno je jasno evidentirati. Primjeren unos kalcija i vitamina D je važan. *Hipokalcijemija* - Važno je prepoznati rizik te se prije mora korigirati unosom kalcija i vitamina D i kroz liječenje pratiti. Bolesnike treba potaknuti na prijavu simptoma hipokalcijemije jer postoje teški prijavljeni slučajevi (većinom u prvim tjednima liječenja), a istodobno liječenje glukokortikoidima dodatni je faktor rizika. *Bolesnici s teškim oštećenjem funkcije bubrega* (klirens kreatinina < 30 ml/min) ili bolesnici koji su na dijalizi imaju povećan rizik za hipokalcijemiju. *Infekcije kože* – Može se razviti infekcija kože (pretežno celulitis) kada odmah treba zatražiti pomoć. *Osteonekroza čeljusti (ONČ)* – Rijetko je prijavljena, ali treba oprez, liječenje treba odgoditi ako ima lezija u ustima. U rizičnih se preporučuje stomatološki pregled te preventivni zahvati i individualna procjena koristi i rizika prije liječenja. Faktori rizika su potentnost i doza lijeka, parenteralni put primjene, rak, komorbiditetna stanja (npr. anemija, koagulopatije, infekcija), pušenje, kortikosteroidi, kemoterapija, inhibitori angiparogeneze, radioterapija glave i vrata, loša oralna higijena, bolesti parodonta, slabo prianjajuće zubne proteze, povijest dentalne bolesti, invazivni stomatološki zahvat (npr. vađenje zuba). Treba izraditi plan zbrinjavanja za bolesnike koji razviju ONČ, razmotriti privremeni prekid terapije, ukoliko je moguće. *Osteonekroza vanjskog slušnog kanala* i *Atipične frakture femura* prijavljene su u bolesnika koji primaju denosumab. *Dugoročna antiresorptivna terapija* - može doprinijeti povećanom riziku. *Istodobno liječenje ostalim lijekovima koji sadrže denosumab* – ne smije. *Upozorenja o pomoćnim tvarima* - sadrži polisorbat 20 koji može uzrokovati alergijske reakcije. **Nuspojave:** Najčešće su mišićno-koštana bol i bol u ekstremitetima. Česte su infekcije mokraćnog te gornjeg dišnog sustava, išijas, gastrointestinalni poremećaji, konstipacija, nelagoda u abdomenu, osip, ekcem, alopecija.

Detaljne informacije o svim nuspojavama molimo pogledajte u odobrenom Sažetku opisa svojstava lijeka.

▼ Ovaj je lijek pod dodatnim praćenjem. Time se omogućuje brzo otkrivanje novih sigurnosnih informacija. Od zdravstvenih radnika se traži da prijave svaku sumnju na nuspojavu za ovaj lijek. Upute za prijavljivanje dostupne su na www.halmed.hr.

Literatura:

1. EMA. EVFRAXY. Izvješće - https://www.ema.europa.eu/en/documents/variation-report/evfraxy-h-c-006526-0000-epar-assessment-report_en.pdf Pristupljeno: 02. ožujka 2026.

EVF-2026-00005-HR, ožujak 2026.

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forxiga
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*uz ostalu standardnu terapiju za ŠBT2, KBB ili ZS.
Dapagliflozin je indiciran za liječenje šećerne bolesti tipa 2, kronične bubrežne bolesti i simptomatskog kroničnog zatajenja srca.[1]
1. Sažetak opisa svojstava lijeka Forxiga.

DKD, dijabetička bolest bubrega; eGFR, procijenjena stopa glomerularne filtracije; HbA1c, glikirani hemoglobin A1c; HFmrEF, zatajivanje srca s blago sniženom ejekcijskom frakcijom; HFpEF, zatajivanje srca s očuvanom ejekcijskom frakcijom; HFrEF, zatajivanje srca s reduciranom ejekcijskom frakcijom; KBB, kronična bubrežna bolest; KRM, kardiorenometabolički; LVEF, ejekcijska frakcija lijevog ventrikula; NT-proBNP, N-terminalni pro-B-tip natriuretski peptid; ŠBT2, šećerna bolest tip 2; UACR, omjer koncentracije albumina i kreatinina u urinu; ZS, zatajivanje srca.

Sastavni dio ovog promotivnog materijala za lijek Forxiga predstavlja skraćeni odobreni Sažetak opisa svojstava lijeka Forxiga, sukladno članku 14. i članku 15. Pravilnika o načinu oglašavanja o lijekovima ("Narodne novine" broj 43/15). Prije propisivanja lijeka Forxiga molimo pročitajte zadnji odobreni Sažetak opisa svojstava lijeka i Uputu o lijeku. Samo za zdravstvene radnike.

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HbA1c, glikozilirani hemoglobin

Reference: 1. Sažetak opisa svojstava lijeka Toujeo, 3/2026. 2. Home PD, et al. *Diabetes Care* 2015;38(12):2217-2225. 3. Matsuhisa M, et al. *Diabetes Obes Metab* 2016;18(4):375-383. 4. Danne T, et al. *Diabetes Care* 2020;43(7):1512-1519. 5. Riddle MC, et al. *Diabetes Care* 2014;37:2755-2762. 6. Yki-Jarvinen H, et al. *Diabetes Care* 2014;37:3235-3243. 7. Bolli GB, et al. *Diabetes Obes Metab* 2015;17:386-394. 8. Becker RHA, et al. *Diabetes Care* 2015;38(4):637-43. 9. Singh R, et al. *Eur Endocrinol* 2018;14:47-51. 10. Pohlmeier H, et al. *J Diabetes Sci Technol* 2017;11;263-269.

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[Δ]Indiciran u odraslih osoba s utvrđenom aterosklerotskom kardiovaskularnom bolešću ili visokim rizikom za njezin nastanak kako bi se snižavanjem razine LDL-K smanjio kardiovaskularni rizik.[1,2]

*Vrijednost korigirana placebom u randomiziranom, dvostruko slijepom, placebom kontroliranom ispitivanju faze II, u kojem se procjenjivalo snižavanje LDL-K kombinacijom bempedoatne kiseline (180 mg), ezetimiba (10 mg) i atorvastatina (20 mg). Trojna terapija (n=41); placebo (n=20).[3]

[†]Bolesnici uključeni u CLEAR Outcomes ispitivanje bili su intolerantni na statine, s početnom vrijednošću LDL-K ≥100 mg/dL; na početku ispitivanja 22,7% ih je primalo statin, a 11,5% ezetimib. Rezultati prikazuju opaženo relativno smanjenje KV rizika.[4]

KV: kardiovaskularni; **LDL-K:** kolesterol lipoproteina niske gustoće

1. NILEMDO® Sažetak opisa svojstava lijeka 2. NUSTENDI® Sažetak opisa svojstava lijeka 3. Rubino J et al. Atherosclerosis. 2021; 320:122-128. 4. Nissen SE et al. Engl J Med. 2023;388:1353-64.

Samo za zdravstvene radnike: Sastavni dio ovog materijala su zadnji odobreni Sažetak opisa svojstava lijeka i Uputa o lijeku koje možete pronaći na internetskoj stranici Europske agencije za lijekove www.ema.europa.eu te vas molimo da ih pročitajte prije primjene lijeka. Lijek se izdaje na recept. Broj odobrenja: EMEA/H/C/004958, EMEA/H/C/004959. Nositelj odobrenja: Daiichi Sankyo Europe GmbH, Zielstattstrasse 48, 81379 München, Njemačka: Predstavnik nositelja odobrenja: Zentiva d.o.o., Av. V. Holjevca 40, 10000 Zagreb, Tel: + 385 1 6641 830, e-mail: pv-Croatia@zentiva.com. Šifra i datum odobrenja: 000787861-09-2025.

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NAPOMENA: Upućujemo zdravstvene radnike na posljednje cjelokupne sažetke opisa svojstava lijekova te upute o lijekovima za lijekove Plivit® D3, Teriparatid PLIVA i Ponlimsi® koji su dostupni na internetskoj stranici Agencije za lijekove i medicinske proizvode (www.halmed.hr). **Način izdavanja:** na recept, u ljekarni. **Datum sastavljanja:** ožujak 2026. OSTA-HR-00177

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1. Carlson AL, et al. Diab Tech and Therap. 2021; DOI 10.1089/dia.2021.0319

* Pogledajte korisnički vodič za sustav – značajka SmartGuard™. Potrebna je određena interakcija između korisnika i sustava.

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