



OSCON MF
OSIJEK STUDENT CONGRESS

TAILORED TO YOU:

FROM GENOME INSIGHT
TO INTELLIGENT
IMMUNE CARE

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8TH INTERNATIONAL TRANSLATIONAL
MEDICINE CONGRESS OF STUDENTS AND
YOUNG PHYSICIANS

OSCON



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GENERAL INFORMATION



DATE

March 26th - March 28th, 2026



MAIN VENUE

Faculty of Civil Engineering and
Architecture Osijek

Vladimira Preloga 3, Osijek



TOPIC

TAILORED TO YOU:

From Genome Insight to Intelligent
Immune Care



GUEST ATTENDANCE POLICY

All registered participants are welcomed
to all events and lectures.

Wearing official conference badges is
obligatory for entering any events.



LANGUAGE

English

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8TH INTERNATIONAL TRANSLATIONAL MEDICINE
CONGRESS OF STUDENTS AND YOUNG PHYSICIANS



WELCOME MESSAGES

March 26th - March 28th
2026



KATARINA VUKOVIĆ

President of the
Organising Committee



*Dear colleagues and friends,
it is my great honor and privilege to welcome you on behalf of the Organising Committee of the Osijek Student Congress, better known as OSCON.*

Over the years, OSCON has grown into a vibrant international platform that brings together students, young researchers, and mentors with a shared passion for science, innovation, and progress in medicine.

This year's theme invites us to step into the future of medicine—a future that is no longer distant, but already unfolding. It reflects the beginning of a new era in which our understanding of the human body reaches deeper, molecular levels, and therapies become tailored to each individual patient.

At its core are innovations that unite science, technology, and clinical practice, transforming the way we approach health and disease. At the same time, it serves as a powerful reminder for young doctors that medicine is not about treating a disease, but about caring for the patient as a whole.

What once started as an idea has grown into a tradition that continues to inspire, educate, and connect generations.

Thank you for being a part of our story and for helping to shape a better tomorrow.

TAMARA STANOJLOVIĆ

President of the
Scientific Committee



*Dear colleagues,
On behalf of the Scientific Committee, it is my great pleasure to welcome you to the eighth edition of OSCON. This year's congress is held under the theme "Tailored to You: From Genome Insight to Intelligent Immune Care," highlighting the growing role of precision medicine and individualized therapeutic approaches in modern healthcare.*

Advances in molecular biology, data science, and immunology are rapidly transforming the way we understand disease and approach treatment. The scientific program, therefore, focuses on how these developments translate into clinical practice and how modern biomedical discoveries are shaping the future of diagnosis and therapy.

This year's program features nine invited lectures by distinguished experts and fourteen interactive workshops, offering participants both broader scientific insight and opportunities for practical, hands-on learning. While the lectures explore emerging concepts in contemporary medicine, the workshops encourage active participation and the development of clinical skills in smaller groups.

Student research remains at the heart of OSCON. Participants will present their work through electronic and printed poster sessions, while a special session will showcase presentations of the ten highest-rated abstracts.

More than a scientific meeting, OSCON is a place for discussion, collaboration, and new ideas. These days will bring new knowledge, inspiration, and meaningful professional connections.

Thank you for being part of OSCON 2026.

8TH INTERNATIONAL TRANSLATIONAL MEDICINE
CONGRESS OF STUDENTS AND YOUNG PHYSICIANS



ABOUT US

March 26th - March 28th

2026

FACULTY OF MEDICINE OSIJEK



The Faculty of Medicine of University of Osijek is the youngest medical faculty in Croatia. It was officially opened in 1968 as a branch of the School of Medicine in Zagreb, and in 2018 it celebrated 20 years as the independent medical faculty. The largest and main base of the faculty is the University Hospital Centre Osijek. The Faculty of Medicine in Osijek is the only medical faculty in Croatia that shares the same campus with its main teaching base, which is located within the University Hospital Centre complex.

Apart from a three-year undergraduate programme and two-year graduate study programmes (Biomedical-Laboratory Technologies) and a six-year long first-degree study programme in Medicine, the Faculty of Medicine offers several postgraduate specialist study programmes and doctoral study programme.

The aim is to provide students the highest level of knowledge and training adequate for the professions they are pursuing. In addition, the Faculty of Medicine Osijek has defined main research areas and has been profiled by several interdisciplinary groups that link basic and clinical studies (translational medicine). We would also like to point out that, our two professors' emeriti Antun Tucak and Savo Jovanović contributed to the faculty with their work and effort.

The Josip Juraj Strossmayer University of Osijek is a university located in Osijek, making it the flagship institution of higher education in Slavonia, and one of the largest and oldest universities in Croatia. It was founded in 1775 and it is organized in 12 faculties, 4 departments and one academy. University is a medium-size in comparison to other European Universities. It is ranked as the Croatia's fourth best university among the 49 ranked institutions of tertiary education from the country.

University of Osijek has been developing into a modern European institution of higher education, and it is becoming a regional centre of knowledge, research and excellence. All efforts are directed towards the constant increase of teaching and studying quality.

The University offers a high student standard concerning accommodation, learning facilities and other student services.

Since over 20 000 students study at University of Osijek, did you know that the city itself is known as the City of Students?

UNIVERSITY OF OSIJEK



CITY OF OSIJEK



Osijek is a modern Central European city with 17 city parks and gardens which make Osijek one of the greenest cities in Croatia. The City of Osijek is also famous for secession (a variation of art nouveau). The promenade along the Drava river is one of the longest walking trails in Croatia. Given the city of Osijek's long history, there's a variety of sights such as Tvrdica, a fortified part of the city from the 18th century.

Some of the most valuable examples of Baroque architecture in Croatia, such as the statue of Holy Trinity and General's-headquarters are located in Tvrdica and were printed on 200 kuna bills, which was Croatia's official currency prior euro.

The tradition of higher education in Osijek exists since 1707 and today our university with its 17 faculties and departments is one of the most important scientific centres in Croatia.

Croatia is a country on the north-western edge of the Balkan peninsula and has a population of 4,05 million people and covers an area of 56,594 km². Five countries border Croatia: Bosnia and Herzegovina, Hungary, Montenegro, Serbia, and Slovenia, and it shares a sea border with Italy. The country offers a long coastline with over a thousand islands along its coast at the Adriatic Sea (Mediterranean Sea), a variety of medieval towns and villages, and a pleasant Mediterranean climate.

The capital of Croatia is Zagreb, placed in the northwest of the country. Croatia's second-largest city is Split, situated on a peninsula in the southern part of Adriatic Sea. Remaining two largest cities are the Port of Rijeka located on the northern shore of the Adriatic Sea and Osijek in the far east of Croatia. Other well-known towns include Dubrovnik, Makarska, Poreč, Rovinj, Opatija and Zadar. Croatia has declared its independence of Socialist Federal Republic of Yugoslavia (SFRY) in 1991.

The Croatian War of Independence started right after that, ending four years later with Croatia as a victor. Despite Croatia's hard history that has left enormous consequences on the country, it could not affect on Croatia's natural beauty that contains some of the prettiest natural wonders in the world! Alongside with its natural beauty, culture. Cultural Heritage and beautiful coastline are only some of the reasons why Croatia is certainly worth visiting.

CROATIA



8TH INTERNATIONAL TRANSLATIONAL MEDICINE
CONGRESS OF STUDENTS AND YOUNG PHYSICIANS



PROGRAMME

March 26th - March 28th

2026

THURSDAY

MARCH 26TH

09:00 - 11:00

REGISTRATIONS

Lobby (G)

11:00 - 11:30

OPENING CEREMONY

CROATIAN LANGUAGE OPENING SPEECH

0.3 Hall (G)

11:45 - 13:15

LECTURE SESSION I.

0.3 Hall (G)

11:45 - 12:15

PROF. DRAGAN PRIMORAC, MD, PHD

Artificial Intelligence and Bioinformatics in Personalized Medicine: From Genome to Therapy

12:15 - 12:45

PROF. JANOŠ TERZIĆ, MD, PHD

Gut Microbiota Carcinogen Metabolism Causes Distal Tissue Tumours

12:45 - 13:15

VJEKOSLAV KOPAČIN, MD, PHD

The Third Dimension of Radiology: Visualization, Planning, and 3D Printing for Personalized Treatment

13:15 - 14:00

APPETIZERS BREAK

Lobby (G)

13:15 - 14:00

STUDENT SESSION (1)

PRINTED POSTER SESSION

Lobby (G)

14:00 - 15:45

STUDENT SESSION I.

E-POSTER PRESENTATIONS

0.2 Hall, II.51 (G)

16:00 - 19:15

WORKSHOPS I.

16:00 - 17:30

SESSION I.

THE SURGICAL BACKBONE III.43 (G)

THE HIDDEN CAUSE III.45 (G)

POINTS OF PRECISION III.46 (G)

SESSION II.

BRINGING DNA TO LIGHT Microbiology and Biology Laboratory (M)

WHEN ALGORITHMS ARE NOT ENOUGH Skills Cabinet (M)

17:45 - 19:15

20:00

SOCIAL PROGRAMME

Pub Quiz

Campus Caffè

A - Faculty of Agrobiotechnical Sciences Osijek

G - Faculty of Civil Engineering and Architecture Osijek

M - Faculty of Medicine Osijek

FRIDAY

MARCH 27TH

09:00 - 10:45

STUDENT SESSION II.

II.50, II.51, II.52 (G)

E-POSTER PRESENTATIONS

10:45 - 11:15

STUDENT SESSION (2)

Lobby (G)

PRINTED POSTER SESSION

11:30 - 13:00

LECTURE SESSION II.

Aula Magna (A)

11:30 - 12:00

PROF. TATJANA CRNOGORAC- JURČEVIĆ, MD, PHD

Towards Early Detection of Pancreatic Cancer

12:00 - 12:30

BEATA HALASSY, PHD

Oncolytic Virotherapy: Cancer Treatment Using Viruses

12:30 - 13:00

ASSOC. PROF. SUZANA MIMICA, MD, PHD

Why One Drug Doesn't Fit All: Prescribing Through the Lens of Pharmacogenetics

13:00 - 14:00

LUNCH BREAK

Lobby (G)

14:00 - 14:45

STUDENT SESSION (3)

Lobby (G)

PRINTED POSTER SESSION

15:00 - 18:15

WORKSHOPS II.

15:00 - 16:30

SESSION I.

IMMUNOTHERAPY UNDER THE SPOTLIGHT
II.50 (G)

UNDER PRESSURE II.51 II.52 (G)

BYPASS THE LIMITS III.43 (G)

16:45 - 18:15

SESSION II.

DECODING THE SCAN II.53 (G)

ENGINEERING IMMUNITY Microbiology
and Biology Laboratory(M)

21:00

SOCIAL PROGRAMME

Pivnica Bure Bar

OSCON Party

A - Faculty of Agrobiotechnical Sciences Osijek

G - Faculty of Civil Engineering and Architecture Osijek

M - Faculty of Medicine Osijek

SATURDAY

MARCH 28TH

09:30 - 11:30

STUDENT SESSION III.

0.2 Hall (G)

Top 10 Oral Presentations

11:45 - 13:15

LECTURE SESSION III.

0.3 Hall (G)

11:45 - 12:15

PROF. IGOR AURER, MD, PHD

CAR-T Therapy: Practical Aspects and Clinical Application

12:15 - 12:45

PROF. GORAN MAROŠEVIĆ, MD, PHD

Walking on the Edge: Radiotherapy in Clinical Practice

12:45 - 13:15

KATA MEĐUGORAC, MD

Immunotherapy as a Model of Precision Treatment: How to Select the Right Patient for the Right Therapy

13:15 - 13:45

STUDENT SESSION (4)

Lobby (G)

PRINTED POSTER SESSION

13:15 - 13:45

COFFEE BREAK

Lobby (G)

13:45 - 16:30

WORKSHOPS III.

13:45 - 15:00

SESSION I.

FROM CUT TO CONTINUITY III.43 (G)

KIDNEY CODES III.44 (G)

HEAL AND SEAL III.45 (G)

15:15 - 16:30

SESSION II.

TRACING THE CELLS III.46 (G)

16:30

CLOSING CEREMONY

0.2 Hall (G)

20:00

SOCIAL PROGRAMME

Projekt 9, Osijek

Gala dinner

A - Faculty of Agrobiotechnical Sciences Osijek

G - Faculty of Civil Engineering and Architecture Osijek

M - Faculty of Medicine Osijek

VENUE LOCATIONS

OSCON 2026



**Faculty of Agrobiotechnical
Sciences Osijek**

VLADIMIRA PRELOGA 1,
OSIJEK



**Faculty of Civil Engineering
and Architecture Osijek**

VLADIMIRA PRELOGA 3,
OSIJEK



Faculty of Medicine Osijek

JOSIPA HUTTLERA 4,
OSIJEK

8TH INTERNATIONAL TRANSLATIONAL MEDICINE
CONGRESS OF STUDENTS AND YOUNG PHYSICIANS



SPEAKERS

March 26th - March 28th

2026

VJEKOSLAV KOPAČIN, MD, PHD



LECTURE TITLE

The Third Dimension of Radiology: Visualization, Planning, and 3D Printing for Personalized Treatment

Vjekoslav Kopačin, MD, is a consultant radiologist at the Department of Diagnostic and Interventional Radiology, University Hospital Centre Osijek. He graduated from the Faculty of Medicine, University of Osijek, in 2011 and began his medical career in emergency medicine, working with the Osijek-Baranja County Emergency Medical Service, where he gained extensive experience in acute and critical care. He later completed residency training in Clinical Radiology at UHC Osijek and has been working as a specialist radiologist since 2020. His primary professional focus is interventional radiology, with a particular interest in minimally invasive vascular and non-vascular procedures that offer effective, patient-centred alternatives to conventional surgical approaches. In parallel with his clinical work, Dr. Kopačin has been actively involved in medical education since 2020 as an assistant lecturer at the Department of Biophysics and Radiology at the Faculty of Medicine, University of Osijek. He is currently enrolled in the postgraduate doctoral program in Biomedical Sciences and Health. His professional interests extend to innovative applications of 3D printing and advanced visualization techniques in medicine, particularly for procedural planning and teaching. Outside the hospital, he enjoys travelling, watching TV series, and spending time outdoors on long walks with his wife and their dog, Gizmo.

PROF. JANOŠ TERZIĆ, MD, PHD

LECTURE TITLE

Gut Microbiota Carcinogen Metabolism Causes Distal Tissue Tumours

Janoš Terzić, MD, PhD, is a Croatian physician and biomedical scientist, and a Professor at the Department of Immunology and Medical Genetics, School of Medicine, University of Split. He graduated from the University of Zagreb in 1991 and earned his PhD seven years later. Professor Terzić founded the Laboratory for Cancer Research and initiated the NobelST program in Split, fostering international collaboration and innovation in cancer research. Under his leadership, his research team made a major discovery on tumor formation in chronic inflammation, published in Nature in 2024. He has also contributed to research at the University of California San Diego, the University of Connecticut Health Center, and the Max Planck Institute. His work has resulted in over 40 scientific publications, with a strong citation record in Web of Science. Professor Terzić combines clinical expertise with academic leadership, mentoring students and young researchers while advancing biomedical science. He has received multiple awards for scientific excellence, including the Croatian State Award for Science, and remains committed to translating research discoveries into meaningful clinical and societal impact.



PROF. DRAGAN PRIMORAC,
MD, PHD

LECTURE TITLE

Artificial Intelligence and Bioinformatics in Personalized Medicine: From Genome to Therapy

Professor Dragan Primorac, MD, PhD, is a pediatrician, geneticist, and forensic expert, internationally recognized for his pioneering work in personalized medicine, forensic genetics, and regenerative medicine. Over the past several years, he has consistently been ranked among the top 2% of scientists worldwide based on career-long and single-year impact metrics. He holds faculty positions at The Pennsylvania State University (Eberly College of Science) and the University of New Haven (USA), and serves as professor at medical schools in Split, Rijeka, and Osijek, Croatia, as well as in Germany. He has also been a Visiting Professor at Xi'an Jiaotong University, China, and in 2021 became the first European Professor Emeritus at the National Forensic Science University of India. Professor Primorac is the author of nearly 300 publications and 30 books or chapters, cited more than 10,600 times. His research has appeared in leading journals including Science and Nature, and he has given invited talks at over 150 international conferences. Early in his career, he was a pioneer in DNA-based identification of human remains from mass graves and contributed to major molecular genetics studies in the US. He also co-authored landmark research on European genetic history and the early migration of modern humans out of Africa. Since 2017, his focus has been on mesenchymal stem cells, regenerative medicine, and personalized therapies. His work includes the first molecular demonstration of cartilage regeneration using micro-fragmented adipose tissue, development of genetic screening for prevention of sudden cardiac death in athletes, and advanced regenerative treatments for severe COVID-19. Beyond research, Professor Primorac co-founded the International Society of Applied Biological Sciences (ISABS), collaborates with the Mayo Clinic, and serves on the Executive Committee of the International Consortium for Personalized Medicine (IC PerMed). He is founder of St. Catherine Specialty Hospital in Croatia and served as Minister of Science, Education, and Sports (2003–2009). He has received numerous national and international awards recognizing his contributions to science, education, and society.

**ASSOC. PROF. SUZANA MIMICA,
MD, PHD**

LECTURE TITLE

Why One Drug Doesn't Fit All: Prescribing Through the Lens of Pharmacogenetics



Suzana Mimica, MD, PhD, is an Associate Professor of Clinical Pharmacology and Toxicology at the Faculty of Medicine, University of Osijek, and a senior clinical pharmacologist at the University Hospital Centre Osijek. She graduated with distinction from the Faculty of Medicine, University of Zagreb, in 1996 and completed her specialization in Clinical Pharmacology and Toxicology in 2004. At UHC Osijek, she established the Diagnostic and Therapeutic Unit for Clinical Pharmacology within the Department of Internal Medicine, which she now leads. Since 2004, she has served as chair of the Hospital Drug Committee. Since December 2024, she has been the Assistant Director for Quality, actively contributing to patient safety and institutional quality standards. Professor Mimica is heavily involved in medical education, teaching basic and clinical pharmacology at undergraduate, postgraduate, specialist, and doctoral levels in Croatian and German. She also holds an academic position at the Faculty of Medicine, University of Rijeka. She participates in specialist and postgraduate programs in drug development and clinical pharmacy in Zagreb and at the University of Tartu, Estonia. Her professional work also extends to national and European regulatory agencies, including the European Medicines Agency and HALMED. Her primary focus in clinical and research work is on pharmacotherapy in older adults and medication safety, with a strong emphasis on preventing adverse drug reactions in vulnerable patient groups.

**PROF. TATJANA CRNOGORAC-
JURČEVIĆ, MD, PHD**

LECTURE TITLE

Towards Early Detection of Pancreatic Cancer

Tatjana Crnogorac-Jurčević, MD, PhD, is a Professor of Molecular Pathology and Biomarkers at the Barts Cancer Institute, Queen Mary University of London. She graduated from the Medical Faculty at the University of Zagreb and earned her PhD at the Imperial College School of Medicine in London. After completing postdoctoral research in molecular biology at the CNRS in Toulouse, France, and in molecular oncology at the CRUK laboratory at Hammersmith Hospital, London, she joined the Barts Cancer Institute in 2004.

Her research focuses on the molecular pathology of pancreatic cancer, aiming to develop biomarkers for early and noninvasive detection of this aggressive disease in urine samples. Professor Crnogorac-Jurčević's work connects fundamental research with clinical applications, advancing precision medicine in oncology. In addition to her research, she is actively involved in teaching, leading Problem-Based Learning, and the Student Selective Component for undergraduate students. She is also the Module Leader for Genomic Approaches to Cancer, a core module across multiple programs, including Cancer and Molecular Pathology and Genomics, Cancer and Molecular and Cellular Biology, and Cancer and Therapeutics courses, helping train the next generation of cancer researchers and clinicians.



BEATA HALASSY, PHD

LECTURE TITLE

Oncolytic Virotherapy: Cancer Treatment Using Viruses



Beata Halassy, PhD, is a Scientific Adviser with tenure and Head of a research team and laboratory at the Centre for Research and Knowledge Transfer in Biotechnology, University of Zagreb. She trained as a molecular biologist at the Faculty of Science, University of Zagreb, where she obtained her PhD in Biological Sciences. After 19 years in biopharmaceutical R&D focused on vaccines and plasma-derived medicinal products, her research now centres on virus–host interactions and the therapeutic exploitation of viruses. Her current work focuses on oncolytic virotherapy, investigating the use of viruses as anticancer agents. She is the inventor of patented technology in virus purification, protected by granted European and US patents, and currently leads five complementary projects in oncolytic virotherapy. Dr. Halassy has published 78 peer-reviewed papers and has an h-index of 21.

KATA MEDUGORAC, MD

LECTURE TITLE

Immunotherapy as a Model of Precision Treatment: How to Select the Right Patient for the Right Therapy

Dr. Kata Medugorac was born in Mostar, Bosnia and Herzegovina, on April 8, 1991. She completed her secondary education at the Gymnasium of the Sisters of Mercy in Ljubuški and the Secondary Music School in Široki Brijeg in 2009. In the same year, she enrolled at the Faculty of Medicine at the University of Zagreb, where she graduated in 2015. Her medical degree thesis „Gastric Cancer,” was completed under the mentorship of Professor Mate Majerović at the University Hospital Centre Zagreb, Department of Surgery. She completed her internship at the University Hospital Centre Zagreb, where she first developed a strong interest in medical oncology. From June 2016 to February 2019, she worked as a general practitioner at the Zagreb Center Health Center. In February 2019, she began her residency in Medical Oncology at the University Hospital Centre Zagreb, and in January 2025, she passed the specialist examination in Medical Oncology with excellent results. Throughout her specialization, she has been actively involved in professional education as a lecturer at numerous courses and congresses in Croatia and abroad. Her primary areas of professional interest include sarcomas, melanoma, and rare tumors. She is the author and co-author of several scientific publications and actively participates in clinical research conducted at the University Hospital Centre Zagreb, including involvement in the EUMelaReg registry.



PROF. GORAN MAROŠEVIĆ, MD, PHD

LECTURE TITLE

Walking on the Edge: Radiotherapy in Clinical Practice



Prof. Goran Marošević graduated from the School of Medicine and subsequently began his professional career in the field of oncology and radiation therapy. He completed his specialization in Radiation Oncology at the University Clinical Center Tuzla, where he worked as a physician, resident, and later as a board-certified radiation oncologist. During his professional development, he received further training at leading oncology centers worldwide, including Houston Methodist Hospital (USA), with a particular focus on implementing advanced, high-precision radiotherapy techniques. Since 2013, he has worked within the Affidea network, where he served as a Radiation Oncologist and Medical Director of the Radiotherapy Center in Banja Luka. He also held the position of International Coordinator of radiotherapy centers in Bosnia and Herzegovina, Poland, and Turkey. He currently serves as the Medical Director of the Special Oncology Hospital – Medikol Medical Center project in Zagreb and is also the President of the Management Board of PZU Medikol Salinea in Živinice.

PROF. IGOR AURER, MD, PHD

LECTURE TITLE

CAR-T Therapy: Practical Aspects and Clinical Application

Igor Aurer, MD, PhD, is a Full Professor of Hematology at the Faculty of Medicine, University of Zagreb, and Head of the Department of Hematology at University Hospital Centre Zagreb. He graduated from the Faculty of Medicine, University of Zagreb, in 1987 and completed postgraduate training in internal medicine and hematology at the UHC Zagreb. He was awarded a Fulbright scholarship and spent two years at the Division of Hematology-Oncology at the UCLA School of Medicine in Los Angeles. He earned his PhD in 1992 with a dissertation focused on the activation of oncogenes in acute myeloid leukemia. His primary clinical and scientific interest is lymphoma, an area in which he has made a significant impact. Professor Aurer has authored numerous scientific publications, more than 60 of which are indexed in Current Contents, and his work has been cited over 1,200 times in the Web of Science (h-index 18). He has supervised several doctoral theses and led multiple nationally funded research projects. He is President of the Croatian Cooperative Group for Hematologic Diseases (KroHem) and actively participates in European lymphoma research networks. Since 2020, under his leadership, CAR T-cell therapy has been successfully implemented at UHC Zagreb for selected patients with acute lymphoblastic leukemia and diffuse large B-cell lymphoma.



8TH INTERNATIONAL TRANSLATIONAL MEDICINE
CONGRESS OF STUDENTS AND YOUNG PHYSICIANS

OSCON



WORKSHOPS

March 26th- March 28th

2026

THE HIDDEN CAUSE

WORKSHOP: LAPAROSCOPY AS A DIAGNOSTIC AND TREATMENT APPROACH IN ENDOMETRIOSIS

LEADERS: Andrea Teodosić, MD; Sandra Lea LuciĆ, MD

INTRODUCTION: Endometriosis is a common yet often underdiagnosed gynecological disease that significantly affects the quality of life and fertility of women of reproductive age. Chronic pelvic pain, dysmenorrhea, dyspareunia, and infertility represent only part of the burden associated with this disease. While non-invasive methods may raise suspicion, laparoscopy remains the gold standard for definitive diagnosis and treatment.

DESCRIPTION: This workshop provides a comprehensive overview of endometriosis, including its pathophysiology, clinical presentation, and management strategies. Special focus is placed on laparoscopy as the key diagnostic and surgical tool. By combining theoretical introduction with laparoscopy simulation, participants will learn to recognize endometriotic lesions, understand classification systems, and explore treatment options.

TRACING THE CELLS

WORKSHOP: CERVICAL CHANGES, HPV INFECTION, AND PAP TEST DEMONSTRATION

LEADERS: Andrea Teodosić, MD

INTRODUCTION: Cervical cancer is one of the most preventable malignancies due to effective screening methods and vaccination against human papillomavirus (HPV). The Pap test plays a crucial role in the early detection of precancerous and malignant cervical lesions. Understanding the link between HPV infection, cytological abnormalities, and cervical pathology is essential for accurate interpretation of screening results and appropriate clinical decision-making.

DESCRIPTION: This workshop provides an introduction to cervical cancer screening focusing on HPV infection, its oncogenic potential, and the natural history of cervical intraepithelial changes. Participants will learn about cytological classifications, common abnormalities, their clinical significance and the practical aspects of performing a Pap test as well as how screening results guide further clinical decisions.

IMMUNOTHERAPY UNDER THE SPOTLIGHT

WORKSHOP: IMMUNOTHERAPY IN DERMATOLOGY

LEADERS: Ivana Krajina, MD, PhD

INTRODUCTION: Immunotherapy is a modern therapeutic approach in dermatology that modulates the immune system to treat skin diseases, improving outcomes in conditions such as skin cancer, autoimmune, and chronic inflammatory skin disorders.

DESCRIPTION: The workshop features an expert lecture and an interactive session. Participants will learn mechanisms of action, indications, and clinical application of immunotherapy in the management of autoimmune and inflammatory skin diseases. Then, they will actively engage in an interactive quiz based on clinical case presentations. Therapeutic decisions, potential adverse effects, and practical use in everyday dermatological practice will be discussed, helping attendees gain a clear understanding of how immunotherapy shapes modern patient care.

DECODING THE SCAN

WORKSHOP: CASE-SOLVING RADIOLOGY WORKSHOP

LEADERS: Ivo Vincetić, MD

INTRODUCTION: Radiological imaging is essential across many areas of clinical medicine. As artificial intelligence becomes increasingly integrated into image interpretation, understanding how AI-generated analysis compares to human clinical reasoning is becoming increasingly important.

DESCRIPTION: This interactive case-solving workshop invites participants to analyze a variety of radiological images, including fractures and other common diagnostic findings. Students will interpret cases, propose diagnoses and clinical considerations, and then compare their conclusions with AI-generated solutions. The workshop highlights both the potential and limitations of AI in medical imaging, emphasizing the continued importance of clinical judgment.



KIDNEY CODES

WORKSHOP: HLA-BASED APPROACHES TO PERSONALIZED KIDNEY THERAPY

LEADERS: Ivana Tolj, MD

INTRODUCTION: The Human Leukocyte Antigen (HLA) system is central to immune recognition and transplantation medicine. Accurate HLA matching in kidney transplantation is crucial for reducing the risk of rejection and improving graft survival. Beyond transplantation, HLA typing increasingly contributes to individualized therapeutic strategies in nephrology.

DESCRIPTION: This interactive workshop explores the clinical relevance of HLA typing beyond transplantation. Key concepts will be introduced through brief quizzes and discussions, covering HLA classes and their impact on clinical decision-making. In the practical session, students will analyze two clinical cases using 3D kidney models, discussing how immunogenetic information can guide personalized treatment options. Participants will actively engage in group discussions, linking molecular insights to real patient scenarios.



BYPASS THE LIMITS

WORKSHOP: CORONARY ARTERY BYPASS GRAFT SIMULATION

LEADERS: Matija Drinković, MD; Stjepan Cvetković, MD; Domagoj Leko, MD

INTRODUCTION: Wouldn't it be great if we could give you a chance to become a cardiac surgeon for a day? Could you imagine bringing the operating room to you? During this workshop, our cardiac surgeons will guide you through the creation of a proximal anastomosis of the coronary artery bypass grafting using a saphenous vein.

DESCRIPTION: To make this workshop as realistic and useful as possible, we will be using swine hearts with a portion of the aorta still attached and human saphenous veins as a graft. You will see microvascular surgical instruments specifically used for such procedures, and it is up to you to try to save your patient.

NOTE: Only 4th-, 5th-, and 6th-year students with previous experience in surgical suturing may apply.





BRINGING DNA TO LIGHT

WORKSHOP: GENOMIC DNA ISOLATION WORKSHOP

LEADERS: Edi Rođak, PhD

INTRODUCTION: If we were to stretch out the DNA from just one human cell, it would be about 2 meters long. But when we look at a biological sample, where is this invisible thread hidden? How can we transform an oral swab into a sample where the genetic material becomes visible to the naked eye?

DESCRIPTION: This workshop aims to demystify the process of genetic extraction. We will guide you through the critical steps of cellular lysis, enzymatic digestion, and the principles of DNA adsorption onto silica-gel columns. This way, you will master the fundamental techniques required to obtain pure DNA - the starting point for all modern genomic research.

WHEN ALGORITHMS ARE NOT ENOUGH

WORKSHOP: ADVANCED LIFE SUPPORT WORKSHOP

LEADERS: Davor Klepo, MD; Josip Kajan, MD; Student Society for Anesthesiology "ANEZIOS" (Faculty of Medicine Osijek)

INTRODUCTION: Advanced Life Support is built on algorithms, and protocol, but real emergencies don't follow scripts. When seconds matter, ALS demands more than memorization - it demands clinical judgment, adaptability, and confidence under pressure.

DESCRIPTION: This workshop challenges the algorithm-first mindset through realistic ALS scenarios where uncertainty is expected. Through guided discussion and hands-on scenarios, participants will strengthen rhythm recognition, improve resuscitation quality, and develop effective decision-making under pressure. The content and pace will be adapted to participants' knowledge level, ensuring both a strong foundation and meaningful clinical challenge. Focus is placed on translating guidelines into real-world action while addressing common pitfalls and human factors in resuscitation.

NOTE: Only 4th-, 5th-, and 6th-year students with prior knowledge may apply.



POINTS OF PRECISION

WORKSHOP: INJECTION TECHNIQUES WORKSHOP

LEADERS: Maja Kožul, MD

INTRODUCTION: Botulinum toxin injections are widely known for their aesthetic applications, but their role in medicine extends far beyond cosmetic procedures. From neurology to rehabilitation and pain management, precise injection techniques play an important therapeutic role. Safe and effective treatment requires precise anatomical knowledge and proper injection technique.

DESCRIPTION: This hands-on workshop introduces participants to injection techniques using silicone head models. Students will learn anatomical landmarks, injection planning, and principles of safe and precise application. The workshop covers both aesthetic and therapeutic indications, highlighting the medical use of botulinum toxin in various clinical conditions.



ENGINEERING IMMUNITY

WORKSHOP: CAR T-CELL THERAPY WORKSHOP

LEADERS: Assist. prof. Stefan Mrđenović, MD, PhD; Sara Bonet, MD; Lea Atlagić, MD

INTRODUCTION: Discover CAR T-cell therapy, a groundbreaking approach that reprograms a patient's immune system to precisely target cancer cells.

DESCRIPTION: In this interactive workshop, participants will gain a clear understanding of T-cell biology, CAR construct design, clinical indications, and major complications such as cytokine release syndrome. The first part of this session will provide a concise overview of the scientific and clinical foundations of adoptive cell therapy. In the second part, under expert guidance, participants will perform venipuncture, process blood samples, centrifuge and isolate the buffy coat, and seed cells onto culture medium. Gain practical insight into the laboratory techniques that bring advanced immunotherapies from concept to clinical reality!



FROM CUT TO CONTINUITY

WORKSHOP: BOWEL ANASTOMOSIS WORKSHOP

LEADERS: Dejan Hil, MD; Marija Trconić, MD; Veronika Šikić, MD

INTRODUCTION: Bowel anastomosis is a fundamental skill in abdominal surgery, where precision, tissue handling, and proper technique directly influence patient outcomes. Creating a secure and functional connection between two segments of the gastrointestinal tract requires more than sutures - it requires an understanding of tissue behavior, tension, and continuity.

DESCRIPTION: This hands-on workshop focuses on practicing bowel anastomosis techniques using real biological models. Participants will learn the basic principles of gastrointestinal suturing: tissue alignment, suture spacing, tension control, and anastomotic integrity. The workshop is designed to provide initial practical experience with bowel anastomosis in a controlled, educational environment.

NOTE: Only 5th- and 6th-year students with previous experience in surgical suturing may apply.



HEAL & SEAL

WORKSHOP: ESSENTIAL SUTURING SKILLS WORKSHOP

LEADERS: Student Society for Surgery "INCISIOS" (Faculty of Medicine Osijek)

INTRODUCTION: Heal & Seal is a hands-on workshop that teaches practical wound management and suturing skills. Created by students for students, it helps participants gain confidence and know-how to assess, manage, and close wounds safely.

DESCRIPTION: In this workshop, you will go step by step through the clinical process: assess wounds, decide when suturing is needed, prepare the wound, and perform safe closure. Using silicone models, you'll practice instrument handling, placing stitches, tying knots, and managing suture tension. Whether it's your first time or you're brushing up on skills, this session gives you a chance to learn, experiment, and build confidence in a friendly, supportive environment.



THE SURGICAL BACKBONE: FROM LYMPH NODE TO TRANSPLANT

WORKSHOP: SURGERY IN ONCOLOGY AND TRANSPLANTATION

LEADERS: Branka Balaban, MD; Student Anatomy Society (Faculty of Medicine in Rijeka); Student Surgery Society (Faculty of Medicine in Rijeka)

INTRODUCTION: Surgery remains the cornerstone of modern medicine, linking diagnosis with definitive treatment. At its core lies a deep understanding of anatomy—essential for both oncologic precision and transplant success.

DESCRIPTION: In this hands-on session, participants will take on the role of a diagnostic surgeon, performing lymph node harvesting and bone marrow biopsy on porcine models while identifying key anatomical landmarks. After reviewing histological findings, the focus shifts to transplant technique, with practice in vascular and ureteric suturing on porcine kidneys. By uniting anatomical knowledge with technical skill, this workshop highlights the foundations of surgical excellence.

UNDER PRESSURE

WORKSHOP: POLYTRAUMA ASSESSMENT AND INITIAL MANAGEMENT WORKSHOP

LEADERS: Assist. Prof. Jelena Kovačević, MD, PhD; Dragan Sljepčević, MD; Mario Barukčić, MD

INTRODUCTION: Polytrauma patients are among the greatest challenges in emergency care. Multiple injuries, unstable vital signs, and limited time demand a structured and prioritized approach. Early recognition of life-threatening conditions and clear decision-making are essential for improving outcomes.

DESCRIPTION: This workshop focuses on the systematic initial assessment and management of polytrauma patients. Through case-based discussion and hands-on training, participants will practice trauma evaluation, injury prioritization, and clinical decision-making in realistic scenarios. Emphasis will be placed on the ABCDE approach, teamwork, and timely recognition of conditions requiring immediate intervention.

NOTE: Only 4th-, 5th-, and 6th-year students with prior knowledge may apply. Please prepare for the workshop using the material provided upon registration.

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BS05 Comparative study of the locus coeruleus in wild-type and PACAP gene knock-out mice with aging

Marcell Schmidt, Dániel Pham, Andrea Tamás

BS06 Investigating the role of Hemokinin-1 in Thermoregulation

David Vince Simon

CASE REPORT

CR00 A novel FGD1 mutation in a patient with Aarskog-Scott syndrome: Expanding the mutational and phenotypic spectrum

Ivano Schneider, Katarina Galinec, Marko Ivković, Marko Trbojević, Silvija Pušeljčić

CR01 When Treatment Becomes the Trigger: Immune-Related Colitis in Lung Adenocarcinoma Treated with Immunotherapy

Matias Andjal, Leon Davidović, Filip Ovničević, Matija Peić, Ivana Canjko

CR02 Epithelioid Angiosarcoma Mimicking Large-Vessel Vasculitis – A Case Report

Maritea Arelić, Marija Ban, Rea Divjanović, Ines Žitko, Filip Mirić

CR03 When a Mass Isn't What It Seems: A Case of Mediastinal Paraganglioma

Borna Atlagić, Magdalena Sikirić, Lea Jukić, Darija Alpeza, Ivana Fumiš

CR04 VEXAS Syndrome: A Case Report

Vanessa Augustić, Mia Mihaela Mašina, Vladimir Cvitanović, Lucija Marojević, Filip Mirić

CR05 Supraglottic stenosis as a rare complication of cicatricial mucosal pemphigoid: a case report

Fran Babić, Hana Marković, Petra Galić, Tomislav Ognjenčić, Željko Zubčić

CR06 Pancreatic Metastasis of Renal Adenocarcinoma More Than Two Decades After Nephrectomy – A Case Report

Marija Bačlija, Ivan Budimir

CR07 Targeted therapy in cholangiocarcinoma

Klara Balog, Petra Kolundžić, Dora Domijan, Dora Bedenic, Jasmina Marić Brozić

CR08 Brachial artery embolism in a patient with metastatic TNBC and bone marrow infiltration

Luka Balog, Lovro Balen, Mislav Blažević, Alen Beneš, Tajana Silovski

CR09 The Role of Comprehensive Genomic Profiling on Circulating Tumour DNA in the Management of Metastatic Breast Cancer

Petra Banovec, Mia Bakula, Natalija Dedić Plavetić

CR10 Foodborne Pathogen: A Fatal Case of *Listeria monocytogenes* Meningoencephalitis in an Immunocompromised Adult

Sara Banjeglav, Brigita Kupanovac, Luka Švitek, Bernarda Kapular, Mihaela Zlosa

CR11 Tuberculous Meningitis in an Immunocompetent Patient from a High-Burden Tuberculosis Setting: A Case Report

Matija Barić, Barbara Hunjadi, Fabijan Teskera, Luka Švitek, Barbara Grubišić

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Borna Borić, Vladimir Cvitanović, Tihana Majetić, Filip Mirić

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ABSTRACTS

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BASIC SCIENCE

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BS01

Acinetobacter Strikes Back: Rise of Colistin Resistance

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Introduction: *Acinetobacter spp.* are strictly aerobic, Gram-negative coccobacilli. In recent years, it has emerged as an important cause of bloodstream infections in hospitalized patients, particularly in intensive care settings. Because of its remarkable ability to develop resistance to major classes of antibiotics, colistin remains a crucial last-line treatment for multidrug-resistant *Acinetobacter* infections. Therefore, the aim of this study was to assess the prevalence of *Acinetobacter spp.* and the emergence of colistin resistance.

Materials and methods: This clinical study included 38,378 blood culture samples collected at University Hospital Centre Osijek between May 2020 and December 2025. Blood samples were inoculated into blood culture bottles and incubated. Positive cultures were Gram-stained, subcultured on conventional agar media, and identified using conventional methods. Susceptibility was tested on Mueller-Hinton agar and interpreted according to European guidelines.

Results: A total of 6,636 positive blood culture bottles were detected during the study period, representing overall incidence of 17%. In 2020, *Acinetobacter* represented 17% of isolates. This peaked at 25% in 2021, then dropped to 13% in 2022. The downward trend continued, reaching 4% in 2023 and a low of 3% in 2024, before slightly increasing to 6% in 2025. *A. baumannii* was the most frequently isolated species. Regarding colistin resistance, rates were 0.7% in 2020, 0.3% in 2021, 5.7% in 2023, and as high as 7.5% in 2024, while no colistin resistance was detected in 2022 and 2025.

Conclusion: Although the overall prevalence of *Acinetobacter* isolates decreased in the later years, the increase in colistin resistance in 2023 and particularly in 2024 is concerning. Monitoring antimicrobial susceptibility is essential, as the emergence of colistin resistance significantly limits therapeutic options.

Keywords: Acinetobacter; Colistin; Microbial Sensitivity Tests

BS02

Investigation of a short PACAP fragment in ischemic retinopathy

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Introduction: Inadequate blood supply of the retina can quickly lead to visual impairment or even blindness. In clinical practice there is no truly effective, long-term, non-invasive therapy for these conditions, and their pathogenesis is not fully understood. Pituitary adenylate cyclase activating polypeptide (PACAP) is a neuropeptide with cytoprotective and anti-inflammatory effects. Among its three receptors, mostly the PAC1 receptor is responsible for the protective functions.

Materials and Methods: In our research, the effects of a short, cyclic, less known, PAC1 receptor agonist PACAP fragment was investigated in an ischemic retinopathy mouse model. In a group of CD1-IGS mice permanent unilateral common carotid artery occlusion (UCCAO) was performed. Some of the animals were treated with PACAP1-5 eyedrops twice a day (Control-Systane: n=13, Control-PACAP1-5: n=13, UCCAO-Systane: n=10, UCCAO-PACAP1-5: n=10). Optical coherence tomography (OCT) was used to determine the thickness of retinal layers on days 0, 14 and 28. After the 28 days period, retinas were isolated and immunohistochemical examinations were performed.

Results: OCT measurements show a significant degeneration of the retina ($p=0.032$) however, this difference was not observed in the PACAP1-5 treated group. Degeneration also affected the somas of the ganglion cells ($p=0.004$), but the extent of the damage was reduced when the PACAP1-5 eye drop was applied ($p=0.024$). Furthermore, the expression of calbindin by the horizontal cells was significantly lower in the UCCAO group ($p<0.01$) and this change did not appear in the PACAP1-5 treated group.

Conclusion: Based on our results, the investigated short PACAP fragment may be a promising therapeutic molecule in ischemic retinopathy.

Keywords: Ischemia; PACAP; Retina

BS03

Surprising behavioral consequences of the chemogenetic silencing of the ventral tegmental area in rats

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Introduction: In this study, we silenced the ventral tegmental area (VTA) using Designer Receptors Exclusively Activated by Designer Drugs and examined the induced, supposedly mania-like symptoms from a behavioral and pharmacological perspective. The dopaminergic theory of bipolar disorder posits state-dependent fluctuations in dopamine transmission, where (hypo)manic episodes are associated with heightened dopaminergic activity.

Materials and methods: Rats received stereotaxic injections of 500 nl adeno-associated viral vectors into the VTA, transfecting inhibitory hM4D(Gi) receptors and mCherry (n=10) or mCherry alone (n=8; controls) into neurons. 30 minutes prior to the experiments, animals were treated subcutaneously with deschloroclozapine (DCZ; 0.03/0.1/0.3 mg/bwkg) actuator or vehicle. Motivation and attention were assessed using the rodent psychomotor vigilance task (PVT). For pharmacological validation we combined the noradrenaline-dopamine reuptake inhibitor bupropion (5/15 mg/bwkg) with DCZ. High-dose DCZ was then administered for 10 days to evaluate subchronic effects of VTA modulation.

Results: In PVT, the hM4Di group performed significantly more premature trials with significant reduction in the number of missed trials, especially in higher (>5) bins, following acute DCZ treatment. These changes were further enhanced by combination treatment with bupropion. During subchronic treatment, these differences between groups diminished, however, on the post-treatment days the number of missed trials significantly increased in the hM4Di group compared to controls.

Conclusion: Acute VTA silencing resulted in hypomania-like behavior and sensitivity towards a monoaminergic drug. This apparent contradiction may reflect on predominantly GABAergic transfection and secondary dopaminergic disinhibition. Evolving tolerance during subchronic treatment and post-treatment passivity may indicate compensatory dopaminergic adaptations. These findings would also be consistent with the dopaminergic theory of bipolar disorder, and further suggests the role of VTA dysfunction in its pathogenesis.

Keywords: Bipolar Disorder; Chemogenetics; Ventral Tegmental Area

BS04

Pharmacological blockade of a neuroinflammatory pathway to relieve chronic stress-induced pain in a mouse model

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Introduction: Fibromyalgia, a primary chronic pain condition affecting a significant part of the population, is still a therapeutically challenging disease. The exact pathophysiological mechanisms remain unclear, but chronic stress is recognized as a major causative factor. Neuroinflammation seems to have an essential role in the development of stress-induced pain, where inflammasomes, as well as proinflammatory cytokines, such as interleukin-1 (IL-1), are involved. Our hypothesis was that the inhibition of the NLRP3 inflammasome pathway by MCC950 and the blockade of IL-1 receptors by anakinra can alleviate chronic stress-induced mechanical and cold hyperalgesia in a mouse model of fibromyalgia.

Materials and methods: Male C57Bl/6 mice were exposed to chronic immobilization stress for two weeks. Mechanical hyperalgesia was measured using a dynamic plantar aesthesiometer (DPA), cold hyperalgesia was assessed weekly using the hind paw withdrawal latency test. Mice received daily intraperitoneal injections of MCC950 (10 mg/kg)/anakinra (10 mg/kg) or vehicle (n=7–10/group). At the end of the experiment, open field and tail suspension tests were conducted to assess anxiety- and depression-like behaviors.

Results: MCC950 and anakinra abolished the approximately 20% stress-induced mechanical pain ($p < 0.0001$ and $p = 0.0001$). 60-70% cold hyperalgesia was not affected by MCC950 but was significantly reduced by the anakinra treatment by the second week ($p = 0.1337$ and $p = 0.0008$). No significant differences were observed between groups in anxiety- or depression-like behaviors.

Conclusion: Pharmacological management of fibromyalgia is an unmet medical need; it is essential to develop a targeted and effective therapy that addresses the pathogenesis of the disease. These preclinical findings suggest that anakinra and MCC950 may represent a promising therapeutic strategy for relieving stress-induced pain.

Keywords: Anakinra, Chronic Pain, Fibromyalgia, Inflammasomes

BS05

Comparative study of the locus coeruleus in wild-type and PACAP gene knock-out mice with aging**Marcell Schmidt¹; Dániel Pham¹; Andrea Tamás¹**

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Introduction: The neuroprotective effect of pituitary adenylate cyclase-activating polypeptide (PACAP) has been demonstrated in several Parkinson's disease models, and its deficiency is known to accelerate aging processes and increase vulnerability in PACAP knockout (KO) mice. Our previous studies described age-dependent neuronal loss and an increase in the number of microglia in the substantia nigra (SN) and ventral tegmental area (VTA) in PACAP KO mice. Our present aim was to observe age-related morphological changes in the locus coeruleus (LC), which, as part of the noradrenergic system, is also involved in the pathogenesis of Parkinson's disease.

Materials and methods: Our studies used young (4 months old) and old (1.5 years old) wild-type (WT) [n=5-4] and PACAP KO [n=8-6] mice. Noradrenergic neurons in the LC were labeled with tyrosine hydroxylase (TH), while microglia were labeled with Iba1. Microglia were classified based on morphological criteria.

Results: In 1.5-year-old PACAP KO mice, we detected a significant decrease in TH+ cell numbers with age and compared to wild-type animals of the same age. We observed a significant increase in the number of microglia with age in PACAP KO mice, which were morphologically inactive.

Conclusion: Our results suggest that in the absence of endogenous PACAP, age-related neurodegenerative processes are accelerated in the locus coeruleus. The observed differences are consistent with the changes previously observed in the SN and VTA, supporting the broad neuroprotective and immunoregulatory role of PACAP. Furthermore, the involvement of the LC suggests that in the absence of endogenous PACAP, not only the dopaminergic but also the noradrenergic system becomes more vulnerable, which supports the hypothesis that endogenous PACAP may play an important role in the mechanism of age-related neurodegeneration.

Keywords: Pituitary Adenylate Cyclase-Activating Polypeptide; Parkinson Disease; Neurodegenerative Diseases

BS06

Investigating the role of Hemokinin-1 in Thermoregulation

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Introduction: Hemokinin-1 (HK1) is a tachykinin-family neuropeptide known to exert several functions in the central nervous system. While some studies have suggested its involvement in nociceptive pathways, its potential role in thermoregulation has received limited attention. Based on its high expression in the thalamus—a key region in temperature regulation—HK1 represents a strong candidate for a regulatory role in thermal homeostasis. Therefore, our study aimed to elucidate its potential involvement in thermoregulatory mechanisms.

Materials and Methods: Experiments were conducted on adult male and female HK1 gene-deficient (HK1 KO) and wild-type (WT) mice. We compared core temperature responses of HK1 KO and WT mice following intraperitoneal administration of low (120 µg/kg) and high (5 mg/kg) doses of lipopolysaccharide (LPS). Additionally, in a polymicrobial sepsis model induced by cecal ligation and puncture (CLP), we examined body temperature, survival, and tissue cyclooxygenase-1 (COX-1) expression via PCR in HK1 KO and WT mice.

Results: Our findings show that under physiological conditions, there was no difference between HK1 KO and WT groups in response to either cold or heat exposure. However, under pathophysiological stress, i.e., systemic inflammation, we found significant differences. In the late phase of the multiphasic febrile response following low-dose LPS, the temperature rise was significantly attenuated in the HK1 KO group. Furthermore, in response to high-dose LPS, HK1 deficiency prevented the development of hypothermia.

Conclusion: These results suggest that HK1 may have both pyrogenic and cryogenic properties. Further studies are warranted to elucidate the precise mechanisms underlying its role in thermoregulation.

Keywords: Body Temperature Regulation; Fever; Hypothermia; Lipopolysaccharides

CASE REPORT

CR00

A novel FGD1 mutation in a patient with Aarskog-Scott syndrome: Expanding the mutational and phenotypic spectrum

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Introduction: Aarskog-Scott syndrome (AAS), also known as faciogenital dysplasia, is a rare X-linked disorder caused by pathogenic variants in the *FGD1* gene. It is generally characterized by short stature, hypertelorism, shawl scrotum, and brachydactyly, with wide phenotypic variability among patients. This case report presents a patient with a novel *FGD1* pathogenic variant and aims to provide new insights into the genetics of AAS.

Case report: A 12-year-old male patient born from the third pregnancy of non-consanguineous parents, a healthy father and mother, with a phenotype similar to the patient is presented. The patient was born with visible dysmorphic features, including a cleft lip and soft and hard palate. He underwent three surgical corrections and was followed by a multidisciplinary team since birth. Initially, he presented to the genetics clinic at age 3 with a cleft of both the soft and hard palate and was later referred again by the endocrinology clinic. Clinical exome sequencing was performed, and a novel c.527dup variant in the *FGD1* gene was discovered. Based on phenotypic features such as hypertelorism, flat face, mild ptosis of the right eyelid, irregular dentition, and short and broad hands and feet, the presence of a neurobehavioral disorder (ADHD), as well as a pathogenic *FGD1* variant, a diagnosis of AAS was made.

Conclusion: This case report expands the *FGD1* mutational spectrum and highlights the importance of detailed clinical evaluation in patients with suspected AAS. The novel pathogenic variant may help link phenotype to mutation and improve recognition of atypical AAS presentations.

Keywords: Aarskog Syndrome; Cleft Palate; Mutation; Phenotype

CR01

When Treatment Becomes the Trigger: Immune-Related Colitis in Lung Adenocarcinoma Treated with Immunotherapy

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Introduction: Immunotherapy is a form of oncologic treatment that harnesses the immune system in the fight against cancer. The most commonly used agents are immune checkpoint inhibitors that block programmed cell death protein 1 (PD-1) and programmed cell death ligand 1 (PD-L1). The aim of this case report is to highlight the adverse effects of immunotherapy in order to reduce morbidity in oncologic patients.

Case report: We report the case of a 68-year-old female patient who initially presented with persistent cough and unintentional weight loss over three months. Following the initial presentation, the patient underwent further evaluation after a suspicious infiltrate was detected on chest radiography, raising concern for a malignant process. As part of the bronchocytological evaluation, cytological analysis confirmed the diagnosis of poorly differentiated non-small cell lung carcinoma, consistent with adenocarcinoma. Computed tomography demonstrated metastatic involvement of the brain and the left adrenal gland. To guide treatment decisions, immunohistochemical analysis was performed on a biopsy sample, revealing 100% PD-L1 expression, after which pembrolizumab therapy was initiated. Following the 10th cycle of immunotherapy, the patient experienced diarrhea with blood streaks, tenesmus, and anorexia. Laboratory investigations showed markedly elevated fecal calprotectin (1485 µg/g), leading to colonoscopic assessment, which revealed hyperemic, edematous, friable mucosa of the rectum and sigmoid colon, accompanied by ulcerations throughout the descending colon. The conducted investigations indicated a diagnosis of ulcerative colitis, most likely induced by immunotherapy. The patient was subsequently started on infliximab, which resulted in remission of her ulcerative colitis.

Conclusion: This case demonstrates that while immunotherapy can provide significant oncologic benefit, it may also trigger severe immune-related toxicities. Recognizing and promptly managing such events is crucial to minimizing morbidity and preserving therapeutic efficacy.

Keywords: Infliximab; Immunotherapy; Pembrolizumab; Ulcerative Colitis

CR02

Epithelioid Angiosarcoma Mimicking Large-Vessel Vasculitis – A Case Report

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Introduction: Angiosarcoma is a rare, highly malignant tumor of endothelial origin with heterogeneous clinical presentation and nonspecific symptoms that may mimic systemic inflammatory and autoimmune diseases. The epithelioid variant represents a significant diagnostic challenge. The aim of this case report is to highlight diagnostic pitfalls in distinguishing immunological from malignant diseases and to emphasize the importance of a multidisciplinary approach for timely and accurate diagnosis.

Case report: We present a 70-year-old female patient hospitalized at the Department of Rheumatology and Clinical Immunology due to fever, anemia, myalgia, cholestatic liver injury, and elevated inflammatory markers, including C-reactive protein, erythrocyte sedimentation rate, leukocytosis, and thrombocytosis. Large-vessel vasculitis was suspected, and glucocorticoid therapy was initiated, resulting in clinical improvement and regression of inflammatory parameters. Extensive diagnostic work-up did not confirm vasculitis. Computed Tomography (CT) angiography of the supra-aortic branches showed no pathological changes, while CT of the thorax revealed thrombotic masses in the infrarenal segment of the abdominal aorta. Blood and urine cultures were sterile; serological testing for hepatitis B and C, HIV, antinuclear antibodies, and antineutrophil cytoplasmic antibodies was negative. Radiological evaluation for knee pain revealed osteolytic changes in the proximal tibia. Bone scintigraphy and CT suggested a tumorous process, followed by bone biopsy. Histopathological and immunohistochemical analysis confirmed epithelioid angiosarcoma. Magnetic Resonance Imaging demonstrated multiple lesions in the proximal tibia and distal femur, with skeletal dissemination and suspected pulmonary metastases.

Conclusion: This case shows that rare malignant tumors may clinically mimic immunological diseases and lead to diagnostic pitfalls. In patients with persistent systemic symptoms, thrombotic phenomena, and atypical bone lesions, it is essential to broaden the differential diagnosis.

Keywords: Angiosarcoma; Bone Neoplasms; Differential Diagnosis; Immunohistochemistry; Vasculitis

CR03

When a Mass Isn't What It Seems: A Case of Mediastinal Paraganglioma

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Introduction: Mediastinal paragangliomas are rare neuroendocrine tumors that typically present with nonspecific clinical manifestations. They are frequently associated with hereditary syndromes and germline mutations, which have important implications for prognosis and family screening. Diagnosis relies on histopathology, immunohistochemistry and molecular within a multidisciplinary approach.

Case report: A 57-year-old female presented with a six-month history of productive cough without additional symptoms. In April 2024, a thoracic multislice computed tomography revealed a 49x38x47 mm mass in the middle mediastinum. Repeated imaging in May 2025 confirmed persistence of the lesion. After multidisciplinary evaluation, surgical excision was performed. The pathological specimen consisted of multiple gray-white tissue fragments. Histopathological evaluation revealed a highly cellular tumor composed of cells forming large irregular clusters separated by fibrovascular septa. The cells had abundant eosinophilic cytoplasm, while the nuclei showed mild to moderate pleomorphism with characteristic “salt-and-pepper” chromatin. Immunohistochemically, tumor cells were positive for INSM1, CD56, chromogranin, and GATA3, and negative for epithelial and lymphoid markers. The Ki-67 proliferation index was 4.5%. These findings were consistent with paraganglioma. Given the association of paragangliomas with hereditary syndromes, next-generation sequencing was performed to assess potential underlying genetic alterations relevant for prognosis, surveillance, and familial counselling. No variants of pathogenic or likely pathogenic significance and no variants of uncertain clinical significance associated with paraganglioma were identified. The patient was included in multidisciplinary follow-up with planned radiologic monitoring, and no recurrence has been detected.

Conclusion: This case highlights the diagnostic challenges of mediastinal tumors and underscores the importance of integrating histopathology, immunohistochemistry, and molecular testing for accurate diagnosis. Multidisciplinary follow-up with radiologic monitoring is recommended to detect potential recurrence.

Keywords: Mediastinum; Neuroendocrine Tumors; Paraganglioma

CR04

VEXAS Syndrome: A Case Report

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Introduction: Vacuoles, E1 enzyme, X-linked (Xp11.3), autoinflammatory, somatic (VEXAS) syndrome is a recently described autoinflammatory disease caused by somatic mutations in UBA1. It is characterized by hematologic and inflammatory manifestations such as recurrent fever, relapsing polychondritis, and myelodysplastic syndrome. This case report's objective is to outline the clinical signs and therapies of this little-known illness.

Case report: We present a case of a 63-year-old man with multiple comorbidities admitted for relapsing polychondritis, pulmonary nodules and infiltrates, and hilar/mediastinal lymphadenopathy. With ambulatory introduced prednisone the patient was stable, however a few days before hospitalization he developed a fever with a cough and high inflammatory parameters. Pulmonary infiltrates were visible on initial chest X-ray and after giving a combined antibiotic therapy of ceftriaxone and levofloxacin, the patient became afebrile and inflammatory parameters dropped. A chest computerized tomography (CT) scan revealed intrapulmonary infiltrates, and a bronchoscopy was indicated but unremarkable. During his stay, a bone-marrow puncture was performed which showed hypercellularity with toxic change of granulocytic lineage and polymorphic megakaryocytes. While in hospital, methotrexate was introduced in addition to his chronic prednisone therapy. DNA genetic testing confirmed positive UBA1. After the release, due to worsening of symptoms, auricle pain and erythema, nose, eye, hip and knee pain a request was made for the introduction of tocilizumab into therapy.

Conclusion: A newly defined illness called VEXAS syndrome is brought on by acquired mutations in the UBA1 gene, which causes inflammatory symptoms that affect several organs. In order to provide appropriate care, further research and medical professional education are required.

Keywords: Polychondritis, Relapsing; UBA1 protein, human; VEXAS syndrome

CR05

Supraglottic stenosis as a rare complication of cicatricial mucosal pemphigoid: a case report

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Introduction: Cicatricial mucosal pemphigoid is a rare chronic autoimmune disease clinically manifested by ulcerations with a tendency to leave scars on the affected tissue. It most commonly affects the oral and ocular mucosa, while involvement of the upper respiratory tract is rare but potentially life-threatening due to the development of supraglottic stenosis and consequent respiratory failure.

Case report: A 71-year-old female patient presented with gradually progressive dyspnea and dysphagia lasting approximately one year. During the last three months, painful oral ulcerations developed and were confirmed with clinical examination. In her medical history, she denied previous upper respiratory tract diseases and exposure to radiotherapy. Two months later, clinical deterioration occurred with more pronounced dyspnea. Endoscopic examination revealed a membranous formation that significantly narrowed the supraglottic airway. Due to threatening respiratory failure, an emergency tracheotomy with tracheostomy formation and laryngomicrosurgery with excision of the membrane with a carbon dioxide laser were performed. Dermatovenereological work-up revealed the diagnosis of cicatricial mucosal pemphigoid. Three months after surgery, restenosis occurred, which led to retracheotomy. Rituximab therapy was initiated for five months postoperatively because that is autoimmune disease where B – lymphocytes create autoantibodies against structures in basal membrane of mucosa, however, repeated laryngomicrosurgery was performed 12 months after the initial intervention. Following the third dose of rituximab, the patient showed no signs of supraglottic restenosis at the last follow-up.

Conclusion: This case report highlights the importance of considering cicatricial mucosal pemphigoid in the differential diagnosis of supraglottic stenosis. Although rare, this complication can be life-threatening, and timely surgical intervention combined with biological therapy and an individualized approach is crucial for preventing restenosis and improving outcomes.

Keywords: Autoimmune Diseases; Rituximab; Tracheostomy

CR06

Pancreatic Metastasis of Renal Adenocarcinoma More Than Two Decades After Nephrectomy – A Case Report

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Introduction: Metastatic tumors of the pancreas account for 2–5% of all pancreatic neoplasms. The most common pancreatic metastases originate from renal cell carcinoma (RCC), melanomas, colorectal carcinomas, breast carcinomas, and sarcomas. Metastatic RCC relatively rarely involves the pancreas, but when surgically resected, it is associated with prolonged survival. A long latency period between nephrectomy and pancreatic metastasis is not exceptional in RCC, but it may complicate diagnosis and delay appropriate management.

Case report: A 76-year-old man with a history of right-sided nephrectomy for RCC performed more than 20 years earlier presented with obstructive jaundice. An abdominal multislice computed tomography scan was performed and revealed a 4-cm expansile mass in the pancreatic head. Endoscopic ultrasound demonstrated choledocholithiasis and a 3.5-cm hypoechoic lesion in the uncinate process. Fine-needle biopsy using a 22-gauge needle confirmed the diagnosis of metastatic RCC. Due to spontaneous passage of the stone, the planned endoscopic retrograde cholangiopancreatography was not performed. The patient was presented at a multidisciplinary oncology team meeting. Surgical resection was considered but given the patient's advanced age, significant comorbidities, and overall clinical status, stereotactic body radiation therapy was selected as the most appropriate treatment approach.

Conclusion: This case demonstrates the possibility of metastatic RCC occurring many years after nephrectomy. It also highlights stereotactic body radiation therapy as a viable treatment modality for patients who are not optimal surgical candidates due to comorbidities or tumor characteristics. Long-term follow-up remains essential in patients treated for RCC.

Keywords: Jaundice, Obstructive; Kidney Neoplasms; Neoplasm Metastasis; Nephrectomy

CR07

Targeted therapy in cholangiocarcinoma

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Introduction: Cholangiocarcinoma is a rare and aggressive malignant tumor often diagnosed at an advanced stage. Advances in molecular profiling contributed to therapeutic options, but clinical responses remain variable.

Case report: A 55-year-old woman, who presented with jaundice lasting three weeks, underwent Endoscopic Retrograde Cholangiopancreatography for choledocholithiasis with biliary bypass placement. Radiological imaging showed a hypovascular lesion measuring 20x21 mm located on the junction of IVa and VIII liver segments, later histologically confirmed as intrahepatic cholangiocarcinoma, with lymphadenopathy. Tumor markers alpha-fetoprotein and carbohydrate antigen 19-9 were elevated. The initial biopsy revealed features of active chronic hepatitis. A cytological finding that was obtained via liver endoscopic ultrasound suggested a neuroendocrine tumor, and positron emission tomography/computed tomography (PET/CT) demonstrated pathological fluorodeoxyglucose uptake in lymph nodes. Repeat biopsy confirmed intrahepatic cholangiocarcinoma, a malignant epithelial tumor of biliary origin. The tumor was staged as cT1aN1M0 (stage IIIB). Chemotherapy with gemcitabine and cisplatin was carried out and resulted in regression after three cycles. Unfortunately, PET/CT showed disease progression after the fifth cycle. Fibroblast growth factor receptor 2 (FGFR2) genetic alteration was identified through genomic profiling, leading to targeted therapy with pemigatinib. Pemigatinib is an oral selective FGFR 1-3 inhibitor used to treat carcinomas with FGFR2 alterations. Near-complete regression was achieved after five cycles of pemigatinib, but disease progression reoccurred after an additional three cycles. The multidisciplinary team decided to conduct third-line therapy with the CapeOx protocol.

Conclusion: This case underscores the diagnostic and therapeutic complexity associated with cholangiocarcinoma. Despite the identification of the FGFR2 alteration, which enabled targeted therapy and an initial treatment response, the disease failed to regress. This outcome shows the unpredictability of cholangiocarcinoma and emphasizes the need for new therapeutic options.

Keywords: Cholangiocarcinoma; Molecular Targeted Therapy; Receptor, Fibroblast Growth Factor, Type 2

CR08

Brachial artery embolism in a patient with metastatic TNBC and bone marrow infiltration**Luka Balog¹; Lovro Balen¹; Mislav Blažević¹; Alen Beneš¹; Tajana Silovski^{1,2}***1 - School of Medicine, University of Zagreb, Zagreb, Croatia**2 - University Hospital Centre Zagreb, Department of Oncology, Zagreb, Croatia*

Introduction: Triple negative breast cancer (TNBC) is the most aggressive subtype of breast cancer with the poorest outcomes. In metastatic setting, the course of disease is often accompanied by clinical complications including hypercoagulable or prothrombotic state explained by chronic inflammation, cytokine production and platelet activation.

Case report: A 53-year-old woman was diagnosed with TNBC and treated with neoadjuvant chemotherapy, followed by surgery and adjuvant radiotherapy. One year later, disease progression occurred with metastases in retro-orbital space, mediastinum, and bones. After a year of stable metastatic disease controlled by targeted therapy talazoparib (given for her BRCA positive status) – due to disease progression, further therapy was continued with antibody-drug conjugate sacituzumab-govitecan. During which, the patient developed pulmonary embolism and Low Molecular Weight Heparin therapy was started. A month later, bone marrow infiltration was cytologically confirmed. Laboratory results showed pancytopenia requiring discontinuation of anticoagulation therapy. A week later, the patient experienced sharp pain and tingling in the right upper arm. A Computed tomography-angiography revealed an occlusion extending from the right axillary artery into the brachial artery, measuring 8.5 cm in length. Platelets were measured at only $20 \times 10^9/L$. A right brachial artery embolectomy was performed, during which the patient received 12 units of platelet concentrate. Anticoagulation with Unfractionated Heparin was introduced, with close monitoring of platelet counts and Activated Partial Thromboplastin Time. The systemic anticancer treatment was also continued.

Conclusion: This case highlights the range of complications that can arise during treatment of metastatic disease, including pulmonary embolism and critical limb ischemia despite profound thrombocytopenia. Continuing effective antineoplastic therapy, despite thrombotic events and cytopenias, remains the primary clinical goal.

Keywords: Breast Neoplasms; Thrombocytopenia; Embolectomy

CR09

The Role of Comprehensive Genomic Profiling on Circulating Tumour DNA in the Management of Metastatic Breast Cancer

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Introduction: As the most commonly diagnosed malignancy worldwide and leading cause of cancer-related mortality among females, breast cancer represents a significant public health issue. This case report illustrates how genomic profiling enables personalized treatment of breast cancer and shows that, even in metastatic disease, therapeutic advances can nowadays allow long-term survival.

Case report: A 52-year-old woman was diagnosed with left-sided breast carcinoma in 2005 and underwent breast-conserving surgery followed by adjuvant chemotherapy and radiotherapy. In 2006, bilateral oophorectomy was performed due to ovarian metastases. Additionally, extensive cytoreductive abdominal surgeries were undertaken after the detection of peritoneal and retroperitoneal metastases in 2017. Histopathological examination of metastatic lesions confirmed oestrogen receptor-positive and HER2-negative disease with a high Ki-67 proliferation index (35%). Consequently, fulvestrant monotherapy was implemented in 2018, but further progression led to addition of palbociclib. Long-term disease control was achieved with paclitaxel between 2021 and 2024, receiving 53 cycles in total. Given the newly diagnosed cases of early-onset breast cancer among two close relatives, one diagnosed at the age of 52 and the other in their forties, comprehensive circulating tumour DNA (ctDNA) sequencing was conducted and revealed a BRCA1 exon 4-6 deletion as the only actionable mutation. This finding qualified the patient for treatment with a poly (ADP-ribose) polymerase (PARP) inhibitor and talazoparib was initiated in December 2025 with immediate biochemical regression. Germline multigene panel testing confirmed a hereditary BRCA1 mutation, leading to cascade genetic testing of family members.

Conclusion: Accurate molecular characterization of metastatic breast cancer guides optimal treatment selection and improved outcomes. This case report demonstrates that comprehensive ctDNA genomic profiling identifies actionable tumour-specific alterations and plays a key role in tailoring personalized therapy.

Keywords: Breast Neoplasms; Genes, BRCA1; Genetic Testing

CR10

Foodborne Pathogen: A Fatal Case of *Listeria monocytogenes* Meningoencephalitis in an Immunocompromised Adult

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Introduction: *Listeria monocytogenes* meningoencephalitis (LMM) is a central nervous system infection caused by the facultative intracellular bacterium *Listeria monocytogenes*, which is transmitted through consumption of contaminated food or from mother to fetus. Although somewhat rare, it progresses rapidly and is often fatal, especially in high-risk groups, including pregnant women, infants, the elderly, and immunocompromised individuals.

Case report: A 64-year-old immunocompromised woman, with multiple myeloma and other comorbidities, was examined on the third day of illness. Her symptoms included fever, altered mental status, disorientation, general weakness, headache, loss of appetite, and acute diarrhea. Central nervous system infection was suspected, and empiric antimicrobial therapy, including ampicillin, was initiated. Laboratory findings showed leukopenia (white blood cells $3.2 \times 10^9/L$), thrombocytopenia (platelets $105 \times 10^9/L$), elevated C-reactive protein (135 mg/L), and mildly increased procalcitonin (0.33 µg/L). Cerebrospinal fluid analysis revealed gram-positive *Listeria monocytogenes* rods with pleocytosis dominated by polymorphonuclear cells ($592 \times 10^6/L$), proteinorachia (1.737 g/L), hypoglycorrachia (0.88 mmol/L), and high lactate (10.22 mmol/L). Computed tomography of the brain identified a zone of acute ischemia in the left cerebellum without signs of hemorrhage. During hospitalization, the patient's neurological condition worsened rapidly, with generalized seizures associated with severe hypokalemia and metabolic acidosis. Despite receiving targeted antimicrobial therapy and supportive measures, the patient passed away on the 5th day after admission.

Conclusion: This case highlights the importance of early recognition and treatment of LMM to prevent serious neurological complications and death. Although appropriate empiric therapy was initiated immediately upon hospital admission, the delay from symptom onset could have contributed to the fatal outcome in this patient.

Keywords: Gram-Positive Rods; Infectious Encephalitis; *Listeria Monocytogenes*

CR11

Tuberculous Meningitis in an Immunocompetent Patient from a High-Burden Tuberculosis Setting: A Case Report

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Introduction: Tuberculous meningitis is a rare, but severe manifestation of *Mycobacterium tuberculosis* infection, associated with high mortality and risk of permanent neurological sequelae. Clinical presentation is often nonspecific, resulting in delayed diagnosis and treatment.

Case report: We present the case of 36-year-old immunocompetent man from the Philippines, with no known comorbidities, who presented with a one-month history of progressive headaches, low-grade fever, respiratory symptoms and significant weight loss (15 kg over 3 months). Despite an initially unremarkable neurological examination and normal early neuroimaging, his clinical condition deteriorated with the development of meningeal signs, nausea, vomiting, and altered mental status. Cerebrospinal fluid (CSF) analysis revealed marked pleocytosis (813 x 10⁶/L) with neutrophilic predominance (66%), markedly elevated protein levels (2.96 g/L), decreased glucose concentration (2.2 mmol/L), and increased lactate levels (6.69 mmol/L). Extensive microbiological testing of the CSF was negative for common bacterial, viral, and fungal pathogens. The diagnosis of tuberculous meningitis was established by detection of *Mycobacterium tuberculosis* DNA in the CSF using real-time polymerase chain reaction. Contrast-enhanced brain magnetic resonance imaging demonstrated numerous small ring-enhancing lesions up to 5 mm in diameter in both supra- and infratentorial regions, accompanied by diffuse leptomeningeal enhancements. Following the diagnostic confirmation, standard four-drug antituberculosis therapy was initiated along with adjunctive corticosteroids, anti-edematous, and supportive treatment. At the time of writing, the patient is still undergoing treatment; partial to full recovery is expected.

Conclusion: Timely application of molecular diagnostics enabled rapid and accurate pathogen identification despite initially negative conventional microbiological results. Tuberculosis should be considered in the differential diagnosis of central nervous system infections, particularly in patients from high-prevalence countries.

Keywords: Polymerase Chain Reaction; Tuberculosis, Central Nervous System; Tuberculosis, Meningeal

CR12

Balanced Robertsonian Translocation in Childhood: Silent Phenotype, Significant Consequences

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Introduction: Robertsonian translocations represent a frequent type of structural chromosomal rearrangement in the general population, with an estimated incidence of approximately 1 in 1,000. Although often clinically silent, their early identification is important due to potential long-term reproductive implications. This case report aims to highlight the value of early diagnosis and raise awareness of a relatively common chromosomal rearrangement that may otherwise remain unrecognized.

Case report: We present the case of a 9-year-old boy referred to the genetics outpatient clinic for assessment of bilaterally small, intermittently painful testes. A pediatric surgeon and endocrinologist initially evaluated the patient. Physical examination revealed prepubertal genitalia, with both testes in the scrotum but reduced in size, right testicle 1,3 x 0,7 cm and left 1,4 x 0,7 cm, without dysmorphic features or neurological deficits. Growth and psychomotor development were normal. Testicular ultrasound demonstrated bilaterally small testes with homogeneous parenchyma and normal vascularization. Endocrine evaluation showed gonadotropin and testosterone levels consistent with prepubertal values. Karyotype analysis was initially performed to exclude Klinefelter syndrome, leading to the incidental detection of a 45, XY, der(13;14)(q10;q10) karyotype, consistent with an apparently balanced Robertsonian translocation between chromosomes 13 and 14. Family history was negative for relevant genetic disorders; however, the mother reported two previous spontaneous miscarriages, raising the possibility of an inherited chromosomal rearrangement. No congenital anomalies or developmental comorbidities were identified. Patient remains under follow-up with pediatric endocrinology and surgery.

Conclusion: This case illustrates that balanced Robertsonian translocation 45, XY, der(13;14)(q10;q10) may present with subtle clinical signs in childhood while remaining endocrinologically silent. Despite normal development, the finding has important reproductive implications for both male and female carriers, underscoring the need for long-term follow-up and genetic counseling.

Keywords: Genetic Counselling; Karyotype; Testis

CR13

Distant metastases in silent prostate adenocarcinoma: importance of early detection**Ivan Barna¹; Anamarija Bećarević¹; Ema Purgar¹; Martina Britvar²; Irena Zagorac^{1,2,3}***1 - Faculty of Medicine, University of Osijek, Osijek, Croatia**2 - Department of Pathology and Forensic Medicine, University Hospital Osijek, Osijek, Croatia**3 - Faculty of Dental Medicine and Health, University of Osijek, Osijek, Croatia*

Introduction: Prostate adenocarcinoma is a common malignancy in men and a significant source of metastases when unrecognized in time, commonly involving pelvic lymph nodes and the axial skeleton. This case aims to highlight the rare atypical distant metastases as a first sign of carcinoma in patients presenting with mild symptoms of prostatic enlargement.

Case report: We present a case of a 70 year old man, who came to the hospital with a progressively growing, swelling mass of approximately 10 cm on the left side of the neck. Patient reported intermittent pain around the region and unintentional weight loss. Apart from increased urinary frequency, he had no other complaints and had a previously known enlarged prostate, for which he was taking *Serenoa repens* extract. RTG scan showed a spherical soft tissue mass paravertebrally on the left side. Cytological analysis of the mass confirmed a poorly differentiated malignancy. Subsequent biopsy confirmed the metastatic adenocarcinoma, and provided following immunohistochemical markers: positive CKAE1/AE3, ARiPSA, indicating a prostatic origin, while negative markers for other primary sites – including gastrointestinal, lung, and thyroid origins – excluded other malignancies. Further radiological assessment determined additional metastases in levels IV, Va and Vb of cervical lymph nodes, in lower thoracic vertebrae and enlarged lymph nodes along the lesser gastric curvature. Prostate lab results showed highly elevated PSA, above 1500 ng/ml, and prostate biopsy confirmed the suspicion of adenocarcinoma.

Conclusion: Prostate adenocarcinoma can lead to silent development of metastases in areas distant from the primary malignancy even in cases with minimal urinary symptoms pointing to prostate malignancy. A simple PSA lab test could have pointed to existence of said malignancy before it reached its metastatic potential.

Keywords: Prostatic Neoplasms; Adenocarcinoma; Neoplasm Metastasis

CR14

Starry-sky under the microscope: a rare sporadic Burkitt lymphoma in an adult woman

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Introduction: Burkitt lymphoma is an aggressive B-cell non-Hodgkin lymphoma (NHL). Typically, it's Epstein-Barr virus (EBV) associated and endemic to African children or immunodeficiency-related. Rarely, it presents as the sporadic type in Western countries, accounting for 50% of pediatric and only 2% of adult lymphomas, with an incidence of 2.5 per one million adults. Abdomen is the typical primary site, and *MYC* oncogene translocation is characteristic.

Case report: A 34-year-old woman presented with 15 days of flu-like symptoms, diarrhoea and a mass in the lower right quadrant. Her past medical history was unremarkable. An abdominal ultrasound revealed ileocecal invagination, and emergency ileocecal resection was performed. Gross examination showed an exophytic tumor within the cecum, 6 cm in the largest diameter. On the microscopic evaluation, the tumor was composed of medium-sized atypical lymphoid cells and scattered tingible-body macrophages, creating the classic starry-sky pattern. Immunohistochemistry indicated a high-grade B-cell NHL (CD20+, Pax5+, Bcl6+, Bcl2-, Cyclin D1-, CD5-, Ki-67 100%). In situ hybridization was negative for EBV. Further immunohistochemistry showed CD10 and c-MYC positivity, while CD30, CD138 and TdT were negative. These findings corresponded to sporadic Burkitt lymphoma. Finally, from a tumor tissue biopsy, the *MYC* translocation was confirmed using the CytoCell *MYC* fluorescence in situ hybridisation probe. Moreover, serology ruled out human immunodeficiency virus infection. She responded well to the chemoimmunotherapy GMALL 2002 protocol and a positron emission tomography-computed tomography scan from September 2025 showed a complete metabolic response to therapy.

Conclusion: Sporadic Burkitt lymphoma is rare, especially in adults, but highly aggressive. Prompt histopathological differentiation from other high-grade B-cell NHLs ensures patients receive the correct treatment protocol, to which they can respond well if started promptly.

Keywords: Burkitt Lymphoma; Genes, myc; Immunohistochemistry

CR15

Mandibular Osteosarcoma: Rare Jaw Tumor and Diagnostic Challenge

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Introduction: Osteosarcoma is a malignant bone tumor that usually affects long bones. Jaw involvement is uncommon, with an incidence of 1 in 1.5 million people per year. Mandibular osteosarcomas often present with non-specific clinical and radiological features, which can delay diagnosis, as in this case report.

Case report: A 31-year-old woman presented to the emergency clinic with a three-week history of a progressively enlarging, firm swelling on the left side of the lower jaw. Her medical history included Hashimoto's thyroiditis, arterial hypertension, and allergic rhinitis. Orthopantomography demonstrated no definitive pathological changes. Clinical examination identified a tender induration in the left lower vestibule, lateral to the edentulous alveolar ridge, with normal-appearing mucosa and no evidence of infection. Cytological aspiration was performed, and the result was inconclusive; however, mesenchymal cells with discrete atypia were observed, necessitating further investigation. The lesion continued to increase in size, and the patient was presented to a multidisciplinary team. Following, computed tomography was ordered and revealed a lytic lesion of the left mandibular body, periosteal reaction, and soft tissue extension. Incisional biopsy of the soft tissue, periosteum, and mandibular bone demonstrated poorly differentiated tumor tissue with pleomorphic osteoblasts producing osteoid. The tumor cells were immunohistochemically positive for Vimentin and Special AT-rich-sequence-binding protein 2 (SATB2) and negative for epithelial markers, thus confirming the diagnosis of mandibular osteosarcoma. The patient underwent partial mandibulectomy with reconstruction. The postoperative course was uneventful, and psychological support is being provided.

Conclusion: Early diagnosis and multidisciplinary approach are crucial in osteosarcoma management, especially when affecting uncommon sites, as it can be easily overlooked. This case highlights the importance of a multidisciplinary team approach to ensure optimal patient outcomes.

Keywords: Jaw; Neoplasms; Osteosarcoma

CR16

Rare Case of Cerebral Venous Sinus Thrombosis Associated With Epidural Analgesia in the Early Postpartum Period

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Introduction: Cerebral venous sinus thrombosis (CVST) as a postpartum complication following epidural analgesia is extremely rare and may be overlooked because symptoms often resemble benign post-dural puncture headache. Risk for this complication potentially increases due to physiological hypercoagulability, dehydration, and peripartum procedures.

Case report: We present a previously healthy 26-year-old woman who developed a severe headache the second day after an uncomplicated vaginal delivery with epidural anesthesia. The headache was constant, pulsating, temporal and bifrontal and was aggravated by sitting and coughing. It was also accompanied by nausea, vomiting, neck stiffness, shoulder pain, and bilateral tinnitus. The patient denied visual disturbances, seizures or focal deficits. Neurological examination was normal. As initial symptoms resembled post-puncture headache, she was first treated with analgesics and caffeine, but without relief. Worsening symptoms required imaging. Non-contrast Computed Tomography (CT) revealed a hyperdense tubular structure near the posterior falx. CT venography demonstrated a 17-mm filling defect in the mid-superior sagittal sinus with partial cortical vein thrombosis and additional involvement of the occipital sinus. Magnetic Resonance Imaging (MRI) with venography confirmed CVST without ischemia or hemorrhage. Hematologic evaluation for thrombophilia and antiphospholipid syndrome tested negative, ruling out hypercoagulability. She was treated with full-dose low-molecular-weight heparin, resulting in gradual improvement. Follow-up MR venography showed complete thrombus resolution. She was discharged neurologically intact and without symptoms.

Conclusion: This case highlights a rare but important complication of CVST occurring shortly after epidural anesthesia in the postpartum period. Severe or atypical post-puncture headaches should prompt urgent imaging because early anticoagulation enable full recovery.

Keywords: Analgesia, Epidural; Headache; Postpartum Period; Sinus Thrombosis, Intracranial

CR17

Combination of cyclin-dependent kinase 4/6 inhibitors and aromatase inhibitors in treatment of metastatic breast cancer**Katarina Benaković¹; Lucija Gotal¹; Josipa Flam^{1, 2}***1 - Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Oncology, University Hospital Center Osijek, Osijek, Croatia*

Introduction: Breast cancer is the most frequently diagnosed malignancy in woman worldwide. Approximately 70% of breast cancers are hormone-receptor positive and human epidermal growth factor receptor 2 (HER2)-negative. This case presents a treatment of advanced breast carcinoma that consists of endocrine therapy combined with a cyclin-dependent kinase 4/6 (CDK4/6) inhibitor.

Case report: A 31-year-old premenopausal woman was admitted to the emergency department due to a femur fracture. She was admitted to the orthopedic department and underwent hip arthroplasty. Histopathological analysis of the bone sample confirmed metastatic breast carcinoma with estrogen receptor positivity in 100% tumor cells and progesterone receptor in 95% of tumor cells. HER2 was negative, and Ki-67 was 65%. Blood tests revealed mild anemia (hemoglobin 118 g/L), elevated alkaline phosphatase (1,256 U/L), and elevated tumor markers, with CEA at 90 ng/mL and CA 15-3 at 456 U/mL. Ultrasound revealed a 32 mm mass in the right breast, accompanied by positive axillary lymphadenopathy. Computed tomography of the thorax, abdomen and pelvis demonstrated multiple osteolytic lesions throughout the skeletal system. The treatment included ribociclib, letrozole, and zoledronate. The alkaline phosphatase level declined to 852 U/L, and 50% decrease in CA 15-3 was observed. The patient developed liver metastases 31 months after initiation of therapy and was treated with paclitaxel chemotherapy. 13 months later, brain metastases were detected and radiotherapy was administered.

Conclusion: Combination of endocrine-based therapy and CDK4/6 inhibitors led to a positive treatment outcome by delaying the onset of metastatic disease. Positive therapeutic effect highlights the importance of continued advancement of endocrine therapy in breast cancer treatment.

Keywords: Alkaline Phosphatase; Carcinoma; Letrozole; Lymphadenopathy

CR18

Amikacin as a treatment for multidrug-resistant *Pseudomonas aeruginosa* keratitis: A case report

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Introduction: *Pseudomonas aeruginosa* is a ubiquitous Gram-negative bacterium. It is one of the leading pathogens associated with bacterial keratitis, particularly among contact lens wearers. If left untreated, keratitis can rapidly progress, leading to corneal perforation and serious complications such as endophthalmitis. Ocular samples typically demonstrate low rates of antibiotic resistance. However, there are increasing reports of multidrug-resistant (MDR) *P. aeruginosa* keratitis cases associated with poor prognosis.

Case report: A 73-year-old female patient presented with a three-day history of a burning sensation, pain, and redness in her right eye. Her best-corrected visual acuity was hand motion in the right eye and 12/20 in the left eye. Slit-lamp examination of the right eye showed corneal melting, a large central corneal ulcer, and hypopyon. The patient had no known risk factors. She was started on systemic and topical antibiotics. No corticosteroid therapy was administered. Conjunctival swab cultures grew a multidrug-resistant *Pseudomonas aeruginosa* strain sensitive only to amikacin. The hospital pharmacy prepared topical amikacin (50 mg/mL) for off-label use, as commercial eye drops were unavailable. Following topical amikacin treatment, corneal melting was halted, the ulcer re-epithelialized, the hypopyon fully resolved, and corneal perforation was prevented. Due to severe inflammation, the patient developed a mature cataract. Cataract surgery is planned in the future to help restore some vision in the affected eye.

Conclusion: Although topical fluoroquinolones are an established treatment for suspected bacterial keratitis, the emergence of MDR *P. aeruginosa* necessitates heightened clinical vigilance and may require alternative therapeutic strategies to optimize patient outcomes. When certain antibiotics are not commercially available, compounded formulations are required to meet the specific therapeutic needs of individual patients.

Keywords: Amikacin; Keratitis; *Pseudomonas aeruginosa*

CR19

Peroral Endoscopic Myotomy for Recurrent Achalasia in a Middle-Aged Male

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Introduction: Achalasia is a rare disorder of esophageal motility, characterized by the inability of the lower esophageal sphincter to relax and the loss of coordinated esophageal peristalsis. This condition presents with slowly progressive dysphagia affecting both solids and liquids, often accompanied by regurgitation, vomiting, and weight loss. Because symptoms develop gradually and can be nonspecific, establishing a diagnosis requires a combination of clinical evaluation, endoscopy, imaging, and confirmation with high-resolution esophageal manometry. The main goals of treatment are to relieve the functional obstruction at the gastroesophageal junction and improve quality of life, with options including pneumatic balloon dilatation and peroral endoscopic myotomy (POEM).

Case report: We present the case of a 43-year-old man with a 10-year history of worsening dysphagia and recurrent vomiting. His symptoms began insidiously and progressed over time, with frequent regurgitation of undigested food and difficulty swallowing both solids and liquids. The initial evaluation included an esophagogastroduodenoscopy, which ruled out mechanical obstruction and malignancy. An esophagram revealed esophageal dilation with delayed emptying, consistent with a motility disorder, and multislice computed tomography of the thorax and abdomen excluded secondary or extrinsic causes. Esophageal manometry ultimately confirmed the diagnosis of achalasia. Six years earlier, the patient underwent pneumatic balloon dilatation, which initially provided significant relief. However, his symptoms gradually returned, prompting re-presentation with worsening dysphagia and vomiting. Given the relapse, he underwent peroral endoscopic myotomy. The patient achieved complete symptom resolution, normal diet tolerance, weight gain, and improved quality of life.

Conclusion: This case illustrates the relapsing nature of achalasia and the limitations of balloon dilatation for long-term control, while highlighting POEM as a safe and effective minimally invasive option for sustained symptom relief and improved long-term outcomes.

Keywords: Esophageal Achalasia, Manometry, Myotomy

CR20

Rheumatoid meningitis as a rare cause of aseptic meningitis in patients with rheumatoid arthritis

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Introduction: Rheumatoid meningitis is a rare and potentially life-threatening extra-articular manifestation of rheumatoid arthritis. It often presents clinically with nonspecific symptoms and mimics infectious meningitis. For this reason, it represents a diagnostic challenge. Timely recognition is crucial for appropriate treatment and a positive outcome.

Case report: A forty-six-year-old man with a long-standing history of known rheumatoid arthritis, on biological therapy (adalimumab), was admitted to the hospital for examination due to fever and headache and underwent an initial diagnostic work-up resulting in a diagnosis of aseptic meningitis. The first symptoms were detected in December 2023, but there was no noticeable clinical benefit from the antimicrobial therapy. Increased use of corticosteroids led to clinical improvement. Cerebrospinal fluid analysis showed lymphocytic pleocytosis despite the absence of abnormalities in microbiology. Meningeal changes appeared in the form of pachymeningitis on the initial brain magnetic resonance imaging, and follow-up imaging showed regression. The infectious cause was not confirmed after extensive infectious and immunological assessment. Interleukin-6 was elevated in the cerebrospinal fluid, while serum interleukin-6 was within normal limits. This phenomenon occurs because the blood-brain barrier restricts the systemic distribution of locally produced cytokines. Corticosteroid therapy began promptly with a good clinical response in anticipation of suspected rheumatoid meningitis. The patient had no neurological symptoms prior to discharge.

Conclusion: The case emphasizes the need to consider rheumatoid meningitis in the differential diagnosis of aseptic meningitis in patients with rheumatoid arthritis. Elevated interleukin-6 in the cerebrospinal fluid, negative microbiological findings, and a good response to corticosteroids support the diagnosis. Early diagnosis leads to timely treatment with favorable outcomes.

Keywords: Arthritis; Interleukin-6; Meningitis, Aseptic

CR21

Case Report: Prophylactic Immunoglobulin Therapy in Pregnancy After Parental Human Platelet Antigen Genotyping

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Introduction: Fetal and neonatal alloimmune thrombocytopenia (FNAIT) is an immune-mediated condition resulting from transplacental passage of maternal IgG antibodies directed against fetal platelet antigens, most commonly human platelet antigen-1a (HPA-1a). The disorder frequently remains unrecognized during pregnancy, as maternal symptoms are absent, and clinical manifestations typically become evident only after birth, when affected neonates may present with thrombocytopenia or bleeding. This case report presents the antenatal management and outcome of a pregnancy complicated by maternal HPA-1a alloimmunization.

Case report: The patient was followed during her third pregnancy because of a documented history of neonatal alloimmune thrombocytopenia. Her first pregnancy proceeded without complications. In the second pregnancy, the neonate was diagnosed postnatally with HPA-1a-associated alloimmune thrombocytopenia following comprehensive evaluation, including serological testing and HPA genotyping. Treatment consisted of platelet transfusions combined with a three-day course of intravenous immunoglobulin (IVIG), leading to clinical stabilization and recovery. During the third gestation, maternal immunological screening performed at 20 weeks of pregnancy revealed the presence of HPA-1a-specific IgG antibodies, indicating maternal alloimmunization. Given the adverse neonatal outcome in the previous pregnancy, prophylactic IVIG therapy was initiated at 24 weeks of gestation to reduce the risk of fetal platelet destruction. The remainder of the pregnancy was monitored closely and progressed without maternal or fetal complications. Delivery occurred at term by cesarean section, and the neonate showed normal platelet counts with no signs of hemorrhage.

Conclusion: This case underscores the importance of recognizing maternal alloimmunization in pregnancies at risk for FNAIT. Early antibody detection and parental HPA genotyping allow timely initiation of prophylactic IVIG, crucial for preventing immune-mediated fetal and neonatal complications.

Keywords: Immunoglobulins; Pregnancy; Thrombocytopenia, Neonatal Alloimmune

CR22

Intracardiac Metastatic Melanoma Mimicking a Benign Left Ventricular Mass

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Introduction: Intracardiac metastases involving the left ventricle are rare and may radiologically mimic benign cardiac lesions. The aim of this case report is to highlight the limitations of imaging and the key role of histopathology and immunohistochemistry in guiding patient management.

Case report: A 52-year-old male with alcohol-related liver cirrhosis undergoing pre-transplant evaluation was incidentally found to have an intracavitary, pedunculated mass within the inferoapical region of the left ventricle. Further evaluation with cardiac computed tomography and magnetic resonance imaging suggested a differential diagnosis of left ventricular myxoma versus intracardiac thrombus. Urgent surgical intervention was indicated and the mass was excised via median sternotomy under cardiopulmonary bypass using a transatrial approach through the mitral valve. Histopathological examination revealed a tumor composed of atypical cells showing marked nuclear pleomorphism, prominent nucleoli, and increased mitotic activity. Immunohistochemical analysis demonstrated strong and diffuse positivity for S100, SOX10, HMB45, MITF, PRAME and Melan-A, confirming melanocytic lineage and establishing the diagnosis of metastatic melanoma. Molecular analysis identified BRAF V600 mutation, allowing consideration of targeted therapy with BRAF inhibitors in combination with mitogen-activated protein kinase inhibitors. Subsequent oncological workup was initiated to identify the primary tumor site, and the patient is currently awaiting positron emission tomography/computed tomography.

Conclusion: Intracardiac metastatic disease may represent the first manifestation of an occult malignancy and should be considered in the differential diagnosis of cardiac masses. Surgical excision enables comprehensive histopathological and immunohistochemical analysis, facilitating accurate diagnosis and subsequent molecular characterization for appropriate oncological treatment selection.

Keywords: Heart Neoplasms; Melanoma; Neoplasm Metastasis

CR23

Giant Cell Myocarditis Masquerading as MINOCA: A Diagnostic Pitfall

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Introduction: Giant cell myocarditis (GCM) is a fulminant inflammatory cardiomyopathy, representing less than 1% of myocarditis cases, characterized by rapidly progressive heart failure, malignant ventricular arrhythmias, and high mortality despite therapy. Due to its nonspecific clinical presentation, GCM may initially mimic myocardial infarction with non-obstructive coronary arteries (MINOCA), thereby delaying definitive diagnosis.

Case report: A 58-year-old man with arterial hypertension, prior transient ischemic attack, and Hashimoto thyroiditis presented with several months of exertional chest pain, with acute worsening prior to admission. Laboratory tests revealed markedly elevated hs-troponin (>50,000 ng/L) and NT-proBNP (11,853 ng/L). Electrocardiography showed right bundle branch block and inferior Q-waves. Transthoracic echocardiography revealed moderately reduced left ventricular ejection fraction (40%) with rapid functional deterioration. Cardiac magnetic resonance imaging fulfilled Lake Louise criteria for acute myocarditis, demonstrating severe biventricular dysfunction and subepicardial late gadolinium enhancement. Histological examination of endomyocardial biopsy samples revealed focal myocytolysis with a dense inflammatory infiltrate composed predominantly of CD68+ macrophages and CD3+ T lymphocytes, with occasional eosinophils. Scattered multinucleated giant cells were identified, without granuloma formation. *In situ* hybridization for Epstein–Barr virus-encoded RNA was negative, excluding Epstein–Barr virus-associated myocarditis and confirming the diagnosis of GCM. Despite guideline-directed therapy, intravenous immunoglobulins, and high-dose corticosteroids, the patient developed cardiogenic shock with recurrent sustained ventricular tachycardia. Ultimately, a durable biventricular HeartMate 3™ (Abbott) device was implanted as a bridge to transplantation, and the patient remains on the transplant waiting list.

Conclusion: Giant cell myocarditis should be suspected in MINOCA patients with rapidly progressive biventricular dysfunction and malignant ventricular arrhythmias. Early endomyocardial biopsy is essential for definitive diagnosis, enabling timely immunosuppression and guiding life-saving mechanical circulatory support.

Keywords: Cardiomyopathies; Heart-Assist Devices; Myocarditis

CR24

Management Challenges of Multivessel Coronary Disease in Advanced Age

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Introduction: Multivessel disease (MVD) is defined as presence of hemodynamically significant stenoses in two or more major coronary arteries. In elderly patients, treatment strategies are often constrained by comorbidities, recurrent ischemic events and declining left ventricular systolic function (LVSF).

Case report: We present 84-year-old male patient admitted due to progressive dyspnea in supine position, anginal chest pain on minimal exertion and recurrent episodes of stenocardia. Past medical history was significant for recurrent transient ischemic attacks, arterial hypertension, dyslipidemia and chronic kidney disease. Patient had been previously hospitalized for non-ST-elevation myocardial infarctions (05/2025, 09/2025, 10/2025), with documented progressive deterioration of LVSF after each event. On multiple occasions, invasive treatment options were proposed, but patient declined them. During hospitalization, cardiological work-up was performed. Echocardiography confirmed further deterioration of LVSF with ejection fraction of 30%. Coronarography showed no significant change compared to previous examinations, confirming MVD. Following multidisciplinary team discussion, patient was deemed unsuitable for surgical revascularization due to his condition. High-risk percutaneous coronary intervention (PCI) supported by mechanical circulatory assistance with Impella CP was proposed in tertiary-care center, which patient accepted. After transfer to tertiary center, patient experienced cardiorespiratory arrest with return of spontaneous circulation. PCI with Impella CP support was angiographically successful. However, clinical condition progressively worsened with development of sepsis and multiorgan dysfunction. Patient was transferred back to primary institution, where he succumbed to illness shortly thereafter.

Conclusion: This case raises question of whether earlier acceptance of surgical revascularization could have altered clinical outcome. It highlights complexity of decision-making in very elderly patients with advanced MVD, where potential benefits of invasive treatment must be weighed against procedural risk, comorbidities and patient preferences.

Keywords: Heart Failure; Myocardial Infarction; Multiple Organ Failure

CR25

Overlap Syndrome of Psoriatic and Rheumatoid Arthritis Successfully Treated with Janus Kinase Inhibitors: A Case Report

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Introduction: Overlap syndromes, in rheumatology, refer to coexistence of clinical, serological, and/or radiological features of two or more systemic rheumatic diseases in the same patient. Psoriatic arthritis (PsA) and rheumatoid arthritis (RA) share clinical features but differ in immunopathogenesis and optimal targeted therapy.

Case report: A 61-year-old woman with a long standing plaque psoriasis presented with peripheral joint pain, morning stiffness, nail changes, enthesitis, dactylitis and active psoriatic plaques. At disease onset, serological testing showed negative rheumatoid factor (RF) and anti-citrullinated peptide antibodies (ACPA), supporting the diagnosis of PsA. After initial treatment with conventional disease modifying antirheumatic drugs, patient was treated over 7 years with tumor necrosis factor- α inhibitor and interleukin (IL) -17 inhibitor to which she developed secondary failure and recently with IL-23 inhibitor which showed primary failure. Over time, clinical presentation evolved, with development of symmetrical polyarthritis of the small joints of the hands, mainly metacarpophalangeal joints, ulnar deviation, and persistent high disease activity. Serological re-evaluation then revealed newly positive RF and ACPA, leading to diagnosis of a PsA-RA overlap syndrome. This resulted in a more precise therapy, janus kinase inhibitors, “covering” both of these diseases pathogenic pathways which brought significant clinical improvement.

Conclusion: Overlap syndromes represent a significant diagnostic and therapeutic challenge. This case highlights the importance of continuous diagnostic reassessment in patients with inflammatory arthritis which then allows prescription of a more precise and effective therapies. However, further research into disease pathogenesis is essential to recognize predictive factors which could help us choose more targeted therapy and avoid primary or secondary treatment failure.

Keywords: Arthritis, Psoriatic; Arthritis, Rheumatoid; Janus Kinase Inhibitors

CR26

Long-Term Outpatient Management of Bipolar Affective Disorder: A Case Report

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Introduction: Bipolar affective disorder is a chronic psychiatric condition characterized by recurrent episodes of mood elevation and depression, often requiring long-term treatment and follow-up. Regular outpatient monitoring enables early recognition of symptom changes and timely adjustment of therapy, thereby reducing the risk of relapse and hospitalization. The aim of this report is to describe the long-term outpatient management of a patient with bipolar affective disorder and to emphasize the importance of continuity of care.

Case report: A middle-aged man, a professional artist, married and father of one, with bipolar affective disorder (ICD-10: F31) diagnosed in 2021 and a history of psychiatric hospitalization that year, presented in March 2024 with worsening symptoms, including elevated mood, reduced insight, sleep disturbance, and partial temporal disorientation. As no acute risk was identified, treatment was continued on an outpatient basis with close psychiatric follow-up. Over the following months, the patient attended regular outpatient visits, during which several adjustments of pharmacological therapy were made. Treatment included antipsychotics, mood stabilizers, antidepressants, and anxiolytics. Lithium carbonate was maintained throughout follow-up, with repeated measurements showing stable serum concentrations. Gradual clinical improvement was observed, although intermittent subdepressive symptoms were noted. During follow-up, the patient remained oriented, cooperative, without psychotic features or suicidal ideation, and involuntary hospitalization was not required.

Conclusion: This case demonstrates the clinical relevance of structured, long-term outpatient follow-up in patients with bipolar affective disorder. Continuous monitoring and timely modification of pharmacological treatment supported sustained clinical stability and functional preservation, highlighting the importance of continuity of care in chronic psychiatric disease management.

Keywords: Ambulatory Care; Antipsychotic Agents; Bipolar Disorder; Follow-Up Studies; Lithium

CR27

Therapeutic challenges and immunomodulatory treatment - a case of refractory familial Mediterranean fever

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Introduction: Familial Mediterranean fever (FMF) is an autoinflammatory disease caused by dysregulation of innate immune response, in which excessive activation of the inflammasome plays a key role, with a consequent increase in the secretion of interleukin-1 β .

Case presentation: A 37-year-old man has been treated at the Department of clinical immunology and rheumatology since 2018 due to FMF. He initially presented with daily high-grade fever and general algic syndrome. After a detailed clinical work-up, no clear cause of the fever was found, and a working diagnosis of autoinflammatory syndrome was made. Initial treatment included prednisone and colchicine, without any significant clinical effect. Genetic testing confirmed a mutation in the *MEFV* gene, resulting in a diagnosis of FMF. Interleukin-1 (IL-1) blocker, anakinra, was initiated; however, despite the higher dose of the biological drug, colchicine, and glucocorticoids, there wasn't any clinical improvement. The administration of interleukin-6 (IL-6) blocker, tocilizumab, was initially accompanied by a clinical improvement, although the fever persisted with less pronounced algic symptoms. Deterioration of the clinical condition, similar to the initial disease presentation, occurred after one year due to the diminishing effect of the biologic therapy. While hospitalized, the selective Janus kinase 1 inhibitor upadacitinib was administered, resulting in satisfactory subsequent symptom control.

Conclusion: This case illustrates the complexity of treating refractory FMF and highlights the disease's pathophysiological heterogeneity. While IL-1 blockade is the mainstay of FMF treatment, in some patients, other cytokine pathways—including IL-6 and JAK-STAT may dominate and are not fully suppressed by current therapeutic options. Therefore, managing refractory FMF requires a tailored treatment plan and ongoing monitoring of the clinical response and long-term complications like amyloidosis.

Keywords: Biologic Therapy; Familial Mediterranean Fever; Hereditary Autoinflammatory Diseases

CR28

Giant Internal Carotid Artery Aneurysm Mimicking Optic Neuritis

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Introduction: Giant intracranial aneurysms are uncommon and often present with symptoms due to compression of nearby structures rather than rupture. When they involve the optic nerve or chiasm, they can resemble optic neuritis, potentially delaying accurate diagnosis. We present a case of a young adult initially treated for optic neuritis, later found to have a giant internal carotid artery (ICA) aneurysm, and successfully managed with endovascular treatment.

Case report: A 28 – year – old man experienced a 10 – day history of progressive blurred vision in his left eye and worsening frontal and left temporal headache. He was initially hospitalized for suspected optic neuritis and received high – dose corticosteroids, which provided only mild improvement. Serology showed positive immunoglobulin M antibodies for *Toxoplasma gondii*, and antiparasitic therapy was initiated; however, this finding was clinically insignificant. Persistent symptoms prompted magnetic resonance imaging with angiography, which revealed a giant aneurysm in the C5 segment of the left ICA measuring approximately 30 x 26 x 24 millimeters (mm), partially thrombosed with a 15 mm residual lumen. There was a significant mass effect on the optic chiasm and left optic nerve. Computed tomography angiography confirmed a giant aneurysm up to 35 mm with a peripheral thrombus. Neurological examination showed decreased visual acuity in the left eye. After multidisciplinary consultation, the patient underwent successful endovascular embolization. Follow – up imaging showed complete aneurysm exclusion, with no residual flow. The patient remained neurobiologically stable with mild persistent visual impairment but no new deficits.

Conclusion: Giant ICA aneurysms can mimic optic neuritis, especially in young patients. Early neuroimaging is crucial in atypical or treatment–resistant visual symptoms with headache, allowing timely diagnosis and effective intervention for a favorable outcome.

Keywords: Carotid Artery, Internal; Intracranial Aneurysm; Optic Neuritis

CR29

A Rare Presentation of Obstructive Hydrocephalus Due To Atypically Localized Arachnoid Cysts – A case report

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Introduction: Arachnoid cysts, cerebrospinal fluid (CSF) collections within the arachnoid membrane, are the most common incidentally discovered intracranial lesions on neuroimaging. Although predominantly asymptomatic, they may cause hydrocephalus when occurring in unusual sites.

Case report: The case describes an 8-month-old male infant, born of a second pregnancy at 3570 g and APGAR 10, presented with laryngitis. Upon examination, the physician noted megacephaly, a bulging anterior fontanelle and upward gaze palsy. The patient required hospitalization, underwent cranial ultrasonography and computed tomography (CT). Both demonstrated pronounced ventriculomegaly, obstructed ventricular outflow, enlargement of the basal and cerebellomedullary cisterns, consistent with hypertensive hydrocephalus. Ventriculoperitoneal shunt with ultra-small medium-pressure valve with antibiotic-impregnated ventricular catheter was inserted in the frontal horn of the right lateral ventricle. Postoperative magnetic resonance imaging (MRI) displayed a cyst of the third ventricle compressing the cerebral aqueduct, confirming non-communicating hydrocephalus, and a supracerebellar cyst in posterior cranial fossa. Despite CSF drainage, the third ventricle demonstrated ongoing enlargement, while the supracerebellar cyst regressed. Subsequently, the patient underwent endoscopic ventriculocystocisternotomy of the third ventricle arachnoid cyst. Postoperative epileptic seizure was treated with levetiracetam (2*150 mg), which was continued. A subdural hygroma later developed and was drained with a temporary placed catheter. Follow-up ultrasound showed a 12 mm frontoparietal hygroma and a mild leftward midline shift. Head circumference remained unchanged at 51 cm. Neurological examination showed slight tone asymmetry without focal neurological deficits. Further neuroradiological follow-up via ultrasonography and MRI is required.

Conclusion: This case highlights the importance of tailored neurosurgical treatment for atypically localized arachnoid cysts causing hypertensive hydrocephalus in a neurodevelopmentally intact infant and the significance of MRI for comprehensive ventricular assessment and surgical planning.

Keywords: Arachnoid Cysts; Endoscopy; Hydrocephalus; Ventriculoperitoneal Shunt

CR30

Schizoaffective Disorder With Stress-Related Onset And Treatment Resistance: A Case Report

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Introduction: Schizoaffective disorder is a chronic psychiatric condition characterized by the coexistence of psychotic symptoms (hallucinations, delusions, disorganized thinking) and prominent mood episodes (depressive or manic). The disorder is associated with functional impairment and may follow a chronic or episodic course. The aim of this case report is emphasizing the clinical course, precipitating factors and treatment challenges.

Case report: In January 2026, a 47-year-old male economist was admitted for inpatient psychiatric treatment due to progressive worsening of symptoms. The patient presented with predominant anhedonia and abulia, accompanied by insomnia, social withdrawal, impaired self-care, tremor, sweating and palpitations. There was a maternal history of schizophrenia. The first psychiatric symptoms developed in July 2024 following a negative professional evaluation at work, representing a significant psychosocial stressor. Initial manifestations included guilt, anxiety, insomnia and impaired concentration. Afterwards, beliefs of being monitored and investigated at the workplace emerged. The first hospitalization was in December 2024 due to symptom exacerbation. Subsequently, the patient received various pharmacological treatments, including antidepressants, mood stabilizers, sedatives and antipsychotics, with limited improvement. During the January 2026 admission, six sessions of electroconvulsive therapy (ECT) were administered due to treatment resistance, resulting in improved mood and sleep regulation, increased social engagement and functional outcome improvement. Based on the chronic course (>18 months), persistence of psychotic symptoms accompanied by depressive features, the final diagnosis was schizoaffective disorder.

Conclusion: This case emphasizes the diagnostic challenges posed by overlapping affective and psychotic symptoms and supports the role of ECT as a treatment option to reduce symptom severity and improve functional outcome in treatment-resistant cases.

Keywords: Electroconvulsive Therapy; Psychotic Disorders; Schizoaffective Disorder; Stress, Psychological

CR31

Neglected Follow-Up in Drug-Induced Chronic Kidney Disease Leading to End-Stage Renal Disease and Dilated Cardiomyopathy: A Case Report

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Introduction: Neglected chronic kidney disease (CKD) represents a major global health burden, particularly among patients disengaged from long-term care. Inadequately monitored CKD is associated with cardiovascular complications, which increase morbidity and mortality. We present a case of advanced, previously stabilized, subsequently neglected CKD progressing to end-stage renal disease (ESRD) and congestive heart failure with echocardiographic features of dilated cardiomyopathy, highlighting the consequences of loss to follow-up in chronic disease management.

Case report: 32-year-old man with ulcerative colitis treated with mesalazine since 2018 was referred to nephrology for azotemia (urea 23.3 mmol/L, creatinine 520 µmol/L). Renal biopsy confirmed tubulointerstitial nephritis, likely mesalazine-induced, with histological features of CKD. Following treatment with glucocorticoids and supportive therapy, mesalazine was discontinued. Renal function remained stable until June 2020, after which he was lost to follow-up. In December 2025, he presented with ESRD (urine albumin-to-creatinine ratio 347.6, estimated glomerular filtration rate 6.7), uncontrolled hypertension (170/100 mmHg), and decompensated heart failure (dyspnea, peripheral edema, ascites, NT-proBNP >35,000), requiring intensive care. After cardiopulmonary stabilization, hemodialysis was initiated via a central venous catheter. Anemia (hemoglobin 74 g/L) was treated with short-acting erythropoietin. Renal ultrasound showed bilaterally reduced kidney size with thin hyperechogenic cortex. Echocardiography revealed dilated cardiomyopathy with biventricular failure, left ventricle ejection fraction was 18% (normal 50-75%, NYHA II). He is now on stable chronic hemodialysis with plans for peritoneal dialysis.

Conclusion: This case illustrates the importance of continuous follow-up and long-term CKD management. Despite appropriate initial diagnosis and treatment, loss to follow-up likely enabled unrecognized progression, culminating in ESRD and cardiovascular complications. Early surveillance and sustained engagement may prevent irreversible outcomes.

Keywords: Chronic Kidney Disease; Dilated Cardiomyopathy; Heart Failure; Hemodialysis; Tubulointerstitial Nephritis

CR32

Nephrotic syndrome: A hematologic red flag – a case report

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Introduction: Primary AL amyloidosis carries high morbidity and mortality from progressive organ involvement. Renal disease often presents with increasing proteinuria, sometimes reaching nephrotic syndrome, while kidney function may initially remain normal. This preserved function can delay diagnosis of the underlying plasma cell disorder, highlighting the diagnostic challenge of recognizing renal AL amyloidosis early in clinical practice settings.

Case report: Our patient is a 50-year-old female who was referred to a nephrologist due to persistent proteinuria detected in primary care. Past medical history included autoimmune thyroiditis and cholecystectomy. Initial evaluation revealed proteinuria with intermittent erythrocyturia and preserved renal function. Over several months, albuminuria progressed from A2 to A3 category, while serum creatinine remained within the reference range. The patient was initially asymptomatic, without edema, hyperlipidemia, or hypertension. During outpatient follow-up for proteinuria, she developed progressive peripheral edema, periorbital swelling in the morning, foamy urine, and nephrotic-range proteinuria. Due to worsening of albuminuria and clinical signs of nephrotic syndrome, a kidney biopsy was performed. The findings were consistent with AL amyloidosis with associated light chain proximal tubulopathy and monoclonal lambda light chain expression. Flow cytometric immunophenotyping of the bone marrow aspirate revealed a small population (2%) of clonal plasma cells (CD45-, CD56+/-, CD38+, CD138+) with lambda light chain restriction, consistent with an underlying plasma cell dyscrasia. A diagnosis of primary AL amyloidosis with renal involvement secondary to plasma cell dyscrasia was established. The patient was discussed at a hematology multidisciplinary meeting, and treatment with the CyBorD-Dex protocol was initiated.

Conclusion: The heterogeneous and nonspecific presentation of primary amyloidosis often delays diagnosis. In this case, nephrotic syndrome was the key clue leading to definitive diagnosis and treatment initiation.

Keywords: Amyloidosis ; Nephrotic Syndrome ; Paraproteinemias

CR33

What (Not) to Expect When You Are Expecting: Peripartum Unmasking of a Genetic Cardiomyopathy

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Introduction: Dilated cardiomyopathy (DCM) is characterized by left ventricular (LV) dilatation coupled with global or regional systolic dysfunction that cannot be explained solely by abnormal loading conditions. In approximately 30% of cases, a genetic mutation can be found, such as the troponin T (*TNNT2*) gene mutation, which alters the structure of cardiac troponin T, thus impairing the sarcomeric function.

Case report: A 30-year-old female patient presented with progressive dyspnoea and fatigue starting in her third trimester of pregnancy, worsening throughout postpartum. Her history included childhood syncopal episodes and a family history of postpartum cardiomyopathy in maternal grandmother. Despite medical treatment, her condition deteriorated with hypotension, tachycardia and severe dyspnoea at rest, in New York Heart Association class IV, due to which she was referred to a tertiary center. Echocardiography and cardiac magnetic resonance showed moderate LV dilatation, severely reduced LV ejection fraction (20%), global hypokinesia, and severely impaired right ventricular function. The overall findings were consistent with primary DCM with peripartum decompensation. Despite dobutamine infusion, intermittent levosimendan administration, and intravenous diuretic therapy, her condition didn't improve, and, since she was not a candidate for LV assist device (LVAD) therapy, due to biventricular cardiomyopathy, she underwent a successful orthotopic heart transplantation, followed by an uncomplicated early postoperative course. Genetic testing revealed a heterozygous mutation in the *TNNT2* gene. Over the eight years of follow-up, she remained asymptomatic, with preserved graft function and no signs of graft rejection.

Conclusion: This case illustrates the severe clinical trajectory that *TNNT2*-associated cardiomyopathy may take under the hemodynamic and hormonal stressors of pregnancy and childbirth. Establishing genetic aetiology was essential for explaining the severe peripartum course, genetic counselling and long-term risk assessment of family members.

Keywords: Cardiomyopathy, Dilated; Heart Failure; Heart Transplantation; Peripartum Period

CR34

The Double-Edged Sword of Long-term Corticosteroid Therapy in Myasthenia Gravis: A Case for a Personalized Immunomodulatory Approach

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Introduction: Myasthenia gravis (MG) is a chronic autoimmune neuromuscular disease characterized by fluctuating muscle weakness. Although corticosteroids are the gold standard of immunosuppression, their long-term use represents a significant iatrogenic burden. The aim of this case report is to emphasize the necessity of a personalized immunomodulatory approach through the timely introduction of steroid-sparing therapy.

Case report: We present a 65-year-old patient with generalized seropositive MG and seropositive rheumatoid arthritis. Due to the polyautoimmune phenotype, the patient was treated for years with progressively increasing doses of corticosteroids. Long-term administration of prednisone resulted in severe iatrogenic complications, including steroid-induced diabetes mellitus, severe osteoporosis with pathological fractures, and chronic prostatitis due to iatrogenic immunosuppression. On two occasions, the patient developed a severe exacerbation, specifically a myasthenic crisis, requiring hospitalization in the intensive care unit. The clinical presentation was dominated by bulbar symptoms and respiratory insufficiency. In both instances, significant clinical restitution was achieved through the administration of intravenous immunoglobulins. Given the limited efficacy and unacceptable long-term toxicity of corticosteroids, the introduction of azathioprine as a steroid-sparing immunosuppressant became a critical intervention. This personalized approach was aimed at stabilizing the disease, restoring the patient's quality of life, and preventing further iatrogenic damage.

Conclusion: This case illustrates the double-edged sword of long-term corticosteroid therapy. While essential for rapid symptom control, cumulative multisystem complications and secondary infections may paradoxically precipitate further myasthenic crises. To prevent further exacerbations and reduce iatrogenic toxicity, a personalized immunomodulatory approach is essential. This strategy represents the cornerstone for stabilizing the disease and improving the quality of life in such complex patients.

Keywords: Azathioprine; Iatrogenic Disease; Myasthenia Gravis

CR35

Anterior abdominal wall as a rare site of endometriosis: A diagnostic challenge - Case report

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Introduction: Endometriosis is a chronic, estrogen-dependent condition characterized by the presence of functional endometrial tissue outside the uterine cavity. In this case, we aim to present how, on rare occasions, it may occur in extraperitoneal locations, including abdominal muscles.

Case report: We report the case of a 38-year-old female patient who presented to the surgical outpatient clinic with an intermittently painful mass in the umbilical region, initially clinically suspected to be an umbilical hernia. As part of the diagnostic workup, an abdominal multi-slice scan (MSCT) was performed. It revealed a well-defined soft tissue mass in the left paraumbilical region within the subcutaneous tissue adjacent to the rectus abdominis fascia, without communication with intra-abdominal structures. The mass measured 2.1 x 2.7 x 2.7 cm in size. Cytological aspiration of the lesion did not provide a definitive diagnosis, and surgical excision was indicated. Intraoperatively, a cystic lesion with a firm capsule, filled with dark green pasty material, was observed. Histopathological analysis of the excised tissue revealed glandular tissue characteristic of endometrium, leading to a diagnosis of abdominal wall endometriosis. The postoperative course was uneventful, and the patient continued follow-up with her gynecologist.

Conclusion: This case highlights the importance of considering endometriosis in the differential diagnosis of soft tissue masses of the abdominal wall, particularly in women of reproductive age presenting with nonspecific or atypical symptoms. Although rare, extraperitoneal localization of endometrial tissue can clinically mimic other pathological entities, such as umbilical hernia, potentially delaying correct diagnosis. Surgical excision remains the treatment of choice, offering both diagnostic and therapeutic benefits, while histopathological evaluation is essential for definitive confirmation of the diagnosis.

Keywords: Abdominal Wall; Differential Diagnosis; Endometriosis; Umbilical Hernia

CR36

Secondary Hemophagocytic Syndrome as a Presenting Feature of Systemic Lupus Erythematosus

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Introduction: Systemic lupus erythematosus (SLE) is an multisystemic autoimmune disease characterized by the production of autoantibodies and immune complex deposition. Hemophagocytic syndrome is a severe hyperinflammatory disorder marked by immune dysregulation, a cytokine storm, and multiorgan dysfunction due to uncontrolled macrophage and lymphocyte activation. When occurring within autoimmune diseases like SLE, this syndrome is termed macrophage activation syndrome (MAS); notably, its presentation as the initial manifestation of SLE is exceedingly rare.

Case report: We present a 42-year-old woman with unspecified fever, symmetric polyarthralgia, inguinal bilateral and left cervical lymphadenopathy. Initial laboratory results revealed elevated values of inflammatory parameters, lactate dehydrogenase, elevated creatinine with pathological urine sediment and lymphopenia. Microbiological investigations were negative. Multislice Computed Tomography of chest, abdomen and pelvis showed axillary and inguinal lymphadenopathy, hepatomegaly, and left adnexal cysts. Cytology and biopsy of inguinal lymph node was unremarkable. During hospitalization, the patient developed severe pancytopenia and splenomegaly, with elevated ferritin and triglyceride levels. Based on clinical and laboratory findings, MAS was suspected. High-dose corticosteroid pulse therapy and intravenous immunoglobulin were initiated, leading to laboratory improvement. Subsequently during the hospitalization, positive antinuclear antibody and double-stranded deoxyribonucleic acid were received and given the clinical picture, a diagnosis of SLE with MAS as the initial manifestation was established. Treatment was initiated with cyclophosphamide according to the Euro lupus protocol, along with the antimalarial therapy. The patient remained clinically stable and was discharged.

Conclusion: This case illustrates MAS as an extremely rare primary manifestation of SLE, highlighting the necessity of early intervention and aggressive therapy in overlapping autoimmune syndromes.

Keywords: Cytokine Release Syndrome; Lupus Erythematosus, Systemic; Macrophage Activation Syndrome

CR37

From Parenteral Nutrition to Enteral Independence: Intestinal Rehabilitation After Multivisceral Transplantation

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Introduction: Multivisceral transplantation (MVT) is indicated in patients with intestinal failure accompanied by secondary dysfunction of other organs. Due to the high immunogenicity of the intestine, the occurrence of rejection, infections, nutritional challenges, and associated mortality, MVT represents the most complex transplantation procedure. A particular challenge lies in the introduction of oral nutrition.

Case report: A 58-year-old patient underwent MVT, including the liver, stomach, small intestine, pancreas, and colon, due to extensive thrombosis of the splanchnic circulation secondary to antiphospholipid syndrome. Postoperatively, the patient received supportive, prophylactic, and immunosuppressive therapy and was closely monitored. Endoscopic evaluations and biopsies revealed no evidence of acute cellular rejection. The early postoperative phase is critical for transitioning from parenteral to enteral nutrition. Intestinal rehabilitation was initiated on postoperative day five with jejunostomy-administered enteral nutrition. The dosage was gradually increased under close monitoring of the patient's nutritional status and stomal effluent. Oral feeding was introduced on postoperative day 18; however, due to poor tolerance, intake was temporarily limited to water and tea. Modified blended diet was subsequently introduced, supplemented with parenteral support. Although gastric peristalsis recovered slowly, intestinal motility improved with patient mobilization and prokinetic therapy. With controlled oral intake and additional parenteral rehydration, the patient showed gradual improvement, including recovery of renal function, normalization of electrolyte balance, and achievement of complete oral nutrition.

Conclusion: This case highlights the importance of individualized nutritional management in intestinal rehabilitation. When combined with multidisciplinary monitoring, it plays a crucial role in the success of multivisceral transplantation, with enteral nutrition promoting intestinal adaptation and improved outcomes.

Keywords: Antiphospholipid Syndrome; Immunosuppression Therapy; Parenteral Nutrition; Postoperative Care; Transplantation

CR38

Targeting Cardiac Myosin: Mavacamten in Symptomatic Obstructive Hypertrophic Cardiomyopathy

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Introduction: Obstructive hypertrophic cardiomyopathy (HOCM) is a genetic disorder characterized by thickening of the myocardium. It is caused by mutations in sarcomere protein genes. This case report highlights the effect of mavacamten (a selective cardiac myosin inhibitor) in the treatment of symptomatic HOCM by reducing left ventricular outflow tract (LVOT) obstruction.

Case report: A 32-year-old male has been followed since 2016 due to stress-related palpitations, arterial hypertension, and suspected HOCM. Echocardiography (ECHO) revealed asymmetric septal hypertrophy (septal diameter of 22 mm), systolic anterior motion of the mitral valve with mitral valve prolapse (A2/A3), and moderate regurgitation. He was treated with a beta blocker and an ACE inhibitor. In March 2025, he was referred to Hospital Centre Zagreb due to worsening symptoms - New York Heart Association (NYHA) score II. Stress ECHO revealed a LVOT gradient of 37 mmHg at rest, 70 mmHg during Valsalva, and 100 mmHg during exercise. Genetic testing confirmed the presence of *MYBPC3* (c.913_914del, heterozygous) variant. Mavacamten at a dose of 5 mg was started. During follow-up, the dosage had to be increased to 10 mg because of persistent symptoms and a high LVOT gradient. Ejection fraction was preserved throughout (55–65%). Non-sustained ventricular tachycardia was diagnosed during 24-h electrocardiogram monitoring, and an implantable cardioverter-defibrillator was implanted. Follow-up ECHO demonstrated the LVOT gradient of 28 mmHg during Valsalva, NYHA score was I, and the current medication dose was maintained.

Conclusion: Mavacamten is a new medication in cardiology that reduces excessive myocardial contractility by decreasing LVOT obstruction and improving symptoms such as dyspnea, angina, and dizziness. In this case, mavacamten proved to be effective, and this represents a promising future option in the treatment of symptomatic HOCM.

Keywords: Cardiomyopathy, Hypertrophic; Drug Therapy; Genetic Testing

CR39

Measles in the Era of Vaccine Hesitancy: A Case Report

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Introduction: Measles is a highly contagious viral disease that typically presents with high fever and cough, followed by a distinctive rash. It is important to obtain an epidemiological history and patient vaccine status to recognize disease early.

Case report: We present the case of a 43-year-old woman with fever up to 39.5°C, cough, and sore throat for four days prior to examination. Physical examination revealed no abnormalities except pharynx erythema. Patient reported she returned from a trip to Turkey two weeks ago. Vaccination against measles was refused by the parents in the patient's childhood. The patient was discharged with a diagnosis of viral pharyngitis. The next day, patient's condition worsened. Erythematous maculopapular rash developed, initially behind the ears, on the neck and near the hairline. Patient also had bilateral conjunctivitis. Based on the patient's symptoms and epidemiological history, real-time reverse transcription polymerase chain reaction for measles virus was performed on serum and urine samples. The patient received symptomatic treatment, as no specific therapy exists. Later, the rash spread in a descending pattern to the trunk and extremities. Both samples tested positive for the measles virus. Seven days later, as rash was regressing, patient developed blurred vision, headache, and eyelid edema. Keratitis was suspected as a complication of the disease. The patient was referred to an ophthalmologist and treated. However, visual impairment in one eye was permanent.

Conclusion: Measles can be a severe disease with various complications, including pneumonia, encephalitis, or keratitis, as seen in this case; yet disease is fully preventable through vaccination. Unfortunately, it reemerges as a significant health concern due to vaccine hesitancy.

Keywords: Keratitis; Measles; Vaccines

CR40

Mechanical thrombectomy: a bail out procedure when conventional therapy fails

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Introduction: High-risk pulmonary thromboembolism is a life-threatening obstruction of pulmonary arteries, which can lead to acute right heart failure. In this case report we present a young woman that developed high-risk pulmonary thromboembolism in postpartum. Despite conventional fibrinolytic therapy with alteplase, signs and symptoms of acute right heart failure persisted which indicated bail out mechanical thrombectomy.

Case report: A 29-year-old postpartum woman presented to the emergency department with symptoms of dyspnea and pain located in left hemithorax. During physical examination signs of tachycardia and hypoxemia were evidenced. At admission, computed tomography (CT) angiography presented perfusion defect in pulmonary arteries. Right ventricular dilatation and increased afterload were confirmed through transthoracic echocardiography (TTE). Along with hemodynamic instability, a CT scan confirmed a high-risk pulmonary thromboembolism. Upon implementation of fibrinolytic therapy with alteplase, no signs of recovery presented and following control TTE showed no improvement. Bail out mechanical thrombectomy was indicated. Procedure was done successfully following improvements in symptoms. Control angiography showed a significant improvement in recanalization of the right pulmonary artery followed by improvement of right heart function, confirmed by control TTE. Tests for hereditary thrombophilia came negative. Furthermore, other risk factors, other than postpartum and smoking, were not present. In consultation with patient, anticoagulant therapy with strict monitoring was agreed upon, since she planned another pregnancy.

Conclusion: Rapid recognition is crucial in cases like this, along with availability of a bail out procedure if conventional therapy fails. Given that the patient is at an increased risk of thrombogenesis, it is important to prevent future complications through strict monitoring and ordinating anticoagulant therapy in a higher risk state, such as pregnancy.

Keywords: Embolism; Postpartum Period; Thrombectomy

CR41

Infectious-Like Onset of ANCA-Associated Vasculitis with Preserved Renal Histology in an Adolescent

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Introduction: ANCA-associated vasculitis (AAV) represents a group of systemic inflammatory disorders in children and adolescents, typically characterized by renal and pulmonary involvement. Histopathological confirmation by renal biopsy plays a central role in assessing disease severity, guiding treatment decisions, and predicting long-term outcomes.

Case report: A 16-year-old male was initially hospitalized at the infectious disease department in September 2025, due to prolonged fatigue, productive cough, nasal congestion, and blood-tinged nasal discharge, accompanied by subfebrile temperatures up to 38.5 °C. Initial laboratory and serological testing were unremarkable. In November, he was admitted to the pediatric rheumatology department, where a comprehensive workup was performed. Laboratory findings revealed anemia, elevated inflammatory markers, elevated D-dimer levels, and positive p-ANCA antibodies. Urinalysis showed significant erythrocyturia and proteinuria. A High-Resolution Computed Tomography of the chest was performed, revealing bilateral parenchymal abnormalities – ground glass opacities, centrilobular nodules with a „tree in bud“ pattern, and peripheral consolidations. Bronchoscopy with endobronchial biopsy revealed diffuse alveolar hemorrhage. A renal biopsy was performed, but it showed no significant pathological changes. Based on clinical assessments and elevated inflammatory markers, a diagnosis of AAV was made. Treatment was initiated with high-dose intravenous methylprednisolone pulse therapy for remission induction, leading to significant clinical improvement. The patient was discharged on maintenance immunosuppressive therapy with mycophenolate mofetil and methylprednisolone, with planned outpatient rheumatology follow-up.

Conclusion: Adolescents with persistent infectious-like symptoms, elevated inflammatory markers, and erythrocyturia/proteinuria may harbor AAV, even in the absence of renal histopathology. Early high-dose glucocorticoid therapy can rapidly reverse organ involvement.

Keywords: Antibodies, Antineutrophil Cytoplasmic; Biopsy; Bronchoscopy; Methylprednisolone; Vasculitis

CR42

Clinical Results of Stem Cell and Exosome treatment for complex genetic disorders

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Introduction: Managing complex genetic disorders remains highly difficult because of their variable clinical manifestations, and the significant cost of advanced treatment options. It is essential to explore affordable, and accessible therapies, particularly those based on regenerative stem cell approaches using Medicinal Signaling Cells (MSCs).

Case report: A five-year-old girl was diagnosed with Prader–Willi syndrome (PWS) and calpainopathy. Her clinical picture included muscle hypotonia, delayed development, elevated creatine kinase levels, and early obesity onset. Comprehensive genomic testing detected maternal uniparental isodisomy of chromosome 15q, a unique genomic alteration introducing a biallelic frameshift variant in the *CAPN3* gene. This mutation led to the manifestation of two separate yet coexisting disorders. The patient was treated with umbilical cord- and placenta-derived allogeneic MSCs administered intravenously and intramuscularly to achieve sustained regenerative benefits and intranasal administrations were used for maintenance therapy. The patient now exhibits enhanced cognitive, muscular and endocrine function. Awareness of her environment and responses to external stimuli are improved. She completes tasks more efficiently and is able to disengage appropriately when encountering difficulties. Her reaction time has improved, and interactions with her caregivers and peers have become easier and more cooperative. Her verbal communication is clearer and louder. Her muscle tone, strength and endurance have shown visible improvements. She has lost 0.5 kg in body weight.

Conclusion: Both MSCs and MSC-exos have stimulated neuroplasticity which is worth further exploring to establish mechanisms. They also contributed to in vivo muscle repair, assisting in myofiber formation and activation of satellite cells which led to the prevention of metabolic dysfunction and obesity. Due to their wide availability, and safety, these biologics represent a promising direction in personalized regenerative medicine for muscular dystrophies, obesity, and related disorders.

Keywords: Limb-Girdle Muscular Dystrophy Type 2A; Neuronal Plasticity; Prader-Willi Syndrome

CR43

Intratympanic Application of Gentamicin in Meniere's Disease

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Introduction: Ménière's disease is a chronic inner ear disorder characterized by recurrent episodes of vertigo, fluctuating sensorineural hearing loss, and tinnitus. Diagnosis is primarily clinical; however, audiometric testing is essential for confirming sensorineural hearing loss and for monitoring disease progression. Conservative treatment usually includes dietary modification and diuretic therapy, although these measures often provide limited benefit. In patients with persistent or severe symptoms, intratympanic therapy or surgical interventions may be required.

Case report: We present a 55-year-old woman with a 15-year history of Ménière's disease. At onset, audiological and vestibular evaluation showed unilateral labyrinthine hypofunction and fluctuating sensorineural hearing loss, which worsened with each vertigo attack. During acute episodes, spontaneous nystagmus and postural instability toward the affected side were observed. In the chronic phase of the disease, vestibular testing consistently indicated unilateral labyrinthine dysfunction. The Fukuda stepping test resulted in rotation toward the affected ear, confirming persistent vestibular asymmetry. Initial conservative management included high-dose betahistine; however, vertigo attacks remained frequent and severe. Over time, the patient developed significant depressive symptoms related to disease burden. Intratympanic corticosteroid therapy was initiated after insertion of a ventilation tube, which allowed repeated drug administration and helped regulate middle ear pressure. Dexamethasone was given twice weekly, without significant improvement. After discussing risks with the patient, chemical labyrinthectomy with a single intratympanic gentamicin injection was performed. Following treatment, vertigo resolved completely. Residual sensorineural hearing loss of 70 dB persisted, but the patient regained independence and returned to normal daily activities.

Conclusion: In severe, refractory Ménière's disease, intratympanic gentamicin provides effective vertigo control. Ablative treatment should be reserved for carefully selected patients who do not respond to conservative therapy, with close audiological monitoring.

Keywords: Hearing Loss, Sensorineural; Meniere Disease; Tinnitus; Vertigo

CR44

Radiation-Associated Extraskkeletal Osteosarcoma

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Introduction: Radiation-associated extraskkeletal osteosarcoma is a rare but recognized complication of radiotherapy, characterized by the formation of malignant osteoid within soft tissues. This case report highlights the importance of thoroughly understanding the patient's medical history and correlating previous therapeutic interventions with newly emerging clinical symptoms.

Case report: We present the case of a 66-year-old woman with constipation, intermittent rectal bleeding, and a one-month history of dull abdominal pain that worsened two days prior to admission. Her medical history included cervical cancer treated in 1989 with a radical Wertheim hysterectomy, followed by external beam radiotherapy and brachytherapy. Colonoscopy identified a proliferative lesion 8 cm from the anocutaneous line, revealing a rectal lumen defect and an adjacent blind-ending cavity. Based on these findings, urgent surgical intervention was indicated. Intraoperatively, a large, firm pelvic mass infiltrating the rectum and pelvic wall was found. A radical procedure was not performed; instead, a terminal colostomy was created to relieve bowel obstruction, and tumor biopsies were taken. Histopathology confirmed radiation-associated extraskkeletal osteosarcoma. The patient subsequently underwent pelvic exenteration and Bricker ureteroileostomy. Six months later, computerized tomography revealed local recurrence in the left pelvis, complicated by extensive deep vein thrombosis of the left iliac and femoral veins, as well as lumbar vertebral metastases. The patient died from the disease nine months after her initial presentation.

Conclusion: This case demonstrates the potential for post-radiation extraskkeletal osteosarcoma to develop decades after primary cancer treatment. It highlights the diagnostic and therapeutic complexities associated with secondary malignancies in previously irradiated fields. The findings emphasize the necessity for vigilant long-term follow-up and a high index of suspicion for atypical tumor presentations in this patient population.

Keywords: Osteosarcoma; Pelvis; Radiotherapy

CR45

Management of adverse therapeutic symptoms mimicking acute abdomen in a neurologically complex child: A case report

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Introduction: Infantile spasms (IS) present a severe epileptic encephalopathy of infancy, most commonly associated with structural brain abnormalities often requiring diagnostic and therapeutic challenges, which can induce systemic complications. Herein, we report a child with a complex neurological disorder developing symptoms of acute abdomen during treatment of recalcitrant IS.

Case Report: A six-month-old male infant with left-sided neuromotor asymmetry, left-sided optic nerve atrophy and developmental delay was hospitalised for continuous clusters of flexor spasms. The neonatal period was burdened by intracranial haemorrhage due to the rupture of a right-sided cerebral arteriovenous malformation, followed by venous infarction of the right thalamus and subsequent hydrocephalus, requiring a ventriculoperitoneal shunt. Genetic testing did not identify a clear pathogenic variant for neurological disorders. During hospitalisation, electroencephalography revealed hypsarrhythmia with right hemispheric predominance, consistent with infantile spasms. Therapy with levetiracetam, prednisolone, and vigabatrin was initiated, resulting in gradual neurological and electroencephalographic improvement. After three weeks of therapy, the patient presented with abdominal distension lasting for 10 days before the appearance of haematochezia. Abdominal X-ray revealed pneumoperitoneum, confirmed by an abdominal computed tomography, which excluded bowel ischemia and mesenteric vascular thrombosis. In the context of long-term corticosteroid therapy, a steroid-related etiology of abdominal symptoms was suspected. As the patient was clinically stable, tolerated oral intake and showed no signs of peritonitis on abdominal examination, conservative management (including prednisolone tapering) was sufficient and the patient recovered within one week.

Conclusion: This case highlights that complex neurological patients require a comprehensive clinical approach, including genetic analysis for tailoring long-term care and managing challenging adverse treatment symptoms.

Keywords: Abdomen, Acute; Intracranial Arteriovenous Malformations; Intracranial Hemorrhages; Spasms, Infantile

CR46

Integrating Genomic Profiling and Immunotherapy in the Management of a Rare Maxillary Squamous Cell Carcinoma

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Introduction: Malignant tumors of the maxillofacial region are challenging due to complex anatomy, heterogeneous biology, and proximity to critical neuro-orbital structures. Advances in immunogenomics now allow precision oncology approaches, especially when conventional surgery carries high morbidity. We present a case illustrating the role of integrated comprehensive genomic profiling in managing a rare, advanced maxillary squamous cell carcinoma.

Case report: A 64-year-old male patient with prior left-eye vision loss due to trauma presented with progressive facial paresthesia, right-sided facial pain, and worsening vision. Imaging revealed a large, locally advanced tumor of the right maxillary region adjacent to orbital structures. Radical maxillectomy with orbital exenteration was considered but deferred due to the high risk of irreversible vision loss and the patient's preference. Initial systemic chemotherapy produced no meaningful clinical or radiological response. Definitive radiotherapy achieved significant tumor reduction and symptomatic relief. Given chemotherapy resistance, comprehensive genomic profiling was performed, revealing a high tumor mutational burden of 12 mutations per megabase. Based on this, pembrolizumab therapy was initiated. Pembrolizumab blocks the programmed cell death protein 1 receptor on activated T lymphocytes, restoring antitumor immunity. The patient continues on immunotherapy, with evidence of disease stabilization and good tolerability.

Conclusion: This case demonstrates the importance of comprehensive genomic profiling in rare, anatomically complex malignancies. Combining radiotherapy for tumor debulking with immune checkpoint inhibition allowed an effective organ-preserving approach. Demonstrating the translational relevance of immunology in precision oncology can thus convert locally advanced maxillofacial tumors into manageable chronic disease, offering a model for high-risk, rare head and neck cancers.

Keywords: Carcinoma, Squamous Cell; Immunotherapy; Maxillary Neoplasms; Pembrolizumab; Radiotherapy

CR47

From Sore Throat to Septic Shock: A Severe Case of Lemierre's Syndrome in a Previously Healthy Female

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Introduction: Lemierre's syndrome (LS) is a rare, life-threatening condition characterized by septic thrombophlebitis of the internal jugular vein. It most commonly occurs after an oropharyngeal infection and is caused by *Fusobacterium necrophorum*. The condition primarily affects young, previously healthy individuals and can rapidly progress to severe sepsis. Early recognition and aggressive multidisciplinary management are essential.

Case report: This case report presents a 19-year-old previously healthy female who developed high-grade fever and symptoms of pharyngitis, for which oral antibiotics were prescribed. One week later, the patient experienced rapid clinical deterioration with progressive right-sided neck swelling, severe right shoulder and chest pain, and increasing respiratory distress. Her general condition worsened rapidly, with signs of systemic infection. Because of rapid hemodynamic deterioration, the patient was admitted to intensive care unit with septic shock, manifesting as hypotension, oliguria, severe thrombocytopenia, and markedly elevated inflammatory markers. Contrast-enhanced computed tomography revealed extensive right-sided peritonsillar, parapharyngeal, and posterior cervical abscesses with gas formation, thrombosis of the right internal jugular vein, acute anterior mediastinitis, bilateral pneumonia, and evidence of septic embolization. Emergency management included tracheotomy, extensive cervical and mediastinal surgical drainage with ligation of the thrombosed jugular vein, invasive mechanical ventilation, vasopressor support, and continuous veno-venous hemodiafiltration for multiorgan dysfunction. Cultures obtained from abscess material grew *Fusobacterium necrophorum*, supporting the LS diagnosis. Targeted antimicrobial therapy with meropenem and metronidazole resulted in clinical, laboratory, and radiological improvement.

Conclusion: This case highlights the rapid progression and severity of LS, emphasizing the importance of early recognition and aggressive multidisciplinary management. Clinicians should maintain a high level of suspicion for this syndrome in young patients presenting with oropharyngeal infections and neck swelling.

Keywords: *Fusobacterium necrophorum*; Lemierre Syndrome; Thrombophlebitis

CR48

Multiple Intestinal Atresias and Combined Immunodeficiency Syndrome: A Case of Two Siblings

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Introduction: Multiple intestinal atresias (MIA) and combined immunodeficiency syndrome (CID) is a rare autosomal-recessive disorder caused by mutations of the tetratricopeptide 7A (*TTC7A*) gene. The *TTC7A* gene encodes a protein involved in epithelial cell polarization, which, when disrupted, results in abnormal intestinal and thymus epithelium. Herein, we report two siblings with a *TTC7A* gene mutation and a severe clinical presentation.

Case Report: A term female baby, with a late prenatally diagnosed suspicion of intestinal atresia in the 37th week of gestation, underwent surgical treatment on the second day of life due to ileus. She was diagnosed with multiple small bowel atresias and colon agenesis. During the subsequent clinical course, she often presented with intestinal bleeding requiring blood transfusions, and enteral nutrition could not be achieved. Additionally, since birth, the patient suffered from recurrent bacterial infections and hence underwent immunological analysis, which revealed CID (IgA/IgM<0.05 g/L, IgG 7.40 g/L; CD3+ 54%, CD19+ 15%; PHA 7%, ConA 4%). Genetic analysis identified a homozygous *TTC7A*/c.315_318del mutation, confirming the MIA-CID syndrome. Despite treatment with haematopoietic stem cell transplantation, recurrent bacterial infections ultimately led to fulminant sepsis and death at six months of age. The male sibling was born two years later as a preterm at 34 weeks with previously suspected intestinal atresias at the 30th week of gestation. He underwent a similar clinical course with genetic testing confirming MIA-CID syndrome and died at three months of age due to bacterial sepsis.

Conclusion: Regular prenatal follow-up combined with early genetic analysis and family counseling is imperative in rare autosomal recessive diseases such as MIA-CID syndrome, given the 25% occurrence risk in future pregnancies, and may enable targeted therapies to prevent adverse outcomes.

Keywords: Genes, Recessive; Intestinal Atresia; Sequence Deletion; Severe Combined Immunodeficiency

CR49

Is Genetic Testing Mandatory? Phenotypic Diagnosis of Arrhythmogenic Cardiomyopathy in a Patient with Positive Family History

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Introduction: Arrhythmogenic cardiomyopathy (ACM) is an inherited heart disease where the heart muscle is replaced by fatty and fibrous tissue. The majority of cases are right-dominant (arrhythmogenic right ventricular cardiomyopathy, ARVC), with 15% affecting only the left side and the rest being biventricular. According to the 2020 Padua criteria, six diagnostic categories are used, only one involving genetics. A diagnosis of ARVC is established by fulfilling two major, one major and two minor, or four minor criteria from different categories, whereas genetic confirmation is mandatory only in left-dominant phenotypes. We present a case questioning the necessity of genetic testing when phenotypic criteria are met.

Case report: A 28-year-old woman presented with palpitations, dyspnea, chest discomfort, and left arm tingling after light exercise. She had a family history of ACM. Her father and brother had an implantable cardioverter-defibrillator (ICD) implanted due to phenotypic ARVC. The initial electrocardiography (ECG) showed no rhythm abnormalities but revealed inverted T waves in leads V1-V4 without a right bundle branch block. Echocardiography showed a normal left and a dilated right ventricle, with reduced ejection fraction, apical dyskinesia, and akinesia. This was confirmed with magnetic resonance. A 24-hour ECG detected 2,636 ventricular extrasystoles. The patient fulfilled three major (ACM in a first-degree relative, ECG repolarization abnormalities, morpho-functional ventricular abnormalities) and one minor criteria (ventricular arrhythmias) for ARVC, and an ICD was implanted due to high risk of sudden cardiac death.

Conclusion: ARVC diagnosis and ICD indication were based on high-risk phenotypic features, including right ventricular dysfunction and ventricular arrhythmias. Genetic testing was not required for this right-dominant phenotype but remains mandatory for left-dominant ACM to differentiate it from similar pathologies.

Keywords: Arrhythmogenic Right Ventricular Dysplasia; Defibrillators, Implantable; Genetic Testing

CR50

Persistent Graves' disease caused by multiple sclerosis medication alemtuzumab

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Introduction: Graves' disease is an autoimmune disorder in which the immune system produces antibodies that stimulate the thyroid gland to produce more of its own hormones, leading to hyperthyroidism. The aim of this case report is to highlight the potential for this complication during the treatment of multiple sclerosis with alemtuzumab, a humanized monoclonal antibody against CD52.

Case report: We present the case of a 46-year-old male patient with multiple sclerosis diagnosed in 2009, who was on alemtuzumab therapy. In February 2019, 25 months after the second cycle of alemtuzumab, the patient was diagnosed with Graves' disease due to elevated fT3 (>30.72 pmol/L) and fT4 (28.97 pmol/L) levels, suppressed TSH (<0.004 mIU/L), and positive TSH receptor antibodies (19.9 U/L). Ultrasound showed a normal-sized hypoechogenic thyroid gland. Clinically, the patient reported weight loss, sweating, and palpitations. He then started thyrostatic therapy with thiamazole. In August 2021, he was hospitalized due to persistent hyperthyroidism, and after the decision on more radical treatment, he received radioiodine therapy (RAI) in a dose of 5.15 mCi I-131. Unfortunately, his symptoms did not subside, and he was hospitalized again one year later, but additional RAI was postponed due to a multiple sclerosis relapse. After another admission in December 2022, additional RAI was administered in a dose of 7.5 mCi I-131, which he tolerated well. The patient is now on levothyroxine replacement therapy due to the resulting hypothyroidism, and his thyroid hormones are within normal range.

Conclusion: This case highlights the importance of regular monitoring of thyroid function in patients treated with alemtuzumab and the necessity for close collaboration between thyroidologists and neurologists in making therapeutic decisions.

Keywords: Alemtuzumab; Graves Disease; Multiple Sclerosis

CR51

Primary Mediastinal Large B-Cell Lymphoma Refractory to Therapy: A Case Report of CAR-T Selection

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Introduction: Primary mediastinal large B-cell lymphoma (PMBL) is a rare subtype of non-Hodgkin lymphoma that typically occurs in young adults (median age 35 years), with a slight female predominance. Chimeric antigen receptor T-cell (CAR-T) therapy is an adoptive immunotherapy in which autologous T lymphocytes are genetically modified *ex vivo* and reinfused to selectively eliminate tumor cells. We present a case highlighting therapeutic challenges and limitations of CAR-T therapy in refractory PMBL with central nervous system (CNS) involvement and cardiac infiltration.

Case report: A 19-year-old female was diagnosed with bulky PMBL with high-risk clinical and pathological features. Despite intensive immunochemotherapy (R-DA-EPOCH, R-CHOEP, high-dose methotrexate), radiotherapy and salvage regimens, the disease remained refractory with rapid progression. Due to CD30 positivity, she was referred for CAR-T cell therapy with tisagenlecleucel. Leukapheresis was successfully performed; however, rapidly progressive disease required bridging therapy with pembrolizumab. At that time, tumor infiltration involved mediastinal structures, the myocardium and the CNS. Extensive extranodal spread significantly limited CAR-T eligibility, as CNS infiltration increases the risk of severe immune effector cell-associated neurotoxicity, while cardiac involvement raises concerns regarding hemodynamic instability during cytokine release syndrome. Tumor infiltration of the heart with suspected embolization further reflected aggressive disease biology. Despite multidisciplinary management and supportive care, the patient developed acute respiratory failure and circulatory collapse, resulting in fatal outcome before CAR-T cell infusion.

Conclusion: Refractory PMBL represents a biologically aggressive entity requiring individualized treatment strategies. Although CD30 positivity enabled consideration of targeted and cellular therapy, advanced extranodal dissemination, CNS involvement and cardiac infiltration substantially limited significantly limited the feasibility and safety of CAR-T therapy.

Keywords: Central nervous system; Lymphoma, Large B-Cell, Diffuse; Mediastinum; Tisagenlecleucel

CR52

Twin Pregnancy in a Patient with Breast Cancer: A Case Report

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Introduction: Breast cancer (BC) ranks as the most prevalent cancer among women with approximately 5-10% of cases being genetically inherited. Many women encounter difficulties conceiving and carrying to term after undergoing BC treatment.

Case report: In 2008, a 28-year-old woman was diagnosed with grade III (G3), estrogen receptor (ER) low BC with 10% ER and 10% progesterone receptor (PR) expression together with human epidermal growth factor receptor 2 (HER2) negative expression. She underwent breast-conserving surgery and axillary dissection followed by adjuvant chemotherapy (6 cycles of doxorubicin and cyclophosphamide) and radiotherapy. Additionally, she was treated with antihormonal therapy (AT) with goserelin (luteinizing hormone–releasing hormone analog) and tamoxifen (selective estrogen modulator) for 4 years. In 2013, she paused AT to get pregnant and in 2015 she gave birth to healthy twins via in vitro fertilization. Due to positive family history, her mother died of ovarian cancer, she was genetically tested, and germline breast cancer gene 1 (*BRCA1*) mutation was found. In 2019, she had a miscarriage and a month later her breast cancer reappeared, but this time it was G3, triple negative BC (ER, PR and HER2 negative). She completed four cycles of docetaxel and carboplatin neoadjuvant chemotherapy which resulted in complete pathological response. Due to *BRCA1* mutation, she underwent bilateral mastectomy followed by breast reconstruction and prophylactic adnexectomy and is currently disease free.

Conclusion: This case highlights that pregnancy is feasible for young *BRCA* mutation carriers after breast cancer therapy. It adds to the existing evidence suggesting that, when carefully monitored and properly managed, post-treatment pregnancy does not adversely affect cancer prognosis.

Keywords: Breast Neoplasms; Genetic Testing; Mastectomy; Pregnancy, Twin

CR53

Diagnostic Challenges in Muckle–Wells Syndrome: A Case Report

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Introduction: Muckle-Wells syndrome (MWS), a subtype of cryopyrin-associated periodic syndromes, is an autoinflammatory disorder caused by *NLRP3* gene mutations. It is characterized by periodic fevers, chronic urticaria, arthralgia, progressive hearing loss, and amyloidosis. This case report aims to highlight the diagnostic challenges of MWS in a setting with limited access to comprehensive genetic testing, such as Croatia.

Case Report: A 56-year-old male patient was referred for the evaluation of chronic urticaria. Urticarial lesions were non-pruritic, appeared daily in the evening, and resolved spontaneously in the morning. Additional symptoms included symmetric talocrural arthralgia, bilateral deafness, microcytic anemia, elevated sedimentation rate and C-reactive protein. Symptoms had persisted for four years. Family history was unremarkable. The patient underwent an extensive hematological, infectological, dermatological, and immunological investigation, including negative antinuclear antibodies and normal complement (C3 and C4) levels. Skin biopsy was inconclusive, showing differential diagnostic features of vasculitis and neutrophilic dermatosis, and clinical–pathological correlation was therefore required. The tonal audiogram revealed sensorineural hearing loss. He was treated with antihistamines, glucocorticoids, leukotriene receptor antagonists, sulfasalazine, and methotrexate without improvement. Standard genetic testing for common *NLRP3* mutations was negative. However, considering the characteristic clinical presentation, poor response to conventional therapies, and known limitations of routine genetic testing, a clinical diagnosis of MWS was established. He has been treated with the interleukin-1 receptor antagonist anakinra since, and his symptoms completely resolved, except for permanent hearing loss.

Conclusion: MWS is rare and often remains undiagnosed. This case shows the importance of clinical recognition of autoinflammatory syndromes even when standard genetic testing is inconclusive. It also demonstrates the need for the availability of advanced genetic diagnostics to facilitate earlier diagnosis and treatment.

Keywords: Cryopyrin-Associated Periodic Syndromes; Genetic Testing; Interleukin 1 Receptor Antagonist Protein

CR54

Deciding on a priority: a case report of neuromyelitis optica spectrum disorder with multiple positive serological tests

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Introduction: Neuromyelitis optica spectrum disorder (NMOSD) is associated with aquaporin-4 immunoglobulin G antibodies (AQP4-IgG), a key diagnostic biomarker. Thorough patient evaluation is necessary, as therapeutic strategies for NMOSD target B lymphocytes.

Case report: A 63-year-old patient initially presented with left-sided retrobulbar neuritis in 2010 and was treated with pulse corticosteroid therapy. A relapse in 2017 was accompanied by progressive left leg paresis. Magnetic resonance imaging revealed multiple nonspecific brain lesions and spinal cord lesions at the C3–C6 level. NMOSD was suspected and subsequently confirmed by positive AQP4-IgG antibodies, while MOG antibodies were negative. Two additional relapses with left-sided weakness and sphincter dysfunction were treated with corticosteroids. The patient developed deep vein thrombosis, complicating further immunosuppressive treatment. Complexity of the clinical course and associated comorbidities made rituximab initiation a therapeutic challenge. As part of serological screening; anti-Borellia burgdorferi IgG, HIV Ag/Ab combined test, HIV antibody, anti-Toxoplasma IgG and anti-Rubella IgG were found positive indicating a possibility of latent infection or patient's previous exposure to the pathogens. Following a reactive HIV screening assay, confirmatory testing was undertaken. HIV RNA was undetectable and confirmatory serology was negative, consistent with a false-positive screening result in the context of autoimmune disease. An infectious disease specialist was consulted. Taking into account patients disease progression, hepatitis B vaccine was advised but not prioritised over immunotherapy. The patient was deemed eligible for an initial dose of rituximab. Regular follow ups with microbiological testing are scheduled and the immunotherapy regime will be adjusted accordingly.

Conclusion: Interpreting reactive serology in immune-mediated disease is challenging; accurate interpretation is essential to mitigate the risk of infection reactivation during immunosuppressive therapy, requiring a multidisciplinary team.

Keywords: Demyelinating Diseases; Rituximab; Serology

CR55

Giant Cell Tumor of Bone Mimicking Osteosarcoma: Diagnostic and Therapeutic Implications

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Introduction: Giant cell tumor of bone (GCTB) is a generally benign but locally aggressive primary bone tumor that typically affects the epiphysis of long bones in young patients. Radiological findings may overlap with malignant bone tumors, particularly osteosarcoma, making histopathological diagnosis essential. The aim of this case report is to highlight the diagnostic challenges in differentiating GCTB from malignant bone tumors and to emphasize the role of molecular confirmation in guiding targeted therapy.

Case report: A 9-year-old pediatric patient presented with intermittent pain in the right knee following a low-energy fall. Initial radiological evaluation revealed a cystic lesion in the distal right femur. Bone scintigraphy and magnetic resonance imaging raised suspicion of a primary malignant bone tumor measuring 76x47x32 mm. An open biopsy of the distal right femur was performed. Histological examination revealed tumor tissue composed of a fibrohistiocytic stroma without cytological atypia, containing numerous uniformly distributed multinucleated osteoclast-like giant cells. Areas of hemorrhage with erythrocyte extravasation and focal bone fragments were present. Immunohistochemically, mononuclear tumor cells were positive for H3.3 G34W, confirming the diagnosis of GCTB and supporting the presence of the mutation in the *H3F3A* gene. The patient was treated with nine cycles of denosumab as targeted therapy, leading to significant tumor reduction, and subsequently underwent resection of the distal femur with implantation of a right knee endoprosthesis.

Conclusion: This case illustrates the importance of integrating histopathological and molecular findings in the diagnosis of bone tumors with overlapping radiological features. Accurate histopathological evaluation of an early biopsy specimen and multidisciplinary collaboration are essential, as they directly influence patient management.

Keywords: Bone Neoplasms; Giant Cell Tumor of Bone; Immunohistochemistry

CR56

Tailored to patient's needs: A case of difficult-to-treat rheumatoid arthritis managed with five sequential biologic therapies

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Introduction: Rheumatoid arthritis (RA) is a systemic autoimmune disease with progressive joint involvement. In this case report, a patient with refractory RA was treated sequentially with upadacitinib (Janus kinase inhibitor), etanercept and infliximab (Tumor Necrosis Factor- α pathway blockade), tocilizumab (Interleukin-6 receptor inhibitor), and rituximab (Cluster of Differentiation 20-positive B-cell depletion), illustrating the therapeutic challenges in disease control.

Case report: The patient is a 67-year-old woman followed for RA since 1999, with involvement of small joints of the hands, elbows, temporomandibular joints, and costosternal joints, and seropositive status (rheumatoid factor [RF+] and anti-cyclic citrullinated peptide antibodies [anti-CCP+]). Initial treatment included sulfasalazine, followed by methotrexate (MTX) and antimalarial therapy with glucocorticoids. MTX and antimalarials were discontinued due to intolerance and chloroquine maculopathy, respectively. Subcutaneous MTX was introduced in 2019, but disease activity progressed. Upadacitinib was briefly used without benefit and discontinued with MTX due to transaminase elevation and leukopenia. Etanercept, started in 2021, produced transient improvement, followed by secondary loss of response. Infliximab began in 2022 but was stopped due to infusion reaction and refractory hypertension. Subsequent therapy with tocilizumab failed to achieve adequate disease control. Given persistent high disease activity, treatment was switched to rituximab in April 2025, resulting in a good clinical response, with continuation planned every six months.

Conclusion: This case highlights the need for individualized treatment strategies in long-standing RA. Multiple conventional, targeted, and biologic therapies were limited by inadequate response or intolerance. Although biologic therapies with different mechanisms are commonly used in RA, resistance to multiple distinct mechanisms, as seen here, is rare. Tailoring therapy to the patient's clinical course ultimately achieved effective disease control.

Keywords: Arthritis, Rheumatoid; Biological Therapy; Disease Progression

CR57

The road from genotyping to treatment: Personalized mavacamten therapy in a slow metabolizer with hypertrophic obstructive cardiomyopathy

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Introduction: Hypertrophic Obstructive Cardiomyopathy (HOCM) is a genetic cardiac condition defined by left ventricular hypertrophy and is often associated with left ventricular outflow tract (LVOT) obstruction. Novel treatment includes mavacamten, a selective inhibitor of cardiac β -myosin ATPase that reduces myocardial hypercontractility and is primarily metabolized in the liver by the cytochrome P450 2C19 (CYP2C19) enzyme. Therefore, mavacamten's efficacy relies on sufficient CYP2C19 enzymatic function.

Case report: We present a case of a 58-year-old female patient hospitalized due to dyspnea, palpitations, and chest pain with a significant troponin increase. During admission, atrial fibrillation paroxysms were detected and converted to sinus rhythm with amiodarone. Echocardiography revealed HOCM with preserved left ventricular systolic function, presence of systolic anterior motion with basal septum thickness of 22 mm, mild mitral and tricuspid regurgitation, and degenerative atrioventricular sclerosis with normal overall aortic valve function. LVOT peak pressure gradient was measured at 120 mmHg. Coronary angiography excluded coronary artery disease. Cardiac magnetic resonance imaging confirmed the HOCM diagnosis. The patient experienced secondary bradycardia with beta blocker rhythm control therapy and was placed on amiodarone 200 mg daily instead. Pharmacogenetic testing of the *CYP2C19* gene polymorphism was performed, classifying the patient as a slow mavacamten metabolizer. Consequently, initial mavacamten dose was adjusted to patient's genetic traits and set at 2.5 mg daily. At a four-week follow-up, overall clinical improvement was observed and supported by echocardiographic findings of improved LVOT peak pressure gradient at 53 mmHg.

Conclusion: Pharmacogenetics plays a crucial role in modern cardiovascular pharmacology and personalized medicine. In this case of mavacamten, pharmacogenetics allows dose personalization and reduces the risk of therapy-related adverse side effects, laying the foundation for enhanced therapeutic safety.

Keywords: Cardiomyopathy; Hypertrophic; Pharmacogenetics; Precision Medicine

CR58

Severe Growth Discordance in a Bichorionic Twin Pregnancy Caused by a De Novo CACNA1D Variant: A Case Report

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Introduction: Discordant fetal growth in twin pregnancies represents a complex clinical entity with a broad differential diagnosis. When conventional prenatal cytogenetic testing is normal, rare monogenic disorders may remain unrecognized despite severe and progressive phenotypic manifestations. Recent advances in genomic diagnostics allow deeper insight into such conditions and support a personalized approach to perinatal and neonatal management.

Case report: A 34-year-old primigravida with a dichorionic twin pregnancy conceived by *in vitro* fertilization (IVF) was admitted at 26 weeks' gestation due to growth discordance. Maternal hypothyroidism was treated with levothyroxine. First-trimester non-invasive prenatal testing (NIPT) indicated increased risk for trisomy 13 in one fetus. Amniocentesis, karyotyping, and chromosomal microarray were normal in both twins. Mid-pregnancy ultrasound revealed marked hypertrophy (>95th centile), polyhydramnios, and gastrointestinal wall thickening in one twin, while the co-twin showed normal growth with oligohydramnios. Serial Doppler assessments showed progression of polyhydramnios and reversed end-diastolic flow in the umbilical artery, prompting therapeutic amnioreduction and preterm delivery by caesarean section at 31 weeks. Postnatally, the hypertrophic male neonate presented with macrosomia, dysmorphic features, hyperinsulinemic hypoglycemia, seizures, left ventricular hypertrophy, and rapid multi-organ dysfunction despite intensive care. Trio-based genetic testing (affected neonate and both parents) identified a heterozygous de novo likely pathogenic variant in the *CACNA1D* gene, absent in the co-twin.

Conclusion: In cases of severe twin growth discordance with negative standard cytogenetic testing, early comprehensive genomic analysis should be considered. In this patient, identification of a de novo *CACNA1D* variant clarified the etiology and multisystem presentation, and informed postnatal management, prognosis, and genetic counseling by confirming a sporadic event without increased recurrence risk.

Keywords: Fetal Macrosomia; Mutation; Pregnancy, Twin

CR59

When Immunotherapy Spreads: Systemic BCG-itis After Intravesical Treatment for Urothelial Carcinoma

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Introduction: Intravesical *Bacillus Calmette-Guérin* (BCG) therapy is a cornerstone in the management of non-muscle-invasive bladder carcinoma and is generally well tolerated. Although most adverse effects are mild and confined to the urinary tract, rare cases of systemic dissemination of *Mycobacterium bovis* may occur, resulting in systemic BCG-itis with potentially severe consequences. We describe a case of disseminated BCG infection following intravesical BCG therapy, emphasizing diagnostic and management challenges.

Case report: A 79-year-old man with high-grade pT1 urothelial carcinoma of the bladder was treated with transurethral resection followed by intravesical BCG therapy. After the sixth BCG instillation, he developed persistent fever, chills and fatigue that did not respond to antipyretic therapy, leading to hospital admission. Severe acute respiratory syndrome coronavirus 2 infection was confirmed by polymerase chain reaction (PCR), and antiviral therapy together with broad-spectrum antibiotics was initiated; however, the patient's condition deteriorated. Laboratory investigation revealed CRP 100.5 mg/L, leukocytes $2.4 \times 10^9/L$, hemoglobin 118 g/L, platelets $107 \times 10^9/L$, AST 188 U/L, ALT 102 U/L and albumin 30.3 g/L, with hepatosplenomegaly. Blood and urine cultures were initially negative. Computed tomography demonstrated hepatosplenomegaly without infection. Given persistent fever after BCG instillation, disseminated BCG infection was suspected, while bacterial sepsis, viral infection and drug-related fever were excluded. PCR analysis of bone marrow aspirate detected *Mycobacterium bovis* BCG, and urine cultures confirmed growth of pyrazinamide-resistant *M. bovis*. Treatment with isoniazid, rifampicin and ethambutol, together with corticosteroids for suspected systemic inflammatory reaction, resulted in gradual clinical and laboratory improvement. The patient was discharged with continued outpatient therapy.

Conclusion: This case underscores the importance of early clinical suspicion for systemic BCG-itis in patients presenting with unexplained fever after intravesical BCG therapy, as delayed diagnosis may lead to morbidity.

Keywords: Fever of Unknown Origin; Iatrogenic Disease; *Mycobacterium bovis*; Urinary Bladder Neoplasms

CR60

From genome to vision: iris and retinal coloboma with visual impairment in an MAB21L2 gene mutation

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Introduction: Chorioretinal coloboma is a rare developmental defect occurring during embryogenesis. Affected patients typically present with reduced vision, an increased risk of retinal detachment, iris coloboma, nystagmus, and strabismus. *MAB21L2* encodes a protein involved in the regulation of eye and skeletal development. We report a patient with ocular coloboma carrying a rare, previously unreported *MAB21L2* variant and describe the associated phenotype.

Case report: We present a 16-month-old boy born at term after a normal pregnancy, without perinatal risk factors, who was referred for evaluation due to iris coloboma. Family history revealed that the patient's mother had a cleft palate, iris coloboma, and nystagmus. At the time of examination, the patient showed normal psychomotor development, with no additional developmental defects, although difficulty with visual tracking was observed. Ophthalmological examination indicated the presence of bilateral iris colobomas, a smaller corneal diameter, and colobomas in the lower part of the retina with borderline macular involvement. Given the hereditary nature of the condition, genetic testing was performed using an NGS-based multigene panel targeting microphthalmia, anophthalmia, and anterior segment dysgenesis. A heterozygous, likely pathogenic variant in the *MAB21L2* gene, (c.152G>A) was detected.

Conclusion: The identified variant is absent from the Genome Aggregation Database, and in silico analyses predict a deleterious effect on protein function. This finding explains the phenotype in both the patient and his mother and confirms hereditary transmission. The detection of this novel *MAB21L2* variant has important clinical implications, including genetic counselling, assessment of recurrence risk, and consideration of prenatal genetic diagnosis in future pregnancies. Early molecular diagnosis also enables appropriate ophthalmological follow-up due to the risk of retinal complications.

Keywords: Coloboma; Congenital Abnormalities; Eye Abnormalities; Mutation

CR61

Therapeutic Challenges and Risk-Benefit Analysis of Biological Therapy Side Effects in Plantar Psoriasis Management: A Case Report

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Introduction: Bimekizumab is a humanized monoclonal antibody that selectively targets Interleukin-17A and Interleukin-17F, approved for the treatment of moderate to severe psoriasis vulgaris in adults eligible for biological therapy.

Case report: A 56-year-old woman first presented to the Department of Dermatology and Venereology in 2018 with persistent plantar lesions, nail changes and auricular desquamation. She was treated with topical corticosteroids and acitretin, discontinued due to hyperlipidemia. Methotrexate was also stopped because of an inadequate therapeutic response. In March 2024, bimekizumab was initiated according to the standard dosing regimen, leading to complete remission. In December 2024, she developed bilateral otitis externa with persistent discharge and scaling, treated by an otorhinolaryngologist. In July 2025, her condition worsened with the development of hearing impairment. Dermatological examination revealed an alopecic scalp patch with pityriasiform scaling and purulent papules in the gluteal and inguinal regions. Microbiological cultures isolated *Staphylococcus aureus* from the scalp, *Proteus mirabilis* from the gluteal region, and *Proteus mirabilis* and *Candida albicans* from both external auditory canals. The patient was treated with systemic antibiotics, antifungal therapy and topical treatment. Bimekizumab provides dual inhibition of IL-17A and IL-17F. These cytokines are key “soldiers” of the immune system against fungal and certain bacterial infections, explaining the adverse effects observed in this patient. Finally, following consultation with a clinical pharmacologist, recurrent cutaneous infections were associated with bimekizumab therapy, and treatment was stopped.

Conclusion: Understanding the side effects of biological therapies is crucial for effective clinical management. This case underscores the importance of monitoring patients receiving biological therapy and the need for multidisciplinary follow-up, with timely discontinuation if adverse effects occur. The decision to continue or stop treatment must be well communicated with the patient.

Keywords: Bimekizumab; Biological Therapy; Infections; Psoriasis

CR62

Incidental PQ Prolongation Revealing a Pathogenic LMNA Mutation: A Case Report

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Introduction: PQ prolongation, also known as first-degree atrioventricular block, reflects delayed conduction between the atria and ventricles. It is often benign, but can be associated with heart disease or medication effects. Cardiomyopathy is a group of diseases affecting the heart muscle, leading to structural and functional abnormalities. It may cause arrhythmias or progress to heart failure.

Case report: A 40-year-old male was referred for cardiological evaluation after incidental detection of PQ interval prolongation during a routine health check. Electrocardiography revealed sinus bradycardia with a horizontal electrical axis. Holter monitor showed a 7% supraventricular extrasystole burden, while echocardiography excluded structural heart disease. The patient was asymptomatic and tolerated physical activity well. Importantly, at the age of 50, his father developed PQ prolongation, which was followed by total atrioventricular block and ventricular tachycardia two years later and, ultimately, heart transplantation due to advanced dilated cardiomyopathy. Given the family history, genetic testing was performed, revealing a pathogenic mutation in the *LMNA* gene. Considering the genetic findings, high-risk family background, and an *LMNA*-risk score of 13% for life-threatening ventricular tachyarrhythmias at five years, implantation of a dual-chamber implantable cardioverter-defibrillator was indicated for primary prevention of sudden cardiac death. Genetic testing of the patient's children is planned.

Conclusion: *LMNA* mutations are an important cause of dilated cardiomyopathy. Conduction disorders, which often precede the development of dilated cardiomyopathy, and ventricular arrhythmias, whose risk is independent of the degree of systolic dysfunction, often present years before heart failure. This case highlights the importance of clinical context (e.g. family history and patient's age) in assessing patients with usually benign conditions like PQ prolongation. Appropriate genetic testing can help early risk stratification and intervention even in asymptomatic patients.

Keywords: Atrioventricular Block; Cardiomyopathies; Electrocardiography; Genetic Testing; Heart Transplantation

CR63

A Rare Pulmonary Adenocarcinoma Variant: A Case of Fetal Adenocarcinoma

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Introduction: Fetal adenocarcinoma of the lung is a rare variant of pulmonary adenocarcinoma that resembles the pseudoglandular stage of fetal lung development, and may be difficult to diagnose on limited preoperative samples. This case report emphasizes its diagnostic complexity and key histomorphological, immunohistochemical, and molecular features.

Case report: A 63-year-old woman underwent chest computed tomography during preoperative evaluation for a benign gynecological condition, which incidentally revealed a solitary 33 mm lesion in the right upper lung lobe. Positron emission tomography/computed tomography demonstrated increased metabolic activity. The patient underwent right upper lobectomy with mediastinal lymphadenectomy. Grossly, the tumor measured 35 × 35 × 32 mm and was well circumscribed. It consisted of papillotubular and cribriform structures lined by pseudostratified columnar cells with clear to mildly eosinophilic cytoplasm, mild cytological atypia and low mitotic activity. Tumor cells expressed Thyroid Transcription Factor-1, confirming primary pulmonary origin, and were negative for Estrogen Receptor and Paired Box Gene 8, excluding a metastasis of gynecologic primary tumor. Membranous and cytoplasmic β -catenin expression was present, indicating activation of the Wnt/ β -catenin signaling pathway and supporting the diagnosis of fetal-type adenocarcinoma. Programmed Death-Ligand 1 expression was <1%. Lymph nodes were free of metastasis. Next-generation sequencing identified CTNNB1 and PIK3CA mutations. CTNNB1 mutation correlates with β -catenin pathway activation, while PIK3CA mutation suggests PI3K/AKT pathway involvement and potential therapeutic relevance. The final stage was pT2N0 (stage IB). Based on multidisciplinary team consensus, no adjuvant therapy was administered, and the patient remains disease-free.

Conclusion: Accurate diagnosis of fetal adenocarcinoma requires integration of morphology, immunohistochemistry, and molecular findings, as its distinct biology may influence management strategies.

Keywords: Adenocarcinoma; Immunohistochemistry; Lung Neoplasms

CR64

Congenital abdominal wall varicosities as a manifestation of Klippel–Trenaunay syndrome

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Introduction: Klippel-Trenaunay syndrome is a congenital disorder characterized by abnormal development of blood vessels, soft tissues, bones, and, in some case, the lymphatic system.

Case report: A case of a 15-year-old boy with congenital varicose veins of the abdominal wall is described, showing increasing prominence with age. The patient had no other symptoms, and physical examination revealed a soft, non-tender abdomen. Computed tomography (CT) angiography showed agenesis of the left common femoral vein and the infrarenal portion of the inferior vena cava, along with dilated and tortuous collateral veins in the abdomen and pelvis. Additionally, CT angiography revealed hypoplasia of the left renal vein, atrophy of the left kidney, and compensatory hypertrophy of the right kidney. The patient was referred to pediatric nephrology, where a diagnosis of Klippel–Trenaunay syndrome was established. Renal scintigraphy showed a morphologically normal right kidney and a hypoplastic left kidney. Differential renal function assessed by dimercaptosuccinic acid (DMSA) scintigraphy was 76% for the right kidney and 24% for the left kidney. Laboratory findings demonstrated elevated levels of urea, urate, and markers of impaired renal function. Magnetic resonance imaging confirmed that the left kidney was smaller, measuring 7.6 cm in length, with a homogeneously reduced renal parenchymal mass. The patient was advised to modify his dietary habits and to undergo regular clinical and laboratory follow-up.

Conclusion: This case report highlights an atypical presentation of Klippel-Trenaunay syndrome, manifesting primarily as congenital abdominal wall varicosities. Despite minimal clinical symptoms, advanced imaging revealed significant vascular and renal abnormalities, emphasizing the importance of radiological evaluation in patients with congenital vascular anomalies.

Keywords: Congenital Abnormalities; Klippel-Trenaunay Syndrome; Vascular Malformations

CR65

Malignant hyperthermia: a challenge in anesthesia

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Introduction: Malignant hyperthermia (MH) is a rare, potentially fatal, autosomal dominant disorder of skeletal muscle metabolism, typically triggered by volatile anesthetics or depolarizing neuromuscular blockers such as succinylcholine. The disorder is characterized by uncontrolled intracellular calcium release, leading to hypermetabolism, muscle rigidity, rapid hyperthermia, acidosis, and cardiovascular instability. This case report highlights the importance of identifying MH during general anesthesia, even in emergency settings.

Case report: A 56-year-old male was admitted for emergency surgical repair of forearm tendon lacerations sustained while working in his garden. General anesthesia was administered with sevoflurane and succinylcholine. During surgery, a progressive rise in body temperature was observed, prompting intraoperative suspicion of malignant hyperthermia. Postoperatively, the patient failed to regain consciousness and developed severe hyperthermia, tachycardia, and hemodynamic instability. Pulseless ventricular tachycardia progressed to asystole. Immediate cardiopulmonary resuscitation and aggressive cooling measures were initiated; however, the patient died within 20 minutes. Laboratory evaluation was not possible perioperatively due to the rapid deterioration. Autopsy revealed skeletal muscle hemorrhages, pulmonary edema, myocardial fiber fragmentation, hepatic and renal cellular changes, and cerebral edema, all consistent with a hypermetabolic state. Several years later, a close relative tested positive for MH susceptibility, confirming the hereditary nature of the disorder.

Conclusion: This case underscores the need to consider MH in patients exposed to triggering anesthetics, recognize early warning signs, and initiate prompt interventions. It also emphasizes the importance of family screening to prevent similar fatal outcomes. Awareness and preparedness among anesthesiologists are essential to improve patient safety and outcomes.

Keywords: Anesthesia, General; Malignant Hyperthermia; Sevoflurane; Succinylcholine

CR66

Chronic Lymphocytic Leukemia with chromosome 17p deletion: Ibrutinib-induced ventricular fibrillation and device-associated endocarditis management**Klara Grgorinić¹; Lucija Purgar²; Hana Matijaca³***1 - School of Medicine Zagreb, University of Zagreb, Zagreb, Croatia**2 - School of Dental Medicine, University of Zagreb, Zagreb, Croatia**3 - Clinical hospital center of Sisters of Mercy, Zagreb, Croatia*

Introduction: Chronic lymphocytic leukemia with chromosome 17p deletion or *TP53* mutation requires continuous therapy with Bruton tyrosine kinase inhibitors, according to European society for medical oncology guidelines. Ibrutinib is clinically effective but carries a risk of cardiotoxicity, including life-threatening ventricular arrhythmias, even in patients without structural heart disease. Differentiating iatrogenic from intrinsic cardiac pathology is clinically challenging but crucial for appropriate management.

Case report: A 53-year-old male with chronic lymphocytic leukemia with chromosome 17p deletion, started ibrutinib in April 2019. Transthoracic echocardiography and 24-hour Holter monitoring showed preserved left ventricular systolic function with an ejection fraction 60%, mild aortic and mitral regurgitation (Grade 1+), along with occasional ectopic beats (54 premature ventricular complexes and 4 supraventricular ectopic beats). These findings suggested a stable baseline cardiac status and supported the safe initiation of ibrutinib therapy. After four years of continuous ibrutinib therapy with stable hematologic response, the patient experienced acute ventricular fibrillation in April 2023. Immediate defibrillation successfully restored sinus rhythm. Coronary angiography showed non-obstructive right coronary artery stenosis, and echocardiography confirmed preserved ejection fraction ≥ 50 , excluding ischemic and structural changes. The presentation was therefore consistent with ibrutinib-induced arrhythmogenicity. A dual-chamber implantable cardioverter-defibrillator (ICD) was implanted for secondary prevention and therapy switched from ibrutinib to venetoclax, maintaining complete hematologic remission and favorable cardiovascular tolerance. Three months post-ICD implantation, pocket infection with lead-associated endocarditis developed, required device removal, targeted antibiotics, and reimplantation.

Conclusion: Ibrutinib-associated cardiotoxicity may evolve quietly over time, even in patients with normal baseline cardiac function. Early recognition of cardiotoxicity, careful risk assessment, and early transition to safer therapeutic alternatives are key elements in preventing catastrophic outcomes.

Keywords: Cardiotoxicity; Defibrillators, Implantable; Leukemia, Lymphocytic, Chronic, B-Cell; Tyrosine Kinase Inhibitors

CR67

Cerebral venous sinus thrombosis and pulmonary embolism: coincidence or shared etiology?**Vlatka Grozdanić¹; Jakov Jurlina¹; Barbara Ravlić²; Marija Perić Strenja³; Teodora Zaninović Jurjević³***1 - Faculty of Medicine Rijeka, University of Rijeka, Rijeka, Croatia**2 - Faculty of Medicine Osijek, University of J.J. Strossmayer, Osijek, Croatia**3 - Clinic for Cardiovascular Diseases, Clinical Hospital Center Rijeka, Croatia*

Introduction: Venous thromboembolism includes deep-vein thrombosis (DVT) and pulmonary embolism (PE), with PE ranging from low-risk to massive forms causing hemodynamic instability. Cerebral sinus thrombosis (CVT) is a relatively uncommon cerebrovascular condition characterized by occlusion of one or more cerebral veins and/or dural venous sinuses presenting with headache, seizures, or focal deficits. These two share the same pathophysiological basis and represent different localizations of the same systemic thrombotic tendency.

Case report: A 37-year-old woman presented to the emergency room in 2018 with a 20-day history of severe headache, nausea, right retro-orbital pain, and bilateral blurred vision. Cerebral magnetic resonance venography demonstrated partial thrombosis of multiple cerebral venous sinuses. Anticoagulation with low-molecular-weight heparin (LMWH) was initiated, and a thrombophilia work-up was performed. Testing for factor V Leiden and prothrombin gene mutations was negative but identified homozygous form of *PAI-1(4G/4G)* and heterozygous form of *MTHFR C677T*. Long-term anticoagulation with warfarin was initiated. Three years after, warfarin was discontinued due to patient's choice and replaced with dabigatran 150 mg twice daily. In 2024, she represented with hemoptysis, dyspnea at rest and exertion, and right-sided pleuritic chest pain. Computed tomography angiography revealed a right segmental pulmonary embolism while lower-limb Doppler ultrasonography showed no evidence of DVT. Following LMWH therapy, warfarin was reintroduced. Given the recurrent thrombotic events, antiphospholipid syndrome was suspected, with initial results showing a borderline positive anti- β 2-glycoprotein I IgM level (20.44 RU/mL), however, further evaluation is currently underway.

Conclusion: This case highlights the continuum of thrombosis, where CVT and PE may coexist over time. It emphasizes the need to consider etiology in recurrent events and highlights the critical role of appropriate, sustained anticoagulation in secondary prevention.

Keywords: Sinus Thrombosis, Intracranial; Thrombophilia; Venous Thromboembolism

CR68

Molecular and Clinical Profile of Rubinstein–Taybi Syndrome in Pediatric Patients

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Introduction: Rubinstein–Taybi syndrome is a rare genetic disorder affecting growth, development, and physical features, most commonly caused by mutations in the CREBBP gene. Due to its variable presentation, early recognition is essential for diagnosis and management.

Case report: We present a three-year-old girl born by uncomplicated vaginal delivery at 42 weeks. During the neonatal period, she was admitted for a perineal infection caused by *Acinetobacter junii*, successfully treated with meropenem and amikacin. Intrauterine growth restriction and dysmorphic features were observed, including broad and adducted toes, rocker-bottom feet, a saddle-shaped nasal bridge, high-arched palate, elongated philtrum, micro- and retrognathia, and hypertrichosis. Cardiac evaluation revealed a secundum-type atrial septal defect and a persistent wide ductus arteriosus, successfully closed medically with ibuprofen and digitalis. Brain ultrasonography showed agenesis of the corpus callosum. Hematological and biochemical parameters were normal for age. Physical examination revealed poor motor skills and axial hypotonia. At ten months, the traction test showed short-term sitting with trunk anteflexion and underdeveloped protective reactions. At nineteen months, she crawled with abducted knees and a low-lying pelvis, and at three years, she walked with support on a wide base. Genetic analysis identified a heterozygous CREBBP mutation (c.1063C>T, p.Gln355Ter), causing a premature stop codon and a truncated, non-functional protein. In addition to pharmacotherapy with diuretics and inotropes, neurorehabilitation including Bobath, Vojta, sensory integration, and inpatient balneorehabilitation was applied.

Conclusion: Early and consistent physical therapy is essential in managing hypotonic children, enhancing motor development, postural control, and functional skills, and significantly improving long-term outcomes through individualized rehabilitation and targeted therapeutic interventions.

Keywords: Muscle Hypotonia; Physical Therapy Modalities; Rubinstein-Taybi Syndrome

CR69

A Preventable Threat: Invasive Pneumococcal Disease in a Vaccine-Eligible Patient

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Introduction: Despite the availability of effective vaccines and well-established immunization recommendations, *Streptococcus pneumoniae* remains a major cause of severe invasive infections, largely due to suboptimal vaccination coverage among adults at increased risk. Invasive pneumococcal disease incidence is approximately 10–30/100,000 annually, with mortality 15–20%, exceeding 30% in older or comorbid adults. We report a case of life-threatening invasive pneumococcal disease in a vaccine-eligible patient, highlighting missed opportunities for prevention and the continued clinical impact of inadequate adult immunization.

Case report: A 61-year-old woman with multiple comorbidities, including insulin-dependent type 2 diabetes mellitus, arterial hypertension, chronic lymphocytic leukemia, and an ileostomy after colorectal surgery, presented with a five-day history of fever, productive cough, and progressive dyspnea. Initial evaluation on admission revealed hypoxemia, elevated inflammatory markers (C-reactive protein 72 mg/L, leukocyte count $14 \times 10^9/L$, neutrophils 90%), and right-sided pneumonia with pleural effusion on chest X-ray. Nasopharyngeal testing for SARS-CoV-2 was positive, and blood cultures subsequently revealed *Streptococcus pneumoniae*, confirming invasive pneumococcal disease. Besides symptomatic and supportive treatment, the patient was treated with ceftriaxone and remdesivir, with gradual clinical, laboratory, and radiological improvement, and was discharged after 9 days in stable condition with appropriate follow-up. Review of her preventive care revealed that, despite clear indications based on age and comorbid conditions, she had never received pneumococcal vaccination.

Conclusion: This case underscores the persistent burden of invasive pneumococcal disease in high-risk, vaccine-eligible adults and emphasizes the critical importance of systematic identification and immunization of vulnerable populations. Improving adult vaccination coverage is essential to reducing morbidity, healthcare utilization, and potentially life-threatening complications associated with invasive pneumococcal disease.

Keywords: Community-Acquired Pneumonia, *Streptococcus pneumoniae*, Vaccination, Vaccine-Preventable Diseases

CR70

From Genome to Phenotype: Non- Immune Fetal Hydrops Caused by 45,X Monosomy

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Introduction: Non-immune fetal hydrops represents a severe prenatal condition with heterogeneous etiology, increasingly linked to underlying genetic abnormalities. Monosomy X (45,X), classically associated with Turner syndrome, is among the most frequent chromosomal causes of early fetal hydrops due to lymphatic dysgenesis and cardiovascular malformations. We present a case of generalized fetal hydrops with a fatal outcome, highlighting the essential role of postmortem genetic analysis in establishing a definitive diagnosis.

Case report: A 32-year-old gravida 2, para 1 with an unremarkable personal and family history was referred to a fetal-maternal specialist at 21 weeks of gestational age due to suspected multiple fetal anomalies. Ultrasound revealed generalized fetal hydrops, including marked skin edema (>10 mm), ascites, pleural effusion, and a large septated cystic hygroma exceeding fetal body volume. Fetal echocardiography demonstrated a complex congenital heart defect. Maternal serology showed positive Parvovirus B19 IgM, while TORCH screening and immunohematological testing were negative. Findings were not consistent with isolated infectious etiology. Intrauterine fetal demise occurred at 22 weeks of gestational age. Post-abortion cytogenetic analysis using array comparative genomic hybridization (aCGH) identified loss of one X chromosome, confirming a 45,X karyotype.

Conclusion: This case highlights monosomy X as a critical genetic cause of early-onset non-immune fetal hydrops with poor prognosis. It underscores the importance of comprehensive genetic evaluation in cases of fetal hydrops, particularly when ultrasound findings suggest lymphatic and cardiac involvement, with aCGH analysis playing a key role in detecting chromosomal abnormalities not attributable to infectious causes alone. Integrating genomic diagnostics enables accurate etiological clarification, informed parental counseling, and optimized management of future pregnancies.

Keywords: Cytogenetic Analysis; Hydrops Fetalis; Turner Syndrome

CR71

Necrotizing Fasciitis of the Leg in a Diabetic Patient – A Case Report

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Introduction: Necrotizing fasciitis (NF) is a rare, life-threatening soft tissue infection characterized by rapidly progressive necrosis of the fascia and subcutaneous tissues. This case highlights the importance of early diagnosis and urgent therapeutic management in achieving optimal outcomes.

Case report: A 61-year-old male was admitted to hospital, with history of type 2 diabetes mellitus, arterial hypertension and atherosclerosis of peripheral arteries, presented with oedema and ulcer of the left foot, extending to the lateral parts of leg. One week before admission, he sustained a left ankle distortion and two days later, he developed diarrhoea, febrility and loss of appetite. Physical examination revealed erythema and oedema of the left lower leg and foot, with lymphedema and 10x15 cm necrotic ulcer of the foot, extending to the lateral parts of leg. Plain radiography showed no fractures, no further imaging was performed. Laboratory investigations demonstrated leukocytosis ($18 \times 10^9/L$), markedly elevated C-reactive protein (484 mg/L), and procalcitonin (3.7 $\mu g/L$). Due to suspected NF, urgent surgical intervention was performed, including incision and necrectomy of the left lower leg and foot. Intraoperative cultures identified beta-haemolytic group B *Streptococcus* (BHSB), *Candida albicans* and *Klebsiella pneumoniae*. Culture-guided antibiotic therapy including amoxicillin/clavulanate and clindamycin was initiated, targeting BHSB and *Klebsiella pneumoniae*, along with negative-pressure wound therapy and wound care that included debridement, toilette with antiseptic solutions and different medical wound dressings. After 20 days of hospitalization, laboratory values normalized and wound status improved, allowing discharge with home-based care. Follow-ups demonstrated gradual healing, with complete epithelialization after nearly one year.

Conclusion: This case illustrates the successful management of NF in a patient with multiple comorbidities, achieved through prompt debridement, appropriate antibiotic therapy, advanced wound care techniques and prolonged follow-up.

Keywords: Fasciitis, Necrotizing; Negative-Pressure Wound Therapy; Ulcer

CR72

Stevens-Johnson syndrome following pembrolizumab therapy in a 59-year-old patient with rectal adenocarcinoma: urgent management**Matija Herceg¹; Daška Štulhofer Buzina^{1,2}; Antonio Ivanac¹; Mislav Blažević¹; Niko Grubišić Neidhardt¹***1 - School of Medicine Zagreb, University of Zagreb, Zagreb, Croatia**2 - Department of Dermatovenereology, University Hospital Centre Zagreb, Zagreb, Croatia*

Introduction: Stevens-Johnson syndrome (SJS) is a rare, severe, and acute immune reaction characterized by diffuse skin and mucosal lesions, most commonly triggered by medications. Immune checkpoint inhibitors, such as pembrolizumab are increasingly used in oncology and are associated with immune-related adverse events, including severe cutaneous reactions. Antibiotics, antiepileptics, and nonsteroidal anti-inflammatory drugs remain well-known causes of SJS. The combined effect of these therapies may increase the risk of severe drug-induced skin reactions. This case report emphasizes the importance of prompt intervention in the treatment of SJS in oncologic patients receiving combined treatments.

Case report: We present a case of a 59-year-old male patient with a history of rectal adenocarcinoma, who underwent total neoadjuvant therapy, laparotomic tumor reduction surgery, and four cycles of adjuvant pembrolizumab. Immunotherapy was complicated by pulmonary embolism and ileus, necessitating an emergent bipolar ileostomy, after which postoperative antimicrobial prophylaxis was initiated. Six days after the introduction of ciprofloxacin, fluconazole, and metronidazole, the patient developed multiple erythematous bullae and vesicles accompanied by a strongly positive Nikolsky sign. The conjunctivae, oral and genital mucosa were also affected by erosions. Antimicrobial therapy was immediately discontinued, and the patient was managed with supportive care and systemic corticosteroids. The dermatohistopathological findings were consistent with toxic epidermal necrolysis. One week later, significant clinical improvement was observed with gradual resolution of skin and mucosal lesions.

Conclusion: This case highlights the diagnostic and therapeutic challenges in identifying the causative agent of SJS in patients receiving immune checkpoint inhibitors alongside other high-risk medications. Early diagnosis, prompt withdrawal of suspected agents, and multidisciplinary management are essential to improve outcomes.

Keywords: Adverse Drug Reaction; Antibiotics; Immune Checkpoint Inhibitors; Stevens-Johnson Syndrome

CR73

Unraveling the Indefinite Amiodarone-Induced Thyrotoxicosis: A Case Report

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Introduction: Amiodarone-induced thyrotoxicosis (AIT) is a significant adverse effect linked to amiodarone, an iodine-rich antiarrhythmic drug. AIT presents in two forms: type 1, associated with underlying thyroid disorders, and type 2, characterized by subacute thyroiditis. Thyrotoxicosis increases the risk of atrial fibrillation (AF), especially in elderly patients with cardiac conditions. This case report highlights the importance of thorough evaluation for thyrotoxicosis patients.

Case report: A 62-year-old male presented to the emergency room with fatigue, nausea, excessive sweating, myasthenia, and unintentional weight loss. Clinical examination showed hypotension, tachycardia, and AF. Laboratory tests revealed elevated anti-thyroid peroxidase (anti-TPO) antibodies and thyroxine levels, suggesting an autoimmune thyroid disorder. However, the patient was prescribed amiodarone a month prior for ventricular tachyarrhythmia, indicating possible AIT type 1. Yet, a thyroid ultrasound showed decreased vascularity, and the symptoms improved with prednisone therapy, indicative of subacute thyroiditis, common in AIT type 2. After discontinuing amiodarone, he was managed by cardiology and endocrinology and started on bisoprolol for ventricular rhythm control. As of January 2025, he experiences permanent AF but reports feeling well with occasional palpitations.

Conclusion: While amiodarone is a potent antiarrhythmic, it can stimulate the thyroid and lead to AF development, even in previously euthyroid patients. It is important to note that some patients may present characteristics of both type 1 and 2 AIT simultaneously. Although thyroid antibodies may be elevated, these patients still require corticosteroid therapy. Therefore, a coherent and multidisciplinary approach is essential for thyrotoxicosis treatment, to optimize therapy and ensure the early management of side effects.

Keywords: Amiodarone; Atrial fibrillation; Thyrotoxicosis

CR74

Precision in Practice: Tailored Surgical Management of a Bosniak III Renal Cyst

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Introduction: Precision medicine extends beyond molecular profiling and encompasses individualized clinical decision-making in the setting of diagnostic uncertainty. The management of Bosniak III renal cystic lesions exemplifies this challenge, as they represent a biologically heterogeneous group for which imaging alone cannot reliably predict malignant potential. This case report highlights how a patient-centered, robot-assisted organ-preserving surgical strategy can simultaneously address diagnostic ambiguity and optimize functional outcomes in contemporary urological practice.

Case report: We present a case of a 38-year-old male patient with no chronic medical conditions and a suspected penicillin allergy, incidentally, diagnosed with a complex cystic lesion of the upper pole of the right kidney. The patient was asymptomatic, and the lesion was detected during a routine abdominal ultrasound examination performed as a part of a general health check-up. Further diagnostic evaluation was conducted using computed tomography, confirming a 3.1 cm Bosniak III lesion of the upper pole of the right kidney. As papillary carcinoma couldn't be definitively excluded, a Da Vinci robotic-assisted partial nephrectomy was performed. The procedure was completed uneventfully, with preservation of renal function and minimal blood loss. Postoperatively, the patient experienced transient subileus, which was successfully managed conservatively. Histopathological examination confirmed a multilocular cystic renal neoplasm of low malignant potential (ISUP grade 2) with clear resection margins and no lymphovascular invasion. Follow-up ultrasonography and laboratory tests were unremarkable, and the patient reported excellent postoperative recovery.

Conclusion: The Da Vinci robotic system enables precise, personalized surgical intervention in the management of rare renal lesions, therefore preserving renal function, reducing perioperative trauma, and accelerating recovery. Integration of this minimally invasive technique exemplifies the potential of precision medicine to optimize patient outcomes and enhance surgical safety in contemporary urology.

Keywords: Kidney Neoplasms; Minimally Invasive Surgical Procedures; Nephrectomy; Robotic Surgical Procedures

CR75

Therapeutic challenges of immune checkpoint inhibitor therapy in squamous cell carcinoma with chronic inflammatory dermatosis

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Introduction: Squamous cell carcinoma (SCC) may form based on chronic inflammatory dermatoses, which often lead to a more aggressive disease course. Immunotherapy became standard treatment for advanced tumors, however, it may worsen the underlying autoinflammatory disease by disrupting immunoregulation.

Case report: We present a case of a 67-year-old man who has suffered from multiple myeloma and hidradenitis suppurativa for years, presenting with long-standing changes like abscesses and fistulas in the gluteal region. Firstly, he was treated with adalimumab without any significant improvement. The patient underwent multiple fistulotomies. SCC was detected in the gluteal, perianal, and perisacral regions with involvement of the iliac and inguinal lymph nodes. In February 2025, the multidisciplinary tumor board classified the tumor as unresectable and initiated cemiplimab. Upon deterioration, treatment was discontinued. A palliative colostomy and surgical intervention were recommended for quality-of-life improvement. Ulcerations developed on the gluteal region and the right upper leg, followed by repeated bleeding, burning sensation, and pain. The gluteus became erythematous with associated hypergranulations, fetor, fibrinous deposits, and numerous fistulas. Diverse bacterial species were isolated and treated parenterally, and multiple red blood cell transfusions were performed. A surgical procedure was planned in September. However, several complications emerged, including repeated bleeding, heart failure with preserved ejection fraction, and partial respiratory failure. Due to comorbidities, disease extent, and poor general condition, the surgical procedure and further oncological treatment were discontinued. In further management, symptomatic treatment and palliative care were indicated.

Conclusion: This case illustrates therapeutic challenges of advanced SCC arising in chronic inflammatory dermatosis. Although immunotherapy is standard for unresectable disease, its benefit may be limited by clinical deterioration and comorbidities. Individualized, multidisciplinary, palliative management with careful risk–benefit evaluation is recommended.

Keywords: Hidradenitis Suppurativa; Immunotherapy; Squamous Cell Carcinoma

CR76

Compressed, clotted, revealed: May–Thurner syndrome**Ante Ivić¹, Lucija Kadleček¹, Franjo Marković¹, Barbara Kokić¹, Željka Breškić Ćurić^{1,2}***1 - Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Cardiology, University Hospital Centre Osijek, Osijek, Croatia*

Introduction: Deep vein thrombosis (DVT) in young patients warrants evaluation for underlying anatomical and prothrombotic conditions. May–Thurner syndrome (MTS) is caused by extrinsic compression of the left common iliac vein by the right common iliac artery. This condition represents a significant risk factor for left-sided DVT, mostly in young women. This case shows a complex case of subacute iliofemoral DVT following orthopedic surgery, complicated by suspected MTS.

Case report: A 41-year-old woman was admitted with a 3–4-day history of pain, swelling, and discoloration of the left lower limb. Eight weeks earlier, she had surgical fixation of a trimalleolar ankle fracture. Ultrasound with compression revealed extensive subacute thrombosis involving left femoral, popliteal, profunda femoris, iliac veins, and the terminal segment of the great saphenous vein. Laboratory tests showed elevated inflammatory markers and mild anemia. Thrombophilia screening identified heterozygous MTHFR C677T and PAI-1 4G/5G polymorphisms, with a weak association with thrombosis risk, and negative Factor V Leiden and prothrombin G20210A mutations. Mechanical thrombectomy was performed achieving almost complete recanalization. Despite therapeutic low-molecular-weight heparin and mechanical thrombectomy, follow-up ultrasound demonstrated persistent extensive residual eccentric filling defects at the level of the left common iliac vein suggesting MTS. Later it was confirmed by Computed Tomography venography. Ultimately, the patient was discharged with oral anticoagulant therapy - enoxaparin two times daily. On her request patient will continue her treatment in KBC Zagreb.

Conclusion: This case emphasizes the importance of considering May–Thurner syndrome in patients presenting with left-sided DVT, especially in young females. Mechanical thrombectomy with endovascular stenting was performed, achieving almost complete recanalization. Early recognition and endovascular intervention can improve clinical outcomes.

Keywords: Anticoagulants; Thrombectomy; Venous Thrombosis

CR77

Minimally Invasive, Maximally Precise: Da Vinci–Assisted Laparoscopic Radical Prostatectomy in a Patient with Prostate Adenocarcinoma

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Introduction: Da Vinci robotic surgery is an innovative technique. It goes beyond conventional approaches by combining minimally invasive methods with three-dimensional visualization and enhanced surgical precision. This integration not only improves ergonomics for the surgeon but also helps to minimize perioperative blood loss, shorten hospital stays, and accelerate patient recovery. In Croatia, the Da Vinci has been predominantly applied in urology and its use in gynecological procedures is expected to expand soon.

Case report: We present a 72-year-old male admitted to the urology department due to elevated prostate-specific antigen PSA (7.5 µg/L). Multiparametric magnetic resonance imaging (mpMRI) revealed lesions at different locations with imaging features highly suggestive of prostate carcinoma. Histopathological examination following robot-assisted targeted and systematic transperineal prostate biopsy using the Mona Lisa Dx Biobot system revealed acinar adenocarcinoma, Gleason score 3+3=6, one positive core. The patient initially chose active surveillance. Follow-up MRI showed increased prostate volume (65 cm³), with the lesions still visible. PSA rose to 10.68 µg/L, prompting repeat biopsy, which confirmed acinar adenocarcinoma with five positive cores without lymphovascular or perineural invasion. After multidisciplinary consultation, Da Vinci robot-assisted laparoscopic radical prostatectomy without nerve-sparing or lymphadenectomy was performed. The intraoperative and postoperative courses were uneventful. The drain was removed on postoperative day 3 and the urinary catheter on day 5. The patient urinated normally without incontinence or macrohematuria. Pre-discharge abdominal and pelvic ultrasound showed no free fluid.

Conclusion: In this case, the Da Vinci system demonstrated high surgical precision and allowed for a reduced length of hospitalization. Although nerve-sparing techniques can preserve erectile function and promote early continence in selected patients, they were not applied in the current procedure.

Keywords: Adenocarcinoma; Biopsy; Prostatectomy

CR78

Laser Treatment for Refractory Epistaxis in a Patient With Hereditary Hemorrhagic Telangiectasia: A Case Report

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Introduction: Hereditary hemorrhagic telangiectasia (HHT; Rendu–Osler–Weber syndrome) is a rare autosomal dominant vascular disorder characterized by mucocutaneous telangiectasias and visceral arteriovenous malformations (AVMs). Spontaneous and recurrent epistaxis is the most common and earliest manifestation, often leading to frequent emergency room visits, chronic iron deficiency anemia and reduced quality of life. Treatment options include local and systemic conservative therapy and multiple surgical treatment modalities. In cases of severe, refractory epistaxis (transfusion-dependent disease), the angiogenesis inhibitor bevacizumab is used.

Case report: A 57-year-old male with a long-standing history of HHT and recurrent epistaxis presented with progressive disease requiring intravenous iron supplementation and multiple blood transfusions. After confirmation of HHT based on the Curaçao criteria (epistaxis, telangiectasias, visceral AVMs, and family history), systemic bevacizumab therapy (5 mg/kg biweekly for 12 weeks) was initiated, resulting in only partial symptomatic improvement and persistent anemia. Consequently, endoscopic laser coagulation of nasal telangiectasias was performed using a 940-nm diode laser. Following the procedure, the severity and frequency of epistaxis decreased markedly, with rare, brief episodes and normalization of hemoglobin levels for 13 months. After symptom recurrence, repeat laser treatment achieved sustained bleeding control, improvement of anemia, and elimination of transfusion dependence. No further interventions were required during the two years of follow-up.

Conclusion: This case highlights laser coagulation as an effective, mucosa-sparing, and repeatable surgical option for controlling severe HHT-related epistaxis and improving clinical outcomes and quality of life. It also represents the least aggressive surgical method to reduce the frequency and severity of bleeding.

Keywords: Epistaxis; Laser Therapy; Telangiectasia, Hereditary Hemorrhagic

CR79

The importance of antiviral prophylaxis for hepatitis B in patients receiving immunosuppressive therapy

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Introduction: Although immunosuppressive therapy (IST) represents a major therapeutic advance for various conditions, it is associated with an increased risk of reactivation of latent infections, including hepatitis B virus (HBV) infection. The aim of this case report is to highlight the importance of timely antiviral therapy in patients receiving IST.

Case report: A 64-year-old man was diagnosed with diffuse large B-cell non-Hodgkin lymphoma (DLBCL), and IST was initiated according to the standard treatment protocol. Prior to treatment, routine screening tests were performed, including HBV serology, which revealed positivity for hepatitis B surface antigen (HBsAg). According to current clinical guidelines, HBsAg-positive patients who require high-dose combination IST are at high risk of HBV reactivation and should receive prophylaxis with nucleos(t)ide analogues. However, further virological evaluation, including assessment of HBV deoxyribonucleic acid (DNA) levels, was not undertaken, and antiviral prophylaxis was not initiated. After four cycles of IST, the patient developed low-grade fever (37.3°C), mild jaundice, and hepatomegaly. Laboratory evaluation revealed severe hepatocellular injury, with markedly elevated transaminase levels (aspartate aminotransferase 1031 U/L, alanine aminotransferase 1288 U/L). Furthermore, HBV serological testing and polymerase chain reaction (PCR) detection of HBV DNA confirmed hepatitis B reactivation with positive viremia. Antiviral therapy with entecavir was initiated, resulting in gradual clinical and biochemical recovery.

Conclusion: This case report underscores the high risk of hepatitis B reactivation in HBsAg-positive patients undergoing immunosuppressive therapy. Timely HBV screening and initiation of antiviral prophylaxis in accordance with current clinical guidelines are essential to prevent severe, potentially life-threatening, yet preventable complications.

Keywords: Hepatitis B; Hepatitis B Surface Antigens; Immunosuppression Therapy; Lymphoma

CR80

How Many Infections Can an Immunocompetent Person Have Simultaneously?

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Introduction: Chronic respiratory symptoms, fever, and weight loss are a significant diagnostic challenge in elderly patients. Even immunocompetent individuals may develop concurrent or atypical infections, such as tuberculosis (TB) and cytomegalovirus (CMV). This requires a broad differential diagnosis to prevent delays in recognition and treatment.

Case report: A 76-year-old man was admitted to the University Hospital Center “Sestre milosrdnice” with chest pain, dyspnea, dry cough, fever, and an 8-kg weight loss over three months. He had a pacemaker placed in 2023, was diagnosed with late-onset asthma, and treated for pleuropneumonia. The pleural effusion was lymphocytic and exudative. Over the past two years, he experienced high fevers attributed to chronic prostatitis and urethral stricture, which responded well to fluoroquinolones. On admission, the physical examination was unremarkable; however, laboratory results revealed leukocytosis, neutrophilia, and a C-reactive protein of 104 mg/L. Computed tomography revealed a tree-in-bud pattern and centrilobular micronodules, mainly in the right upper lobe. Initial bronchoscopy was inconclusive, but repeated bronchoscopy detected CMV in bronchoalveolar lavage (BAL); Human Immunodeficiency Virus and hepatitis were excluded. Ganciclovir and cefuroxime were started, but switched to amikacin after *Pseudomonas aeruginosa* was found in urine. The patient showed clinical and biochemical improvement, although radiology results remained unchanged. Six weeks later, BAL cultures grew *Mycobacterium tuberculosis*, so standard quadruple antituberculous therapy (isoniazid, rifampin, ethambutol, pyrazinamide) was initiated. The patient tolerated treatment well, became asymptomatic, and showed significant radiological improvement.

Conclusion: This case illustrates that immunocompetent patients can have multiple chronic infections. The initial TB infection likely enabled CMV reactivation, while urethral stricture may have stemmed from genitourinary TB. Fluoroquinolones used for urinary symptoms potentially have temporarily suppressed TB, masking its presence and delaying diagnosis.

Keywords: Coinfection; Cytomegalovirus; Pneumonia; Tuberculosis; Urinary Tract Infections

CR81

When Pneumonia Does Not Respond: Invasive Aspergillosis in a Patient with Chronic Obstructive Pulmonary Disease

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Introduction: Invasive aspergillosis is a life-threatening fungal infection most commonly caused by *Aspergillus fumigatus*. It is rare in healthy individuals, but can occur in immunocompromised patients, including those with severe chronic obstructive pulmonary disease (COPD). This case highlights early recognition and treatment of invasive aspergillosis in COPD.

Case report: A 44-year-old male with COPD on inhaled corticosteroids and bronchodilators presented with a four-day history of dry cough and exertional dyspnea. Additional symptoms included mild headache, lower abdominal pain and leg cramps, one episode of vomiting, watery diarrhea and a fever up to 38.3 degrees Celsius. His medical history revealed a weakly positive cutaneous test for *Aspergillus* seven years earlier. Laboratory findings revealed leukocytosis with neutrophilia, elevated C-reactive protein and lymphopenia. Chest X-ray showed right-sided patchy infiltrates, most likely of inflammatory origin, with upper-lobe consolidation and hilar enlargement consistent with lymphadenopathy. Due to these findings, he was treated with moxifloxacin and ceftriaxone. His condition, laboratory results and chest X-ray worsened, leading to admission to the pulmonology ward after four days. His condition kept worsening and, eight days later, he was transferred to the intensive care unit being unresponsive, hypotensive and dyspnoic. He was immediately intubated. Bronchoalveolar lavage tested positive for *Aspergillus* galactomannan and voriconazole therapy was initiated, with subsequent improvement. One month after admission, he was discharged home. Treatment continued for four months, and he was ultimately referred for evaluation at a pulmonary transplant center due to advanced emphysema.

Conclusion: This case report emphasizes the importance of considering invasive aspergillosis as a potential, often underrecognized, cause of treatment-refractory pneumonia. Early recognition may reduce morbidity, healthcare costs, and severe outcomes.

Keywords: Aspergillosis; *Aspergillus*; Critical Care; Pulmonary Disease, Chronic Obstructive; Voriconazole

CR82

A Master of Disguise: Immunoglobulin G Kappa Multiple Myeloma Mimicking Lymphoplasmacytic Lymphoma

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Introduction: Multiple Myeloma is a plasma cell malignancy commonly presented with anemia, renal impairment, and bone disease. Bing–Neel syndrome describes central nervous system involvement typically associated with Immunoglobulin M (IgM) lymphoplasmacytic lymphoma, with rare reports in non-IgM cases. This report aims to present a case of Immunoglobulin G (IgG) kappa multiple myeloma with an unusual clinical course mimicking lymphoplasmacytic lymphoma and presenting with severe multisystem complications.

Case report: In March 2021, after the second dose of the COVID vaccine, a 69-year-old male patient developed skin rashes, thrombocytopenia, elevated Lactate Dehydrogenase, and splenomegaly, which improved after corticosteroid therapy. In early 2022, he developed progressive neurological symptoms, including hoarseness, followed by bilateral facial nerve palsy, gait instability, and progressive encephalopathy. Guillain–Barré syndrome was suspected, and appropriate therapy was initiated. Imaging showed splenomegaly and lymphadenopathy. During further evaluation, the patient developed fever and pancytopenia, prompting hematology consultation. In May 2022, the patient's condition rapidly worsened with altered mental status, pancytopenia, disseminated intravascular coagulation, acute liver and kidney injury and sepsis, requiring admission to the intensive care unit and transfusion support. Hematologic assessment revealed an IgG kappa monoclonal gammopathy. Bone marrow analysis confirmed kappa-restricted clonal plasma cell infiltration (CD138+, CD20–), supporting the diagnosis of multiple myeloma. Following stabilization, the patient continued hematology-directed treatment.

Conclusion: To our knowledge, this represents one of the very few reported cases of multiple myeloma with such a clinical presentation. Pathological plasma cells and elevated IgG levels were detected in the cerebrospinal fluid, confirming Bing–Neel syndrome.

Keywords: Central Nervous System Diseases; Immunoglobulin G; Multiple Myeloma

CR83

Chemobioimmunotherapy in a Patient with a Rare Site of Metastatic Esophageal Carcinoma – case report

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Introduction: Esophageal cancer is primarily classified into squamous cell carcinoma and adenocarcinoma. Over recent decades, the incidence of esophageal adenocarcinoma, particularly in the distal esophagus, has increased largely due to the rising prevalence of Barrett's esophagus. Despite advances in diagnostics and treatment, prognosis remains poor, especially in advanced disease. The most common metastatic sites include the liver, lungs, lymph nodes and bones. We present a case of extremely rare skeletal metastasis managed using a multimodal treatment approach.

Case report: A 49-year-old male was diagnosed with distal esophageal adenocarcinoma in December 2021. Initial treatment included esophageal stent placement, neoadjuvant chemoradiotherapy and subsequent esophagectomy. Histopathological examination revealed ypT3N0Mx disease, HER2 positivity and R0 resection margins. In October 2022, the patient presented with right knee pain. Biopsy confirmed an osseous metastasis consistent with the primary tumor. Palliative radiotherapy was administered, followed by systemic treatment with FOLFOX and trastuzumab until June 2023. Due to disease progression, FOLFOX plus trastuzumab was reintroduced in May 2024, achieving stable disease. However, isolated local progression of the knee lesion occurred shortly thereafter without evidence of other distant metastases. Cryoablation was attempted without success and a right transfemoral amputation was performed in October 2024. In February 2025, lung and retroperitoneal metastases were detected. Oxaliplatin and fluoropyrimidine-based chemotherapy with trastuzumab was restarted with pembrolizumab added following confirmation of PD-L1 expression (CPS 5). As of January 2026, the patient remains on maintenance therapy with pembrolizumab and trastuzumab with stable disease.

Conclusion: Although survival outcomes in advanced esophageal cancer remain poor, this case demonstrates that a personalized multimodal approach, guided by specific tumor phenotype (HER2 positivity and PD-L1 expression), integrating chemotherapy, immunotherapy and targeted biological therapy, may achieve prolonged disease control.

Keywords: Biological Therapy; Chemotherapy; Esophagus; Immunotherapy; Neoplasm Metastasis

CR84

When Blood Cultures Are Positive but Transthoracic Echocardiography Is Inconclusive: Native Valve Endocarditis Caused by *Gemella morbillorum*

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Introduction: Infective endocarditis (IE) is a serious infection with an in-hospital mortality ranging from 15% to 25%. IE remains a diagnostic challenge, particularly when caused by rare pathogens and presenting with nonspecific clinical features. *Gemella morbillorum*, a commensal organism of the oropharyngeal and gastrointestinal flora, is an uncommon but significant cause of IE. Its subtle echocardiographic findings and indolent clinical presentation may delay diagnosis and complicate management.

Case report: We present the case of a 47-year-old man with multiple comorbidities, including diabetes mellitus, obesity, obstructive sleep apnea, and inherited thrombophilia with a history of recurrent cerebrovascular events, who was admitted with a four-day history of generalized weakness, chills, gastrointestinal symptoms with elevated C reactive protein (195.0 mg/L). The chest X-ray showed an enlarged cardiac silhouette and the urine culture result returned sterile. Blood cultures grew *Gemella morbillorum*. Initial transthoracic echocardiography was inconclusive. Subsequent transesophageal echocardiography (TTE) revealed a small, mobile, chain-like vegetation on the non-coronary cusp of the native aortic valve, accompanied by aortic regurgitation. The patient was evaluated by a multidisciplinary Heart Team, which recommended conservative management. 18F-FDG positron emission tomography/computed tomography showed no pathological metabolic activity in the valvular region, although its sensitivity for small native valve lesions is limited. The patient completed a six-week course of antibiotic therapy with amoxicillin–clavulanate, achieving clinical and laboratory improvement. Follow-up TTE demonstrated no residual vegetations.

Conclusion: This case emphasizes the need for a suspicion of IE caused by rare pathogens, particularly in patients with complex comorbidities and nonspecific clinical presentations. It highlights the crucial role of transesophageal echocardiography and multidisciplinary decision-making in achieving accurate diagnosis and appropriate management, especially when advanced imaging is negative or inconclusive.

Keywords: Echocardiography; Endocarditis; *Gemella*

CR85

When the Skin Reveals Cancer: Paraneoplastic Bullous Pemphigoid as a Diagnostic Challenge

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Introduction: Paraneoplastic bullous pemphigoid is a rare, autoimmune, mucocutaneous, blistering disease that is associated with a confirmed or occult neoplasm. It usually presents as tense blisters and erosions on skin and mucous membranes.

Case report: An 80-year-old man presented to a family medicine outpatient clinic with a pruritic, erythematous rash and a widespread eruption of large bullae involving the upper arms, chest, abdomen, and thighs. Initial management in primary care included topical treatment with a combined corticosteroid–antibiotic preparation (betamethasone and gentamicin) and an oral antihistamine (fexofenadine). Due to the severity and extent of the bullous lesions, he was referred to the emergency dermatology department for further evaluation. Laboratory investigations and a skin biopsy were performed. Laboratory results demonstrated normocytic normochromic anemia, leukocytosis, and elevated segmented granulocytes. Given the characteristic clinical presentation and the patient's advanced age, bullous pemphigoid was suspected. Histopathological examination of the skin biopsy revealed a subepidermal bullous dermatosis consistent with bullous pemphigoid. Direct immunofluorescence demonstrated linear deposition of IgG and C3 along the epidermal basement membrane zone, with IgG deposits identified on the blister roof. Further diagnostic evaluation showed elevated serum levels of the tumor marker CA 72-4, raising suspicion of a paraneoplastic etiology. The patient was therefore referred for gastroenterological assessment. Review of his medical history revealed that the last surveillance colonoscopy had been performed in 2017, demonstrating a high-grade tubular adenoma, with no subsequent follow-up. Extended diagnostic work-up ultimately resulted in the diagnosis of colorectal carcinoma.

Conclusion: Bullous pemphigoid in elderly patients may represent a paraneoplastic manifestation of malignant diseases. The presented case highlights the importance of timely recognition of atypical or treatment-resistant skin changes in family medicine practice, as well as the necessity for comprehensive diagnostic evaluation.

Keywords: Colonic Neoplasms; Paraneoplastic Syndromes; Pemphigoid, Bullous; Pruritus; Skin Diseases

CR86

Herbal supplements as an underrecognized arrhythmogenic risk – case report

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Introduction: Atrial fibrillation (AF) is one of the most common cardiac arrhythmias and is associated with a significantly increased risk of thromboembolic events, particularly ischemic stroke. Management includes anticoagulation and rhythm or rate control, depending on the clinical context.

Case report: A 52-year-old man presented to the emergency medical service with palpitations and chest discomfort. Besides arterial hypertension, his history was unremarkable. Ten days earlier, he had experienced similar symptoms and was diagnosed with paroxysmal AF in the emergency department, where sinus rhythm was restored with a single intravenous dose of 300 mg amiodarone. On the current presentation, auscultation revealed an irregular heartbeat, and a normal lung exam. Blood pressure was 145/90 mmHg, with an oxygen saturation of 98% and a variable heart rate. A 12-lead electrocardiogram confirmed recurrent paroxysmal AF with rapid ventricular response. Detailed history revealed that prior to both episodes, the patient had consumed two liters of homemade “liver detox” tea containing nettle, dandelion, and milk thistle, which raised suspicion of a diet-related trigger for AF. Dandelion and nettle have diuretic properties and may contribute to electrolyte imbalance, especially hypokalemia, a known trigger of AF. Milk thistle has been associated with prolonged warfarin activity and increased bleeding risk, while nettle contains high levels of vitamin K that may promote coagulation. He was transported to the hospital, where pharmacological cardioversion was again achieved with 300 mg of amiodarone, and was discharged in stable condition with recommendations for cardiology follow-up and initiation of rivaroxaban 20 mg and bisoprolol 5 mg once daily.

Conclusion: This case highlights the importance of caution when using dietary supplements and herbal remedies, particularly in individuals predisposed to cardiac arrhythmias.

Keywords: Atrial Fibrillation, Fatty Liver, Hypokalemia

CR87

Chronic Granulomatous Disease as a Differential Diagnosis to Inflammatory Bowel Disease – An Instructive Case Report

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Introduction: Chronic granulomatous disease (CGD) is a rare inborn error of immunity characterised by a defect in the phagocyte respiratory burst, leading to impaired killing of certain bacteria and fungi. It is inherited in an X-linked or autosomal recessive manner. CGD is characterised by recurrent infections and dysregulated inflammation with granuloma formation.

Case report: We present a boy whose gastrointestinal symptoms began when he was two years of age, with stool retention, followed by the development of external haemorrhoids, perianal fissures, and alternating episodes of diarrhoea and constipation. At the age of five, he was hospitalised due to haematochezia, and a colonoscopy revealed diffuse inflammation in the mucosa of the transverse and descending colon, most prominently in the sigmoid colon and rectum. He was initially diagnosed with ulcerative colitis, so mesalazine and metronidazole were started. Histopathology showed granulomatous inflammation, consistent with granulomatous colitis. Despite treatment with corticosteroids, azathioprine, thalidomide, infliximab, and anakinra, sustained clinical remission was not achieved. Genetic testing revealed a hemizygous mutation in the CYBB gene (c.785>G, p. Phe262Cys) responsible for X-linked CGD, after which antimicrobial prophylaxis was initiated. Persistent inflammation on follow-up colonoscopy led to the decision to proceed with hematopoietic stem cell transplantation (HSCT). The patient was successfully transplanted at the age of eight from a matched unrelated donor, and he is now four years post-transplant, without any symptoms.

Conclusion: This case highlights the importance of considering CGD in children with granulomatous, treatment-refractory colitis. Early recognition allows timely use of appropriate antimicrobial prophylaxis and evaluation for definitive therapy. HSCT remains the only curative option for CGD, offering the potential for immune restoration and long-term resolution of inflammatory manifestations.

Keywords: Chronic Granulomatous Disease; Colitis; Genetic Diseases, X-Linked; Hematopoietic Stem Cell Transplantation

CR88

Fibrillary Glomerulonephritis Unmasked by Dabigatran Supersaturation in Acute Kidney Injury

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Introduction: Direct oral anticoagulants revolutionized the prevention of thromboembolic events, particularly in atrial fibrillation (AF). Although safer than vitamin K antagonists, evidence indicates they may cause anticoagulant-related nephropathy, especially dabigatran due to renal clearance. Chronic kidney disease (CKD) and findings of parenchymal renal disease are major risk factors, suggesting underlying glomerular pathology, while atypical associations such as fibrillary glomerulonephritis require further investigation.

Case report: We present a 65-year-old female with an acutisation of CKD and macroscopic hematuria. Medical history included arterial hypertension, AF and chronic cardiomyopathy. The patient had been taking dabigatran for AF for six months. Upon admission due to dabigatran supersaturation, idarucizumab and prothrombin complex concentrate were administered. Due to high bleeding risk, the patient was transferred to the intensive care unit for continuous venovenous hemodiafiltration (CVVHDF) with regional citrate anticoagulation. During hospitalization, two additional hemodialysis sessions were performed. Coagulation parameters significantly improved after two days of CVVHDF. Laboratory tests showed partial improvement of CKD, nephrotic-range proteinuria and 10% dysmorphic erythrocytes. Urgent abdominal ultrasound revealed enlarged kidneys with disrupted corticomedullary differentiation. Treatment with corticosteroids (40 mg) was initiated *ex iuvantibus* and immunological work-up showed no clear criteria for autoimmune disease. Kidney biopsy revealed fibrillary glomerulonephritis and diffuse severe acute tubular injury of unclear etiology. Intermittent urinary infections (*Klebsiella pneumoniae*, *Enterococcus faecium* and *Escherichia coli*) necessitated antibiotics. Following resolution of the urinary infections, rituximab (2x1 g) therapy was considered. Anticoagulation therapy was switched to rivaroxaban. Further follow-up is planned.

Conclusion: This case highlights the complex interplay between renal dysfunction and anticoagulation, demonstrating the potential nephrotoxicity of dabigatran despite its widespread use. It also emphasizes the crucial role of kidney biopsy in identifying rare underlying glomerular diseases such as fibrillary glomerulonephritis.

Keywords: Acute Kidney Injury; Atrial Fibrillation; Dabigatran; Glomerulonephritis; Renal Insufficiency, Chronic

CR89

NIPT limitations, From double Y to double X – a case report

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Introduction: Klinefelter syndrome (KS) is one of the most common sex chromosome aneuploidies in males, typically caused by an additional X chromosome (47,XXY). Mosaic forms, such as 47,XXY/46,XY, are less common and often associated with milder or variable clinical manifestations. KS most commonly presents with primary hypogonadism, including small testes, androgen deficiency, infertility, and gynecomastia, and is associated with an increased risk of neurocognitive and psychosocial difficulties.

Case report: We report the case of a 7-month-old male infant referred for clinical genetic evaluation following a discordant prenatal screening result. Non-invasive prenatal testing (NIPT) suggested an XYY karyotype, but further invasive prenatal testing was declined by the parents. The patient was born at term and followed in a neurodevelopmental clinic due to hypotonia, with early physiotherapy and occupational therapy recommended. The external genitalia were male, while the testes were not reliably palpable. Postnatal conventional karyotyping revealed mosaicism, with 47,XXY detected in 33% and 46,XY in 67% of analyzed cells, confirming mosaic Klinefelter syndrome (mos 47,XXY/46,XY). Parents were counseled regarding the variable clinical course, potential endocrine, reproductive, and neurodevelopmental implications, and the need for regular pediatric endocrinology follow-up and genetic counseling.

Conclusion: This case highlights the importance of early genetic diagnosis of mosaic Klinefelter syndrome, even in the absence of clear clinical signs. Timely identification allows appropriate monitoring, endocrine evaluation, fertility counseling, and psychosocial support. It also emphasizes the limitations of NIPT in accurately distinguishing sex chromosome aneuploidies, reinforcing that NIPT remains primarily a screening tool rather than a definitive diagnostic method. Confirmatory cytogenetic testing, therefore, remains essential to ensure accurate diagnosis.

Keywords: Karyotype; Klinefelter Syndrome; Noninvasive Prenatal Testing

CR90

Genetic atypical hemolytic uremic syndrome successfully treated with complement component C5 inhibitors**Marija Kišmartin¹; Leona Zečević¹; Ivan Dulić¹; Lea Paradinović²; Ivana Tolj^{1,3}***1 - Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia**2 - Emergency Medicine Institute of Osijek-Baranja County, Croatia**3 - Department of Nephrology, University Hospital Centre Osijek, Osijek, Croatia*

Introduction: Hemolytic uremic syndrome (HUS) is a thrombotic microangiopathy characterized by microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury. Atypical hemolytic uremic syndrome (aHUS) accounts for approximately 5–10% of all HUS cases, predominantly within adults. It is most often the result of uncontrolled activation of the alternative complement pathway, while infections, pregnancy, transplantation and drug use can trigger the clinical manifestation of the disease. First-line therapy in aHUS is complement inhibition, as well as plasmapheresis in certain indications.

Case report: A 27-year-old female patient presented to the emergency department with suprapubic and bilateral lower chest pain, fever and hematuria. Since childhood, she had been hospitalized several times for episodes of aHUS with acute renal failure, which temporarily required peritoneal dialysis. At the age of 19, the CFH gene mutation was confirmed, along with her brother's diagnosis. Laboratory tests revealed thrombocytopenia and normocytic normochromic anemia with significant reticulocytosis, consistent with hemolytic anemia. Abdominal ultrasound showed increased echogenicity of the renal parenchyma. Echocardiography was normal. During hospitalization, nine sessions of therapeutic plasmapheresis with fresh frozen plasma were performed, initially with citrate anticoagulation, later with heparin. After approval by the ethics committee, therapy for inhibition of the C5 complement component with ravulizumab was initiated, with prior premedication and vaccination against meningococcal infection. After the introduction of therapy, the patient felt well, with stabilization of the clinical condition. Due to the planned pregnancy, the therapy was replaced with eculizumab. The patient tolerated the maintenance therapy well.

Conclusion: Early C5 inhibition achieved long-term control of genetically determined, relapse-prone and treatment-refractory atypical HUS, stabilizing disease and preserving clinical stability, including during pregnancy planning.

Keywords: Complement C5; Hemolytic-Uremic Syndrome; Thrombocytopenia

CR91

One Gene, Two Brothers, Multiple Faces: X-Linked Ichthyosis Caused by an Xp22.31 STS Deletion – A Case Report

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Introduction: X-linked ichthyosis (XLI) is a genetic disorder characterized by deletions or mutations of the steroid sulfatase (STS) gene at Xp22.31, which leads to a damaged epidermal barrier. Although it is predominantly a cutaneous disorder, its clinical expression is highly variable and can also manifest extracutaneously. The case report aims to demonstrate the phenotypic variability of XLI within the same family, in two brothers with an identical deletion of the STS gene.

Case report: Two brothers, aged 15 months and 4 years and 9 months, were referred for genetic evaluation due to congenital skin abnormalities. Both exhibited persistent dry skin and scaling from early childhood. Dermatological assessment revealed generalized, brownish, polygonal scales, with sparing of the palms and soles. Despite similar cutaneous findings, the clinical courses diverged. The younger brother demonstrated normal psychomotor development and no extracutaneous manifestations during follow-up. In contrast, the older brother experienced epileptic seizures in the neonatal period; brain magnetic resonance imaging revealed chronic, non-progressive post-lesional changes, with subsequent seizure control. Early intellectual development was assessed as below average, and cryptorchidism requiring surgical correction was also present. Chromosomal microarray analysis identified an identical maternally inherited hemizygous deletion of the Xp22.31 region, measuring 1.558 Mb and encompassing the STS gene. Both siblings received symptomatic dermatological management with emollients and continue to receive multidisciplinary follow-up.

Conclusion: This case demonstrates that XLI exhibits marked intrafamilial phenotypic variability despite an identical STS gene deletion. The discrepant extracutaneous manifestations observed in two brothers emphasize that molecular diagnosis alone is not sufficient to predict clinical expression, confirming that XLI is a potentially multisystem disorder that requires individualized, multidisciplinary management.

Keywords: Ichthyosis; Maternal Inheritance; Microarray Analysis; Steryl-Sulfatase

CR92

Treatment challenges in a patient with acute myeloid leukemia – case report

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Introduction: Acute myeloid leukemia (AML) is an aggressive malignant disease of the hematopoietic system characterized by the uncontrolled formation of immature myeloid cells in the bone marrow, leading to the failure of normal hematopoiesis and the development of life-threatening complications. The aim of this case report is to present the challenges in treating adult AML.

Case report: A 68-year-old male was admitted to the Department of Hematology due to significant leukocytosis, anemia, and thrombocytopenia. A cytological puncture of the bone marrow revealed AML with 87% blasts, of which 80% were myeloblasts, according to the immunophenotyping. A TKD mutation in the FTL3 gene was identified indicating biologically more active disease with potential poor prognosis and higher risk of relapse. Due to dyspnea, a chest computed tomography scan was performed, which revealed diffuse centrilobular emphysema. Elevated levels of N-terminal pro-brain natriuretic peptide suggested heart insufficiency, so a cardiology consultation was obtained, and echocardiography revealed signs of pulmonary hypertension and hypertrophy of the left ventricular wall. Infectious complications and compromised cardiological status made the patient unfit for intensive treatment of AML, so venetoclax and azacitidine were prescribed. The chemotherapy protocol was tolerated without complications. Following regression of pneumonic radiological findings, microbiological analysis of the sputum revealed *Pseudomonas aeruginosa*, and ciprofloxacin therapy was initiated. The patient was discharged home shortly afterwards, was later rehospitalized for continued treatment, and is currently in complete remission of the disease.

Conclusion: This case highlights the complexity of managing AML, especially in elderly patients with various infectious and pulmonary complications. Even though this patient was not a good candidate for intensive treatment, venetoclax and azacitidine proved to be effective in inducing remission of AML.

Keywords: Azacitidine; Leukemia, Myeloid, Acute; Mutation; *Pseudomonas aeruginosa*

CR93

Fulminant course of anti-glomerular basement membrane disease in a young patient

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Introduction: Anti-glomerular basement membrane (anti-GBM) disease is a rare autoimmune cause of rapidly progressive glomerulonephritis, characterized by rapid deterioration of renal function and crescent formation on renal biopsy. The disease can affect the kidneys exclusively or manifest as a pulmonary–renal syndrome. The aim of this case report is to describe the clinical course and therapeutic challenges of anti-GBM disease in a young patient.

Case report: We present a 22-year-old female patient with positive family history of Fanconi syndrome and chronic kidney disease, who developed progressive fatigue, abdominal pain, nausea, intermittent fever, oliguria, dark urine, and rapid weight gain with generalized edema over one month. Despite initial outpatient antibiotic treatment, symptoms rapidly worsened. Laboratory evaluation revealed acute kidney injury with serum creatinine level of 229 μmol/L, progressing to anuria and need for urgent hemodialysis. Renal ultrasound showed increased echogenicity of renal parenchyma with reduced corticomedullary differentiation. Immunological workup revealed a markedly elevated anti-GBM antibody titer (166 U/L). Renal biopsy showed crescentic glomerulonephritis with linear IgG deposition, with cellular and fibrocellular crescents in 100% of glomeruli, suggesting highly active disease with minimal chronic changes. Treatment included high-dose intravenous methylprednisolone, cyclophosphamide, and eight sessions of therapeutic plasmapheresis, followed by intravenous immunoglobulin. Anti-GBM antibody titers significantly declined during treatment. High-resolution computed tomography revealed pleural and pericardial effusions, consistent with pulmonary–renal syndrome. Despite aggressive immunosuppressive therapy, the patient remained anuric and dependent on chronic renal replacement therapy.

Conclusion: This case highlights the fulminant course of anti-GBM disease in young patients and underscores the importance of early recognition and timely initiation of immunosuppressive therapy in rapidly progressive glomerulonephritis. Irreversible renal failure may still occur, underscoring the need for early referral to specialized nephrology care.

Keywords: Cyclophosphamide; Glomerulonephritis; Methylprednisolone; Plasmapheresis

CR94

Personalized Immunomodulatory Therapy Guided by Immunogenetic Profiling in Recurrent Peripheral Ulcerative Keratitis: A Case Report

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Introduction: Peripheral ulcerative keratitis (PUK) is a rare but potentially sight-threatening autoimmune corneal disease. Standard corticosteroid therapy is often insufficient, and a personalized approach based on genetic and immunological biomarkers may allow for more precise targeting of the inflammatory response. The aim of this case report is to demonstrate the role of immunogenetic profiling in guiding PUK therapy using the biologic agent adalimumab.

Case report: A 58-year-old patient with recurrent PUK in the right eye, without evidence of systemic autoimmune disease, developed repeated epithelial defects and progression of stromal infiltrates despite intensive topical corticosteroids (prednisolone acetate 1% drops 6×/day) and systemic corticosteroids (oral prednisone 1 mg/kg/day for 4 weeks). Serological and immunogenetic testing revealed HLA-DR4 and HLA-DQ1 haplotypes and elevated serum levels of IL-6 and TNF- α , indicating a predominantly Th17-mediated inflammatory response. Based on this immunological profile, adalimumab therapy was initiated at 40 mg subcutaneously every other week, while systemic corticosteroids were gradually tapered over six weeks. During six months of follow-up, complete healing of the epithelial defect, regression of stromal infiltrates, and absence of disease recurrence were observed. The patient continued regular ophthalmological monitoring, including slit-lamp exams every 4–6 weeks.

Conclusion: This case highlights the importance of integrating genetic and immunological biomarkers into the diagnostic and therapeutic management of PUK. Personalized immunomodulatory therapy may optimize clinical response and reduce the risk of recurrence in selected patients, although conclusions are limited by single-case observation and short-term follow-up. Complete surgical or medical management, when guided by immunogenetic profiling, can help preserve long-term corneal transparency and visual function.

Keywords: Biological Products; Corneal Ulcer; Cytokines; HLA Antigens; Immunosuppression Therapy

CR95

Less Salt, Less Inflammation: Endothelial Function Improvement in a Psoriasis Patient

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Introduction: Psoriasis is a chronic immune-mediated inflammatory skin disease, characterized by erythematous, scaly. Beyond cutaneous involvement, psoriasis is associated with an increased risk of cardiovascular disease. Recent evidence indicates that patients with moderate psoriasis exhibit increased sodium accumulation in the skin.

Case report: We report a case of a 51-year-old male patient with a 20-year long history of psoriasis who participated in a 14-day long low-sodium (LS) dietary intervention. Dietary adherence was confirmed by the reduction in 24-hour urinary sodium excretion. After finishing the dietary intervention, the patients were assessed using microvascular function markers conventionally measured by the laser Doppler flowmetry. It demonstrated enhanced endothelium-dependent vasodilation, with a pronounced increase in post-occlusive reactive hyperemia (PORH), from the value of 98,10 perfusion units (PU) before, to the 220,40 PU after the LS diet. It also demonstrated acetylcholine-induced vasodilation (AChID) improvement from 11,44 to 11,90 measured by PU. Also, augmentation of local thermal hyperemia (LTH) from 6,78 to 11,92 PU was observed. These vascular changes were accompanied by a mild reduction in systemic inflammation, as reflected by a decrease in C-reactive protein levels from 2.70 to 2.07 mg/L.

Conclusion: This case illustrates that, given the critical effect of endothelial dysfunction on psoriasis mortality, LS diet may present a simple adjunctive strategy to modulate vascular risk in this population.

Keywords: Cardiovascular Diseases; Psoriasis; Diet, Sodium-Restricted

CR96

Precision Therapy in Severe Immune-Mediated Dry Eye Disease: A Tailored Approach

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Introduction: Dry eye disease (DED) is a chronic ocular surface condition with a strong inflammatory and immunological basis. Patients with autoimmune diseases often develop more severe and refractory forms of the disease. Therefore, standard symptomatic therapy is frequently insufficient in such cases. Understanding the importance of tear film production and comorbidities is crucial in DED diagnosis and optimal patient management.

Case report: We present a case of a 45-year-old woman with Sjögren's syndrome, an autoimmune disorder, who was experiencing prolonged symptoms of eye burning, foreign body sensation, accompanied by photophobia, with no significant improvement despite treatment with conventional artificial tear therapy. The patient underwent an extensive dry eye assessment, which revealed significantly elevated Ocular Surface Disease Index (OSDI) levels, reduced Schirmer I test values, and a shortened tear break-up time (TBUT). Clinical examination indicated signs of chronic ocular surface inflammation. Based on the patient's phenotype and immunological etiology, personalized therapy was implemented, given the immune-mediated nature of the disease. This included a local immunomodulator cyclosporin A, a short-term application of a local corticosteroid, supportive treatment with preservative-free artificial tears, and finally patient education focusing on disease understanding, treatment adherence, and lifestyle modification. As a result of therapeutic initiation, the patient reported that this individualized treatment led to reduced subjective symptoms, improved tear film stability, good tolerance of therapy, and an overall increase in quality of life.

Conclusion: A tailored therapeutic approach focused on the disease's immune mechanisms illustrates clinical outcomes in patients with severe DED and represents an example of precision medicine in ophthalmology.

Keywords: Adrenal Cortex Hormones; Cyclosporine; Dry Eye Syndromes; Lubricant Eye Drops

CR97

Atypical presentation of *Fusobacterium necrophorum* sepsis without classic Lemierre's syndrome in a 16-year-old male**Marta Koren¹; Petra Kolundžić¹; Mila Kostić¹; Maja Vrdoljak Pažur^{1,2}***1 - School of Medicine, University of Zagreb, Zagreb, Croatia**2 - University Hospital for Infectious Diseases "Dr. Fran Mihaljević", Zagreb, Croatia*

Introduction: *Fusobacterium* species are Gram-negative anaerobic bacteria that form part of the microbiota of the oral cavity, pharynx and gastrointestinal tract. *Fusobacterium necrophorum* sepsis is usually associated with Lemierre's syndrome. It presents with oropharyngeal infection followed by septic thrombophlebitis of the internal jugular vein and metastatic septic complications, usually to the lungs. We report a case of *Fusobacterium necrophorum* sepsis in an immunocompetent patient, presenting with hepatic and splenic abscesses.

Case report: A previously healthy 16-year-old male was admitted with high-grade fever (up to 41°C) and chills, but no other symptoms. Physical examination revealed mild dehydration, erythematous pharynx with enlarged tonsils without exudate, and mild hepatomegaly. Four weeks prior, he had a sore throat treated with amoxicillin and clavulanic acid. Laboratory findings showed leukocytosis ($22.8 \times 10^9/L$) with neutrophilia of 92%, thrombocytopenia ($67 \times 10^9/L$), and elevated C-reactive protein (256.3 mg/L) and procalcitonin (155.2 µg/L). Despite empiric antimicrobial therapy with ceftriaxone, his clinical condition worsened, with somnolence and hypotension, requiring intensive care unit admission and noradrenaline support. Blood cultures grew *Fusobacterium necrophorum*. Abdominal ultrasound revealed splenomegaly with a 3-cm splenic abscess and two hepatic abscesses of 3 and 1 cm. Doppler ultrasound and magnetic resonance imaging of the neck excluded internal jugular vein thrombosis. Antimicrobial therapy with ceftriaxone and metronidazole was administered for two weeks. The patient was discharged on vancomycin and amoxicillin-clavulanate.

Conclusion: This case demonstrates an atypical presentation of *Fusobacterium necrophorum* sepsis. Although classically associated with Lemierre's syndrome, our patient developed sepsis and visceral abscesses despite minimal pharyngeal findings and no internal jugular vein thrombosis. It highlights the importance of recognizing rare manifestations of *Fusobacterium* infection for timely diagnosis and management.

Keywords: Abscess; Adolescent; *Fusobacterium Necrophorum*; Sepsis; Syndrome

CR98

Individualized Treatment of Severe Asthma: Successful Tezepelumab Therapy After Omalizumab Failure

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Introduction: Severe asthma is a condition characterized by poor response to standard therapy, frequent exacerbations and the need for systemic corticosteroids. It is commonly associated with type 2 (T2) inflammation mediated by interleukins 4, 5 and 13. Tezepelumab, a monoclonal antibody targeting thymic stromal lymphopoietin (TSLP), improves clinical outcomes regardless of asthma phenotype. This case report highlights the importance of individualized treatment in patients with evolving asthma phenotypes and the therapeutic effect of tezepelumab after unsuccessful treatment with omalizumab, a monoclonal antibody against immunoglobulin E (IgE).

Case report: A 56-year-old woman with a 10-year history of asthma initially presented with an allergic phenotype. In 2018, diagnostic evaluation revealed elevated IgE levels, sputum eosinophilia (50%), high fractional exhaled nitric oxide (FeNO) values, increased eosinophil count in peripheral blood and reduced lung diffusing capacity for carbon monoxide. Spirometry suggested small airway obstruction with reduced lung volumes. Despite standard inhalation therapy with inhaled corticosteroids, long-acting beta-agonists, long-acting muscarinic antagonists and systemic corticosteroids, the patient experienced frequent exacerbations. Omalizumab therapy administered between 2019 and 2021 resulted in no significant clinical improvement. During follow-up, T2 inflammatory markers regressed, while FeNO remained elevated. In 2024, tezepelumab was initiated at a dose of 210 mg every four weeks. After 12 subcutaneous administrations, the patient achieved significant clinical improvement, resulting in better disease control and the patient currently has no significant respiratory symptoms.

Conclusion: This case emphasizes the importance of continuous clinical and phenotypic assessment in severe asthma and supports an individualized approach using targeted biological therapy.

Keywords: Asthma; Biological Therapy; tezepelumab; Thymic Stromal Lymphopoietin

CR99

Early Clinical Recognition of Beckwith–Wiedemann Syndrome and Personalized Tumor Surveillance

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Introduction: Beckwith–Wiedemann syndrome (BWS) is a congenital, highly variable disorder characterized by dysregulated growth and an increased risk of embryonal tumors, such as Wilms tumor, hepatoblastoma, rhabdomyosarcoma and neuroblastoma. The disorder can result from various genetic/epigenetic alterations, most commonly abnormal methylation patterns at the imprinting control region 1 and imprinting control region 2 (IC2), paternal uniparental disomy within the 11p15.5 chromosomal region, and maternal heterozygous *CDKN1C* pathogenic variant. Early recognition can be challenging due to clinical variability; this case highlights the importance of timely diagnosis for tailoring tumor surveillance.

Case report: We present the case of male neonate transferred to the intensive care unit due to initial diagnosis of omphalocele, with macrosomia and macroglossia. Birth measurements were as follows: weight 4250 g, length 52 cm, and Apgar score 10. On admission, notable findings included low-set ears, micrognathia, macroglossia and omphalocele; the remaining examination was unremarkable. The omphalocele was surgically repaired and well-healed. Transient hypoglycemia was also noted postoperatively. Given features of overgrowth, macroglossia, craniofacial dysmorphism and hypoglycemia, BWS was suspected. The patient underwent multidisciplinary evaluation and genetic testing. Molecular testing revealed hypomethylation in the IC2, consistent with the clinical diagnosis of BWS. According to current guidelines, the patient was enrolled in a structured monitoring program, including regular hematology-oncology follow-up (abdominal ultrasound and serum alpha-fetoprotein monitoring), and endocrinological, nephrological and cardiovascular assessment.

Conclusion: BWS is a genetic disorder with a highly variable clinical spectrum and heterogeneous genetic/epigenetic etiologies. Accurate and early diagnosis is crucial to ensure appropriate patient management, with particular emphasis on individualized and vigilant tumor surveillance to enable the earliest possible detection of embryonal tumors.

Keywords: Beckwith-Wiedemann Syndrome; Genomic Imprinting; Neoplasms

CR100

Meier–Gorlin Syndrome Type 4 Presenting With Febrile Status Epilepticus and Ketotic Hypoglycemia

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Introduction: Meier–Gorlin syndrome (MGS) is a rare autosomal recessive disorder characterized by intrauterine and postnatal growth retardation, facial dysmorphism, and patellar agenesis, with usually preserved or only mildly impaired neurocognitive development. The aim of this report is to describe a boy with MGS type 4 carrying compound heterozygous mutations in the *CDT1* gene, presenting with an unusual clinical course that included febrile epileptic status, ketotic hypoglycemia, and transient hyperammonemia.

Case report: The patient is a boy born at 34 + 4/7 weeks of gestation with intrauterine growth restriction, who required resuscitation and mechanical ventilation at birth, and in whom a syndromic diagnosis was suspected early because of dysmorphic features and patellar agenesis. At the age of 5 years, he presented with febrile epileptic status following prolonged fasting and infection, accompanied by severe ketotic hypoglycemia, metabolic acidosis, hyperammonemia, and cerebral edema. These abnormalities resolved after intensive treatment with intravenous glucose, targeted therapy for hyperammonemia, and supportive measures, without residual neurological deficit. Extended genetic testing (exome sequencing) confirmed a compound heterozygous *CDT1* genotype, and the parents received genetic counseling regarding a 25% recurrence risk and options for prenatal diagnosis.

Conclusion: This case highlights the importance of recognizing the Meier–Gorlin phenotype in children with intrauterine growth restriction and postnatal failure to thrive, as well as the necessity of prompt management of metabolic stress in order to prevent life-threatening complications such as status epilepticus.

Keywords: Exome Sequencing; Fetal Growth Retardation; Hypoglycemia; Meier-Gorlin Syndrome; Status Epilepticus

CR101

Early diagnosis of MYCN-Amplified Retinoblastoma in a 9-Month-Old Infant

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Introduction: Retinoblastoma is a malignant intraocular tumor most commonly diagnosed in young children. A typical clinical sign of retinoblastoma is leukocoria, defined as a white pupillary reflex resulting from tumor reflection through a dilated pupil. Most retinoblastomas arise from biallelic inactivation of the *RB1* gene, while 1.5% of all retinoblastoma tumors show high-level *MYCN* amplification. *MYCN*-amplified, *RB1* proficient tumors present within the first year of life and exhibit a more aggressive clinical and histological phenotype.

Case report: During a systematic examination at the age of 9 months, patient's parents reported strabismus and leukocoria, and the child was referred to the emergency ophthalmology clinic. The ophthalmological examination found a lack of red reflex and a retrolental solid retinal detachment on the right eye, which is why hospitalization was arranged. Magnetic resonance imaging (MRI) of the brain and orbit showed a smaller right eyeball and a soft tissue mass in the retrolental part suggesting a more prolonged disease evolution. During the hospitalization enucleation of the right eye with implantation of a silicone prosthesis and conjunctivoplasty was performed. Histopathological analysis confirmed a diagnosis of histologic grade 3 retinoblastoma. Fluorescence in situ hybridization analysis demonstrated *MYCN* amplification in approximately 90% of tumor cells. Long-term follow-ups are performed by an ophthalmologist every 3 months and by a pediatric hematologist-oncologist every 6 months, with routine MRI of the brain and orbits. The most recent follow-up MRI revealed no evidence of retinoblastoma recurrence.

Conclusion: A detailed ophthalmologic examination with fully dilated pupils is essential for early diagnosis of retinoblastoma. *MYCN*-amplified, *RB1*-proficient tumors, although rare, are aggressive but can achieve favorable outcomes with timely enucleation.

Keywords: Eye Enucleation; FISH Technique; Retinoblastoma

CR102

Twin-twin transfusion syndrome in monochorionic diamniotic twins – a case report**Maja Krišto¹; Vanesa Marin¹; Lucija Lizatović¹; Martina Vulin^{1,2}***1 - Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Gynecology and Obstetrics, University Hospital Centre Osijek, Osijek, Croatia*

Introduction: Monochorionic diamniotic (MCDA) twins are monozygotic, arising from a single fertilized egg splitting later in development, and share the same placenta. Due to placental vascular anastomoses an imbalance in blood exchange occurs, such as twin-twin transfusion syndrome (TTTS). The hallmark of TTTS is the combination of oligohydramnios in the donor twin and polyhydramnios in the recipient twin. If untreated, TTTS is related to high perinatal mortality. Fetoscopic laser ablation (FLA) of placental anastomoses is the preferred treatment for TTTS Quintero stage ≥ 2 , improving overall survival to 70%.

Case report: A 26-year-old woman was admitted to hospital at 29 weeks gestational age (GA) with MCDA twin pregnancy due to threatened preterm labour. The pregnancy was conceived by *in vitro* fertilization. MCDA pregnancy was confirmed at 12 weeks scan. TTTS was diagnosed at 15 weeks GA based on ultrasound findings of amniotic fluid volume discordance and poor visualization of the donor's urinary bladder. At 19 weeks GA, due to TTTS Quintero stage III, FLA of placental vascular anastomoses was performed at the University Hospital Centre (UHC), Sofia, Bulgaria. The next day, intrauterine demise of the recipient twin was confirmed and patient was discharged from UHC for outpatient management. At 30 weeks GA, the patient developed uterine contractions and vaginal bleeding. Cesarean section was performed, resulting in birth of a female preterm neonate.

Conclusion: TTTS is a life-threatening complication of MCDA twin pregnancies. Despite optimal treatment, intrauterine demise of one or both twins may occur. Early diagnosis, close monitoring, and timely referral to specialized centres remain essential. FLA is the most effective treatment of TTTS and results in the survival of at least one twin in about 85% of MCDA pregnancies.

Keywords: Amniotic Fluid; Fetofetal Transfusion; Fetoscopy; Twins

CR103

Treatment of acute cardiogenic shock with advanced methods of mechanical circulatory support

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Introduction: Cardiogenic shock is a clinical syndrome caused by severe cardiac dysfunction, leading to critically reduced cardiac output and inadequate tissue perfusion. This case describes the management of acute cardiogenic shock using mechanical circulatory support.

Case report: A 40-year-old male with a two-year history of compensated left ventricular dysfunction experienced ventricular fibrillation during a soccer match and was successfully defibrillated. He was admitted to University Hospital (UH) Split, where an implantable cardioverter-defibrillator (ICD) was implanted for secondary prevention. Two years later, he presented with progressive heart failure symptoms and a left ventricular ejection fraction (LVEF) below 30%. An additional pacemaker was implanted, and after that, a 6-minute walk test showed 70% of expected with a stable clinical condition. After a further two years, his condition deteriorated significantly, with LVEF declining to 10% and recurrent ventricular arrhythmias, resulting in seven ICD activations. He was emergently supported with an Impella CP (2.5 L/min) and transferred to UH Rebro with a systolic blood pressure of 50 mmHg. There, peripheral veno arterial extracorporeal membrane oxygenation and an Impella 5.5 (5 L/min) were implanted. Due to the need for prolonged mechanical support, escalation to a durable left ventricular assist device (LVAD) was performed. On the second postoperative day, the LVAD triggered a low-flow alarm, prompting surgical intervention for device repositioning. Despite these measures, the patient subsequently died due to multiorgan failure.

Conclusion: This case highlights advances in the management of advanced heart failure using mechanical circulatory support. Mechanical support enabled safe interhospital transport despite profound hemodynamic instability and effectively replaced most of the cardiac output, allowing survival with minimal native cardiac function and providing a potential bridge to decision and possible transplant.

Keywords: Heart-Assist Devices; Shock, Cardiogenic; Ventricular Fibrillation

CR104

A Fork in the Diagnostic Road: Meningoencephalitis in an Immunocompromised Host

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Introduction: In patients with hematological malignancies and symptoms of central nervous system (CNS) infection a diagnostic work-up can be challenging. The primary disease itself may cause meningitis while immunosuppression increases susceptibility to bacterial, viral, fungal, and parasitic pathogens.

Case report: A 76-year-old man with a 3-day history of fever was admitted for evaluation of a possible central nervous system infection, presenting with somnolence and headache. His medical history included B-cell chronic lymphocytic leukemia (B-CLL). Cerebrospinal fluid (CSF) revealed $186 \times 10^6/L$ leukocytes (polymorphonuclear cells 68%, mononuclear cells 32%) and proteinorachia (0.863 g/L). Empiric antimicrobial therapy and supportive measures were initiated. There was no evidence of B-CLL progression in CNS. Due to immunodeficiency, intravenous immunoglobulin was administered. A broad diagnostic work-up was performed. Results showed intrathecal West Nile virus (WNV) IgM synthesis, and CSF reverse transcription polymerase chain reaction was positive for enterovirus. All other microbiological tests came back negative. The patient initially showed significant clinical improvement, but approximately three weeks into treatment, he developed *Clostridioides difficile* colitis, followed by methicillin-resistant *Staphylococcus aureus* sepsis which, despite treatment, led to lethal outcome.

Conclusion: In immunocompromised patients, establishing the precise disease etiology remains challenging when multiple microbiological investigations yield concurrent positive results. Given the acute onset of symptoms, enterovirus appears to be the most likely causative agent; however, WNV cannot be excluded. Clinicians should maintain a broad differential diagnosis of CNS infections in immunocompromised patients until an etiologic agent is identified. In addition, new diagnostic tests with higher specificity and sensitivity are needed.

Keywords: Enterovirus; Leukemia, Lymphocytic, Chronic, B-Cell; Meningoencephalitis; West Nile virus

CR105

From Pleural Effusion to Precision Therapy: An ALK-Positive Lung Adenocarcinoma in a Young Non-Smoker

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Introduction: Lung adenocarcinoma is the most common histological subtype of lung cancer and is increasingly diagnosed in younger non-smokers. In this group, molecular alterations are particularly important. One of the most relevant targetable alterations is anaplastic lymphoma kinase (ALK) rearrangement. The aim of this case report is to present an outcome of advanced ALK-positive lung adenocarcinoma in a young non-smoking patient.

Case report: A 35-year-old male non-smoker was admitted in 2019 due to right-sided pleural effusion accompanied by dry cough, low-grade fever, fatigue, and night sweats. Physical examination revealed decreased breath sounds at the right lung base. Diagnostic and therapeutic thoracentesis revealed hemorrhagic pleural fluid. Cytological analysis confirmed metastatic adenocarcinoma, while immunocytochemistry showed positivity for cytokeratin 7 and thyroid transcription factor-1, suggesting pulmonary origin. Computed tomography of the chest and abdomen demonstrated a tumor in the right lower lung lobe with widespread metastases to lymph nodes, bones, liver, and pleura, as well as signs of carcinomatous lymphangitis. The disease was classified as T4N3M1c, stage IV. Histopathological and molecular testing confirmed non-small cell lung cancer, adenocarcinoma subtype, with positive ALK rearrangement and negative epidermal growth factor receptor mutation status. First-line targeted therapy with alectinib was initiated. During hospitalization, the course was complicated by deep vein thrombosis of the right lower extremity, requiring therapeutic-dose low-molecular-weight heparin. The patient maintained an ECOG status of 0. Follow-up imaging showed marked tumor regression and sustained disease control.

Conclusion: This case highlights the educational value of molecular diagnostics in lung adenocarcinoma, particularly in young non-smokers. Early identification of ALK rearrangement enabled appropriate targeted therapy, leading to excellent clinical response and survival exceeding six years. The patient is still alive despite initially disseminated disease.

Keywords: Adenocarcinoma of Lung; Anaplastic Lymphoma Kinase; Thoracentesis

CR106

Severe rotavirus – associated cerebellitis in a child

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Introduction: Rotavirus cerebellitis is an extremely rare complication of rotavirus gastroenteritis. The clinical presentation is often severe and includes akinetic mutism, tremor and ataxia.

Case report: In August 2023, an 8-year-old boy presented with meningoencephalitic syndrome that occurred several days after frequent vomiting and watery stools. He was somnolent and didn't respond to verbal stimuli. Computed tomography (CT) scan was normal, and lumbar puncture revealed mild pleocytosis (20 cells/ μ L). An extensive workup for possible usual causes of meningoencephalitic syndrome was done, but all came back negative for common pathogens. Polymerase chain reaction (PCR) test for rotavirus P[4] VP4 was positive from cerebrospinal fluid. On the fifth day, the patient had his first bowel movement, and the virus was also detected in the stool. Magnetic resonance imaging (MRI) of the neuroaxis confirmed cerebellitis, and since his symptoms worsened with truncal ataxia, mutism and tetraplegia, intravenous immunoglobulins and methylprednisolone were administered. Verbal communication was severely damaged, and he would sometimes stare and become distant for a few seconds, but the electroencephalogram (EEG) showed no signs of epileptic seizures. Corticosteroid therapy was continued for 6 weeks, and levetiracetam was given to prevent seizures. Very slowly, with extensive physical and speech therapy, his condition started to improve. One year after disease onset, a follow-up MRI revealed signs of cerebellar atrophy.

Conclusion: Severe forms of rotavirus-associated cerebellitis can lead to significant neurological impairment and may require intensive care and immunomodulatory treatment. Although the prognosis is more guarded due to a higher risk of prolonged recovery, residual ataxia, and other long-term neurological sequelae, timely management can still lead to significant improvement.

Keywords: Encephalitis; Methylprednisolone; Rotavirus

CR107

Long-Term Management of Symptomatic Hypocalcemia Due to Pseudohypoparathyroidism: A Case Report

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Introduction: Hypocalcemia is a metabolic disturbance with diverse clinical manifestations, ranging from asymptomatic or mild neuromuscular symptoms to severe tetany and seizures. Chronic hypocalcemia may remain unrecognized when symptoms are intermittent or nonspecific. Pseudohypoparathyroidism is a rare disorder characterized by resistance of target organs to parathyroid hormone, resulting in hypocalcemia despite normal or elevated hormone levels. Case reports provide insight into diagnostic challenges and long-term outpatient management. The aim of this report is to describe the clinical course and management of an adult patient with symptomatic hypocalcemia due to pseudohypoparathyroidism.

Case report: A 28-year-old female patient, with a history of hypothyroidism and asthma, presented in April 2021 following a generalized epileptic seizure. Emergency evaluation revealed marked hypocalcemia with reduced ionized calcium and QT prolongation on electrocardiography, while brain imaging was unremarkable. She reported a history of recurrent tetany, carpopedal spasms, and paresthesias over several years. Intravenous calcium gluconate led to rapid clinical improvement. Endocrine evaluation demonstrated persistently low calcium levels with inappropriately elevated parathyroid hormone, normal renal function, and no structural thyroid abnormalities, consistent with pseudohypoparathyroidism. Treatment with calcitriol, oral calcium, and magnesium supplementation was initiated, alongside continued levothyroxine therapy. During long-term outpatient follow-up, spanning from April 2021 to April 2024, metabolic parameters stabilized and no further seizures or tetanic episodes occurred.

Conclusion: This case emphasizes the importance of considering rare endocrine causes in patients with unexplained seizures and neuromuscular symptoms. Structured long-term outpatient follow-up and targeted metabolic therapy ensured sustained clinical stability and prevention of recurrent neurological manifestations.

Keywords: Calcitriol; Calcium; Hypocalcemia; Pseudohypoparathyroidism

CR108

Targeted therapy in severe psoriasis – a case report

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Introduction: Psoriasis is a chronic immune-mediated inflammatory disease driven by dysregulation of the interleukin (IL)-23/IL-17 axis. While tumor necrosis factor alpha (TNF- α) and IL-23 inhibitors act upstream in the inflammatory cascade, IL-17 is a key effector cytokine directly responsible for psoriatic lesion development and a critical therapeutic target. IL-17 can be produced independently of IL-23 by various immune cells which may contribute to variable clinical responses to IL-23 inhibitor therapy in psoriasis.

Case report: We report a 62-year-old man with a 33-year history of severe refractory psoriasis and an extremely high Psoriasis Area and Severity Index (PASI) score of 47.3, presenting with generalized erythematous-squamous plaques, severe palmoplantar hyperkeratosis, and psoriatic arthritis. In 2018, initial treatments with acitretin, adalimumab (TNF- α inhibitor) and secukinumab (IL-17 inhibitor) were ineffective. Therapy was switched to a second IL-17 inhibitor ixekizumab, resulting in complete remission (PASI 0). However, a severe relapse occurred after one year (PASI 32.4), probably due to anti-drug antibody formation. Afterwards, IL-23 inhibitors were introduced (risankizumab and guselkumab), but without a positive clinical impact. After all therapeutic options were exhausted, it was recommended to wait for next-generation drugs. In 2024, the next-generation IL-17 inhibitor bimekizumab was initiated, leading to complete lesion regression (PASI 0). The therapy has been continued to this day with maintained therapeutic effect.

Conclusion: Given the psoriasis pathophysiology, achieving and maintaining remission with the initial therapy was challenging. Therefore, this case highlights the importance of individualized treatment for each patient, which often requires medication changes. In this case, the lack of response to inhibitors may be due to indirect suppression of IL-17-driven inflammation, while loss of efficacy of the second IL-17 inhibitor may be immunogenicity-related.

Keywords: Biological Products; Interleukin-17; Psoriasis

CR109

Pyonephrosis Complicated by Septic Shock in an Adolescent Girl: A Case Report

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Introduction: Urinary tract infections in adolescence may have a dramatic course when associated with structural obstruction. Unlike early childhood pyelonephritis, commonly ascending in origin, infection in older adolescents requires evaluation for anatomical predisposition. We present a 17.5- year-old girl with pyonephrosis secondary to pelviureteric junction (PUJ) obstruction, complicated by septic shock.

Case report: The patient was admitted with abdominal pain, vomiting and a palpable right-sided mass. Imaging revealed grade IV hydronephrosis with a 15×8.5×22.5 cm cystic dilatation of the right collecting system, markedly thinned parenchyma (≤ 4 mm) and PUJ obstruction, indicating chronic obstructive uropathy as the predisposing factor. Emergency percutaneous nephrostomy was performed, evacuating approximately 1500 mL of purulent urine, and a JJ stent was inserted to secure drainage and preserve function. Postoperatively, septic shock developed, requiring fluid resuscitation and noradrenaline. Broad-spectrum antimicrobial therapy (meropenem and vancomycin) was initiated empirically due to hemodynamic instability and risk of multidrug-resistant pathogens in obstructive urosepsis. Urine culture later confirmed *Escherichia coli*. Given the severity of septic shock and favorable clinical response, antimicrobial therapy was continued without de-escalation. Transient bilateral pleural effusions resolved with conservative management. Dynamic renal scintigraphy (Tc-99m MAG3) showed preserved left kidney function and impaired right kidney function (23% differential function) with persistent obstruction, confirming a severely compromised but functional kidney. At discharge, the patient was afebrile, normotensive (105/70 mmHg), with stable renal function and adequate diuresis; JJ stent remained *in situ* with planned follow-up.

Conclusion: This case illustrates pyonephrosis due to previously unrecognized PUJ obstruction, leading to septic shock and marked functional loss of the affected kidney. Early recognition of obstruction and prompt decompression with empiric antimicrobial therapy are crucial to prevent irreversible renal damage and complications.

Keywords: Pyonephrosis; Shock, Septic; Urinary Tract Infections

CR110

Role of Genomic Imprinting in Autism Spectrum Disorder Due to Maternal 15q11.2–q13.1 Duplication

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Introduction: Maternal 15q duplication syndrome is a genetic disorder characterised by autism spectrum disorder (ASD), hypotonia, developmental delay (DD), and epilepsy resulting from a maternally derived copy of the Prader-Willi/Angelman critical region (PWACR) located on chromosome 15. Maternal interstitial 15q duplication is typically *de novo* mutation but can also be inherited from the mother in approximately 15% of cases. Paternal duplication is presented by ASD in less than 50% of cases or can be clinically unnoticeable.

Case report: A 4-year-old boy was referred to a geneticist due to ASD, DD, and epileptic seizures. Molecular karyotyping and Methylation-specific Multiplex Ligation-dependent Probe Amplification (MS-MLPA) method revealed a duplication from BP1 to BP3 in the 15q11.2-q13.1 region which involves the PWACR. According to the results of methylation profiling, the level of methylation for the *SNRPN-3* and *MAGEL2-1* genes was approximately 70%, indicating the maternal origin. The rest of the family was tested, including an 8-year-old sister who presented with ASD, and parents without any clinical signs. The same mutation was found in both the sister and mother. The mother was counselled about the 50% risk of having a child with this genetic condition. However, the test results can't predict the phenotype severity. The boy was treated with lamotrigine for epilepsy. Both children attended a special educational institution and underwent habilitation.

Conclusion: This report presents the difference between maternal and paternal origin of the 15q duplication and the importance of genetic diagnosis in mothers without clinical signs, because in rare cases, they can be carriers of the mutation and pass it on to offspring with risk of 50%.

Keywords: Autism Spectrum Disorder; Genomic Imprinting; Karyotyping; Prenatal Diagnosis; Segmental Duplications, Genomic

CR111

When "limited" is not limited: Aggressive presentation of CREST syndrome

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Introduction: Limited systemic sclerosis (lcSSc) is an autoimmune connective tissue disease characterised by microvascular dysfunction and fibrosis of the skin and internal organs; however, the term limited refers only to the extent of skin involvement and not to internal manifestations. LcSSc often presents with calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia, collectively called CREST syndrome. We report a case complicated by systemic sclerosis-associated interstitial lung disease (SSc-ILD), pulmonary arterial hypertension (PAH), digital ischaemia, and small intestinal bacterial overgrowth (SIBO).

Case report: A 59-year-old woman with a 15-year history of Raynaud's phenomenon and gastroesophageal reflux disease presented with progressive skin changes. One year earlier, she developed lower-leg lesions following a tick bite, which were treated as acrodermatitis chronica atrophicans. Subsequently, skin thickening of extremities and digital pitting developed along with cachexia and intermittent dysphagia. Initial rheumatological evaluation revealed an active scleroderma pattern on nailfold capillaroscopy. Concurrently, serologic testing revealed the presence of anti-centromere antibodies, supporting a diagnosis of lcSSc. Further assessment revealed SSc-ILD on high-resolution computed tomography, while echocardiography suggested PAH. Magnetic resonance enterography showed small bowel wall thickening with luminal narrowing. SIBO was confirmed by breath testing, reflecting gastrointestinal dysmotility and malabsorption that contributed to cachexia. Initial treatment included hydroxychloroquine and rifaximin. Three months later, digital gangrene developed, prompting initiation of bosentan and mycophenolate mofetil. One year later, the patient achieved clinical remission.

Conclusion: This case illustrates a severe and potentially life-threatening presentation of CREST syndrome, with significant pulmonary, cardiac, and gastrointestinal involvement. It underscores the clinical heterogeneity of lcSSc and its potential for aggressive multisystem disease. Importantly, the case shows that timely initiation of targeted therapy can lead to clinical improvement, even in advanced disease.

Keywords: Acrodermatitis; Gangrene; Gastrointestinal Diseases; Immunotherapy; Scleroderma, Limited

CR112

Chronic Wound - A Disease of Many Faces

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Introduction: A chronic wound is a lesion which does not heal within the expected period of time, often due to repeated trauma, ischemia, infection, or underlying systemic conditions. The prolonged inflammation and impaired tissue repair may obscure or delay the recognition of other pathological processes. Basal cell carcinoma (BCC) is the most common skin cancer, originating from basal epidermal cells and characterized by slow growth and local invasiveness, with a low tendency to metastasize. This clinical case highlights a diagnostic challenge created by the presence of the two conditions.

Case report: A 72-year-old female presented with a chronic wound on the left lower leg that had persisted for three years following local trauma. Initial treatment by a vascular surgeon included twenty sessions of hyperbaric oxygen therapy and varicose vein surgery, resulting in temporary improvement. However, the lesion later enlarged to 5 x 2,5 cm, with bleeding and granulation tissue at the base. Over the next six months, the wound was treated with silver dressings, silicone foam adhesive dressings, neutral moisturizing cream, and venotonic and vasoprotecting medication. The wound decreased in size to 1,2 x 1,5 cm but failed to heal completely. Eventually, an excisional biopsy was performed due to a change in the clinical presentation of the chronic wound, followed by the histopathological confirmation of the underlying BCC. Subsequently, surgical removal of the tumor was performed.

Conclusion: This case emphasizes the importance of considering underlying malignancy in chronic, non-healing wounds, particularly when standard treatment fails. Early biopsy or excision should be considered to avoid delayed diagnosis and ensure appropriate management.

Keywords: Basal Cell Carcinoma; Skin Neoplasms; Wound Healing

CR113

Chronic spontaneous urticaria – a case report

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Introduction: Chronic spontaneous urticaria (CSU) is a Th2-dominant chronic inflammatory disease characterized by recurrent wheals lasting longer than six weeks without an identifiable external trigger. Due to shared Th2-driven mechanisms, CSU frequently coexists with other atopic and eosinophilic diseases as observed in this patient. The aim of this report is to highlight the role of targeted biological therapy in the management of CSU with comorbid atopic disease.

Case report: We present a 42-year-old woman with a 17-year history of allergic rhinitis, nasal polyposis, chronic rhinosinusitis, and allergic asthma. She was initially treated with oral and intranasal corticosteroids and antihistamines. Over the past seven years, she reported a progressive worsening of symptoms despite ongoing therapy. Owing to frequent exacerbations, biological therapy with benralizumab was initiated, resulting in significant clinical improvement. Several months later, the patient developed acute urticaria which persisted and evolved into CSU. Despite treatment with corticosteroids and antihistamines, symptoms persisted. Given the coexistence of CSU, asthma, and nasal polyposis, therapy was switched from benralizumab to omalizumab. After three months, complete resolution of urticarial lesions was achieved, along with a favorable effect on asthma control and nasal polyposis. Continuous treatment with omalizumab, with extended dosing intervals, was maintained due to sustained disease control. Asthma and nasal polyposis also remained clinically well controlled.

Conclusion: This case emphasizes IgE-mediated inflammation as a common pathogenic pathway in chronic spontaneous urticaria, allergic asthma, and chronic rhinosinusitis with nasal polyps. The successful switch to omalizumab resulted in sustained clinical remission and overall disease control, underscoring the importance of individualized targeted biological therapy in patients with overlapping atopic conditions.

Keywords: Biological Products; Chronic Urticaria; Omalizumab

CR114

Spinal Cord Stimulation as a Last-Resort Therapy for Refractory Chronic Low Back Pain: A Case Report

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Introduction: A spinal cord stimulator (SCS) is an implantable medical device that delivers low-amplitude electrical impulses to the spinal cord to modulate pain transmission and reduce chronic pain. It is typically indicated for patients with refractory pain who have not achieved adequate relief from conservative, operative or minimally invasive treatments.

Case report: We present the case of a 57-year-old male with chronic low back pain beginning in 2018. Initial management included analgesic therapy: opioids, paracetamol and NSAIDs in different combinations per os, which provided limited benefit. In 2019, radiofrequency denervation at the L2/L3 level was performed, resulting in only short-term pain relief. Subsequently, the pain progressed and radiated to the hip region, leading to total hip arthroplasty in 2020, due to avascular necrosis of femoral head. Despite this intervention, the patient's pain persisted. In 2022, an interlaminectomy was performed without significant improvement. Further pain management efforts in 2024 included epidural steroid injections and epiduroscopy, neither of which resulted in meaningful pain reduction or functional improvement. After all conservative, operative and minimally invasive treatment options had been exhausted, the patient was evaluated and deemed a suitable candidate for spinal cord stimulation. In October 2025, an SCS device was successfully implanted. The patient experienced immediate and significant pain relief, with notable improvement in daily activities and quality of life.

Conclusion: This case demonstrates that spinal cord stimulation is an effective last-resort therapy for chronic pain management. However, due to its cost and invasive nature, SCS should be considered only after all other treatment modalities have been thoroughly explored.

Keywords: Chronic Pain; Pain Management; Spinal Cord Stimulation

CR115

GAD65 – Positive Ataxia

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Introduction: Anti-glutamic acid decarboxylase 65 (anti-GAD65) ataxia is a rare immune mediated cerebellar ataxia that mostly affects women in their 60s and typically follows a subacute or chronic course. Autoimmunity toward the GAD65 isoform predominantly affects the cerebellum. The main clinical feature is gait ataxia, associated with limb incoordination, dysmetria, oculomotor ataxia, and scanning speech.

Case report: Our patient is a 52-year-old woman admitted to the Department of Neurology at the University Hospital Centre Osijek. Since 2014, she had suffered from diplopia, left-sided incoordination, and tremor. In 2021, tremor involved the entire body, with progressive gait, speech, and swallowing difficulties. Neurological examination revealed nystagmus, diplopia and dysarthric speech, as well as bilateral dysmetria and limb oscillations in an anti-gravity position. It also showed a positive Migazzini test, a positive rebound phenomenon in both upper limbs, upper limb hyperreflexia with pathological reflexes, gait ataxia, and truncal ataxia evident while sitting. Romberg test was positive even with the eyes open, without lateralization. Given the history of Hashimoto's thyroiditis, autoimmune ataxia was suspected, and the diagnosis was confirmed by high titers of anti-GAD65 antibodies in serum. The patient received corticosteroids, physiotherapy, and intravenous immunoglobulins without significant improvement. Subsequent intravenous rituximab kept her condition relatively stable.

Conclusion: As this patient with chronic anti-GAD65 ataxia showed only a limited response to immunotherapy, treatment focused on symptomatic management with physiotherapy, psychotherapy, speech therapy, and medications for stiffness. Because anti-GAD65 ataxia is a rare yet treatable immune-mediated cause of ataxia, early recognition and timely initiation of immunotherapy are crucial, and this subtype should be considered in patients with otherwise unexplained cerebellar ataxia.

Keywords: Autoantibodies; Cerebellar Ataxia; Immunotherapy

CR116

Dose-Adjusted Approach in the Era of Precision Medicine: Managing Complications of DA-R-EPOCH in a Patient with PMBCL**Marko Margetić¹; Lara Rončević¹; Sara Burko¹; Katarina Domjanović¹; Vlatka Periša^{1,2}***1 - Faculty of Medicine, J. J. Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Hematology, Clinic of Internal Medicine, University Hospital Center Osijek, Osijek, Croatia*

Introduction: Primary mediastinal B-cell lymphoma (PMBCL) is an aggressive subtype of diffuse large B-cell lymphoma characterized by a large anterior mediastinal mass with immunophenotypic features. The dose-adjusted rituximab plus etoposide, prednisone, vincristine (Oncovin), cyclophosphamide, and doxorubicin (DA-R-EPOCH) regimen is the standard of care. However, dose escalation leads to severe hematologic toxicities, including prolonged neutropenia and life-threatening infections, requiring precise individualization and supportive care.

Case report: A 46-year-old male patient with cervicofacial edema, bilateral axillary lymphadenopathy, and progressive dyspnea presented to the emergency room. Chest X-ray and computed tomography (CT) revealed a mediastinal mass (88mm) with superior vena cava infiltration and bilateral pleural effusions. Histopathological examination confirmed Ann Arbor stage IV PMBCL, with a high Ki-67 proliferation index of 95% and positive expression of MYC by immunohistochemistry. FISH testing showed presence of MYC, BCL2, and BCL6 rearrangements. Treatment with the DA-R-EPOCH protocol was indicated, and after three cycles, follow-up CT showed significant tumor regression (45 mm), supporting dose escalation up to 140%. Additionally, during the fifth cycle, the patient developed febrile neutropenia (absolute neutrophil count < 0.1), pancytopenia, and right-sided pneumonia. Intervention included antifungal therapy, broad-spectrum antibiotics, and supportive care with granulocyte colony-stimulating factors and irradiated blood component transfusions. After clinical improvement and a reduction in inflammatory markers, chemotherapy was continued at 130%. Six completed cycles led to a good therapeutic response and successful transition to consolidated radiotherapy.

Conclusion: This case emphasizes that early diagnosis, appropriate antimicrobial therapy, and hematopoietic support are essential to reduce immunologic risks and manage potential complications. Additionally, a tailored approach enables the safe delivery of maximal-intensity therapy in achieving complete metabolic remission in high-risk patients.

Keywords: Antineoplastic Combined Chemotherapy Protocols; Febrile Neutropenia; Mediastinal Neoplasms; Precision Medicine

CR117

One Symptom, Two Antibodies: A Personalized Approach to Optic Neuritis

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Introduction: Optic neuritis is a common cause of acute visual loss in young adults and is most frequently associated with demyelinating diseases of the central nervous system. However, advances in immunological diagnostics have revealed distinct pathogenic entities with overlapping clinical presentations but fundamentally different prognostic and therapeutic implications. Among these, myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) represents a separate inflammatory disorder defined by the presence of MOG-IgG antibodies.

Case report: A 19-year-old previously healthy man presented with sudden visual loss in the right eye, severe pain on eye movement, and impaired color and contrast perception lasting three days. Ophthalmological examination revealed reduced visual acuity (0.1), mild optic disc edema, and markedly impaired color vision on Ishihara testing (2/24 plates) in the affected eye. Magnetic resonance imaging of the brain and orbits demonstrated thickening and contrast enhancement of the right optic nerve without demyelinating lesions suggestive of multiple sclerosis. Immunological testing showed negative aquaporin-4 antibodies and high-titer MOG-IgG positivity, establishing the diagnosis of MOG-associated optic neuritis. Initial treatment with high-dose intravenous corticosteroids led to partial visual recovery, followed by an early relapse after treatment cessation. Targeted immunomodulatory therapy was subsequently initiated in collaboration with the Department of Neurology. During 12 months of follow-up, no further relapses occurred, visual acuity improved to 0.9, pain completely resolved, and optical coherence tomography demonstrated preserved optic nerve structure.

Conclusion: This case highlights the importance of immunological biomarkers in the differential diagnosis of optic neuritis. Although clinically indistinguishable at presentation, MOG-associated optic neuritis represents a distinct disease entity requiring a tailored therapeutic approach. Early identification of MOG-IgG enables personalized treatment strategies and significantly improves visual outcomes, underscoring the principles of contemporary personalized medicine.

Keywords: Aquaporin 4; Immunoglobulin G; Immunomodulation; Myelin-Oligodendrocyte Glycoprotein; Optic Neuritis

CR118

Treatment challenges in persistent atopic dermatitis in an adult patient

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Introduction: Atopic dermatitis (AD) is a chronic inflammatory skin disease that most often appears in childhood. In most patients symptoms resolve during adolescence but in some patients they persist. Persistent AD is characterized by a complex immunopathogenesis with chronic immune activation, predominantly Th2 response with the involvement of other signaling pathways (Th1, Th17, Th22). This often requires long-term systemic therapy.

Case report: A 41-year-old male patient has been treated for AD since birth. In 2017. he came to the dermatology department with a clinical picture of generalized AD, previously treated with corticosteroids and local therapy. In March 2021. With a SCORAD value 70.65 cyclosporine therapy was initiated. Due to the longer-term disease control the patient was treated with three different JAK inhibitors in different treatment periods over the following years. Therapy was discontinued due the side effects such as herpetic blepharitis and a gradual loss of efficiency. Herpetic blepharitis was caused by reactivation of herpes simplex virus type 1 (HSV-1), a known risk associated with JAK inhibitor therapy due to immunomodulatory effects on antiviral interferon signaling. Methotrexate was eventually initiated, which patient continues to take to this day. Despite the therapy, SCORAD fluctuates between 20-40.

Conclusion: This case report highlights the complex immunopathogenesis of adult-onset AD. The need for multiple systemic therapy from different therapeutic groups reflects the heterogeneity of the immune pathways and limitations of available therapeutic options making it a clinical and therapeutic challenge.

Keywords: Dermatitis; Skin Diseases; Janus Kinase Inhibitors

CR119

An Autoinflammatory Crisis Masquerading as Septic Shock

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Introduction: Autoinflammatory diseases represent diagnostically challenging disorders characterized by dysregulated innate immune activation. Their clinical presentation frequently mimics severe infection, resulting in delayed recognition and inappropriate therapeutic interventions. Recent advances in genomic medicine have enabled rapid identification of monogenic immune disorders, facilitating targeted therapy even in acute emergency contexts.

Case report: A 23-year-old male patient presented to the emergency department with recurrent high-grade fever, severe abdominal pain, and hypotension. Throughout the preceding year, he had experienced multiple hospitalizations for presumed sepsis, each yielding negative microbiological findings and demonstrating only a transient response to broad-spectrum antibiotics. Physical examination revealed fever, tachycardia, and circulatory shock. Laboratory investigations demonstrated markedly elevated C-reactive protein and ferritin concentrations, neutrophilia, and normal procalcitonin values, suggesting a non-infectious hyperinflammatory process. Contrast-enhanced abdominal computed tomography revealed diffuse serositis without a focal infectious source. Considering the recurrent sterile inflammatory episodes and Mediterranean ancestry, hereditary autoinflammatory syndrome was suspected. Rapid genetic testing identified a pathogenic *MEFV* gene mutation, establishing the diagnosis of familial Mediterranean fever with macrophage activation syndrome precipitating circulatory shock. The immediate administration of high-dose corticosteroids, combined with interleukin-1 receptor antagonist therapy, resulted in rapid clinical improvement, with complete fever resolution and hemodynamic stabilization within 48 hours. The patient was discharged on targeted biologic therapy with comprehensive follow-up.

Conclusion: This case underscores the critical importance of considering autoinflammatory syndromes in patients presenting with recurrent sterile inflammatory episodes, particularly those of Mediterranean ancestry. The integration of rapid genomic testing into acute care settings represents a paradigm shift in emergency medicine, enabling clinicians to differentiate complex immune dysregulation from infectious processes and institute life-saving targeted immunomodulation.

Keywords: Familial Mediterranean Fever; Hereditary Autoinflammatory Diseases; Macrophage Activation Syndrome; Sepsis

CR120

From growth failure to hematopoietic stem cell transplantation: a case report of a patient with Schimke immuno-osseous dysplasia

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Introduction: Schimke immuno-osseous dysplasia (SIOD) is a rare autosomal recessive condition characterized by disproportionate short stature, spondyloepiphyseal dysplasia, steroid-resistant nephrotic syndrome, hyperpigmented skin macules, and immunodeficiency. We report a case that illustrates the complexity of this entity.

Case report: A six-year-old boy was referred to our hospital for evaluation of growth retardation. Physical examination revealed disproportionate short stature and multiple hyperpigmented skin lesions. Radiological findings were suggestive of spondyloepiphyseal dysplasia. Based on the clinical presentation and radiological findings, clinical exome sequencing was undertaken, revealing a nonsense homozygous pathogenic variant in the *SMARCAL1* gene, consistent with a diagnosis of SIOD. Accordingly, an extensive multidisciplinary evaluation was conducted to assess disease-related manifestations. The patient was diagnosed with nephrotic syndrome, hypovitaminosis D, and arterial hypertension. Renal biopsy demonstrated focal segmental glomerulosclerosis, which results in steroid-resistant nephrotic syndrome as a manifestation of the underlying disease, as well as immunocomplex-mediated glomerulonephritis, which was the reason to start corticosteroid therapy. Dual antihypertensive was also initiated. Due to persistent nephrotic-range proteinuria and hypoalbuminemia, albumin replacement therapy was administered. Immunological evaluation demonstrated combined T- and B-cell immunodeficiency with severe functional T-lymphocyte deficiency. Therefore, prophylactic therapy with sulfamethoxazole–trimethoprim was initiated, and the patient has been receiving immunoglobulin therapy. Considering the rapid deterioration of renal function and severe immunodeficiency, the patient was presented to the hematopoietic stem cell transplantation team.

Conclusion: Patients with this diagnosis, particularly those with nonsense mutations, frequently require hematopoietic stem cell transplantation and kidney transplantation from the same donor. Therefore, the definitive plan is to refer this patient to an experienced international center for a combined bone marrow and kidney transplantation.

Keywords: Bone Marrow Transplantation; Kidney Transplantation; Nephrotic Syndrome; Osteochondrodysplasias

CR121

Personalized immunotherapy in successful treatment of myocarditis as first presentation of systemic lupus erythematosus – case report

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Introduction: Systemic Lupus Erythematosus (SLE) is a complex autoimmune disease which targets multiple organs concomitantly and thus complicates diagnosis and treatment. Lupus myocarditis can present as an acute disease, however, can also be subclinical and more difficult to diagnose. A high-mortality cardiovascular complication of SLE is myocarditis, an uncommon but severe complication. We present a case with acute heart failure as a first manifestation of SLE.

Case report: A 46-year-old woman was admitted due to global heart failure. Patient presented with fatigue, dyspnoea, peripheral oedema and gastroenteritis. She had no history of significant comorbidities. Upon admission, a transthoracic echocardiogram showed a decreased left ventricular ejection fraction (LVEF) of 30%, dilation of the left ventricle, severe secondary mitral regurgitation, and mild tricuspid regurgitation. She was also diagnosed with deep vein thrombosis and acute renal failure. Alongside elevated values of NT-pro-BNP, the high values of hs-troponin lead to cardiac magnetic imaging revealing myocarditis as the aetiology of the dilated cardiomyopathy. Further laboratory findings confirmed SLE as the aetiology of heart and renal failure. Initially, she was treated with glucocorticoids and heart failure medications. Due to lack of response to initiated treatment, immunoadsorption therapy alongside intravenous immunoglobulin treatment (IVIg) was initiated, followed with immunosuppressant medication cyclophosphamide. Patient responded favourable and reported a significant clinical improvement, confirmed by echocardiographic improvement of the systolic function of LVEF up to 50% and improvement of renal function.

Conclusion: Our case emphasizes both, the complicated differential diagnosis of cardiac involvement in SLE, and the beneficial role of up-titration and personalized SLE treatment with immunosuppressant medication cyclophosphamide, immunoadsorption therapy and IVIg treatment, as well as glucocorticoids in acute cases with cardiac involvement to achieve a more favourable outcome.

Keywords: Immunotherapy; Lupus Erythematosus, Systemic; Myocarditis; Personalized medicine

CR122

Basal cell adenocarcinoma of the parotid gland: a case report**Nina Matić¹ ; Jana Mateljić²; Drago Boščić³***1 - Health Center Zagreb – Center, Zagreb, Croatia**2 - Family Medicine Practice of Sanja Klajić Grotić, MD, Zagreb, Croatia**3 - Department of Otorhinolaryngology and Head and Neck Surgery, Dubrava University Hospital, Zagreb, Croatia*

Introduction: Basal cell adenocarcinoma (BCAC) is a rare malignant neoplasm that accounts for approximately 1-2% of all salivary gland tumors. It can affect any salivary gland, but the parotid gland is the most common site. BCAC typically presents as a slow-growing, painless mass. Although the tumor may be locally invasive with reported recurrences, it has low potential for distant metastasis. Cytologic diagnosis is challenging because of morphological similarities with salivary tumors that occur more often, such as adenoid cystic carcinoma and pleomorphic adenoma.

Case report: A 71-year-old female patient presented with a palpable, painless mass below her right ear, which she had first noticed approximately two months prior to examination. No other symptoms were reported. An ultrasound showed an irregularly shaped, inhomogeneous lesion, measuring 20x16 mm, with poor vascularization. No pathologically enlarged lymph nodes were detected. Fine-needle aspiration cytology suggested a differential diagnosis of pleomorphic adenoma with cylindromatous features or adenoid cystic carcinoma. The patient underwent a right superficial parotidectomy. Postoperative histopathological diagnosis confirmed basal cell adenocarcinoma. The tumor was completely removed with no evidence of metastasis, and no further treatment was required. The patient recovered well postoperatively, without complications, and remains under regular follow-up with no signs of disease recurrence.

Conclusion: Due to its cytomorphological similarity with more prevalent salivary gland tumors, basal cell adenocarcinoma presents a diagnostic challenge. This case highlights limitations of fine-needle aspiration cytology in establishing a preoperative diagnosis of basal cell adenocarcinoma and shows the essential role of histopathological examination following surgical excision.

Keywords: Adenocarcinoma; Fine-Needle Biopsy; Parotid Gland

CR123

When the Past Returns: Late Pulmonary Metastasis of Ovarian Endometrioid Adenocarcinoma

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Introduction: Pulmonary metastases of ovarian adenocarcinoma pose significant diagnostic challenge when they occur decades after treatment of the primary tumor. Accurate diagnosis is essential, as pulmonary lesions in elderly patients are frequently misinterpreted as primary lung carcinoma, leading to inappropriate treatment.

Case Report: A 75-year-old female smoker was referred for evaluation of a persistent cough unresponsive to antibiotics. Her medical history included total hysterectomy with bilateral adnexectomy performed 30 years ago for endometrioid ovarian adenocarcinoma, followed by adjuvant chemotherapy. Chest computed tomography (CT) revealed a solitary mass measuring 28x32x36 mm in the lower lobe of the left lung. A CT-guided transthoracic biopsy revealed tumour tissue composed of gland-forming atypical epithelial cells. However, the tumor origin remained unclear on initial immunohistochemical evaluation. The patient underwent left lower lobectomy with complete tumor resection. Histopathological examination revealed a tumour composed of atypical epithelial cells forming glandular and cribriform structures. Immunohistochemically, tumour cells displayed positivity for cytokeratin7, paired-box gene 8, β -catenin and vimentin. Wild type p53 expression was observed. Estrogen receptor was positive in more than 95% and progesterone receptor was positive in 70% of tumor cells. These findings supported endometrioid differentiation, consistent with pulmonary metastasis from the previously diagnosed ovarian carcinoma.

Conclusion: This case underscores the importance of comprehensive evaluation of pulmonary tumors in patients with a remote oncologic history. Recognition of extremely late metastatic recurrence is essential to ensure correct tumor classification, and guide appropriate patient management.

Keywords: Adenocarcinoma; Immunohistochemistry; Neoplasm Metastasis

CR124

Immunotherapy in Advanced Squamous Cell Carcinoma of the Head and Neck: A Case Report

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Introduction: Squamous cell carcinoma is a common malignant tumor of the head and neck, characterized by aggressive local growth and recurrence. In locally advanced or inoperable disease, immunotherapy with PD-1 receptor inhibitors represents a therapeutic advancement.

Case report: In March 2023, a 66-year-old man presented with progressive hoarseness. Computed tomography (CT) of the neck revealed a tumor involving the right vocal cord and glottis with extension to the arytenoid cartilage. In April 2023, the patient underwent tracheotomy, total laryngectomy, and total thyroidectomy. Histopathological analysis confirmed squamous cell carcinoma T4aN0M0, with a combined positive score (CPS) of 20. After multidisciplinary team (MDT) evaluation, adjuvant radiotherapy was indicated with a total dose of 66 Gy delivered in 33 fractions. After that, the patient noticed a lesion on the right auricle. CT imaging demonstrated infiltration of the auricle and parotid gland with perineural spread, and histopathological examination confirmed cutaneous squamous cell carcinoma. Due to inoperability and inability to deliver additional radiotherapy, the MDT recommended immunotherapy with cemiplimab at a dose of 350 mg every three weeks. After seven cycles, complete regression of cutaneous neck metastases and the tumor of the right auricle was observed, and treatment was continued for a planned duration of two years. CT imaging revealed an inoperable recurrence of squamous cell carcinoma of the larynx (first primary carcinoma). Despite a CPS value of 20, treatment with cetuximab, was initiated due to concerns regarding unpredictable long-term adverse effects of prior immunotherapy.

Conclusion: This case report demonstrates the efficacy of cemiplimab in inoperable cutaneous squamous cell carcinoma. Management of laryngeal carcinoma requires an individualized approach, and sequential treatment guided by clinical course and MDT decision-making represents a rational therapeutic strategy.

Keywords: Cemiplimab; Immunotherapy; Squamous Cell Carcinoma

CR125

Iatrogenic Kaposi's Sarcoma in a Patient with Myasthenia Gravis: A Case Report

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Introduction: Kaposi's sarcoma (KS), caused by human herpesvirus 8 (HHV-8), is a lymphangioproliferative tumor that typically presents as cutaneous or mucosal patches and nodules. There are four types of KS: classic, endemic, immunosuppressive, and acquired immunodeficiency syndrome-related KS. While immunosuppressive KS commonly occurs after organ transplantation, iatrogenic cases unrelated to transplantation are rare. Here, we present a rare case of corticosteroid-induced KS in a patient with myasthenia gravis (MG).

Case report: A 64-year-old patient with anti-N-AChR-positive generalized MG on oral corticosteroids presented with erythematous changes on the right heel. Dermatological examination suggested KS, and excision confirmed nodular KS with a tumor thickness of 0.9 mm. Immunohistochemical analysis was positive for CD31, CD34, and HHV-8, with no dermal infiltration or endothelial proliferation observed. Steroid withdrawal and substitution with pyridostigmine bromide was considered but not pursued due to MG-related visual problems. Alternative strategies, including the use of other immunosuppressive agents, were also discussed but deemed unsuitable given the patient's clinical status. Corticosteroids were therefore continued, and follow-up examinations revealed disease progression with two purple macules on the back of the left lower leg, which were completely excised, confirming KS. The patient remains under close monitoring, including dermatological examinations, lymph node ultrasonography, and computed tomography (CT) scans, with no signs of further disease progression.

Conclusion: Iatrogenic KS is a rare adverse event in immunosuppressed patients, most commonly post-organ transplantation, but also occurring in those treated for proliferative or immune-mediated disorders. In patients with MG receiving chronic immunosuppressive therapy, particularly corticosteroids, individual risk assessment and careful monitoring are crucial, especially in those predisposed to HHV-8 reactivation. Management requires a careful balance to control both conditions, potentially involving new immunosuppressives or anti-tumor agents.

Keywords: Glucocorticoids; Herpesvirus 8, Human; Myasthenia Gravis; Sarcoma, Kaposi

CR126

Severe psoriasis vulgaris in an adolescent: therapeutic transition from methotrexate to biologic therapy**Lara Milostić¹; Elena Vujnović¹; Iva Strelec¹; Lucija Pešo¹; Marijana Vičić²***1 - Faculty of Medicine, University of Rijeka, Rijeka, Croatia**2 - Department of Dermatovenereology, University Hospital Centre Rijeka; Faculty of Medicine, University of Rijeka, Rijeka, Croatia*

Introduction: Psoriasis vulgaris is a chronic inflammatory skin disease that can significantly impair quality of life in patients. Moderate-to-severe forms, especially when associated with joint symptoms or inadequate response to topical therapy, require systemic treatment. This case report highlights therapeutic challenges and the role of biologic therapy in an adolescent with severe psoriasis vulgaris.

Case report: A 15-year-old girl presented with worsening psoriasis lasting over six months, accompanied by severe evening pruritus, impaired daily functioning and school attendance, and intermittent joint pain. Previous topical therapy with betamethasone and calcipotriol had become ineffective. At the presentation, Body Surface Area (BSA) involvement was 12%, and Dermatology life Quality Index (DLQI) score was 19. Following an appropriate baseline evaluation, systemic therapy with methotrexate was initiated at a dose of 7.5 mg once weekly, with folic acid supplementation and regular emollient use. At follow-up month later, complete regression of erythematous squamous lesions was observed, with residual post-inflammatory hypopigmentation. The disease remained stable during regular three-month follow-ups until February 2025, when clinical exacerbation occurred, characterized by multiple erythematous squamous plaques on the neck, trunk, and extremities, without subjective symptoms. Due to insufficient therapeutic response, biologic therapy was considered, and approval was requested in March 2025. From April 2025, the patient was treated with ixekizumab at a dose of 80 mg. At follow-up in January 2026, complete skin clearance was achieved, and the patient tolerated the therapy well without adverse effects. BSA and DLQI score were 0.

Conclusion: This case underscores the importance of timely therapeutic escalation in severe pediatric psoriasis vulgaris. Biologic therapy with ixekizumab proved to be a safe and effective option in an adolescent with inadequate response to methotrexate, leading to sustained disease control.

Keywords: Biological Therapy; Methotrexate; Psoriasis

CR127

Late-Onset Bilateral Pulmonary Embolism Following Remission of Small Cell Lung Cancer

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Introduction: The occurrence of bilateral pulmonary embolism (PE) after remission of small cell lung cancer (SCLC) is rare and poorly studied, as most researches focus on active disease rather than the post-remission period. Although the risk of venous thromboembolism remains elevated in cancer survivors, particularly during the first five years after treatment, bilateral PE in this setting is exceptionally rare.

Case report: This report describes a 73-year-old male with a history of SCLC previously treated with chemotherapy and radiotherapy, who was considered to be in remission for six months and under regular oncological follow-up. Despite being regarded as disease-free, he developed progressive symptoms suggestive of PE. Diagnostic evaluation revealed markedly elevated D-dimer levels. Chest X-ray demonstrated a right infrahilar consolidation suspicious for PE, which was confirmed by computed tomography pulmonary angiography showing bilateral pulmonary embolism with signs of right ventricular overload. Given the high-risk of PE and contraindication to systemic thrombolysis due to underlying malignancy, the patient underwent mechanical thrombectomy. Following the procedure, he achieved hemodynamic stabilization. Transthoracic echocardiography performed three days later demonstrated normal dimensions and contractility of both the right and left ventricles. The patient was subsequently discharged home on long-term anticoagulant therapy consisting of enoxaparin 80 mg twice daily.

Conclusion: This case illustrates a rare but serious complication in patients with SCLC in remission, highlighting the need for continued vigilance for thromboembolic events even after successful oncological treatment. It also demonstrates the role of interventional therapy in selected cases of massive or bilateral PE. Given the scarcity of reported cases, this report provides valuable insight for clinical practice and future research.

Keywords: Pulmonary Embolism; Small Cell Lung Carcinoma; Thrombectomy

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Immunotherapy-Guided Treatment in Breast Cancer with Evolving Tumor Biology: A Case Report

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Introduction: Breast cancer is a dynamic disease that requires an individual multimodal approach. Immunotherapy has emerged as a new promising treatment option, improving outcomes in patients with aggressive triple-negative breast cancer, associated with short progression-free survival and overall survival.

Case report: Woman born in 1969 presented in 2008 with a confirmed lesion in her left breast. Quadrantectomy and removal of eight axillary lymph nodes were performed. Histopathological analysis confirmed invasive carcinoma of no special type, with ER 30%, PR 10%, and positive HER2/neu. She underwent six cycles of epirubicin and cyclophosphamide (EC protocol) in 2009, followed by radiotherapy (RT). Radiological examinations were unremarkable after the completion of therapy and remained stable until 2015, when she reported discomfort in the left breast. Magnetic resonance imaging showed a spiculated lesion and core biopsy confirmed malignancy. Left mastectomy was performed, followed by four cycles of EC and twelve cycles of paclitaxel. Follow-up assessments confirmed another positive therapy response. A new axillary lesion was found in 2019 and removed surgically. Histopathology confirmed triple-negative, PD-L1 positive carcinoma. She underwent RT, declining chemotherapy. Subsequent positron emission tomography-computed tomography done in 2020 showed no active disease, but in 2021 pulmonary and mediastinal metastatic lesions were detected. Instead of standard guideline-based chemotherapy, the oncology board decided to initiate treatment with atezolizumab in combination with nab-paclitaxel as first-line therapy, which was continued for two years until 2023. Since then, the patient is monitored two to three times a year with stable radiological findings and no disease progression.

Conclusion: Choosing immunotherapy over standard guideline-based chemotherapy demonstrates its growing value in treating triple-negative breast cancer and potential to achieve disease control, even in complex cases such as this one.

Keywords: Breast Neoplasms; Combined Modality Therapy; Immunotherapy; Neoplasm Metastasis; Radiotherapy

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Late development of cyanosis in Fontan circulation

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Introduction: The late development of pulmonary fistulas is a recognized complication of Fontan circulation. Chronic venous hypertension and altered pulmonary blood flow may promote the formation of intrapulmonary venovenous fistulas, leading to progressive hypoxemia and cyanosis.

Case report: We present an 18-year-old patient born with a single ventricle – the left ventricular type, a rudimentary right ventricle, and a common atrioventricular valve, who underwent staged surgical palliation with establishment of Fontan circulation. Two years later, the patient developed reduced arterial oxygen saturation (SpO₂ 88–89%). Subsequent cardiac magnetic resonance imaging demonstrated aortopulmonary collateral vessels and pathological venovenous connections within the lungs consistent with intrapulmonary fistulas. Transcatheter coil embolization was performed, resulting in temporary clinical stabilization. Fourteen years later, the patient re-presented with fever, exertional intolerance, and cough, following resolution of the infection, gradual development of cyanosis and a decrease in SpO₂ to 85% were observed. Computed tomography findings suggested the presence of a diffuse capillary intrapulmonary arteriovenous shunt. Subsequent catheterization confirmed abnormal right-to-left intrapulmonary communications, described angiographically as venovenous collaterals. Hemodynamic assessment demonstrated normal mean pressures within the Fontan circuit (8 mmHg) and pulmonary arteries (7 mmHg), and low pulmonary capillary wedge pressure (2–3 mmHg). Sildenafil therapy was initiated, resulting in an increase in SpO₂ to 91%. The patient is currently on the waiting list for interventional closure of the identified fistulas scheduled in a catheterization lab.

Conclusion: This case demonstrates late progression of intrapulmonary fistulas in a Fontan patient despite preserved hemodynamic parameters, emphasizing that clinically significant shunting may develop even in the absence of elevated Fontan pressures or pulmonary vascular resistance, and requires ongoing long-term surveillance.

Keywords: Congenital Heart Defect; Cyanosis; Fontan Procedure

CR130

First Report of Linezolid-Resistant Vancomycin-Resistant *Enterococcus* (LR-VRE) in a Tertiary Care Centre in Eastern Croatia**Vanja Mitrović¹; Maja Bogdan^{2,3}; Domagoj Drenjančević^{2,3}; Christian Kurina⁴; Marko Živkov^{2,3}***1 - Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Medical Microbiology and Infectology, Faculty of Medicine Osijek, Osijek, Croatia**3 - Department of Clinical Microbiology and Hospital Infections, University Hospital Centre Osijek, Osijek, Croatia**4 - Institute of Emergency Medicine of the Osijek-Baranja County, Osijek, Croatia*

Introduction: Vancomycin-resistant *Enterococcus* (VRE) represents a major challenge in hospital settings, particularly when resistance extends to last-line agents such as linezolid. Linezolid-resistant VRE strains remain rare but carry significant clinical and epidemiological implications. We report the first detection of such an isolate in our institution, underscoring the need for vigilant surveillance and antimicrobial stewardship.

Case report: We present a case involving a patient admitted to University Hospital Centre Osijek due to elective surgery of sigmoid colon adenoma. Postoperative complications in form of wound dehiscence and peritonitis required multiple reoperative surgeries, VAC (Vacuum Assisted Closure) therapy, and finally abdominal wall reconstruction. Also, multiple VAC device changes were performed, with intraoperative sampling for microbiological analysis. In one of the intraoperative samples, vancomycin-resistant *Enterococcus faecium* and multidrug-resistant *Pseudomonas aeruginosa* were isolated. The linezolid resistance of the enterococcal isolate was unexpected, and the strain was sent to the Reference centre for antibiotic resistance surveillance, where resistance was confirmed by disk diffusion (zone of inhibition: 8mm) and gradient test (minimum inhibitory concentration: 8.0 µg/mL) methods. Antibiotic therapy was adjusted according to microbiological findings in consultation with infectious disease specialists. Patient received high doses of daptomycin. Finally, his overall condition improved, and he was discharged from the hospital.

Conclusion: The detection of a linezolid-resistant VRE isolate underscores the evolving resistance landscape and the need for robust microbiological surveillance. Confirmatory testing and timely communication between laboratory and clinicians were key to guiding therapy. Ongoing monitoring is necessary to prevent local dissemination of such high-risk strains.

Keywords: Drug Resistance; *Enterococcus faecium*; Linezolid

CR131

Immunodeficiency as the Basis of Prolonged and Recurrent Sepsis in a Preterm Neonate

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Introduction: Preterm infants have an immature immune system characterized by reduced function and low immunoglobulin G (IgG) levels due to insufficient transplacental transfer, increasing their risk for severe, prolonged, and recurrent infections. In such cases, immunodeficiency should be considered as a potential underlying cause. Maternal autoimmune diseases and hypertensive pregnancy disorders can further impair placental immunoglobulin transfer, contributing to neonatal immunological immaturity.

Case report: A male, preterm neonate, was delivered by cesarean section at 30 weeks due to HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets). The pregnancy was complicated by maternal Hashimoto's thyroiditis, thrombophilia, and gestational diabetes. Initial management included non-invasive respiratory support and empirical antimicrobial therapy, with cranial ultrasound showing a grade I intraventricular hemorrhage. On day 12, the infant developed profound apnea, cardiorespiratory arrest, and septic shock with *Escherichia coli* bacteremia. Intensive targeted antimicrobial and supportive treatment was initiated. The clinical course was complicated by profound thrombocytopenia, anemia, coagulopathy, macrohematuria, peripheral ischemic necrosis of the toes, and progression of the cerebral hemorrhage to grade III. Due to cholestatic hepatopathy and recurrent infections, an extensive metabolic and immunological workup was performed, ruling out conditions like galactosemia and cystic fibrosis. Next-Generation Sequencing was also performed and showed no abnormalities. A second life-threatening sepsis episode with *Klebsiella pneumoniae* occurred on day 38. Diagnostic testing revealed persistently low IgG levels in both the infant and mother. Consequently, intravenous immunoglobulin replacement therapy was initiated upon discharge from the unit.

Conclusion: This case highlights the need for prompt immunological evaluation in preterm infants with recurrent severe sepsis. Immunological immaturity, potentially exacerbated by maternal autoimmune disease, significantly impacts clinical outcomes. Early recognition of immunodeficiency is crucial for prognosis and targeted intervention.

Keywords: Autoimmune Diseases; Cerebral Intraventricular Hemorrhage; Immunoglobulins, Intravenous; Sepsis

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Avoiding the Biopsy Trap: An Atypical Musculoskeletal Infection Mimicking a Soft Tissue Tumor

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Introduction: Echinococcosis is a parasitic disease caused by *Echinococcus granulosus*, characterized by hydatid cyst formation whose rupture, spontaneous or iatrogenic, may trigger life-threatening anaphylaxis. While the liver and lungs are most involved, musculoskeletal echinococcosis is very rare even in endemic regions, accounting for 0.9% to 2.5% of all cases. This report describes a soft tissue tumor that turned out to be of parasitic origin, underscoring the need for heightened diagnostic vigilance when evaluating lesions that mimic musculoskeletal neoplasms.

Case report: A 39-year-old woman presented with non-specific systemic symptoms, including myalgia, fatigue, and low-grade fever accompanied by a painless, progressively enlarging mass in the right shoulder region, lasting two months. The lesion was moderately firm, with unremarkable overlying skin. Laboratory investigations, including inflammatory markers, were within normal reference ranges. Ultrasound imaging demonstrated a well-encapsulated mass of unknown etiology measuring approximately 2.5 cm³ within the deltoid muscle. Due to a differential diagnosis that included benign and malignant soft tissue tumors, as well as parasitic lesions, surgical excision was indicated. Consequently, complete surgical excision of the pseudotumor was performed and subsequent histopathological examination revealed it to be a parasitic cyst with a fibrous capsule. Serological testing confirmed *Echinococcus granulosus*. The patient received symptomatic treatment initially, followed by albendazole therapy for other possible loci after disease confirmation.

Conclusion: Soft tissue lesions with tumor-like characteristics and accompanying non-specific symptoms should prompt consideration of echinococcosis in the differential diagnosis. Early recognition is crucial to guide optimal management, allowing for definitive surgical treatment while avoiding biopsy and cyst rupture, thereby reducing the risk of severe complications.

Keywords: Echinococcosis; Hydatid Cyst; Shoulder; Soft Tissue Neoplasms; Therapeutics

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Genicular artery embolization in symptomatic knee osteoarthritis in 74-year-old woman**Rebeka Nađ¹; Katja Lovoković²; Luka Zmaić³; Luka Ileš⁴; Vjekoslav Kopačin¹***1 - University Hospital Center Osijek, Osijek, Croatia**2 - Poliklinika Slavonija, Osijek, Croatia**3 - Health Centre Osijek-Baranja County, Osijek, Croatia**4 - Institute of Emergency Medicine of Vukovar-Srijem County*

Introduction: In recent years, the role of neoangiogenesis in the pathophysiology of knee osteoarthritis (KOA) has gained increasing attention. Genicular artery embolization (GAE) has emerged as a novel, minimally invasive technique aimed at reducing synovial arterial flow and subsequent inflammation. While surgical treatment such as total knee arthroplasty remains the standard option for advanced KOA, GAE represents a potential bridging or interim therapy for patients who do not yet meet criteria for surgery and are refractory to conservative management, including intra-articular corticosteroids and hyaluronic acid injections.

Case report: We present the case of a 74-year-old woman with long-standing bilateral knee pain due to KOA, unresponsive to conservative therapy. The patient underwent GAE on both knees within a two-month interval. Pre-procedurally, the average Visual Analogue Scale (VAS) score for both knees was 6.5, and the average Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) score was 72.8, with higher values recorded for the right knee. The procedures were performed using a transfemoral approach without perioperative complications, and the patient was discharged the following day. After one month, the mean VAS score for both knees dropped to 4, and the mean WOMAC score to 35.41, indicating a notable improvement in pain and function. The patient reported reduced pain in the right knee and occasional discomfort in the left knee only during prolonged activity.

Conclusion: This case report illustrates a significant reduction in pain intensity and improvement in functional status following GAE in an elderly KOA patient. It should, however, be considered a potential temporary or adjunctive therapy rather than an alternative to total knee arthroplasty.

Keywords: Embolization, Therapeutic; Genicular Artery Embolization; Osteoarthritis, Knee; Pain Management

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Individualized neuropsychiatric response to semaglutide: Integrating neurobiological, neuroimmunological and genetic insights

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Introduction: Semaglutide, a *glucagon-like peptide 1* (GLP-1) receptor agonist, is widely used for glycemic control in type 2 diabetes and is often associated with beneficial effects on mood. However, rare neuropsychiatric reactions may occur in patients with latent emotional vulnerability. The aim of this case report is to show the importance of individualized GLP-1 agonist therapy to ensure safety and efficacy in patients with preexisting psychiatric conditions.

Case report: We present the case of a 52-year-old woman with longstanding type 2 diabetes, diabetic retinopathy, and a previously stable anxiety disorder in remission. The initial introduction of semaglutide in 2021 was limited by gastrointestinal side effects, without any neuropsychological manifestations. Reintroduction in May 2024 resulted, within several weeks in affective lability, impulsivity, irritability and reduced social inhibition, inconsistent with her previous psychiatric profile. Somatic and laboratory evaluations revealed no organic cause, suggesting activation of latent psychodynamic vulnerabilities. Stabilization was achieved with escitalopram in standard doses, and alprazolam for acute symptom control. After 4-6 weeks her psychiatric condition stabilized and controlled response to semaglutide was continued, emphasizing the need for a multidisciplinary and personalized approach in patients with a psychiatric history.

Conclusion: Neurobiologically, GLP-1 agonists modulate dopaminergic and serotonergic pathways within the limbic system, while neuroimmunological effects through microglial activation and pro-inflammatory cytokines may further increase neural sensitivity. Individual genetic predisposition and immune profiles may influence susceptibility to pharmacological effects, highlighting the importance of integrating genomic and immunological insights into therapeutic planning.

Keywords: Neurobiology; Pharmacogenetics; Precision Medicine; Semaglutide

CR135

From Cardioprotection to Autoimmunity: Statin-Induced Myopathy in a Female Patient

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Introduction: Immune-Mediated Necrotizing Myopathy (IMNM) is a rare autoimmune muscle disease causing severe, symmetrical weakness in proximal muscles due to muscle fiber death and high creatine kinase levels, often linked to statin use. The estimated incidence of statin-induced IMNM is approximately 2–3 cases per 100,000 patients exposed to statins. The aim of this case report is to raise awareness of statin-induced IMNM as a distinct clinical entity, emphasizing the diagnostic challenges and therapeutic approach.

Case report: A 58-year-old woman with type 2 diabetes and dyslipidemia initially presented with arthralgia, myalgia, increased fatigability, and proximal muscle weakness involving the thigh and upper arm muscles. Laboratory investigations demonstrated elevated serum creatine kinase levels of 3,924 U/L, while thyroid-stimulating hormone and vitamin D levels were within the normal range. Further history revealed that the symptoms began after the initiation of atorvastatin therapy and persisted despite its discontinuation. Consequently, the patient was referred to rheumatologic assessment, which included Human Leukocyte Antigen (HLA) typing. The positive HLA findings in our patient suggest a genetic predisposition that may have facilitated the development of an IMNM following statin exposure. A muscle biopsy of the thigh was performed, and the histopathological findings together with immunophenotyping results, along with positive Anti-3-hydroxy-3-methylglutaryl-coenzyme A reductase (anti-HMGCR) antibodies, were consistent with the referring diagnosis of IMNM. Therapy with methylprednisolone and azathioprine was initiated, resulting in improvement of both muscle strength and serum creatine kinase levels, which were 176 U/L.

Conclusion: Although statins are the cornerstone of cardiovascular disease prevention, the development of statin-induced IMNM necessitates clinical reevaluation. Discontinuation of statins and initiation of alternative therapies allows safe achievement of target cholesterol levels.

Keywords: Creatine Kinase; HLA Antigens; Muscle Weakness; Statins

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Overlapping autoimmunity and demyelination: a case report of diagnostic and therapeutic challenges

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Introduction: MOG (myelin oligodendrocyte glycoprotein) antibody-associated disease (MOGAD) is a rare inflammatory demyelinating disease of the central nervous system caused by anti-MOG autoantibodies. The aim of this case report is to show the importance of early recognition of MOGAD in patients with multiple autoimmune comorbidities.

Case report: We present a 58-year-old woman with rheumatoid arthritis (RA), followed by an immunologist since 2009. She was treated with tumor necrosis factor-alpha (TNF- α) inhibitors, achieving low disease activity. Her comorbidities included autoimmune thyroiditis, arterial hypertension, hyperlipidemia, and status post cardiac myxoma surgery. Over the past three years, she reported intermittent bilateral lower foot discomfort, right-sided facial and tongue numbness, and episodic visual disturbances. Repeated magnetic resonance imaging (MRI) examinations of the central nervous system revealed chronic brain lesions initially interpreted as predominantly vascular, with possible demyelinating features. Subsequent MRI of the cervical and thoracic spine demonstrated multiple intramedullary T2 hyperintense lesions suggestive of a primary demyelinating process. Therefore, TNF- α inhibitor therapy was discontinued. A comprehensive diagnostic workup including cerebrospinal fluid analysis, autoimmune and infectious serology, evoked potentials, and repeated MRI studies was performed. Anti-MOG antibodies were detected in the serum. Other potential diagnoses were excluded by extensive diagnostic evaluation. Based on clinical presentation, radiological findings, and serological results, the patient fulfilled the currently accepted diagnostic criteria for MOGAD. Given the coexistence of RA and MOGAD, rituximab was identified as a therapeutic option indicated for both conditions.

Conclusion: This case highlights the importance of multidisciplinary collaboration in patients with multiple autoimmune diseases and demonstrates that similar treatment may be applicable across different conditions. Additionally, it highlights the ongoing uncertainty regarding the underlying cause of demyelinating lesions, whether related to autoimmunity or immunosuppressive therapy.

Keywords: Autoimmune Diseases; Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease; Rituximab

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Not Every New Lesion Is a Metastasis: Four Primary Malignancies in a Single Patient

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Introduction: Multiple primary malignancies are a rare clinical occurrence, especially when they develop sequentially within the same patient over time. The aim of this case is to highlight the importance of considering additional primary malignancies and to emphasize the role of thorough diagnostic evaluation.

Case report: We present the case of a 70-year-old male patient with a history of multiple malignancies, including a gastric gastrointestinal stromal tumor, supraglottic laryngeal squamous cell carcinoma, and prostate adenocarcinoma. During routine follow-up, a pulmonary lesion was detected, raising suspicion for either metastatic disease or a newly developed primary lung cancer, given the patient's oncological background. Chest computed tomography (CT) revealed a 1.5 cm peripheral consolidative lesion with irregular margins in the apicoposterior segment of the left upper lobe. Bronchoscopy showed no pathological findings. A CT-guided transthoracic needle biopsy was then performed, confirming lung adenocarcinoma. A positron emission tomography/computed tomography (PET/CT) scan showed minimal metabolic activity in the lesion with no abnormal uptake in regional lymph nodes. PET/CT findings and immunohistochemically confirmed thyroid transcription factor-1 positivity favored a primary lung adenocarcinoma over metastatic disease. The patient then underwent thoracic surgical treatment consisting of left upper lobectomy with mediastinal lymphadenectomy. Histopathological examination revealed a well-differentiated papillary non-small cell lung adenocarcinoma (grade 2), staged as pT2aN0M0 (IB), with negative anaplastic lymphoma kinase rearrangement and programmed death-ligand 1 expression <1%. Postoperatively, the patient recovered well and remained in good general condition. After a multidisciplinary team discussion, no adjuvant chemotherapy was indicated.

Conclusion: This case illustrates the diagnostic challenge of distinguishing metastatic disease from multiple primary malignancies. A comprehensive diagnostic approach, including careful evaluation of new lesions, multidisciplinary input, and histopathological confirmation, is essential to ensure accurate diagnosis and appropriate treatment.

Keywords: Adenocarcinoma of Lung; Differential Diagnosis; Multiple Primary Neoplasms

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From Molecular Profiling to Targeted Therapy in Rapidly Progressive Breast Cancer

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Introduction: Rapidly progressing tumors with limited response to standard treatments represent a significant clinical challenge. In such cases, identification of actionable molecular alterations may enable an individualized therapeutic approach. Somatic gene panel testing provides insight into the tumor's genetic profile and may reveal potential targets for personalized interventions.

Case report: A 44-year-old female patient was referred to the oncology center after surgical excision of a primary breast tumor at an outside institution, where postoperative histopathology confirmed invasive ductal carcinoma of the breast, not otherwise specified (NOS), with strong hormone receptor expression and HER2-negative status. As initial therapy, she received pseudoneoadjuvant doxorubicin, cyclophosphamide and paclitaxel, followed by a right skin-sparing mastectomy with axillary dissection, with no residual disease. She then completed endocrine therapy with tamoxifen, and subsequently anastrozole with goserelin for five years. At routine follow-up, computed tomography revealed two liver lesions and suspicious lymph nodes. Biopsy confirmed metastatic breast carcinoma in the liver and fulvestrant–ribociclib therapy was started. The patient showed poor response, progressing within two months with new hepatic metastases – repeated biopsy was identical to prior one. Given the rapid progression and limited response to standard treatment, the patient underwent somatic gene panel testing, performed on tissue obtained from the metastatic liver biopsy. The analysis identified several variants of predictive significance for this tumor type, including *PTEN* deletion, *MDM2* amplification, and *CDKN2A/B* deletion. Based on these molecular findings, the tumor qualified for capivasertib–fulvestrant therapy, resulting in radiologic regression of all hepatic metastases.

Conclusion: This case emphasizes the importance of somatic gene panel testing in refractory disease, demonstrating how molecularly guided therapy can uncover effective treatment options and achieve meaningful clinical and radiologic responses when conventional strategies fail.

Keywords: Breast Cancer; Genetic Testing; Molecular Targeted Therapy; Neoplasm Metastasis; Personalized medicine

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Genomically Profiled p53-Mutant Endometrial Cancer Discovered During Surgery for Suspected Benign Ovarian Tumor

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Introduction: Molecular classification of endometrial cancer based on DNA polymerase epsilon (*POLE*), DNA polymerase delta 1 (*POLD1*), mismatch repair (MMR), and tumor protein p53 gene (*TP53*) status enables more precise prognostic assessment and individualization of oncological treatment. Particularly clinically relevant are cases in which highly aggressive tumor protein p53 (p53)-mutant tumors present clinically and radiologically in a nonspecific manner, appearing as benign adnexal processes. The aim of this case report is to emphasize the importance of integrating genomic insights into decisions regarding surgical and adjuvant treatment in apparently low-risk gynecological tumors.

Case report: A 66-year-old female patient with arterial hypertension and type 2 diabetes mellitus was referred due to a suspected tumor of the right ovary with normal tumor markers. Transvaginal ultrasound and computed tomography of the abdomen and pelvis revealed an anechoic formation of the right adnexa inseparable from the uterus, with no signs of distant disease and a radiologically normal endometrium. Median infraumbilical laparotomy was performed with total abdominal hysterectomy and bilateral adnexectomy; intraoperative analysis of the right ovary confirmed a benign cystadenofibroma. Definitive histopathological findings revealed G2 endometrioid adenocarcinoma of the endometrium, confined to the endometrial layer, with myometrial invasion, without involvement of the cervix, adnexa, or lymphovascular invasion. Immunohistochemical and molecular analysis confirmed estrogen receptor (ER) positivity, progesterone receptor (PR) negativity, intact MMR status, absence of *POLE/POLD1* mutations, and p53-mutant tumor type, which classified the patient in the high-risk group. Accordingly, adjuvant chemotherapy was initiated.

Conclusion: This case highlights that benign adnexal findings and normal markers can mask aggressive p53-mutant endometrial cancer. Routine genomic characterization is therefore crucial for identifying patients requiring intensive surveillance.

Keywords: Endometrial Neoplasms; Immunotherapy; Molecular Targeted Therapy; Carcinoma, Endometrioid

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Hepatocellular Carcinoma in a Patient With Early-Treated Tyrosinemia Type 1: a Case Report

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Introduction: Tyrosinemia type 1 (HT1) is a rare autosomal recessive disease caused by fumarylacetoacetate hydrolase deficiency. If untreated, the consequent accumulation of toxic metabolites leads to progressive liver disease, tubulopathy, neurologic crises and, frequently, to hepatocellular carcinoma (HCC). Although early treatment substantially reduces the risk, hepatocellular carcinoma can still develop in rare cases.

Case report: A 7-month-old girl was diagnosed with HT1 after evaluation for increased aminotransferases found during respiratory infection. She was immediately started on nitisinone therapy combined with a tyrosine- and phenylalanine-restricted diet and remained clinically stable during long-term follow-up. During a routine check-up at age 14, an elevated alpha-fetoprotein (AFP) level of 88 µg/L was found, suggesting possible liver malignancy. Magnetic resonance imaging of the liver revealed multiple hypervascular lesions, with the largest measuring less than 2 centimeters in diameter. Whole-body imaging showed no evidence of extrahepatic disease. A multidisciplinary team, including international experts in tyrosinemia management, recommended transplantation as the treatment of choice, given the presence of multifocal disease confined to the liver and the high risk of disease recurrence. Histopathological examination of the explanted liver found two HCC nodules in the right lobe, without lymphovascular invasion and with clean margins. Postoperatively, she was maintained on immunosuppressive therapy and continues long-term surveillance. Eight years after transplantation, she remains clinically well.

Conclusion: This case reinforces the fact that, although highly effective, early treatment does not eliminate the risk of HCC in these patients. It highlights the importance of structured, lifelong clinical and biochemical assessments, along with regular imaging. Such an approach can enable timely and curative interventions resulting in optimal patient care.

Keywords: alpha-Fetoproteins; Carcinoma, Hepatocellular; Liver Transplantation; Tyrosinemias

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Esophageal variceal bleeding because of portal hypertension and portal vein cavernoma in a bone marrow transplant patient

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Introduction: Varices are dilated veins in the distal esophagus or proximal stomach caused by increased portal venous pressure. Acute variceal bleeding is a medical emergency requiring prompt intervention.

Case report: A 41-year-old male was admitted to the Emergency Department due to hematemesis, which was preceded by a brief loss of consciousness. On admission, the patient reported normal appetite, bowel movements, and urination. He was hospitalized at the Department of Gastroenterology. After hemodynamic stabilization, endoscopic ligation of esophageal varices was performed, successfully stopping the bleeding, and pharmacological therapy for portal hypertension was continued. Since 2017, the patient has been monitored for thrombosis of the portal vein and the main trunk of the superior mesenteric vein, resulting in cavernous transformation and the development of collateral circulation. At that time, splenomegaly and esophageal varices were confirmed. Hematological evaluation in 2019 revealed Janus kinase 2- positive essential thrombocythemia. Due to secondary myelofibrosis, the patient underwent allogeneic hematopoietic stem cell transplantation in February 2024. Later that year, a stent was placed around the common bile duct due to venous compression. During the current hospitalization, a bone marrow biopsy confirmed preserved hematopoietic function, while laboratory findings indicated pancytopenia due to hypersplenism. Given these findings, therapeutic splenectomy and the placement of a splenorenal shunt have been considered as a treatment option for the patient.

Conclusion: This case highlights the importance of timely diagnosis and a multidisciplinary approach in managing esophageal varices associated with portal hypertension. Acute variceal bleeding requires prompt intervention, such as endoscopic ligation. The patient's complex hematological background complicates the clinical picture and necessitates an individualized approach. Planned treatment options are essential for reducing the risk of portal hypertension-related complications and improving prognosis.

Keywords: Bone Marrow Transplantation; Esophageal and Gastric Varices; Hypertension, Portal

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Pleural Effusion as the First Clue of Multiple Myeloma: A Case Report

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Introduction: Multiple myeloma is a plasma cell malignancy characterized by bone marrow infiltration, monoclonal immunoglobulin (M-Ig) production, and multisystem involvement. It primarily affects the skeletal system, kidneys, and hematopoietic function. This case reports an unusual presentation of multiple myeloma with pleural involvement.

Case report: We present a 76-year-old woman with a history of hypertension and osteopenia who was admitted in August 2025 to the emergency room due to interscapular and lumbar pain lasting for ten days. Tests revealed macrocytic anemia along with left-sided pneumonia with a pleural effusion. Vertebroplasty of the seventh thoracic vertebra was performed for vertebral fracture and pain. Within one month, she presented twice with chest pain, fatigue, weakness, anemia, and loss of weight. A repeat X-ray showed nodular shadows on the lungs and bilateral pleural effusion, so the patient was referred for a pleural puncture. Cytological puncture revealed numerous polymorphic atypical plasma cells with malignant nuclei and mitoses. Immunophenotyping confirmed that 58% of cells expressed antigens related to aberrant plasma cells. Bone marrow aspiration demonstrated infiltration by plasma cells, and immunofixation identified M-IgG gammopathy; therefore, the diagnosis of multiple myeloma with plasma cell leukemia was established. The patient was not a candidate for transplantation due to advanced age, and treatment was initiated according to the Daratumumab-Lenalidomide-Dexamethasone protocol with supportive therapy. During follow-up, gradual regression of pleural effusions was observed, with clinical improvement and reduction of skeletal pain.

Conclusion: This case emphasizes the importance of considering multiple myeloma in patients with unexplained cytopenias, pathological fractures, and persistent pleural effusion. Early recognition enables hematologic treatment, which may lead to regression of pleural effusion, clinical improvement, and pain relief, even in non-transplant candidates.

Keywords: Multiple Myeloma; Plasma Cells; Pleural Effusion

CR143

Juvenile Idiopathic Arthritis: From Pathophysiological Insight to Smart Immune Therapy – Case Report

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Introduction: Juvenile idiopathic arthritis (JIA) is the most common chronic inflammatory rheumatic disease in childhood. Clinical presentation depends on genetic factors and incompletely elucidated immunological mechanisms resulting in different subtypes and extraarticular manifestations. American College of Rheumatology (ACR) recommends disease-modifying antirheumatic drugs (DMARDs) in achieving stable clinical and laboratory remission. In patients with chronic anterior uveitis unresponsive to conventional DMARD methotrexate, TNF inhibitors adalimumab and infliximab as biological DMARDs are next options. Subsequent options are not strictly defined and depend on the clinical judgment of the expert team.

Case report: A 19-year-old male who initially presented with right knee swelling and chronic anterior uveitis at the age of 3 is an example of a difficult-to-treat patient. He was initially treated with methotrexate and adalimumab, followed by infliximab due to uncontrolled uveitis. Months after complete remission, he developed anti-infliximab antibodies, resulting in subclinical disseminated intravascular coagulation with diffuse petechiae, mild thrombocytopenia, decreased fibrinogen, and increased D-dimers. Infliximab was discontinued with regression of petechiae and normalization of coagulation parameters. 14 relapses of arthritis and/or chronic uveitis required therapy changes: tocilizumab (IL-6 inhibitor), abatacept (T-cell costimulation modulator), golimumab (TNF- α inhibitor), and tofacitinib (JAK inhibitor) combined with intermittent courses of intraocular, intraarticular, and systemic glucocorticoids and subtotal right knee synovectomy. Exhausting all options, after 15 years of treatment, infliximab with mycophenolate mofetil was cautiously reintroduced and currently maintains disease remission without coagulopathy.

Conclusion: JIA is a heterogeneous disease that often requires therapy switching due to changes in immunological phenotype during the disease course. In this case report we emphasize the importance of tailored, treat-to-target immunotherapy, especially in difficult-to-treat patients, with consideration to chronic uveitis as the most common and extremely serious vision-threatening complication.

Keywords: Arthritis, Juvenile; Biological Products; Immunotherapy; Precision Medicine

CR144

Overlapping MEFV-negative Familial Mediterranean Fever and Adult-onset Still's disease- a case report

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Introduction: Familial Mediterranean fever (FMF) is a monogenic autoinflammatory disease caused by *MEFV* gene mutation, leading to periodic fever, systemic inflammation, and increased risk of renal failure due to amyloidosis. A positive *MEFV* gene test is not required for diagnosis, as negative forms exist in which diagnosis is based on clinical presentation. In contrast, adult-onset Still's disease (AOSD) is a polygenic disorder characterized by daily fever, transient maculopapular rash, and arthritis. Both conditions involve excessive IL-1 activation, resulting in overlapping symptoms and complicating diagnosis, particularly in atypical FMF presentations.

Case report: A 35-year-old male patient has experienced septic-type fevers, abdominal pain, and enterocolitis four times a year since 11 months old. Gluten enteropathy and Crohn's disease had been ruled out. FMF was suspected; however, genetic testing did not reveal *MEFV* mutations. Laboratory tests showed consistently elevated acute phase reactants and hyperferritinemia. A periodic fever syndrome was suspected, and repeated whole-genome sequencing is pending. Initially, the patient was treated with colchicine, but inflammatory markers remained elevated. After one year, colchicine was discontinued due to the development of rhabdomyolysis. Subsequently, suspecting AOSD, the patient was treated with high doses of glucocorticoids and nonsteroidal anti-inflammatory drugs, with a good response. However, after glucocorticoid tapering, the symptoms returned. Finally, the patient was started on anakinra (an IL-1 receptor antagonist), which resulted in complete regression of his symptoms.

Conclusion: The overlap of diseases with the same inflammatory mechanism can make diagnosis and treatment challenging, especially when symptoms are variable. This case underscores that genetic negativity and atypical clinical presentation do not exclude the diagnosis, and highlights the importance of targeted biological therapy, such as IL-1 inhibition, in achieving symptom control.

Keywords: Familial Mediterranean Fever; Interleukin 1 Receptor Antagonist Protein; Still's Disease, Adult-Onset

CR145

Leigh syndrome caused by a pathogenic MT-ND3 variant: a pediatric case report

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Introduction: Leigh syndrome is a rare neurodegenerative disorder caused by impaired mitochondrial energy production, commonly presenting in infancy or early childhood as subacute necrotizing encephalopathy. The aim of this report is to describe the clinical presentation and diagnostic approach in a child with Leigh syndrome.

Case report: A five-year-old boy was referred for neuropediatric evaluation showing impaired eye coordination with additional progressive gait instability and speech regression. Pregnancy and delivery were uncomplicated. Early psychomotor development was normal. During the evaluation the child was lethargic with delayed responses to questions. Neurological examination revealed a deviation of the left eye and ptosis of both eyelids, ataxic gait, and impaired coordination. Brain magnetic resonance imaging demonstrated bilateral symmetric lesions of the caudate nucleus, putamen, hypothalamus, and brain stem with signs of cytotoxic edema, consistent with mitochondrial encephalopathy. Based on the clinical and neuroradiological findings, whole-exome sequencing was performed both on the boy and his parents. A pathogenic mitochondrial DNA variant, *MT-ND3 m.10197G>A*, was identified with a heteroplasmy level of 91% in the patient, whereas the mother had 59% heteroplasmy thus confirming Leigh syndrome. The patient continued the already started physical therapy and regular neurological follow-ups with the initiation of mitochondrial cocktail therapy consisting of coenzyme Q10, B vitamins, antioxidants, magnesium, and essential fatty acids to support mitochondrial function and energy production.

Conclusion: Leigh syndrome should be suspected in children with progressive neurological deterioration, including developmental regression accompanied with characteristic bilateral basal ganglia lesions and/or brainstem on neuroimaging and possible metabolic abnormalities. The symptoms can be detected later in childhood, although first signs appeared early. Appropriate genetic counselling of the whole family is necessary for possible treatments, prognosis and family planning.

Keywords: Leigh Disease; Mitochondrial Diseases; Mitochondrial Genes; Pediatrics

CR146

Polypharmacy After Myocardial Infarction: A Case of Unrecognized Adverse Drug Reactions and Drug–Drug Interactions

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Introduction: Guideline-directed medical therapy after myocardial infarction improves prognosis but often results in polypharmacy, increasing the risk of adverse drug reactions (ADRs) and drug–drug interactions (DDIs).

Case report: A 63-year-old woman with hypertension, diabetes, depression, and a history of smoking was referred for evaluation of suspected ADRs. In September 2025, she was hospitalized for ST-elevation myocardial infarction and underwent percutaneous coronary intervention. At discharge, therapy included acetylsalicylic acid, prasugrel, perindopril, metformin/empagliflozin, trimetazidine, esomeprazole, and atorvastatin/ezetimibe, while chronic therapy with amlodipine, carbamazepine, quetiapine, trazodone, and clonazepam was continued. Shortly thereafter, she developed headaches and persistent insomnia, leading to escalation of trazodone therapy. Following a second coronary angiography in December 2025, isosorbide mononitrate and ranolazine were introduced. Together with previously initiated medications, this resulted in a cascade of ADRs. The patient subsequently experienced severe headaches, ongoing insomnia, gastrointestinal bloating, and a genital fungal infection. Headache was attributed to isosorbide mononitrate, while other symptoms were initially unrecognized as drug-related. Carbamazepine, a potent cytochrome P450 3A4 inducer, reduced exposure to both ranolazine and atorvastatin, resulting in reduced ranolazine efficacy and insufficient LDL-cholesterol lowering, which prompted initiation of bempedoic acid requiring out-of-pocket payment. Ranolazine could have contributed to metformin-related gastrointestinal symptoms, while the genital fungal infection represented a known ADR of empagliflozin. Insomnia was suspected to be related to trimetazidine. Discontinuation of isosorbide mononitrate, ranolazine, trimetazidine, and bempedoic acid, along with switching lipid-lowering therapy to rosuvastatin/ezetimibe, resulted in symptom improvement without compromising guideline-directed post–myocardial infarction therapy.

Conclusion: This case highlights the importance of systematic medication review in patients with polypharmacy, particularly when introducing new therapies, to prevent prescribing cascades and preserve patients' quality of life and treatment adherence.

Keywords: Drug Interactions; Myocardial Infarction; Polypharmacy

CR147

From a Common Painkiller to Stevens–Johnson Syndrome

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Introduction: Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare, life-threatening mucocutaneous adverse drug reactions. Although most commonly triggered by antibiotics and anticonvulsants, nonsteroidal anti-inflammatory drugs may induce these conditions. There is increasing evidence supporting a critical role of genetic susceptibility particularly human leukocyte antigen (HLA) - associated immune mechanisms, culminating in a delayed, T-cell-mediated hypersensitivity reaction.

Case report: We report a 35-year-old male admitted with fever and a widespread mucocutaneous reaction following ibuprofen intake. Initial dermatological evaluation suggested SJS, with rapid progression to SJS/TEN overlap and subsequent transfer to the intensive care unit due to sepsis. Extensive epidermal denudation involved the face, trunk, extremities, and oral mucosa, accompanied by significant ocular symptoms. During hospitalization, repeated microbiological cultures of blood and skin were obtained, yielding *Acinetobacter baumannii* from blood and skin swabs, with additional isolation of *Klebsiella pneumoniae*, vancomycin-resistant *Enterococcus faecium*, and *Candida species*. The patient received multidisciplinary care, including broad-spectrum antibiotics, intravenous immunoglobulins, albumin supplementation, specialized wound care with silver-impregnated hydrofiber dressings. He remained hemodynamically stable throughout intensive care treatment. Almost whole re-epithelialization was achieved after three weeks, and the patient was transferred to the dermatology ward for continued targeted antimicrobial therapy.

Conclusion: SJS represents a T-cell-mediated type IV hypersensitivity reaction in which genetically determined immune pathways promote keratinocyte apoptosis. This case highlights the unpredictable nature of ibuprofen-induced SJS and underscores the potential contribution of pharmacogenetic susceptibility. Improved understanding of genetic risk factors may enable future prevention strategies, personalized drug prescribing, and reduced morbidity associated with severe cutaneous adverse drug reactions.

Keywords: Ibuprofen; HLA Antigens; Stevens-Johnson Syndrome; Toxic Epidermal Necrolysis

CR148

Adult-Onset Still's Disease – A Case Report

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Introduction: Adult-onset Still's disease (AOSD) is a rare systemic inflammatory disorder characterised by inflammatory polyarthritis, daily intermittent fever, and a transient salmon-pink maculopapular rash. The etiology of AOSD remains unknown. The prevailing hypothesis suggests that AOSD is a reactive syndrome in which various infectious agents may act as triggers in individuals with a genetic background.

Case report: We present a case of a 35-year-old male patient who was admitted with a diagnosis of fever of unknown origin, initially suspected to be due to a bacterial infection. Before admission, the patient experienced shortness of breath, a fever exceeding 39°C for three days, and a maculopapular rash. Despite receiving antibiotics, the fever persisted. Heart echocardiography revealed a significant pericardial effusion and right atrial collapse, along with a small pleural effusion. Laboratory tests showed neutrophilia, elevated acute phase reactants, hyperferritinaemia, and increased liver enzymes. An extensive workup excluded infections, lymphoproliferative disorders, and connective tissue diseases, ultimately leading to a diagnosis of AOSD. Initial treatment included high doses of glucocorticoids, nonsteroidal anti-inflammatory drugs (NSAIDs), and colchicine, followed by the addition of tocilizumab. One year later, the patient showed a positive response but experienced a relapse, which prompted a switch to anakinra. Four years later, there have been no signs of disease relapse.

Conclusion: This case demonstrates the importance of recognising the broad spectrum of diseases that can present with relatively general symptoms. In cases of vague fever, often accompanied by a characteristic maculopapular rash, adult-onset Still's disease should be considered, although it is extremely rare.

Keywords: Fever; Pericardial Effusion; Still's Disease, Adult-Onset

CR149

X-linked agammaglobulinemia in a male infant – from clinical suspicion to targeted genetic diagnosis

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Introduction: X-linked agammaglobulinemia, also known as Bruton's disease, is a rare primary immunodeficiency, caused by mutations in the Bruton tyrosine kinase (BTK) gene. These mutations lead to defective B-cell development and reduced or absent serum immunoglobulin levels. Bruton's disease usually presents in male infants aged 6-12 months, once the mother's antibodies no longer provide adequate protection, resulting in recurrent infections, such as pneumonia, bronchitis, otitis, conjunctivitis or sinusitis.

Case report: An eleven-month-old male patient was examined by pediatric infectious disease specialist due to an acute febrile illness lasting for four days without a leading symptom. Initial laboratory findings showed agranulocytosis and agammaglobulinemia with IgG <0.1 g/L, IgA 0.06 g/L and IgM 0.05 g/L. Past medical history revealed mild neutropenia at 7 months of age and otitis with perforated tympanic membrane at 8 months of age. After consultation with pediatric immunologist, X-linked agammaglobulinemia was suspected and immunoglobulin replacement therapy was introduced, starting with intravenous human immunoglobulin at 1g/kg and continued with 20mL subcutaneous human immunoglobulin every two weeks. Additional immunological work up showed absent B lymphocytes. Targeted genetic analysis revealed hemizygous variant c.1072C>T(p.Leu358Phe) in BTK gene, which is likely pathogenic. Genetic analysis of the mother is pending, to see if the mutation is inherited or occurred de novo. In a 3-month follow-up by the pediatric immunologist, immunoglobulin G levels show significant improvement at 11.89 g/L and the patient did not have any infections.

Conclusion: X-linked agammaglobulinemia is a serious condition, but if discovered early and treated correctly, with immunoglobulin replacement therapy and timely antibiotics in case of suspected infections, patients can have normal life quality and expectancy.

Keywords: Genome; Immunoglobulins; Primary Immunodeficiency Diseases

CR150

Genetic characteristics of hereditary angioedem

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Introduction: Hereditary angioedema with normal C1 inhibitor (HAE-nC1INH), previously referred to as HAE type III, is a rare disorder characterized by normal C1 inhibitor (C1-INH) levels and function. The disease course is marked by recurrent, self-limited episodes of subcutaneous and submucosal edema, primarily affecting the skin, gastrointestinal tract mucosa, and upper airway. HAE-nC1INH is associated with mutations in several genes, most commonly factor XII, but also plasminogen, angiopoietin-1, kininogen-1, myoferlin, and heparan sulfate glucosamine 3-O- sulfotransferase 6.

Case report: A 25-year-old woman with recurrent non-urticarial angioedema initially presented with facial swelling accompanied by influenza-like symptoms. Antihistamines and systemic corticosteroids were administered, however the symptoms recurred despite given therapy. Complement laboratory findings were within reference ranges, including complement C4 levels, C1-INH concentration, and functional activity. Tumor markers and other laboratory parameters were also normal. She experienced multiple attacks involving the lips, eyelids, tongue, and extremities, some of which required emergency care. Prophylaxis with danazol was initiated but discontinued due to poor tolerance and insufficient efficacy. Tranexamic acid was introduced but proved ineffective, with continued episodes. Patient was treated successfully with on-demand icatibant 30 mg subcutaneously leading to rapid regression. Due to high disease activity, long-term prophylaxis was accomplished with berotralstat after which episode frequency decreased and the patient achieved a stable clinical course with continued access to icatibant (Firazyr) for emergency events. Genetic testing for known mutations associated with HAE-nC1INH was negative.

Conclusion: Diagnosis of HAE-nC1INH is challenging to confirm despite normal laboratory findings of complement activity and function. It is necessary to carry out genetic testing with analysis of relevant genes.

Keywords: Angioedemas; Icatibant; Quality of Life

CR151

Precision Genomics as a Gateway to Safe Motherhood: Preimplantation Genetic Testing in Incontinentia Pigmenti

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Introduction: Incontinentia pigmenti (IP) is a high-penetrance, X-linked dominantly inherited disorder with a prevalence of 1/143.000. It affects the skin and its appendages, eyes, and central nervous system, occurring at a 20:1 female-to-male ratio. The aim of this case report is to emphasize the role of preimplantation genetic testing (PGT) as a reproductive option in severe hereditary disorders by selecting the embryos predicted to be free of a specific genetic condition.

Case report: We present the case of a 31-year-old woman who had two miscarriages in early weeks of gestation. As a newborn, she was diagnosed with IP by skin biopsy. Presently, she had hypopigmented lesions on the arms and legs, the pattern follows Blaschko lines. Given the nature of the disease, cardiological, ophthalmological, and neurological evaluations were performed, including brain MRI, with normal findings. Her partner was healthy. Considering the reproductive history, the patient was referred for diagnostic genetic testing. Long-range PCR and gel electrophoresis have shown the common deletion affecting exons 4 to 10 of the *IKBKG* gene in a heterozygous state. The risk of transmitting the variant to an offspring is 50%, with a note that male offspring usually miscarry. Since the *IKBKG* pathogenic variant was identified, preimplantation genetic testing was possible, and the patient was referred to PGT abroad.

Conclusion: IP is a rare genetic neuroectodermal disorder that occurs primarily in females. Most male conceptuses miscarry, resulting in pregnancies at a higher risk of spontaneous abortion. Detection of the pathogenic variant is mandatory for PGT, which ensures healthy offspring for affected families.

Keywords: Genetic Testing, Incontinentia Pigmenti, Mutation, Preimplantation Diagnosis

CR152

Immune Dysregulation in Common Variable ImmunoHeterozygositynodeficiency Associated with TACI and RUNX1

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Introduction: Common Variable Immunodeficiency (CVID) is a spectrum of immune dysregulation characterized by low levels of serum immunoglobulins. 20–30% of patients carry a genetic background associated with autoimmune manifestations, enteropathy, cytopenias, and more severe disease. Heterozygous variants in *TNFRSF13B*, encoding the TACI receptor, disrupt B-cell homeostasis and predispose to inflammatory and lymphoproliferative complications that may mimic hematologic or metabolic malignancies. This case is presented to clarify the diagnostic challenges when CVID-related inflammatory manifestations overlap with clinical features of systemic malignancy.

Case report: We present a 52-year-old male patient diagnosed with CVID in 2013. Despite receiving immunoglobulin replacement therapy (IRT), he developed a complex multisystemic phenotype characterized by bronchiectasis, interstitial lung disease, hepatosplenomegaly and cytopenias. He was treated with glucocorticoids and azathioprine with little effect, therefore azathioprine was switched to mycophenolate mofetil. Diagnostic reassessment was prompted by progressive pancytopenia and persistent elevation of alkaline phosphatase (ALP). Bone scintigraphy revealed markedly increased radiotracer uptake in the skull and femora, raising suspicion of Paget's disease, while pancytopenia required exclusion of myelodysplastic syndrome (MDS). Exome sequencing identified heterozygous *TNFRSF13B* (TACI) and *RUNX1* variants, with the latter linked to myeloid malignancy predisposition. ALP isoenzyme analysis confirmed a hepatic origin, suggestive of CVID-associated liver disease, while bone marrow biopsy ruled out MDS. Pancytopenia was attributed to immune-mediated dysregulation and iatrogenic myelosuppression from mycophenolate mofetil. Clinical stabilization followed mycophenolate mofetil withdrawal and initiation of parenteral iron therapy, with low-dose glucocorticoids and IRT.

Conclusion: This case demonstrates how complex CVID with autoimmune manifestations can convincingly mimic systemic malignancies. Identification of the *TNFRSF13B* variant contributed to resolving diagnostic uncertainty. These findings underscore the necessity of genotype-driven surveillance to accurately interpret complex CVID phenotypes.

Keywords: Azathioprine; Colitis; Cytopenia; Glucocorticoids

CR153

Undiagnosed Hereditary Thrombophilia as a Contributor to Septic Shock and Massive Pulmonary Embolism in a Surgical Patient

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Introduction: Hereditary thrombophilia is a genetic disorder that increases thromboembolic risk and often goes undiagnosed until significant clinical stress occurs. In surgical patients, infection, chronic inflammation, and obesity further increase hypercoagulability. This case highlights the role of thrombophilia and obesity as contributing risk factors for severe thromboembolic complications.

Case report: A 29-year-old woman (body mass index 34) with suppurative hidradenitis and pilonidal disease was admitted to the Intensive Care Unit after excision of a recurrent pilonidal sinus. She deteriorated rapidly postoperatively, developing septic shock and massive bilateral pulmonary embolism confirmed by computed tomography (CT) angiography. Due to hemodynamic instability, systemic thrombolysis with alteplase was administered. CT revealed a gluteal abscess at the surgical site, requiring emergency revision with incision and drainage. Despite broad-spectrum antibiotics, she progressed to multiorgan failure, necessitating prolonged mechanical ventilation, high-dose vasopressors, and continuous renal replacement therapy. During intensive care, she developed extensive necrotizing soft tissue infection and progressive peripheral ischemia, requiring multiple necrectomies and vacuum-assisted closure therapy. Given the severity of her condition, extended thrombophilia testing was performed and revealed heterozygous mutations in Factor V Leiden (G1691A), MTHFR C677T, and PAI-1 4G/5G. Histopathology showed extensive tissue necroses with thrombi in small and medium-sized vessels. Due to the poor prognosis, a multidisciplinary team decided to continue palliative care.

Conclusion: In surgical patients with risk factors or clinical signs of hypercoagulability, a comprehensive diagnostic workup, including analysis of coagulation factor mutations, is essential. Individuals with confirmed mutations and their family members should receive counselling and personalized, intensified thromboprophylaxis. This approach helps identify high-risk individuals, prevent serious thromboembolic events, and provide tailored treatment during periods of increased risk, such as acute infection or surgery.

Keywords: Mutation; Obesity; Sepsis; Thrombophilia

CR154

Mature cystic teratoma of the oral cavity in an infant: a case report

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Introduction: Teratomas are rare embryonic tumors that develop from germ cells. Oral cavity teratomas are extremely rare in infants compared with the more common ovarian and sacrococcygeal forms, while sublingual presentations are even rarer. These abnormalities may impair airway patency, tongue movement, and feeding, requiring immediate medical attention. The aim of this case report is to present a rare mature cystic sublingual teratoma in an infant, highlighting its clinical presentation, diagnostic challenges, and surgical management.

Case report: A male infant born after an uncomplicated pregnancy and vaginal delivery at 38 weeks presented at nine months of age with a sublingual mass discovered during hospitalisation for upper respiratory infection and bronchiolitis. Drooling and food retention beneath the tongue were among progressively worsening feeding difficulties. Earlier, bilateral sublingual protrusions were observed following frenulotomy at two months of age; however, the initial ultrasound examination was considered insignificant. A midline lobulated sublingual cystic lesion measuring 25 × 25 × 27 mm, with minor septations and small hemorrhagic patches, displacing the tongue superiorly, was identified by contrast magnetic resonance imaging (MRI). Initially, a ranula was suspected. Complete surgical excision via an intraoral approach was performed with careful preservation of surrounding tissues. Histopathological examination revealed a cystic lesion with skeletal muscle at the margins, as well as seromucinous glands, ciliated respiratory epithelium, gastrointestinal-type epithelium, folliculosebaceous structures, and stratified squamous epithelium, consistent with a mature cystic teratoma.

Conclusion: This case illustrates the importance of considering mature cystic teratoma in the differential diagnosis of sublingual masses in an infant with feeding difficulties. Early contrast MRI and timely complete surgical excision enabled definitive diagnosis, restoration of normal feeding, and an excellent postoperative outcome.

Keywords: Infant; Magnetic Resonance Imaging; Mouth Neoplasms; Oral Surgical Procedures; Teratoma

CR155

Treatment of Type 1 Diabetes Mellitus Using an Advanced Hybrid Closed-Loop System

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Introduction: Type 1 diabetes mellitus (T1DM) requires precise glucose regulation. The Medtronic MiniMed 780G insulin pump with the hybrid closed-loop (HCL) system enables automatic insulin delivery adjustments based on continuous glucose monitoring (CGM). This HCL system, featuring autocorrection boluses, safely improves glycemic control and helps people with T1DM consistently achieve international clinical targets.

Case report: An 18-year-old male patient (body mass index 18.94 kg/m²) with T1DM since childhood is presented. He was treated with continuous subcutaneous insulin infusion using an external pump. Despite the glycated haemoglobin (HbA1c) value of 7.1%, analysis of CGM data from a flash glucose monitoring system (FGM Libre) revealed poor regulation quality. The percentage of time in range (TIR) was only 56% (recommended >70%), and a high time above range (TAR) of 44% indicated frequent hyperglycemia. Therefore, the patient was transitioned to the Medtronic MiniMed 780G insulin pump, and after a few days, the SmartGuard system was activated, which functions as HCL system. By the next follow-up, excellent glycemic control was achieved. The glucose management indicator (GMI) was 6.5%, meeting the American Diabetes Association goals. The glucose coefficient of variation (CV) was 32.5% (below the 36% threshold), and the TIR reached 83%. The risk of hyperglycemia was significantly reduced. Continuation of the same therapy with regular six-month check-ups was recommended.

Conclusion: This case illustrates how the HCL system addresses the issue of suboptimal control, despite an apparently acceptable HbA1c. It emphasizes the importance of CGM parameters, such as TIR, TBR, and CV, for a comprehensive assessment of glycemic regulation quality.

Keywords: Continuous Glucose Monitoring; Diabetes Mellitus, Type 1; Hyperglycemia

CR156

Enzyme replacement therapy in adenosine deaminase-deficient severe combined immunodeficiency

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Introduction: Adenosine deaminase-deficient severe combined immunodeficiency (ADA-SCID) is a rare inherited disorder of purine metabolism resulting in profound impairment of both cellular and humoral immunity. If left untreated, the disease is typically fatal within the first few years of life due to severe, recurrent infections. Available therapeutic strategies include hematopoietic stem cell transplantation (HSCT), recently developed gene therapy and enzyme replacement therapy (ERT).

Case report: We report the case of a 28-year-old male patient with ADA-SCID who has been receiving continuous enzyme replacement therapy since early childhood. The patient presented in early infancy with failure to thrive and recurrent, severe respiratory infections resistant to antibiotic and immunoglobulin therapy. The family history was positive for immunodeficiency, with two relatives who died in early childhood. The diagnosis was established by demonstrating absent ADA activity in erythrocytes. Since the patient had no HLA-identical donor, HSCT was not indicated. He was consequently started on ERT and antimicrobial prophylaxis. The current standard of care is elapegademase-lvrl, a recombinant pegylated formulation of adenosine deaminase with an extended half-life and no animal-derived components. Despite ongoing therapy, the patient still has lymphopenia and occasional respiratory and herpesvirus infections, as well as human papillomavirus-associated cutaneous warts.

Conclusion: Survival into adulthood illustrates the effectiveness of modern recombinant therapy in correcting metabolic toxicity and restoring immune function in patients with ADA-SCID. Early diagnosis, prompt initiation of treatment, and lifelong multidisciplinary care are essential to improve prognosis and quality of life in this otherwise fatal disease.

Keywords: Adenosine deaminase deficiency; Enzyme Replacement Therapy; Inborn Error of Immunity; Severe Combined Immunodeficiency

CR157

Balancing the Benefits and Risks: A Case of Pembrolizumab-Associated Immune Hepatitis in Breast Cancer

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Introduction: With the expanding use of immunotherapy, an increasing number of patients are exposed to the risk of immune-mediated liver injury. Immune-mediated hepatitis is a common adverse effect of checkpoint inhibitors, including pembrolizumab, a Programmed Cell Death Protein 1 inhibitor. It may range from asymptomatic liver enzyme elevations to fulminant liver failure.

Case report: A 41-year-old woman was admitted to the Oncology Clinic after a core needle biopsy confirmed grade 2 triple-negative invasive carcinoma of the right breast. She was scheduled to receive 12 cycles of paclitaxel with carboplatin and pembrolizumab, followed by 4 cycles of adriamycin and cyclophosphamide combined with pembrolizumab. After the third cycle, aspartate aminotransferase rose to 111 U/L, alanine aminotransferase to 200 U/L, and gamma-glutamyl transferase to 256 U/L. Therapy was delayed, and intravenous methylprednisolone 80 mg was administered for 4 days. A computed tomography scan of the thorax, abdomen, and pelvis revealed two atypical hepatic hemangiomas. On the third day, prednisone 60 mg was initiated. A contrast-enhanced liver ultrasound confirmed four focal lesions resembling hemangiomas. As liver enzymes remained elevated, chemotherapy continued, but pembrolizumab was discontinued. The patient completed all planned chemotherapy cycles and was referred for breast segmentectomy.

Conclusion: Although pembrolizumab is an effective treatment for various cancers, it can cause serious adverse effects, including immune-mediated hepatitis. Continuous laboratory monitoring before and during therapy, along with thorough diagnostic evaluation, is essential for early detection and timely management of organ damage.

Keywords: Breast Neoplasms; Hepatitis; Immune Checkpoint Inhibitors; Immunotherapy

CR158

Atypical Presentation of Multiple Sclerosis

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Introduction: Multiple sclerosis is a chronic immune-mediated inflammatory disorder of the central nervous system (CNS). Diagnosis of relapsing–remitting multiple sclerosis (RRMS) requires fulfillment of the revised McDonald criteria. Atypical presentations may mimic neoplastic or other inflammatory CNS conditions, complicating diagnosis and therapeutic decisions.

Case Report: We present the case of a 43-year-old male patient diagnosed with RRMS and initially treated with dimethyl fumarate. Despite therapy, he developed dysphagia, dysarthria, ataxia, and left-sided internuclear ophthalmoplegia and was consequently started on high-dose intravenous corticosteroid pulse therapy, resulting in partial clinical improvement. Additional diagnostic workup for myasthenia gravis, Behçet's disease and sarcoidosis was performed and yielded negative results. The existing immunomodulatory therapy was continued, and the patient remained clinically stable. Follow-up magnetic resonance imaging revealed a 1-cm enlargement of the pontine lesion. Biopsy was precluded due to the lesion's brainstem location. Reassessment of prior diagnostics confirmed fulfillment of the McDonald criteria, with positive oligoclonal bands and an elevated kappa index. Extensive evaluation excluded neuroborreliosis, neurotropic viral infections, lymphoma, tuberculosis, as well as antibodies associated with Neuromyelitis Optica Spectrum Disorder and Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease. Ongoing clinical and radiological progression warranted escalation to B-cell-depleting therapy.

Conclusion: Although the patient fulfills the diagnostic criteria for RRMS, the clinical course demonstrates atypical features. This pattern of lesion evolution is unusual for RRMS and raises concern for alternative or overlapping pathological processes, warranting further diagnostic evaluation and close monitoring.

Keywords: Brain Stem; Demyelinating Diseases; Multiple Sclerosis

CR159

When Energy Metabolism Fails the Retina: A New IDH3A Variant Linking Retinal Dystrophy and Mitochondrial Encephalopathy

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Introduction: The macula represents the most metabolically demanding region of the retina, requiring uninterrupted adenosine triphosphate production to sustain photoreceptor function. The *IDH3A* gene encodes the catalytic subunit of mitochondrial isocitrate dehydrogenase (IDH), a key enzyme involved in the tricarboxylic acid cycle and cellular energy metabolism. The pathogenic variant *IDH3A* c.364G>A, p.(Ala122Thr) has been previously associated with a milder form of retinitis pigmentosa (RP), whereas the clinical relevance of the *IDH3A* c.293C>T p.(Pro98Leu) variant remains unclear.

Case report: We report the case of a six-year-old girl referred for comprehensive ophthalmological and genetic evaluation. The patient presented with congenital nystagmus at four months of age, followed by nyctalopia. At the age of three, she developed acute cerebellitis of unknown etiology, subsequently manifesting delayed psychomotor development suggestive of infantile mitochondrial encephalopathy. Ophthalmological assessment revealed severe visual impairment, photophobia, and nystagmus. Ultra-widefield imaging depicted bilateral, sharply demarcated macular atrophic lesions, consistent with macular pseudocoloboma. Optical coherence tomography confirmed extensive bilateral atrophy of the neurosensory retina, retinal pigment epithelium, and choriocapillaris. The phenotype was consistent with RP. Genetic analysis identified compound heterozygosity for *IDH3A* c.364G>A, p.(Ala122Thr) and *IDH3A* c.293C>T p.(Pro98Leu). At present, treatment remains exclusively supportive, as targeted gene augmentation therapy is not yet available for the *IDH3A* gene.

Conclusion: While the *IDH3A* c.364G>A, p.(Ala122Thr) variant is associated with predominantly retinal disease and residual enzymatic activity, this case suggests that the presence of the *IDH3A* c.293C>T p.(Pro98Leu) variant might correlate with a more severe multisystem mitochondrial disease, potentially including neurological involvement. Further studies are needed to determine the clinical relevance and pathogenic potential of this variant.

Keywords: Mitochondrial Diseases; Ophthalmology; Retinal Dystrophies; Retinitis Pigmentosa

CR160

The Clinical Path to Clarity: Step-by-Step Diagnosis of Rare GQ1b Encephalitis

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Introduction: Numerous pathologies can mimic symptoms or Magnetic resonance imaging (MRI) findings of multiple sclerosis (MS), including rare neuro-immunological disorders such as Bickerstaff Brainstem Encephalitis (BBE). BBE is traditionally characterized by progressive ophthalmoplegia, ataxia, altered consciousness, and positive anti-GQ1b antibodies. However, these antibodies are not exclusive to BBE, as it is part of a broader spectrum of GQ1b-antibody syndromes.

Case Report: We report a 37-year-old male who presented to the Emergency Department with paraesthesia of the left hemibody and cheeks. His condition progressed to include tinnitus, autophony, and vertigo. Given the presentation, MS was initially suspected. Brain MRI revealed a hyperintense lesion in the right pons, while MR spectroscopy showed decreased N-acetyl-aspartate and increased choline, suggesting a demyelinating process. The patient received high-dose intravenous methylprednisolone (1g daily for five days), resulting in clinical improvement. However, the absence of intrathecal oligoclonal bands and normal kappa index shifted the clinical focus away from MS. Extensive differential diagnostic work-up, including neuroborreliosis, Neuromyelitis Optica Spectrum Disorder, MOG-antibody-associated disease, and paraneoplastic syndromes, was negative. Electromyoneurography was unremarkable. Immunological testing returned positive for anti-GQ1b antibodies suggesting BBE. However, as BBE criteria were incomplete, the antibody profile established a final GQ1b encephalitis diagnosis. Subsequent intravenous immunoglobulin therapy led to clinical improvement and initial lesion regression on MRI.

Conclusion: This case illustrates the structured clinical pathway required to reach a rare diagnosis. It emphasizes the importance of incorporating immunological findings into the broader clinical picture to ensure diagnostic accuracy in daily practice.

Keywords: Autoantibodies; Demyelinating Diseases; Encephalitis; Gangliosides; Multiple Sclerosis

CR161

Tailored Genetic Analysis for Diagnosis of a Very Rare Immunodeficiency

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Introduction: Immunodeficiency, centromeric instability, facial dysmorphism (ICF) syndrome type 1 is a rare autosomal recessive disorder caused by mutations in the DNA methyltransferase 3 beta gene. It is characterised by severe hypogammaglobulinemia, instability of heterochromatin in the centromeric regions of chromosomes 1 and 16, and specific craniofacial and skeletal anomalies.

Case report: A male patient presented in infancy with recurrent otitis media. Diagnostic workup revealed severe hypogammaglobulinemia, for which intravenous immunoglobulin replacement therapy was initiated, reducing the frequency of infections. By the age of 7, the child developed conductive hearing loss due to recurrent infections, apraxia, and speech difficulties. The patient has certain facial abnormalities such as low-set and deformed ears, bilateral prominent epicanthus, a small chin and partial toe syndactyly. Due to hypogammaglobulinemia and characteristic facial dysmorphisms, ICF syndrome was suspected, and targeted cytogenetic analysis was performed. Karyotyping showed that 50% of cells had a normal male karyotype 46, XY, while the remaining 50% of analyzed metaphases exhibited instability of the heterochromatic centromeric regions of chromosomes 1qh and 16qh, which is a characteristic of ICF syndrome type 1. The abnormalities manifested as heterochromatin decondensation, most frequently at 1qh, resulting in breaks in homologous and nonhomologous chromosomes, monosomies and trisomies of the entire chromosome 1 or more often, partial q arm abnormalities, isochromosomes 1q and 1p, and multiple copies of chromosome 1 forming rosette structures. Despite regular immunoglobulin replacement treatment, the patient has chronic conjunctivitis and has developed bronchiectasis, which is a common complication in individuals with hypogammaglobulinemia.

Conclusion: This case highlights the importance of targeted genetic and cytogenetic diagnostics to ensure better and individually tailored care.

Keywords: Agammaglobulinemia; Craniofacial Abnormalities; Genomic Instability; Primary Immunodeficiency Diseases

CR162

Immunotherapy Outcomes in Immune Complex–Mediated Diffuse Sclerosing Glomerulonephritis

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Introduction: Diffuse sclerosing glomerulonephritis represents a form of immune complex–mediated glomerular disease, characterized by sclerotic involvement of more than 50% of glomeruli and chronic renal parenchymal damage. Persistent immune-mediated injury may result in irreversible loss of renal function, clinically manifesting as chronic kidney disease. We present a case of immune complex–mediated glomerulonephritis, focusing on the outcomes of immunosuppressive therapy.

Case report: A 27-year-old woman was admitted to the nephrology department for evaluation of renal function due to newly diagnosed arterial hypertension, with blood pressure values reaching 180/100 mmHg. Laboratory findings revealed a serum creatinine level of 178 $\mu\text{mol/L}$ and an estimated glomerular filtration rate of 33 mL/min/1.73 m² (chronic kidney disease, stage 3b), with microalbuminuria, erythrocyturia, and severe proteinuria. Arterial hypertension was well controlled with perindopril and amlodipine; however, kidney biopsy was indicated due to impaired renal function. Preliminary histopathological findings suggested tubulointerstitial nephritis, and corticosteroid therapy was initiated. Subsequent electron microscopy demonstrated immune complex–mediated diffuse sclerosing glomerulonephritis, with fibrotic changes in 68% of glomeruli and segmental sclerosis in 18%, accompanied by glomerular hypertrophy and mesangial hypercellularity. After immunology consultation, mycophenolate mofetil was introduced as a glucocorticoid-sparing therapy. Immunosuppressive treatment resulted in a reduction of albuminuria and proteinuria; however, therapy was discontinued due to reactivation of varicella-zoster virus, leukopenia, and anemia, leading to a subsequent increase in proteinuria. Rituximab was initiated as second-line immunosuppressive therapy.

Conclusion: Although mycophenolate mofetil is frequently used for immune complex–mediated glomerular disease, careful assessment of the benefit–risk ratio is essential, particularly with adverse effects. This case report highlights the need for an individualized approach to immunosuppressive therapy, with readiness for treatment modification and use of alternative agents when indicated.

Keywords: Glomerulonephritis; Immunotherapy; Renal Insufficiency, Chronic

CR163

Hypomelanosis as a positive predictor of favorable outcomes in stage III-IV melanoma patients treated with immunotherapy – a case report

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Introduction: Melanoma is a skin cancer with a rising global incidence. Before the arrival of immune checkpoint inhibitors, such as monoclonal antibodies directed against the programmed cell death protein-1 (anti-PD-1 antibodies), the prognosis for advanced disease was poor. The complex interplay between melanoma biology and antitumor immunity prevents durable disease control, highlighting the need for genetic, serologic, and clinical biomarkers to predict therapeutic response and outcomes.

Case report: We present a 71-year-old woman with a large tumorous mass in the left inguinal region. Core biopsy with histopathologic and molecular analyses confirmed metastatic melanoma without B-Raf proto-oncogene, serine/threonine kinase mutation, while dermatologic and gynecologic assessments failed to identify a primary lesion. According to the Tumor-Node-Metastasis classification, melanoma was staged as T0N3bM1a (stage IVA). Baseline positron emission tomography–computed tomography (PET-CT) with fluorodeoxyglucose (FDG) demonstrated disease confined to the left inguinal and iliac lymph nodes. Treatment consisted of four cycles of combined immunotherapy (nivolumab and ipilimumab), followed by six cycles of nivolumab monotherapy. The patient developed immune-related cutaneous manifestations, including eyelash depigmentation and vitiligo-like hypopigmentation on the face and body. Follow-up PET-CT revealed a near-complete loss of FDG uptake with a marked tumor reduction. Per the Immune response evaluation criteria in solid tumors, three additional cycles of ipilimumab are planned, followed by reassessment.

Conclusion: Based on our clinical experience, vitiligo-like hypo- and depigmentation during immunotherapy correlates with strong antitumor activity and favorable outcomes in advanced melanoma. Recent meta-analyses support this, linking treatment-induced hypomelanosis to improved response and survival with immune checkpoint inhibitors. These findings highlight hypomelanosis as a potential prognostic biomarker to guide therapy and optimize management.

Keywords: Hypomelanosis; Immune Checkpoint Inhibitors; Immunotherapy; Melanoma

CR164

Posterior Reversible Encephalopathy Syndrome Following Bilateral Nephrectomy: A Life-Threatening Presentation

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Introduction: Posterior reversible encephalopathy syndrome (PRES) is a life-threatening, acute neurological disorder presenting with encephalopathy, visual impairment, and characteristic neuroradiological findings. With adequate treatment, it quickly resolves. Hypertension, kidney disease, and immunosuppressants are common etiological factors, while neuroimaging is critical for timely recognition and complication avoidance.

Case report: We present a 59-year-old patient with adequately controlled hypertension, on maintenance hemodialysis (HD) following a bilateral nephrectomy and subsequent cystectomy due to carcinoma. He presented to the emergency room after a post-HD seizure. Laboratory tests suggested significant metabolic abnormalities. Neurological assessment suspected uremic encephalopathy and indicated emergency HD under sedation due to extreme restlessness. Although Cerebrospinal fluid analysis was unremarkable, magnetic resonance imaging of the brain revealed bilateral posterior corticosubcortical T2-weighted Fluid-Attenuated Inversion Recovery hyperintensities consistent with PRES. Thereupon, the patient was transferred to the Department of Intensive Neurological Treatment with prominent left-sided neurological deficits. Additionally, transcranial color Doppler revealed reduced flow in the right internal carotid, and multiple cerebral arteries. Significant hypertension (200/90 mmHg) developed, alongside psychiatric disturbances partially provoked by uremic alteration. These included delusional beliefs of being on a train, followed by passersby intending to harm him. Antipsychotics and a benzodiazepine were introduced in addition to regular HD sessions. During his stay, psychiatric symptoms and hypertension gradually normalized. Follow-up neuroimaging showed complete regression of PRES-related changes, allowing discharge the following day, with only minor neurological deficits.

Conclusion: This case elucidates PRES not merely as a neuroradiological entity, but a heterogeneous syndrome which may include severe psychiatric symptomatology. Early diagnosis, alongside urgent correction of blood pressure and metabolic factors resulted in complete radiological recovery and a favorable clinical outcome consistent with the reversible nature of the syndrome.

Keywords: Delusions; Nephrectomy; Posterior Leukoencephalopathy Syndrome; Renal Dialysis

CR165

Saving the Kidney, Avoiding the Surgery: Percutaneous Cryoablation for a Large Renal Tumor - A Case Report

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Introduction: Cryoablation is a minimally invasive treatment method where the utilization of low temperatures induces coagulation necrosis and subsequent systemic immune response. This procedure is performed under local anesthesia, making it suitable for patients at high risk for general anesthesia. The aim of this case report is to describe the effectiveness of cryoablation as a treatment method comparable to surgical treatment.

Case report: The patient is a 75-year-old male with a right kidney tumor measuring 71 x 65 x 58 mm. Due to the patient's comorbidities, cryoablation has emerged as a safe alternative to surgical treatment. To reduce peri- and postprocedural bleeding risk in the setting of a large tumor, tumor-feeding renal artery branches were embolized with a liquid embolic agent and microspheres before cryoablation. Under computed tomography (CT) guidance, percutaneous cryoablation of the tumor was performed using six cryoprobes, with alternating cycles of freezing and thawing. Temperatures range from – 40°C to – 20°C in the freezing zone. Cryoprobes freeze tumor tissue, triggering necrosis and apoptosis and releasing tumor antigens that activate the immune system against residual tumor cells. Follow-up CT, 24 hours post-procedure, showed no local complications. At one-month follow-up, the CT scan showed a size reduction to 66 x 59 x 60 mm. The patient is in good general condition and pain-free.

Conclusion: Due to the extremely low temperatures, cryoablation induces tumor cell necrosis and stimulates an anti-tumor immune response. The combination of cryoablation and embolization is an excellent treatment alternative to surgery in high-risk patients with mostly equal treatment results.

Keywords: Cryoablation; Embolization, Therapeutic; Kidney Neoplasms

CR166

ACTA1-Related Congenital Myopathy Presenting in the Neonatal Period: A Case Report**Tiyya Selimović¹; Ana Vučaj¹; Morena Sirotić¹; Matej Raguž¹; Dorotea Drašković^{1,2}***1 - Faculty of Medicine Rijeka, University of Rijeka, Rijeka, Croatia**2 - Department of Paediatrics, Clinical Hospital Center Rijeka, Rijeka, Croatia*

Introduction: ACTA1-related congenital myopathies are rare neuromuscular disorders with a wide phenotypic spectrum, frequently presenting at birth with severe hypotonia and respiratory insufficiency. Early diagnosis is challenging due to overlap with other causes of neonatal hypotonia but is essential for guiding management and genetic counseling.

Case report: A male term neonate presented with profound generalized hypotonia, absent spontaneous movements, and severe respiratory failure requiring ventilatory support. Neurological examination revealed axial and limb hypotonia, preserved consciousness, and reduced reflex activity. Despite initial non-invasive ventilation, the patient developed progressive chronic respiratory insufficiency with hypercapnic respiratory failure (pH 7.20, pCO₂ >12 kPa), necessitating tracheostomy and long-term invasive mechanical ventilation. Feeding difficulties resulted in inability to tolerate oral intake, and a percutaneous endoscopic gastrostomy (PEG) was placed to ensure nutrition. An extensive diagnostic work-up, including metabolic, infectious, cardiologic, and neuroimaging evaluations, excluded secondary causes of hypotonia. Brain imaging demonstrated asymmetrical ventriculomegaly without raised intracranial pressure. Whole-exome sequencing identified a heterozygous pathogenic missense variant in the ACTA1 gene (c.282C>A; p.Asn94Lys), confirming ACTA1-related congenital myopathy. During hospitalization, the patient experienced infectious complications, including episodes of sepsis, successfully treated with antimicrobial therapy guided by susceptibility testing, including cefepime, meropenem, and vancomycin. At discharge, the infant was hemodynamically stable, tracheostomized, PEG-fed, and dependent on home mechanical ventilation. A multidisciplinary follow-up plan was established, and genetic counseling provided to the family.

Conclusion: This case underscores the importance of early genetic testing in neonates with severe hypotonia and respiratory failure. Early diagnosis of ACTA1-related congenital myopathy helps guide prognosis, optimize clinical management, support timely multidisciplinary care and provide essential genetic counseling for the family.

Keywords: Myopathies, Congenital; Rare Diseases; Whole Exome Sequencing

CR167

IgA Vasculitis Mimicking Inflammatory Bowel Disease:: A Case Report

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Introduction: Inflammatory bowel disease (IBD) is a collective term for a group of conditions that cause swelling and inflammation in the gastrointestinal tract. IBD primarily includes Crohn's disease and ulcerative colitis. Henoch-Schönlein purpura, also known as Immunoglobulin A vasculitis (IgAV), is a type of blood vessel inflammation caused by the accumulation of IgA antibodies in small vessels. In this case we present the unusual way in which IgAV presented itself as a condition like IBD.

Case report: A 35-year-old man presented to the emergency department with right lower abdominal pain lasting four days, accompanied by nausea, vomiting and diarrhea, and with left axillary edema. An urgent computed tomography abdominal scan was performed, which showed localized thickening and hyperemia of the small intestinal wall with enlarged abdominal lymph nodes, consistent with inflammatory process. Laboratory findings showed increased C-reactive protein and elevated leukocytes. During hospitalization, an esophagogastroduodenoscopy was performed, which revealed several ulcerations of the gastric mucosa. Patient declined recommended colonoscopy. Histopathological examination (HPE) of the mucosa indicated probable IBD, but also possible autoimmune enteropathy. Magnetic resonance enterography was also performed, which described mesenteric panniculitis. During hospitalization, the patient developed a purpuric rash and proteinuria. For this reason, skin and kidney biopsies were performed. HPE of the skin was consistent with leukocytoclastic vasculitis, and HPE of the kidneys was consistent with IgA nephropathy. Given the clinical picture and comprehensive diagnostic work-up, a diagnosis of IgAV was made.

Conclusion: This case demonstrates that IgAV may initially present with gastrointestinal manifestations closely resembling IBD. Awareness of this diagnostic overlap is important to avoid misdiagnosis and ensure appropriate management.

Keywords: Abdomen, Acute; Autoimmune Diseases; IgA Vasculitis; Inflammatory Bowel Diseases

CR168

Ocular Adverse Effects of Adalimumab Therapy: A Case Report

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Introduction: Tumor necrosis factor- α (TNF- α) inhibitors, such as adalimumab, are widely used in the treatment of autoimmune diseases. Although these agents are effective in controlling systemic inflammation, paradoxical immune-mediated adverse effects may occur, including ocular complications. Early recognition of such manifestations is essential to prevent visual impairment and enable individualized therapeutic decision-making.

Case report: A 34-year-old patient with Crohn's disease receiving adalimumab therapy developed new-onset ocular symptoms after approximately 10 months of continuous treatment. Ophthalmological examination revealed acute unilateral anterior uveitis, accompanied by retinal microvascular changes, including areas of capillary flow alteration and reduced perfusion on optical coherence tomography angiography. No macular edema or signs of retinal ischemia were detected. Infectious causes were excluded through comprehensive laboratory testing. A multidisciplinary evaluation involving rheumatology and ophthalmology suggested a paradoxical immune-mediated reaction to TNF- α inhibition rather than an extra-articular manifestation of the underlying disease. Based on the patient's clinical presentation and immune status, management was tailored individually, including temporary discontinuation of adalimumab, initiation of topical corticosteroid therapy (prednisolone acetate 1% eye drops), and close ophthalmological follow-up, resulting in gradual resolution of ocular inflammation. Adalimumab was not reintroduced, and no recurrence of ocular symptoms was observed.

Conclusion: This case highlights anterior uveitis and retinal microvascular alterations as potential ocular adverse effects of adalimumab therapy. Ophthalmological manifestations may serve as early indicators of paradoxical immune activation during biological treatment and emphasize the need for careful monitoring and multidisciplinary management of patients receiving TNF- α inhibitors. Awareness of therapy-related ocular complications is crucial for optimizing patient outcomes through timely recognition and individualized immunomodulatory decision-making.

Keywords: Adalimumab; Case Reports; Drug-Related Side Effects and Adverse Reactions; Tumor Necrosis Factor Inhibitors; Uveitis

CR169

Hypersensitivity in Emergency Medicine: The Importance of a Personalized Diagnostic Approach

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Introduction: Drug hypersensitivity reactions represent a significant clinical challenge, particularly in emergency settings where multiple medications are often administered simultaneously. Identifying the causative drug is crucial for patient safety and further therapeutic decisions. The aim of this paper is to highlight the importance of an individualized and structured pharmacological approach in identifying the causative drug in suspected acute drug hypersensitivity during emergency management of traumatic injury.

Case report: A 33-year-old man with no previously known drug allergies was admitted to the Emergency Department with multiple open wounds of the left hand caused by a firework explosion. He was hemodynamically stable. Initial management included paracetamol, ibuprofen, tetanus prophylaxis, and empirical intravenous antibiotics (gentamicin, metronidazole, and cefazolin). The wounds were surgically treated under local anesthesia with lidocaine. Approximately 15 minutes after antibiotic administration, the patient developed facial urticaria without respiratory or hemodynamic compromise. Symptoms resolved after chlorpyramine and methylprednisolone. He was discharged with oral amoxicillin/clavulanic acid and clindamycin, which he tolerated well. Due to suspected hypersensitivity, the patient was referred to a clinical pharmacology clinic. Skin tests for beta-lactams, tetanus vaccine, local anesthetics, metronidazole, and gentamicin were negative. During a single-blind intravenous provocation with cefazolin, generalized urticaria with predominant facial involvement occurred and resolved after oral antihistamine administration. Further testing excluded hypersensitivity to other drugs and cephalosporins. Avoidance of cefazolin was recommended, while other medications were considered safe.

Conclusion This case highlights the importance of targeted diagnostics in acute care and polypharmacy. Controlled intravenous provocation testing enables reliable identification of the causative drug and safe planning of future therapy; hypersensitivity was confirmed to be selective for cefazolin, while other cephalosporins and penicillins were tolerated.

Keywords: Cefazolin; Drug Hypersensitivity; Polypharmacy; Skin Tests; Urticaria

CR170

TRUST THE SEQUENCE, NOT THE TREE: A case of autosomal dominant polycystic kidney disease with a negative family history**Jana Skender¹; Neva Rašperić¹; Ivan Stanušić¹; Nikola Zagorec²; Ivica Horvatić²***1 - School of Medicine, University of Zagreb, Zagreb, Croatia**2 - Department of Nephrology and Dialysis, University Hospital "Dubrava", Zagreb, Croatia*

Introduction: Autosomal dominant polycystic kidney disease (ADPKD) is the most common genetic renal disease, mostly due to pathogenic variants in *PKD1* and *PKD2*.

Case report: 29-year-old man presented with pain in the right lower quadrant, accompanied by increased urinary frequency without dysuria. Laboratory workup showed normal kidney function, serum creatinine 84 µmol/L, estimated glomerular filtration rate 107 ml/min/1.73m², bland urine sediment and no proteinuria. The patient had a one-year history of hypertension, which had been well controlled with a combination of perindopril/amlodipine. Four years ago, a routine abdominal ultrasound revealed multiple bilateral kidney cysts, which is why the patient was referred to a nephrologist. Magnetic resonance imaging revealed numerous bilateral renal cysts, height-adjusted total kidney volume was 344 ml/m, and several hepatic cysts. Family history was negative for ADPKD or kidney failure as well as screening for kidney cysts in parents and siblings. Whole exome sequencing revealed pathogenic variant in *PKD1* (c.4041_4042del, p.His1347Glnfs*83) thus confirming ADPKD. Prior to this presentation, his medical history was negative for episodes of gross hematuria, urinary tract infections, urolithiasis or back pain. Furthermore, time of flight magnetic resonance angiography identified a small infundibulum or aneurysm of the anterior communicating artery, requiring further follow-up. Considering the Mayo Imaging Classification (Class 1C) and additional risk factors of rapid disease progression (Predicting Renal Outcome in PKD score of 7), treatment with tolvaptan was started.

Conclusion: In this case, ADPKD was most likely due to de novo variant in *PKD1*, although mosaicism in parents can not be excluded. Genetic testing confirms a diagnosis when family history is negative and may guide risk stratification and treatment.

Keywords: Hypertension; Magnetic Resonance Imaging; Polycystic Kidney, Autosomal Dominant

CR171

Chronic recurrent multifocal osteomyelitis: when a fracture is not a fracture, when a tumor is not a tumor**Ivan Sladoja¹; Ivana Groznica¹; Barbara Veselovac¹; Ana Mehičić²; Vjekoslav Wertheimer^{1,2}***1 - Faculty of Medicine, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Orthopedics and Traumatology, University Hospital Center, Osijek, Croatia.*

Introduction: Chronic recurrent multifocal osteomyelitis (CRMO) is a rare, sterile, inflammatory bone disorder predominantly affecting children. It is thought to result from imbalanced cytokine production and enhanced osteoclast differentiation. This case illustrates the diagnostic challenges of CRMO, which frequently mimics traumatic, infectious, or neoplastic bone lesions.

Case report: A 10-year-old boy presented with pain and edema of the dorsal left foot and impaired gait following minor trauma. Initial radiographs demonstrated periosteal reaction and cortical disruption of the distal metaphysis of the fourth metatarsal, and an acute fracture was suspected. After immobilization, follow-up imaging showed cortical thickening interpreted as callus formation, correlating with reported clinical improvement. However, two months later, symptoms recurred. Repeat radiographs revealed atypical bone remodeling with significant expansion, irregular cortical thickening, and focal osteolytic lesions, raising suspicion of malignancy and prompting further evaluation with magnetic resonance imaging. The metatarsal lesion was described as highly suspicious for sarcoma or, potentially, osteomyelitis. Technetium-99m scintigraphy demonstrated increased uptake in the left fourth metatarsal, calcaneus, tibia, and right sacroiliac joint. Laboratory inflammatory markers, including erythrocyte sedimentation rate, C-reactive protein, and leukocyte count, were within normal limits. To exclude malignancy, a surgical biopsy of the metatarsal was performed, confirming a sterile inflammatory process. Histopathological analysis demonstrated predominantly chronic osteomyelitis. The patient reported immediate postoperative pain relief, presumably due to surgical decompression of intraosseous pressure. Following a multidisciplinary review, a diagnosis of CRMO was established.

Conclusion: CRMO remains an uncommon and diagnostically challenging condition due to its heterogeneous presentation and ability to mimic traumatic, infectious, or malignant lesions. Combined with limited awareness, this frequently results in delayed diagnosis, unnecessary interventions, and postponed treatment initiation.

Keywords: Diagnosis, Differential; Metatarsal Bones; Osteomyelitis

CR172

Beyond Conventional Immunosuppression: Successful Management of Refractory Multisystem Sarcoidosis with Adalimumab

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Introduction: Sarcoidosis is a multisystem, immune-mediated granulomatous disorder with highly variable clinical expression, most often affecting the lungs, skin, joints, and nervous system. Disease variety and unpredictable therapeutic responses contribute to a number of significant, challenging cases.

Case report: A patient with sarcoidosis presented with an acute exacerbation of chronic dyspnea that had been present for the previous three years. The dyspnea now occurred with minimal effort, including walking or speaking, without any chest pain. Chest X-ray and spirometry were within normal limits. Prior to this exacerbation, the patient had already been diagnosed with peroneus longus tenosynovitis, small fiber polyneuropathy, and seborrheic dermatitis with episodic flare-ups. Additional symptoms included ankle edema, paresthesia of the feet, and pain in the ankles and heels. Initial treatment included glucocorticoids and methotrexate 20 mg with folic acid supplementation, which was ended after five months due to gastrointestinal intolerance and limited efficacy. Afterwards, the patient developed worsening morning stiffness and progressive bilateral talocrural, knee, metacarpophalangeal, and hand joint pain, with a Visual Analogue Scale (VAS) score >9/10, requiring ambulation with crutch support. Leflunomide was initiated for sarcoidosis-associated arthritis and synovitis, but it did not provide clinical benefit. After four months, the patient was hospitalized for dyspnea, tachycardia, and dizziness, with unremarkable diagnostic findings. Due to persistent disease activity and functional decline, adalimumab 40 mg was initiated and administered subcutaneously every two weeks, resulting in significant clinical improvement without febrile episodes, dermatologic reactions, or other adverse effects.

Conclusion: This report emphasizes the importance of individualized treatment strategies in sarcoidosis. Recognizing clinical diversity, focusing on patient-reported outcomes, and adjusting treatment beyond standard immunosuppression are important in refractory disease, where biologic agents may provide clinical improvement and functional recovery.

Keywords: Adalimumab; Arthralgia; Dyspnea; Sarcoidosis

CR173

Dry Cough: Not Always a Lung Disease

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Introduction: Autoimmune Polyglandular Syndrome type 2 (APS-2) is a rare autoimmune disorder defined by the coexistence of primary adrenal insufficiency, which is mandatory for diagnosis, with autoimmune thyroid disease and/or type 1 diabetes mellitus. It usually manifests in adulthood and is the most common adult polyglandular autoimmune syndrome, with a prevalence of about 1:1000.

Case presentation: A 30-year-old man presented with a three-month history of progressive dyspnea and persistent dry cough, accompanied by electrolyte disturbances. Symptoms became evident after an appendectomy, while preoperative assessment first identified elevated lupus anticoagulant (LAC) levels and mild renal impairment, raising suspicion of antiphospholipid syndrome and acute renal insufficiency. Worsening hypoxemia and cyanosis prompted computed tomography pulmonary angiography, which revealed bilateral multifocal ground-glass opacities and mediastinal lymphadenopathy. As COVID-19 pneumonia and asthma were suspected, inhaled corticosteroid/long-acting β_2 -agonist therapy was initiated. However, further deterioration required hospitalization. Physical examination revealed diffuse skin hyperpigmentation with black linear discoloration of the lips. Laboratory investigations demonstrated elevated creatinine along with hyponatremia and hyperkalemia, positive antinuclear antibodies, and LAC. Endocrine evaluation revealed hypocortisolism, hyperthyroidism, and reduced free testosterone levels with elevated luteinizing hormone, while abdominal computed tomography excluded structural adrenal pathology. A diagnosis of APS-2 was established, comprising Addison's disease, Graves' disease, and primary hypogonadism in association with antiphospholipid syndrome. Glucocorticoid and mineralocorticoid replacement therapy led to normalization of laboratory results and resolution of pulmonary infiltrates. Thiamazole and hydroxychloroquine were subsequently introduced.

Conclusion: This case illustrates that chronic dry cough and radiological pulmonary abnormalities may be manifestations of underlying systemic endocrine and autoimmune disorders rather than primary pulmonary pathology. A holistic, multidisciplinary diagnostic approach is essential to prevent misdiagnosis and delayed recognition of potentially life-threatening systemic disease.

Keywords: Addison Disease; Antiphospholipid Syndrome; Autoimmune Diseases; Graves Disease

CR174

Autoimmune Lymphoproliferative Syndrome Presenting with SLE-Like Features in a Pediatric Patient

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Introduction: Autoimmune lymphoproliferative syndrome (ALPS) is an inherited disorder of immune dysregulation caused by defects in the FAS signaling pathway, leading to impaired lymphocyte apoptosis and accumulation of autoreactive CD4⁻CD8⁻TCRαβ⁺ double-negative T cells (DNT). Clinically, ALPS is characterized by lymphoproliferation, cytopenias, autoimmunity, and an increased risk of lymphoma and may mimic systemic autoimmune diseases, including systemic lupus erythematosus (SLE). Definitive diagnosis requires targeted immunological and genetic testing.

Case report: We present a boy born in 2015 who was referred to an immunologist at the age of 5 due to dysgammaglobulinemia. Initially asymptomatic, he was monitored over several years, during which laboratory abnormalities emerged, including polyclonal hypergammaglobulinemia, normocytic anemia, and accelerated erythrocyte sedimentation rate. Over time, antinuclear antibodies were detected with intermittent elevations of anti-double-stranded DNA antibodies, raising suspicion of SLE. Mild organ involvement, including borderline hepatosplenomegaly and generalized lymphadenopathy, supported this consideration, although clinical lupus manifestations were lacking. The coexistence of long-standing dysgammaglobulinemia, anemia, elevated vitamin B12 levels, and lymphoproliferation prompted evaluation for ALPS. Further work-up demonstrated increased DNT cells, significantly elevated vitamin B12 and soluble Fas ligand levels, all of which are established biomarkers for ALPS. Targeted genetic analysis identified a heterozygous *FAS* mutation (c.536T>G;p.Leu179Arg). At the age of 10, he developed immune thrombocytopenia resistant to intravenous immunoglobulins and systemic corticosteroids. He responded excellently to sirolimus, which is the treatment of choice in ALPS-associated cytopenias.

Conclusion: This case illustrates how ALPS can mimic pediatric SLE. Recognition of characteristic biomarkers and confirmation of a pathogenic *FAS* mutation were crucial for establishing the correct diagnosis. Targeted therapy with sirolimus achieved sustained remission of refractory cytopenia, highlighting the importance of timely genetic evaluation in suspected inborn errors of immunity.

Keywords: Autoimmune Lymphoproliferative Syndrome; Autoimmunity; fas Receptor; Lymphoproliferative Disorders; Sirolimus

CR175

Chorea as the Primary Manifestation of Neuropsychiatric Systemic Lupus Erythematosus: A Case Report

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Introduction: Systemic lupus erythematosus (SLE) is a chronic, multisystem autoimmune disease characterised by the production of autoantibodies against nuclear antigens, deposition of immune complexes, and chronic inflammation affecting multiple organs, most commonly the skin, joints, and kidneys. Neuropsychiatric SLE (NPSLE) refers to neurological syndromes involving the central, peripheral, and autonomic nervous systems, as well as psychiatric syndromes observed in patients with SLE. Movement disorders are a rare presentation of NPSLE, with chorea as the most common manifestation.

Case report: We present the case of a 68-year-old female with involuntary choreiform movements of the hand and face and dysarthria, who had a history of transient ischemic attacks. Magnetic resonance imaging demonstrated microangiopathic changes in the brain, and genetic testing excluded Huntington's disease. Elevated antiphospholipid antibodies (APA) were found, leading to referral to an immunologist. Five years earlier, she had a positive antinuclear antibody titer of 1:320 with cytoplasmic and homogeneous fluorescence, but did not follow up. Further tests showed elevated anti-double-stranded DNA and APA antibodies, and clinical examination revealed involuntary movements and mild polyarthritis. The workup showed no signs of involvement of other organs. Ultimately, she was diagnosed with NPSLE and secondary antiphospholipid syndrome. Treatment included high-dose glucocorticoids, mycophenolate mofetil, hydroxychloroquine, warfarin, and acetylsalicylic acid, resulting in significant clinical improvement.

Conclusion: Symptoms of SLE can resemble those of many other diseases and conditions, making accurate diagnosis challenging. This case underscores the importance of considering a broad spectrum of diseases, including SLE, when encountering a rare neurological presentation.

Keywords: Antiphospholipid Syndrome; Central Nervous System Diseases; Chorea; Lupus Erythematosus, Systemic

CR176

The Importance of Early Diagnosis of Severe Combined Immunodeficiency

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Introduction: Severe combined immunodeficiency (SCID) is the most severe form of congenital immunodeficiency, characterized by profound impairment of T-lymphocyte development and function. Newborn screening enables early identification of affected infants before the onset of life-threatening infections. If unrecognized and untreated, children with SCID usually die within the first two years of life.

Case report: A male newborn was hospitalized at 20 days of age due to bronchiolitis accompanied by generalized rash, liver lesion, and eosinophilia, which was interpreted as Drug Reaction with Eosinophilia and Systemic Symptoms. During evaluation, cytomegalovirus (CMV) infection was confirmed. He was discharged home after clinical improvement. At five months of age, he was rehospitalized due to tachypnoea, dyspnoea, and dry cough. Radiological examination revealed non-homogenous pulmonary opacities, while laboratory findings showed leucocytosis with relative lymphocytosis and normal inflammatory markers. Further evaluation revealed hypogammaglobulinemia, low CD4 lymphocytes, athymia, disseminated CMV infection, *Pneumocystis jirovecii* pneumonia, and disseminated *Bacillus Calmette-Guerin* infection, leading to suspicion of SCID. Genetic analysis confirmed a mutation in the *IL2RG* gene (c.265T>G, p.Tyr89Asp), the most common form, which is inherited in an X-linked manner. The patient had maternal T cell engraftment, explaining the rash, eosinophilia, and liver lesion during the neonatal period. At 10 months of age, a successful hematopoietic stem cell transplantation was performed. Due to a positive family history, the patient's younger brother was diagnosed in the second week of life. Antimicrobial prophylaxis and immunoglobulin replacement therapy were initiated immediately, isolation measures were implemented, and the child underwent transplantation at five months of age without prior infectious complications.

Conclusion: This case highlights the need to consider implementation of newborn screening in order to allow timely curative treatment and prevent severe infectious complications.

Keywords: Cytomegalovirus Infections, Hematopoietic Stem Cell Transplantation, Opportunistic Infections, Severe Combined Immunodeficiency

CR177

From Genomic Diagnosis to Curative Therapy: A Severe Early Presentation of X-Linked Hyper-IgM Syndrome

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Introduction: X-linked hyper-IgM syndrome is a rare primary immunodeficiency caused by mutations in the CD40LG gene, resulting in defective immunoglobulin class-switch recombination and combined humoral and cellular immune dysfunction. Diagnosis relies on characteristic immunoglobulin profiles, flow cytometric assessment of CD40 ligand expression, and confirmatory genetic testing.

Case report: A 5.5-month-old male infant was admitted to a tertiary pediatric intensive care unit for progressive respiratory failure following two weeks of poor feeding, irritability, and failure to thrive. He rapidly developed hypoxemia requiring mechanical ventilation. Chest imaging demonstrated bilateral patchy pulmonary infiltrates. Immunological testing revealed profound hypogammaglobulinemia with absent IgG and IgA and preserved IgM levels, raising suspicion of a primary immunodeficiency. Polymerase chain reaction analysis confirmed *Pneumocystis jirovecii* pneumonia, along with disseminated cytomegalovirus (CMV) infection and CMV pneumonitis. Rhinovirus was also identified. Immunological workup showed lymphopenia with reduced T-cell and NK-cell counts and relative B-cell expansion, as well as markedly decreased CD40 ligand (CD154) expression on activated T lymphocytes. The child had thymus (seen on chest X-ray and on ultrasound). Fluorescence in situ hybridization excluded maternal lymphocyte engraftment. Based on the clinical course, immunological findings, and severe opportunistic infections he was diagnosed with X-linked hyper-IgM syndrome, which was genetically confirmed one month later. The patient was stabilized with targeted antimicrobial and antiviral therapy and intravenous immunoglobulin replacement. At 1 year and 7 months of age, he underwent allogeneic hematopoietic stem cell transplantation from a matched unrelated donor, achieving full donor chimerism.

Conclusion: This case illustrates a severe early-onset presentation of X-linked hyper-IgM syndrome with life-threatening opportunistic infections, in which integrated immunologic and genomic diagnostics enabled timely treatment with an excellent clinical outcome.

Keywords: Hyper-IgM Immunodeficiency Syndrome; CD40 Ligand; *Pneumocystis jirovecii*; Hematopoietic Stem Cell Transplantation

CR178

Diabetic ketoacidosis triggered by sepsis leading to cardiopulmonary arrest**Lucija Stanić¹; Romana Serdarušić¹; Anita Božičević Vadjlaja²***1 - Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Emergency, University Hospital Centre Osijek, Osijek, Croatia*

Introduction: Diabetic ketoacidosis (DKA) is often triggered by infection and may progress to sepsis, which in turn exacerbates metabolic decompensation. Meanwhile, hyperglycemia, acidosis and dehydration impair the immune response, increasing the risk of septic shock and mortality.

Case report: The 41-year-old patient was admitted to the Intensive Care Unit for internal medicine patients via the Emergency Department due to immeasurably high blood glucose levels, sepsis, septic shock and DKA. Upon admission, he was uncooperative due to the severity of his condition and treatment was immediately initiated. The patient received a continuous intravenous infusion of short-acting insulin (NovoRapid, 10 IU in 500 ml of saline) along with additional fluid replacement with 500 ml of saline and intravenous administration of 20 mmol potassium chloride. Simultaneously, oxygen therapy was provided at a flow rate of 6 L/min. Throughout the hospitalization, the patient remained hemodynamically unstable and required continuous vasoactive support due to pronounced hypotension, while respiratory function remained stable. His general condition progressively deteriorated, with a drop in arterial blood pressure despite high doses of vasoactive agents. He developed bradycardia and asystole, after which cardiopulmonary resuscitation was immediately initiated. Despite all intensive care interventions and treatment measures, the patient ultimately died in the setting of cardiopulmonary arrest.

Conclusion: DKA is a severe metabolic complication with the potential to develop into sepsis. When combined with profound hemodynamic instability DKA may result in cardiopulmonary arrest and death. Any case of DKA with signs of infection and hypotension should prompt prompt immediate and intensive multidisciplinary management.

Keywords: Cardiopulmonary Resuscitation; Diabetic Ketoacidosis; Heart Arrest; Shock

CR179

When Transverse Myelitis Is More Than Isolated Spinal Disease: A Case of Seronegative NMOSD

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Introduction: Transverse myelitis is a rare inflammatory disorder of the spinal cord causing motor, sensory, and autonomic dysfunction. Longitudinally extensive lesions, particularly when associated with optic nerve involvement, should raise suspicion of neuromyelitis optica spectrum disorder (NMOSD), including seronegative forms.

Case report: A 41-year-old man with long-term alcohol and psychoactive substance abuse and no relevant family neurological history was hospitalized due to subacute neurological symptoms. In April 2025, he underwent surgery for a right tibial fracture under spinal anaesthesia. In May, he developed periumbilical paresthesias with caudal spread, progressive lower limb weakness, gait impairment, and urinary retention while being treated for right leg cellulitis. Neurological examination revealed flaccid paraparesis, sensory loss below Th10–Th11, and urinary retention. Spinal magnetic resonance imaging demonstrated longitudinally extensive transverse myelitis (LETM), with a focal lesion at C6 and thoracic involvement from Th2 to Th11, without spinal cord compression. Cerebrospinal fluid analysis revealed mild lymphocytic pleocytosis and elevated protein levels. Pulse corticosteroid therapy led to partial neurological recovery. In June, the patient sustained traumatic rectal and bladder lacerations following a fall attributed to persistent lower limb weakness, necessitating a Hartmann procedure. During follow-up, he experienced recurrent episodes of painful visual loss consistent with optic neuritis, each resolving after corticosteroid therapy. Immunological testing revealed negative aquaporin-4 and myelin oligodendrocyte glycoprotein antibodies, with serological evidence of prior varicella-zoster virus infection. Based on clinical and radiological findings and treatment response, seronegative NMOSD was diagnosed. Spastic paraparesis (EDSS 4.5) prompted rituximab therapy, achieving clinical stabilization.

Conclusion: LETM should be recognized as an early warning sign of a potentially relapsing inflammatory disease of the central nervous system. Early recognition enables timely immunotherapy initiation, potentially preventing permanent neurological disability.

Keywords: Myelitis, Transverse; Neuromyelitis Optica; Paraparesis, Spastic; Rituximab

CR180

Manifestations of Common Variable Immunodeficiency (CVID) in a 67-year-old male: a case report**Ivan Stanušić¹; Franjo Lapić¹; Jana Skender¹; Neva Rašperić¹; Ana Gudelj Gračanin²***1 - School of Medicine, University of Zagreb, Zagreb, Croatia**2 - Department of Clinical Immunology and Rheumatology, University Hospital "Sveti Duh", Zagreb, Croatia*

Introduction: Common Variable Immunodeficiency (CVID) is the most common symptomatic primary immunodeficiency in adulthood. It is characterized by hypogammaglobulinemia, recurrent infections, and frequent autoimmune and inflammatory complications. Due to the heterogeneous clinical picture, diagnosis is often delayed, which can lead to the development of severe complications.

Case report: A 67-year-old patient with a history of frequent pneumonias and autoimmune thyroiditis presented with hemorrhoidal bleeding and subconjunctival hemorrhage of the left eye due to severe secondary immune thrombocytopenia resulting from humoral immunodeficiency and from recent administration of influenza and SARS-CoV-2 vaccines to the patient. Treatment with corticosteroids was initiated with only a transient therapeutic response, and after a renewed drop in platelet count, intravenous immunoglobulin therapy was initiated, after which a significant increase in platelet count occurred. Immunohematological testing in the patient's plasma demonstrated antiplatelet antibodies. Further laboratory testing revealed hypogammaglobulinemia (Immunoglobulin G serum concentration was 4,74 g/L and Immunoglobulin M concentration was 0,11 g/L), which led to the conclusion that immune thrombocytopenia was an autoimmune manifestation of CVID. The patient's symptoms had drastically improved, he was discharged, and treatment with intravenous immunoglobulin therapy was started. However, this therapy was discontinued after three months due to its adverse effects, subcutaneous immunoglobulin therapy was initiated instead, which in his case is a lifelong replacement therapy due to his primary hypogammaglobulinemia.

Conclusion: This case demonstrates the importance of considering CVID in elderly patients with recurrent infections and autoimmune manifestations because early recognition of the disease and early initiation of immunoglobulin therapy are crucial for preventing infections, reducing autoimmune complications, and improving the patient's quality of life.

Keywords: Autoimmune Disease; Hypogammaglobulinemia; Thrombocytopenia

CR181

Myeloproliferative Neoplasm Complicated by Severe Cardiomyopathy and Pregnancy-Related Adverse Outcomes

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Introduction: Myeloproliferative neoplasms (MPNs) are clonal hematopoietic stem cell disorders characterized by proliferation of one or more myeloid lineages and require long-term, individualized treatment. The aim of this case is to present the clinical course of an MPN patient complicated by cardiomyopathy, adverse pregnancy outcomes, and therapy-related side effects.

Case Report: In March 2010, a 20-year-old female patient was diagnosed with a myeloproliferative neoplasm after incidentally discovered thrombocytosis. Diagnostic evaluation revealed *Janus Kinase 2* mutation positivity, negative *BCR-ABL*, normal karyotype (46,XX), and bone marrow findings consistent with essential thrombocythemia. The patient was lost to follow-up after May 2011. In September 2013, she was hospitalized due to newly diagnosed cardiomyopathy of unclear etiology with severely reduced ejection fraction ranging from 10% to 15% and pulmonary hypertension. Laboratory findings showed persistent leukocytosis, erythrocytosis, and thrombocytosis, without splenomegaly. Therapeutic phlebotomies were initiated along with hydroxyurea and acetylsalicylic acid. At the 2019 evaluation, no cardiological symptoms or significant cardiac pathology were documented. In February 2020, pegylated interferon alfa was recommended instead of hydroxyurea due to pregnancy planning. In October 2020, the patient developed preeclampsia at 33 weeks of gestation requiring emergency cesarean section and antihypertensive therapy. Due to alopecia, treatment was continued with ropeginterferon. After its introduction, psychological restlessness occurred and required psychiatric treatment. Subsequently, fetal loss occurred at 10 weeks of gestation in January 2025. Further follow-up was arranged.

Conclusion: This case demonstrates the complexity of long-term management of myeloproliferative neoplasms, particularly in patients with cardiovascular and pregnancy-related complications and therapy-related adverse effects. The findings emphasize the importance of continuous follow-up, multidisciplinary care, and individualized therapeutic strategies.

Keywords: Cardiomyopathies; Myeloproliferative Disorders; Pregnancy Complications

CR182

Familial Hypercholesterolemia and Modern Therapeutic Intervention

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Introduction: Familial hypercholesterolemia (FH) is the most common monogenic metabolic disorder and a leading genetic cause of premature death. Primarily caused by mutations in the *low-density lipoprotein (LDL) receptor* gene, and less frequently in the *APOB* or *PCSK9* genes, FH results in lifelong LDL cholesterol (LDL-C) elevation. In Croatia, approximately 10,000-18,000 individuals are affected, according to the Croatian Institute of Public Health (2023). FH accelerates atherosclerotic cardiovascular disease (ASCVD); heterozygous forms double LDL-C levels, while rare homozygous forms carry a poor prognosis. Current guidelines emphasize aggressive LDL-C targets, often unattainable with conventional therapy, highlighting the role of small interfering RNA (siRNA) in high-risk patients.

Case report: We present a 60-year-old patient with arterial hypertension, type 2 diabetes, and established ASCVD (status post ST-elevation myocardial infarction (STEMI) with percutaneous coronary intervention (PCI) of the right coronary artery). Despite treatment with maximally tolerated doses of rosuvastatin and ezetimibe, LDL-C remained elevated. FH was diagnosed via Dutch Lipid Clinic Network Score. The clinical course was complicated by a third-degree intermittent atrioventricular (AV) block, requiring a dual-chamber (DDD) permanent pacemaker. Given this high-risk profile and failure to reach target LDL-C levels, inclisiran was introduced as an add-on therapy. As an siRNA, inclisiran inhibits hepatic synthesis of the PCSK9 protein, increasing LDL-receptor density and clearance. After the initial doses, LDL-C decreased from 3.6 mmol/L to 1.9 mmol/L. This approach was essential to stabilize plaque progression where oral therapies reached their limits.

Conclusion: This case emphasizes inclisiran's importance in high-risk FH. Its twice-yearly dosing ensures sustained LDL-C reduction and addresses medication adherence, offering a vital pathway for improving the prognosis in complex coronary patients.

Keywords: Hypercholesterolemia, Familial; PCSK9 Inhibitors; Receptors, LDL; RNA, Small Interfering

CR183

The Role of CGM Systems in Reducing Glycemic Variability and Hypoglycemia Risk in Geriatric Patients

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Introduction: In 2024, Diabetes Mellitus was the fourth leading cause of death in Croatia. Roughly 1 in 5 people aged 65-99 live with diabetes. For elderly patients, glycemic control is challenging due to age-related hypoglycemia unawareness, where weakened hormonal responses mask early warning signs, often making confusion or sudden falls the first visible symptoms.

Case report: We present an 87-year-old male with a 30-year history of Type 2 Diabetes Mellitus (T2DM) and comorbidities: heart failure, chronic kidney disease, and atrial fibrillation. His intensive insulin therapy and declining renal function significantly heightened the risk of hypoglycemia. Diary entries revealed frequent blood glucose drops to 3 mmol/L after meals, resulting in mood shifts and disruptions to daily activity. Such episodes carry risks, as mortality rates of up to 10% are linked to associated cardiac events. Upon initiating continued glucose monitoring (CGM), the target Time in Range (TIR) was set at 3.9–10.0 mmol/L. Post-implementation, the patient achieved 82% TIR, 0% Time Below Range (TBR), and 18% Time Above Range. His glycemic variability remained below 30%, indicating high stability.

Conclusion: CGM systems are a vital tool for the early detection of asymptomatic hypoglycemia and stabilizing glycemia in elderly populations with T2DM. This technology identifies glucose trends that traditional diaries miss, preventing life-threatening cardiovascular and neuroglycopenic incidents. Our findings suggest that for patients with hypoglycemia unawareness and significant comorbidities, CGM is essential for minimizing glycemic variability and reducing neuroglycopenic risks.

Keywords: Diabetes Mellitus, Type 2; Blood Glucose; Hypoglycemia; Aged, 80 and over

CR184

Surgical management of hand pseudosyndactyly in dystrophic epidermolysis bullosa using an anterolateral thigh flap

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Introduction: Dystrophic epidermolysis bullosa (DEB) results from mutations in the COL7A1 gene and causes chronic wounds, progressive scarring, joint contractures and pseudosyndactyly progressing to a "mitten hand" deformity. In hand reconstruction, the anterolateral thigh (ALT) flap is preferred over split thickness skin grafts because it provides stable, well vascularized tissue that is more resistant to secondary contractions, limiting the often-expected recurrence of pseudosyndactyly and improving long-term functional outcomes.

Case report: A 38-year-old male with DEB sought medical attention for severe functional impairment of the left hand due to recurrence of pseudosyndactyly and contractures. He had previously undergone multiple finger separation procedures, all complicated by graft failure and recurrence. Clinical examination revealed near-complete fusion of the digits and profound loss of hand function. Surgical reconstruction of the left hand started by releasing the scar tissue, correcting basal syndactyly of the third to fifth digit, and deepening interdigital spaces. Afterwards soft-tissue reconstruction was performed by using vascularized tissue from the ALT flap allowing simultaneous finger separation. During hospitalization, the patient received thromboprophylaxis, antibiotic therapy and analgesics with ongoing wound care. The postoperative course was uneventful, followed by regular graft healing and significant improvement in hand grip and range of motion. The next follow-up is planned in three months with attention to possible pseudosyndactyly relapse.

Conclusion: This case highlights the advantages of using the ALT flap in hand reconstruction. Surgical intervention is crucial for patients with DEB as it provides a worthwhile improvement in symptoms, function and appearance for several years. The use of vascularized flaps represents a valuable option in the treatment of complex hand deformities in DEB.

Keywords: Epidermolysis Bullosa; Plastic Surgery Procedures; Surgical Flaps

CR185

Thromboembolism in a Young Woman: The Interplay of Genetic and Acquired Risk Factors

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Introduction: Deep vein thrombosis and pulmonary embolism are rare in young adults. The presence of genetic factors, such as factor II (FII) mutations and antiphospholipid syndrome, substantially increases the risk of venous thromboembolism, particularly when combined with acquired risk factors, including hormonal contraception.

Case Report: A 23-year-old woman presented with pain and edema of the left thigh that persisted despite ibuprofen use. Six months prior, she had started using a contraceptive patch. Family history was significant for a father with thrombosis and a confirmed heterozygous FII mutation. The patient was hemodynamically stable. Laboratory evaluation revealed elevated D-dimer (3135 ng/mL), while the coagulation profile remained within reference limits. Venous ultrasound demonstrated thrombosis of the left common and external iliac veins, with noncompressible common and superficial femoral veins. Multislice Computed Tomography (MSCT) confirmed venous thrombosis and identified a filling defect in the lobar branch of the right pulmonary artery, consistent with pulmonary embolism. The patient was initiated on low-molecular-weight heparin and underwent mechanical thrombectomy. She was subsequently discharged on apixaban for six months, with follow-up recommended and hormonal contraception discontinued. Hematology consultation advised further evaluation for thrombophilia and antiphospholipid syndrome, with results pending.

Conclusion: This case underscores the importance of early recognition of venous thromboembolism in young individuals with familial thrombophilia. The combination of genetic predisposition and acquired risk factors, such as hormonal contraception, emphasizes the need for individualized monitoring and genetic assessment of family members.

Keywords: Pulmonary embolism; Thromboembolism; Thrombophilia

CR186

X-linked Thrombocytopenia – Instructive Example of the Importance of Genetic Diagnostics

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Introduction: X-linked thrombocytopenia (XLT) is a rare inherited disorder that may occur isolated or as part of Wiskott-Aldrich syndrome. The main characteristic of XLT is isolated thrombocytopenia with small platelets. It results from a mutation in the gene encoding the WAS protein, located on the X chromosome.

Case report: A male newborn was diagnosed with thrombocytopenia ($60 \times 10^9/L$) at four weeks of age. Antiplatelet antibodies were initially negative, but repeat testing was positive, raising suspicion of autoimmune thrombocytopenia. At two years of age, the boy developed Kawasaki disease and received immunomodulatory treatment with intravenous immunoglobulins, after which his platelet count temporarily normalized, a finding typically seen in immune thrombocytopenia. At three years of age, he was hospitalized due to severe thrombocytopenia ($<5 \times 10^9/L$) and epistaxis. Platelet transfusion was administered due to acute bleeding. Bone marrow aspiration showed moderately preserved thrombocytopoiesis with reduced numbers of megakaryocytes. Due to the unusual clinical course and the presence of microthrombocytes, genetic analysis was performed and showed a mutation in the WASP gene which manifests as X-linked thrombocytopenia or Wiskott-Aldrich syndrome. The boy did not meet clinical criteria for Wiskott-Aldrich syndrome (eczema, microthrombocytopenia, and immunodeficiency). At the age of four, our patient successfully underwent allogeneic hematopoietic stem cell transplantation. The mutation was inherited from his mother, a healthy carrier of WASP mutation. During family planning, the parents opted for preimplantation genetic diagnosis. Consequently, the patient's brother was born as a second, healthy child without the mutation.

Conclusion: This case demonstrates that genetic analysis is sometimes essential for establishing an accurate diagnosis and for the optimal treatment approach. It also highlights the importance of appropriate genetic counselling in family planning.

Keywords: Hematopoietic Stem Cell Transplantation, Thrombocytopenia, Wiskott-Aldrich Syndrome

CR187

Diffuse Large B-Cell Lymphoma Treated With R-CHOP After Kidney Transplantation

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Introduction: Posttransplant lymphoproliferative disorders are serious complications of long-term immunosuppression after solid organ transplantation. One of the complications is diffuse large B-cell lymphoma (DLBCL) so this case highlights the importance of early management.

Case report: A 64-year-old male patient who had suffered from two kidney infections in his youth, had undergone hemodialysis and then a kidney transplant in 2015 was hospitalized due to worsening renal function. Multislice computed tomography revealed a retroperitoneal mass next to the transplanted kidney, paraaortic lymphadenopathy, splenic lesions, and a small liver lesion. In September 2023, a computed tomography-guided biopsy of the retroperitoneal mass confirmed diffuse large B cell non-Hodgkin lymphoma at Ann Arbor stage IV. Treatment with the R-CHOP protocol was initiated from October 2023 to April 2024. During treatment, the patient was hospitalized due to prolonged fever of unclear origin. A polymerase chain reaction detected a SARS-CoV-2 infection with concurrent cytomegalovirus (CMV) and Epstein-Barr virus (EBV) viremia, which were treated with remdesivir and valganciclovir. Subsequently, the patient developed deep vein thrombosis of the left calf, which was managed with anticoagulant therapy. At the conclusion of the treatment in May 2024, a positron emission tomography-computed tomography was performed, showing complete metabolic remission (Deauville score 2). Subsequent follow-up demonstrated a stationary state of the graft without active lymphoproliferative disease and negative CMV and EBV viremia.

Conclusion: This case highlights the importance of monitoring the condition of patients after solid organ transplantation and the importance of a multidisciplinary approach where successful treatment of infections and aggressive chemoimmunotherapy resulted in recovery with preserved renal function.

Keywords: Antineoplastic Combined Chemotherapy Protocols; Kidney Transplantation; Lymphoma; Lymphoproliferative Disorders; Opportunistic Infections

CR188

Pulse wave velocity-guided personalized management in a patient with arterial hypertension

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Introduction: Arterial hypertension is a major risk factor for cardiovascular morbidity and mortality. In addition to elevated blood pressure, detection of subclinical target organ damage significantly improves risk stratification. Pulse wave velocity (PWV) is a noninvasive biomarker of arterial stiffness and, according to the 2023 ESH Guidelines, a key marker of hypertension-mediated organ damage. This case highlights the role of PWV in tailoring antihypertensive therapy to the individual patient, exemplifying the move toward precision medicine.

Case report: A 48-year-old male patient was diagnosed with stage 1 arterial hypertension with initial office blood pressure values of approximately 148/92 mmHg and body mass index of 36.5 kg/m², corresponding to class II obesity. His medical history was unremarkable, with no diabetes, chronic kidney disease, or previous cardiovascular events. Laboratory evaluation, including renal function and lipid profile, was within normal limits. As part of an extended cardiovascular assessment, carotid-femoral PWV was measured and found to be elevated at 11.4 m/s, indicating increased arterial stiffness and subclinical vascular damage. Based on this finding, initial dual antihypertensive therapy was started using a fixed-dose combination of an angiotensin-converting enzyme inhibitor and a calcium channel blocker. Therapy was titrated over several weeks with the goal of achieving systolic blood pressure values closer to the lower end of the recommended range. At follow-up, blood pressure values improved to approximately 130/80 mmHg. PWV was not remeasured during the short-term follow-up period.

Conclusion: This case demonstrates that PWV measurement, although it does not change the standard initial antihypertensive regimen, provides essential information on subclinical vascular damage. This enables a more accurate assessment of cardiovascular risk and helps guide individualized treatment intensity, therapeutic targets, and monitoring frequency.

Keywords: Hypertension; Pulse Wave Analysis; Vascular Stiffness

CR189

Bilateral Pneumonia and Respiratory Insufficiency in a CVID Patient who Discontinued Immunoglobulin Replacement Therapy: A Case Report

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Introduction: Common Variable Immunodeficiency (CVID) is a primary immune disorder characterized by impaired antibody production, specifically of immunoglobulin (Ig) types IgG, IgM and IgA, leading to recurrent, severe infections, particularly affecting the respiratory system. In this case report, we present how discontinuation of immunoglobulin replacement therapy can lead to the complications described.

Case report: A 46-year-old female with known CVID, previously maintained on intravenous immunoglobulin (IVIG) replacement therapy, discontinued treatment on her own. She presented to the emergency department with fever, productive cough, fatigue and progressive dyspnea. Upon admission, she was febrile, tachypnoeic, hypotensive and hypoxaemic. Chest X-ray showed bilateral inflammatory infiltrates and laboratory findings showed systemic inflammation. Computed Tomography Pulmonary Angiography (CTPA) excluded pulmonary thromboembolism. A nasopharyngeal swab polymerase chain reaction (PCR) test detected *Haemophilus influenzae*, supporting a bacterial aetiology in the specified clinical and radiological context. Empiric broad-spectrum antimicrobial therapy was initiated due to clinical severity and subsequently de-escalated to amoxicillin/clavulanate based on microbiological results and clinical improvement. IVIG replacement was re-initiated during hospitalization. The patient improved clinically, with decreased oxygen requirements and reduced inflammatory markers, and was discharged with a plan for continued immunoglobulin replacement and follow-up.

Conclusion: This case emphasizes the importance of following prescribed therapy regimens correctly in order to avoid unnecessary and life-threatening complications. Consistent long-term immunoglobulin therapy in CVID patients is crucial for preventing severe infections and reducing hospital admissions.

Keywords: Common Variable Immunodeficiency; Intravenous Immunoglobulin; Pneumonia; Respiratory Insufficiency

CR190

Granulomatosis with Polyangiitis: A Challenging Case with Renal, Pulmonary, and Otologic Manifestations

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Introduction: Granulomatosis with polyangiitis (GPA), formerly known as Wegener's Granulomatosis, is a rare systemic autoimmune disease characterized by granulomatous inflammation, primarily affecting the respiratory tract and kidneys. This case highlights the clinical complexity of GPA, therapeutic challenges, and the importance of long-term multidisciplinary care.

Case report: A 69-year-old female diagnosed with GPA in September 2024 at an external institution presented with pulmonary, sinonasal, and renal involvement. Laboratory tests showed significant proteinuria (2341 mg/day) and elevated anti-neutrophil cytoplasmic antibodies (ANCA) with anti-proteinase 3 (PR3) specificity (375 AU/mL). Renal biopsy revealed pauci-immune rapidly progressive glomerulonephritis. Pulmonary function tests showed moderate-to-severe obstruction and reduced diffusion capacity. Therapy included induction with cyclophosphamide until March 2025, followed by maintenance with azathioprine, which was discontinued due to inefficacy. Mycophenolate mofetil was introduced in November 2025. The patient also received intravenous immunoglobulin therapy for secondary hypogammaglobulinemia. During the disease course, she developed chronic otitis media with tympanic membrane perforation and progressive bilateral hearing loss. At the most recent follow-up, while under ongoing immunosuppressive therapy, she remained clinically stable. Immunoglobulin G levels were within normal range, ANCA/PR3 titers decreased, and proteinuria was reduced but persisted (1370 mg/day). Repeated imaging showed partial regression of pulmonary infiltrates. Her bilateral hearing loss remained. The next step involves rituximab to optimize disease control while minimizing immunosuppressive risk.

Conclusion: Although Granulomatosis with polyangiitis remains a challenging and potentially life-threatening systemic disease, advances in immunosuppressive therapy have significantly improved the prognosis. This case emphasizes the critical role of early diagnosis, close monitoring, and personalized treatment in achieving effective disease control.

Keywords: Anti-Neutrophil Cytoplasmic Antibody-Associated Vasculitis; Granulomatosis with Polyangiitis; Hearing Loss; Immunosuppression Therapy; Rituximab

CR191

Like Mother, Like Daughter: A Rare Case of Hereditary Autoinflammatory Disease

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Introduction: Autoinflammatory diseases are disorders of innate immunity often caused by specific gene mutations. Their low prevalence and similarity of symptoms between subgroups makes diagnosis difficult. Tumor necrosis factor (TNF) receptor-associated periodic syndrome (TRAPS) is a hereditary autoinflammatory disease characterized by recurring fevers, arthralgia, abdominal pain and rash with other nonspecific symptoms. Early diagnosis and treatment are key as it can cause severe organ damage.

Case report: A 13-year-old female presented with abdominal pain and fever followed by increased C-reactive protein (CRP). Infection was ruled out and additional gastroenterological workup was unremarkable. These episodes recurred over the years, but the diagnosis remained unknown. Regarding medical history, the patient had a tonsillectomy and appendectomy in childhood and is being treated for juvenile arthritis. At 20-years-old the patient was diagnosed with ulcerative colitis due to aphthous lesions found on ileocolonoscopy, CRP of 260 mg/L and calprotectin >1000 µg/g. Treatment consisted of azathioprine and high dose glucocorticoids. Symptoms subsided during therapy, but intensified after steroid reduction. The patient's mother was previously diagnosed with TRAPS as genetic testing found a mutation specific for the disease (heterozygous variant rs104895224 in gene *TNFRSF1A*). The same mutation was found in our patient, which lead to the same diagnosis. Due to suspected concomitant ulcerous colitis, the patient received three doses of infliximab and developed hyperinflammatory reaction. After changing the treatment to tocilizumab the patient is well and in permanent remission.

Conclusion: Rare diseases such as TRAPS often remain undiagnosed for years and lead to inadequate treatment and reduced quality of life. Detailed family history and genetic testing are essential for early diagnosis, while keeping in mind that the same mutation can lead to different phenotype expression.

Keywords: Fever; Hereditary Autoinflammatory Diseases; Immunity, Innate; Mutation; Tumor Necrosis Factor-alpha

CR192

Personalized management of eosinophilic esophagitis following acute impaction: A case report

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Introduction: Eosinophilic esophagitis (EoE) is a chronic, immune-mediated clinicopathological disease recognized in young adults. This case illustrates the transition from emergency management to a personalized diagnostic and therapeutic approach, thereby preventing disease progression and enhancing long-term outcomes..

Case report: A 24-year-old male with no significant medical history was admitted due to acute dysphagia and inability to swallow solids and liquids after eating lamb. He reported retrosternal discomfort but denied previous episodes. Emergency upper gastrointestinal endoscopy revealed complete distal esophageal obstruction by a food bolus, which was removed using a retrieval basket and forceps. At the impaction site, a mucosal laceration (15–20 mm) was treated with endoscopic clips. Additionally, endoscopic features suggestive of EoE, including esophageal trachealization, were noted. Contrast-enhanced computed tomography (CT) of the chest revealed pneumomediastinum, and the patient was managed conservatively with analgesia, antibiotics, proton pump inhibitors (PPIs), and close monitoring. Following a normal follow-up esophageal transit study, the patient was discharged on the eighth day. One month after the initial endoscopy, a follow-up esophagogastroduodenoscopy was performed, and biopsies were obtained according to the EoE protocol. The Endoscopic Reference Score (EREFS) was 3. Histopathological analysis revealed dense eosinophilic infiltration >70 eosinophils per high-power field (HPF), with eosinophilic microabscesses, confirming the diagnosis of EoE. Following histological confirmation, therapy with PPIs and an elimination diet was initiated.

Conclusion: This case illustrates that acute food bolus impaction may represent the first manifestation of EoE. Histological confirmation enabled a transition from emergency intervention to a personalized management strategy, including dietary modification and pharmacotherapy, thereby reducing the risk of recurrence and long-term esophageal remodeling.

Keywords: Endoscopy, Gastrointestinal; Eosinophilic Esophagitis; Esophageal Diseases; Precision Medicine

CR193

Targeted BRAF and MEK Inhibitor Therapy in a Patient with Melanoma of Unknown Primary

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Introduction: Melanoma of unknown primary is a rare condition in which patients present with melanoma metastasis, yet the primary site cannot be discovered through physical exams or imaging. Mutations in the gene *BRAF* are detected in approximately 50% of cutaneous melanomas and have led to the evolution of targeted therapy, which has enhanced treatment effectiveness and overall therapy results.

Case report: A 61-year-old man presented with a suspicious preauricular mass. Biopsy and histopathological analysis revealed melanoma metastasis. A preoperative positron emission tomography/computed tomography (PET/CT) scan showed lesions in the left parotid gland and left cervical region II, as well as enlarged lymph nodes showing abnormal radiotracer uptake. The patient initially decided not to go through with the surgery. Nevertheless, when the mass was eventually removed, pathology confirmed parotid melanoma metastasis, and genetic testing revealed a *BRAF* codon 600 mutation (V600E/E2/D). A complete dermatological examination did not reveal a primary melanoma. During the subsequent radical left neck dissection, two lymph nodes containing melanoma were removed. The multidisciplinary melanoma team defined the tumor as stage III and suggested adjuvant therapy using BRAF and MEK inhibitors. The patient received dabrafenib and trametinib for one year. At a dermatological follow-up seven months after the end of treatment, he developed diffuse vitiligo, which was considered an immune-mediated response. The patient remains in remission, with advised dermatological and oncological follow-ups every six months.

Conclusion: The detection of the *BRAF* mutation enabled the use of targeted therapy using BRAF and MEK inhibitors, even though the primary site was never discovered. This combination blocks the MAPK pathway activated by BRAF, and thus helps to manage uncontrolled cell growth, as well as improve the treatment outcome.

Keywords: Melanoma; Mutation; Neoplasms, Unknown Primary

CR194

A Patient With Ulcerative Colitis and High Cardiovascular Risk Initiated on Immunomodulatory Therapy Through General Practitioner Intervention

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Introduction: Personalized medicine increasingly relies on understanding individual immune and genetic profiles, particularly in patients with chronic inflammatory diseases and multiple comorbidities. This case illustrates how immunomodulatory treatment improved outcomes in a complex patient, with a central role of the general practitioner (GP).

Case report: A 61-year-old woman with a long-standing history of ulcerative colitis, psoriasis, arterial hypertension, dyslipidemia and established coronary artery disease was followed in a primary care setting. She previously underwent percutaneous coronary intervention with drug-eluting stent implantation to the left anterior descending artery after acute coronary syndrome. Despite adherence to dietary recommendations, regular physical activity and high-intensity statin therapy, low density lipoprotein cholesterol levels remained above recommended targets for very high cardiovascular risk, suggesting a genetically driven dyslipidemia. At the same time, intestinal disease activity progressed, with frequent bloody stools and impaired quality of life. Based on clinical deterioration, the GP initiated and suggested targeted immunomodulatory therapy. Following gastroenterology evaluation, therapy directed at tumor necrosis factor alpha, adalimumab, was initiated, resulting in rapid clinical improvement and remission of gastrointestinal symptoms. Due to persistent hypercholesterolemia despite lifestyle measures and pharmacological treatment, a gene-based lipid-lowering therapy targeting proprotein convertase subtilisin/kexin type 9 synthesis, inclisiran, was introduced following primary care recommendation and specialist assessment. Both therapies were well tolerated. No significant adverse effects or infectious complications were recorded. Continuous follow up in general practice ensured monitoring, coordination between specialties and patient education.

Conclusion: This case demonstrates how effective navigation of the healthcare system by the GP enables timely initiation of two modern immunomodulatory therapies. Active coordination within primary care supported rapid access to advanced treatment and a patient centered approach to complex chronic disease management.

Keywords: Adalimumab; Cardiovascular Diseases; Colitis, Ulcerative; Immunomodulation; Proprotein Convertase 9

CR195

To be or not to be - biologic agents in the treatment of atopic dermatitis: a case report**Graciela Špionjak¹ ; Josipa Vasilj¹; Antoaneta Taslidžić¹; Melita Vukšić Polić^{2,3}***1 - Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Dermatology and Venereology, University Hospital Center Osijek, Osijek, Croatia**3 - Department of Dermatovenereology, Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia*

Introduction: Atopic dermatitis (AD) is a chronic inflammatory skin disease characterised by epidermal barrier dysfunction and immune dysregulation in genetically predisposed individuals, presenting as eczema, xerosis, and pruritus. Barrier impairment allows antigen entry, triggering T helper cell responses, with Th2-mediated inflammation (IL-4, IL-5) in acute lesions and Th1-mediated inflammation (IFN- γ , IL-12) in chronic lesions. Janus kinase (JAK) inhibitors target the JAK-mediated pathway, achieving effective AD control. However, they increase the risk of infection, as shown in this case.

Case report: We report a case of a 29-year-old male with severe, lifelong AD, significantly aggravated during the last five years. Previous topical treatments, antihistamines, and phototherapy provided only short-term benefit, leading to the use of systemic methotrexate (October 2022 – September 2023) for more durable control. This was partially effective but discontinued due to gastrointestinal side effects. There was no personal or family history of other atopic diseases. Laboratory findings revealed elevated total IgE (345 kU/L) and positive IgG gliadin antibodies without clinical evidence of celiac disease. Disease severity was high (SCORAD 63.6, DLQI 20). In May 2024, therapy with the JAK inhibitor abrocitinib (200 mg daily) was initiated, leading to rapid disease control. In January 2025, the patient developed right-sided blepharitis with periorbital edema, pustules, and secondary bacterial (*Staphylococcus aureus*) and suspected herpes infection. Abrocitinib was temporarily discontinued and antiviral and antibiotic therapy initiated. Following infection resolution, abrocitinib was reintroduced with excellent response. Long-term antiviral prophylaxis resulted in sustained remission without recurrence at follow-up in July 2025.

Conclusion: AD is a recurrent inflammatory skin disease that impairs quality of life; JAK inhibitors are effective but require careful monitoring and a multidisciplinary approach.

Keywords: Dermatitis, Atopic; Inflammation; JAK inhibitors

CR196

Cerebral Amyloid Angiopathy - Related Inflammation: Case report**Ela Štimac¹; Elena Vujnović¹; Ivona Žuža¹; Ines Strenja^{1, 2}***1 - Faculty of Medicine in Rijeka, Rijeka, Croatia**2 - Department of Neurology, Clinical Hospital Center Rijeka, Rijeka, Croatia*

Introduction: Cerebral amyloid angiopathy (CAA) is a cerebrovascular disorder characterized by an abnormal buildup of β -amyloid ($A\beta$) in the leptomeninges and the smaller- to medium-sized blood vessels of the brain. Cerebral amyloid angiopathy-related inflammation (CAA-RI) is a less common but aggressive manifestation of CAA caused by an autoimmune reaction to the $A\beta$ deposits in the affected vessels. The primary symptoms commonly observed in CAA-RI include headaches, seizures, focal neurological impairments, and a gradual to sudden cognitive decline.

Case report: A 74-year-old patient presented with flashing colorful lights, headaches and cognitive decline with behavioral changes in the last months. During his examination, he was delirious and bilateral papilledema was found. Computerized tomography (CT) showed an irregular hypodense area of white matter, with preserved overlying cortex, indicating cerebral edema. Brain magnetic resonance imaging scans showed multiple oval, focal areas of low signal on susceptibility-weighted imaging, cortical siderosis and vasogenic edema, consistent with CAA-RI. He was treated with 1,0 g intravenous methylprednisolone for five days, then switched to oral therapy with a gradual dose decrease. He was released neurologically intact. A month later he was brought in by emergency services because of aggressive behavior and confusion. Emergent CT showed a resolution of cerebral edema, and his neurological exam was intact, indicating a separate psychiatric issue. He was admitted to psychiatry where symptoms improved with oxazepam and quetiapine. He was transferred for prolonged inpatient psychiatric treatment at the specialized hospital Insula on the island of Rab.

Conclusion: CAA-RI should be suspected in patients with cerebral amyloid angiopathy who present with acute/subacute neurological symptoms. Recognizing this syndrome is important since patients can benefit from treatment with steroids and immunosuppressive therapy.

Keywords: Brain Edema; Cerebral Amyloid Angiopathy; Immunosuppression Therapy

CR197

Sequential Targeted Therapy Achieving Prolonged Disease Control in EGFR-Mutant Metastatic Lung Adenocarcinoma

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Introduction: Epidermal growth factor receptor (EGFR)-mutated non-small cell lung cancer (NSCLC) shows high initial sensitivity to EGFR tyrosine kinase inhibitors (TKIs), followed by acquired molecular resistance. Optimal sequencing of targeted therapies, particularly in patients with central nervous system (CNS) involvement and complex resistance mechanisms, remains a major clinical challenge. We report a longitudinal case demonstrating prolonged disease control through molecularly guided sequential treatment.

Case report: In August 2022, a patient was diagnosed with metastatic right-sided lung adenocarcinoma with pleural effusion and pulmonary, bone, and hepatic metastases. Molecular testing revealed an EGFR exon 19 deletion, with negative anaplastic lymphoma kinase (ALK), ROS proto-oncogene 1 (ROS1), and programmed death-ligand 1 (PD-L1) expression. First-line afatinib achieved marked clinical and radiological response, maintaining disease control for approximately 10 months. In June 2023, progression occurred with cerebellar metastasis and pericardial effusion. Rebiopsy confirmed acquisition of the EGFR T790M mutation, and second-line osimertinib resulted in durable systemic and intracranial disease control without radiotherapy. Subsequent hepatic progression after prolonged osimertinib exposure prompted next-generation sequencing, identifying EGFR exon 19 deletion, T790M, C797S mutation, and EGFR amplification. Based on multidisciplinary tumor board recommendations, treatment with amivantamab combined with chemotherapy, followed by amivantamab and lazertinib, was initiated, leading to radiological regression of pulmonary, hepatic, and brain metastases and sustained Eastern Cooperative Oncology Group (ECOG) performance status 0.

Conclusion: This case underscores the importance of repeated molecular profiling and rational sequencing of targeted therapies in achieving long-term disease control in advanced EGFR-mutant NSCLC, even with CNS involvement and complex resistance patterns.

Keywords: Adenocarcinoma of Lung; Drug Resistance, Neoplasm; Epidermal Growth Factor Receptor; Molecular Targeted Therapy; Non-Small Cell Lung Cancer

CR198

Inflammatory Bowel Disease and Chronic Myeloid Leukemia: Coexistence Highlighting Immune Dysregulation and Targeted Therapy

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Introduction: Inflammatory bowel disease (IBD) is a chronic immune-mediated condition associated with systemic inflammation and an increased risk of malignancy. While links between IBD and certain haematological malignancies have been reported, the relationship with chronic myeloid leukemia (CML) remains incompletely understood. Chronic inflammation and long-term immunosuppressive therapy may contribute to genomic instability and clonal haematopoietic expansion.

Case report: We report a female patient born in 1980, diagnosed with IBD in 2007, initially classified as ulcerative colitis. In 2013, the diagnosis was revised to Crohn's disease based on capsule endoscopy showing active jejunal inflammation and perianal fistulas. She was treated with systemic glucocorticoids and azathioprine, and infliximab was started in October 2023 due to persistent disease activity. At that time, laboratory tests revealed leukocytosis with thrombocytosis, prompting haematological evaluation. In May 2024, CML was diagnosed following bone marrow examination, with polymerase chain reaction confirming the Philadelphia chromosome (*BCR-ABL* p210 transcript). Treatment with imatinib at a dose of 400mg daily was initiated in June 2024. Also in June 2024, the patient underwent colonic resection with a latero-lateral anastomosis for Crohn's-related stenosis at the splenic flexure. However, the postoperative imaging revealed an enterocutaneous fistula. Definitive surgical treatment, including resection of the fistula and previous anastomosis with ileosigmoid reconstruction, was performed in November 2025. Due to development of antibodies against infliximab, ustekinumab was started in December 2025, while imatinib for CML was continued.

Conclusion: This case highlights the coexistence of long-standing IBD and CML and the importance of looking into haematological abnormalities in patients with chronic inflammatory conditions, especially those receiving immunosuppressive therapy. Reporting such cases may help to understand a potential link and supports further research.

Keywords: Crohn Disease; Immunosuppression Therapy; Inflammatory Bowel Diseases; Leukemia, Myelogenous, Chronic, BCR-ABL Positive

CR199

Systemic Juvenile Idiopathic Arthritis: From Inflammatory Phenotype to Diagnosis

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Introduction: Systemic juvenile idiopathic arthritis (sJIA) is a rare autoinflammatory disease characterized by quotidian fever, arthritis, and evanescent rash, with the potential development of life-threatening macrophage activation syndrome (MAS), reported to occur in up to 40% of patients. Due to its nonspecific clinical presentation and significant overlap with infectious, malignant, and other inflammatory conditions, early diagnosis remains challenging. This case emphasizes the importance of immune-oriented diagnostic reasoning in complex inflammatory syndromes.

Case report: We report the case of a 15-year-old male with no significant medical history, admitted for a three-week history of high-grade fever up to 39°C, migratory polyarthritis involving both large and small joints, and a transient macular rash. Between febrile episodes, the patient remained in good general condition. Laboratory evaluation revealed elevated inflammatory markers: C-reactive protein (64.4 mg/L), leukocytosis ($14 \times 10^9/L$), marked hyperferritinemia (ferritin 1909 µg/L), mildly elevated liver enzymes (aspartate aminotransferase 39 U/L, alanine aminotransferase 55 U/L), and increased fibrinogen levels (4.9 g/L). Extensive microbiological investigations were negative, while serological testing suggested prior Epstein-Barr virus infection. Given the combination of prolonged fever, arthritis, rash, and pronounced hyperferritinemia, sJIA with high clinical suspicion of MAS was considered. Therapy was initiated with methylprednisolone 1 mg/kg/day; however, due to clinical worsening, pulse methylprednisolone therapy (1 g/day for three days) was administered. The patient was discharged on tapering methylprednisolone and weekly subcutaneous methotrexate (20 mg), with ongoing follow-up in pediatric rheumatology clinic.

Conclusion: This case highlights the diagnostic challenges of sJIA and emphasizes hyperferritinemia as a key biomarker of immune dysregulation. Integrating clinical features with laboratory immune profiles enables timely risk assessment and supports individualized therapeutic strategies within contemporary precision immune care.

Keywords: Arthritis, Juvenile Idiopathic; Hyperferritinemia; Macrophage Activation Syndrome

CR200

A Case Report of Cerebral Venous Sinus Thrombosis Presenting with Otagia and Headache

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Introduction: Cerebral venous thrombosis is a rare form of cerebrovascular disease, accounting for less than 1% of all strokes, and may occur at any age. It is characterised by thrombus formation within the cerebral veins and dural sinuses. The clinical presentation is variable; patients may complain of headache, unilateral weakness, or may present with epileptic seizures or a comatose state. This case report highlights the importance of early recognition and treatment of this condition.

Case report: In this case report, we present a 60-year-old patient who presented to a general practitioner with a four-day history of headache partially responsive to ibuprofen, accompanied by otalgia and ipsilateral temporal pain. Otoscopic, meningeal, and neurological examinations were unremarkable. Due to the unclear aetiology and poor response to analgesics, the patient was referred to the emergency department. Magnetic resonance imaging of the brain and magnetic resonance venography confirmed thrombosis of the transverse and sigmoid sinuses, the jugular bulb, and the internal jugular vein, in the context of cerebrovascular disease. Anticoagulant therapy with low-molecular-weight heparin was initiated promptly and subsequently transitioned to rivaroxaban. Statin therapy was initiated due to hyperlipidaemia. No underlying cause of the thrombosis was identified, and the patient was discharged in good condition.

Conclusion: Cerebral venous thrombosis is an exceptionally rare presentation of cerebrovascular disease and, as such, it is important to recognise it promptly. Any severe headache that does not fully respond to analgesics and is accompanied by negative meningeal signs should be considered a cerebrovascular event until proven otherwise.

Keywords: Headache; Stroke; Thrombosis

CR201

Tailored Immunotherapy in Refractory Follicular Lymphoma: From Antibodies to CAR-T Cells

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Introduction: Follicular lymphoma is a type of non-Hodgkin lymphoma (NHL) that develops because of uncontrolled B lymphocyte proliferation. Low-grade NHL develops slowly, often without symptoms for years and is usually resistant to therapy. Modern drugs, including cutting-edge monoclonal antibodies, show promising therapeutic results.

Case report: The patient is a 36-year-old male who was admitted to the department of hematology in November 2023. because of lymphadenopathy and hepatosplenomegaly. After detailed examination, a stage IV, bulky, low-grade follicular lymphoma was found with suspected spreading to the bladder. Due to a high-risk disease according to Follicular Lymphoma International Prognostic Index, the patient was started on O-CHOP (obinutuzumab, cyclophosphamide, doxorubicin, vincristine and prednisone) protocol. After one cycle the patient was switched to R-CHOP protocol where obinutuzumab was substituted with rituximab due to a strong infusion reaction. End-of-treatment positron emission tomography/computed tomography in February 2025 showed progressive, metabolically active disease involving lymph nodes above and below the diaphragm, as well as the spleen, liver, and bone marrow. Considering these findings, the patient was started on ESHAP (etoposide, methylprednisolone, cytarabine and cisplatin) protocol in March 2025. After two cycles the chosen therapy was replaced with mosunetuzumab, a bispecific CD20/CD3 antibody, because of clinical evidence of disease progression (pleural effusion and palpable splenomegaly extending 12 centimeters under the left costal margin). After drug application the patient suffered from grade 2 cytokine release syndrome that was successfully managed with tocilizumab, antipyretics and dexamethasone. The patient is now clinically stable and in the process of receiving chimeric antigen receptor T-cell therapy.

Conclusion: This case illustrates the shift toward patient specific immunotherapy in refractory follicular lymphoma, showcasing how a tailored treatment strategy is critical to achieve satisfactory therapeutic outcomes.

Keywords: Antibodies, Monoclonal; Cytokine Release Syndrome; Immunotherapy; Lymphoma, Follicular

CR202

A Perfect Stroke Imitator: Internuclear Ophthalmoplegia caused by Myasthenia Gravis

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Introduction: Internuclear ophthalmoplegia (INO) is an eye movement disorder caused by a lesion of the medial longitudinal fasciculus (MLF) in the brainstem. It is characterized by impaired adduction of the ipsilateral eye with nystagmus of the abducting eye. A similar clinical picture can occur even without an MLF lesion, due to weakness of the extraocular muscles. The most common causes are myasthenia gravis (MG) and Guillain-Barré syndrome, especially the Miller-Fisher variant. This is called pseudo-internuclear ophthalmoplegia. The aim of this case report is to emphasize the importance of considering MG as a possible cause of isolated eye movement abnormalities.

Case report: A 58-year-old man presented with diplopia. Neurological examination revealed bilateral ptosis and bilateral INO, while the rest of the examination was unremarkable. The patient had multiple vascular risk factors; therefore, a lacunar ischemic stroke was initially suspected. Magnetic resonance imaging (MRI) of the brain showed no acute lesions. A prostigmin test was performed, and within 15 minutes there was a significant improvement in ptosis and eye movement. This test indicated myasthenic syndrome; thus, further tests were done. Finally, antibodies to nicotinic acetylcholine receptors confirmed the diagnosis of ocular myasthenia gravis. Therapy with acetylcholinesterase inhibitors and corticosteroids led to the patient's complete recovery.

Conclusion: Isolated ocular disorders, especially in patients with multiple vascular risk factors, can be misleading and suggest a cerebrovascular diagnosis. When a brain MRI does not show acute lesions and other neurological symptoms are absent, other, possibly neuromuscular, diseases should be suspected. In summary, a wide range of differential diagnoses must be considered to ensure appropriate care and treatment.

Keywords: Diplopia; Myasthenia Gravis; Ophthalmoplegia

CR203

Ventricular fibrillation caused by epicardial pacemaker lead fracture in a child with complex congenital heart disease**Karla Trampus¹; Katarina Jelečević¹; Hana Marković¹; Valentina Mirković¹; Krešimir Šantić^{1,2}***1 - Faculty of Medicine, University of Josip Juraj Strossmayer, Osijek, Croatia**2 - Department of Pediatrics, Clinical Hospital Centre Osijek, Osijek, Croatia*

Introduction: Permanent pacing in children with complex congenital heart disease carries a risk of device-related complications. Although epicardial pacing systems are frequently used, lead fracture is rare and may result in life-threatening events. This case highlights epicardial ventricular lead fracture as a potential cause of sudden cardiac arrest in the pediatric population.

Case report: We report the case of a 9-year-old boy with total anomalous pulmonary venous return associated with complete atrioventricular block, complicated by chronic heart failure. The patient had undergone four previous cardiac surgeries, including implantation of an epicardial permanent pacemaker and replacement of a mechanical mitral valve. He collapsed suddenly at school and was found in ventricular fibrillation. Return of spontaneous circulation was achieved through immediate bystander and subsequent professional cardiopulmonary resuscitation with defibrillation. On admission, the patient was unconscious, hypothermic, severely bradycardic, and hemodynamically unstable, with markedly reduced myocardial contractility. Transthoracic echocardiography demonstrated reduced ejection fraction and significant valvular dysfunction. Electrocardiographic monitoring revealed pacemaker spikes without ventricular capture. Chest radiography confirmed fracture of the epicardial ventricular lead, resulting in loss of sensing, non-capture, and critically increased lead impedance. Recurrent ventricular tachyarrhythmias required antiarrhythmic therapy, external pacing, and vasoactive support. The patient was stabilized and transferred to a reference pediatric cardiac center for definitive management, including implantation of a new permanent pacemaker, with temporary ventricular pacing used as a bridging measure.

Conclusion: This case highlights epicardial pacemaker lead fracture as a life-threatening complication that can lead to sudden cardiac arrest in children, underscoring the importance of early recognition and timely management to improve outcomes.

Keywords: Heart Arrest, Radiography, Ventricular Fibrillation

CR204

The "Operation of the Century" as a Definitive Solution for Secondary Hip Osteoarthritis Due to Developmental Dysplasia of the Hip

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Introduction: Developmental dysplasia of the hip (DDH) is the most common congenital disorder of the locomotor system and a leading cause of early-onset secondary hip osteoarthritis (OA). When joint-preserving options have been exhausted, total hip arthroplasty (THA) – famously described as the “operation of the century” by The Lancet – remains the definitive treatment option. The aim of this case report is to highlight the role of THA in managing DDH-related secondary hip OA.

Case report: A 42-year-old woman with a history of DDH treated in infancy with a Pavlik harness presented with severe groin pain, daily analgesic use, and a marked decline in hip function and quality of life. At the time of her birth, routine hip ultrasonography using the Graf method was not yet widely available in Croatia. A positive family history was noted, as her mother had DDH and underwent THA at the age of 40. Radiographic assessment demonstrated a Wiberg angle of 17°, confirming acetabular dysplasia. Due to the limited success of conservative treatment, THA was performed as the definitive intervention. The patient was mobilized on postoperative day one using two forearm crutches, discharged on postoperative day four, and underwent guided physiotherapy to support recovery. At the three-month follow-up, she reported high satisfaction, walked without crutches or gait abnormalities, and had a pain-free hip with a normal range of motion.

Conclusion: This case emphasizes THA as a successful treatment for secondary OA due to DDH. By effectively relieving chronic pain and restoring full mobility, this procedure remarkably improves the patient's quality of life. Furthermore, this case demonstrates that optimal outcomes rely on appropriate postoperative care and individualized rehabilitation.

Keywords: Arthroplasty, Replacement, Hip; Congenital Abnormalities; Developmental Dysplasia of the Hip; Osteoarthritis

CR205

Syndrome of the Trephined: a Rare Decompressive Craniectomy Complication Following an Intracranial Arteriovenous Malformation Rupture – A Case Report

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Introduction: Syndrome of the trephined, also known as the sinking skin flap syndrome, is a delayed neurological deterioration after craniectomy, caused by an atmospheric pressure exerted on the unprotected cerebral parenchyma. The condition is potentially reversible following timely cranioplasty.

Case report: This case describes a 45-year-old woman who developed progressive neurological deficits following emergency left frontotemporoparietal decompressive craniectomy for a rupture of an arteriovenous malformation (AVM). The feeding artery was embolized and the AVM subsequently resected. Postoperative magnetic resonance imaging (MRI) demonstrated areas of encephalomalacia with gliosis within the presumed eloquent and motor regions of the re-expanding left hemisphere, correlating with right sided hemiparesis and verbal unresponsiveness. Periventricular T1-weighted hypointensity suggested hypertensive hydrocephalus, prompting ventriculoperitoneal shunt (VPS) placement. Postoperative recovery was complicated by an occipital pressure ulcer, thereby preventing timely cranioplasty. After healing, autologous bone flap was reimplanted. However, re-craniectomy was shortly required due to epidural hematoma. One month later, patient deteriorated to a stuporous state with decorticate posturing and marked skin-flap sinking. Emergency computed tomography demonstrated parenchymal compression with 11 mm midline shift, confirming syndrome of the trephined. Urgent cranioplasty using customized polymethylmethacrylate implant was performed. Follow-up MRI demonstrated parenchymal re-expansion corresponding with neurological improvement, but contrast enhancement warned of a developing epidural empyema. Repeat craniectomy was required, during which the VPS was removed and replaced with external ventricular drainage, which was removed after hydrocephalus resolved. At age 45, she was transferred to nursing facility for adequate 24-hour palliative support, while further therapeutic options are evaluated.

Conclusion: This case illustrates the manifestation of the syndrome of the trephined, where timely cranioplasty remains the treatment of choice; however, comorbidities and postoperative complications increase the risk of suboptimal outcome.

Keywords: Arteriovenous Malformations; Decompressive Craniectomy; Epidural Abscess; Intracranial Pressure; Postoperative Complications

CR206

Ovarian Hyperandrogenism - A Potential New Feature of Smith-Magenis Syndrome

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Introduction: Primary amenorrhea with progressive virilization in adolescent girls is rare and alarming. Differential diagnosis includes androgen-secreting ovarian or adrenal tumors, adrenal steroidogenesis defects, or, rarely, polycystic ovary syndrome (PCOS). Smith-Magenis syndrome (SMS) is a rare genetic disorder characterized by developmental delays, dysmorphic facial features, behavioral abnormalities and congenital anomalies.

Case Report: We report a 14-year-old girl with SMS (*RAI1*-c.1228C>T) referred for primary amenorrhea and progressive virilization. Puberty began at age 11 with pubarche preceding thelarche and no menarche. Clinical examination revealed breast development (Tanner IV), pubic and axillary hair (Tanner V), severe hirsutism (Ferriman-Gallwey score 22), and clitoromegaly. Laboratory tests showed markedly elevated testosterone (10 nmol/L; n.v. 0.1-1.7), androstenedione (19 nmol/L; n.v. 1.3-7), and inhibin B (186 ng/L; n.v. <83), with normal Dehydroepiandrosterone sulfate, estradiol and gonadotropins. Pelvic magnetic resonance imaging revealed bilaterally enlarged ovaries with polycystic morphology and stromal hypertrophy without a defined mass. Exploratory laparoscopy with biopsy confirmed ovarian hyperthecosis. Therefore, to suppress ovarian androgens, gonadotropin-releasing hormone analogue was introduced, but with only partial response. Given the ovarian size, limited medical response, and absent fertility potential due to primary disease, bilateral salpingo-oophorectomy was performed. Androgen levels normalized promptly postoperatively, and estrogen replacement therapy was initiated.

Conclusion: To our knowledge, this is the first reported patient with SMS who presented with severe hyperandrogenemia and progressive virilization due to underlying ovarian hyperthecosis, a condition within the PCOS spectrum. Only one previous case of PCOS in SMS has been documented, but without such a severe clinical presentation. The pathophysiological link between *RAI1* mutations and PCOS/hyperthecosis remains unknown. This report expands the phenotypic spectrum of SMS and suggests that severe ovarian hyperandrogenism could be considered a potential feature of this syndrome.

Keywords: Hirsutism; Hyperandrogenism; Polycystic Ovary Syndrome; Smith-Magenis syndrome; Virilism

CR207

Secondary Hemophagocytic Lymphohistiocytosis Associated with a Heterozygous Perforin Variant: A Case Report

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Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a hyperinflammatory condition that results in increased phagocytic activity of natural killer cells and CD8+ cytotoxic cells. These cells produce cytolytic enzymes like perforin that destroy hematopoietic cells. Secondary HLH presents in adulthood with cytopenias, fever, hypertriglyceridemia and splenomegaly.

Case report: A 31-year-old woman with a medical history of multiple sclerosis and prior tuberculous cervical lymphadenopathy presented to the emergency department with a high fever. Laboratory findings revealed significant thrombocytopenia ($46 \times 10^9 /L$), leukopenia ($1.13 \times 10^9 /L$), normal hemoglobin and an elevated C-reactive protein (28 mg/L). Tryglicerides and ferritin (1479 $\mu g/L$) were markedly elevated. Computed tomography imaging revealed hepatosplenomegaly and para- aortocaval lymphadenopathy. Despite antibiotic and corticosteroid therapy, leukopenia and thrombocytopenia persisted. A bone marrow biopsy revealed abundant hematopoiesis with clear signs of hemophagocytosis, leading to a diagnosis of secondary HLH. Genetic testing identified a heterozygous variant of PRF1 K285D – an in-frame deletion in the *PRF1* gene, which is typically associated with HLH. Treatment was initiated with etoposide, pulse-dose corticosteroids and cyclosporine. Although ferritin and triglyceride levels improved, the patient developed acute kidney failure due to cyclosporine, causing discontinuation of the drug. Over two years, the patient's condition fluctuated, requiring massive transfusion support - 117 units of platelets, 6 units of erythrocytes. An allogeneic hematopoietic stem cell transplant (HSCT) was planned. Her asymptomatic brother was initially considered as a donor but carried the same *PRF1* variant, disqualifying him. A matched unrelated donor was used. The transplant was successful, and the patient achieved remission.

Conclusion: This case underlines the complexity of HLH triggered by underlying genetic predispositions. It highlights the critical importance of an individualized approach to diagnosis and the necessity of genetic screening of potential family donors.

Keywords: Hematopoietic Stem Cell Transplantation; Hemophagocytic; Lymphohistiocytosis; Remission Induction

CR208

Tailoring Therapy Starts with Tailoring the Diagnosis: POEMS Syndrome

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Introduction: Polyneuropathy, organomegaly, endocrinopathy, monoclonal plasma cell disorder and skin changes (POEMS) syndrome is a rare paraneoplastic disorder. It is difficult to diagnose because its symptoms can vary and resemble many other diseases.

Case report: A 60-year-old woman with hypothyroidism presented with paresthesias and pain in both lower legs beginning in November 2018. She was hospitalized and treated with plasmapheresis under the diagnosis of acute polyradiculoneuritis. Due to persistent symptoms pulse corticosteroid therapy was initiated in November 2019 and the condition was subsequently managed as chronic inflammatory demyelinating polyneuropathy (CIDP). Intravenous immunoglobulin therapy administered in June 2020 showed no significant benefit. During rehospitalization between September and October 2020 electromyography confirmed progression of CIDP. MSCT (multi-slice computed tomography) revealed ascites, pleural and pericardial effusions and splenomegaly. Spinal MRI (magnetic resonance imaging) demonstrated a cystic lesion of the L1 vertebral body. Cerebrospinal fluid analysis showed elevated protein levels. A small M-spike (monoclonal protein band) was found on serum protein electrophoresis. Ophthalmologic examination revealed bilateral papilledema with partial corticosteroid response. Hematological evaluation prompted by suspected POEMS syndrome led to initiation of RVD therapy (bortezomib, lenalidomide, dexamethasone) per hematology council decision in November 2021. After seven RVD cycles, in February 2022, bone marrow immunophenotyping revealed no plasma cell proliferation confirming suitability for peripheral blood stem cell collection. Autologous stem cell transplantation was indicated due to good response to induction and POEMS diagnostic criteria, enabling remission in eligible patients. The patient remains clinically stable on lenalidomide maintenance therapy.

Conclusion: This case illustrates how clinical/laboratory overlap with other diseases delays POEMS diagnosis and treatment. Timely multidisciplinary recognition enables accurate diagnosis, targeted therapy and better outcomes, confirming that adapting therapy begins with adapting the diagnosis.

Keywords: Hematology; Papilledema; Polyneuropathies; Skin; Syndrome

CR209

Fetal Intracranial Hemorrhage Associated with Suspected Neonatal Alloimmune Thrombocytopenia: A Fatal Perinatal Case

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Introduction: Neonatal alloimmune thrombocytopenia (NAIT) is recognized as the most common cause of spontaneous fetal intracranial hemorrhage (ICH) in otherwise uncomplicated pregnancies. However, antenatal diagnosis remains challenging due to nonspecific clinical signs and limited availability of confirmatory testing. The aim of this case report is to highlight the diagnostic complexity and poor prognosis of massive fetal ICH potentially related to immune-mediated thrombocytopenia.

Case report: A multiparous woman with two previous uncomplicated pregnancies presented in the third trimester with significantly reduced fetal movements over a two-day period. Cardiotocography (CTG) demonstrated narrowed variability with a baseline fetal heart rate of 145 bpm. Pregnancy ultrasound revealed severe macrocephaly with a head circumference of 40.9 cm. Fetal neurosonography demonstrated a large heterogeneous lesion occupying the right cerebral hemisphere, consistent with massive intracranial hemorrhage, accompanied by a pronounced midline shift and compression of the falx cerebri. The right lateral ventricle and midline structures were not visualized, while the left lateral ventricle was dilated. Posterior fossa structures were displaced caudally, and hepatosplenomegaly was present. Maternal laboratory evaluation revealed anemia, leukocytosis, preserved coagulation parameters, and significantly elevated D-dimer levels. In the absence of maternal trauma, infection, hypertensive disease, or known coagulopathy, NAIT was considered the leading etiological hypothesis, and parental human platelet antigen genotyping results are pending. Due to non-reassuring CTG findings and extremely poor neurological prognosis, an emergency cesarean section was performed. A live, preterm female neonate was delivered with profound neurological depression and died on the second postnatal day despite intensive care.

Conclusion: This case illustrates a catastrophic presentation of fetal ICH associated with NAIT and emphasizes the importance of considering immune-mediated thrombocytopenia in cases of unexplained fetal cerebral hemorrhage.

Keywords: Antigens; Fetus; Intracranial Hemorrhages; Newborn; Thrombocytopenia

CR210

Effects of 2-week low dietary salt intake in a female patient with psoriasis: a case report

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Introduction: Psoriasis is a chronic, immune-mediated inflammatory skin disease, characterized by erythematous-squamous plaques. Psoriasis represents a model of IL-23–Th17-mediated inflammation. In recent years, evidence has emerged that salt promotes Th17 differentiation while impairing regulatory T cell function, potentially aggravating inflammatory diseases. Therefore, salt restriction may induce immunological changes relevant to psoriasis pathogenesis.

Case report: A 67-year-old female patient presented to the dermatology department in October 2021 with a three-year history of unrecognized psoriasis. Physical examination revealed erythematous-squamous lesions involving scalp, trunk and extremities, with a Psoriasis Area and Severity Index (PASI) score of 11.2. The patient was enrolled in a study investigating the effects of low-salt dietary intake in psoriasis and followed a 14-day low-salt diet. Salt intake was reduced for 14%, as assessed by the change in 24-h urine sodium excretion. Body weight decreased by 2 kg compared to baseline. No topical or systemic therapy was used two weeks prior, nor during the study. After two weeks, laboratory tests showed a 50% reduction in the frequency of Th17 cells determined by flow cytometry analysis of peripheral blood mononuclear cells as IL17+RORγt+ cells (0.18% vs. 0.9%) and a decrease in serum IL-23 concentration (70 vs. 52 pg/mL), along with clinical improvement of the disease, reflected by a 43% reduction in the PASI score (11.2 vs. 6.4).

Conclusion: This case report demonstrates that a short-term isolated low-salt diet can induce measurable immunological changes, including reduced Th17 frequency and lower IL-23 levels, alongside clinical improvement of psoriasis. However, due to the single-patient design, short follow-up, and limited control of confounding factors, the findings should be confirmed in larger studies.

Keywords: Diet, Sodium-Restricted; Psoriasis; Th17 Cells

CR211

From Autoimmunity to Spirochetes: Chronic Lyme Disease Presenting with Scleroderma-Like Skin Changes

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Introduction: Acrodermatitis chronica atrophicans (ACA) is an uncommon late cutaneous manifestation of Lyme borreliosis that typically affects the lower limbs and may mimic scleroderma-like dermatoses. It is rarely recognised and reported in Croatia. We report ACA in a 60-year-old woman with CREST syndrome (calcinosis, Raynaud's phenomenon, oesophageal dysmotility, sclerodactyly, telangiectasia).

Case report: A 60-year-old woman with established CREST syndrome, long-standing Raynaud's phenomenon, and severe digital vasculopathy reported a tick bite followed by erythema migrans on the upper arm in 2021, treated with azithromycin 500 mg once daily for three days. Four years later, she developed progressive reddish-livid discolouration and swelling of the lower legs and feet, with skin tightness, burning pain, and paraesthesia. Additional features included ankle arthralgia and lower-limb myalgia, as well as early cutaneous wrinkling and atrophy, prompting suspicion of cutaneous sclerosis. Lyme serology demonstrated both immunoglobulin M (IgM) and immunoglobulin G (IgG) positivity. A punch biopsy from the affected lower leg excluded scleroderma and supported cutaneous borreliosis consistent with ACA. Doxycycline 100 mg twice daily for 28 days led to regression of oedema and discolouration and improvement of neuropathic and musculoskeletal symptoms. Follow-up serology showed IgM seroreversion with persistently positive IgG, as expected after treatment.

Conclusion: ACA is an under-recognised late manifestation of Lyme borreliosis in Croatia and may mimic scleroderma-like disease, even in patients with established CREST syndrome. A focused exposure history and early skin biopsy support timely anti-borrelial therapy and limit diagnostic anchoring on autoimmunity. Further surveillance and multicentre reporting are needed to clarify local burden and outcomes.

Keywords: Borrelia Infections; Post-Lyme Disease Syndrome; Scleroderma, Localized

CR212

Early-Onset Homozygous Familial Hypercholesterolemia Requiring Long-Term LDL Apheresis from Childhood

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Introduction: Homozygous familial hypercholesterolemia (HoFH) is a rare genetic disorder characterized by abnormally high levels of low-density lipoprotein (LDL) cholesterol. Without treatment, this life-threatening condition leads to premature cardiovascular disease. Despite intensive pharmacological therapy, patients with a severe form of inherited hypercholesterolemia may require LDL apheresis to achieve adequate lipid control. Early diagnosis, long-term follow-up, and a multimodal approach are key to optimal management.

Case report: Since early childhood, a 24-year-old male patient with HoFH, and a null mutation confirmed by genetic testing resulting in complete LDL receptor deficiency, has been treated with intensive lipid-lowering medications, including rosuvastatin (40 mg), ezetimibe (10 mg), and strict dietary measures, combined with weekly LDL apheresis. Monitoring included lipid profile, liver enzymes, and glucose levels before and after each apheresis session. Initial cardiovascular evaluation indicated mild (grade I) aortic and mitral regurgitation, which later progressed to moderate-to-severe (grade II–III), accompanied by concentric left ventricular hypertrophy with preserved systolic function. Carotid Doppler imaging showed concentric fibrous atherosclerotic plaques without hemodynamically significant stenosis. Follow-up examination revealed progression to cardiac remodeling, prompting the introduction of ramipril (1.25 mg). Following its inclusion on the list of very expensive medications, treatment was supplemented with alirocumab, a proprotein convertase subtilisin/kexin type 9 inhibitor (300 mg monthly), further to optimize lipid levels in combination with ongoing apheresis. Given the severity of the disease, lomitapide and evinacumab are considered as potential treatment options pending national reimbursement approval.

Conclusion: This case report underscores the clinical complexity of HoFH and the progression of cardiovascular complications. Combining early and sustained LDL apheresis with novel anticholesterol therapy is essential for managing high-risk patients and addressing cardiovascular risk in young adults.

Keywords: Cholesterol, LDL; Heart Disease Risk Factors; Hypercholesterolemia

CR213

When Infection Mimics Malignancy: *Fusobacterium nucleatum* Brain Abscesses Presenting as Suspected Lung Cancer with Brain Metastases

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Introduction: Multiple brain lesions with acute focal neurological deficits often suggest malignancy, especially in patients with systemic symptoms and cancer risk factors. However, infections can mimic metastatic disease clinically and radiologically. *Fusobacterium nucleatum*, an oral anaerobe, is increasingly recognized as a rare cause of brain abscesses, often without evident dental infection in immunocompetent patients.

Case report: We report a 44-year-old male with hypertension, tobacco use and recent weight loss who presented with abdominal pain, fever, night sweats and shivering. Ultrasound revealed an anterior abdominal wall fluid collection, therefore oral amoxicillin was prescribed. Twelve days later, he developed right-sided weakness and speech difficulty. Examination revealed diffusely decreased breath sounds. Chest imaging showed a right lung mass suspicious for primary lung cancer, while brain computed tomography revealed multiple ring-enhancing brain lesions with oedema and subfalcine herniation, prompting admission for suspected brain metastases. Subsequent magnetic resonance imaging suggested multiple cerebral and cerebellar abscesses. He experienced his first seizure, and antiepileptic therapy was initiated. Despite empiric broad-spectrum antibiotic and antifungal therapy, brain imaging showed progression, requiring urgent neurosurgical decompression, while chest radiograph demonstrated infiltrate regression. Microbiological analysis of intraoperative aspirate identified *Fusobacterium nucleatum* by 16S rRNA sequencing, while histopathology excluded malignancy. Targeted antimicrobial therapy was started, followed by transfer to a tertiary infectious diseases centre. One year later, successful cranioplasty with an autologous bone graft was performed. Postoperatively, infection developed, necessitating bone flap removal. He recovered with residual supranuclear facial paresis, mild motor dysphasia, and severe right-sided spastic hemiparesis.

Conclusion: This case highlights the critical role of comprehensive diagnostic evaluation and a coordinated multidisciplinary approach in managing complex brain abscesses, enabling timely intervention, targeted therapy, and optimal neurological recovery.

Keywords: 16S rRNA; Brain Abscess; Brain Metastases; Dysphasia; *Fusobacterium nucleatum*

CR214

Treatment of a Perforating Corneal Injury by Amniotic Membrane Transplantation

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Introduction: Infectious keratitis is a rapidly progressive, vision-threatening condition and constitutes an ophthalmic emergency. *Pseudomonas aeruginosa* is among the most aggressive bacterial pathogens, capable of causing corneal perforation within 24–48 hours if untreated. Corneal perforation represents a serious complication requiring prompt intervention. The aim of this abstract is to present the timely management of a rapidly progressing pseudomonal infection using amniotic membrane transplantation and to highlight its role in preserving vision.

Case report: A 70-year-old man was admitted to the ophthalmology ward with decreased vision, photophobia, and pain in the left eye, lasting for three days. On examination, visual acuity was significantly reduced; the patient could perceive only hand movements, but not fingers, letters, or shapes. Slit-lamp examination revealed a corneal ulcer with a white-yellow infiltrate. The Seidel test was positive, indicating perforation or leakage of aqueous humor. Material from the ulcer was collected for culture and antibiogram to identify the causative organism. Urgent treatment was initiated, including empirical therapy followed by targeted therapy according to the results of the antibiogram. Culture confirmed *Pseudomonas aeruginosa*. Topical 0.5% moxifloxacin was administered every hour as part of therapy. The corneal perforation was closed with amniotic membrane transplantation. A multilayered amniotic graft was used and sutured with monofilament sutures. At the follow-up examination after one month, the amniotic membrane had almost completely resorbed, and after two months, visual acuity was 0.2. The ulcer had completely healed.

Conclusion: Corneal perforation is an urgent ophthalmic condition. The exceptional biological properties of the amniotic membrane make it an excellent choice for the treatment of corneal perforation and for preventing severe complications such as blindness.

Keywords: Cornea; Cornea Ulcer; Eye Pain; Infection; Visual Acuity

CR215

Incidental Familial Discovery of Fabry Disease: Cascade Screening, Multisystemic Manifestations, and Enzyme Replacement Therapy Switch Due to Hypersensitivity

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Introduction: Fabry disease (FD) is an X-linked lysosomal storage disorder caused by pathogenic variants in the GLA gene, resulting in alpha-galactosidase A deficiency and the accumulation of globotriaosylceramide, which is associated with an increased risk of early cerebrovascular events, myocardial infarction, neurologic damage, and progressive renal disease that may advance to end-stage renal failure requiring dialysis. The multisystem nature of the disease often delays diagnosis, particularly in non-classical phenotypes.

Case report: A 45-year-old male underwent family-based genetic screening following his mother's incidental diagnosis of Fabry disease during evaluation for an acute viral illness, when chest imaging revealed cardiomegaly and genetic testing identified a pathogenic GLA variant. Prominent clinical manifestations of Fabry disease include lifelong acroparesthesias, anhidrosis, genital angiokeratomas, and cornea verticillata. In this patient, echocardiography confirmed concentric left ventricular hypertrophy consistent with FD cardiomyopathy. Enzyme assay demonstrated profoundly reduced alpha-galactosidase A activity (2 nmol/mg protein/h). Agalsidase beta (Fabrazyme) was initiated but discontinued after eleven infusions due to hypersensitivity reactions (urticaria, flushing, and dizziness). Therapy was subsequently switched to pegunigalsidase alfa (Elfabrio), which he tolerates well, with clinical stabilization and a reported reduction in acroparesthesias. Additional manifestations of the disease include mild sensorineural hearing loss and small fiber neuropathy causing distal lower limb paresthesias, which prevent barefoot walking.

Conclusion: This case underscores the importance of cascade genetic screening in identifying Fabry disease in at-risk relatives. Early diagnosis, based on the detection of a pathogenic GLA variant, reduced enzyme activity, and evidence of target organ involvement, is crucial for the timely initiation of therapy and may help prevent irreversible cardiac, renal, and neurologic complications.

Keywords: Enzyme Replacement Therapy; Fabry Disease; Genetic Testing; Hypersensitivity

CR216

Cat got your eyesight? – A case report of neuroretinitis caused by cat scratch disease

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Introduction: Cat scratch disease (CSD), caused by *Bartonella henselae*, typically causes skin lesions and self-limiting lymphadenopathy. This case describes CSD neuroretinitis, which can present before characteristic signs, such as the macular star, become apparent.

Case Report: A 26-year-old male presented to the ophthalmology ward with painful left ocular motility and a five-day-old scotoma. Initial ophthalmological examinations revealed neuroretinitis and vitritis of unknown etiology. Blood work showed a mildly elevated C-reactive protein value (43.8 mg/L). Serology, physical examinations, and brain/orbit computed tomography scans were unremarkable. Empiric therapy with azithromycin, trimethoprim/sulfamethoxazole, and ciprofloxacin, as well as potent corticosteroids, resulted in no clinical improvement. After one week, a neurologist noted only a decreased pupillary reaction to light and a persistent central visual field impairment; patient's scotoma remained present. Repeat blood work, lumbar puncture, and magnetic resonance imaging (MRI) of the brain and cervical spine, and MR angiography were unremarkable. During a subsequent fundoscopic examination, a macular star was discovered, resulting from lipid exudation in the macula and optic disc edema. Upon thorough epidemiologic history, the patient revealed frequent contact with a four-month-old kitten. Immunofluorescent assays showed a positive IgM and an IgG titer >1:512 against *Bartonella henselae*. The patient was treated for 6 weeks with doxycycline, rifampin, and systemic corticosteroids. After treatment, the scotoma persisted for approximately one year, and the patient's visual acuity recovered over the same period.

Conclusion: CSD can be an unlikely cause of ocular pathology. Efficient diagnosis relies on detailed epidemiological histories and continuous monitoring for late-appearing signs of the disease, like the macular star.

Keywords: Bartonella henselae; Cat-Scratch Disease; Infectious Diseases; Neuroretinitis; Scotoma

CR217

Crohn's Disease or Behçet's Disease? A Case of Multiple Liver Abscesses and Recurrent Aphthous Stomatitis.**Friedrich von Hoffmeister¹ ; Dorian Österreicher² ; Helena Grbavac³ ; Petra Čačić⁴ ; Josip Grbavac^{1,5}***1 - Medical faculty of Osijek, University of Josip Juraj Strossmayer Osijek**2 - Department of Neurology, Clinical medical center Osijek**3 - Community Health Centre, Osijek-Baranja County, Osijek**4 - Department of Gastroenterology and Hepatology, University Hospital Centre Sisters of Charity, Zagreb**5 - Department of Gastroenterology, General hospital Vinkovci, Croatia*

Introduction: Behçet's disease is a rare, severe and chronic multi-systemic vasculitis. In this case we present an initially perplexing case of Behçet's disease appearing with gastrointestinal symptoms mimicking irritable bowel disease, sepsis-like symptoms as well as liver abscesses making an initial diagnosis difficult.

Case Report: A 19-year-old man presented with a one-month history of watery diarrhea occurring 2-3 times daily, with nighttime fever of up to 39.3°C, significant weight loss of up to 9 kg and conjunctivitis in both eyes. Initial imaging revealed multiple liver lesions suggestive of pyogenic abscesses, accompanied by elevated C-reactive protein and cholestatic enzymes. Due to the clinical triad of chronic diarrhea, weight loss, and elevated fecal calprotectin, the primary working diagnosis was irritable bowel disease. However, extensive endoscopic and histological evaluation ruled out classic irritable bowel disease phenotypes. Macroscopically, discrete aphthous ulcers were found in the colon and esophagus. Histological analysis showed only nonspecific inflammation. With a notable absence of granulomas or crypt architectural distortion required for irritable bowel disease. On antibiotic therapy clinical remission was achieved. However, the patient experienced a severe relapse five months later, manifesting with worsening oral/ gastrointestinal ulcers/aphthae and arthralgia. Re-evaluation classified the liver lesions as aseptic abscesses associated with vasculitis. Diagnosis of Behçet's disease was established based on the International Criteria for Behçet's Disease (ICBD). Switching from antibiotics to systemic corticosteroids resulted in rapid remission. Therapy with TNF inhibitors is planned after further consultation.

Conclusion: This case illustrates the capacity of Behçet's disease to present with hepatic lesions that indistinguishably mimic bacterial abscesses. It highlights that elevated fecal calprotectin, and gastrointestinal symptoms lack specificity and mandate rigorous histopathological evaluation to exclude irritable bowel disease.

Keywords: Behcet Syndrome; Crohn Disease; Liver Abscess

CR218

Complex Multimodal Treatment of Multiple Malignancies in a Young Patient management

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Introduction: Immunotherapy represents a modern therapeutic approach in oncology, particularly in the treatment of biologically aggressive tumors and those for which standard therapeutic options are insufficiently effective. Despite high efficacy, immunotherapy may be associated with serious immune mediated adverse events.

Case report: A 31 year old female patient was treated in 2011 for primary carcinoma of the right breast. She underwent right breast quadrantectomy followed by adjuvant chemotherapy consisting of six cycles according to the fluorouracil epirubicin cyclophosphamide protocol and radiotherapy (RT) with a total dose(TD) of 50 Gy. In 2021, a pigmented skin lesion on the right breast was excised, and histopathological analysis confirmed cutaneous melanoma stage pT2b, without indication for systemic therapy. During follow up, breast ultrasound revealed a hypoechoic lesion in the left breast. Core needle biopsy confirmed triple negative carcinoma, and neoadjuvant chemotherapy was initiated with four cycles of the doxorubicin cyclophosphamide protocol followed by twelve cycles of paclitaxel. Due to positive family history, the multidisciplinary team recommended bilateral mastectomy. Adjuvant RT was administered with a TD of 43.2 Gy and a boost dose of 12 Gy, followed by six cycles of capecitabine. In 2023, a bluish firm skin lesion was observed on the left breast. Excisional biopsy confirmed metastatic melanoma. Dual immunotherapy with nivolumab and ipilimumab was initiated. Despite favorable response, treatment was discontinued due to severe immune mediated colitis with acute pancreatitis. As a BRAF mutation was detected, targeted therapy with BRAF and MEK inhibitors was initiated, consisting of dabrafenib 75 mg and trametinib 2 mg.

Conclusion: This case highlights the complexity of treating a young patient with multiple malignancies, requiring individualized approach.

Keywords: Breast Neoplasms; Immunotherapy; Metastatic Melanoma

CR219

Fibrillary glomerulonephritis successfully treated with rituximab**Antun Vrdoljak¹; Klara Zima¹; Filip Vlašić¹; Nikola Zagorec²; Ivica Horvatić^{1,2}***1 - School of Medicine Zagreb, University of Zagreb, Zagreb, Croatia**2 - Department of Nephrology and Dialysis, University Hospital Centre Zagreb, Zagreb, Croatia*

Introduction: Fibrillary glomerulonephritis (FGN) is a remarkably rare type of glomerulonephritis characterized by deposition of fibrillary material within glomeruli with DNAJB9 being the most abundant component. There are no specific recommendations on how FGN should be treated and prognosis remains poor. Here, we report a case of FGN successfully treated with rituximab.

Case report: A 72-year-old female, with a history of arterial hypertension, presented with lower limb edema and blood pressure dysregulation of several months duration. Clinical workup showed normocytic anemia with hemoglobin measuring 98 g/L, renal insufficiency with serum creatinine up to 298 $\mu\text{mol/L}$ and urea 36.5 mmol/L, nephrotic range proteinuria of 8 g/day and dysmorphic erythrocyturia. Additional screening for malignancy, monoclonal gammopathies, infections and immunological diseases was unremarkable. Kidney biopsy was performed due to nephrotic-nephritic syndrome revealing mesangial expansion due to deposition of periodic acid-Schiff positive and Kongo negative material being positive on immunohistochemistry for DNAJB9. Presence of randomly oriented fibrils, measuring 8-15 nm in diameter, average 13 nm in mesangial space and glomerular basement membranes, has been confirmed by electron microscopy. The patient was treated with methylprednisolone and rituximab in dosage of 2x1000 mg two weeks apart. Six months later, stabilization of kidney function with serum creatinine around 200 $\mu\text{mol/L}$ and significant reduction of proteinuria measuring < 1g/day was achieved.

Conclusion: FGN is a rare glomerular disease whose etiology is unclear as well as treatment. A thorough workup is necessary in order to exclude other underlying conditions such as malignancies and paraproteinemia. DNAJB9 analysis is crucial for diagnosis of FGN. B cell depletion achieved by rituximab was beneficial in our case. Clinical trials are needed in future to define treatment of FGN.

Keywords: Glomerulonephritis, Heat-Shock Proteins, Nephrotic Syndrome, Rituximab

CR220

Improved growth and quality of life following vosoritide therapy in a patient with achondroplasia

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Introduction: Achondroplasia is the most common genetic disorder causing disproportionate dwarfism. The cause is an *FGFR3* gene mutation, which encodes the protein crucial for bone tissue development. The typical phenotype includes an average-sized trunk, rhizomelic limb shortening, and an average height of 131 cm. These patients often have issues with hypotonia and spinal canal stenosis, leading to cord compression and chronic pain. Vosoritide is a modified C-type natriuretic peptide analog that stimulates chondrocyte proliferation and differentiation, leading to bone growth. It can be administered from 4 months of age until epiphyseal closure.

Case report: We present a case of a 14-year-old boy with achondroplasia confirmed with genetic testing. His medical history included ventriculomegaly, thoracolumbar kyphosis, hypoacusis, and spinal canal stenosis. At 12 years of age, the patient's height was 122.1 cm standing and 80.6 cm sitting, and his arm span was 107.6 cm. After educating the parents, the first dose was administered, and regular checkups scheduled every three months. He reported no adverse effects, and 22 months after starting therapy, achieved significant growth of 14.5 cm while standing, 7.5 cm while sitting, and 13.3 cm in arm span. He reported feeling well with ability to walk and run for longer without any leg or back pain. He also had more muscle strength, started playing football, and riding the bike without the need for accommodations. Due to good tolerance and therapy effects, therapy was continued.

Conclusion: This case demonstrates the benefits of vosoritide therapy in not only promoting growth but also improving the quality of life for the patient, who was able to enjoy usual childhood activities.

Keywords: Achondroplasia; Adolescent; Receptor, Fibroblast Growth Factor, Type 3; Quality of Life

CR221

The role of pharmacogenetics in clinical therapy assessment

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Introduction: Pharmacogenetic testing enables better understanding of interindividual variability in drug metabolism, potentially explaining reduced efficacy or increased risk of adverse effects. This case report emphasizes the clinical relevance of *CYP2D6* variability in optimizing tamoxifen therapy and highlights the importance of individualized, multidisciplinary therapeutic decision-making.

Case report: The patient, born in 1970, was referred by her psychiatrist to a clinical pharmacologist due to the ineffectiveness of antidepressants. In January 2024, she underwent surgery for right breast cancer (pT1b, pN0, luminal subtype, human epidermal growth factor receptor 2 (HER2)-negative), followed by radiotherapy. Since February 14, 2024, she has been receiving adjuvant antihormonal therapy with tamoxifen 10 mg orally twice daily. Her regular therapy includes mirtazapine, pregabalin, diazepam as needed, tamoxifen, methotrexate with folic acid, non-steroidal anti-inflammatory drugs as needed, esomeprazole. A clinically relevant interaction between mirtazapine and tramadol was observed, prompting recommendations to rationalize analgesic therapy and referral for pharmacogenetic testing. Pharmacogenetic testing showed an unpredictable *CYP2D6* phenotype (*CYP2D6* *3,*4,*6,*9,*10,*41: *1/*4; *CYP2D6* *5 (del), xN (CNV in2, in6, ex9) x3), making accurate phenotype prediction impossible. The findings suggest potentially reduced conversion of tamoxifen to its active metabolite, endoxifen, which could decrease therapeutic efficacy. Recommendations included avoiding *CYP2D6* inhibitors and considering alternative endocrine therapy or increasing the tamoxifen dose to 40 mg orally once daily. At follow-up, continuation of mirtazapine was advised and oncological reevaluation was recommended. Given her good general condition, with an Eastern Cooperative Oncology Group (ECOG) performance status of 0, the oncologist recommended continuation of tamoxifen 10 mg orally twice daily.

Conclusion: This case highlights the clinical relevance of pharmacogenetics in therapy optimization, while emphasizing that treatment decisions should remain individualized, multidisciplinary, and guided by ongoing clinical monitoring.

Keywords: Breast Neoplasms; Cytochrome P-450 CYP2D6; Pharmacogenetics; Tamoxifen

CR222

When Midazolam Lasts Too Long: A Case Report of Prolonged Sedation After Liver Resection

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Introduction: Benzodiazepines (BZDs) are sedative-hypnotic agents that have been in clinical use for a long time. BZDs, usually midazolam, are administered during the perioperative period because they have anxiolytic properties and are believed to improve the patient experience. When using midazolam, no long-term effects are expected.

Case report: A 76-year-old male with liver cirrhosis underwent a scheduled liver resection indicated by hepatocellular carcinoma and was given midazolam 7,5 mg per os before the surgery. After surgery, he was transferred to the intensive care unit (ICU), intubated, sedated, and receiving analgesics. Computed tomography was done to explore possible haemorrhagic or ischemic surgical complications, and both were negative. Only a small part of the remaining right liver lobe was found to be ischemic. On the third postoperative day, sedation was discontinued, and the patient regained consciousness in the evening hours. The day after, he became somnolent, was given flumazenil and naloxone, and became awake. On the fifth postoperative day, the patient became comatose and was given flumazenil in bolus; a continuous flumazenil IV drip was introduced that stayed for five days. On the eleventh day of ICU stay, flumazenil drip was discontinued, and two days later, the patient was transferred to the Department for Hepatobiliary Surgery.

Conclusion: This case illustrates the importance of careful management of patients with drastically reduced liver function. The patient presented with liver cirrhosis and a right-sided liver lobectomy, which left him with only 35% of liver function. Rarely used continuous IV flumazenil was the key that enabled this patient to regain consciousness earlier.

Keywords: Benzodiazepines; Flumazenil; Hepatectomy; Midazolam

CR223

Ultra-Massive Transfusion Following Abdominal Gunshot Injury: A Case Report

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Introduction: Abdominal gunshot wounds are associated with complex injuries and a high risk of delayed hemorrhage. Even after prompt damage control surgery (DCS), occult secondary vascular lesions may progress to catastrophic bleeding and trauma-induced coagulopathy. This case illustrates a dramatic, massive hemorrhage after an abdominal gunshot wound.

Case report: A previously healthy young male sustained a high-velocity gunshot wound to the left lower abdomen. On arrival, he was hypotensive and tachycardic. The patient was quickly taken to the operating room (OR), where he underwent DCS, during which all primary bleeding sources were surgically controlled. In the OR, he received packed red blood cells (pRBCs), fresh frozen plasma (FFP), platelets (PLTs), prothrombin complex concentrate (PCC), and fibrinogen to combat blood loss and trauma-induced coagulopathy. In the intensive care unit (ICU), initial hemodynamic improvement was quickly followed by a gradual drop in hemoglobin levels despite blood transfusions. An urgent abdominal computed tomography scan showed active bleeding in the retroperitoneum, indicating unrecognized secondary vascular injuries. He was brought back for a second DCS due to early signs of hemorrhagic shock. The massive transfusion protocol was initiated, including again, pRBCs, FFP, PLTs, PCC, and fibrinogen. The presence of acute bleeding and additionally oozing indicated an ongoing trauma-related coagulopathy. After achieving temporary control and packing, he was moved to the ICU, where his condition again worsened with increased hypotension, thrombocytopenia, and elevated lactate levels. A third emergent DCS revealed extensive secondary bleeding from the left femoral venous system, which was ligated and repacked under ongoing massive transfusion.

Conclusion: This case underscores the need for vigilant surveillance, rapid imaging, and a low threshold for re-exploration after abdominal gunshot trauma.

Keywords: Blood Coagulation Disorders; Shock, Hemorrhagic; Vascular System Injuries; Wounds, Gunshot

CR224

Successful Treatment of Severe Psoriasis with Risankizumab in a Patient with Asbestosis: A Case Report

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Introduction: Psoriasis is a chronic, immune-mediated skin disease characterized by erythematous, scaly plaques. Its immunopathogenesis involves dysregulated immune pathways, particularly the Th17/interleukin-23 (IL-23) axis. Risankizumab is a humanized monoclonal antibody that targets IL-23 and is highly effective in treating psoriasis. However, the use of IL-23 inhibitors in patients with interstitial lung disease (ILD), including asbestosis, raises concerns regarding infection risk and potential pulmonary disease exacerbation.

Case report: A 71-year-old man was referred to the dermatology department in March 2021 for psoriasis, which had been present since 2020. He presented with erythematous, scaly papules and plaques, predominantly affecting the scalp and extensor surfaces of the extremities along with nail changes including hyperkeratosis, onychodystrophy, and onycholysis. Psoriasis Area and Severity Index (PASI) 9, Dermatology Life Quality Index (DLQI) 5. A skin biopsy confirmed psoriasis vulgaris. His medical history was notable for occupational exposure to asbestos with radiologically confirmed, clinically stable asbestosis. Initially, he was treated with topical corticosteroids and vitamin D analogues, without significant clinical improvement. Over time, due to disease progression (PASI 25, DLQI 18) and contraindications to conventional systemic therapy, treatment with the IL-23 inhibitor risankizumab was initiated in June 2025. After administration of two doses at weeks 0 and 4, complete regression of psoriasis was achieved, and therapy has since been continued at 12-week intervals.

Conclusion: This case illustrates the effectiveness and good tolerability of risankizumab in a patient with severe psoriasis and asbestosis, without evidence of pulmonary deterioration during follow-up. IL-23 inhibition may therefore represent a safe and effective therapeutic option for carefully selected psoriasis patients with stable ILD.

Keywords: Asbestosis; Psoriasis; Risankizumab

CR225

A Pediatric Case of Shiga-toxigenic *Escherichia coli* Associated Haemolytic-Uraemic Syndrome with Early Neurological Involvement

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Introduction: Haemolytic-uraemic syndrome (HUS) is an acute complication following an infection with Shiga-toxigenic *Escherichia coli* (STEC). It is characterized by haemolytic anaemia, thrombocytopenia and acute kidney injury. Rapidly progressive HUS may be empirically treated with ravulizumab, a terminal complement-inhibiting monoclonal antibody, until complement-mediated disease is excluded. This case highlights early neurological involvement in typical HUS.

Case report: A two-year-old male was brought to the hospital after seven days of having multiple episodes of watery diarrhoeas. After two asymptomatic days he started vomiting and developed a petechial rash and oedema on hands and feet. Stool came back positive for STEC. Blood work showed haemolytic anaemia, severe thrombocytopenia (35x10⁹/mL) and high lactate-dehydrogenase levels (854 IU/L). Creatinine and urea levels were elevated and the patient was oliguric. During the first day the child developed generalized tonic-clonic seizure, which later progressed to left-side twitching together with consciousness impairment following body stiffness. Magnetic resonance imaging (MRI) of the brain revealed mild symmetric cytotoxic oedema in the thalamic area and around globus pallidus. Ophthalmologic examination revealed visual impairment with pupillary light reflex preserved. Seizures were treated with levetiracetam and midazolam. Patient went through three cycles of plasmapheresis and, due to neurological involvement, first dose of ravulizumab (600 mg i.v.) was given. Complement-mediated HUS was excluded after complement study, following discontinuation of ravulizumab therapy. Oliguria was managed with furosemide, without need for dialysis. Platelet and erythrocyte concentrates were given three and four times. Laboratory findings recovered rather quickly; however, neurological and visual recovery was very slow, and the follow-up MRI was normal.

Conclusion: STEC-HUS can present with early and severe neurological complications. Prompt recognition and multidisciplinary management is essential for avoiding irreversible damage.

Keywords: Antibodies, Monoclonal; Hemolytic-Uremic Syndrome; Plasmapheresis; Shiga-Toxigenic *Escherichia coli*

CR226

When thrombotic thrombocytopenic purpura is not TTP: malignancy-associated thrombotic microangiopathy: A case report

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Introduction: Thrombotic microangiopathy (TMA) represents a heterogeneous group of disorders characterized by microangiopathic hemolytic anemia, thrombocytopenia, and organ dysfunction. While thrombotic thrombocytopenic purpura (TTP) is a medical emergency requiring urgent plasma exchange, malignancy-associated TMA poses a distinct diagnostic and therapeutic challenge. This case highlights the importance of differentiating secondary TMA from primary TTP, as management and prognosis differ significantly.

Case report: A 62-year-old male with a history of hyperuricemia was admitted for evaluation of jaundice and right upper quadrant abdominal pain. Initial laboratory findings showed moderate normocytic normochromic anemia and mild thrombocytopenia. Computed tomography revealed thoracic and abdominal lymphadenopathy, borderline splenomegaly, biliary tract abnormalities, pancreatic and colorectal wall changes, diffuse skeletal sclerotic lesions suggestive of metastases, and prostate enlargement. During hospitalization, the patient developed rapidly progressive anemia, thrombocytopenia, and unconjugated hyperbilirubinemia. Further evaluation demonstrated reticulocytosis, markedly elevated lactate dehydrogenase, low haptoglobin, schistocytes on peripheral blood smear, and negative direct and indirect antiglobulin tests. ADAMTS13 activity was 38%, excluding TTP and supporting secondary TMA with no detectable inhibitors. Bone marrow cytology revealed clusters of poorly differentiated malignant cells consistent with metastatic adenocarcinoma of unknown primary origin. The patient developed transient neurological symptoms without radiological evidence of acute ischemia. Based on clinical, laboratory, and imaging findings, secondary TMA associated with advanced malignancy and partially compensated disseminated intravascular coagulation was diagnosed. Plasma exchange was not initiated due to limited expected benefit. Despite supportive treatment, the patient's condition deteriorated, and he died shortly after transfer to palliative care.

Conclusion: This case underscores the importance of recognizing malignancy-associated TMA as a distinct entity from TTP. Accurate differentiation is crucial to avoid ineffective therapies and to guide clinical decision-making in patients.

Keywords: Adenocarcinoma; Disseminated Intravascular Coagulation; Thrombotic Microangiopathies

CR227

When Youth Offers No Protection: Early-Onset Colorectal Carcinoma in a Young Adult

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Introduction: Colorectal carcinoma (CRC) is a major global health concern, ranking as the third most commonly diagnosed cancer worldwide, with a significantly increased risk after the age of fifty. However, a growing number of young individuals are presenting with CRC. Our aim is to highlight an early onset advanced CRC with no hereditary factors involved.

Case report: This case presents a 25-year-old male, current smoker, with no significant personal medical history, and with a negative family history of malignancy, who underwent comprehensive gastroenterological evaluation due to periumbilical abdominal pain. During colonoscopy, a pathologically altered segment of sigmoid colon was observed, encircling the lumen and causing luminal narrowing. The biopsy was obtained, and histopathological findings revealed malignant process. Computed tomography of the thorax, abdomen, and pelvis showed a stenosing tumor of the sigmoid colon, without evidence of dissemination. Consequently, a rectosigmoid resection was performed with an end-to-end colorectal anastomosis. Intraoperative exploration of the abdominal cavity revealed abnormal appearance of the pelvic peritoneum, which was excised because of suspected carcinomatosis. Histopathological examination of the resected colon segment confirmed an advanced-stage mucinous adenocarcinoma of the colon. The pelvic peritoneum specimen contained signet ring cells within mucinous tissue, confirming carcinomatosis. Tumor molecular profiling showed no mutations in the *KRAS, NRAS* or *BRAF* genes. Consequently, the multidisciplinary team recommended treatment with conventional chemotherapy protocol in combination with targeted anti-EGFR therapy, cetuximab, for five cycles.

Conclusion: This case highlights the importance of considering colorectal malignancy in differential diagnosis in young adults. In the era of targeted therapies and the rising incidence of CRC among younger individuals, early diagnosis is of crucial importance.

Keywords: Abdominal Pain; Adenocarcinoma, Mucinous; Colon, Sigmoid; Colonoscopy; Colorectal Cancer

CR228

Bleeding Dry: A Case of Acquired Hemophilia A**Petra Zemljak¹; Doris Barunčić¹; Anamarija Živković¹; Slavica Kvolik^{1,2}***1 - Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia**2 - Department of Anesthesiology, Resuscitation and Intensive Care, University Hospital Centre Osijek, Osijek, Croatia*

Introduction: Acquired hemophilia A (AHA) is a rare, but serious bleeding disorder. Unlike its hereditary variant, it is not caused by a genetic deficiency of coagulation factor VIII (FVIII), but by development of anti-FVIII autoantibodies in other conditions. Given its frequent association with autoimmune diseases and malignancies, it appears in both sexes. It presents as spontaneous bleeding in previously unaffected patients, and if unrecognized may have serious consequences.

Case report: We present the case of a 68-year-old female patient admitted to the hospital due to diffuse edema and hematoma of the left arm that has appeared at the venipuncture site 2 days prior. Patient's history shows that she was previously diagnosed with rheumatoid arthritis, IgA nephropathy, thyrotoxicosis and asthma. Upon examination in the Emergency Department the left arm was edematous and livid and the left hand was pale with palpable peripheral pulsations. Due to compromised limb perfusion, emergency fasciotomy was performed alongside anemia and hemorrhagic diathesis correction. Afterwards, the patient develops severe hypotension and tachycardia and was treated via QuickClot Control with dressing tamponade of the surgical wound. Bloodwork showed prolonged activated partial thromboplastin time (aPTT ratio 1.66), decreased FVIII (6%), and positive Bethesda test (1 BU/ml). In agreement with hematologist, the patient was treated first with recombinant FVIII, other blood products as needed, later adding factor VIIa concentrate and methylprednisolone. However, only after the administration of rituximab, a monoclonal antibody targeting antibody-producing CD20 positive B-lymphocytes, the patient's laboratory values, and bleeding slowly improved and surgical wound healed.

Conclusion: While uncommon, AHA is life-threatening if unrecognized. This case emphasizes the importance of timely diagnosis and treatment, as well as a potentially therapy-resistant nature of AHA.

Keywords: Antibodies; Factor VIII; Hemophilia A; Rituximab

CR229

What may be hidden behind hypertension in young man?

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Introduction: Hypertension in young individuals requires further evaluation due to common secondary etiology. Autosomal dominant polycystic kidney disease (ADPKD), mostly due to variants in PKD1 and PKD2, is the most common hereditary cause of kidney failure, characterized by bilateral renal cysts. Progressive cysts growth leads to kidney enlargement and development of chronic kidney disease often with early hypertension. This report highlights the importance of early recognition of an underlying condition and timely referral for optimal outcomes.

Case report: A 25-year-old male patient was referred to a nephrologist for further hypertension work-up. Over the past 6–12 months, the patient has experienced evening headaches, fatigue and elevated blood pressure (BP) of 140/91 mmHg. Kidney ultrasound revealed numerous bilateral renal cysts and kidney function was preserved with unremarkable urinalysis. Lifestyle modifications were proposed along with initiation of antihypertensive therapy (perindopril/indapamide). During follow-up, home BP values were satisfying. Due to unclear family history of polycystic kidney disease, genetic testing was performed revealing a pathogenic truncating variant PKD1 (c.5026del, p.Leu1676Trpfs*46) causing ADPKD. The magnetic resonance imaging of the kidneys was performed to assess kidney volume and cyst burden. The total kidney volume was 551 mL corresponding to Mayo Imaging Classification class 1C, with a Predicting Renal Outcomes in ADPKD (PROPKD) score of 7, indicating increased risk of ADPKD progression thus treatment with tolvaptan is indicated in this patient.

Conclusion: When not being at risk (unclear family history of ADPKD in our case), individuals with ADPKD may remain unrecognized with significant delay in diagnosis and appropriate treatment. Hypertension appears much earlier in individuals with ADPKD and may be its first sign. For that reason, hypertension in young people always requires further evaluation.

Keywords: Genetic Testing; Hypertension; Polycystic Kidney, Autosomal Dominant; TRPP Cation Channels

CLINICAL MEDICINE

 OSCON

CM01

Changes in the Hematopoietic and Coagulation Systems in Patients with Colorectal Cancer After Neoadjuvant Therapy

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Introduction: Colorectal cancer (CRC) is a leading cause of morbidity and mortality. Neoadjuvant therapy (NCRT) plays an important role in CRC treatment but affects the hematopoietic and coagulation systems. Von Willebrand factor (vWF) is a marker of endothelial activation and thrombotic risk. The hypothesis of this study was that patients who received NCRT would show more pronounced changes in hematopoietic and coagulation parameters compared to a control group. The aim was to examine these changes in surgical CRC patients after NCRT.

Materials and methods: Following Ethics Committee approval, 16 CRC patients scheduled for elective surgery were enrolled. Patients were divided into two groups: those who had received NCRT and a control group without NCRT. The mean age was 64.6 years (range 46–80). Leukocyte, hemoglobin, platelet count, and vWF levels were analyzed preoperatively, on the first postoperative day, and before hospital discharge.

Results: Patients who received NCRT had lower leukocyte counts before surgery (4.1 vs $7.3 \times 10^9/L$, $p=0.006$) and postoperatively (8.9 vs $15.9 \times 10^9/L$, $p=0.04$) compared to controls. Platelet counts were lower before surgery (194 vs $305 \times 10^9/L$, $p=0.012$) and postoperatively (149 vs $269 \times 10^9/L$, $p=0.01$). Hemoglobin levels did not differ significantly between groups. Median hemoglobin was 128 and 132 g/L preoperatively, and 107 and 110 g/L postoperatively, in the NCRT and control groups, respectively. No significant preoperative anemia was observed. Higher vWF levels were observed in the NCRT group (2.4 vs 1.7 , $p=0.04$).

Conclusion: These results support the hypothesis that neoadjuvant therapy induces more pronounced changes in the hematopoietic system in CRC patients. Elevated vWF levels may indicate endothelial activation and a potentially increased thrombotic risk during the perioperative period.

Keywords: Colorectal Surgery; Neoadjuvant Therapy; von Willebrand Factor

CM02

When resistance escalates: clinical challenges of multiple carbapenemases in carbapenem-resistant *Enterobacteriaceae*

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Introduction: *Enterobacteriaceae* are Gram-negative, rod-shaped bacteria and common causative agents of nosocomial infections, which are often treated with carbapenems, broad-spectrum β -lactam antibiotics. In recent years, the production of carbapenemases has led to an increasing prevalence of carbapenem resistance in certain isolates. The aim of this clinical study was to evaluate the frequency of isolates producing multiple carbapenemases and the available therapeutic options.

Materials and methods: Between 2020 and 2025 at University Hospital Centre Osijek, a total of 895 carbapenem-resistant *Enterobacteriaceae* (CRE) isolates were processed using routine microbiological methods, such as culture, Matrix-Assisted Laser Desorption/Ionization Time-of-Flight (MALDI-TOF) mass spectrometry and lateral flow detection of genes, with one or more carbapenemases detected: *Klebsiella pneumoniae* carbapenemase (KPC), New Delhi metallo- β -lactamase (NDM), Verona integron-encoded metallo- β -lactamase (VIM), imipenemase (IMP), and oxacillinase (OXA), followed by antimicrobial susceptibility testing.

Results: Analysis of CRE isolates over the study period identified 11 (1%) as producing multiple carbapenemases. The first CRE isolates with multiple carbapenemases were identified in 2022. The most common combination was NDM+KPC, detected in 5 (45%) isolates. Other combinations were NDM+OXA-48 in 4 (36%) isolates, KPC+OXA-48 in 1 (9%) isolate, and OXA-48+VIM in 1 (9%) isolate. Furthermore, 9 (82%) of the 11 isolates were *Klebsiella pneumoniae*, of which 7 (78%) were collected in 2025. The remaining affected bacteria were *Enterobacter cloacae* and *Klebsiella oxytoca*, with one isolate each. Antibiograms of all isolates with multiple carbapenemases showed resistance to all tested antibiotics apart from colistin.

Conclusion: The production of multiple carbapenemases represents a significant therapeutic challenge by severely limiting treatment options due to extensive antibiotic resistance in CRE. Active patient screening with surveillance cultures remains crucial for the timely detection of CRE carriers before infection develops.

Keywords: beta-Lactam Resistance; Carbapenem-Resistant *Enterobacteriaceae*; Colistin; Microbial Sensitivity Tests

CM03

Hidden Invaders: Trends and Antimicrobial Resistance of Anaerobic Bloodstream Infections

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Introduction: Anaerobic bacteria are an often underrecognized cause of bloodstream infections, reported to account for approximately 0.5–13% of adult cases. Among anaerobic pathogens causing bloodstream infections, *Bacteroides* species are most frequently reported. The aim of this study was to determine the frequency, temporal trends, and antimicrobial susceptibility patterns of anaerobic bacteria isolated from blood cultures over a six-year period.

Materials and methods: This retrospective study included blood cultures collected from hospitalized patients at the University Hospital Center Osijek between 2020 and 2025. During the study period, 38,378 blood cultures were processed, 6,636 of which were positive, yielding 42 anaerobic isolates included in this study. Blood cultures were analyzed using conventional microbiological techniques, and the results were descriptively synthesized.

Results: Anaerobic bacteria represented 0.6% of all positive blood cultures. The annual proportion of anaerobic isolates among positive blood cultures showed year-to-year variability and an overall upward trend, increasing from 0.1% in 2020 to 0.2% in 2021 and 0.7% in 2022, decreasing to 0.1% in 2023, and subsequently rising to 1.2% in 2024 and 1.3% in 2025. Of the 42 anaerobic isolates recovered from blood cultures, *Bacteroides fragilis* was the predominant species (35.7%), followed by *Bacteroides thetaiotaomicron* (16.7%). Overall, non-susceptibility was most frequent to clindamycin (40.5%), followed by amoxicillin–clavulanic acid (28.6%), penicillin (26.2%), and amoxicillin (21.4%); non-susceptibility to other tested agents was rare ($\leq 14.3\%$). Notably, anaerobic blood culture isolates showed resistance to clindamycin and penicillins, while no resistance to metronidazole was observed.

Conclusion: These results emphasize the importance of blood cultures and susceptibility testing, as *Bacteroides* species can rapidly acquire resistance, supporting metronidazole as a reliable option for empiric therapy.

Keywords: Bacteremia; *Bacteroides fragilis*; Blood Culture; Clindamycin; Metronidazole

CM04

Innovative Approaches in Post-Stroke Neurorehabilitation – Reactivation of Innate Movement Patterns

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Introduction: Post-stroke spasticity limits functional independence and delays neurological recovery. Therapeutic approaches based on ontogenetic motor development aim to reactivate innate movement patterns to support tone regulation and motor control. Autoreflex Prenatal and Postnatal Therapeutic Positions (APPP) use targeted reflex activation to influence neuromuscular regulation. This study evaluated the effect of this reflex-based intervention on spasticity reduction and functional recovery after stroke, hypothesizing earlier and greater improvement compared to conventional therapy alone.

Materials and Methods: Forty-six post-stroke patients with mild to severe spasticity completed a 3-week inpatient rehabilitation program. The intervention group (n = 25) received APPP-based therapy, while the control group (n = 21) underwent conventional physiotherapy. Spasticity was assessed using the Modified Ashworth Scale (MAS) at baseline, mid-intervention (1.5 weeks), and at discharge.

Results: Spasticity significantly decreased over time in the whole cohort (Friedman test: $\chi^2(2) = 80.419$, $p < 0.001$). Within-group analysis confirmed significant reductions across all three time points in both the control group ($\chi^2(2) = 35.547$, $p < 0.001$) and the experimental group ($\chi^2(2) = 45.340$, $p < 0.001$). Between-group comparison of overall change (discharge – baseline) demonstrated a significantly greater reduction of spasticity in the experimental group treated with APPP (Mann–Whitney U = 8.000, Z = -5.638, $p < 0.001$). The mean change score was -1.14 ± 0.78 in the control group compared to -9.88 ± 3.98 in the experimental group.

Conclusion: APPP-based reflex therapy resulted in a significantly greater reduction of spasticity compared to conventional rehabilitation. Early modulation of muscle tone may enhance motor control and support improved functional recovery after stroke.

Keywords: Physical Therapy Modalities; Recovery of Function; Reflex; Rehabilitation

CM05

Ligand Expression Profiles in the Immune Microenvironment of Muscle-Invasive Bladder Cancer

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Introduction: Immunotherapy enhances endogenous antitumor immune response and has demonstrated efficacy in several solid tumors, increasing interest in the immune microenvironment of bladder cancer. In addition to the well-established role of Programmed Death-Ligand 1 (PD-L1), growing attention is being directed toward novel immune checkpoint ligands including T-cell Immunoreceptor with Immunoglobulin and Immunoreceptor Tyrosine-based Inhibitory Motif domains (TIGIT), T-cell Immunoglobulin and Mucin-domain containing-3 (TIM-3), Lymphocyte Activation Gene-3 (LAG-3) as potential targets for the development of new immunotherapeutic approaches. In this study, we performed an immunohistochemical analysis of PD-L1, TIGIT, TIM-3 and LAG-3 in Muscle-Invasive Bladder Cancer (MIBC) specimens to evaluate their expression patterns within the tumor microenvironment.

Materials and Methods: Eight MIBC samples were selected from the archival repository of the Department of Pathology, University Hospital Centre “Sestre milosrdnice”. Inclusion criteria were >40% tumor tissue on a representative hematoxylin and eosin stained slide, confirmed muscularis propria invasion and visible intratumoral/peritumoral lymphocytic infiltration. Immunohistochemistry on formalin-fixed, paraffin-embedded tissue assessed PD-L1, TIGIT, TIM-3, and LAG-3 expression, which was quantified in immune microenvironment components using the immune cell score under high-power magnification.

Results: TIM-3 showed the highest expression, being positive in all samples, with 10–50% of immune cells stained. PD-L1 demonstrated expected expression patterns, with 1–20% positivity in seven samples. TIGIT was detected in seven samples, although at lower levels (1–5%). LAG-3 showed the lowest expression, with positivity observed in only two samples (1% and 3%).

Conclusion: This pilot study, through immunophenotyping, identifies the presence of immune checkpoint ligands (PD-L1, TIGIT, TIM-3) in MIBC, highlighting them as potential therapeutic targets. Further validation in larger cohorts is needed, including immune profiling and molecular subtyping.

Keywords: Immunohistochemistry; Immunotherapy; Ligands; Urinary Bladder Neoplasms

CM06

Artificial intelligence reveals surprising epidemiological patterns in the distribution of malignant hematological diseases - a step closer to better prevention

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Introduction: Multiple myeloma (MM), chronic lymphocytic leukemia (CLL) and acute myeloid leukemia (AML) belong to the most common malignant hematologic diseases. Despite the multifactorial etiology, some aspects, such as environmental factors, could have a pivotal role in their development. Various types of pesticides and toxins from the soil are strongly connected with some solid and non-solid neoplasms. The aim of this study was to determine the geographical distribution of MM, CLL and AML in eastern Croatia, assuming homogeneous distribution.

Materials and methods: Archival data were collected from three registers of patients who were diagnosed with MM (07/2017 – 06/2023), CLL (01/2014 – 06/2019) and AML (01/2018 – 06/2023), who were being treated in the Department of hematology in University Hospital Center Osijek. Using the artificial intelligence (AI) tool "ChatGPT" it was performed a mapping of geographic distribution and an AI statistical analysis of the probability of clustering of patient distribution in certain geographical locations in eastern Croatia, bringing them into relationship with major local hydrological systems.

Results: 307 patients were included, apart from 59 with MM, 79 with AML, and 170 with CLL. Global spatial analysis demonstrated a significant deviation from random distribution. The Quadrat test revealed pronounced heterogeneity in point density across spatial units ($\chi^2 = 1603.9$; $df = 35$; $p < 0.000001$). Kernel Density Estimation identified high-density hotspots along all Slavonic riverbeds, especially the River Drava and in proximity to major urban centers of eastern Slavonia.

Conclusion: Combining multiple statistical methods and AI analysis, we perceive that the geographic distribution of hemato-oncological patients has a specific pattern, so we refuted our hypothesis. Future research should be focused on the correlation of possible etiological factors and specific geographic distribution of these hematologic diseases

Keywords: Artificial Intelligence; Leukemia, Lymphoid; Leukemia, Myeloid; Multiple Myeloma

CM07

Kappa Free Light Chains – A Quantitative Alternative to Oligoclonal Bands in Multiple Sclerosis

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Introduction: Multiple sclerosis (MS) is characterized by intrathecal immunoglobulin synthesis. Oligoclonal bands (OCB) are the standard cerebrospinal fluid (CSF) marker, but their qualitative nature has increased interest in kappa free light chains (kFLC), recently incorporated into the 2024 McDonald criteria. This study evaluated the diagnostic performance of the kFLC index compared with OCB in patients undergoing evaluation for suspected MS.

Materials and methods: This cross-sectional study included 90 patients who underwent lumbar puncture between 2024 and 2025 during diagnostic evaluation of demyelinating disease. MS diagnosis was established according to the 2017 McDonald criteria. The MS group (n = 35) was compared with controls (n = 55) with non-inflammatory neurological disorders. Patients with systemic autoimmune or infectious conditions were excluded. Paired CSF and serum samples were obtained simultaneously. kFLC levels were measured by nephelometry, and the kFLC index was calculated. OCB were detected by isoelectric focusing with immunoblotting; positivity was defined as ≥ 2 CSF-restricted bands. Diagnostic performance was assessed using ROC analysis.

Results: The kFLC index was significantly higher in the MS group compared to controls (median 16.78, IQR 53.35 vs. 2.58, IQR 5.47; $p < 0.001$). OCB positivity was more frequent in MS patients ($p = 0.003$). ROC analysis demonstrated good diagnostic accuracy of the kFLC index (AUC = 0.78; 95% CI: 0.65–0.91), slightly higher than OCB (AUC = 0.74). The optimal kFLC cutoff value was 5.1, with 80% sensitivity and 69% specificity.

Conclusion: The kFLC index demonstrated diagnostic performance comparable to OCB in suspected MS. As a quantitative biomarker, kFLC may contribute to a more standardized and accessible assessment of intrathecal immunoglobulin synthesis, although further validation in larger patient cohorts is needed.

Keywords: Biomarkers; Cerebrospinal Fluid; Immunoglobulin Light Chains; Multiple Sclerosis; Oligoclonal Bands

CM08

Adaptation and Validation of the Scleroderma Skin Patient-Reported Outcome Questionnaire into Hungarian

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Introduction: Systemic sclerosis (SSc) is a chronic autoimmune connective tissue disease characterized by progressive fibrosis of the skin and internal organs, musculoskeletal involvement, and vascular abnormalities. Cutaneous involvement may be associated with significant impairment in quality of life from psychological, physical, and social perspectives. The Scleroderma Skin Patient-Reported Outcome (SSPRO) questionnaire was developed to assess these aspects. This study aimed to adapt and validate the SSPRO into Hungarian.

Methods: A total of 60 patients with SSc [female n=53, mean age 58.1 ± 13.4 years, diffuse cutaneous SSc (dcSSc) n=31 (51.67%)] and 31 age- and sex-matched healthy controls were enrolled. The 18-item SSPRO questionnaire was translated and culturally adapted using the OMERACT filter.

Results: The questionnaire significantly differentiated SSc patients from healthy controls (20.5 [11–32] vs. 11 points [7–17], $p < 0.007$). No significant difference was found between limited cutaneous SSc and dcSSc subgroups. A strong correlation was observed between SSPRO scores and reference questionnaires (Health Assessment Questionnaire Disability Index: $\rho = 0.499$, $p < 0.001$; Short Form Health Survey 36 Physical Component: $\rho = -0.570$, $p < 0.001$; Dermatology Life Quality Index: $\rho = 0.754$, $p < 0.001$, respectively). During the assessment of reproducibility, a high intraclass correlation coefficient was found (Cronbach's $\alpha = 0.894$, $p < 0.001$). Principal component analysis identified three subdomains.

Conclusion: The adaptation and validation of the SSPRO questionnaire into Hungarian was successful. It demonstrated good discriminative ability by significantly differentiating patients with SSc from healthy controls and showed strong correlations with established quality of life measures. Its high internal consistency supports reliability. The questionnaire may serve as a simple, practical and clinically applicable tool for routine assessment of skin related quality of life in SSc.

Keywords: Patient Reported Outcome Measures; Quality of Life; Systemic Sclerosis

REVIEW

R01

Managing Hepatofugal Portal Flow in Liver Transplantation and Retransplantation: Surgical Options to Restore Portal Inflow

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Introduction: Portal inflow failure after liver transplantation (LT) or retransplantation (re-LT), including hepatofugal portal flow, can cause graft hypoperfusion and dysfunction. This review summarizes surgical strategies to restore portal perfusion, focusing on venous conduits, renal vein–based reconstructions, collateral control/inflow modulation, and portal vein arterialization.

Review: We searched PubMed/Medline (2020–2025), prioritizing systematic reviews, multicenter series, and technical reports on portal inflow salvage in LT/re-LT. Search terms included: *portal steal, hepatofugal flow, renoportal anastomosis, jump graft, portal vein arterialization, portal vein thrombosis, portosystemic shunt, liver transplantation*. We included a 2025 case report on post-transplant hepatic venous outflow obstruction treated with a retrohepatic tissue expander. Portal hypoperfusion most often reflects chronic portal vein thrombosis, large spontaneous portosystemic shunts with “portal steal,” or complex mesentericoportal anatomy encountered in retransplantation. When feasible, physiological portal reconstruction is preferred (thrombectomy and anastomotic revision). If the portal vein is non-reconstructable, inflow can be re-established with venous conduits such as superior mesenteric vein-to-portal “jump” grafts or interposition vein grafting. In dominant splenorenal shunting with absent hepatopetal flow, renal vein–based strategies (e.g., renoportal anastomosis) and/or selective collateral ligation/clamping may restore portal perfusion. Endovascular options (angioplasty/stenting or thrombus-directed therapies) can complement surgery in selected lesions. In extreme salvage scenarios, portal vein arterialization has been described as a graft-saving bridge, requiring close postoperative surveillance for flow-related complications.

Conclusion: In LT/re-LT, early recognition and etiology-driven escalation from reconstruction to salvage techniques can preserve graft perfusion and avoid graft loss. A multidisciplinary algorithm integrating transplant surgery, interventional radiology, and intensive Doppler-based surveillance is essential in complex cases. Future multicenter studies are needed to define flow targets and clarify outcomes of renal vein–based pathways and portal vein arterialization.

Keywords: Endovascular Procedures; Liver Transplantation; Portal Vein; Portosystemic Shunt, Surgical; Thrombosis

R02

Liquid Biopsy and Artificial Intelligence: The Future of Non-Invasive Cancer Detection – A Narrative Review

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Introduction: Cancer remains a leading cause of mortality worldwide, with early detection crucial for treatment and survival. While tissue biopsy remains a gold standard in histological and molecular diagnostics, liquid biopsy (LB) has emerged as a promising, less invasive alternative for early cancer detection. This concept is particularly relevant in the context of artificial intelligence (AI), which provides the tools necessary for robust biomarker data analysis and accurate pattern recognition. This review aims to present original studies using non-invasive methods in cancer detection.

Review: PubMed was searched for original studies on non-invasive cancer detection using LB and AI, published 2019 – 2025, with the keywords “liquid biopsy”, “cancer”, and “machine learning”. Reviews, case reports, and studies not addressing early detection were excluded; nine original studies were included, and results were presented descriptively. Three of the selected studies focused on early detection of pancreatic cancer; two used exosome-based approaches, while one focused on circulating microRNA (miRNA). All achieved high accuracies in early-stage disease. Two additional studies illustrated different LB approaches in lung cancer; one demonstrated a high accuracy in cancer detection by combining immune cell metabolic profiling and machine learning (ML), while the other successfully utilized a cell-free DNA (cfDNA) ML model in disease progression. An exosome-derived miRNA signature was also developed for the detection of early-stage gastric cancer. Finally, three studies demonstrated effective pan-cancer detection strategies using cfDNA, exosomal miRNAs and platelet RNA ML models.

Conclusion: This review illustrates that studies using liquid biopsy approaches, including cfDNA, exosomal biomarkers, and AI models demonstrate promising diagnostic accuracy in cancer detection, supporting the transition toward non-invasive diagnostic strategies with the potential to improve cancer screening and patient outcomes.

Keywords: Cell-Free Nucleic Acids; Liquid Biopsy; Machine Learning; MicroRNAs; Neoplasms

R03

Obesity Paradox – Better Survival and Increased Risk of Complications in Obese Surgical Patients

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Introduction: Despite the “obesity paradox” - better survival after surgery for some obese compared to normal weight cancer patients, the incidence of complications is higher in obese patients. This review aims to highlight underlying mechanisms and outcomes of obese surgical patients.

Review: Hyo Jin et al. (2024) performed an observational study of adult patients undergoing major surgery using the 2016 to 2019 National Surgical Quality Improvement Program database. The outcome was 30-day mortality. Their findings suggest better survival in most adult obese surgical patients, yet the trend dissipates with high procedural complexity and comorbidity burden. However, Hirano et al. (2023) and Mullen et al. (2009) have confirmed increased production of reactive oxygen species in inflamed adipose tissue, leading to pronounced oxidative stress, endothelial dysfunction, chronic inflammation, and adipokine dysregulation. Obesity is associated with a higher prevalence of insulin resistance, which impairs endothelial function, increases thrombogenicity, increases the incidence of infections, delays wound healing, and increases the rate of reoperation (Prieto et al., 2014). Fanaki et al. (2025) established perioperative ERAS protocols for obese patients, thereby minimizing complications and enhancing recovery. In addition to metabolic disturbances, obese patients demonstrate an increased risk of respiratory and cardiovascular complications, including hypoventilation under anaesthesia, reduced functional residual capacity, arrhythmias, and cardiac dysfunction.

Conclusion: In the last 10 years, publications confirmed that obesity represents a significant risk factor for carcinogenesis and contributes to increased perioperative morbidity in surgical patients through a complex interaction of molecular mechanisms. An individualized perioperative approach is essential to reduce risk and improve surgical outcomes, especially in morbidly obese patients.

Keywords: Intensive Care, Surgical; Obesity; Oxidative Stress; Perioperative Care; Postoperative Complications

R04

State-of-the-Art Pharmacoeconomics: Balancing Cost and Efficacy in the Era of Advanced Therapy Medicinal Products

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Introduction: Advanced therapy medicinal products (ATMPs) comprise three main types: gene therapy medicines, somatic-cell therapy medicines, and tissue-engineered medicines. Although ATMPs and orphan medicines are not synonymous, some ATMPs target rare diseases previously considered incurable and consequently may be designated as “orphan drugs”, often entailing high costs and the need for robust pharmacoeconomic evaluation. Thus, this narrative literature review aims to explore and descriptively present the pharmacoeconomic challenges and evaluation methods associated with ATMPs, focusing on their cost-effectiveness and reimbursement implications.

Review: A literature search was conducted in the PubMed database. Articles published in English between 1 January 2020 and 31 December 2025 were included. All studies focusing on pharmacoeconomic evaluations, cost analyses, and health technology assessments of ATMPs were selected for further assessment. ATMPs face significant hurdles in clinical trial design, particularly randomized controlled trials, due to small, homogenous patient populations. To address this, real-world evidence is increasingly incorporated into regulatory processes and decisions for orphan drugs. Pharmacoeconomic evaluations, including cost-utility analyses and cost-effectiveness analyses, guide funding/reimbursement decisions. However, conventional willingness-to-pay thresholds often fail to capture the unique value of ATMPs, necessitating adjusted criteria to better reflect their therapeutic and economic impact. Pharmacoeconomics represents one component of the broader decision-making process, while Health Technology Assessment integrates clinical, economic, societal and ethical factors to guide reimbursement and policy decisions. Pharmacoeconomic analyses remain essential for comparing alternative therapeutic options.

Conclusion: This review highlights that, as novel ATMPs continue to emerge, in-depth pharmacoeconomic evaluations, combined with real-world evidence that captures not only effectiveness and safety but equally patient-reported outcomes, and health technology assessments, are essential to inform reimbursement decisions and facilitate access to patient-tailored treatments.

Keywords: Costs and Cost Analysis; Economics; Pharmacoeconomics; Precision Medicine; Rare Diseases

R05

The Role of Sleep in the Genetic Regulation of Tumor Angiogenesis and Immune Response to Tumor

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Introduction: Sleep disturbances are related to dysfunction of the body and increased number of diseases, especially cancer, by causing irregular circadian rhythms thus affecting cell proliferation, angiogenesis and DNA repair, key factors in tumor development. The aim of this paper is to briefly explain how sleep is related to circadian genes involved in tumor angiogenesis and immune response to tumor at the molecular level.

Review: PubMed search (key words: Angiogenesis, Circadian Rhythm, Immunotherapy, Melatonin, Sleep) results presented the connection between sleep disruption and alteration in gene expression related to cell growth and circadian rhythm important in tumor development. Some of the more important circadian genes regulate growth signaling, DNA repair and cell cycle. When sleep is disturbed, DNA repair is damaged and tumor growth is stimulated by increased angiogenesis and oxidative stress. One possible link is suppression of melatonin by disruption of sleep which affects its modulatory effect of circadian gene activity and ability to inhibit angiogenesis by downregulating *vascular endothelial growth factor-A* (VEGF-A), other proangiogenic factors and genes related to tumor biology (such as *Per2*, *TNF-α*, *p53*, *PDGF-C* and *Ang*). Importantly, melatonin inhibits the phosphorylation of *endothelial growth factor receptor* (EGFR) and its downstream effectors. Melatonin synergistically increases cytotoxic and apoptotic effect of monoclonal antibody therapy against EGFR. Sleep deprivation decreases NK and CD3⁺-T cell number and increases immunosuppressive CD11b⁺ cells in tumor tissue promoting tumor progression, possibly via glucocorticoids' regulatory effects.

Conclusion: Understanding sleep's effects on circadian gene regulation, angiogenesis and immunity is necessary to facilitate prevention and therapy of tumors. Future studies should investigate strategies to optimize sleep to improve immunotherapy outcomes and tumor control.

Keywords: Angiogenesis; Circadian Rhythm; Immunotherapy; Melatonin; Sleep

R06

Digital Stress and the Epigenome: Exploring the Molecular Footprint of Social Media Exposure

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Introduction: Over recent years, the digital environment has become the most influential psychosocial factor in terms of its impact on human society. The increase in social media use increases cyberbullying, internet addiction (IA), online harassment, and digital stress overall. The aim of this abstract is to present a literature review of the latest research on epigenetic changes induced by social media.

Review: A literature search was conducted using PubMed, Scopus and Web of Science database searching a combination of the following keywords: “digital stress”, “epigenetic footprint”, and “social media”. Reviewed were papers published between January 2020 and November 2025, a total of 13 papers were found that analyze digital stress, of which 6 were selected and whose results are descriptively presented in this review. Inclusion criteria focused on studies that specifically analyze digital stress and its molecular footprint such as DNA methylation of psychosocial stress-responsive genes. Selected studies were conducted with a focus on epigenetic modulations caused by methylation of the oxytocin receptor (*OXTR*), dopamine transporter (*DAT1*), serotonin transporter (*SLC6A4*), and brain derived neurotrophic factor (*BDNF*) genes. The *OXTR* and *DAT1* genes showed coherence of methylation with the level of IA, however, due to methodological heterogeneity, the *SLC6A4* and *BDNF* genes still remain unconfirmed biomarkers related to digital stress. The resulting epigenetic modifications of *OXTR* and *DAT1* genes were associated with the behavioral manifestation of depression and anxiety.

Conclusion: Digital stress, although still an understudied epigenetic factor, shows certain results in its impact on the epigenetic profile of the subjects. Further scientific research could significantly expand the existing knowledge and clarify still unexplained effects of digital stress.

Keywords: Digital stress; Genes; Internet addiction; Methylation

R07

European viper envenomation in children – a review with accentuating clinical experience from Croatia

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Introduction: *Vipera ammodytes* is the most venomous European snake. Paediatric envenomation is significant due to the specific clinical grading, severe clinical presentation and frequent local complications. The aim of this review is to summarize the clinical and therapeutic characteristics of European viper envenomation in children.

Review: We analysed a total of 208 published original scientific papers, reviews and case reports describing paediatric viper envenomation in Europe from 1979 until 2024 available in *PubMed* and *Web of Science*. We used combination of terms: “*Vipera*”, “*Vipera ammodytes*”, “snake envenomation”, “neurotoxicity”, “Europe”, “child”, “paediatric”, “antivenom” only in the work titles and abstracts, afterwards eliminating works without clinical and therapeutic data. Most paediatric snakebites occur during warm months, affect the upper limbs and result in moderate to severe envenomation, but fatal cases are rare. Local swelling, discoloration of skin and ecchymosis at the bite place, usually occur within two to four hours. Available data from Croatia show that most common local complications were haemorrhagic blisters (20%) followed by compartment syndrome (7.5%). The predominant general complication was cranial nerve paresis/paralysis (11.2%), usually manifested as the oculomotorius nerve lesion with bilateral palpebral ptosis that recovered approximately 72 hours after the bite. All children treated in Croatia until 2020 received antivenom produced by the Institute of Immunology in Zagreb without complications after providing it. However, since 2020, this antivenom has no longer been produced, and the data on the efficacy of alternative antivenoms are limited.

Conclusion: This review highlights specific manifestations and complications of *Vipera ammodytes* bites in children and importance of timely antivenom administration. Future research should focus on comparing antivenom efficacy and collecting multicenter pediatric data to improve treatment outcomes.

Keywords: Antivenins; Child; Snake Bites; *Vipera*

R08

Personalizing fluoropyrimidine-based therapy through pre-emptive *DPYD* pharmacogenetic testing: A systematic review of cost-effectiveness and cost-utility analyses

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Introduction: Pre-emptive *DPYD* testing is important for identifying patients at increased risk of severe fluoropyrimidine-related toxicity and for guiding safer, individualized dosing. Thus, our aim was to assess the cost-effectiveness of pre-emptive *DPYD* testing prior to fluoropyrimidine-based therapy.

Review: We conducted a systematic literature review using PubMed/MEDLINE, searched on 12 January 2026 to identify studies reporting cost-effectiveness or cost-utility analyses of pre-emptive *DPYD* testing prior to fluoropyrimidine-based therapy. Eligible studies were published from 1 January 2020 onwards. The following search strategies were applied: (1) *DPYD* AND cost*; and (2) *DPYD* AND econom*. The initial search yielded 48 and 20 records. Seven studies met inclusion criteria, comprising six cost-effectiveness analyses and one cost-utility analysis. Six studies employed Markov models, while one used a decision-analytic model. All studies were conducted from a national healthcare perspective, including Qatar, Australia, the United Kingdom (with additional perspectives from the Netherlands and Hungary), Italy, Iran, and the United States. Three studies focused on colorectal cancer populations, three on breast cancer, and one study included all solid tumors. Despite substantial heterogeneity in study designs, populations, and model inputs, all included studies consistently reported that pre-emptive *DPYD* testing prior to fluoropyrimidine-based therapy is likely to be cost-effective, with incremental cost-effectiveness ratios below established willingness-to-pay thresholds, regardless of tumor type or stage.

Conclusion: Overall, the available evidence indicates favorable economic value for pre-emptive *DPYD* testing. These findings support the economic justification for routine implementation of *DPYD* testing prior to fluoropyrimidine-based therapy in national healthcare settings.

Keywords: Dihydropyrimidine Dehydrogenase; Fluorouracil; Neoplasms; Pharmacoeconomics; Pharmacogenetics

R09

Freezing the Pain: Cryoneurolysis in Modern Pain Management – An Educational Overview

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Introduction: Cryoneurolysis is a modern, minimally invasive pain treatment option considered when conservative methods do not lead to satisfactory pain reduction. It uses extremely low temperatures to modify the conduction of pain impulses resulting in cryoanalgesia. The aim of this educational review is to demonstrate the underlying mechanism that enables a high-safety profile with maximum efficiency.

Literature review: Cryoneurolysis is an image-guided procedure that applies extreme cold to targeted nerves, inducing reversible Wallerian degeneration to achieve analgesia. A diagnostic block using a local anesthetic, with or without a corticosteroid, around the afferent nerve is recommended. A greater reduction of pain observed by visual analog scale is considered an indication for cryoneurolysis. Under image guidance, a cryoprobe is introduced to the targeted afferent nerve. The probe contains an integrated neurostimulator, allowing confirmation of sensory nerve involvement while avoiding motor fibers. Formation of intraneural ice crystals is achieved by using high-pressure gas. The analgesic effect of cryoneurolysis persists until regeneration of the affected nerve occurs, lasting couple of weeks to months. Cryoneurolysis is applicable for use in chronic musculoskeletal pain, cancer pain and postoperative pain. Available evidence suggests a favorable safety profile, with predominantly transient sensory adverse effect reported. Cryoneurolysis offers an individualized pain management option for patients who cannot achieve adequate relief with long-term analgesic therapy or are not candidates for surgical treatment.

Conclusion: This overview illustrates how cryoneurolysis offers a long-lasting effect with fewer complications compared to traditional treatment. Reversible Wallerian degeneration relieves pain, without major safety concerns. At the same time, it enables reduction of analgesics and opioids usage.

Keywords: Cryotherapy; Pain Management; Wallerian Degeneration

OTHER

OSCON

O01

Introducing Diazoxide Choline Extended-Release Tablet in Children with Prader-Willi Syndrome: To Expect a Revival or To Be Restrained?

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Introduction: Prader-Willi syndrome (PWS) is a rare neurobehavioral and metabolic disorder caused by the loss of paternal alleles on 15q1-q13. PWS is characterized by hypotonia, neurocognitive and endocrine problems, and hyperphagia leading to severe obesity. Diazoxide choline extended-release tablet (DCCR) is the first FDA-approved pharmacotherapy for hyperphagia in PWS, penetrates the blood-brain barrier, activates ATP-sensitive potassium channels (KATP), and suppresses hypothalamic orexigenic neuropeptide secretion (neuropeptide Y and agouti-related protein), thus reducing hyperphagia. Peripheral KATP activation in the dorsal motor nucleus of the vagus, pancreatic β -cells, and adipocytes could decrease hyperinsulinemia and adiposity, while enhancing insulin and leptin sensitivity, and promoting satiety.

Materials and methods: Upon parental consent, ten children, five boys and five girls, will be enrolled into cohorts: progressive weight gain for children aged 4–12 years, or severe obesity for children over 12 years. All participants will receive DCCR for two years. Primary outcomes will include changes in hyperphagia, as assessed by the Hyperphagia Questionnaire for Clinical Trials, and weight changes. Secondary endpoints will include Global Impression Scores, changes in physiological composition, behaviour, and hormones. Safety monitoring will include serial fasting plasma glucose and HbA1c measurements.

Results: Treatment is expected to reduce hyperphagia and body fat, particularly in younger participants. These effects are expected to result from combined central hypothalamic actions and improvements in hyperinsulinemia and leptin resistance, accompanied by decreases in circulating insulin and leptin concentrations. Additional benefits will include improved behavior, metabolism, reduced food-related outbursts, and more focus on daily activities.

Conclusion: This study aims to further define the efficacy and safety profile of DCCR in children with PWS and evaluate its potential to reduce obesity-related complications, improve quality of life, and ease caregiver burden.

Keywords: Hyperphagia; Obesity; Prader-Willi Syndrome

O02

Leaving home, mental adjustment and academic success in university students

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Introduction: The beginning of university studies is one of the most important life transitions and often involves leaving the parental home and possible adaptation difficulties. We examined the relationship between leaving home, mental adjustment and academic success.

Materials and methods: A cross-sectional study was conducted among students at Josip Juraj Strossmayer University of Osijek using an anonymous online questionnaire developed by the authors in June and July 2024. Results are presented both descriptively and analytically.

Results: A total of 515 students completed the questionnaire, of whom 69.9% (360) were female. The median age of the participants was 21 years (interquartile range 20-23). Overall, 30.6% of students were from biomedical and health-related fields. 65% of students had moved out of the parental home to study at university. Moving out at the age of 18 years was the most common (28.7%). Half of the students who left reported easy and rapid adaptation to the new environment, and almost half reported no changes in mood during the first weeks of the academic year. Biomedical students more frequently reported low mood. There was no difference between students who left the parental home and those who did not in their assessment of academic performance at university compared with their high school performance. Students in biomedical and health-related fields more frequently reported academic achievement similar to that in secondary school.

Conclusion: The study illustrates that, despite leaving the parental home and experiencing slower adaptation to a new environment, most students did not report changes in academic performance compared to their performance in secondary school.

Keywords: Academic Performance; Residence Characteristics; Students

O03

From Trembling Hands to Confident Talks: Soft-Skills Training for Precision-Immunology Communication

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Introduction: Precision immunology and genome-informed immune care increasingly rely on communicating complex, individualized findings to clinical teams and patients. Yet public-speaking anxiety is common in health-science training and can undermine scientific communication. We evaluated whether small-group, practice-intensive soft-skills courses improve presentation competence and reduce anxiety. We hypothesized significant gains in presentation skills and reductions in public-speaking, social, and general anxiety.

Materials and methods: We conducted a longitudinal pre–post quantitative evaluation over one curriculum-embedded semester. Participants were 127 medical students (88 women, 39 men; mean age 23 years). At the start and end of the course, students completed self- and peer-assessments using validated questionnaires: The Public Speaking Competency Rubric (PSCR+), the Personal Report of Confidence as a Speaker scale (12-item; PRCS-12), the Brief Fear of Negative Evaluation Scale (BFNE), and the State–Trait Anxiety Inventory (STAIT-5). Pre–post differences were tested using paired comparisons.

Results: All instruments demonstrated statistically significant improvements from baseline to the post-course assessment ($p < .001$). The PSCR+ indicated higher communication confidence and delivery skills, proven by both self- and peer-ratings. PRCS-12 scores decreased, reflecting reduced public-speaking anxiety. BFNE scores declined as well, particularly in fear of observation and negative evaluation. STAIT-5 scores also decreased, indicating reduced general anxiety.

Conclusion: Practice-intensive soft-skills training strengthened presentation competence and reduced multiple anxiety domains, supporting the hypothesis. Embedding this training may enhance the communication capacity needed for patient-tailored explanations of immune and genomic data in precision immunology.

Keywords: Anxiety; Communication; Education, Medical; Students, Medical

O04

Digital Twin Technology in Personalized Precision Cardiometabolic Care: An Educational Review of Current Applications and Future Perspectives

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Introduction: Digital Twin (DT) technology refers to the creation of dynamic virtual replicas of individuals that integrate physiological, behavioral, clinical, metabolic, genetic, psychosocial, and environmental data. Given the global burden of cardiometabolic diseases, DTs offer a transformative framework for personalized treatment and prevention. This overview outlines the core components and advantages of DT technology with a focus on cardiometabolic care.

Review: This overview provides an integrated synthesis of DT and its use in the cardiometabolic field. Recent studies underscore the transformative potential of DT-guided interventions in managing cardiometabolic diseases, demonstrating significant clinical improvements in glycemic control, adiposity markers, blood pressure, and lipid profiles, ultimately leading to reduced cardiovascular risk. Unlike traditional population-based models that fail to reflect individual disease trajectories, DTs enable personalized simulation of disease progression in response to lifestyle changes and individual treatment planning by modeling the effects, sequence, and timing of therapeutic interventions. Additionally, visualization of outcomes enhances patient's understanding and motivation*. Beyond clinical care, DTs support drug development through *in silico* testing, synthetic control groups and head-to-head comparison. From an economic and policy perspective, continuous assessment of patient trajectories informs dynamic reimbursement and more efficient resource allocation. Despite these advantages, ethical considerations related to data use, transparency, and accountability persist. In addition, regulatory frameworks for DT-based support systems continue to evolve and require dedicated education of healthcare professionals.

Conclusion: DT technology represents a transformative approach for personalized precision-based cardiometabolic care. Although early evidence demonstrates multiple benefits, successful implementation will demand attention to ethical, regulatory, and educational constraint.

Keywords: Drug Development; Education; Hypertension; Obesity; Type 2 Diabetes Mellitus

O05

High Levels of Depression Symptoms and Suicidal Ideation Among Medical Students: A Cross-Sectional Study

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Introduction: Introduction: The prevalence of depression and the level of suicidal ideation among medical students in Croatia have not been sufficiently investigated. Therefore, this study aimed to assess depressive symptoms and suicidal ideation among medical students at the University of Osijek, Croatia, hypothesizing a high prevalence of depressive symptoms and suicidality.

Materials and methods: This cross-sectional study was conducted between September 29 and October 5, 2025, among medical students at the Faculty of Medicine in Osijek, Croatia. All 444 students were invited to participate in an anonymous survey via official university email addresses, and 260 participated (response rate: 58.6%). The questionnaire was completed using Google Forms. Depressive symptoms and suicidal ideation were assessed using the validated Croatian version of the Patient Health Questionnaire-9 (PHQ-9). Ethical approval was obtained from the Ethics Committee of the Faculty of Medicine, University of Osijek (Approval code: 2158-61-46-25-154).

Results: The median age was 21 years, with 197 females and 63 males. According to the PHQ-9, 54.2% reported depressive symptoms, including 9.2% with moderately severe or severe symptoms. Additionally, 12.8% reported thoughts of being better off dead or self-harm in the previous two weeks. Female students had significantly higher PHQ-9 scores (median [IQR]: 5 [3–9]) than male students (median [IQR]: 4 [2–8]) ($p = 0.01$, Mann–Whitney U test).

Conclusion: High levels of depressive symptoms and suicidal ideation were observed among medical students. Female students demonstrated significantly higher levels of depression, highlighting the need for targeted mental health screening, prevention strategies, and support programs within medical education.

Keywords: Cross-Sectional Studies; Depression; Students, Medical; Suicidal Ideation

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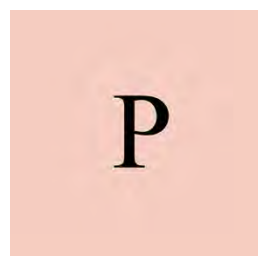
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