BOOK OF ABSTRACTS

NEURI 2026

STUDENT CONGRESS OF NEUROSCIENCE



24th APRIL - 26th APRIL 2026

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NEURI CONGRESS

Rijeka - Rab



EDITOR-IN-CHIEF

Renata Miskrić

EDITORIAL BOARD

Vladimir Cvitanović

PUBLISHER

Faculty of Medicine Rijeka
Braće Branchetta 20
51000 Rijeka

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NEURI CONGRESS

STUDENT CONGRESS OF NEUROSCIENCE

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24th APRIL - 26th APRIL 2026



GENERAL INFORMATION

DATE

24th APRIL - 26th APRIL 2026

VENUE

- FACULTY OF MEDICINE RIJEKA
- INSULA COUNTY HOSPITAL, RAB





TOPICS

NEUROSCIENCE, NEUROLOGY, PSYCHIATRY, NEUROSURGERY



LANGUAGE

OFFICIAL LANGUAGE OF THE CONGRESS IS ENGLISH

CONGRESS FORMAT

IN-PERSON





CONTACT INFORMATION

neuricongress@gmail.com

COMMITTEES

SUPERVISION TEAM
- Renata Miskrić
- Vladimir Cvitanović
- Maximilian Wechsler

MEDIA AND DESIGN
- Ana Bardić
- Karlo Đaković
- Eleonora Chersi
- Maritea Arelić
- Lucija Marojević
- Vanesa Augustić
- Helena Pejić Dragović

COORDINATORS OF PARTICIPANTS
- Paulina Kušan
- Magdalena Kadović
- Lana Sabati

TECHNICAL SUPPORT
- Maximilian Wechsler
- Gabrijela Šoštarić
- Izak Feher
- Matea Cica
- Lucija Španić

COORDINATORS OF RESOURCES
- Tihana Majetić
- Maja Tomljenović
- Ana Modrčin
- Martina Linić
- Karla Kleščić

SCIENTIFIC COMMITTEE
- Rea Krmpotić
- Nico Lohfink
- Lori Lovrenčić
- Mia Mašina
- Hana Dovolić
- Marija Ševo
- Florian Liebau
- Sonja Farkaš
- Matija Jovanović
- Annabel Heman-Ackah

COORDINATORS OF PARTICIPANTS
- Ines Žitko
- Tea Elez
- Eni Šebelja
- Milana Vukmirović

WELCOME MESSAGE

Dear colleagues, distinguished lecturers, and dear participants,

It is my great pleasure to welcome you to the 15th Student Congress of Neuroscience – NeuRi 2026.

NeuRi was founded with the vision of creating a platform where students, young researchers, and healthcare professionals can come together to exchange knowledge, share research, and inspire one another. Over the years, the congress has grown into an international meeting that encourages collaboration across disciplines while fostering a passion for neuroscience and scientific discovery.

Each abstract reflects the hard work, commitment, and innovative thinking of its authors. We are proud to showcase a diverse collection of research spanning various fields of neuroscience, highlighting both fundamental science and clinical practice.

On behalf of the Organizing Committee, I would like to express my sincere gratitude to all authors for submitting their work, to our Scientific Committee for ensuring a high standard through the review process, and to our lecturers, workshop leaders, sponsors, and partners for their invaluable support. Most importantly, I would like to thank every member of the Organizing Committee and our volunteers, whose dedication and teamwork made this congress possible.

I hope this congress will inspire new perspectives, meaningful collaborations, and lasting friendships, while this Book of Abstracts serves as a valuable record of the scientific contributions presented during NeuRi 2026.

Thank you for being part of our congress. I wish you a productive, inspiring, and enjoyable NeuRi 2026.

Renata Miskrić
President of the Organizing Committee

NEURI 2026

15TH STUDENT CONGRESS OF NEUROSCIENCE

APRIL
24.-26.04.2026.

PROGRAMME

The Faculty of Medicine of the University of Rijeka and Insula County Hospital Rab

FRIDAY - APRIL 24
Faculty of Medicine Rijeka

9:00-12:00

Registrations

12:00-12:30

Opening ceremony

12:30-13:30

Lecture cycle 1
1. Hrvoje Šimić
2. Josipa Kajić

13:30-13:40

Coffee break

13:40-14:40

Lecture cycle 2
1. Sandra Blažević Zelić
2. Jelena Radić Nišević
3. Mirza Pojskić

14:40-14:55

Official Photoshoot

15:00-16:00

Lunch

16:00-17:00

Student Sessions 1

NEURI 2026

15TH STUDENT CONGRESS OF NEUROSCIENCE

APRIL
24.-26.04.2026.

PROGRAMME

The Faculty of Medicine of the University of Rijeka and Insula County Hospital Rab

FRIDAY - APRIL 24
Faculty of Medicine Rijeka

17:00-18:00

Workshops

18:00-19:00

Student Sessions 2

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at 21

Karaoke at River Pub
Ul. Frana Supila 12, 51000, Rijeka

NEURI 2026

15TH STUDENT CONGRESS OF NEUROSCIENCE

APRIL
24.-26.04.2026.

PROGRAMME

The Faculty of Medicine of the University of Rijeka and Insula County Hospital Rab



SATURDAY - APRIL 25
Insula County Hospital Rab

6:30-9:45

Drive to Rab

9:45-10:00

Arriving at Insula

10:00-10:15

Welcome speech

10:30-11:30

Lecture cycle 3

1. Vesna Šendula-Jengić
2. Ana Kapor Tomić
3. Marija Tomac Stojmenović

11:30-11:45

Coffee break

11:45-12:45

Tour of the hospital

12:45-13:45

Lunch

14:00

Island of Rab

16:00-18:15

Drive to Rijeka

21:00

Gala Dinner at Botel Marina

NEURI 2026

15TH STUDENT CONGRESS OF NEUROSCIENCE

APRIL
24.-26.04.2026.

PROGRAMME

The Faculty of Medicine of the University of Rijeka and Insula County Hospital Rab



SUNDAY - APRIL 26
Faculty of Medicine Rijeka

9:00-10:00

Student sessions 3

10:00-10-30

Zdravi doručak

10:30-11:30

Lecture cycle 4
1. Eliša Papić
2. Slavica Kovačić
3. Marija Janev

11:30-12:10

NeuRi Social & Photo Time

12:10-13:10

Lecture cycle 5 (Lecture + Roundtable)
1. Marko Šestan

13:10-14:10

Lunch

14:10-15:30

Lecture cycle 6
1. Marija Pranjić
2. Iris Martinović - Sentinel Uma (Lecture + Roundtable)

NEURI 2026

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PROGRAMME

The Faculty of Medicine of the University of Rijeka and Insula County Hospital Rab



SUNDAY - APRIL 26
Faculty of Medicine Rijeka

15:30-15:40

Coffee break

15:40-16:40

Workshops

16:40

Closing ceremony

LECTURES

24th APRIL - 26th APRIL 2026

Prof. Vesna Šendula-Jengić, MD, PhD, psychiatrist

Prof. Vesna Šendula Jengić, MD, PhD, is a distinguished psychiatrist with extensive clinical and leadership experience at the Insula County Specialist Hospital. Guided by a biopsychosocial approach, she has dedicated her professional career to the understanding, treatment, rehabilitation, resocialization, and destigmatization of individuals with a wide spectrum of mental health disorders.

After graduating from the Faculty of Medicine in Rijeka in 1983, she completed her specialization in psychiatry (1990), subspecialization in forensic psychiatry (2007), and obtained both her MSc and PhD degrees (1990; 2008). She continuously enhances her knowledge through various educational programs and professional development activities.

During her long-term leadership of the Insula County Specialist Hospital (2001–2025), the hospital was renovated and transformed in accordance with high contemporary standards. Alongside her extensive clinical work and leadership responsibilities, Prof. Šendula Jengić is dedicated to scientific and academic work. She is the author of numerous professional and scientific papers published in indexed journals and other reputable publications.

She shares her valuable knowledge and clinical experience with students at the Faculty of Medicine in Rijeka and the Faculty of Health Studies in Rijeka, where she teaches as an adjunct full professor.

New literacy or addiction in the digital era: what can we learn and/or relearn?

From a psychiatrist's perspective, there are many challenges regarding the impact of the digital era on the human brain. Are we facing a new "digital normality"? Should some diagnoses be reframed?

From childhood to adulthood, we live with new digital "friends" and altered realities—does this permanently change or shape our brains? Is there a connection between pharmacogenic and non-pharmacogenic addictions? What works in terms of prevention, behavioral reshaping, and the potential benefits of digital well-being, if any?

What truly works? What can we learn—and what should we relearn?

Ana Kapor-Tomić, MD, psychiatrist

Ana Kapor-Tomić, MD, is a psychiatrist at Insula County Specialist Hospital with professional experience predominantly in the field of biological psychiatry. She was born in Sarajevo in 1986 and graduated from the Faculty of Medicine in Novi Sad in 2013. She completed her specialization in psychiatry in 2021 and has five years of independent clinical experience. Her work also includes experience with migrant populations, which is especially challenging in the psychiatric world.

Can AI replace a psychiatrist?

The author in her presentation outlines the potential advantages and risks of using AI in the treatment of mental disorders . Are diagnostic tools and therapeutic possibilities more precise? Is so-called countertransference avoided or is the therapeutic relationship, as the foundation of understanding, empathy, and the therapeutic process and treatment outcomes, still missing? In what way can AI help in the process and when does it pose a risk?

The author presents an interesting case report.

Marija Tomac Stojmenović, MD, internist

Marija Tomac Stojmenović, born in 1983 in Rijeka, is a specialist in internal medicine currently working at Insula County Specialist Hospital. She graduated from the Faculty of Medicine, University of Rijeka, in 2007 and completed her specialization in General Internal Medicine at the University Hospital Centre Rijeka in 2019. She has also undertaken postgraduate studies in General Internal Medicine at the Faculty of Medicine in Rijeka.

Dr. Tomac Stojmenović is actively involved in scientific and professional work, with several publications in peer-reviewed journals, particularly in the fields of cardiology and internal medicine cases.

The Influence of Mental Processes on Cardiovascular Pathophysiology
Or what mind does to a heart ...

Cardiovascular diseases (CVDs) remain the predominant cause of mortality worldwide, accounting for approximately 20 million deaths each year. In recent decades, increasing scientific attention has been directed toward elucidating the role of psychosomatic determinants in both the initiation and progression of cardiovascular pathology. Psychosomatic components of CVDs refer to the complex interplay between psychological states, emotional responses, and social environments with underlying physiological mechanisms, which collectively may elevate susceptibility to cardiac and vascular disorders.

Conditions such as chronic psychological stress, depressive disorders, and anxiety syndromes have been identified as significant contributors to the pathogenesis of cardiovascular abnormalities. These conditions may be conceptualized as disorders with psychosocial origins that manifest through measurable organic dysfunction.

A central role is attributed to the hypothalamic–pituitary–adrenal (HPA) axis, whose dysregulation is frequently associated with sustained exposure to stressors and psychological burden. Prolonged activation of this neuroendocrine system results in elevated secretion of glucocorticoids, particularly cortisol, which adversely affects immune competence, promotes pro-inflammatory states, and disrupts physiological homeostasis.

Clinically, psychosomatic influences may present as somatic symptomatology modulated by psychological factors, including chronic pain syndromes, gastrointestinal dysregulation, and respiratory disturbances. Importantly, persistent exposure to stress, anxiety, or emotional strain may precipitate or aggravate cardiovascular conditions such as arterial hypertension and ischemic heart disease.

Chronic emotional stress and anxiety perpetuate continuous activation of the sympathetic “fight-or-flight” response, characterized by increased secretion of catecholamines, notably adrenaline, alongside cortisol. Adrenaline induces tachycardia, peripheral vasoconstriction, and elevated arterial pressure, thereby imposing increased hemodynamic load on the myocardium and vascular system. Over time, these effects contribute to the development of hypertension and accelerate atherogenic processes.

Simultaneously, cortisol enhances inflammatory pathways, facilitating the formation and progression of atherosclerotic plaques within arterial walls. This pathophysiological cascade ultimately heightens the risk of acute cardiovascular events, including myocardial infarction, cerebrovascular insult, and other related disorders.

Sandra Blažević Zelić, MD, PhD

Sandra Blažević Zelić, MD, PhD, is a Croatian psychiatrist and academic affiliated with the University of Rijeka Faculty of Medicine and the Clinical Hospital Centre Rijeka. She graduated in medicine in 1996 and also holds a degree in psychology, reflecting a strong interdisciplinary foundation in mental health. She completed her specialization in psychiatry, followed by subspecializations in biological psychiatry and addiction medicine, and obtained her PhD in 2015. She was appointed Research Associate in 2018 and currently holds the academic title of Assistant Professor (Docent). Her professional career has been primarily based at the Clinical Hospital Centre Rijeka, where she has developed extensive clinical experience in psychiatry, particularly in biological psychiatry and substance-related disorders. Alongside her clinical work, she is actively involved in teaching at the University of Rijeka, contributing to courses in psychological medicine, psychiatry, and communication skills in both Croatian and English-language medical programs. Dr. Blažević Zelić has a well-established research profile, with publications in internationally indexed journals and participation in numerous scientific conferences and research projects. Her work focuses on schizophrenia, psychopharmacology, and the interaction between psychological and somatic factors in mental health. She is also engaged in professional organizations and contributes to the broader dissemination of psychiatric knowledge.

DIGITAL DEMENTIA

The lecture deals with the current topic of the impact of digital media on learning processes and academic success and will also discuss the fact that time spent on screens can lead to addiction, anxiety, and/or lack of socialization.

The lecturer will highlight the main signs of digital dementia, how to recognize it, and how to prevent it, and all of this will be supported by existing research on the subject, from a socially important field.

Jelena Radić Nišević, MD, PhD

Jelena Radić Nišević, MD, PhD, docent at the Department of Pediatric Neurology and Metabolism graduated from the Faculty of Medicine in Rijeka in 2006. Since 2014, she has been working as a specialist pediatrician at the Department of Pediatric Neurology and Metabolism, Clinic of Pediatrics, Clinical Hospital Center Rijeka, which is also the Reference Center of the Ministry of Health for epilepsy and convulsive disorders in developmental age.

In 2016, she completed a two-month training in neurological and neurodevelopmental disorders in children at Great Ormond Street Hospital in London. Since 2015, she has held a joint appointment at the Department of Pediatrics.

She obtained her PhD in 2017, and in 2019 she became a subspecialist in pediatric neurology. She has published multiple scientific papers in national and international journals and has actively participated in numerous domestic and international professional and scientific conferences, where she has received awards.

Early Symptoms and Treatment Outcomes of Neuronal Ceroid Lipofuscinosis Type 2: Croatian Experience

Late infantile neuronal ceroid lipofuscinosis type 2 (CLN2) is a rare neurodegenerative disease that typically occurs in children between the ages of 2 and 4, leading to epileptic seizures and progressive loss of language and motor functions. As the disease progresses, affected individuals usually develop blindness and ultimately die in late childhood.

Treatment with intracerebroventricular cerliponase alfa has been shown to slow the deterioration of motor and language functions compared to the natural course of the disease.

The aim of this lecture is to highlight the early symptoms of CLN2 that aid in early diagnosis and timely initiation of treatment in children with specific medical indications, as well as to identify medical contraindications for enzyme replacement therapy.

We will present the experience of the Clinical Hospital Center Rijeka, which is also the Reference Center for the administration of cerliponase alfa in our country.

Dr.sc. Marija Pranjić

Dr. Marija Pranjić is a postdoctoral researcher at the Laboratory for Cognitive Neuroscience at Harvard Medical School and at Boston Children's Hospital, and the course leader of Clinical Neuroscience of Music at Harvard. Her scientific research focuses on the neurocognitive mechanisms of neurodevelopmental disorders, with particular emphasis on the impact of music and auditory perception on early brain development, as well as on the biomedical application of music in neurological rehabilitation. Dr. Pranjić publishes in leading scientific journals, and her research is supported through prestigious international grants such as the GRAMMY Museum Grant, the MITACS Globalink Research Award, and Harvard's Interdisciplinary Mind Grant. Prior to completing her PhD in neuroscience and music at the University of Toronto, Dr. Pranjić earned a degree in piano performance from the Academy of Music in Zagreb and completed postgraduate studies in music therapy at Anglia Ruskin University in Cambridge (United Kingdom). Strongly engaged in education and science communication, Dr. Pranjić is the founder of the Med&X Accelerator initiative, where she leads programs of clinical and scientific training for Croatian students at prestigious hospitals and universities worldwide.

Neuroscience of Music: From the Laboratory to Clinical Practice

Over the past two decades, the field of music neuroscience has undergone significant development, connecting science and art while opening new possibilities in research, education, and therapy. Studies show that listening to and performing music involve the simultaneous activation of sensory, motor, and cognitive systems, making music a unique model for studying how the brain predicts, learns, and adapts to the environment from childhood to old age. This lecture provides an overview of current knowledge on the effects of music on the brain and their potential for biomedical application — from assessing neurodevelopmental risk to developing interventions based on music and rhythm. Through examples from clinical practice, it will demonstrate how findings from fundamental research can be translated into concrete approaches in diagnosis, education, and rehabilitation.

Eliša Papić, MD, neurology specialist

Eliša Papić completed his medical degree at the Faculty of Medicine in Rijeka (2011–2017).
He is a neurology specialist at the Department of Neurology, Clinical Hospital Center Rijeka, with a particular professional and research interest in neurodegenerative and paroxysmal disorders, as well as functional neurological disorders. He is also a PhD candidate at the Faculty of Medicine in Rijeka. Alongside his medical career, he is an award-winning writer with several novels and a musician. He is currently engaged in clinical work, research, and the creation of music, prose, and poetry.

Functional neurological disorders – from diagnosis to treatment

Functional neurological disorders (FND) are common conditions at the interface of neurology and psychiatry. Historically described as “hysteria” by Jean-Martin Charcot and later explored by Sigmund Freud, FND is now understood through neurobiological models emphasizing altered brain network function rather than structural disease. Despite advances, FND remains challenging. Patients often undergo extensive investigations and experience delayed diagnosis and stigma. Modern diagnosis relies on positive clinical signs, not exclusion. Features such as internal inconsistency, Hoover’s sign in functional weakness, and distractibility or entrainment in functional tremor support the diagnosis. Current models highlight abnormalities in attention, salience processing, and sense of agency, reflecting disrupted predictive coding. Treatment is individualized and multidisciplinary, centered on clear explanation of the diagnosis, physiotherapy targeting normal movement patterns, and psychological support when indicated.
Targeted interventions such as distraction techniques, recontextualization, and graded exercises can lead to significant and sustained improvement. Recognizing FND as a rule-in diagnosis with effective treatment strategies is essential for improving patient outcomes.

Marija Janev, MD

Marija Janev, MD has over three years of experience in family medicine. She obtained her medical degree at the Faculty of Medicine in Rijeka and completed part of her studies through an Erasmus exchange at Acibadem University in Istanbul. Her clinical background includes training in non-invasive cardiology at the Polyclinic for Cardiovascular Diseases “Sveti Nikola” in Korčula. During the COVID-19 pandemic, she volunteered at a respiratory intensive care unit in Rijeka, working with mechanically ventilated patients. She also completed volunteer work at Segev Clinic, an aesthetic dermatology clinic in Tel Aviv.

She worked in family medicine at the Health Center Samobor (Bregana) from December 2022 to February 2026 and is currently employed at the Health Center of Split-Dalmatia County.

She is fluent in English (C1) and has advanced proficiency in German, Dutch, and Italian (B2).

The visualization of movement

A personal lecture on neurological rehabilitation after an 8 cm glioblastoma diagnosis and urgent surgery in the temporoparietal region following a seizure. It describes the initial motor, sensory, and balance deficits, followed by a long phase of compensation and later intensive rehabilitation focused on re-learning everyday movements. The lecture focuses on neuroplasticity and the re-education of movement through manual therapy, highlighting the brain’s role in motor control and coordination. It explores how mirror neurons, visual feedback, and repetition of functional, familiar movements supported the recovery of gait and hand function.

It also discusses the use of mental imagery and movement visualization techniques to improve motor planning and reduce pathological movement patterns.

Josipa Kajić, MD, neurosurgery resident
Josipa Kajić is a third-year neurosurgery resident at the Department of Neurosurgery, University Hospital Marburg. She graduated from the Medical Faculty, University of Rijeka in 2023. In addition to her training, she is involved in academic writing and has contributed to projects in spine surgery. During her student years, she was actively engaged in neuroscience education, serving both on the organising committee and as president of NeuRi Congress, an experience that played a key role in shaping her future career path. She is particularly interested in making clinical reasoning more accessible to students and early-career clinicians.

What I've Learned About Recognizing Neurosurgical Emergencies (So Far)
Early recognition of neurosurgical emergencies remains one of the most time-critical challenges in daily clinical practice. While classical teaching emphasizes clear diagnostic criteria, real-world presentations are often less straightforward, and delays in recognition can result in irreversible neurological injury. In many cases, timely decision-making—including the indication for emergency surgical intervention—directly determines patient outcome. The concept that "time is brain" (and, by extension, time is spine) is not theoretical, but a daily clinical reality. This presentation reflects on early clinical experiences in identifying neurosurgical emergencies, focusing on pattern recognition, diagnostic uncertainty, and common pitfalls. Four representative conditions are discussed: epidural hematoma, malignant middle cerebral artery infarction, cerebellar hemorrhage, and cauda equina syndrome. These examples illustrate different mechanisms of neurological deterioration, including rapid intracranial mass effect, space-occupying ischemic edema, posterior fossa compression, and spinal canal compromise. Rather than providing an exhaustive review, the talk emphasizes how these conditions may initially present in subtle or misleading ways. Particular attention is given to early warning signs, the limitations of relying on single findings, and the importance of maintaining a low threshold for imaging and timely escalation.
Grounded in early training, this perspective highlights both structured frameworks and uncertainty in clinical decision-making, with the aim of supporting students and early-career clinicians in recognizing what cannot be missed.

Slavica Kovačić, MD, PhD, Assistant Professor at the Department of Radiology

Slavica Kovačić, MD, PhD, is a radiology specialist at the University Hospital Centre Rijeka and Assistant Professor at the Faculty of Medicine, University of Rijeka. She graduated from the University of Rijeka School of Medicine in 1996 and obtained her PhD in Biomedicine in 2017. She completed her radiology specialization in 2007, followed by subspecialty training in neuroradiology and interventional radiology.

In addition to her clinical work, she is actively involved in undergraduate and postgraduate teaching and has contributed to numerous scientific publications in radiology. She has extensive experience in organizing professional and scientific events, including serving as president of national and international congresses in interventional radiology, and is an active member of several national and European radiological societies.

Endovascular Treatment of Stroke with Mechanical Thrombectomy

Mechanical thrombectomy is a minimally invasive endovascular procedure used in the treatment of acute ischemic stroke, enabling rapid and effective removal of a thrombus from occluded cerebral arteries, particularly in cases of large vessel occlusion. Timely diagnosis and appropriate patient selection are essential for optimal outcomes, with a central role played by modern neuroimaging techniques. The presentation will include illustrative examples of non-contrast brain CT and CT perfusion imaging, as well as an overview of the thrombectomy procedure itself. In addition, key findings from landmark randomized clinical trials, including MR CLEAN trial, ESCAPE trial, DAWN trial, and DEFUSE 3 trial, will be presented to provide students with an understanding of the clinical relevance and evidence base supporting this treatment approach.

Hrvoje Šimić, MD, PhD, Assistant proffessor of Neurosurgery

Hrvoje Šimić, MD, PhD, is a neurosurgery specialist at the Clinical Hospital Center Rijeka and Full Professor at the Faculty of Medicine, University of Rijeka, where he serves as Chair of the Department of Neurosurgery. He graduated from the University of Rijeka School of Medicine in 2006, completed his neurosurgery specialization in 2015, and obtained his PhD in 2018. His clinical work focuses primarily on brain tumors and complex spine surgery, with additional experience in neurotrauma and pediatric neurosurgery. In addition to his clinical work, he is actively involved in undergraduate and postgraduate teaching. He has completed numerous international courses, training programs, and fellowships in neurosurgery, including advanced training in minimally invasive spine surgery and complex spinal procedures. He has authored and co-authored multiple scientific publications in the field of neuroscience and clinical medicine. For his scientific contribution, he was awarded the Best Young Scientist award in Clinical Science by the University of Rijeka. He is dedicated to continuous professional development, academic excellence, and advancing modern neurosurgical practice through clinical work, education, and research.

Why do we experience back pain?

Back pain is one of the most common health problems and does not arise from a single cause. It develops through a combination of changes in the spine and the way the nervous system processes pain.

This topic can be understood using the Kirkaldy–Willis model, which describes how the spine changes over time, and the role of the dorsal root ganglion, which can amplify pain signals when sensitized.

Together, these mechanisms explain why back pain can vary greatly between individuals and why symptoms do not always match imaging findings.

Back pain results from the interaction between structural changes and neural sensitivity, not from a single anatomical problem.

Mirza Pojskić, MD, neurosurgeon

Dr. Mirza Pojskic is a Privatdozent (Assistant Professor) and Teaching Coordinator at the Department of Neurosurgery, University Hospital Marburg, Germany. He completed his medical degree at the University of Sarajevo (2011), board certification in neurosurgery (2018), and habilitation (2023) in Marburg. He further specialized in neurocritical care and completed international fellowships in the US, France, and the UK.

With over 3,300 performed procedures, his clinical focus spans complex spine surgery, skull base surgery, neuro-oncology, and advanced surgical technologies (neuronavigation, augmented reality, robotics).

Dr. Pojskic has authored 76 scientific publications, 10 textbook chapters, and edited a Springer textbook on neurosurgical technology. He serves as Co-Chair of the WFNS Materials, Drugs and Devices Committee, an EANS examination board member, Visiting Professor in Sarajevo, and Vice President of the Association of Bosnian and Herzegovinian Physicians in Germany (BHÄG). He actively leads global neurosurgery training initiatives, including spine surgery courses across Africa.

Advances in Spine Surgery: From Navigation to Robotics, Augmented Reality, and AI-Assisted Decision Making- Clinical evidence, workflow integration, and future directions

Advances in spine surgery over the past decades have been driven by the progressive integration of imaging, navigation, robotics, augmented reality (AR), and, more recently, artificial intelligence (AI). This lecture provides a clinically grounded overview of how these technologies are currently applied, where they add value, and where their limitations remain. Neuronavigation represents the foundation of modern technology-assisted spine surgery, particularly in complex anatomy, deformity, and revision cases. Its accuracy, however, is highly dependent on image quality and registration, emphasizing that workflow remains a critical determinant of success. Building on this, robotic systems have been introduced to improve the reproducibility and precision of surgical execution, especially for pedicle screw placement. While robotic-assisted techniques consistently demonstrate high accuracy and reduced radiation exposure, their impact on clinical outcomes remains variable and largely indication-dependent. Augmented reality extends navigation by integrating imaging data directly into the surgeon's field of view, enhancing intraoperative orientation and workflow continuity. Its value is most evident in anatomically challenging scenarios and minimally invasive approaches, although it remains dependent on accurate imaging and is limited by anatomical shifts during surgery. The integration of intraoperative imaging and ultrasound offers potential solutions toward more dynamic navigation. Artificial intelligence currently plays a supportive role, primarily in preoperative planning, risk stratification, and pattern recognition. Despite rapid technical development, real-time intraoperative AI applications remain limited, and clinical implementation is constrained by data quality, validation, and regulatory considerations.

Overall, these technologies improve precision and standardization but do not replace surgical judgment. Their effective use depends on appropriate patient selection, structured workflows, and critical evaluation of their true clinical benefit. The future of spine surgery will likely be defined not by autonomy, but by the intelligent integration of technology into surgeon-led decision-making.

Iris Martinović, mag. pharm., body psychotherapy trainee
Iris is a body-oriented psychotherapist whose work is rooted in a deep curiosity about what lies beneath our thoughts, emotions, and behaviors. With a background in science and a degree from the Faculty of Pharmacy and Biochemistry, University of Zagreb, she naturally approaches people and their challenges by exploring underlying causes and deeper patterns. She completed a four-year training in body psychotherapy (practitioner level), accredited by the European Association for Body Psychotherapy and recognized by the Croatian Chamber of Psychotherapists, along with Propedeutics of Psychotherapy. She continues to deepen her work through ongoing education, supervision, and years of workshops focused on movement, body awareness, and emotional work. After working in a pharmacy, she realized that supporting people through conversation was the most meaningful part of her work, which led her to fully dedicate herself to psychotherapy. Today, she helps clients explore deeper patterns, release emotional blocks, and build a more balanced and connected relationship with themselves.

Burn out - catch it early
In this short lecture, we will explore what happens beneath the surface as we begin to slide toward burnout—often long before we recognize it ourselves. We will focus on identifying early warning signs, from subtle physical changes to emotional and cognitive shifts, and examine how patterns such as guilt and perfectionism can contribute to the process. Participants will gain insight into common underlying causes and leave with practical tools for early intervention and effective symptom management, supporting both wellbeing and resilience in demanding environments.
In the discussion, we will explore our relationship with fatigue and reflect on relatable experiences.

Marko Šestan, DR. SC, PhD, Docent at the Department of Histology and Embryology

Dr Marko Šestan is a Docent at the Department of Histology and Embryology, Faculty of Medicine, University of Rijeka, where he returned following postdoctoral training as an EMBO Fellow at the Champalimaud Foundation in Lisbon. He graduated in 2012 from the Faculty of Veterinary Medicine, University of Zagreb, and obtained his PhD in 2018 under the mentorship of Professor Bojan Polić.

He has been awarded several prestigious research grants, including the Acteria project (2021–2023), the EMBO Installation Grant (2026–2031), and the ERC Starting Grant (2025–2030), as well as multiple awards, notably the Croatian State Award for Science. He is the author of 13 scientific publications, including seven papers published in journals with an impact factor exceeding 20. His work has been cited 1,134 times, with an h-index of 11.

He serves as a course leader and actively contributes to teaching across several study programmes at the Faculty of Medicine, University of Rijeka.

Can the brain form memories of past infections?

Everyone has heard of Pavlov's, or conditioned, reflex. But can it also act on our brain during viral infections? In this lecture, I will introduce you to one of the mechanisms through which our brain can 'remember' a viral infection and help accelerate recovery.

WORKSHOPS

24th APRIL - 26th APRIL 2026

SYNESTHESIA: CONNECT THE UNCONNECTABLE

Katja Katavić1, Donna Karan1, Natalia Kučić2
1 University of Rijeka, Faculty of Medicine, Rijeka, Croatia
2 University of Rijeka, Faculty of Medicine, Department of Physiology and Immunology, Rijeka, Croatia
Presenting author: Katja Katavić, katjak.skole@gmail.com

Introduction
The key to strong clinical reasoning and differential diagnosis is the ability to perceive connections others overlook and recognize patterns before they fully unfold. Synesthesia, traditionally defined as cross-activation between sensory and associative cortices, demonstrates the brain's intrinsic tendency toward association and integration. While typically studied as a neurocognitive curiosity, it can also serve as a conceptual framework for enhancing learning, memory consolidation, and problem-solving in medical education. Exploring this principle allows learners to understand how associative thinking supports deeper clinical insight and knowledge integration.

Materials and Methods
The workshop is designed as an interactive, participatory session that combines a brief introduction to synesthesia with practical, experience-based learning. Participants take part in three structured, game-based activities using a digital platform to connect concepts, recognize patterns, and build associations. All responses are anonymous and displayed in real time, allowing participants to observe how ideas develop collectively in an open and supportive environment. Each individual is encouraged to follow their authentic approach, illustrating how future clinicians may differ in reasoning and problem-solving when faced with the same clinical scenarios.

Literature Review

The literature reviewed for this workshop indicates that synesthesia, beyond its perceptual aspects, illuminates how the brain integrates information, reinforces memory, and promotes innovative problem-solving. It also suggests that individuals with synesthetic tendencies demonstrate superior associative learning, efficient multisensory processing, and enhanced divergent thinking. Such neural flexibility highlights the potential of cultivating associative skills to strengthen reasoning, conceptual connections, and adaptability in medical education.

Conclusion
Viewing synesthesia as a broader principle of association may provide a useful educational model for strengthening clinical reasoning, memory integration, and interdisciplinary thinking. Encouraging students to recognize and develop their own associative pathways may improve depth of understanding particularly in an era characterized by information overload and fragmented attention.

Keywords:
Clinical reasoning; Cognition; Memory; Neuronal Plasticity; Synesthesia

“NeuroSuture: Advanced Surgical Suturing Techniques,,- Student Section for Neurosurgery

Rea Krmpotić1, Paulina Kušan1
1Faculty of Medicine, University of Rijeka, Croatia
Corresponding author: Rea Krmpotić, rea.krmpotic@gmail.com

Workshop

This advanced surgical suturing workshop is designed for medical students who wish to refine their practical skills and develop a deeper understanding of wound closure techniques. Organized and led by the Student Section for Neurosurgery, the workshop provides hands-on training with individualized guidance and continuous feedback.

Participants will practice advanced suturing techniques used in everyday surgical practice, including superficial, subcutaneous, and intradermal sutures. The focus will be on proper technique, tissue handling, knot security, developing fine motor skills, precision, and efficient instrument handling. In addition to the practical component, the workshop includes a brief theoretical introduction covering suture materials, needle selection, and indications for different closure techniques.

The aim of this workshop is to equip students with practical skills applicable to clinical settings, strengthen their confidence in performing surgical procedures, and encourage interest in neurosurgery through hands-on experience.

Keywords: Neurosurgery; Sutures; Surgical Instruments

Cognitive tests workshop – MiniCOG, MOCA, MMSE

Eliša Papić1,2, Vladimira Vuletić1,2
1Clinic for Neurology, Clinical Hospital Center Rijeka, Rijeka, Croatia
2Department of Neurology, Faculty of Medicine, University of Rijeka, Rijeka, Croatia

Introduction: Dementia encompasses a group of progressive neurodegenerative disorders characterized by decline in memory, executive function, language, and behavior. Its growing prevalence presents a major clinical challenge, particularly in early and accurate diagnosis, where symptom overlap and comorbidities often obscure the picture. Cognitive screening tools are central to initial assessment and clinical decision-making. In our practice at the Clinic for Neurology in Rijeka, evaluation is conducted within our Day Clinic, enabling comprehensive workup including standardized cognitive testing, laboratory analysis, lumbar puncture for dementia biomarkers, as well as multimodal neuroimaging (MRI, PET), neurophysiological studies (EEG, evoked potentials), and neuropsychological assessment.

Workshop Description: This workshop focuses on the practical application of commonly used cognitive screening tools, including the Mini-Cog, Mini-Mental State Examination (MMSE), and Montreal Cognitive Assessment (MoCA). Participants will gain hands-on experience in administering, scoring, and interpreting these tests, with emphasis on their strengths, limitations, and appropriate clinical use. The session will highlight common pitfalls, interpretation nuances, and strategies for efficient bedside cognitive assessment.

Conclusion: Accurate use and interpretation of cognitive screening tools are essential for early detection and differentiation of dementia syndromes. Strengthening these practical skills directly enhances clinical confidence and diagnostic precision.

Keywords
Cognitive Dysfunction; Early Diagnosis; Dementia; Mental status and dementia tests;Neuropsychological tests

"Everything You Need to Know About Traumatic Brain Injury" – RiStart

Lea Cofek1, Mia Kontuš1, Pulina Kušan1, Mara Paravić1, Sara Schubert1
1Faculty of Medicine, University of Rijeka, Croatia
Corresponding author: Lea Cofek; lea.cofek@gmail.com

Workshop

The workshop, "Everything You Need to Know About Traumatic Brain Injury," provides a comprehensive and accessible overview of traumatic brain injury (TBI), containing causes and effects. Participants will learn how TBIs occur, from mild concussions to more severe forms of brain trauma and explore the physical and cognitive changes that may result. The session highlights the importance of early recognition of symptoms and offers guidance on appropriate immediate responses and when to seek medical care.

In addition, the workshop introduces current approaches to treatment, rehabilitation, resuscitation and long-term management, emphasizing the role of multidisciplinary care and individualized recovery plans. The content is grounded in real-life examples and practical applications. By the end of the workshop, attendees will have a deeper understanding of TBI and feel more confident in diagnosing and managing TBIs.

Keywords: Diagnosis; Emergencies; Resuscitation; Traumatic Brain Injury

The Neural Code

Natalija Frančin[1], Ana Šmit[1], Ana Vučaj[1], Leona Ostojić[1], Marija Barbarić[1]
1 University of Rijeka, Rijeka, School of Medicine, Croatia
Corresponding author: Natalija Frančin, natalija.francin1@gmail.com

Workshop
The Neural Code is an interactive, case-based workshop designed to engage medical students in neuroradiology through a gamified escape room format. The activity combines principles of neuroscience, radiological interpretation, and clinical reasoning, encouraging participants to apply their knowledge in a dynamic and time-pressured environment. Students work in teams of five and are challenged to complete a series of structured tasks within 15 minutes. Each phase focuses on a different aspect of neuroimaging and neurological thinking. By combining radiology with interactive problem-solving, The Neural Code fosters teamwork, communication, and critical thinking. The workshop aims to bridge the gap between theoretical knowledge and practical application, while highlighting the importance of accurate image interpretation in neurological diagnosis. This innovative format demonstrates the potential of gamification in medical education, offering an engaging and memorable learning experience for students interested in neurology, neuroradiology, and clinical sciences.

Keywords: Diagnosis; Neurology; Radiology; Simulation Training

Mindfulness Workshop – An Introduction to Mindfulness in Clinical Practice and Everyday Life

Marija Šaravanja, Nikoleta Koljanin

Workshop:
The Student Section for Psychiatry and Psychological Medicine (SZMEDRI) is organizing a one-hour workshop at the NeuRi Congress dedicated to mindfulness as a psychological skill increasingly applied in clinical practice and mental health promotion. The workshop will include a brief theoretical introduction to the concept of mindfulness, its neurobiological and psychological foundations, and an overview of evidence regarding its effectiveness in reducing stress, anxiety, and depressive symptoms.
The emphasis of the workshop will be on experiential learning through guided mindfulness exercises, including focused breathing attention, the body scan technique, and a brief exercise in non-judgmental observation of thoughts and emotions. Participants will have the opportunity to reflect on their experience and discuss the potential applications of mindfulness techniques in everyday life, the academic setting, and future clinical practice.
The aim of the workshop is to introduce participants to the fundamentals of mindfulness and to encourage the development of self-regulation skills, emotional resilience, and stress prevention through simple, practical techniques.

How the Brain Becomes a Signal: From Neuronal Activity to Neurosurgical Monitoring

Iman Bećirčić, Electrical Engineering student

Workshop

This workshop explores how neuronal activity is transformed into measurable electrical signals and how these signals are used in modern clinical practice. It bridges neuroscience and engineering through the fundamentals of electroencephalography (EEG) and brain imaging (MRI/fMRI), showing how these methods capture both the timing and location of brain activity. Through intuitive explanations and examples, participants will see how these technologies are used in preoperative brain mapping, and neurosurgical decision-making, as well as how combining EEG and imaging is shaping the future of diagnostics.

HOW (IN)VISIBLE IS THE PROBLEM?
Epidemiological Indicators of Child and Adolescent Mental Health

prof. dr. sc. Zorana Kušević
doc. dr. sc. Dunja Jurić Vukelić

Workshop

Drawing on epidemiological data, this interactive workshop examines key indicators of child and adolescent mental health and how to interpret them in both clinical and public health contexts. The core of the session is a guided, participant-led exploration of psychosocial factors in students' own environments that may contribute to risk or resilience, and how these realities translate into what we observe, record, and measure. Particular attention will be given to the distinction between transient symptoms and clinically significant disorders, as well as to the shifting boundaries between typical adolescent development and psychopathology. We will also consider why some difficulties remain under-recognized in statistics (e.g., stigma, help-seeking patterns, access to services) and how these factors shape epidemiological trends. The session emphasizes cautious, developmentally informed reasoning when moving from population-level data to individual assessment, and includes brief case vignettes and audience participation.

SCIENTIFIC COMMITTEE

- prof. dr. sc. **Ingrid Škarpa-Prpić,**MD, PhD, co-president of the Scientific Committee
- dr. sc. **Bobana Samardžija**, co-president of the Scientific Committee
- prof. **Vesna Šendula-Jengić**, MD, PhD, psychiatrist
- **Josipa Kajić,** MD, neurosurgery resident
- **Mirza Pojskić,** MD, neurosurgeon
- **Igor Salopek,** MD, psychiatrist
- **Maja Juković**, mag. pharm. inv
- **Matea Kršanac**, univ. mag. biotech. in med.

24th APRIL - 26th APRIL 2026

ABSTRACTS

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BASIC NEUROSCIENCE

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OUTCOMES FOLLOWING PERINATAL ASPHYXIA: A ONE-YEAR STUDY AT CLINICAL HOSPITAL CENTER RIJEKA

Jelena Mrkić1, Dorotea Drašković2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Department of Pediatrics, Clinical Hospital Center Rijeka, Rijeka, Croatia

Presenting author: Jelena Mrkić, jelena.mrkic123@gmail.com

Introduction
Perinatal asphyxia is a leading cause of neonatal morbidity and mortality and is often associated with hypoxic-ischemic encephalopathy (HIE), with an incidence in Croatia of approximately 2–3 per 1000 live births. Timely recognition and intervention are crucial to reduce neurological impairment. This study aims to assess the incidence, clinical characteristics, and neurodevelopmental outcomes of newborns with perinatal asphyxia over one year at a tertiary care center.

Materials and Methods
A retrospective cohort study was conducted at Clinical Hospital Center Rijeka from April 2024 to April 2025. 29 newborns were included in the study.
Inclusion criteria were low Apgar scores, umbilical vein pH <7.1, need for postnatal resuscitation and clinical signs of HIE. Collected data included gestational age, umbilical vein pH, Apgar scores, resuscitation, HIE severity (according to the Thompson score), which ranges from 0–22, with higher scores indicating more severe encephalopathy, application of therapeutic hypothermia, cranial ultrasound findings and motor milestone assessment in first year of life.

Results
In the analysed cohort, the median gestational age was 38 weeks (range 36–40 weeks), with a mean umbilical vein pH of 7.0 (range 6.8–7.3). Mean Apgar scores were 5 at 1 minute and 7 at 5 minutes. Four infants (13.8%) required resuscitation at birth. Severe and moderate HIE developed in those 4 cases (13.8%). Three of them were treated with therapeutic hypothermia. Mild HIE was diagnosed in 25 infants (86.2%). Abnormal cranial ultrasound findings were observed in 7 cases (24.1%); 2 infants (6.9%) were from moderate/severe group and 5 (17.2%) were from mild asphyxia group. Signs of impaired motor function were observed in 11 infants (37.9%); 1 with severe psychomotor delay and 10 with asymmetry in postural development.

Conclusion
Although most infants were classified as mild HIE, over one-third showed impairment of motor function or abnormal findings on neuroimaging. These findings indicate that even mild HIE may be associated with clinically relevant neurodevelopmental abnormalities. Structured follow-up is therefore essential, particularly with early identification and targeted interventions such as physiotherapy and neurodevelopmental support. However, the results should be interpreted with caution due to the small sample size and retrospective study design.

Keywords:
Apgar Score; Asphyxia; Hypothermia, Induced; Hypoxia-Ischemia, Brain; Neuroimaging

ACUTE METHAMPHETAMINE EXPOSURE INDUCES SELECTIVE REORGANIZATION OF POST TRANSLATIONAL MODIFICATIONS ON HISTONE 3

Marta Medija1, Antea Marjanović1, Bruno Diklić1, Ana Filošević Vujnović1, Rozi Andretić Waldowski1
1 Faculty of Biotechnology and Drug Development, University of Rijeka, Croatia
Presenting author: Marta Medija, marta.medija@biotech.uniri.hr

Introduction

Methamphetamine (METH) addiction is a chronic, relapsing disorder characterized by persistent alterations in gene expression leading to maladaptive neuronal plasticity. Short-term METH exposure modifies neuronal transcription through epigenetic histone post-translational modifications (PTMs) without altering DNA sequence. Histone 3 (H3) PTMs are particularly sensitive to METH and regulate genes involved in synaptic plasticity and dopamine signaling, making it a prime target for quantitative analysis.

Materials and Methods

To analyze H3 PTMs after one and two exposures to volatilized METH (vMETH), histones were acid-extracted from male *Drosophila* heads. Twenty-seven methylation, acetylation and phosphorylation marks on H3 were quantified using enzyme-linked immunosorbent assays (ELISA). To further characterize global modification patterns, H3 was separated by gel electrophoresis, excised, and subjected to in-gel tryptic digestion, and analyzed by matrix-assisted laser desorption ionization – time of flight mass spectrometry (MALDI-TOF).

Results

Our results show that vMETH induces changes in methylation, acetylation, and phosphorylation patterns, which allows restricted gene activation without complete distruption of chromatin structure. Specifically, dynamic silencing marks (H3K9me1/2, H3K27me1/2) decrease, while H3K9me3 and H3K27me3 remain stable, indicating that certain genes become more accessible while inactive DNA is preserved. Reduced H3K4me1/3 and H3K36me2/3, alongside stable H3K4me2 and H3K36me1, suggest that genes are selectively activated at their promoters, with limited transcriptional elongation. Decreased K9ac and K14ac modifications indicate reduced baseline gene transcription, whereas stable K18ac and adaptive changes in K56ac suggest that chromatin remain flexible and capable of responding to signals. Increased H3S28 phosphorylation after both exposures is consistent with dopamine-dependent, CREB-mediated transcription. Furthermore, MALDI-TOF analysis revealed broader peptide peaks (2200–2400 m/z) following vMETH exposure, indicating additional modified H3 forms.

Conclusion

Acute METH exposure induces coordinated and selective reorganization of H3 modifications, consistent with activation of conserved dopamine-dependent transcriptional pathways, highlighting potential epigenetic targets involved in addiction.

Keywords:
AMPHETAMINE-RELATED DISORDERS; DROSOPHILA MELANOGASTER; HISTONES; METHYLATION; PROTEIN PROCESSING, POST-TRANSLATIONAL

Gut microbiota as a modulator of radiotherapy efficacy in gliomas and brain metastases

Tea Franjković1, Damir Vučinić2
1 School of medicine, University of Rijeka, Rijeka, Croatia
2 Special Hospital Radiochirurgia Zagreb, Sveta Nedelja, Croatia
Presenting author: Tea Franjković, teafranjkovic22@gmail.com

Introduction
Radiotherapy is a cornerstone of treatment for gliomas and brain metastases. However, treatment response remains heterogeneous and is influenced by tumor biology, immune status, and the systemic inflammatory milieu. Growing evidence indicates that the gut microbiota, through immune modulation and gut–brain axis signaling, may influence radiosensitivity and therapeutic outcomes in central nervous system tumors.

Materials and Methods
A structured narrative literature review was conducted using PubMed MEDLINE. The search strategy combined terms related to gut microbiota, microbiome, radiotherapy, glioma, and brain metastases. Original research articles, translational studies, and relevant review papers published in English were considered. Reference lists of key publications were manually screened to identify additional studies.

Literature Review
A total of 48 publications were reviewed, including clinical studies, preclinical experimental models, and high-quality review articles. Clinical evidence linking gut microbiota composition to radiotherapy response in brain metastases remains limited; however, the most relevant clinical study to date demonstrated a significant association between pretreatment gastrointestinal microbiome profiles and radiotherapy response in patients with lung cancer and brain metastases, suggesting a potential predictive role of the microbiome for local control and treatment efficacy. In gliomas, preclinical and translational studies indicate that gut microbiota composition and derived metabolites, such as short-chain fatty acids (SCFAs) and tryptophan metabolites, can modulate antitumor immunity, including TLR–MYD88–NF-κB signaling, AHR activation, and HDAC inhibition, as well as influence DNA damage response pathways relevant to radiotherapy.. Emerging data demonstrating microbial signals within primary and metastatic brain tumors further supports a biologically plausible interaction between the microbiota and the tumor microenvironment.

Conclusion
Current evidence suggests that gut microbiota may modulate the efficacy of radiotherapy in both gliomas and brain metastases. Nevertheless, significant heterogeneity in study design, microbiome assessment methods, and clinical endpoints limits translation into routine practice. Well-designed prospective studies with standardized radiotherapy outcomes and control of confounding factors are required to validate microbiota-based biomarkers and explore therapeutic modulation strategies.

Keywords: Brain Neoplasms; Glioma; Microbiota; Brain-Gut Axis; Radiotherapy

EFFECT OF M6A RNA MODIFICATION ON PSYCHOSTIMULANT-INDUCED BEHAVIOR IN THE DROSOPHILA MELANOGASTER

Nina Milotić1, Ana Filošević Vujnović1, Rozi Andretić Waldowski 1
1 Faculty of Biotechnology and Drug Development at the University of Rijeka, Croatia
Presenting author: Nina Milotić, nmilotic@gmail.com

Introduction

Epitranscriptomic modifications, particularly m6A RNA methylation, are the most abundant intracellular modification found in eukaryotic messenger RNA (mRNA). m6A RNA methylation is a reversible process controlled by coordinated activity of writers (methyltransferases), erasers (demethylases) and readers (RNA-binding proteins that interpret the modifications). Previous studies in vertebrate models have implicated m6A RNA in the regulation of gene expression within neural circuits associated with drug reward. However, the behavioral impact of m6A perturbation in invertebrate models, particularly in relation to psychostimulant-induced phenotypes, remains largely unexplored.

Materials and Methods

In this study, we investigated how disruption of m6A RNA deposition and recognition affects cocaine (COC)-induced behaviors in Drosophila melanogaster. We used Mettl3 null mutants (deficient in m6A deposition) and YTHDF ΔCAT mutants (defective in cytosolic m6A recognition), in comparison with wild-type (wt) controls. Behavioral responses to cocaine were assessed using two validated paradigms: FlyBong for locomotor activation and sensitization, and FlyCAFÉ (CApillary FEeding (CAFÉ) assay) for measuring voluntary preferential consumption (VPC).

Results

FlyBong assays revealed that YTHDF ΔCAT mutants, but not Mettl3 null mutants, exhibited reduced sensitivity to cocaine and failed to develop normal locomotor sensitization compared to wild-type flies. In contrast, FlyCAFÉ experiments demonstrated a significant reduction in COC VPC in both YTHDF ΔCAT and Mettl3 null mutants.

Conclusion

Our findings highlight distinct roles for m6A RNA modifications in modulating COC-induced behaviors: YTHDF-mediated cytosolic recognition is critical for acute responses, while both m6A deposition and recognition are necessary for long-term drug preference. Future work should focus on identifying specific m6A-regulated transcripts and their spatial-temporal translation dynamics to better understand how epitranscriptomic mechanisms shape addiction-relevant plasticity.

Keywords:
Drosophila Melanogaster; Feeding-Related Behavior; m6A RNA Modification; Motor Activity; Substance-Related Disorder

CIRCADIAN MODULATION OF SEIZURE OCCURRENCE IN EPILEPSY

Brozović Jakov1, Baljak Bruna1, Habek Nikola1,2
1 University of Zagreb School of Medicine, Zagreb, Croatia
2 Croatia Institute for Brain Research, Scientific Centre of Excellence for Basic, Clinical and Translational Neuroscience, Zagreb, Croatia
Presenting author: Jakov Brozović, brozović.jakov@gmail.com

Introduction
Epilepsy is among the most dynamic disorders in neurology characterized by episodes of abnormal neural excitability. A traditional view holds that seizures occur randomly, but evidence supports temporal organization in seizure occurrence.

Materials and Methods
A systematic review following PRISMA guidelines was conducted using the PubMed database for studies published between 2015 and 2026 with the keywords: “epilepsy”, “circadian “rhythm”, and “chronobiology”. Full-text human observational studies were included, while reviews, case reports, animal studies, studies lacking quantitative data on seizure-timing were excluded. In total, 43 studies were analyzed.

Literature Review
Circadian rhythms are mainly generated by the suprachiasmatic nuclei, where clock genes can regulate seizure threshold, but the underlying mechanisms remain incompletely defined. Hormones like cortisol and melatonin also modulate seizure occurrence within cycle.
Seizure susceptibility varies with sleep stage, with interictal epileptiform discharges increasing during NREM sleep, whereas REM sleep is inhibitive, and up to 90% of seizures occur within two hours of awakening.
The diurnal pattern of seizure occurrence is influenced by the type of epilepsy and onset zone, with generalized seizures occurring mainly during wakefulness and focal epilepsies showing distinct temporal patterns depending on the seizure onset zone. Studies show that frontal lobe seizures predominate during sleep (60-72%), temporal and occipital lobe seizures during wakefulness (65%), mesial temporal lobe seizures show bimodal peaks, while parietal seizures lack definite pattern. However, small cohorts and non-longitudinal monitoring limit understanding.
Literature proposes chronotype-based therapy as personalized approach, aligning treatment timing with individual circadian patterns to improve seizure control, with future strategies potentially including circadian cycle stabilization or targeting molecular circadian clock mechanisms.

Conclusion
Epilepsy exhibits temporal organization influenced by circadian rhythms, with seizure patterns varying according to epilepsy type. Chronotherapy-based therapeutic strategies and forecasting seizures based on epilepsy type may improve seizure control and optimize clinical outcomes.

Keywords:
Chronotherapy; Circadian Rhythm; Epilepsy; Seizure; Sleep

NEUROLOGY

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LATE NEUROBORRELIOSIS PRESENTING AS SUBACUTE BILATERAL UPPER LIMB PARESIS IN AN ELDERLY PATIENT

Natalija Frančin1, Anja Janković1, Tea Elez1, Niko Kirinčić1, Lari Gorup (mentor)1, 2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Center Rijeka, Department of Infectious Diseases, Rijeka, Croatia
Presenting author: Natalija Frančin, natalija.francin1@gmail.com

Introduction

Neuroborreliosis is a neurological manifestation of Lyme disease caused by *Borrelia burgdorferi* and occurs in approximately 10–15% of untreated infections. It typically presents with meningoradiculitis, cranial neuropathy, or headache. However, isolated progressive upper limb paresis without systemic symptoms is uncommon, particularly in elderly patients. Early recognition is essential because delayed diagnosis may result in persistent neurological deficits.

Case Report

A 78-year-old woman presented with a three-week history of progressive bilateral hand weakness and paresthesia, more pronounced on the left. Two months earlier, she had sustained a tick bite on the right upper arm followed by expanding erythema consistent with erythema migrans, treated with azithromycin for five days. She remained afebrile and denied headache. Neurological examination revealed reduced upper limb strength, with rapid dropping of the arms in antigravity position. Electromyoneurography demonstrated mild bilateral neurogenic changes at C8. Routine laboratory findings and inflammatory markers were normal. Prior serum testing from an external private laboratory showed positive *Borrelia burgdorferi* IgG (Western blot). Lumbar puncture at the Department of Infectious Diseases revealed marked hyperproteinorrachia (2583 mg/L) and pleocytosis (283 cells/μL; 86% mononuclear). Microbiological evaluation, including multiplex PCR and testing for *Treponema pallidum* and tuberculosis, was negative. Intrathecal anti-Borrelia IgG synthesis was confirmed (IgG index 21; positive >2), establishing late neuroborreliosis. The patient received four weeks of intravenous ceftriaxone according to guidelines, with additional doxycycline due to positive rickettsial serology. Consequently, complete neurological recovery occurred.

Conclusion

Neuroborreliosis should be considered in patients with unexplained progressive radiculoneuropathy, even in the absence of systemic signs. Diagnosis requires cerebrospinal fluid analysis with confirmation of intrathecal antibody synthesis. Early recognition and prompt, guideline-based antibiotic treatment are crucial to achieve complete neurological recovery and prevent long-term complications.

Keywords:
Borrelia Burgdorferi; Ceftriaxone; Paresis; Paresthesia; Spinal Puncture

ACUTE ISCHEMIC STROKE FROM PARADOXICAL EMBOLISM IN A YOUNG PATIENT WITH FORAMEN OVALE

Antea Grizelj1, Ivana Gladović1, Branko Malojčić1, 2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, University Hospital Center Zagreb, Zagreb, Croatia
Presenting author: Antea Grizelj, antea.grizelj@gmail.com

Introduction

Acute ischemic stroke in young adults is frequently classified as cryptogenic with no clear etiology. In up to one-third of these patients, a patent foramen ovale (PFO) can be detected. However, establishing a causal relationship is challenging due to its prevalence in the general population. PFO may facilitate paradoxical embolism, allowing venous thrombi to bypass the pulmonary circulation and enter the arterial system, representing a potential stroke mechanism in young individuals.

Case Report

A 26-year-old female was admitted for management of acute ischemic stroke. Neurological examination revealed sensorimotor aphasia, leftward head and gaze deviation, right facial paresis, and right-sided hemiplegia. Non-contrast computed tomography (CT) and CT angiography (CTA) revealed areas of ischemic demarcation in the left insular and opercular regions, temporal lobe, lentiform nucleus, and caudate nucleus due to “T” occlusion of the left internal carotid artery and left middle cerebral artery. Emergency reperfusion therapy, including endovascular mechanical thrombectomy, was performed with successful recanalization. Her medical history was notable for bilateral pulmonary embolism associated with left-sided deep vein thrombosis three years earlier and was prescribed chronic anticoagulation therapy with rivaroxaban. During hospitalization, CT pulmonary angiography revealed recurrent pulmonary embolism involving the left pulmonary artery. Medication adherence and appropriate dosing of rivaroxaban were verified. Extensive cardiological, neuroradiological and hematological evaluation, including thrombophilia testing, were performed to determine stroke etiology. A transcranial Doppler bubble test demonstrated a moderate right-to-left shunt consistent with a patent foramen ovale. In the absence of other stroke mechanisms, paradoxical embolism through PFO was considered a possible underlying cause.

Conclusion

This case highlights paradoxical embolism through PFO as a potential mechanism of stroke in young patients, particularly those with prior venous thromboembolism. The coexistence of pulmonary embolism and embolic large-vessel stroke despite ongoing anticoagulation emphasizes the importance of comprehensive etiological evaluation and optimized anticoagulant therapy and possible PFO closure for secondary stroke prevention.

Keywords:
Embolism, Paradoxical; Foramen Ovale, Patent; Ischemic Stroke; Pulmonary Embolism; Ultrasonography, Doppler, Transcranial

An unusual clinical course of Miller Fisher syndrome with three distinct episodes: a case report

Lucija Skejić1, Anđela Krišto1, Vana Košta1,2
1 University of Split, School of Medicine, Split, Croatia
2 University Hospital of Split, Department of Neurology, Split, Croatia
Presenting author: Lucija Skejić, skejiclucija@gmail.com

Introduction

Miller Fisher syndrome (MFS) is a subtype of Guillain-Barré syndrome (GBS) and is characterised by the triad of ophthalmoplegia, ataxia and areflexia, often following infection. The worldwide incidence of MFS is 1 per 1,000,000 and recurrent episodes occur in 10% of cases. The occurrence of more than two episodes is exceptionally rare.

Case Report

Previously healthy 34-year-old man presented to the Emergency department in 2003 with diplopia and gait instability, preceded by sore throat. In 2004, the patient had same episode with additional dyspnea and was hospitalized at the Department of Neurology and diagnosed with MFS. During both episodes, the patient was treated with intravenous immunoglobulins and achieved complete recovery. In January 2026, the patient presented with ataxia, visual disturbances, dysphagia, and paresthesias. Medical history revealed a sore throat and temperature 20 days prior to admission. Neurological examination showed mydriasis without light reflexes, bilateral abducens nerve palsy, paresis of upward vertical gaze, and partial ptosis of the right eyelid. The muscle strength was normal with generalized areflexia and paresthesias of the hands and feet. Gait ataxia was also present. Laboratory blood tests and brain computed tomography were normal. Magnetic resonance showed no infratentorial abnormalities but several supratentorial punciform subcortical lesions. A lumbar puncture was performed and showed albuminocytologic dissociation (white blood cells 2x106/L, proteins 627 mg/L). Serological analysis detected positivity for anti-ganglioside antibodies GM1, GT1a, GD1a, and GQ1b. Human Leukocyte Antigen (HLA) typing identified HLA-DRB1*04 and HLA-DRB1*10 alleles. Treatment with intravenous human immunoglobulin was initiated at a dose of 0.4 g/kg for five days, along with physical therapy. The patient achieved complete recovery.

Conclusion

Although GQ1b antibodies are specific for MFS, the significance of other antibodies is still unknown. Some studies showed an association between recurrent MFS and HLA-DR2, while further research is needed to evaluate other HLA alleles.

Keywords:
Ataxia; Autoantibodies; Immunoglobulins; Miller Fisher Syndrome; Ophthalmoplegia

ISCHEMIC SPINAL CORD INFARCTION WITH DELAYED MAGNETIC RESONANCE IMAGING FINDINGS; CASE REPORT

Petar Panjak1, Antonio Perišić1, Matea Starešinić1, Valentino Rački1,2, Zoran Tomić2
1 Faculty of Medicine Rijeka, Rijeka, Croatia
2 Department of Neurology, Clinical Hospital Center Rijeka, Rijeka, Croatia
Presenting author: Petar Panjak, ppanjak@student.uniri.hr

Introduction
Ischemic spinal cord infarction is a rare condition caused by occlusion in the arteries supplying the spinal cord (most commonly in the extravertebral arteries) along with embolic events and vascular malformations. Patients typically present with paresis or paralysis, sensory disturbances, and autonomic dysfunction. Symptoms may vary depending on the extent of spinal cord involvement. This case report describes a patient presenting with nonspecific acute tetraparesis and highlights the diagnostic challenges in identifying spinal cord infarction.

Case Report
A 52-year-old man presented to the emergency department with acute-onset numbness that initially affected the left side of the body and later progressed to the right. Neurological examination observed tetraparesis accompanied by paresthesias below the C6 level. The positioning-supination test and Mingazzini maneuver showed sinking of the arms and legs. Upon admission, an extensive radiological and laboratory workup was performed. Computed tomographic angiography (CTA) of the thoracoabdominal aorta, brain CT and CTA of intracranial arteries revealed no pathological findings, and coagulation tests were normal. Magnetic resonance imaging (MRI) of the cervical and thoracic spine demonstrated degenerative changes, without evidence of spinal cord involvement. The patient was subsequently hospitalized at the Department of Neurology, where cerebrospinal fluid analysis was performed. It revealed positive IgM serology for *Borrelia burgdorferi*, and doxycycline was given; however, without significant improvement. Two days later, repeat cervical spine MRI demonstrated newly developed linear confluent ischemic lesions, extending from C3 to Th1. Patient was given low-molecular-weight heparin and antiplatelet therapy, combined with active physical rehabilitation. With continued therapy, the patient demonstrated significant neurological improvement and was eventually discharged home.

Conclusion
Acute nonspecific tetraparesis represents a diagnostic challenge, especially when initial MRI findings are normal, as ischemic lesions may evolve and present over time. Therefore, repeated imaging and early clinical suspicion are crucial for timely diagnosis, improved prognosis and optimal recovery.

Keywords:
Cerebrospinal Fluid; Infarction; Magnetic Resonance Imaging; Paresis; Spinal Cord

INTRAMEDULLARY SPINAL CORD TUMOR MIMICKING LUMBOISCHIALGIA: A DIAGNOSTIC CHALLENGE

Anamarija Bećarević1, Ivan Barna1, Ema Purgar1, Ana Šestanj1, Tihana Gilman Kuric1,2
1 Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
2 Department of Neurology, University Hospital Center Osijek, Osijek, Croatia
Presenting author: Anamarija Bećarević, anamarija.becarevic1@gmail.com

Introduction

Low back pain with radicular symptoms is a common neurological complaint, most often associated with degenerative spinal disease. Because such findings are frequent on imaging, they are often assumed to explain lower limb weakness or sensory disturbances. However, rare intramedullary spinal cord lesions may present similarly, posing a diagnostic challenge and potentially delaying treatment. Recognition of atypical clinical features and careful clinicoradiological correlation are therefore essential.

Case Report

A 64-year-old male presented with acute low-back pain followed by progressive numbness and weakness of the right leg. He had no prior history of chronic low back pain or neurological deficits. Although lumbar magnetic resonance (MR) imaging showed multilevel degenerative changes, there was no significant nerve root compression to explain the progressive monoparesis and sphincter dysfunction. Despite symptomatic therapy, neurological deficits progressed, with right-sided monoparesis, sensory impairment predominantly affecting the right lower limb, and urinary retention. Neurophysiological testing showed absent somatosensory evoked potentials on the right and abnormal conduction on the left. Because clinical deterioration was inconsistent with lumbar imaging findings, further evaluation was performed. Thoracic MR imaging revealed a 10-mm intramedullary expansile lesion at Th11 with surrounding spinal cord edema and signs of hemorrhage. MR angiography excluded vascular malformations. Neurosurgical consultation indicated operative treatment, and the patient was referred for surgical management.

Conclusion

Intramedullary spinal cord tumors may initially mimic common lumbar syndromes and represent an important diagnostic pitfall. Progressive motor deficit, sphincter dysfunction and clinicoradiological mismatch should be considered red flags warranting extended spinal imaging. Early diagnosis enables timely neurosurgical treatment, which is crucial for preventing neurological deterioration and improving functional outcomes. Over-reliance on common degenerative findings may delay recognition of serious intramedullary pathology.

Keywords:
Clinical Alarms; Diagnosis, Differential; Low Back Pain; Paresis; Spinal Cord Neoplasms

INFLUENZA A-ASSOCIATED MILLER FISHER SYNDROME: EXPANDING THE SPECTRUM OF POST-VIRAL NEUROLOGICAL COMPLICATIONS

Lea Radočaj1, Matea Rabotegl, Iva Predovan1, Niko Jukić1, Sven Lasta2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, Clinical Hospital Dubrava, Zagreb, Croatia
Presenting author: Lea Radočaj, leaaradocaj@gmail.com

Introduction

Miller Fisher Syndrome (MFS) is a rare, acute paralytic neuropathy considered a clinical variant of Guillain-Barré Syndrome (GBS). It is characterized by a triad: ataxia, areflexia and ophthalmoplegia. MFS is typically an immune-mediated response triggered by a previous infection which leads to the production of antiganglioside antibodies that attack the peripheral nerves. While the prognosis is generally favorable with intravenous Immunoglobulin (IVIG) treatment, the diagnostic process can be complex.

Case Report

A 62-year-old male patient was admitted to the Neurology Department following a referral from the University Hospital for Infectious Diseases. Initially, he presented with symptoms of influenza A, including high fever, general malaise and a dry cough. Shortly after the onset of the viral illness he developed neurological symptoms. Upon neurological examination, the patient exhibited the classic signs of MFS. Notable findings included right eye ptosis and double vision when looking vertically, absent patellar and Achilles tendon reflexes on the right side, with diminished reflexes on the left side and positive sign of ataxia. Multislice computed tomography and Magnetic Resonance Imaging of the brain were normal. Lumbar puncture revealed albuminocytologic dissociation which is an indication of GBS/MFS. Electromyoneurography showed sensory neural lesions and prolonged distal latencies, compatible with acute polyradiculoneuritis. Interestingly, while antiganglioside antibodies were negative, the patient tested positive for *Borrelia burgdorferi* antibodies, although further testing is still in progress. The patient was treated with a 5-day course of IVIG. He also received oseltamivir due to the influenza A. Following the initiation of IVIG, the patient showed significant improvement. He was eventually stabilized, became afebrile, and was discharged for outpatient physical therapy and further follow-up.

Conclusion

This case highlights the importance of monitoring for neurological complications after common viral illness and also confirms IVIG treatement as the gold standard for stabilizing patients and accelerating recovery in GBS variants.

Keywords:
Ataxia; Immunoglobulins, Intravenous; Miller Fisher Syndrome; Ophthalmoplegia

Bilateral Facial Nerve Palsy as a Manifestation of Neuroborreliosis in a Child: A Case Report

Gabrijela Šoštarić1, Dorotea Živalj1, Maša Vujnović1, Martina Linić1, Ema Šlabek1,2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Department of Pediatrics, Mother and Child Care Hospital Rijeka, Rijeka, Croatia
Presenting author: Gabrijela Šoštarić, gabrijela.sostaric26@gmail.com

Introduction
Bilateral facial nerve palsy is rare in children, accounting for approximately 2% of all facial palsy cases. Unlike unilateral involvement, bilateral palsy often suggests an underlying systemic disease, most commonly neuroborreliosis. Early recognition is essential for prompt targeted therapy and optimal neurological recovery.

Case Report
An 11-year-old boy was initially treated for recurrent parotitis with oral cefpodoxime and metronidazole. During treatment, he developed right-sided peripheral facial palsy which, despite physical therapy, progressed to bilateral involvement. Clinical features included an inability to completely close the eyes and significantly impaired mimetic musculature function (House-Brackmann Grade IV). Upon admission, the patient was afebrile, without headaches or meningeal signs, and exhibited normal motor findings. Despite no history of tick bites, lumbar puncture confirmed neuroborreliosis. Cerebrospinal fluid analysis showed lymphocytic pleocytosis (672×10^6/L), elevated protein (1274 mg/L), and blood- cerebrospinal fluid barrier dysfunction. Serology confirmed neuroborreliosis with positive Western blot and intrathecal antibody synthesis. Cranial computed tomography was normal. Audiological evaluation revealed a present stapedial reflex on the right and an absent reflex on the left, with recovery after seven days. Due to a poor response to initial parenteral antibiotics and physical therapy, hyperbaric oxygen therapy (HBOT) was introduced. The patient completed a series of 10 HBOT sessions at 2.4 ATA, with a total of 60 minutes of oxygen breathing time per session, administered in a fractionated 20/5 protocol. Subsequently, the patient was discharged on oral doxycycline with continued rehabilitation. A one-month follow-up showed significant improvement in mimetic function, a symmetrical smile, and near-complete eyelid closure.

Conclusion
Bilateral facial palsy in children warrants urgent cerebrospinal fluid evaluation for Borrelia burgdorferi, even without a tick-bite history. Early targeted antibiotics combined with rehabilitation may result in favorable recovery despite severe presentation.

Keywords:
Cerebrospinal Fluid; Child; Facial Paralysis; Hyperbaric Oxygenation; Lyme Neuroborreliosis

HIRAYAMA DISEASE: HIGHLIGHTING THE ROLE OF CLINICAL AND ELECTROPHYSIOLOGICAL EXAMINATION

Ivan Barna1, Anamarija Bećarević1, Ema Purgar1, Ana Šestanj1, Tihana Gilman Kuric1,2
1 Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
2 Department of Neurology, University Hospital Center Osijek, Osijek, Croatia
Presenting author: Ivan Barna, ivan.barna1610@gmail.com

Introduction

Hirayama disease, or monomelic amyotrophy, is a rare motor neuron disorder characterized by progressive weakness and atrophy of the distal upper limb, caused by dynamic compression of the lower cervical spinal cord during neck flexion. The aim of this case report is to emphasize the importance of careful clinical examination, detailed history, electrophysiological testing and targeted imaging in providing an accurate diagnosis.

Case Report

We present a case of a 21-year-old man, who reported right distal upper limb weakness, and was initially managed under the suspicion of a right ulnar mononeuropathy. Despite physical therapy and after several months, the patient developed progressive atrophy in right-sided myotomes associated with thenar, hypothenar and interosseous musculature, all innervated by different nerves but same segments. Further examination showed no sensory dysfunction nor upper motor neuron damage. Needle electromyography demonstrated chronic denervation changes in thenar, hypothenar and interosseous muscles, consistent with segmental anterior horn cell lesion. Sensory and motor nerve conduction studies were within normal limits, indicating preserved peripheral nerve integrity and arguing against peripheral neuropathy or focal mononeuropathy. These findings, considering patient's age and gender, are characteristic of Hirayama disease. Cervical spine magnetic resonance imaging (MRI) with additional flexion imaging was performed, as required when Hirayama disease is suspected. MRI showed anterior dural shift in lower cervical segments, venous plexus enlargement and cord atrophy, confirming the diagnosis.

Conclusion

Hirayama disease is often misdiagnosed as a common neuropathy or radiculopathy. However, atrophy of muscles innervated by different peripheral nerves but arising from the same lower cervical segments, preserved sensation and normal nerve conduction studies together with a history of a young male with recent growth spurt and symptom worsening during neck flexion should raise suspicion of this condition. In such cases, cervical MRI in neck flexion is crucial for establishing the correct diagnosis.

Keywords:
Anterior Horn Cells; Magnetic Resonance Imaging; Motor Neuron Disease; Monomelic Amyotrophy, Spinal Muscular Atrophies of Childhood

DIAGNOSTIC CHALLENGE IN A CHILD WITH FRIEDREICH'S ATAXIA – CASE REPORT

Morena Sirotić1, Ana Vučaj1, Tiyya Selimović1, Krešimir Serdarušić1, Jelena Radić Nišević1,2
1Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2Department of Pediatrics, Mother and Child Care Hospital Rijeka, Rijeka, Croatia
Presenting author: Morena Sirotić, morena.sirotic7@gmail.com

Introduction
Friedreich's ataxia (FRDA) is the most common autosomal recessive ataxia of childhood, usually caused by a biallelic GAA repeat in the FXN gene. It presents with progressive ataxia, sensorimotor neuropathy, skeletal deformities, and frequent cardiac involvement. In about 4% of patients, FRDA is due to rare structural or sequence variants in the FXN gene in a compound heterozygous state, which may be missed on initial genetic testing and pose a diagnostic challenge. This case highlights the importance of careful neurological assessment and expanded genetic testing in children with progressive ataxia.

Case Report
A girl born in 2016 presented at the age of three with progressive gait disturbance. Her parents reported gradually worsening balance, frequent falls, and increasing difficulty climbing stairs. Neurological examination revealed a wide-based ataxic gait, positive Romberg sign, lower limb areflexia, tremor, impaired coordination (finger-to-nose and heel-to-shin tests), pes cavus, and scoliosis. Brain and spinal MRI showed no structural abnormalities. Electroneuromyography indicated sensorimotor neuropathy. During the diagnostic workup, hypertrophic non-obstructive cardiomyopathy with preserved systolic function was detected, further strengthening the suspicion of Friedreich's ataxia. Whole genome sequencing was performed but initially revealed no pathogenic variant. However, due to the typical neurological presentation, additional targeted genetic testing was undertaken. This confirmed a heterozygous GAA repeat expansion in the FXN gene along with a heterozygous structural deletion of exon 5 of the FXN gene, thereby establishing the diagnosis of Friedreich's ataxia caused by a rare molecular mechanism.

Conclusion
In children with progressive ataxia, normal neuroradiological findings and an initially negative exome analysis do not exclude Friedreich's ataxia. Rare FXN gene variants, present in about 4% of patients, require targeted genetic testing. Careful neurological evaluation and sustained clinical suspicion are essential for identifying rare genetic mechanisms of Friedreich's ataxia.

Keywords:
Friedreich-Like Ataxia; FXN protein, human; Gait Disorder, Neurologic; Hypertrophic Cardiomyopathy; Testing, Genetic

A RARE CASE OF EARLY-ONSET HEMIMEGALENCEPHALY COMPOUNDED BY PHARMACORESISTANT NEONATAL EPILEPSY AND ATRIAL FLUTTER

Ivona Vrbančić 1, Jana Vidić1, Lea Županović1, Ivan Lehman2, Nada Sindičić Dessardo1,3
1 University of Zagreb School of Medicine, Zagreb, Croatia
2 University Hospital Centre Zagreb, Department of Neurology, Zagreb, Croatia
3 University Hospital Centre Zagreb, Department of Neonatology, Zagreb, Croatia
Presenting author: Ivona Vrbančić, ivonavrbancic12@gmail.com

Introduction:
Hemimegalencephaly is a rare malformation of cortical development characterized by unilateral hemispheric enlargement and dysplasia, strongly associated with early-onset epilepsy and neurological deterioration. Cardiac rhythm disturbances have only rarely been reported in this context.

Case Report:
A male neonate was born at 38+6 weeks of gestation via cesarean section following a low-risk pregnancy (Apgar scores 10/10). At the fourth hour of life, he presented with sudden apnea and marked cyanosis, followed by recurrent left-sided myoclonic jerks and horizontal nystagmus. Due to oxygen desaturation (82%), supplemental oxygen was initiated, and the infant was admitted to the neonatal intensive care unit (NICU). Cranial ultrasound suggested a congenital brain anomaly, and subsequent magnetic resonance imaging with time-of-flight angiography confirmed right-sided hemimegalencephaly. Video-electroencephalogram demonstrated continuous high-amplitude epileptiform activity over the right hemisphere without contralateral generalization. The infant was subsequently transferred to the Department of Pediatric Neurology for further management. Despite multi-drug therapy (phenobarbital, levetiracetam, phenytoin, valproic acid, vigabatrin, midazolam) and a ketogenic diet, epilepsy remained pharmacoresistant. On the 49th day of life, the infant developed atrial flutter, which was treated with sotalol. Whole-genome sequencing from peripheral blood was negative. The patient is currently being evaluated for neurosurgical intervention.

Conclusion:
This case highlights the complexity of hemimegalencephaly compounded by pharmacoresistant epilepsy and atrial flutter. A shared pathogenic mechanism involving somatic mosaic activation of the PI3K-AKT-mTOR pathway affecting both cerebral and cardiac tissue is biologically plausible. As blood-based genetic tests may be negative in mosaicism, brain tissue analysis during surgery remains crucial for further pathogenesis elucidation.

Keywords: Atrial Flutter; Epilepsy; Hemimegalencephaly; Infant, Newborn

CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY

Dominik Lukičić1, Matea Marović1, Branko Mišel1,2
1 School of Medicine, University of Zagreb
2 Clinic for infectious diseases "Dr. Fran Mihaljevic"
Presenting author: Dominik Lukičić, lukicicdomi@gmail.com

Introduction

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an autoimmune disorder of the peripheral nervous system characterized by progressive or relapsing weakness and sensory dysfunction lasting longer than eight weeks. It is caused by immune-mediated demyelination of peripheral nerves, leading to slowed nerve conduction and impaired motor and sensory function. Treatment typically includes intravenous immunoglobulin (IVIG), corticosteroids, or other immunosuppressive therapies, and many patients respond well with early diagnosis and management.

Case Report

An 11-year-old female patient presented with undefined symptoms of weakness and poor muscle control of the extremities (mainly in the legs), throat pain, fever (38.3°C), difficulty walking, and pain that did not respond to daily analgesic therapy. Ten days earlier, she had received antibiotic therapy for a UTI, but the symptoms of weakness, pain, and loss of control persisted. A pelvic X-ray revealed inflammation of the SIJ, raising suspicion of sacroiliitis; however, MRI showed demyelination of the motor and sensory nerves, which added Guillain-Barré syndrome (GBS) or chronic inflammatory demyelinating polyneuropathy (CIDP) to the differential diagnosis. After extensive testing, it was determined that the disease affected the peripheral nervous system and was associated with positive immunoglobulins, pathological electroneuromyography (ENMG), and chronic pain with a good response to immunosuppressants. Once CIDP was diagnosed, continuous IVIG (intravenous immunoglobulin) therapy (2 g/kg over 2 days) was initiated during hospitalization. The illness persisted with pain and neurological symptoms, but without blood, CSF, or MRI abnormalities; therefore, corticosteroids were reintroduced. At present, the patient is asymptomatic and responding well to treatment.

Conclusion

An 11-year-old girl developed weakness after an infection. MRI showed demyelination, and tests confirmed chronic inflammatory demyelinating polyneuropathy. Anti-ganglioside IgM antibodies – whose pathogenicity depends on complement activation and ganglioside complexes – supported immune-mediated neuropathy. IVIG and corticosteroids led to remission, highlighting antibody-driven peripheral nerve dysfunction in GBS/CIDP spectrum disorders.

Keywords:
Autoantibodies; CIDP; Gangliosides; GBS; Peripheral Autoimmune Demyelinating Disease;

Logopenic Primary Progressive Aphasia as an Early Manifestation of Alzheimer's Disease

Ema Purgar1, Anamarija Bećarević1, Ana Šestanj1, Ivan Barna1, Tihana Gilman Kuric1,2
1 *Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia*
2 Department of Neurology, University Hospital Center Osijek, Osijek, Croatia
Presenting author: Ema Purgar, ema.purgar153@gmail.com

Introduction
Primary progressive aphasia (PPA) is a neurodegenerative disease characterized by a gradual decline in language and speech functions, while other cognitive domains remain relatively preserved. The logopenic variant of PPA (lvPPA) primarily presents with word-finding difficulties and is often underrecognized, as early symptoms may be misattributed to depression, stress, or normal aging.

Case Report
We present a 65-year-old male with a one-year history of progressively impaired speech production. The symptoms were primarily characterized by word retrieval difficulties, while speech comprehension and independence in daily activities remained preserved. There was no history of stroke, head trauma, or seizures. Neurological examination showed pronounced anomic pauses and reduced spontaneous speech. Comprehensive neuropsychological assessment (MMSE, ACE-R) revealed moderate cognitive decline with dominant language impairment and milder deficits in working memory and executive functions. Visuospatial functions remained intact. Orientation in time and space was impaired, atypical for early PPA but consistent with underlying Alzheimer's pathology. Family history was positive for similar cognitive symptoms in the patient's father. Magnetic resonance imaging (MRI) of the brain showed mild atrophy in the parenchyma with multiple subcortical T2 hyperintensities consistent with gliotic lesions of vascular etiology, without evidence of acute ischemia or expansive process. These findings do not explain the language impairment, supporting a neurodegenerative etiology. Based on medical history, clinical presentation, neuropsychological profile, and MRI findings, a diagnosis of lvPPA within early-onset Alzheimer's disease (AD) was established. Cholinesterase inhibitor therapy was initiated. Cerebrospinal fluid AD biomarkers were planned for diagnostic confirmation. They are expected to show decreased Aβ42 levels along with elevated t-tau and p-tau, a profile indicative of cerebral amyloid deposition and tau-mediated neurodegeneration consistent with the diagnosis.

Conclusion
This case highlights lvPPA as an early manifestation of Alzheimer's disease. While there is currently no cure, early recognition of language-led cognitive decline enables timely speech-language strategies intervention, appropriate patient counseling, and participation in clinical research on disease-modifying therapies.

Keywords:
Alzheimer Disease; Aphasia, Primary Progressive; Cholinesterase Inhibitors; Speech

WERNICKE ENCEPHALOPATHY IN A PATIENT WITH CLINICALLY QUIESCENT CROHN'S DISEASE

Lana Halle1, Katarina Dejanović2, Željka Belošić Halle3
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 School of Medicine, University of Zagreb, Zagreb, Croatia
3 Department of Gastroenterology and Hepatology, Clinical Hospital "Sveti Duh", Zagreb, Croatia
Presenting author: Lana Halle, lana.halle83@gmail.com

Introduction
Wernicke's encephalopathy (WE) is an acute neurological disease presenting with ophthalmoplegia, ataxia, and altered mental status caused by thiamine depletion. Thiamine is a vitamin that functions as a coenzyme responsible for glucose metabolism and therefore vastly affects the central nervous system dependent on glucose. Its deficiency is most commonly associated with chronic alcoholism and rarely occurs in patients with Crohn's disease (CD).

Case Report
The patient had a 15-year history of CD with poor response to multimodal therapy and multiple abdominal surgeries for strictures and fistulae. His last surgery was a right-sided hemicolectomy with ileotransverse anastomosis. This was followed by stenosis and ileus requiring further ileal and transverse colon resection with colostomy and ileostomy. He declined stoma reversal. Although clinical remission was achieved, endoscopy showed aphthous ileal ulcers with chronic histopathological changes. He refused maintenance therapy and remained malnourished and underweight due to his complicated disease course. In his forties, he presented to the emergency department with progressive fatigue, weakness, paresthesia, and painful cramps in the extremities. Previously, he had no history of vomiting, diarrhea, or infection. On examination, he was confused but cooperative, with carpopedal spasm, nystagmus, and absent Achilles reflexes. Laboratory testing revealed severe hypocalcemia and hypomagnesemia. Magnetic resonance imaging (MRI) of the brain revealed a hypointense signal in the splenium of the corpus callosum. His neurological status rapidly deteriorated to unresponsiveness, and he was admitted to the intensive care unit, where intravenous magnesium, calcium, and high-dose thiamine were initiated. Neurological symptoms improved within days, and he was discharged two weeks later in good mental and functional condition on maintenance thiamine therapy.

Conclusion
Wernicke's encephalopathy can occur in Crohn's disease due to malnutrition, even without alcoholism. Because symptoms are often atypical, early recognition and thiamine therapy are crucial to prevent irreversible neurological damage.

Keywords:
Crohn Disease; Magnetic Resonance Imaging; Malnutrition; Thiamine Deficiency; Wernicke Encephalopathy

KETOGENIC DIET AND EPILEPSY

Bruna Baljak1, Jakov Brozović1, Nikola Habek1,2
1 University of Zagreb, School of Medicine, Zagreb, Croatia
2 Croatia Institute for Brain Research, Scientific Centre of Excellence for Basic, Clinical and Translational Neuroscience, Zagreb, Croatia
Presenting author: Bruna Baljak, bruna.baljak11@gmail.com

Introduction
Epilepsy is one of the most prevalent neurological disorders. Despite pharmacotherapy being the first-line treatment, about 30% of patients remain drug-resistant. The ketogenic diet (KD) is one of the non-pharmacological therapies for refractory epilepsy.

Materials and Methods
A literature search was conducted in the PubMed database using the keywords “ketogenic diet”, “epilepsy”, “seizures”, and “metabolism”. Only literature published in the last five years was included, yielding 171 results.

Literature Review
The KD consists of a low-carbohydrate, high-fat, and moderate-protein intake, causing a metabolic shift from glucose to ketone utilization. Ketones act through multiple mechanisms, including modulation of neurotransmission; for example, they reduce glutamate release by inhibiting vesicular glutamate transporter 1. Furthermore, they improve mitochondrial function, reduce oxidative stress and neuroinflammation, and alter the gut microbiota. Conditions that strongly support KD induction are developmental and epileptic encephalopathies, such as Lennox-Gastaut syndrome, as well as metabolic diseases that cause epilepsy, including glucose transporter 1 deficiency and pyruvate dehydrogenase complex deficiency. Around half of patients experience more than a 50% reduction in seizure frequency, and a few randomized controlled trials report cognitive benefits in children and adolescents. However, approximately one-third of patients discontinue the KD, mainly due to side effects. Common short-term adverse effects include constipation and vomiting, while long-term risks involve hypoproteinemia, mineral deficiencies, and dyslipidemia. A modified KD with a less restrictive regimen and milder side effects contributes to a better quality of life and long-term adherence to therapy, but further optimization remains a challenge for the future. Evidence on the use of the KD in the elderly population is lacking in the current literature.

Conclusion
Evidence suggests that the KD has significant beneficial effects on reducing seizure frequency in patients with refractory epilepsy. Nevertheless, serious side effects limit the use of the KD and decrease long-term adherence.

Keywords: Diet, Ketogenic; Drug Resistant Epilepsy; Epilepsy; Quality of Life; Seizures

AUTOIMMUNE TEMPORAL LOBE EPILEPSY AND AMYGDALA ENLARGEMENT

Tin Trobić1, Korina Pervan1, Marko Šarić1, Željka Petelin Gadže2, Andreja Bujan Kovač2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, University Hospital Centre Zagreb and School of Medicine, University of Zagreb, Referral Centre of Ministry of Health of the Republic of Croatia for epilepsy, Affiliated Member of the ERN EpiCARE, Zagreb, Croatia
Presenting author: Tin Trobić, tin.trobic@gmail.com

Introduction
Amygdala enlargement (AE) is increasingly recognized as a distinct epileptogenic substrate within the spectrum of temporal lobe epilepsy (TLE). Its etiology remains remarkably heterogeneous, encompassing inflammatory, autoimmune, neoplastic, and structural pathologies. Among autoimmune causes, anti-glutamic acid decarboxylase 65 (anti-GAD) antibodies are associated with limbic encephalitis and drug-resistant epilepsy. Recent literature suggests that AE-associated TLE may represent a unique clinical subtype, often characterized by specific semiology and potential resistance to conventional anti-seizure medications (ASMs).

Case Report
We report the case of a 19-year-old male with no family history of epilepsy, who presented with focal seizures starting at age 13. The seizures were characterized by an epigastric aura, ascending paresthesias, derealization, and "dreamy state" phenomena, followed by impaired awareness, staring, perioral pallor, and postictal confusion sometimes evolving into bilateral tonic-clonic seizures. Interictal electroencephalography (EEG) demonstrated right frontotemporal epileptiform discharges. Over several years, multiple ASMs were trialed, including levetiracetam, carbamazepine, oxcarbazepine, clobazam, zonisamide, brivaracetam, and valproate; however, only partial seizure control was achieved. Serial magnetic resonance imaging (MRI) consistently revealed bilateral amygdala enlargement without further structural abnormalities. Due to clinical worsening in 2023, a comprehensive re-evaluation was performed. Serological testing identified a high titer of anti-GAD antibodies (>2000 IU/mL). Extensive diagnostic workup, including cerebrospinal fluid (CSF) analysis, oncological screening, and systemic autoimmune panels, remained unremarkable. Neuropsychological assessment indicated significant attentional deficits and reduced working memory. Following the diagnosis of anti-GAD-associated autoimmune TLE, immunotherapy with intravenous immunoglobulins was initiated. Despite a transient improvement lasting one year, seizure frequency subsequently increased, necessitating the planned administration of rituximab.

Conclusion
This case emphasizes the clinical significance of amygdala enlargement as a radiographic marker for anti-GAD-associated temporal lobe epilepsy. Recognizing AE as a potential indicator of autoimmune etiology is crucial for facilitating early diagnosis and the prompt initiation of targeted immunomodulatory therapy, which may improve long-term neurological outcomes in drug-resistant cases.

Keywords:
Amygdala; Autoantibodies; Drug Resistant Epilepsy; Epilepsy, Temporal Lobe; Glutamate Decarboxylase

SPORADIC CREUTZFELDT-JAKOB DISEASE – CASE REPORT

Anđela Krišto1, Lucija Skejić1, Vana Košta1,2
1 University of Split, School of Medicine, Split, Croatia
2 University Hospital of Split, Department of Neurology, Split, Croatia
Presenting author: Anđela Krišto, andela.kristo01@gmail.com

Introduction
Prion diseases, also called spongiform encephalopathies, are rare, fatal neurological conditions with the most common being Creutzfeldt-Jakob disease (CJD).They are characterized by rapidly progressive dementia, myoclonus and ataxia. While definite diagnosis can only be made through brain tissue testing, clinical criteria for the probable CJD include characteristic cerebrospinal fluid (CSF) findings (14-3-3 protein, S100 protein, Tau protein, Neuron-specific enolase and Real-time quaking-induced conversion (RT-QuIC)), electroencephalogram (EEG) periodic sharp wave complexes and magnetic resonance imaging (MRI) hyperintensities in the caudate nucleus and putamen or two or more cortical regions.

Case Report
A 75 year-old man with no prior history of neurological disease was admitted to the Department of Neurology with rapidly progressive cognitive decline, ataxia and dysarthria. An extensive diagnostic evaluation was conducted. MRI of the brain showed restricted diffusion in the left caudate nucleus and anterior putamen. Continuous EEG monitoring confirmed diffuse slowing with dysrhythmia and grouping of slower waves, findings consistent with encephalopathy. The complete autoimmune encephalitis panel was negative, and extensive laboratory workup results were in normal reference ranges. While awaiting results of CSF testing from the external reference institution, National Reference Center for Human Spongiform Encephalopathies in Göttingen, the patient was treated *ex juvantibus* with corticosteroids and intravenous immunoglobulins without improvement. Treatment was complicated by a urinary infection and myoclonus which resolved with antiepileptic therapy. Upon arrival of positive RT-QuIC and 14-3-3 results, alongside characteristic brain MRI, clinical and paraclinical findings, it was concluded the patient has Creutzfeldt-Jakob disease. Given that CJD is invariably fatal, symptomatic and supportive treatment was recommended.

Conclusion
This case highlights the challenge of early recognition of CJD due to its rarity and nonspecific symptoms. It underscores the importance of prompt diagnosis using EEG, MRI, and CSF findings that enables families to prepare emotionally and organize optimal supportive care given its invariably fatal course.

Keywords:
Ataxia; Brain Diseases; Creutzfeldt-Jakob Syndrome; Dementia; Prion Diseases

WHEN EPILEPSY SIGNALS CANCER: AN ANTI-HU PARANEOPLASTIC SYNDROME WITHOUT DETECTABLE MALIGNANCY

Livia Badurina1, Petra Bilosnić1, Ana Deškin1,Ivana Bašić1,2,3
1 University of Zagreb, School of Medicine, Zagreb, Croatia
2 University hospital Dubrava, Department of Neurology, Reference center of the Ministry of Health of the Republic of Croatia for preoperative treatment of patients with pharmacoresistant epilepsy, Zagreb, Croatia
3 J. J. Strossmayer University of Osijek School of Medicine, Osijek, Croatia
Presenting author: Livia Badurina, livia.badurina@gmail.com

Introduction
Paraneoplastic neurological disorders (PND) are a heterogeneous group of immune-mediated disorders associated with underlying malignancies, often characterized by specific onconeural antibodies. They typically present with acute or subacute neurological symptoms and may precede tumor detection. Diagnostic evaluation can be guided by the Paraneoplastic Neurological Syndrome-Care (PNS-Care) Score, which evaluates the clinical phenotype, antibody type, and presence of malignancy. Anti-neuronal nuclear antibodies type 1 (ANNA-1) are rare and most commonly associated with small-cell lung cancer (SCLC). ANNA-1–related syndromes most often present as limbic encephalitis or encephalomyelitis with cognitive and behavioral symptoms, while epileptic seizures as an initial manifestation are uncommon.

Case Report
A 48-year-old woman was admitted to the Neurology Clinic due to recurrent episodes of altered awareness and loss of consciousness. Her history included insomnia and migraine. She was diagnosed with focal epilepsy with impaired consciousness seizures. Over the following year, seizures increased despite antiseizure therapy, accompanied by cognitive, psychiatric symptoms, and transient paresthesia. Extensive neurological investigations, including brain MRI and cerebrospinal fluid analysis, excluded alternative diagnoses. ANNA-1 positivity was detected, confirming a PND diagnosis. Screening for underlying malignancy, including tumor markers and whole-body PET-CT, was negative. The patient was started on pulse corticosteroid therapy with methylprednisolone; further treatment will depend on her clinical response. She remains under close follow-up with planned periodic screening for underlying malignancy.

Conclusion
This case highlights the importance of recognizing PND as a potential cause of adult-onset focal epilepsy. Epileptic seizures are an uncommon initial manifestation of PND, particularly in the presence of ANNA-1. In this case, the clinical course and ANNA-1 positivity supported a PND diagnosis despite no detectable malignancy. Because paraneoplastic neurological syndromes may precede malignancy by several years, long-term screening for underlying malignancy is essential. Early recognition and follow-up are crucial to optimize outcomes and enable timely malignancy detection.

Keywords:
Cognitive Dysfunction; Epilepsy; Nervous System; Paraneoplastic Syndrome; Seizure

EARLY RECOGNITION OF NEURONAL CEROID LIPOFUSCINOSIS TYPE 2 AFTER INITIAL SEIZURE: A CASE REPORT

Antonia Mišić1, Marija Bunić1, Dora Gašparović1, Ena Ivanković1, Mia Damašek1,2
1 Medical faculty Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia
2 Department of Pediatrics, University Hospital Centre Osijek, Osijek, Croatia
Presenting author: Antonia Mišić, antoniamisic61@gmail.com

Introduction

Neuronal ceroid lipofuscinosis type 2 (CLN2) is a rare recessive neurodegenerative disorder caused by mutations in the *CLN2* gene, leading to deficiency of the enzyme tripeptidyl peptidase 1 (TPP1). The disease typically presents in early childhood with epileptic seizures, progressive language delay, deterioration of vision and motor disorders. The average time that passes between the first symptoms and diagnosis is approximately two years, which postpones treatment in the early stages with specific enzyme replacement therapy.

Case Report

A 3.5-year-old boy was hospitalized after experiencing his first generalized motor seizure with loss of consciousness. Upon admission, his physical and neurological examinations showed no abnormalities. Brain computed tomography revealed mild asymmetry of the cerebral hemispheres and lateral ventricles, as well as a small arachnoid cyst without clear clinical significance. On the third day of hospitalization, he experienced another seizure, semiologically identical to the first one. An electroencephalogram was performed on three different occasions, showing dysrhythmia in the temporoparietal and centrofrontal regions, more pronounced on the left side, without specific epileptiform elements. Psychodiagnostic evaluation revealed delayed speech and language skills. Brain magnetic resonance imaging demonstrated signs of early cerebellar atrophy, enlarged cerebrospinal fluid spaces, and reduced cerebellar parenchyma. Considering the child's age, seizures, delayed language development and cerebellar atrophy, CLN2 was suspected. A dried blood sample showed significantly reduced TPP1 enzyme activity and confirmed the suspicion. The diagnosis was finally confirmed by identification of a mutation in the *CLN2* gene, just two months after the first seizure, prompting the immediate start of therapy.

Conclusion

This case highlights the importance of considering CLN2 in children presenting with seizures and developmental delay. Early testing and confirmation allow timely initiation of the disease-specific therapy, which may slow down the progression of neurodegeneration and improve the outcome of the disease.

Keywords:
Enzyme Replacement Therapy; Language Development Disorders; Neuronal Ceroid-Lipofuscinoses; Seizures; Tripeptidyl-Peptidase 1

PICA SYNDROME – A LIFE THREATENING CONDITION MASQUERADING AS PERIPHERAL VERTIGO

Lea Županović1, Ivona Vrbančić1, Dora Vlašić1, Jana Vidić1, Boris Šimunjak2,3
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Otorhinolaryngology and Head and Neck Surgery, Clinical Hospital Sveti Duh, Zagreb, Croatia
3 School of Medicine, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
Presenting author: Lea Županović 1, lzupanovic1@student.mef.hr

Introduction
Acute vestibular syndrome (AVS) is characterized by the acute onset of continuous vertigo, typically accompanied by nausea, vomiting, and nystagmus. The underlying cause may be peripheral, such as vestibular neuritis, or central, most commonly due to posterior circulation stroke. Among these, infarction in the territory of the posterior inferior cerebellar artery (PICA) classically results in lateral medullary (Wallenberg) syndrome, characterized by a combination of vestibular, cerebellar, sensory, and bulbar deficits.

Case Report
A 54-year-old woman presented to the emergency department with sudden-onset vertigo characterized by a rightward leaning sensation, nausea, and vomiting that began in the morning. Initial non-contrast brain computed tomography (CT) revealed no acute abnormalities. Neurological examination showed no clear focal deficits, except for unidirectional horizontal-torsional nystagmus with a left-beating fast phase. Following referral to the otorhinolaryngology department, clinical examination demonstrated a positive head impulse test on the affected side, leading to suspicion of vestibular neuritis. Symptomatic treatment with thiethylperazine and intravenous methylprednisolone sodium succinate was initiated. On the following day, the patient reported persistent vertigo with newly developed dysarthria, dysmetria and right-sided weakness. Neurological reassessment prompted brain magnetic resonance imaging (MRI) and intracranial angiography, performed on the second day of hospitalization, which demonstrated an acute ischemic lesion in the right posterolateral medulla within the territory of the posterior inferior cerebellar artery, consistent with PICA syndrome. The patient was subsequently treated with dual antiplatelet therapy for secondary stroke prevention, along with continued symptomatic antiemetic treatment. Following the treatment, the patient's condition improved, and she was discharged in stable clinical condition.

Conclusion
This case emphasizes the importance of differential diagnosis in AVS, as cerebellar strokes can mimic peripheral vestibular disease. Early MRI should be considered in patients with persistent or evolving symptoms to prevent misdiagnosis and improve outcomes.

Keywords:
Nystagmus, Pathologic; Lateral Medullary Syndrome; Vertigo; Vestibular Neuronitis; Magnetic Resonance Angiography

REVERSIBLE BILATERAL THALAMIC MRI LESIONS IN AN ISOLATED EPILEPTIC SEIZURE: CASE REPORT

Sonja Palija1, Sara Mrkonjić1, Eliša Papić1,2
1 The Faculty of Medicine of the University of Rijeka, Rijeka, Croatia
2 Department of Neurology, Clinical Hospital Center of Rijeka, Rijeka, Croatia
Presenting author: Sonja Palija, sonja.palija3@gmail.com

Introduction
Incidental neuroradiological findings during reevaluation of patients with a single isolated seizure can present complex differential diagnostic challenges, particularly in the absence of clinical correlations. Bilateral thalamic and periaqueductal MRI lesions may suggest metabolic or neoplastic etiologies, including Wernicke's encephalopathy and low-grade glial tumors.

Case Report
A 70-year-old patient was followed after a single generalized seizure in 2019, treated with lamotrigine. Over six years, no seizure recurrence occurred, and multiple EEGs remained normal. In March 2025, prior to planned therapy withdrawal, brain MRI revealed bilateral hyperintensities in the posterior thalami, periaqueductal region, and mamillary bodies, without diffusion restriction, post-contrast enhancement, or perfusion abnormalities. Neurological examination was without abnormalities (MoCA 28/30), and the patient was asymptomatic. Given the lesion morphology, Wernicke's encephalopathy was considered in the differential diagnosis despite absent clinical signs, and vitamin B1 supplementation was initiated, with no radiological change. Due to persistent lesions and absent clinical correlations, a low-grade glial tumor was also suspected. The patient was presented at a multidisciplinary board, where biopsy and surgical options were discussed. Lamotrigine was gradually reduced and completely discontinued in June 2025. During subsequent follow-up, the patient remained asymptomatic, and a 2026 control MRI revealed complete regression of lesions, excluding neoplastic etiology. Interestingly, the temporal correlation between lamotrigine discontinuation and the spontaneous regression of MRI lesions raises the question of a potential, yet unproven, pharmacological effect of lamotrigine on these neuroradiological findings.

Conclusion
This case highlights that reevaluation after a single seizure may reveal unexpected MRI findings, emphasizing cautious interpretation in the absence of clinical correlates and independent consideration of epileptic syndrome and structural changes. It also illustrates how careful longitudinal follow-up can help prevent unnecessary invasive procedures, such as brain biopsy.

Keywords:
Epilepsy; Lamotrigine; Magnetic Resonance Imaging; Seizures; Thalamus

POSTHERPETIC AUTOIMMUNE ENCEPHALITIS IN AN INFANT - CASE REPORT

Olivia Perković1, Jelena Radić Nišević2,3
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinic of Pediatrics, Clinical Hospital Centre Rijeka, Rijeka, Croatia
3 Department of Pediatrics, Faculty of Medicine, University of Rijeka, Rijeka, Croatia
Presenting author: Olivia Perković, perkovic.olivia@gmail.com

Introduction
Neonatal infection with herpes simplex virus (HSV), especially type 2, may result in severe meningoencephalitis with permanent neurological damage. An autoimmune variant - anti-N-methyl-D-aspartate (NMDA) encephalitis, associated with a more severe disease prognosis, arises in a significant percentage of patients.

Case Report
A female term infant, born vaginally, was hospitalized at the age of 17 days due to fever and urinary tract infection, treated with cefotaxime. The next day, frequent convulsions alongside fever occur in the form of leftward gaze fixation, twitching of left limbs and face with hypersalivation, anticipating seizures. Midazolam, phenobarbitone, and levetiracetam were initiated. Initial blood laboratory findings were normal. Lumbar puncture was performed alongside empirical intravenous acyclovir therapy for three weeks, continued orally for six months. Multiplex polymerase chain reaction (PCR) of cerebrospinal fluid (CSF) confirmed HSV-2. The father reported recurrent skin lesions, corresponding to genital herpes, none visible in the mother. The initial brain ultrasound was normal. However, the initial brain magnetic resonance imaging (MRI) showed multiple embolic ischemic areas with subdural and intraventricular hemorrhage. A month later, a follow-up MRI described extensive bilateral postinflammatory encephalomalacia, dominantly in the right hemisphere, involving temporal, parietal, and frontal lobes, cortical hemosiderin deposits, and extraaxial hemorrhage. The initial electroencephalography (EEG) was abnormal. The infant required respiratory support and was placed on non-invasive ventilation (NIV). At the age of 3 months, the infant was transferred to the Fran Mihaljević Reference Center in Zagreb due to involuntary movements, and autoimmune encephalitis was confirmed with positive anti-NMDA antibodies (detected in the CSF and serum). Prompt immunomodulatory treatment was started, involuntary movements were reduced but severe encephalopathy with profound developmental delay persists.

Conclusion
Early recognition and prompt immunomodulatory therapy are crucial for limiting permanent neurological sequelae due to postherpetic encephalitis onset, alongside the significant importance of adequate heteroanamnesis in pediatric age.

Keywords:
Anti-N-Methyl-D-Aspartate Receptor; Convulsions; Encephalitis; Herpes simplex virus 2; Infant

CEREBRAL HEMORRHAGES AS A CONSEQUENCE OF CEREBRAL AMYLOID ANGIOPATHY – CASE REPORT

Ana Vučaj1, Morena Sirotić1, Tiyya Selimović1, David Bonifačić1,2, Ines Strenja1,2
1Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2Department of Neurology, Clinical Hospital Centre Rijeka, Rijeka, Croatia
Presenting author: Ana Vučaj, anavucaj4@gmail.com

Introduction

Cerebral small vessel disease (cSVD) is major public health problem, responsible for over 25% of ischemic strokes, most spontaneous intracerebral hemorrhages (ICH), and at least 20% of dementias. It is broadly classified into cerebral amyloid angiopathy (CAA) and non-amyloid cSVD associated with aging, hypertension or genetic factors. CAA is a leading cause of non-traumatic convexity subarachnoid hemorrhage (cSAH) and spontaneous lobar ICH in the elderly. In addition to severe neurological deficits following ICH, cSAH often presents with transient focal neurological episodes (TFNE), the so-called "amyloid spells," or acute headaches. Early diagnosis and a multidisciplinary approach are crucial for optimizing outcomes and secondary prevention.

Case Report

A 71-year-old man was admitted after sudden tingling and weakness in left arm lasting 15 minutes, consistent with TFNE. The symptoms spread to the left corner of the mouth, which subsequently drooped.The symptoms briefly recurred on arrival at the Emergency Department. Non-contrast brain CT revealed a fresh right supratentorial frontotemporal subarachnoid hemorrhage (SAH). One month earlier, he had been hospitalized for speech disturbance and headache due to a left temporo-occipital lobar ICH, when CAA had been diagnosed. On admission, he was conscious, with pre-existing sensorimotor aphasia and a suspected right homonymous hemianopsia, without new deficits. Digital subtraction angiography excluded aneurysm and arteriovenous malformation. Brain MRI confirmed a subacute left temporo-occipital ICH (41×28×29 mm), bilateral frontal extra-axial SAH with superficial siderosis, and severe microangiopathy (Fazekas grade 3), consistent with CAA. Laboratory findings were unremarkable except for moderately reduced renal function. During hospitalization, the patient underwent physical, occupational and speech therapy rehabilitation. At discharge, mild sensorimotor dysphasia and right homonymous hemianopsia persisted.

Conclusion

This case highlights complex CAA presentation with multiple hemorrhages and fluctuating symptoms without vascular malformations. Multimodal imaging is essential for diagnosis. Early rehabilitation and risk-factor control enabled good outcome, but follow-up remains necessary due to recurrence risk.

Keywords:
Aphasia; Cerebral Amyloid Angiopathy; Cerebral Hemorrhage; Cerebral Small Vessel Diseases; Intracranial Hemorrhages

PRRT2-Associated Infantile Epilepsy: From Focal Seizures to Genetic Confirmation

Katarina Ivezić1, Magdalena Borić1, Petra Guljaš1 , Lucija Klajn1, Mia Damašek1,2
1 Faculty of Medicine Osijek, J.J. Strossmayer University of Osijek, Osijek, Croatia
2 Department of pediatric, Division of Neuropediatrics, genetic, metabolic disease and endocrinology, Clinical Hospital Centre Osijek, Osijek, Croatia
Presenting author: Katarina Ivezić, katarinaivezic74@gmail.com

Introduction

Infantile epilepsy comprises a heterogeneous group of neurological disorders characterized by recurrent, unprovoked seizures and often presents a diagnostic challenge due to diverse etiologies and normal neuroimaging findings. Because this developmental period involves rapid brain maturation, seizures may influence neurodevelopmental outcomes. Among genetic etiologies, PRRT2-associated epilepsy is an important cause of self-limited infantile epilepsy, typically presenting within the first year of life with focal seizures and favorable outcomes. Advances in molecular diagnostics have improved identification of causes and prognostic assessment.

Case Report

At the age of six months, a previously healthy girl with an unremarkable perinatal history and normal early neurodevelopment presented with recurrent focal seizures. The episodes were characterized by impaired awareness with orofacial automatisms, eyelid fluttering, eye deviation, and perioral cyanosis. Brain MRI and cardiologic evaluation were normal. Initial EEG demonstrated focal abnormalities in the left frontocentrotemporal region without generalized epileptiform discharges, suggesting focal seizure onset. Treatment with levetiracetam was initiated with good tolerability and seizure control. A seizure recurrence occurred at 16 months of age, accompanied by transient focal EEG abnormalities, after therapy adjustment, stable seizure control was achieved. Follow-up EEG recordings showed no epileptiform activity. Genetic testing using an epilepsy gene panel identified a heterozygous frameshift variant in the PRRT2 gene. During follow-up until nearly four years of age, no further seizures were documented. Neurological examination remained normal and cognitive development was age-appropriate, with no signs of regression.

Conclusion

This case highlights the importance of recognizing the clinical features of focal seizures and early EEG changes in infancy, as well as the role of genetic testing in confirming the underlying etiology. PRRT2-associated early-onset epilepsy may follow a benign course with a favorable therapeutic response. Early molecular diagnosis contributes to better understanding of disease progression and facilitates treatment planning.

Keywords: Electroencephalography; Epilepsy; Genetic Testing; Infant; PRRT2 Protein

WHEN GRAVITY BECOMES A CHALLENGE: A DECADE-LONG CASE OF SEVERE AUTONOMIC FAILURE

Marko Roso1; Lara Rončević1; Tomislava Tretinjak1; Lara Uglik1; Sanja Bekić1, 2
1Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia
2Specialist Practice of Family Medicine dr. Sanja Bekić, Višnjevac, Croatia
Presenting author: Marko Roso, marko.roso8@gmail.com

Introduction
Neurogenic orthostatic hypotension (NOH) is a debilitating condition caused by autonomic dysfunction (AD), leading to recurrent episodes of syncope. Early symptom recognition, appropriate diagnostic methods, and exploration of potential treatment options are of utmost importance, especially when optimal management of this rare condition remains uncertain.

Case Report
We present a case of a 75-year-old woman with NOH caused by severe AD. In 2013, she started experiencing vertigo, decreased sweating, blurred vision, vomiting, and syncope after 3-4 minutes of standing. As a result, she was only able to walk 50 meters. Autonomic assessment confirmed severe impairment of adrenergic and cardiovagal function, while magnetic resonance imaging of the brain revealed gliotic changes. Transcranial color Doppler could not be performed during head-up tilt. However, supine measurements showed reduced flow in the right vertebral and both posterior cerebral arteries. Over the following years, her clinical course was complicated by marked blood pressure variability, ranging from 55/undetectable diastolic pressures to 190/112 mmHg. Subsequently, Holter monitoring documented a reverse-dipping pattern consistent with NOH complicated by hypertension. Therapeutically, midodrine, abdominal corset, and fludrocortisone partially improved mobility. In 2019, she experienced a subsegmental pulmonary embolism, after which anticoagulation was initiated. Meanwhile, further testing fulfilled the criteria for antiphospholipid syndrome. In the absence of additional findings, it was considered concomitant, although a contributory role could not be entirely excluded. Upon follow-up in November 2025, with only two brief syncopes and several presyncopes in the past year, she was able to walk indoors with a cane and sit for 30 minutes, requiring only outdoor assistance.

Conclusion
This case demonstrates that rare conditions such as NOH require a highly individualised, multidisciplinary approach. Proper symptomatic therapy was found due to the efforts of various physicians. Even though the underlying cause remained undetermined despite a decade-long extensive evaluation, the patient reported clinical improvement.

Keywords:
Hypotension, Orthostatic; Syncope; Antiphospholipid Syndrome; Pure Autonomic Failure

NEW-ONSET EPILEPSY AS A REVERSIBLE CAUSE OF COGNITIVE IMPAIRMENT – A CASE REPORT

Petra Bilosnić1, Ivana Bašić1,2,3
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, University hospital Dubrava, Reference centre of the Ministry of Health of the Republic of Croatia for preoperative treatment of patients with pharmacoresistant epilepsy, Zagreb, Croatia
3 J. J. Strossmayer University of Osijek School of Medicine, Osijek, Croatia
Presenting author: Petra Bilosnić, petra.bilosnic@gmail.com

Introduction
Cognitive dysfunction in the elderly is often attributed to neurovascular disease and neurodegenerative disorders. However, epilepsy is also frequent disease in the elderly in whom seizures often present atypically as brief episodes of confusion and memory lapses rather than overt convulsions. Unrecognized seizures can result in cognitive dysfunction and may mimic dementia.

Case Report
A 72-year-old woman presented in June 2025 with difficulties in recent memory and disorientation to time. She underwent an initial diagnostic work-up for cognitive disorder during which she experienced bilateral tonic-clonic seizure arising from sleep. This prompted prolonged video-EEG monitoring. During monitoring, thirty focal seizures with impaired consciousness were observed in a period of 24 hours. The semiology was subtle, presenting as short episodes of confusion, without prominent motor features, of which patient was unaware. EEG showed bilateral frontocentrotemporal dysrhythmia, more pronounced on the right, with ictal right frontotemporal lateralization. MRI showed no significant findings. Laboratory tests, including cerebrospinal fluid analysis, revealed no abnormalities. Cognitive testing MoCA (The Montreal Cognitive Assessment) indicated a mild cognitive disorder. Treatment with zonisamide (ZNS) was initiated. Three months later, the patient reported improvement in her memory and repeated video-EEG demonstrated reduction in seizure frequency. Therapy was switched to brivaracetam (BRV). After introduction of BRV additional seizure reduction was observed, with only one seizure over 36 hours of monitoring. The patient's memory is expected to further improve with seizure control.

Conclusion
A high seizure frequency disrupts the normal functioning of neuronal networks, which may impair the encoding of new information and memory retrieval. Each seizure is associated with a transient disturbance of consciousness, followed by postical confusion and memory impairment. This may prevent brain function recovery and lead to cognitive dysfunction, which highlights the importance of considering new-onset focal epilepsy as a reversible cause of cognitive dysfunction in elderly.

Keywords:
Cognitive dysfunction; Electroencephalography; Epilepsy; Seizure

AUTONOMIC DYSFUNCTION IN LATERAL MEDULLARY INFARCTION FOLLOWING VERTEBRAL ARTERY DISSECTION

Ivana Gladović1, Antea Grizelj1, Branko Malojčić1, 2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, University Hospital Centre Zagreb, Zagreb, Croatia
Presenting author: Ivana Gladović, ivanagladovic2@gmail.com

Introduction

Vertebral artery dissection (VaD) is a recognized cause of posterior circulation stroke that frequently involves the lateral medulla. Lateral medullary (Wallenberg) syndrome classically presents with vertigo, sensory deficits, bulbar symptoms, and cerebellar signs. Autonomic dysfunction in VaD may arise from direct injury to perivascular sympathetic fibers surrounding the vertebral artery or, indirectly, from ischemic involvement of descending sympathetic pathways within the lateral medulla. Although lateral medullary syndrome is well described, systematic autonomic evaluation is rarely performed in clinical practice, potentially leading to underrecognition of sympathetic dysfunction in these patients.

Case Report

A 59-year-old woman with a history of hyperlipidemia and infrarenal abdominal aortic aneurysm was hospitalized due to spontaneous left VaD. She presented with a sudden-onset left-sided headache and vertigo with rotatory nystagmus. CT angiography confirmed VaD without evidence of parenchymal brain lesions at initial presentation. During follow-up, she reported right-sided clumsiness, burning pain in the right leg, reduced pain and temperature sensation on the right side of the trunk and extremities, and increased sweating on the right side of the body. Neurological examination confirmed the sensory deficit, along with mild truncal instability on Romberg testing. Autonomic testing revealed orthostatic intolerance with marked blood pressure variability without compensatory heart rate increase. Cold stress testing failed to elicit an appropriate sympathetic vasomotor response. Computerized color telethermography (CCTT) demonstrated relative hypothermia of the right side of the body. Brain MRI subsequently demonstrated a minimal post-ischemic lesion in the left lateral medulla. With continued anticoagulation therapy and supportive management, the patient's neurological status gradually stabilized, with partial symptom improvement during follow-up.

Conclusion

This case highlights autonomic dysfunction as a clinically relevant but often underrecognized manifestation of lateral medullary infarction. In the context of VaD, targeted autonomic testing may reveal sympathetic disturbances not evident on routine neurological examination, thereby improving clinical recognition of brainstem stroke.

Keywords:
Autonomic Nervous System Diseases; Brain Stem Infarctions; Lateral Medullary Syndrome; Sympathetic Nervous System; Vertebral Artery Dissection

EARLY CENTRAL HYPERTONIA IN DISTAL ARTHROGRYPOSIS: A CASE SUGGESTIVE OF FREEMAN-SHELDON SYNDROME

Erik Šepić1*, Matej Raguž2, Tiyya Selimović2, Krešimir Serdarušić2, Jelena Radić Nišević2, 3
1 Specialist Office of Family Medicine Jadranka Karuza, Rijeka, Croatia
2 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
3 Clinic for Pediatrics, Clinical Hospital Center Rijeka, Rijeka, Croatia

Introduction

Freeman-Sheldon syndrome (FSS), or distal arthrogryposis type 2A, is a rare congenital disorder primarily characterized by craniofacial anomalies and distal contractures. Although traditionally considered a musculoskeletal condition, increasing evidence suggests significant neuromotor involvement. We present an infant with prominent neurological manifestations in the context of suspected FSS.

Case Report

A female infant was born at 41+6 weeks by caesarean section with a birth weight of 3560 g and an Apgar score of 9/10). Early neonatal adaptation was stable. Later, feeding difficulties and insufficient weight gain became noticeable. At four months of age, neurological examination demonstrated generalized hypertonia with distal predominance and a flexor-dominant posture (clenched fists with elbow flexion). Ulnar deviation of the wrists with digital contractures was observed. Spontaneous movements were reduced. Head control was poor, and gross motor milestones were delayed. Inspiratory stridor had been present since birth, raising concern for neuromuscular contribution to airway dysfunction. Neuroimaging revealed no structural brain abnormalities, and metabolic screening was unremarkable. No epileptiform activity was documented. Due to the presence of this disorders, a syndromic neuromuscular disorder was suspected. Genetic evaluation begun with an inspection of bodily features. Craniofacial deformities were observed and described: low-set earlobes, nose, and hairline; thick auricular helices; and a retracted mandible. A set of traits not typical for any particular genetic illness. Molecular genetic testing was initiated to investigate pathogenic variants associated with distal arthrogryposis, MYH3 mutations linked to FSS in particular. The results are pending.

Conclusion

This case emphasizes the importance of careful neurological assessment in infants presenting with distal contractures and early motor delay, even when neuroimaging and metabolic studies are normal. Prominent hypertonia, abnormal motor patterns, and feeding and airway difficulties may reflect underlying neuromotor dysfunction. Recognition of these features is essential to support a timely intervention and optimize developmental outcomes.

Keywords
Arthrogryposis; Genetic testing; Hypertonia; Motor skills disorders; Neuromuscular diseases.

DIAGNOSTIC CHALLENGES IN COMBINED ADEM AND TRANSVERSE MYELITIS: THE IMPACT OF A LANGUAGE BARRIER

Anđela Jurić1, Angela Kalpić1, Lucija Kovačić1 , Dora Komić1 , Krešimir Čaljkušić1,2
1 *School of Medicine, University of Split, Split, Croatia*
2 *Department of Neurology, University Hospital Centre Split, Split, Croatia*
Presenting author: Anđela Jurić, andela.juric002@gmail.com

Introduction

Acute disseminated encephalomyelitis (ADEM) is an inflammatory demyelinating syndrome that affects the white matter of the central nervous system multifocally, while transverse myelitis affects only the spinal cord. Both occurring simultaneously is a rare but serious condition that requires prompt recognition and treatment.

Case Report

A young Peruvian woman was admitted to the Department of Neurology due to an acute onset of paraplegia. Two days prior to admission, she experienced headaches, back pain, photophobia, and nausea. Taking her medical history was hindered due to a lack of Spanish speaking personnel, so English was used for communication. Getting her prior medical information was impossible due to lack of communication between institutions. Shortly after admission, she developed two generalized epileptic seizures. Urgent magnetic resonance imaging of the spine revealed hyperintense lesions in the cervical and thoracic spinal cord suggestive of transverse myelitis. Cerebrospinal fluid (CSF) analysis revealed marked pleocytosis (640/L) and proteinorachia (1775 mg/L). Empirical antiviral and antibiotic therapy were initiated. CSF cultures were sterile, and multiplex polymerisation chain reaction (PCR) was negative. Subsequent MRIs showed progression of lesions previously found in the thoracic spinal cord, and hyperintensity lesions supratentorial and infratentorial which are consistent with a differential diagnosis of, respectfully, transverse myelitis and ADEM. The patient underwent plasmapheresis cycles followed by intravenous immunoglobulin therapy. Physical rehabilitation was initiated, and antidepressant treatment was introduced after psychiatric consultation. Seizures did not recur, and her mental status stabilized. At discharge, her condition improved regarding her sensory loss, flaccid paraplegia and absence of deep tendon reflexes persisted. She was discharged with gastroprotection and anticonvulsants. Her rehabilitation continued at a tertiary neurology center.

Conclusion

This case shows the importance of a structured diagnostic approach in complex neuroinflammatory disorders and highlights the additional challenges posed by language barriers in obtaining accurate clinical information and ensuring timely management.

Keywords:
Acute disseminated encephalomyelitis (ADEM); Demyelinating disease; Neuroinflammation; Paraplegia; Transverse myelitis

HIPPOCAMPAL-SPARING WHOLE BRAIN RADIOTHERAPY IN TRIPLE-NEGATIVE BREAST CANCER WITH BRAIN METASTASES: THERAPEUTIC POTENTIAL

Matea Starešinić1, Petar Panjak1, Marta Starešinić2, Alojzije Košić1,3, Ivana Mikolašević1,3,4
1 University of Rijeka, Faculty of Medicine, Rijeka, Croatia
2 University of Rijeka, Faculty of Philosophy, Department of Psychology, Rijeka, Croatia
3 Clinical Hospital Centre Rijeka, Tumor Clinic, Rijeka, Croatia
4 Clinical Hospital Centre Rijeka, Department of Gastroenterology, Rijeka, Croatia
Presenting author: Matea Starešinić, staresinicmatea1@gmail.com

Introduction
Whole brain radiotherapy represents the standard therapy in the treatment of multiple brain metastases, its drawback is the high irradiation of the hippocampus, which leads to neurocognitive decline. In younger patients, it is necessary to preserve neurocognitive function, and for this reason hippocampal sparing is used through prior planning and calculation of the avoidance zone. Triple-negative breast cancer is an extremely aggressive tumor that metastasizes to the brain very rapidly and quickly leads to neurological complications.

Case Report
A 36-year-old female patient presented to the oncology outpatient clinic with newly developed neurological symptoms. She has a four-year history of triple-negative breast cancer, previously treated with neoadjuvant therapy and surgery. Over the past three months, she had been experiencing severe headaches, nausea, and increased somnolence, along with noticeable behavioral changes reported by family members. In light of the newly developed neurological symptoms, brain MRI was performed, revealing more than twenty secondary oval lesions of varying sizes distributed across both cerebral and cerebellar hemispheres. Following a multidisciplinary team decision, and considering the patient's young age, preservation of neurocognitive function was identified as a critical therapeutic objective. Whole brain radiotherapy with hippocampal sparing was planned, delivered at a total dose of 30 Gy in 10 fractions. Special consideration was given to treatment planning, including delineation of an avoidance zone around the hippocampus with 5 mm margins and a maximum dose below 16 Gy. After radiotherapy, systemic therapy with sacituzumab govitecan was initiated, and four cycles were administered. This treatment was selected due to its activity against extracranial disease and its ability to act across the disrupted blood-brain barrier, contributing to overall tumor burden reduction. Two months after completion of combined treatment, follow-up brain MRI demonstrated complete regression of multiple secondary lesions in both supratentorial and infratentorial regions.

Conclusion
Hippocampal-sparing WBRT may be considered in selected patients with metastatic triple-negative breast cancer to support intracranial disease management while addressing the importance of preserving neurocognitive function.

Keywords:
Breast Cancer; Hippocampus; Neoadjuvant Therapy; Radiotherapy

ANTI-NMDAR ENCEPHALITIS

Dora Komić1, Anđela Jurić1, Angela Kalpić1, Irma Nina Orlandić1, prim. Eugenija Marušić, dr.med.1,2
1 - University of Split, School of Medicine, Split, Croatia
2 - University Hospital of Split, Department of Pediatrics – Institute of Neurology, Split, Croatia
Presenting author: Dora Komić, dorakomic@gmail.com

Introduction

Autoimmune encephalitis refers to acute to subacute, progressive inflammation of the brain associated with antibodies against neuronal cell surface and synaptic proteins, most commonly being anti-NMDAR encephalitis. Complex neuropsychiatric symptoms are the most common presenting features. Early diagnosis and prompt immunotherapy treatment can be beneficial for the outcome.

Case Report

A seventeen-year-old girl was hospitalized for dysarthria and skin paresthesia. Upon admission, an extensive diagnostic evaluation was performed. Neurological examination revealed cortical language processing impairment, cognitive dysfunction, comprehension difficulties, palmar hyperhidrosis, and reduced gross muscle strength. Psychiatric manifestations or epileptic seizures were not present. The EEG was low-voltage and dysrhythmic, without epileptogenic elements, and the fundus was normal. Laboratory findings were normal except for decreased vitamin B12 and folate levels, which were corrected with supplementation. Cerebrospinal fluid analysis revealed pleocytosis, type 2 oligoclonal IgG bands, and negative multiplex PCR for common encephalitis and meningitis pathogens. Brain MRI revealed bilateral occipital peritrigonal hyperintensities suggestive of possible glial lesions. Serologically, the finding of intrathecal synthesis of IgG on EBV was also observed, but the clinical significance was assessed as questionable. Diagnostic work-up has been extended to rule out paraneoplastic syndrome. Finally, anti-NMDAR antibodies were detected in serum and cerebrospinal fluid, which confirmed the diagnosis of autoimmune anti-NMDAR encephalitis. Treatment was initiated with pulsed corticosteroid therapy, followed by oral corticosteroids and IVIG. Due to neurological deterioration, plasmapheresis with repeated IVIG was initiated, resulting in significant clinical improvement.

Conclusion

This case stands out because it presents anti-NMDAR encephalitis with an atypical presentation without dominant psychiatric symptoms and epileptic seizures. The importance of timely recognition of the disease and the need for detailed and extensive diagnostic examination is emphasized in order to prove the correct diagnosis and apply therapy for a clinically better outcome.

Keywords

Encephalitis, Anti-N-Methyl-D-Aspartate Receptor Encephalitis, Dysarthria, Paresthesia, Immunotherapy

IDIOPATHIC OPSOCLONUS-MYOCLONUS-ATAXIA SYNDROME IN AN INFANT- A CASE REPORT

Paulina Kušan1, Mirjam Lovaković1, Rea Krmpotić1, Igor Prpić1,2
1 University of Rijeka, Faculty of Medicine, Rijeka, Croatia
2 Clinical Hospital Center Rijeka, Department of Pediatrics, Rijeka, Croatia
Presenting author: **Paulina Kušan1**, pkusan@uniri.hr

Introduction
Opsoclonus-myoclonus-ataxia syndrome (OMAS) is a rare neuroimmunological disorder characterized by opsoclonus, myoclonus and cerebellar ataxia often accompanied by behavioral disturbances and developmental regression. In children, OMAS may occur as a paraneoplastic syndrome, primarily associated with neuroblastoma, as well as a postinfectious or idiopathic immune-mediated condition.

Case Report
A 10-month-old male infant was admitted to the pediatric neurology department because of tremorous movements. Two weeks before admission parents noticed behavioral changes, frequent crying, irritability, sleep-related jerks and agitation. Over the preceding three days opsoclonus, impaired visual fixation and mild photophobia occurred. The child was born at 35+2 weeks of gestation from an uneventful pregnancy. Developmental milestones at that point were appropriate for age. On examination, continuous blinking, opsoclonus, head tremor, generalized tremor after manipulation and truncal ataxia were observed. Electroencephalogram in awake and sleeping states and brain ultrasound were normal. Cerebrospinal fluid (CSF) analyses revealed pleocytosis with lymphocyte domination and elevated lactate levels. CSF glucose levels were normal. Autoimmune and infectious antibody panels were negative. Imaging studies, including neck and abdomen ultrasound, whole-body magnetic resonance and I-23 MIBG scintigraphy, excluded neuroblastoma. Initial treatment with high doses of corticosteroids had little effect so intravenous immunoglobulins and methylprednisolone were introduced and led to clinical improvement and a decrease in Mitchell-Pike OMAS score from 4 to 2. Maintenance therapy consisted of prednisone and symptomatic treatment for sleep disturbances. Over the following months, opsoclonus and myoclonus resolved but ataxia and sleep disturbances persisted. Gradual improvements in coordination and behavior were observed correlating with a decrease in OMAS score to 1-2.

Conclusion
This case illustrates a rare case of idiopathic non-paraneoplastic OMAS in infancy and highlights the importance of early diagnosis, malignancy exclusion and timely immunotherapy, while also reflecting limitations of current diagnostic protocols that rely largely on exclusion based diagnosis. Despite appropriate treatment some neurological and behavioral disorders may remain, emphasizing the need for continuous neurodevelopmental monitoring and long-term follow-up care.

Keywords:
Ataxia; Immunotherapy; Neuroblastoma; Neuroinflammatory diseases; Opsoclonus-myoclonus syndrome; Paraneoplastic syndrome

FROM DIAGNOSTIC CHALLENGE TO TARGETED THERAPY: DYSTONIA TYPE 30 IN PEDIATRICS; CASE REPORT

Mirjam Lovaković1, Paulina Kušan1, Rea Krmpotić1, Igor Prpić1,2
1Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2Clinical Hospital Center Rijeka, Department of Neurology and Pediatric Psychology, Rijeka, Croatia
Presenting author: Mirjam Lovaković, mlovakovic@uniri.hr

Introduction

Dystonia type 30 is a movement disorder characterized by involuntary muscle contractions resulting from the simultaneous activation of agonist and antagonist muscles. It may be presented as either focal or generalized and is often exacerbated by voluntary actions such as writing, walking, and other purposeful movements. The purpose of this case study is to highlight the importance of a thorough and accurate diagnosis.

Case Report

A 14-year-old boy presented to the pediatric neurology department in March 2023. with involuntary movements of the right hand. Neurological evaluation identified dystonic movements affecting the right hand, with no additional neurological deficits. Comprehensive initial workup including physical examination, laboratory studies, electromyography and nerve conduction studies (EMNG), and brain magnetic resonance imaging (MRI) revealed no abnormalities and were age appropriate. Family history was negative for dystonia or other movement disorders. Initial genetic testing for common dystonia-associated mutations was performed and no mutations were found. “Over the two following years”, the patient developed compensatory strategies, learning to write and eat with non- dominant left hand and play handball with affected right hand. The dystonic movements remained and were characteristically action-induced, being more prominent during voluntary hand use. In September 2025, the patient transferred care to another institution where next-generation sequencing (NGS) was performed. This revealed a rare heterozygous pathogenic variant in the VPS16 gene with incomplete penetrance. The mutation is known for causing dystonia type 30, which is characterized by early onset with median of 12 years. The patient was initiated on oral baclofen therapy at a starting dose of 25 mg in the morning, with potential for dose escalation to maximum of 125 mg daily based on clinical response. Despite therapy, no significant clinical improvement was observed in following months.

Conclusion

In conclusion, clinicians should follow their clinical instinct and continue diagnostic evaluation when all aspects of the presentation suggest a particular diagnosis, even if early investigations are inconclusive. Timely identification of the underlying mutation enables earlier treatment, rehabilitation planning, and adequate genetic counseling for the family.

Keywords: Baclofen; Dystonia; Tremor; Sequence Analysis;

RESISTANT ESSENTIAL TREMOR AND NEW THERAPEUTIC OPTIONS

Mihaela Perković1, Petra Petravić1, Lana Papić1, Srđana Telarović1,2
Faculty of Medicine, University of Zagreb, Zagreb, Croatia1
Clinical Hospital Center Zagreb, Clinic for Neurology, Zagreb, Croatia2
Presenting author: Mihaela Perković, mihaela.perkovic99@gmail.com

Introduction

Essential tremor (ET) is a common movement disorder characterized by involuntary, rhythmic oscillations, most often action and postural. Standard therapy includes propranolol, primidone, clonazepam, anxiolytics and other symptomatic drugs. Despite the available options, some patients develop refractory disease. In such cases, advanced treatment methods such as Deep Brain Stimulation (DBS), magnetic resonance-guided focused ultrasound (MRgFUS) or stereotactic radiosurgery (Gamma Knife) and stereotactic radiotherapy should be considered.

Case Report

We present a 70-year-old male patient with long-standing ET and functional impairment. The patient has multiple comorbidities: chronic myeloproliferative neoplasm, MGUS, prior cerebrovascular accident, hypertension, disc protrusions, polyneuropathy, osteopenia and lumbosacral syndrome.

Numerous therapeutic attempts have been made over the years, including propranolol, primidone, clonazepam, gabapentin and clozapine, without satisfactory clinical effect. Propranolol and oxazepam were discontinued due to lack of clinical effectiveness. The Dopamine Transporter Scan (DAT-SCAN) findings were unremarkable.

Neurological status revealed mixed tremor, predominantly bilateral action tremor of the hands with a postural component. Tremor is also present at rest without rigidity and bradykinesia. The patient was unable to perform the Archimedean spiral adequately, indicating marked impairment.

Due to refractory symptoms, invasive methods were considered. DBS is the standard for severe, refractory ET with proven long-term efficacy. MRgFUS enables ultrasonic thalamotomy without craniotomy but requires strict selection criteria. Gamma Knife thalamotomy offers a non-invasive radiosurgical alternative, especially in patients with increased operational risk, while radiotherapy techniques are still being developed.

After multidisciplinary evaluation, invasive treatment options including DBS and stereotactic radiosurgery were proposed. However, the patient is currently not inclined toward invasive procedures. Pharmacological therapy was adjusted with the introduction of alprazolam, including initiation of anxiolytic therapy, with follow-up scheduled in 3-4 months.

Conclusion

This case illustrates the therapeutic challenges in elderly patients with refractory essential tremor and multiple comorbidities. Treatment decisions must be individualized, balancing efficacy, procedural risk and patient preference. In this case, although advanced interventions were proposed, the patient currently opts for conservative management with continued follow-up.

Keywords:
Deep Brain Stimulation; Essential Tremor; Movement Disorders; Radiosurgery; Stereotaxic Techniques

SUCCESSFUL TREATMENT OF VENTRICULITIS CAUSED BY EXTENSIVELY DRUG-RESISTANT ACINETOBACTER BAUMANNII WITH MULTI-ROUTE COLISTIN: CASE REPORT

Vita Pervan1, Dorotea Topić1, Marina Bralić2, Vladimira Vuletić2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Department of Neurology, Faculty of Medicine, University of Rijeka, Rijeka, Croatia
Presenting author: Vita Pervan, pervanvita13@gmail.com

Introduction

Intracerebral hemorrhage (ICH) represents a severe form of acute brain injury characterized by sudden bleeding within the cerebral tissue, most commonly due to arterial hypertension. ICH may be associated with intraventricular hemorrhage (IVH), which can result in hydrocephalus. In cases of hydrocephalus, the insertion of an external ventricular drain (EVD) permits CSF drainage and intracranial pressure monitoring but risks new hemorrhage, catheter misplacement, and infection.
This case report presents a patient with ICH and IVH who required ventriculostomy, along with the subsequent complications and multidisciplinary management.

Case Report

A 57-year-old patient was admitted to the emergency department due to sudden onset of left-sided weakness accompanied by repeated vomiting. The neurological examination revealed motor dysphasia, left homonymous hemianopia, left central facial palsy, and left-sided hemiplegia. His medical history revealed ischemic heart disease with prior myocardial infarction and coronary intervention, as well as arterial hypertension. The patient's level of consciousness gradually deteriorated, necessitating endotracheal intubation and mechanical ventilation. Urgent neuroradiological imaging showed intracerebral hemorrhage extending to the ventricles, and neurosurgical consultation led to aseptic insertion of EVD. By the fifth day of treatment, the patient's neurological status improved, and he successfully weaned off mechanical ventilation. Four days later, the patient developed a high fever, prompting samples of CSF and blood to be taken for culture, and the start of empirical IV antibiotic treatment. Consequently, the patient's condition deteriorated, necessitating re-intubation and mechanical ventilation. Following the positive identification of highly resistant Acinetobacter baumannii complex in both blood and CSF cultures, meropenem was substituted with colistin. Colistin was administered via multiple routes including intravenous combined with intraventricular administration, that led to a gradual improvement in the patient's condition.

Conclusion

This case highlights the significance of early recognition and prompt management of complications, particularly infections. It also emphasizes the essential role of timely coordinated multidisciplinary care in enhancing outcomes for patients with brain hemorrhage.

Keywords:
Acinetobacter baumannii; External ventricular drain; Cerebral hemorrhage; Intraventricular hemorrhage; Ventriculitis

DISARMING A TICKING TIMEBOMB: SIMULTANEOUS ARTERIAL DISSECTIONS WITH PSEUDOANEURYSMS IN A YOUNG WOMAN

Lara Rončević1, Marko Roso1, Dina Šišljagić1,2, Zdravka Krivdić1,3, Mihael Mišir1,4
1 Faculty of Medicine Osijek, J.J. Strossmayer University of Osijek, Osijek, Croatia
2 Department of Gynecology, Clinical Hospital Center Osijek, Osijek, Croatia
3 Department of Radiology, Clinical Hospital Center Osijek, Osijek, Croatia
4 Department of Neurology, Clinical Hospital Center Osijek, Osijek, Croatia
Presenting author: Lara Rončević, ronceviclara@gmail.com

Introduction
Cerebral arterial dissections (CAD) are rare and frequently unrecognized due to nonspecific symptoms despite multi-vessel pathology. CAD is sometimes complicated by luminal stenosis, occlusion, or pseudoaneurysm, posing a substantial risk of stroke.

Case Report
A previously healthy 40-year-old female presented with left eyelid ptosis and a three-day headache. Two weeks earlier, after bending toward her dog, she developed right-sided neck pain radiating to the forehead. Neurological examination revealed isolated left-sided Horner's syndrome without other deficits. Brain computed tomography (CT) was normal, while CT angiography (CTA) demonstrated 60% stenosis of the left internal carotid artery (ICA) in the C1 segment with pseudoaneurysm (4.9mm), responsible for Horner's syndrome, and stenosis of the right vertebral artery (VA) in the V3 segment with pseudoaneurysm (6mm), explaining neck pain. Three-dimensional time-of-flight magnetic resonance angiography and T1-weighted fat-suppressed sequences confirmed ICA and VA dissections. As endovascular intervention was deferred in the absence of stroke, selection between antiplatelet and anticoagulation therapy posed a major dilemma. She was initially treated with therapeutic doses of low-molecular-weight heparin. Based on vascular findings, bleeding risk, and age, therapy was switched to dabigatran. Follow-up CTA showed progression of left ICA stenosis (80%) and enlargement of the pseudoaneurysm (7×3mm), while the right VA remained unchanged. Anticoagulation therapy was continued due to clinical stability. Two months later, evaluation showed regression of left ICA stenosis (50%) and reduction of the right VA pseudoaneurysm. The patient remained stable, with mild ptosis due to Horner's syndrome, and continued anticoagulation therapy for stroke prevention. Switching to long-term antiplatelet therapy remains possible in due time, and stenting in case of embolic strokes.

Conclusion
While CAD posed a therapeutic challenge, individualized management with urgent anticoagulation proved crucial for vascular stabilization and stroke prevention. This case represents a favorable outcome despite the complexity and potential life-threatening nature of the underlying vascular pathology.

Keywords:
Anticoagulants; Carotid artery diseases; Horner syndrome; Stroke; Vertebral artery dissection

Atypical Presentation of Sporadic Creutzfeldt-Jakob Disease - A Case Report

Vanessa Lukas1, Anamarija Gerenčir1, Sanja Svalina2
1 Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
2 Department of Neurology, National Memorial Hospital Vukovar, Vukovar, Croatia
Presenting author: Vanessa Lukas, vanessa.lukas01@gmail.com

Introduction
Sporadic Creutzfeldt-Jakob disease (sCJD) is a rare, fatal prion-mediated neurodegenerative disorder occurring in 1–2 cases per million. It typically presents with rapidly progressive dementia, myoclonus, and cerebellar dysfunction. Characteristic findings include periodic sharp-wave complexes (PSWCs) on electroencephalogram (EEG) and cortical ribboning with basal ganglia hyperintensity on magnetic resonance imaging (MRI). We report a case of sCJD with atypical imaging findings that initially suggested cerebrovascular disease.

Case Report
A 77-year-old male with hypertension, ischaemic heart disease, and hypothyroidism presented with imbalance, psychomotor slowing, and evening disorientation. Examination revealed temporal disorientation, impaired vibration sense, distal hypoesthesia, and positive Romberg sign. Initial brain computed tomography, MRI and EEG findings were consistent with cerebrovascular disease, and vascular cognitive impairment was diagnosed. Treatment with donepezil, vortioxetine, and acetylsalicylic acid was initiated. Over three months, rapid deterioration occurred with severe insomnia, wandering, frequent falls, weight loss, behavioural disorganisation, and multifocal myoclonus. Upon hospitalisation, he was somnolent with incoherent speech, axial rigidity, paratonia, and prominent myoclonus. sCJD became the leading diagnostic suspicion given the rapid progression from mild cognitive symptoms to severe neurological dysfunction. Multifocal myoclonus, rapidly progressive dementia, and extrapyramidal signs fulfilled core clinical criteria for prion disease. Serial EEG showed diffuse dysrhythmia with lateralised changes without PSWCs. Real-time quaking-induced conversion (RT-QuIC) assay was performed to detect pathological prion protein, as it represents the most accurate sCJD biomarker. Both cerebrospinal fluid and nasopharyngeal samples were tested to maximise diagnostic sensitivity. RT-QuIC from both sources confirmed sCJD.

Conclusion
This case demonstrates that sCJD may be obscured by atypical imaging and EEG findings, making rapid clinical deterioration the key diagnostic clue. RT-QuIC proved essential in confirming the diagnosis, reinforcing the value of advanced biomarkers in diagnostically challenging cases. Despite its low incidence, sCJD warrants consideration in patients with unexplained rapid cognitive decline, especially when myoclonus and extrapyramidal signs are present.

Keywords:
Biomarkers; Cerebrovascular Disorders; Creutzfeldt-Jakob Syndrome; Prion Diseases

MULTIPLE SCLEROSIS-THE DISEASE WITH A THOUSAND FACES-CASE REPORT

Franka Radas1, Klaudija Duka Glavor2
1Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2Department of Health Studies, University of Zadar, Zadar, Croatia
Presenting author: Franka Radas, radas.franka@gmail.com

Introduction
Multiple sclerosis (MS) is a chronic autoimmune disease affecting the central nervous system, characterized by inflammation, demyelination, gliosis, and neuronal loss. This condition manifests with a wide range of neurological symptoms, bladder dysfunction and cognitive impairment. A variety of symptoms from the spectrum of psychiatric diseases may arise as a result of brain affection, which can occasionally be concealed and make it challenging to identify multiple sclerosis.

Case Report
A 30-year-old woman notices a deficit in working memory and learning curve with bladder incontinence. She was treated by a psychiatrist for bipolar disorder, with normal MSCT and MRI of the brain. Ten years later, MSCT of the brain describes lesions interpreted as vascular damage. The psychiatrist continues to treat depression. A few years later, she noticed facial asymmetry, spasm on the right, blinking of the eyes, and lower jaw. The neurologist indicated an MRI of the brain and cervical spine according to the demyelination protocol, which showed numerous round, oval, and irregular T2 and FLAIR hyperintense demyelination lesions throughout the brain and cervical spine. Lumbar puncture was performed. Cerebrospinal fluid electrophoresis with immunofixation reveals intrathecal synthesis of oligoclonal bands. Pulsed glucocorticoid therapy was applied, then teriflunomide treatment continued, along with depression therapy. A control MRI of the brain in 2023 showed disease progression, therefore ofatumumab was included. MS is currently in remission.

Conclusion
The patient has relapsing-remitting multiple sclerosis - unrecognized cognitive relapses, incontinence, and hemifacial spasms. Due to cognitive symptoms, multiple sclerosis was recognized late. This case reminds us of the importance of a proper history taking, a multidisciplinary approach in the treatment, while avoiding stigmatization of the psychiatric patient, for timely and correct treatment.

Keywords:
Multiple sclerosis; Neurobehavioral manifestations; Patient care team; Person with psychiatric disorders

SERIAL MRI ANALYSIS IN INFLAMMATORY CEREBRAL AMYLOID ANGIOPATHY – THE KEY TO DIAGNOSIS AND THERAPEUTIC RESPONSE

Matej Raguž1; Ines Strenja1,2; David Bonifačić1,2, Tea Tui Stanić1, Tiyya Selimović1
1 – Faculty of Medicine Rijeka, University of Rijeka, Rijeka, Croatia
2 – Department of Neurology, Clinical Hospital Center Rijeka, Rijeka, Croatia
Presenting author: Matej Raguž, mraguz79@gmail.com

Introduction
Inflammatory cerebral amyloid angiopathy (ICAA) is a rare, potentially reversible subtype of cerebral amyloid angiopathy characterized by an inflammatory response to β-amyloid deposition within cortical and leptomeningeal vessels. Clinically, it presents with subacute cognitive decline, focal neurological deficits, headache, and seizures. The aim of this report is to emphasize the importance of comparative analysis of serial MRI findings in diagnosing and monitoring therapeutic response in ICAA.

Case Report
A 67-year-old hypertensive man was admitted with acute left-sided weakness, headache, and vomiting. Neurological examination revealed left hemiparesis, homonymous hemianopsia, and ataxic gait. Brain MRI (04/2016) demonstrated numerous bilateral parieto-occipital cortico-subcortical T2* hypointensities, more pronounced on the right, consistent with microhemorrhages. Extensive asymmetric T2/FLAIR white matter hyperintensities with marked vasogenic edema and mild mass effect were present, without diffusion restriction or pathological enhancement. These findings supported the diagnosis of inflammatory CAA. High-dose corticosteroid therapy resulted in rapid clinical improvement. Follow-up MRI (05/2016) showed substantial regression of vasogenic edema and resolution of mass effect, while chronic microhemorrhagic foci persisted without new lesions. Long-term imaging follow-up (2020–2021) demonstrated stable distribution of microbleeds and persistent periventricular and subcortical white matter lesions (Fazekas 2), along with mild to moderate cortical (GCA 1–2) and medial temporal atrophy (MTA 1–2), without evidence of active inflammation. Neuropsychological testing confirmed mild cognitive impairment (MMSE 28/30) with stable course on donepezil. At the most recent follow-up in 2022, the patient demonstrated mild cognitive impairment (MMSE 28/30) without evidence of further decline compared to prior evaluations, and continued on the same therapeutic regimen.

Conclusion
This case underscores the value of serial MRI in diagnosing and monitoring ICAA. Early recognition and prompt immunosuppressive treatment may lead to significant clinical and radiological improvement and a more favorable functional outcome with reduced permanent neurological deficit.

Keywords:
Amyloid beta-Peptides; Cerebral Amyloid Angiopathy; Cognitive Dysfunction; Magnetic Resonance Imaging

DIAGNOSTIC CHALLENGES OF PRIMARY CENTRAL NERVOUS SYSTEM LYMPHOMA IN AN IMMUNOCOMPETENT PATIENT: A CASE REPORT

Marko Opačak1, Jakov Paić1, Zdravka Poljaković Skurić1,2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, University Hospital Center Zagreb, Zagreb, Croatia
Presenting author: Marko Opačak, marko29opacak@gmail.com

Introduction

Primary central nervous system lymphoma (PCNSL) is a rare and aggressive extranodal non-Hodgkin lymphoma confined to the central nervous system. Immune dysfunction is the only established risk factor, and PCNSL occurs more frequently in immunocompromised patients. Clinical presentation is nonspecific, and although neuroimaging plays a crucial role in the initial diagnostic evaluation, definitive diagnosis requires histopathological confirmation by brain biopsy. Approximately 85–90% of PCNSL lesions are located supratentorially, making infratentorial presentations uncommon and diagnostically challenging. We report a case of PCNSL in an immunocompetent man with an atypical infratentorial lesion.

Case Report

A 59-year-old man presented with a four-month history of gait instability with right-sided veering, followed by worsening neurological symptoms, including nausea, vomiting, diplopia, right-sided facial and upper limb paresthesia, and dysarthria. Emergency brain computed tomography revealed a hyperdense infratentorial parasagittal lesion adjacent to the right aspect of the fourth ventricle, suggestive of a neoplasm. Subsequent contrast-enhanced brain magnetic resonance imaging (MRI) demonstrated an irregular infratentorial lesion along the right aspect of the fourth ventricle, extending toward the medulla oblongata. The lesion was hypointense on T1-weighted images and heterogeneously hyperintense on T2/FLAIR sequences, with diffusion restriction and irregular contrast enhancement involving the cerebellar peduncles, accompanied by minimal perifocal edema. Cerebrospinal fluid analysis was unremarkable, and whole-body PET/CT showed no extracranial disease. Surgery was planned for diagnosis. A follow-up contrast-enhanced brain MRI performed three weeks later demonstrated disease progression, with newly developed supratentorial enhancing lesions. Stereotactic biopsy confirmed primary diffuse large B-cell lymphoma of the central nervous system. The patient's condition deteriorated, with impaired consciousness progressing to stupor, requiring intensive care management. Chemotherapy was not initiated due to advanced disease and poor clinical status. The patient died two months after admission.

Conclusion

This case highlights that PCNSL should be considered among the differential diagnoses of an infratentorial brain lesion of unknown etiology, even in immunocompetent patients. Early clinical suspicion of PCNSL, timely stereotactic biopsy of the lesion and histopathological confirmation remain essential steps in the diagnostic algorithm of this rare intracranial malignancy.

Keywords:
Primary Central Nervous System Lymphoma; Diffuse Large B-Cell Lymphoma; Magnetic Resonance Imaging; Stereotactic Biopsy

PAROXYSMAL KINESIGENIC DYSKINESIA TYPE 1 IN A PATIENT WITH LONG-STANDING EPILEPSY

Zora Ripić1, Eliša Papić2,3
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Center Rijeka, Clinic of Neurology, Rijeka, Croatia
3 Department of Neurology, Faculty of Medicine, University of Rijeka, Rijeka, Croatia
Presenting author: Zora Ripić, zripic@student.uniri.hr

Introduction
Paroxysmal kinesigenic dyskinesia type 1 is rare and often underdiagnosed movement disorder characterized by brief, recurrent episodes of involuntary movements triggered by sudden voluntary actions. It is most associated with autosomal dominant pathogenic variants in the proline-rich transmembrane protein 2 (PRRT2) gene and is frequently misdiagnosed, particularly in patients with coexisting epilepsy.

Case Report
We present a case of a 37-years old women with a long-standing history of focal epilepsy, first diagnosed in 1995. Her epileptic seizures were characterized by involuntary motor manifestations affecting the right side of the body and had been well controlled for several years, with no recent seizure activity. The patient relocated two years ago and was referred for neurological follow up. Since 2021, following a breast biopsy as a physically stressful event, she reported recurrent episodes of sudden twisting and dystonic posturing affecting the left arm and left leg, occasionally accompanied by repetitive tapping movements. The episodes occurred without loss of consciousness and were predominantly triggered by sudden movement or stress, with partial voluntary suppression. The involuntary movements were intermittent and more frequent during periods of increased stress, suggesting a possible role of the biopsy as an indirect precipitating factor. Unlike her previous epileptic seizures, these symptoms were left-sided and phenomenologically distinct, raising suspicion of a comorbid paroxysmal movement disorder. Electroencephalography following patient sleep deprivation demonstrated paroxysmal activity predominantly over the right frontoparieto-occipital regions. Brain magnetic resonance imaging revealed only small nonspecific subcortical white matter changes and a minor post-ischemic gliotic lesion in the right cerebellum. Neuropsychological assessment showed mild deficits in attention, alongside prominent psychosomatic features. Given the clinical dissociation between right-sided epileptic seizures and left-sided paroxysmal movements, genetic testing was performed and identified a heterozygous pathogenic variant in the PRRT2 gene, confirming PKD 1. Based on genetic findings and clinical phenotype, antiepileptic therapy was transitioned from lamotrigine to carbamazepine, following the gradual cross-titration protocol.

Conclusion
This case highlights the importance of recognizing PKD1 in patients with epilepsy and new lateralized motor symptoms, emphasizing the diagnostic value of genetic testing for targeted treatment.

Keywords:
Dyskinesias; Epilepsy; Epilepsies, partial; Movement disorders; Seizures

SEVERE GUILLAIN-BARRE SYDROME WITH PROLONGED VENTILATORY DEPENDENCE AND REMARKABLE FUNCTIONAL RECOVERY

Maria Perić1, Lana Papić1, Mihaela Perković1, Sara Drnasin2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2Department of Neurology, University hospital "Sestre milosrdnice", Zagreb, Croatia
Presenting author: Maria Perić, maria.peric@gmail.com

Introduction
Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness and possible respiratory and autonomic involvement. Several clinical and electrophysiological factors have been associated with worse outcomes, including advanced age, rapid progression to peak weakness, need for mechanical ventilation, severe weakness at presentation, axonal involvement on electrophysiological studies, and marked autonomic dysfunction.

Case Report
A 71-year-old man was admitted with rapidly progressive symmetrical limb weakness, distal paresthesias, and dysphagia. Neurological examination revealed severe flaccid tetraparesis with generalized areflexia. Cerebrospinal fluid (CSF) analysis demonstrated albuminocytologic dissociation with markedly elevated protein at 1.99 g/L. Electroneuromyography showed a predominantly demyelinating polyradiculoneuropathy with secondary axonal involvement, consistent with acute inflammatory demyelinating polyradiculoneuropathy (AIDP). Despite sequential treatment with plasmapheresis and intravenous immunoglobulins, the disease progressed to acute respiratory failure requiring intubation and mechanical ventilation. Ventilation was required for 75 days — unusually prolonged even in severe GBS — and tracheostomy was performed due to prolonged ventilatory dependence. The course was complicated by severe autonomic instability and infectious complications, including bacterial pneumonia and candidemia. After stabilization, the patient underwent prolonged multidisciplinary rehabilitation, including intense physiotherapy, respiratory therapy, and swallowing rehabilitation. He was successfully weaned from mechanical ventilation but discharged in a severely disabled condition with tracheostomy and percutaneous endoscopic gastrostomy (PEG) in place. Over 18 months of outpatient rehabilitation and family-supported daily exercises, gradual neurological improvement led to PEG removal, independent ambulation, and near-complete recovery of motor strength.

Conclusion
This case demonstrates that even in an elderly patient with GBS and multiple adverse prognostic features — prolonged mechanical ventilation, axonal involvement, severe autonomic instability, and infectious complications — meaningful long-term recovery remains achievable. Persistent severe disability and only partial recovery are typically expected after prolonged ventilatory dependence. Nevertheless, sustained improvement over 18 months led to near-complete functional independence. The case highlights the prolonged, rehabilitation-dependent trajectory of recovery in severe GBS and cautions against early prognostic pessimism.

Keywords:
Guillain-Barre Syndrome; Mechanical Ventilation; Rehabilitation; Polyradiculoneuropathy

RELAPSING-REMITTING MULTIPLE SCLEROSIS IN A PEDIATRIC PATIENT

Lucija Kovačić1, Dora Komić1, Angela Kalpić1, Irma Nina Orlandić1, prim. Eugenija Marušić, dr.med.1,2
1 – University of Split, School of Medicine, Split, Croatia
2 – University Hospital of Split, Department of Pediatrics – Institute of Neurology, Split, Croatia
Presenting author: Lucija Kovačić, kovaciclucija02@gmail.com

Introduction
Multiple sclerosis (MS) is a chronic autoimmune disease affecting the central nervous system, characterised by inflammation, demyelination, gliosis, and neuronal loss. While commonly diagnosed in young adults, about 3-10% of all cases affect children. Symptoms at presentation are generally similar to those in adults. However, pediatric MS often follows a relapsing–remitting course with a high relapse rate and frequently presents with cerebellar or brainstem symptoms.

Case Report
A twelve-year-old boy was transferred to the University Hospital of Split for further diagnostic evaluation of left-sided hemiparesis. Prior to transfer, laboratory findings and multi-slice computed tomography (MSCT) with angiography were performed, and no abnormalities were detected. Upon admission, an urgent brain magnetic resonance imaging (MRI) revealed nine lesions bilaterally, consistent with demyelinating changes. Cerebrospinal fluid analysis showed mild pleocytosis without proteinorachia, with a markedly elevated Kappa index. The presence of type 2 oligoclonal bands indicated intrathecal synthesis of immunoglobulins and an inflammatory process in the central nervous system. Based on the clinical presentation, MRI findings, and cerebrospinal fluid results, a diagnosis of relapsing–remitting multiple sclerosis was suspected. The patient was treated with high-dose methylprednisolone, alongside gastroprotection, as first-line therapy. Due to clinical deterioration, a follow-up brain MRI was performed, demonstrating progression of the lesion in the right internal capsule, consistent with ongoing disease activity despite treatment. Subsequent management included plasmapheresis followed by intravenous immunoglobulin administration. Vitamin D, folic acid, and B-complex vitamins were also administered. The patient's clinical status has shown a gradual improvement since then.

Conclusion
Pediatric-onset multiple sclerosis is a rare and diagnostically challenging disease, as its symptoms often overlap with other pediatric neurological disorders. This case underscores the importance of raising awareness of MS in children. Early diagnosis and prompt therapeutic intervention are crucial for improving long-term prognosis and minimising disability.

Keywords:
Hemiplegia; Magnetic Resonance Imaging; Multiple Sclerosis; Oligoclonal Bands; Plasmapheresis

INTRACRANIAL DISSECTION AS CAUSE OF A CEREBROVASCULAR INSULT IN YOUNG MALE, CASE REPORT

Angela Kalpić1, Anđela Jurić1, Lucija Kovačić1, Irma Nina Orlandić1, Krešimir Čaljkušić1,2
1 School of Medicine, University of Split, Split, Croatia
2 Department of Neurology, University Hospital Split, Split, Croatia
Presenting author: Angela Kalpić, angelakalpiccc@gmail.com

Introduction

Cerebrovascular insult in young patients is rare and often caused by atypical etiologies, such as intracranial or cervical artery dissection, patent foramen ovale, prothrombotic disorders, and substance abuse. All of which require prompt recognition and intervention. This case report presents a young patient with intracranial arterial dissection.

Case Report

A twenty-year-old male patient was admitted in 2024 to the Department of Neurology, University Hospital Split, after being found unresponsive. He underwent surgery for a left-sided ganglioma in 2017. Upon admission, he responded to painful stimuli. Left central facial paresis, hypotonia, and weakness in the left extremities were present. Emergency brain multi-slice computed tomography (MSCT) indicated a hyperacute ischemic stroke in the entire middle cerebral artery (MCA) supply area. Mechanical thrombectomy was unsuccessful, and thromboembolic etiology was excluded. Further evaluation confirmed intracranial dissection. Follow-up MSCT demonstrated the development of early uncal herniation. Urgent decompressive craniectomy was performed with intensive anti-edematous therapy and analgesia. Postoperatively, cerebral oedema gradually diminished, and the patient was taken off mechanical ventilation. Upon awakening, he was somnolent with left hemiplegia (1/5), a positive Babinski sign on the left and atypical on the right. Speech therapy assessment verified moderate motor dysphasia. Physical therapy resulted in improvement of motor strength to 3/5. Three months later, cranioplasty was performed. One and a half years after the stroke, spastic left hemiparesis (3/5), hemiparesthesia, and discrete asymmetry of the left oral commissure were still present. Mild dysarthria and structural epilepsy persisted.

Conclusion

This case highlights intracranial dissection as the cause of stroke in a young patient, where rapid decompressive craniectomy and anti-edematous therapy saved the patient's life and prevented further damage. Despite early diagnosis, a multidisciplinary approach, and extensive rehabilitation, the neurological deficits are still significant, emphasizing the need for long-term follow-up and ongoing support.

Keywords:
Brain Edema; Decompressive Craniectomy; Dissection, Blood Vessel; Infarction, Middle Cerebral Artery; Rehabilitation

THE ROLE OF SPEECH-LANGUAGE PATHOLOGISTS IN EARLY RECOGNITION OF BULBAR-ONSET ALS: A CASE REPORT

Jana Macinger1, Karolina Lice2
1 Faculty of Speech and Language Pathology, University of Rijeka, Rijeka, Croatia
2 Speech-Language Pathology Service for Diagnostics and Therapy, SUVAG Polyclinic for Hearing and Speech Rehabilitation, Zagreb, Croatia
Presenting author: Jana Macinger, jana.macinger@gmail.com

Introduction

Amyotrophic lateral sclerosis is a progressive neurodegenerative disorder affecting upper and lower motor neurons. In 20–30% of patients, it initially manifests with bulbar symptoms, including speech or swallowing impairment. As bulbar-onset ALS is associated with faster decline, early recognition is essential.

Case Report

A 78-year-old male was first evaluated by an otorhinolaryngologist in October 2024 due to progressive speech difficulties and voice changes. He was diagnosed with dysphonia and referred for neurological evaluation and speech-language assessment. Neurological consultation recommended further testing, no severe neurological disorder was suspected.
During speech-language evaluation in January 2025, articulatory movement assessment revealed reduced lingual range of motion. The tongue was unable to elevate to the upper lip, hard or soft palate. Protrusion was limited and unstable, lateral range of motion was reduced, bilateral fasciculations were visible at rest. Speech was mildly hypernasal with reduced vocal intensity. Phonation time was shortened. Articulatory precision began to deteriorate, most noticeably in the vibratory phoneme /r/. Increased salivation and early signs of swallowing difficulty were reported.
These findings suggested lower motor neuron involvement. Speech-language therapy was initiated prior to neurological diagnosis to slow symptom progression and preserve speech motor function, voice quality and swallowing ability. Further neurological evaluation was encouraged.
A comprehensive diagnostic work-up excluded alternative causes. Neurological examination revealed tongue and distal limb muscle atrophy, with right arm fasciculations and semipronation in the AG position. Electromyoneurography confirmed generalized motor neuron involvement, leading to ALS diagnosis in late 2025 and initiation of riluzole therapy.
Clinical changes were documented via video. As of March 2026, verbal communication is maintained using strategies taught in therapy, while swallowing ability remains unchanged.

Conclusion

This case highlights the role of speech-language pathologists in the early recognition of progressive bulbar symptoms of ALS. Careful clinical assessment can facilitate timely referral and diagnosis, enabling early multidisciplinary management.

Keywords:
Amyotrophic Lateral Sclerosis; Speech-Language Pathology; Dysarthria; Motor Neuron Disease; Speech Disorders

TINNITUS AND AUTOPHONY AS KEY DIAGNOSTIC CLUES IN SUPERIOR SEMICIRCULAR CANAL DEHISCENCE SYNDROME

Gabriela Večerić1; Ivana Ivošl; Zvonimir Popović2,3
1 Faculty of Medicine, University of Josip Juraj Strossmayer, Osijek, Croatia
2 Department of Neurology, University Hospital Center Osijek, Croatia
3 Department of Anatomy and Neuroscience, Faculty of Medicine, University of Josip Juraj Strossmayer, Osijek, Croatia
Presenting author: Gabriela Večerić, gabrielaveceric07@gmail.com

Introduction
Superior semicircular canal dehiscence syndrome (SCDS) is a rare condition that is caused by a thinning or absence of the part of the temporal bone overlying the superior semicircular canal. It's primarily congenital and often asymptomatic, but symptoms can develop afer infection or head trauma. This syndrome allows sound and pressure to pass abnormally into and out of the inner ear to other parts of the brain, causing hearing and balance problems. This case report highlights the importance of considering this syndrome in patients presenting with tinnitus and autophony.

Case Report
A 29 - year old male was admitted to the Department of Neurology with a one-year history of tinnitus in the right ear, intermittent vertigo, insomnia and a dull headache behind the right ear. Neurological examination revealed poor ocular convergence with bilateral involvement, particularly the right eye. During the diagnostic evaluation, brain magnetic resonance imaging (MRI) was performed, which revealed previous ischemic lesion in the right occipital lobe. Serological tests detected anti-treponemal antibodies but cerebrospinal fluid analysis was negative, thereby excluding active central nervous system involvement. Time of *Treponema pallidum* infection is unknown. Audiovestibular test battery was normal. After six months, the patient reported worsening of symptoms, including autophony. MRI showed no progression of the previously described lesion which was considered to be a consequence of previous *Treponema pallidum* infection. A computed tomography of temporal bones revealed SCDS of the right temporal bone. The patient was treated with acetazolamide without effect. Afterwards, surgical treatment involving transmastoid approach and covering the canal defect was performed and caused alleviation of his previous symptoms.

Conclusion
This case highlights the challenges of diagnosing patients with SCDS which often include multidisciplinary approach. As this syndrome can impact patient's daily functioning, the proper diagnosis and treatment are essential.

Keywords:
Dizziness; Headache; Semicircular Canal Dehiscence; Tinnitus

TINNITUS AND AUTOPHONY AS KEY DIAGNOSTIC CLUES IN SUPERIOR SEMICIRCULAR CANAL DEHISCENCE SYNDROME

Gabriela Večerić1; Ivana Ivošl; Zvonimir Popović2,3
1 Faculty of Medicine, University of Josip Juraj Strossmayer, Osijek, Croatia
2 Department of Neurology, University Hospital Center Osijek, Croatia
3 Department of Anatomy and Neuroscience, Faculty of Medicine, University of Josip Juraj Strossmayer, Osijek, Croatia
Presenting author: Gabriela Večerić, gabrielaveceric07@gmail.com

Introduction
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Case Report
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Conclusion
This case highlights the challenges of diagnosing patients with SCDS which often include multidisciplinary approach. As this syndrome can impact patient's daily functioning, the proper diagnosis and treatment are essential.

Keywords:
Dizziness; Headache; Semicircular Canal Dehiscence; Tinnitus

CHALLENGES IN DIAGNOSING AND TREATING MOGAD – A RARE AND COMPLICATED AUTOIMMUNE DISEASE OF THE CNS

Marta Lugarić1, Ivo Todorić1, Sofija Tomas1, Barbara Barun MD, PhD1,2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 University Hospital Centre Zagreb, Department of Neurology, Zagreb, Croatia
Presenting author: Marta Lugarić, marta.lugaric03@gmail.com

Introduction

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a rare autoimmune inflammatory disease in which the immune system produces anti-MOG antibodies that attack myelin oligodendrocyte glycoprotein, a component of myelin found in the CNS. It is typically presented with attacks of unilateral or bilateral optic neuritis, acute disseminated encephalomyelitis (ADEM) and transverse myelitis.

Case Report

A 25 year-old female patient was admitted to the Clinic of Neurology in KBC Zagreb with walking disabilities and low-grade fever. Three months prior to current hospitalization she had been diagnosed with bilateral recurrent optic neuritis with negative anti-AQP4 and anti-MOG antibodies that was treated with methylprednisolone. Upon admission, MRI showed cortical and juxtacortical white matter demyelination lesions. On the second day of hospitalization the patient developed high fever (39°C), and CSF analysis revealed pleocytosis (3471 cells/μL) with a predominance of polymorphonuclear cells (2421/μL) and 1050 mononuclear cells, indicating CNS inflammation compatible with encephalitis, which was treated with acyclovir (3x750 mg i.v.) and ceftriaxone (2x1g i.v.). Throughout the rest of the hospitalization her walking disabilities progressed to complete paraplegia and anti-MOG antibodies were found, due to seroconversion that occurs later. She was immediately started on the treatment consisting of 7 cycles of plasmapheresis, 10 days of methylprednisolone 1000mg, 2days of 2g/kg IVIG and one dose of tocilizumab 400mg i.v. Her condition stabilized and the control MR performed a few months later showed a significant regression of previous lesions, without the appearance of new ones. She continued further therapy with tocilizumab and oral prednisolone.

Conclusion

This case illustrates a rare but potentially severe condition with its usual presentation which, due to the wide spectrum of symptoms, can often be misdiagnosed with other similar neuroimmunological diseases. Although there is no typical cure, already mentioned immunosuppressive treatments can resolve symptoms and slow disease progression.

Keywords

Demyelinating Diseases; Encephalitis; Immunosuppression Therapy; Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease

BRAIN METASTASIS MIMICKING A CEREBROVASCULAR ACCIDENT - A CASE REPORT

Lucija Cvitak1, Klara Cifrek1, Sara Ćepić1, Marta Defar1, Marko Vukadinović2

1School of Medicine, University of Zagreb, Zagreb, Croatia
2Health Centre Zagreb - Centar, Zagreb, Croatia
Presenting author: Lucija Cvitak, lucy.cvitak@gmail.com

Introduction

Brain metastases are an unfavourable prognostic sign, irrespective of the tumour they originate from. Most common tumours that give brain metastases are primary lung and breast cancers and melanoma. Most brain metastases present with seizures or headaches, but focal neurological deficits such as upper or lower limb weaknesses can pose a diagnostic dilemma, especially in absence of other symptoms indicative of the primary tumour.

Case report

A 75-year-old male patient presented to the emergency department with left-sided hemiparesis, inverted left foot, left-sided hypoesthesia, and a positive Babinski sign on the left. Given the lateralized neurological deficits and the patient's age, an ischemic stroke was suspected. A brain computed tomography (CT) scan excluded intracranial hemorrhage, but revealed a well-defined, relatively round, centrally hypodense lesion measuring 21x23x23 mm in the right thalamus. The lesion was surrounded by prominent vasogenic oedema and was highly suspected of a neoplasm or a metastasis. Magnetic resonance imaging (MRI) showed post-contrast peripheral enhancement, confirming the diagnosis of a brain metastasis. Subsequent CT of the chest, abdomen and pelvis revealed an 18x14x38 mm necrotic tumour mass in the lower lobe of the right lung. Histopathological examination after endobronchial ultrasound-guided transbronchial needle aspiration confirmed non–small cell lung carcinoma (NSCLC). Due to the patient's progressive symptoms leaving him bed-bound and cachectic, systemic chemotherapy for NSCLC was contraindicated. He underwent stereotactic radiosurgery (SRS) for the solitary brain metastasis, delivered in three fractions of 8 Gy each, along with dexamethasone therapy. Follow-up brain MRI three months after SRS showed a significant regression of the lesion, now measuring 15x12 mm. The NSCLC therapy is still pending.

Conclusion

This case emphasizes the importance of distinguishing stroke from tumor-related deficits in patients with hemiparesis. For neurologists, maintaining a broad differential diagnosis is essential, as brain metastasis may be the first clinical sign of an underlying malignancy.

Keywords
Diagnosis, Differential; Lung Neoplasms; Neoplasm Metastasis; Radiosurgery; Stroke

CAROTID FIBROMUSCULAR DYSPLASIA – A CASE REPORT

Antonio Perišić1, Petar Panjak1, Matea Starešinić1, Slavica Kovačić1,2
1 University of Rijeka, Faculty of Medicine, Rijeka, Croatia
2 University Hospital Centre Rijeka, Department of Radiology, Rijeka, Croatia
Presenting author: Antonio Perišić, a.perisic02@gmail.com

Introduction
Fibromuscular dysplasia (FMD) is a non-atherosclerotic, non-inflammatory arteriopathy characterized by abnormal cellular proliferation and fibrosis within the arterial wall, leading to segmental stenosis, aneurysm formation, arterial dissection, and tortuosity. It most commonly affects the renal, carotid, and vertebral arteries, producing the characteristic “string-of-beads” appearance on imaging. Patients with carotid involvement may present with headache, pulsatile tinnitus, transient ischemic attack, ischemic stroke, or cervical artery dissection. About 90% of FMD patients are women. This case report highlights the importance of considering FMD in the differential diagnosis when patients present with these symptoms.

Case Report
A 49-year-old female patient was suddenly presented with right-sided limb weakness and speech difficulties suggestive of acute cerebral infarction, which persisted until the patient’s arrival at the Clinic of Neurology. Upon admission, NIHSS score was 3 and mRS was 0, while the following brain MRI showed acute ischemia signs on left part of pons. Her medical history was notable for well-controlled hypertension treated with antihypertensive therapy. During hospitalization at the Neurology Clinic, CT angiography revealed bilateral fibromuscular dysplasia of the carotid arteries with the characteristic “string-of-beads” appearance. The patient received prompt medical treatment and recovered without significant neurological deficits, except for occasional episodes of mild dizziness. Antiplatelet therapy with aspirin was initiated in addition to her previous medication, and physical therapy was recommended. At present, percutaneous transluminal angioplasty is not indicated since the medical treatment proved to be sufficient, and the patient will continue regular follow-up at the Neurology Clinic.

Conclusion
This case emphasizes carotid fibromuscular dysplasia, which most commonly shows bilateral involvement, as an important non-atherosclerotic cause of cerebral infarction. Recognition of its characteristic imaging features is essential, as management differs from that of atherosclerotic disease and is primarily medical. Increased awareness of this condition may improve diagnostic accuracy and contribute to more effective secondary stroke prevention.

Keywords:
Angiography; Aspirin; Fibromuscular Dysplasia; Infarction; Stroke

SUSPECTED FAMILIAL CEREBRAL CAVERNOMAS PRESENTING WITH DIPLOPIA AND NEVUS FLAMMEUS: A CASE REPORT

Marija Kišmartin1, Iva Kristić1, Lea Ćorluka1, Silva Guljaš1,3, Ružica Palić Kramarić1,2,4
1 Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia
2 University Hospital Centre Osijek, Department of Neurology, Osijek, Croatia
3 University Hospital Centre Osijek, Department of Radiology, Osijek, Croatia
4 Faculty of Dental Medicine and Health, J.J. Strossmayer University of Osijek, Department of Clinical Medicine, Osijek, Croatia
Presenting author: Marija Kišmartin, marija.kismartin@gmail.com

Introduction
Familial cerebral cavernous malformation is an autosomal dominant inherited disorder associated with mutations in the KRIT1, CCM2, and PDCD10 genes in 85–90% of patients. The number of cerebral and/or spinal cavernous malformations varies from a single lesion to several hundred and may occur in other organs. The disease is mostly diagnosed within young adults due to focal neurological deficits, headaches or seizures. However, up to 50% of patients remain asymptomatic.

Case Report
A 24-year-old, otherwise healthy man presented to the emergency with diffuse headache, blurred vision and diplopia on extreme left gaze for the past two days. He reported a possible contributory head injury during boxing the day before symptom onset. His uncle died from stroke-related complications at the age of 40. Computed tomography of the brain revealed cavernomas in the central pons affecting the abducens nucleus and in the left parietal region. The hospital course proceeded without neurological or systemic deterioration. Further evaluation revealed mutations of factor II (prothrombin), MTHFR and PAI-1. Therapy with neurobiotin, diazepam and paracetamol was initiated. Brain magnetic resonance imaging (MRI) demonstrated eighty supra- and infratentorial cavernomas with subacute hemorrhage zones. Findings from transcranial color doppler and color doppler of the carotid and vertebral vessels, three-dimensional time-of-flight MR angiography, echocardiography, holter electrocardiography and chest radiography were unremarkable. A lumbosacral nevus flammeus was diagnosed. Accordingly, a medical genetics consultation was requested. Brain MRI of the patient's parents was recommended, with additional genetic testing in order to confirm the suspected diagnosis, if cavernomas are identified. Further follow-up was planned with the aim of initiating optimal therapy. Walking is recommended instead of boxing. In case of immobility, anticoagulant therapy is advised.

Conclusion
Multidisciplinary evaluation is crucial in young patients, as diplopia, especially with external signs like a nevus flammeus, may signal serious neurovascular or genetic pathology requiring prompt diagnostic workup.

Keywords:
Abducens Nucleus; Diplopia; Genetics, Medical; Headache; Magnetic Resonance Imaging

CHALLENGING DIAGNOSIS: MULTIFOCAL OLIGODENDROGLIOMA MIMICKING INFECTIOUS DISEASE - A CASE REPORT

Dora Gašparović1, Marija Bunić1, Ena Ivanković1, Antonia Mišić1, Marta Petek Vinković1,2
1 Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
2 Department of Intensive Neurological Treatment, Clinical Hospital Centre Osijek, Osijek, Croatia
Presenting author: Dora Gašparović, dora.gasparovic@outlook.com

Introduction
Oligodendroglioma is a primary brain tumor that typically presents as a slow-growing, solitary lesion; however, atypical presentations with multifocal lesions and obstructive hydrocephalus may mimic infectious or inflammatory diseases, delaying diagnosis and requiring integration of radiological, microbiological, and histopathological findings.

Case Report
A 37-year-old woman presented to the Department of Neurology in February 2025 with diplopia, vertigo, gait instability, and mild right-sided hemiparesis following a brief febrile episode (38.5°C). Neurological examination revealed left oculomotor nerve palsy, dystaxia and impaired vertical gaze. Brain magnetic resonance imaging (MRI) showed multiple subependymal and basal ganglia lesions, the largest (22 mm) in the left mesencephalon, with contrast enhancement and edema causing aqueductal compression and obstructive hydrocephalus, initially suggestive of parasitosis. An external ventricular drain was placed, requiring several revisions due to persistent hydrocephalus. Computed tomography confirmed ventricular dilatation and stable lesions. Laboratory findings were largely unremarkable, with mildly elevated IgE raising suspicion of parasitic infection; however, extensive microbiological and serological testing excluded it. Hematological workup, including bone marrow analysis and immunophenotyping, excluded malignancy. Radiologists in Osijek and Zagreb were repeatedly consulted, as brain MRI findings strongly suggested parasitic infection. Biopsy was initially deferred due to high risk and potential parasite dissemination. Due to lack of improvement and mild deterioration, biopsy was performed, confirming oligodendroglioma WHO grade II. Radiotherapy and temozolomide were initiated. Follow-up brain MRI in July 2025 showed mostly stable disease with mild regression of one temporal lesion and no hydrocephalus. In January 2026, the patient remained in good general condition and continued temozolomide therapy.

Conclusion
This case illustrates an atypical presentation of a low-grade glioma mimicking infection, highlighting the importance of considering neoplastic etiology despite suggestive findings. Neuroradiologically, the case posed a diagnostic challenge for radiologists and clinicians. It was concluded that biopsy would provide greater clinical benefit, enabling appropriate treatment, which was subsequently administered.

Keywords:
Brain Neoplasms; Hydrocephalus; Oligodendroglioma; Parasitic Disease; Temozolomide

DIAGNOSTIC CHALLENGES IN A POST-DNET PEDIATRIC PATIENT: DIFFERENTIATING EPILEPTIC RELAPSE FROM PSYCHOGENIC NON-EPILEPTIC EVENTS

Lucija Mitrović1, Natalija Grdsanjski1, Karla Kleščić1, Veronika Hanić1, Jelena Radić Nišević2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Centre Rijeka, Department of Pediatric Neurology, Rijeka, Croatia
Presenting author: Lucija Mitrović, lucijamitrovic3@gmail.com

Introduction
Dysembryoplastic neuroepithelial tumors (DNETs) are rare, low-grade glioneuronal tumors in children, commonly associated with focal epilepsy. Although surgical resection often results in seizure freedom, late recurrence or persistent epileptogenicity may occur. Differentiating epileptic seizures from psychogenic non-epileptic seizures (PNES) is essential but can be particularly challenging in patients with prior structural lesions.

Case Report
A 14-year-old female with a history of right temporal DNET resected in 2015 remained seizure-free, leading to antiseizure medication withdrawal in 2019. In December 2024, she developed recurrent paroxysmal episodes occurring in clusters, characterized by tongue paresthesia, left-sided weakness, brief impaired awareness, falls, and tremulous movements. Some episodes showed atypical features, including occurrence in social settings, partial responsiveness, and preserved awareness, raising suspicion for PNES; in one instance, she asked peers to record the event prior to onset. Neurological examination was normal. Prolonged video-EEG monitoring did not capture a typical clinical event. Wake EEG was limited by artifacts, while sleep EEG showed stable background without asymmetry and isolated right frontotemporal sharp waves without clinical correlation. No definitive ictal pattern or clear PNES semiological markers were recorded. Given recurrent events, interictal discharges, and structural background, a mixed etiology (epileptic relapse vs PNES) was considered. Therapy was adjusted with lamotrigine titration, oxcarbazepine tapering, and continuation of pregabalin due to its adjunctive antiseizure and anxiolytic effects. Repeat MRI and psychiatric evaluation were recommended.

Conclusion
This case highlights the complexity of distinguishing epileptic relapse from PNES in post-DNET patients. A multidisciplinary approach integrating clinical, electrophysiological, imaging, and psychiatric assessment is crucial for accurate diagnosis and tailored management.

Keywords:
DNET; pediatric epilepsy; PNES; video-EEG; seizure recurrence

ISCHEMIC STROKE ASSOCIATED WITH PATENT FORAMEN OVALE AND PROTHROMBIN GENE MUTATION

Lana Papić1, Maria Perić1, Petra Petravić1, Sara Drnasin2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, University Hospital "Sestre milosrdnice", Zagreb, Croatia
Presenting author: Lana Papić, lana.papic123@gmail.com

Introduction

Paradoxical embolism is a rare but clinically important cause of ischemic stroke. It occurs when a venous thrombus enters arterial circulation through an intracardiac shunt, most commonly a patent foramen ovale (PFO). PFO is present in about 25% of the general population but becomes clinically relevant in patients with venous thrombosis or prothrombotic conditions. Inherited thrombophilias, such as prothrombin gene mutations, increase the risk of venous thrombus formation and may contribute to complex thromboembolic events.

Case Report

A 52-year-old male was admitted with sudden neurological deficit. The NIH Stroke Scale (NIHSS) score on admission was 1. Brain magnetic resonance imaging showed acute infarction in the left thalamus. Computed tomography angiography revealed bilateral segmental pulmonary embolism, and evaluation confirmed deep vein thrombosis of the lower limb. Transthoracic echocardiography demonstrated a patent foramen ovale with right-to-left shunt. Thrombophilia testing identified heterozygous prothrombin (factor II G20210A) mutation. The combination of deep vein thrombosis, pulmonary embolism, and cerebral infarction strongly suggested paradoxical embolism. The patient initially received intravenous thrombolysis. Because venous thromboembolism was present, therapeutic anticoagulation was subsequently initiated. During treatment, he developed a large retroperitoneal hematoma. Anticoagulation was temporarily withheld, and the patient was managed conservatively with monitoring. After stabilization and radiological regression of the hematoma, anticoagulation was cautiously reintroduced. Following clinical and hemorrhagic stabilization, percutaneous PFO closure was performed, with timing guided by bleeding risk. At discharge, the NIHSS score was 0. During follow-up, the patient remained neurologically stable without recurrent thromboembolic events.

Conclusion

This case highlights the combined effect of inherited thrombophilia and PFO in systemic embolism. The presence of deep vein thrombosis, pulmonary embolism, and cerebral infarction represents a diagnostic triad of paradoxical embolism. Prothrombin gene mutation increases the risk of venous thrombosis, while PFO enables emboli to enter arterial circulation and cause ischemic stroke. Recognition of both factors was essential for individualized management, including anticoagulation and percutaneous PFO closure, resulting in a favorable outcome without recurrence.

Keywords:
Brain infarction; Ischemic stroke; Patent Foramen Ovale; Hypercoagulability; Deep vein thrombosis

WHEN VISION AND GAZE CONTROL DIVERGE: HEIMANN-BIELSCHOWSKY PHENOMENON IN MULTIPLE SCLEROSIS

Marko Šarić1, Tin Trobić1, Korina Pervan1, Mašan Sredanović2, Mario Habek1,2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 University Hospital Center Zagreb, Department of Neurology, Referral Center for Demyelinating Diseases of Central Nervous System, Zagreb, Croatia
Presenting author: Marko Šarić, smarko046@gmail.com

Introduction
The Heimann–Bielschowsky phenomenon (HBP) is characterized by slow, large-amplitude, pendular vertical oscillations of one eye, typically occurring in an eye with severe, long-standing visual loss. It is considered a form of dissociated vertical nystagmus associated with disruption of visual fixation and central ocular motor control.

Case Report
A 63-year-old man with a 3-year history of primary progressive multiple sclerosis (MS) and on treatment with ocrelizumab reported newly noticed involuntary right eye movements.
Eye movement examination revealed a unilateral rotatory pendular nystagmus confined to the right eye, present in the primary gaze position. Corrected visual acuity in the right eye was 20/200. Visual evoked potentials (VEP) showed severely abnormal responses in the right eye, with an absent N75–P100–N135 complex, while left-eye responses showed only prolonged P100 latency. Brain MRI axial T2-weighted sequences demonstrated a focal hyperintense lesion in the right dorsolateral medulla oblongata. This case supports the hypothesis that the HBP reflects a combined effect of impaired visual afferent input and abnormal central oculomotor control within the brainstem. The right dorsolateral medullary lesion involves the vestibular nuclei, which provide essential input to ocular motor integrators via the medial longitudinal fasciculus, forming a plausible anatomical substrate for monocular pendular nystagmus. Isolated dysfunction of either system alone is insufficient to produce this phenomenon. Furthermore, prolonged P100 latency, reflecting chronic structural and functional damage along the visual pathway, may lead to asymmetric deprivation of visual afferent input, destabilizing central gaze-holding networks and thereby predisposing to the HBP.

Conclusion
This case shows that disrupted feedback between central oculomotor control and sensory pathways can cause unilateral rotatory monocular pendular nystagmus in a patient with MS. The disorder likely reflects chronic dysfunction rather than an acute new lesion. VEP and targeted brainstem imaging may help clarify its underlying mechanisms.

Keywords:
Pendular Nystagmus; Multiple Sclerosis; Brainstem; Evoked Potentials, Visual

SUCCESSFUL TREATMENT OF CHEMOTHERAPY-INDUCED HEADACHE WITH ATOGEPANT: A CASE REPORT

Lucija Tomulić1, Tea Tomičević1, Valentino Rački1,2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Department of Neurology, Clinical Hospital Center Rijeka, Rijeka, Croatia
Presenting author: Lucija Tomulić, lucija.tomulic@gmail.com

Introduction
Headache is a frequent but under-recognized adverse effect of chemotherapy, occasionally presenting with migrainous features even in patients without a prior headache history. Management is often challenging, as such headaches may be severe and refractory to standard analgesic therapies. Calcitonin gene-related peptide (CGRP) antagonists have proven efficacy in migraine, but their role in chemotherapy-induced headache has not been established.

Case Report
A 32-year-old woman undergoing chemotherapy for breast cancer developed a new-onset severe headache seven days after chemotherapy administration. The headache was hemicranial and associated with marked photophobia and nausea, fulfilling clinical characteristics of migraine. The patient had no personal history of primary headache disorders. Neurological evaluation revealed no focal deficits, and routine analgesics, including non-steroidal anti-inflammatory drugs and paracetamol, were ineffective. Atogepant is an oral CGRP receptor antagonist that was initiated prophylactically on the day of chemotherapy. Following initiation of atogepant, headache intensity decreased by more than 50%. Although mild headache persisted, it became manageable and was no longer accompanied by photophobia or nausea. Atogepant was taken from the day of chemotherapy up to seven days afterward, with intermittent breaks due to the absence of continuous headache. The treatment was well tolerated, and no adverse effects were reported.

Conclusion
This case suggests that atogepant may represent a novel and effective therapeutic option for chemotherapy-induced headache with migrainous features, particularly when conventional analgesics fail. CGRP pathway modulation could play a role in managing secondary headaches related to chemotherapy, warranting further systematic investigation.

Keywords:
Calcitonin Gene-Related Peptide Receptor Antagonists; Chemotherapy; Headache; Migraine Disorders

VIRAL ENCEPHALITIS FOLLOWING HEMATOPOIETIC STEM CELL TRANSPLANTATION - SHORT REVIEW OF THERAPEUTIC OPTIONS

Korina Pervan1, Marin Rimac1, Marko Šarić1, Tin Trobić1, Ivana Mareković1,2
1 School of Medicine, University of Zagreb, 10000 Zagreb
2 Department of Clinical Microbiology, Infection Prevention and Control, University Hospital Centre Zagreb, 10000 Zagreb
Presenting author: Korina Pervan, kpervan@student.mef.hr

Introduction

Viral encephalitis is a rare complication following hematopoietic stem cell transplantation (HSCT), occurring in 1-1.2% of recipients and most commonly presenting with fever, seizures, and cranial nerve palsies. Incidence is the highest among recipients of cord blood transplants, and human herpesvirus 6 (HHV6) is the most common cause. Treatment remains challenging because of the diversity of viral causes and limited therapeutic options in immunocompromised HSCT recipients.

Materials and Methods

A comprehensive search was conducted to identify relevant literature sources published between January 2010 and December 2025. The databases searched included PubMed, Scopus, Web of Science, Embase, and Google Scholar. Case reports, editorials, letters to the editor, conference abstracts and non-human studies were excluded. We incorporated a combination of keywords such as hematopoietic stem cell transplantation, viral causative agents and viral encephalitis. A total of 14 papers were selected.

Literature Review

Due to the variety of causes, multiple therapeutic strategies exist. Foscarnet is considered the first-line treatment for HHV6 due to its lower immunosuppressive effect and superior penetration across the blood–brain barrier, whereas ganciclovir is preferred for cytomegalovirus (CMV) infections. Both can be used in the treatment of Epstein-Barr viral encephalitis, with rituximab as an additional option. Acyclovir and valacyclovir remain the treatments of choice for Herpes simplex and Varicella zoster virus encephalitis. JC and BK encephalitis is treated with cidofovir. At present, evidence supporting prophylactic interventions mostly remains limited to ciprofloxacin for BK virus and letermovir for CMV. Virus-specific T-cells are a newly proposed treatment, requiring further evidence.

Conclusion

Heterogeneous causes of viral encephalitis, low incidence, and the absence of large-scale studies mean that the evidence guiding prophylaxis or therapy is limited. Current management relies largely on small case series, extrapolation from other viral infections, and expert opinion, highlighting the need for further research.

Keywords:
Antiviral Agents; Encephalitis, Viral; Hematopoietic Stem Cell Transplantation; Herpesvirus 6, Human

Hemorrhagic Cavernous Malformations versus Demyelinating Disease: A Diagnostic Challenge in Acute Brainstem Syndrome

Tiyya Selimović1, Morena Sirotić1, Lana Oštro2, Petra Črnac2 , Sanja Tomasović2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Department of Neurology, Sveti Duh University Hospital, Zagreb, Croatia
Presenting author: Tiyya Selimović, tiyya.se@gmail.com

Introduction

An acute change in neurological status with brainstem localization requires urgent etiological evaluation, particularly when neuroimaging findings suggest both vascular and demyelinating substrate. Both cavernous malformations with hemorrhagic transformation and demyelinating disease may clinically appear similar. The aim of this case report is to highlight a diagnostic challenge of separating cavernomas with hemorrhage from demyelinating lesions as potential underlying causes of a patient's symptoms.

Case Report

A 43 year old patient was admitted to the Department of Neurology with sudden-onset imbalance, diplopia, nausea, and vomiting. Neurological examination revealed left internuclear ophthalmoplegia, mild right-sided arm paresis with brisk deep tendon reflexes on the right side and ataxic gait. Despite the patient's relatively young age vascular risk factors included arterial hypertension, type 2 diabetes mellitus, and hyperlipidemia. Initial non-contrast MSCT showed centrally located hyperdense pontine lesions and a partially calcified vermian lesion suggestive of cavernous malformations, with possible intralesional hemorrhage. CT angiography of intracranial and cervical vessels did not show stenosis, occlusion, aneurysm, or AV malformation. Brain MRI (3T) demonstrated multiple susceptibility-weighted hypointense lesions in the brainstem, deep gray nuclei and cerebellum consistent with cavernomas, some with hemorrhagic transformation. Additionally, more than 20 bilateral periventricular and subcortical T2/FLAIR hyperintense lesions were observed, several oriented perpendicular to the ventricles, raising suspicion of demyelinating disease. Given the significant clinical overlap between demyelinating and hemorrhagic etiologies, further diagnostic evaluation will be performed after radiological stabilization, including lumbar puncture to support a definitive diagnosis.

Conclusion

This case highlights the significant diagnostic challenge posed by the clinical overlap of hemorrhagic cavernous malformations and demyelinating lesions in acute brainstem presentation. A structured diagnostic approach, including follow-up imaging and cerebrospinal fluid analysis is essential to establish the definitive etiology and guide appropriate management.

Keywords:
Ataxia; Demyelinating Diseases; Diplopia; Internuclear Ophthalmoplegia

ASSOCIATION BETWEEN CHARCOT – MARIE – TOOTH TYPE 1 AND MULTIPLE SCLEROSIS

Matea Marović1, Dominik Lukičić1, Ivan Adamec1,2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Neurology, University Hospital Centre Zagreb, Zagreb, Croatia
Presenting author: Matea Marović, matea.marovic2003@gmail.com

Introduction

Multiple sclerosis (MS) is a chronic, inflammatory, demyelinating, immune-mediated disease of the central nervous system with the most common clinical presentation being relapsing-remitting MS (RRMS), producing symptoms such as muscle weakness, sensory loss and ataxia. Charcot – Marie – Tooth (CMT) is an inherited heterogenous group of demyelinating peripheral neuropathies, mainly presenting with distal limb weakness, and muscle atrophy. We present a case of rare association of MS and CMT1a with peripheral myelin protein (PMP) mutation.

Case Report

A 40-year-old female first presented with right sided facial numbness. Magnetic resonance imaging (MRI) demonstrated supratentorial and right pontine T2 hyperintense lesions. Lumbar puncture showed intrathecal synthesis of oligoclonal IgG bands. Having met the 2017 McDonald criteria, the patient was diagnosed with RRMS and treatment with dimethyl fumarate was initiated. Her history revealed that for the prior 12 months she gradually developed bilateral calf atrophy, foot muscle weakness, and difficulties walking, findings not attributable to central lesions of MS but consistent with a peripheral neuropathy pattern. Given the positive family history of CMT, gene analysis was performed discovering PMP22 gene duplication leading to the diagnosis of CMT type 1a. The patient exhibited no signs of clinical or radiological MS disease activity during a 4-year follow up. However, symptoms of CMT gradually progressed leading to further weakness of the feet and development of hand muscle atrophy.

Conclusion

Our patient presents a rare case of co-occurrence of MS and CMT1a. An increasing number of reports associate the PMP22 gene mutation with central nervous system demyelinating disorders which may suggest causative link between these two demyelinating diseases. Emerging research has shown a link between CMTX1, another type of CMT caused by mutations in Gap Junction Beta-1 gene, and MS-like lesions on MRI, suggesting that different types of CMT may be linked with MS or MS-like imaging findings.

Keywords:
Charcot-Marie-Tooth Disease; Demyelinating Diseases; Multiple Sclerosis; Myelin Proteins; Oligoclonal Bands

Internal Carotid Artery Dissection As A Cause Of Transient Neurological Deficits – A Case Report

Ana Šmit1, Laura Čajo2 , Ana Vučaj1, David Bonifačić3 , Ines Strenja3
1 – Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 – Faculty of Medicine, University of J.J. Strossmayer, Osijek, Croatia
3– Department of Neurology, Clinical Hospital Center Rijeka, Rijeka, Croatia
Presenting author: Ana Šmit1, ana.smit11@gmail.com

Introduction
Internal carotid artery (ICA) dissection is a leading cause of ischemic stroke in young and middle-aged adults. It often presents with nonspecific symptoms such as cervical pain, headache, and transient neurological deficits. Early recognition using multimodal neuroimaging is crucial to prevent permanent neurological impairment.
Case Report
WeWe present a 43-year-old male smoker with dyslipidemia, admitted due to transient speech disturbance. Ten days prior, he developed posterior neck pain after prolonged driving, which persisted for one week. Three days before admission, he performed neck massage with partial relief. On the day of admission, he experienced sudden inability to speak with incoherent, effortful speech lasting 30 minutes, accompanied by mild imbalance. Symptoms resolved before arrival. Non-contrast brain CT was unremarkable. CT angiography revealed dissection of the extracranial segment of the left ICA with preserved intracranial flow via collaterals. The patient was not a candidate for mechanical thrombectomy. Brain MRI showed a small acute ischemic lesion in the posterior left superior temporal gyrus, with findings consistent with post-dissection occlusion. Dual antiplatelet therapy and high-intensity statin therapy were initiated. Neurological examination at discharge was normal, and follow-up was arranged.
Conclusion
ICA dissection should be suspected in young patients with cervical pain and transient neurological symptoms, especially after mechanical neck strain. Multimodal imaging allows early diagnosis and assessment of lesion severity. Prompt antiplatelet therapy can achieve favorable outcomes and prevent progression of ischemic injury.

Keywords:
Carotid Artery, Internal; Dissection; Statins; Ischemic Attack, Transient; Neck Pain;

A RARE NEURO-OPHTHALMOLOGICAL PRESENTATION OF PLEXIFORM NEUROFIBROMA IN A PATIENT WITH NF1

Paolo Stroligo[1]*, Karlo Žepina1, Maja Novak-Stroligo, MD2
[1]Medical Faculty, University of Rijeka, Rijeka, Croatia
[2]Department of Ophthalmology, Rijeka, Croatia
Presenting author's email: paolostroligo@gmail.com

Introduction: Neurofibromatosis type one (NF1) is an autosomal dominant disorder. Incidence is 1 in 3000, and it has heterogeneous clinical presentation, including café-au-lait spots, Lisch nodules, neurofibromas, optic glioma, bone dysplasia and axillary freckling. Plexiform neurofibroma is the most infiltrative neurofibroma and has the highest risk of malignancy.

Case report: A 34-year-old patient was diagnosed with neurofibromatosis type one and epilepsy since childhood. She presents with periorbital changes, such as ptosis of the eyelid, disturbed vision in the left eye, and suspected hydrocephalus with VP shunting. In anamnesis, we find that she already had two operations for ptosis reduction and neurofibroma reduction. On physical examination, a significant ptosis of the left eyelid and soft tissue thickening of the periorbital region are seen, along with limited elevation and abduction of the eyeball that causes diplopia. Brain and orbital MRI were performed, revealing a plexiform neurofibroma with invasion of the periorbital structures. A considerable degree of involvement warrants referral to an oculoplastic surgeon. Reduction with correction of ptosis is planned in a multidisciplinary approach due to the infiltrative nature of plexiform neurofibroma. In the end, ptosis was mostly corrected, and the patient's sight is much better, but diplopia persists. Neurofibroma was confirmed pathohistologically. This case is critical because of the pronounced neuro-ophthalmologic manifestations in NF1, which require a balanced diagnostic and therapeutic approach.

Conclusion: This case illustrates the complexity of managing neuro-ophthalmological manifestations in NF1. Optimal patient outcome relies on a multidisciplinary and individualized strategy for the best quality of life.

Key words:
Blepharoptosis; Diplopia; Eye Neoplasms; Neurofibroma, Plexiform; Neurofibromatosis 1

DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN A CASE OF SEVERE RECURRENT TOLOSA-HUNT SYNDROME

Borna Borić1, Andrijana Andrešić1, Luka Barać1, Vladimir Cvitanović1, Hrvoje Budinčević2
1 Faculty of Medicine, University of Rijeka, Rijeka, Republic of Croatia
2 Sveti Duh University Hospital. Department of Neurology, Zagreb, Republic of Croatia
***Presenting author:** Borna Borić, borna.boric@gmail.com

Introduction

Tolosa-Hunt Syndrome (THS) is an uncommon condition characterized by painful ophthalmoplegia caused by idiopathic granulomatous inflammation of the cavernous sinus, superior orbital fissure, or orbital apex. While typically steroid-responsive, the management of recurrent or refractory cases remains a significant clinical challenge. This case highlights THS severity, where refractory inflammation led to irreversible sensory and motor deficits despite intensive immunosuppression.

Case Report

A 76-year-old female with hypertension and Hashimoto's thyroiditis presented with symptoms dating back to March 2025, characterized by recurrent episodes of painful left-sided ophthalmoplegia and diplopia. The disease followed a relapsing-remitting course with three distinct hospitalizations. Initial neurological examinations consistently revealed ptosis and impaired ocular motility. Despite multiple cycles of pulsed intravenous methylprednisolone and maintenance therapy with Methotrexate, which provided only transient symptomatic relief, her condition progressively worsened. By September 2025, the patient experienced a complete amaurosis of the left eye. The definitive diagnosis was established using 3 Tesla Magnetic Resonance Imaging (MRI) of the orbits, which demonstrated asymmetric thickening and post-contrast enhancement of the left cavernous sinus, extending into the orbital apex and superior orbital fissure, along with characteristic dural tailing. Laboratory findings supported the inflammatory etiology, showing an elevated erythrocyte sedimentation rate and high titers of Anti-Thyroid Peroxidase/Anti-Thyroglobulin antibodies, while thyroid-associated orbitopathy and orbital malignancies were excluded. During her January 2026 admission, clinical findings confirmed permanent complete left-sided ophthalmoplegia (cranial nerves III, IV, and VI) and amaurosis. The patient was managed with 60 mg of oral Prednisone. Current therapeutic outcomes are limited to pain control and managing secondary complications, such as exposure keratopathy and corneal scarring, caused by the permanent loss of the blink reflex.

Conclusion

This case illustrates that Tolosa-Hunt Syndrome is not always a benign, steroid-responsive condition. Recurrent inflammation can lead to permanent cranial nerve damage and irreversible amaurosis. It highlights the need for aggressive initial treatment and long-term multidisciplinary follow-up to mitigate the risk of catastrophic visual outcomes.

Keywords:
Blindness; Cavernous Sinus; Corticosteroids; Ophthalmoplegia; Tolosa-Hunt Syndrome

WHEN STROKE STRIKES EARLY: THE HIDDEN ROLE OF PATENT FORAMEN OVALE; A CASE REPORT

Maja Tomljenović[1]* ,Marija Ševo[1], Ines Strenja [1,2],David Bonifačić [1,2]
[1]Faculty of Medicine, University of Rijeka, Rijeka, Croatia
[2]Clinical Hospital Center Rijeka, Department of Neurology, Rijeka, Croatia
***Presenting author** : Maja Tomljenović, tomljenovicmaja13@gmail.com

Introduction : A patent foramen ovale (PFO) is a residual, functionally significant interatrial channel at the fossa ovalis, resulting from incomplete septal fusion. While physiological in fetal circulation, its persistence in adults is associated with paradoxic embolism, particularly cryptogenic stroke. PFO is identified in 10-15% cases via transthoracic echocardiography (TTE) and observed in up to 27% autopsies, representing an established cause of stroke in young adults.

Case presentation : A 42-year-old man presented with sudden dysarthria and right-sided weakness. On admission, 4 hours after symptom onset, his score on National Institutes of Health Stroke (NIHSS) was 11. Non-contrast brain computed tomography revealed an acute ischemic stroke in the left middle cerebral artery (MCA) with a large penumbra. Intravenous thrombolysis (Tissue Plasminogen Activator) was administered, followed by mechanical thrombectomy which resulted in subsequent clinical improvement. In addition to the left MCA infarction, magnetic resonance imaging demonstrated multiple acute ischemic lesions in both hemispheres. Doppler ultrasonography of the carotid and vertebral arteries was performed with a transcranial doppler emboli detection (bubble test), which detected cerebral emboli (grade 4). Moreover, the patient underwent physical therapy, was discharged with significant neurological improvement (NIHSS 2) on dual antiplatelet therapy and referred to a cardiologist. TTE and transesophageal echocardiography confirmed the presence of PFO. Percutaneous PFO closure with a patch device was indicated. The patient remained under follow-up by both neurology and cardiology department.

Conclusion : Ischemic stroke in young adults requires a systematic diagnostic approach, especially when an embolic mechanism is suspected in the absence of traditional risk factors. Evaluation should include neuroimaging, cardiac rhythm monitoring, and echocardiography with bubble study. PFO should be considered in cryptogenic cases as its recognition enables appropriate secondary prevention and may improve long-term outcomes.

Keywords: Dysarthria, Foramen Ovale, Patent; Stroke; Thrombectomy; Ultrasonography, Doppler

KAPPA FREE LIGHT CHAINS – A QUANTITATIVE ALTERNATIVE TO OLIGOCLONAL BANDS IN MULTIPLE SCLEROSIS

Jelena Mićić1, Miloš Danilović2, Ljiljana Arsenović3, Evica Dinčić2
1 Faculty of Medicine, Military Medical Academy, University of Defence, Belgrade, Serbia
2 Department of Neurology, Military Medical Academy, Belgrade, Serbia
3 Institute of Biochemistry, Military Medical Academy, Belgrade, Serbia
Presenting author: Jelena Mićić, jelenamicic25@gmail.com

Introduction
Multiple sclerosis (MS) is an demyelinating disease 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 16.8 vs. 2.6, $p < 0.001$). OCB positivity was also 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, cost-effective marker with faster availability, the kFLC index may facilitate earlier diagnostic evaluation. These findings support the clinical utility of kFLC as an alternative biomarker in MS diagnostics, although further validation in larger patient cohorts is needed.

Keywords:
Biomarkers; Cerebrospinal Fluid; Immunoglobulin Light Chains; Multiple Sclerosis; Oligoclonal Bands

EMERGENCY MRI IN CT-NEGATIVE CEREBELLAR STROKE: A DECISIVE DIAGNOSTIC STEP

Tihana Majetić1, Andrijana Andrešić1, Borna Borić1, David Bonifačić1,2, Ines Strenja1,2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Centre Rijeka, Department of Neurology, Rijeka, Croatia
Presenting author: Tihana Majetić, tmajetic02@gmail.com

Background
Computed tomography (CT) is the standard first-choice imaging method for suspected acute stroke. However, in cases of posterior circulation stroke, early ischemic lesions often remain undetectable on CT, creating a critical diagnostic blind spot in the hyperacute phase. Magnetic resonance imaging (MRI) provides better sensitivity for detecting acute ischemia, yet it is rarely performed emergently within routine stroke protocols. Consequently, clinically apparent posterior circulation infarctions may initially escape radiological confirmation, potentially delaying appropriate therapeutic decisions.

Case Report
A female patient presented with sudden dizziness, right-sided instability, dysarthria, and mild right-sided central hemiparesis (National Institutes of Health Stroke Scale, NIHSS 4), consistent with a posterior circulation stroke. Admission blood pressure was significantly elevated (230/120 mmHg). The neurological examination revealed a shallow right nasolabial fold, subtle pronation of the right upper limb, slight lowering of the right leg during the Mingazzini maneuver, and lateropulsion to the right, with normal sensation. The initial CT scan showed no signs of acute ischemia or bleeding, only chronic microangiopathic changes. However, persistent focal deficits prompted suspicion of posterior circulation ischemia. An emergency MRI, rarely used in hyperacute stroke diagnosis, was performed. Diffusion-weighted imaging (DWI) confirmed ischemia in the right cerebellar hemisphere within the posterior inferior cerebellar artery (PICA) territory. Corresponding fluid-attenuated inversion recovery (FLAIR) hyperintensity without DWI/FLAIR mismatch indicated symptom onset beyond 4.5 hours, ruling out intravenous thrombolysis. No hemorrhage or hydrocephalus was observed; moderate chronic microangiopathy (Fazekas grade 2) was present. Treatment included dual antiplatelet therapy, high-dose statins, and optimized antihypertensive medication. Early rehabilitation followed. At discharge, neurological deficits had improved to an NIHSS score of 3, and the patient was functionally independent (modified Rankin Scale 0).

Conclusion
When clinical suspicion exceeds negative CT findings, emergency MRI can detect elusive posterior circulation infarctions, enhance treatment planning, and prevent potentially harmful diagnostic delays in cerebellar stroke.

Keywords: Brain Infarction; Computed Tomography; Diffusion Magnetic Resonance Imaging; Ischemic Stroke; Magnetic Resonance Imaging

WHEN OPTIC NEURITIS ISN'T MULTIPLE SCLEROSIS

Ana Bardić1*, Andrijana Andrešić1, Luka Barać1, Borna Borić1, Hrvoje Budinčević2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Sveti Duh University Hospital, Department of Neurology, Zagreb, Croatia
***Presenting author:** Ana Bardić, ana.bardic77@gmail.com

Introduction
Retrobulbar optic neuritis (RON) is inflammation of the optic nerve behind the eyeball, often associated with demyelinating diseases. It presents with periocular pain, visual disturbances, and color desaturation, most commonly in multiple sclerosis (MS), where it is usually unilateral and mild. Neuromyelitis optica spectrum disorder (NMOSD), mediated by aquaporin-4 (AQP4) antibodies, is generally more severe, often bilateral, may involve the optic chiasm, and frequently results in incomplete recovery and permanent visual loss. In our case, RON affected only one eye. Diagnosis was based on clinical assessment, neuroimaging including magnetic resonance imaging (MRI), visual evoked potentials, and AQP4 serology.

Case Report
A 28-year-old woman with a history of right-eye retrobulbar optic neuritis presented with right periocular pain, transient visual desaturation, and intermittent central and peripheral scotomas over two weeks. Examination showed visual acuity of 1.0 in the right eye and 0.9 in the left eye, with normal anterior segment and fundus. Ishihara testing showed minor color vision deficits. VEP revealed retrochiasmal demyelination of the right optic nerve. MRI of the brain and spinal cord showed stable periventricular hyperintensities on T2-weighted and Fluid-Attenuated Inversion Recovery (FLAIR) sequences, without contrast enhancement or new lesions, excluding multiple sclerosis. Laboratory tests were positive for AQP4 antibodies, while cerebrospinal fluid analysis was unremarkable. The patient was treated with high-dose intravenous corticosteroids, resulting in symptom improvement. Long-term immunosuppression with azathioprine (Imuran 100 mg) was initiated to reduce the risk of relapse. Follow-up exams, including visual fields, perimetry, and serial MRIs, remained stable with no new deficits.

Conclusion
This case illustrates recurrent RON as a presentation of AQP4-positive NMOSD, underscoring the importance of multimodal diagnostic assessment, including MRI, VEP, and serologic testing. Stable MRI lesions and positive AQP4 antibodies guided early immunosuppressive therapy, leading to visual recovery. Continuous neurologic and ophthalmologic monitoring, together with immunosuppressive treatment, is essential to prevent relapses and preserve visual function in patients with NMO.

Keywords:
Aquaporin 4; Demyelinating Diseases; Multiple Sclerosis; Neuromyelitis Optica; Optic Neuritis

CASE REPORT: DIAGNOSTIC DILEMMA IN ACUTE COMA - TUMOR OR HEMORRHAGE?

Magdalena Borić1, Katarina Ivezić1, Dora Gašparović1, Katarina Benaković1, Anita Božičević Vadlja2
1 Faculty of Medicine Osijek, University of Josip Juraj Strossmayer, Osijek, Croatia
2 Department of Emergency Medicine, University Hospital Centre Osijek, Osijek, Croatia
Presenting author: Magdalena Borić, magb3207@gmail.com

Introduction

Acute coma is a life-threatening neurological emergency requiring immediate stabilization and urgent etiological clarification. The differential diagnosis includes vascular events, seizures, and intracranial space-occupying processes. Prompt neuroimaging is essential to distinguish between these entities and guide further management.

Case Report

A 53-year-old patient was brought to the emergency department after sudden collapse and loss of consciousness. On admission, he was comatose with a Glasgow Coma Scale score of 4, showing no verbal response and minimal reaction to painful stimuli. The abrupt onset initially raised strong suspicion of an acute vascular event, particularly subarachnoid hemorrhage. However, certain neurological findings were atypical for a pure vascular etiology: deviation of the head and ocular bulbs, asymmetric pyramidal signs (positive Babinski sign on the left), and bilaterally constricted pupils with reduced light reactivity suggested a focal structural lesion with possible mass effect. An epileptic seizure with postictal coma was also considered; however, the severity of impaired consciousness together with focal neurological deficits raised concern for an underlying intracranial process. The patient received osmotherapy with mannitol, antiepileptic therapy, and supportive care. Due to neurological deterioration and impaired airway protection, orotracheal intubation was required. Urgent computed tomography and magnetic resonance imaging (MRI) demonstrated a large right frontal intra-axial mass with extensive perilesional edema, significant midline shift, and subfalcine herniation, consistent with critically elevated intracranial pressure. Advanced MRI techniques, including perfusion imaging and spectroscopy, showed increased choline, reduced N-acetylaspartate, and elevated relative cerebral blood volume, findings highly suggestive of a high-grade glioma. Electroencephalography revealed focal abnormalities over the right hemisphere. Neurosurgical consultation deemed the lesion inoperable, and stereotactic biopsy was recommended for definitive histopathological confirmation.

Conclusion

In comatose patients with an abrupt onset, vascular causes are often presumed; however, the presence of focal neurological deficits or atypical clinical features should promptly raise suspicion of an underlying intracranial mass. This case highlights the importance of early neuroimaging in the emergency triage of comatose patients, as timely differentiation between vascular, epileptic, and space-occupying etiologies is crucial for appropriate management and outcome. Not all acute comas are vascular—early recognition of red flags for mass effect is critical in emergency triage.

Keywords:
Brain Neoplasms; Coma; Glasgow Coma Scale; Intracranial Pressure

CLINICAL PRESENTATION OF CEREBELLAR ATROPHY WITH SENSORY NEUROPATHY: A CASE REPORT

Tea Elez1, Paula Kapović2, Natalija Frančin3, Andrea Elez4
1 Faculty of Medicine: University of Rijeka, Rijeka, Croatia
2 Faculty of Medicine: University of Rijeka, Rijeka, Croatia
3 Faculty of Medicine: University of Rijeka, Rijeka, Croatia
4 General Hospital Zadar: Zadar, Croatia
Presenting author: Tea Elez, tea.elez1809@gmail.com

Introduction
Cerebellar atrophy is a neurological condition characterized by progressive degeneration of cerebellar neurons, leading to impaired coordination, gait instability, dysarthria, and balance disturbances. It may arise from hereditary, degenerative, autoimmune, toxic, or paraneoplastic causes. The coexistence of cerebellar involvement with peripheral neuropathy presents a diagnostic challenge and requires a comprehensive evaluation and careful clinical assessment.

Case Report
A 36-year-old female presented with progressive lower limb weakness, more pronounced during physical exertion such as climbing stairs, along with difficulty squatting. The symptoms had been gradually worsening over time, affecting her daily activities. Initial MRI revealed thoracic disc protrusions (Th6–Th7, Th7–Th8). Anti-GAD antibodies were positive. Neurological examination showed abnormalities on the Mingazzini test and increased muscle tone in the lower limbs. Further evaluation with brain and cervical spine MRI demonstrated marked cerebellar atrophy in the infratentorial region. Electromyoneurography (EMNG) revealed mild chronic neural lesions in the upper limbs and moderate to severe chronic lesions in the lower limbs, predominantly affecting distal muscles. Sensory conduction studies showed reduced amplitudes and mildly slowed velocities. Based on clinical, radiological, and neurophysiological findings, a diagnosis of probable spinocerebellar ataxia was considered. Given the presence of anti-GAD antibodies, an autoimmune etiology was strongly suspected, although a genetic cause could not be excluded. Therefore, genetic testing for hereditary ataxias, including Friedreich's ataxia, was recommended. The patient was started on physical therapy, inpatient rehabilitation, vitamin D and E supplementation, and clonazepam, with partial symptomatic improvement.

Conclusion
This case highlights the importance of recognizing combined cerebellar and peripheral nervous system involvement in patients with progressive gait disturbance. It also emphasizes the need to consider both autoimmune (anti-GAD–related) and genetic causes in the differential diagnosis of spinocerebellar ataxia. Early identification of a potentially autoimmune etiology is particularly important, as it may influence treatment decisions and improve patient outcomes.

Keywords: Cerebellar Atrophy; Electromyography; Peripheral Nervous System Diseases; Spinocerebellar Ataxia

MULTIPLE RECURRENT STROKES AS THE FIRST MANIFESTATION OF OCCULT LUNG CANCER

Luka Barać1, Ana Bardić1, Vladimir Cvitanović1, Tihana Majetić1, Darija Ćorić2
1 University of Rijeka, Faculty of Medicine, Rijeka, Republic of Croatia
2 General Hospital Zadar, Department of Neurology, Zadar, Republic of Croatia
***Presenting author:** Luka Barać, lukabaracps@gmail.com

Introduction
Ischemic stroke is most commonly caused by atherosclerosis or cardioembolism; however, a subset of cases is associated with malignancy-related hypercoagulability. This etiology should be considered in patients presenting with recurrent or multifocal infarcts in different vascular territories without an identifiable cause. Cancer-associated stroke accounts for approximately 5–10 % of ischemic strokes and may represent the first manifestation of an occult malignancy.

Case Report
A 66-year-old woman was admitted to the Department of Neurology at General Hospital Zadar with acute speech disturbance that began the same morning. One month earlier, she had experienced a right occipital ischemic stroke with residual left homonymous hemianopsia and was discharged on aspirin and atorvastatin. Previous evaluation revealed no significant carotid stenosis, normal cardiac findings, and sinus rhythm.
On admission, neurological examination showed dysarthria, ataxia, incomplete right oculomotor nerve palsy, and pre-existing hemianopsia. Brain computed tomography (CT) demonstrated new bilateral thalamic hypodensities. Magnetic resonance imaging (MRI) confirmed multiple acute ischemic lesions in different vascular territories, including bilateral thalamic, frontal, and cerebellar regions. CT angiography revealed distal occlusion of the right middle cerebral artery.
Cardiac evaluation, including electrocardiography and transesophageal echocardiography, showed no embolic source or patent foramen ovale. Given recurrent multifocal infarctions without a clear etiology, a hypercoagulable state was suspected. Elevated tumor markers, carcinoembryonic antigen (CEA) and cytokeratin 19 fragment 21-1 (CYFRA 21-1), prompted further evaluation. Chest and abdominal CT revealed a suspicious pulmonary mass with lymphadenopathy. Endobronchial ultrasound-guided biopsy confirmed non-small cell lung cancer.
The patient received secondary stroke prevention and supportive therapy and was referred for oncological treatment. She was discharged with persistent neurological deficits and later died due to disease progression.

Conclusion
This case highlights the importance of considering cancer-associated hypercoagulability in patients with recurrent or multifocal ischemic strokes without an identifiable cause. Infarcts affecting multiple vascular territories over a short period should prompt extended diagnostic evaluation, including malignancy screening. Early recognition of occult cancer may influence therapeutic strategy and prognosis. In selected patients, anticoagulation should be considered instead of standard antiplatelet therapy.

Keywords:
Brain Ischemia; Lung Neoplasms; Paraneoplastic Syndromes; Stroke; Thrombophilia

UNEXPLAINED RAPID COGNITIVE DECLINE REVEALING CREUTZFELDT-JAKOB DISEASE

Lea Ćorluka1, Dora Gašparović1, Marija Kišmartin1, Antonia Mišić1, Tihana Gilman Kuric1,2
1 University of Osijek, Faculty of Medicine, Osijek, Croatia
2 Department of Neurology, University Hospital Center Osijek, Croatia
Presenting author: Lea Ćorluka, leacorluka6@gmail.com

Introduction
Creutzfeldt-Jakob disease (CJD) can be difficult to recognize when early symptoms fall outside the typical pattern, broadening the differential diagnosis toward neurodegenerative, autoimmune, infectious, or metabolic etiologies. Recognition usually follows the appearance of characteristic Magnetic Resonance Imaging (MRI) and Electroencephalography (EEG) abnormalities. As CJD is a rapidly progressive, fatal prion disease with no curative treatment, early identification is essential to ensure timely supportive and palliative care.

Case Report
A 63-year-old woman was admitted to the Department of Neurology with vertigo, cognitive decline, apathy, and hallucinations. The patient, brought in by her daughter, has displayed significant changes in mental state and behavior. She expressed concern about her health, fearing issues with her head. The daughter's observations noted a decline in the patient's apathy, interest in activities, daily living skills, and memory, as she struggles to remember simple actions like whether she has had coffee. Neurological examination revealed hypomimia, occasional bilateral resting tremor, and ataxic gait. Extensive somatic and laboratory evaluation ruled out major differential diagnoses, including autoimmune and viral encephalitis, paraneoplastic syndromes, and metabolic encephalopathies. In the following days her condition deteriorated further, marked by the emergence of myoclonus, occasional bilateral myoclonic jerks, and mutism, directing clinical suspicion toward prion disease. Brain MRI revealed bilateral caudate and putaminal hyperintensities with diffusion restriction and cortical ribboning, whereas EEG showed diffuse dysrhythmia with periodic spike-wave complexes (supporting the diagnosis of CJD). With the emergence of a typical clinical, radiological, and EEG pattern, the diagnosis of CJD was confirmed.

Conclusion
This case illustrates the rapid progression and diagnostic challenges of CJD, particularly with atypical early symptoms. Multimodal assessment with MRI, EEG, and laboratory markers is essential to distinguish CJD from other differential diagnoses. Early recognition is crucial to guide appropriate supportive and palliative care.

Keywords:
Creutzfeldt-Jakob Disease; Dementia; Myoclonus; Ataxia; Mutism

Overlap of Multiple Sclerosis and Systemic Lupus Erythematosus; A Case Report

Marija Ševo1, Maja Tomljenović1, Ines Strenja 1,2, , David Bonifačić1,2
1 Faculty of medicine, University of Rijeka, Rijeka, Croatia
2 Department of Neurology, Clinical Hospital Center of Rijeka, Rijeka, Croatia
Presenting author: Marija Ševo, marijasevo14@gmail.com

Introduction
Multiple sclerosis (MS) and systemic lupus erythematosus (SLE) are autoimmune diseases that can affect the nervous system. MS is characterized by demyelination of the central nervous system, whereas SLE is a B-cell-driven systemic disorder that can involve neuropsychiatric manifestations. Although each disease is relatively common, their coexistence in the same patient is extremely rare. This case report describes overlap of MS and SLE, highlighting the challenges of establishing an accurate diagnosis and selection of appropriate therapy.

Case Report
A 63-year-old male patient first presented in 2016 with neurological symptoms, characterized by paresthesia in the extremities exacerbated by neck flexion. Cervical magnetic resonance imaging (MRI) demonstrated demyelinating lesions involving the medulla oblongata and cervical spinal cord at levels C1-C7. Brain MRI showed multiple supratentorial and infratentorial demyelinating lesions without post-contrast enhancement. A follow-up MRI in 2017 demonstrated no progression of previously described lesions. Lumbar puncture revealed the presence of multiple cerebrospinal fluid–restricted oligoclonal IgG bands (type 2 pattern). Due to objectively quantified xerostomia, positive anti-Ro (SSA) antibodies and clinical suspicion of a neurological manifestation of Sjögren's syndrome, the patient was further evaluated by immunologist. During further evaluation, the patient developed pain in the small joints of the hands. Laboratory analysis revealed positive anti-dsDNA antibodies, and in February 2019, SLE was diagnosed. In March 2023, the patient was evaluated at the Croatian National Referral Center for Demyelinating Diseases at University Hospital Centre Zagreb. Based on the McDonald criteria, the diagnosis of relapsing-remitting multiple sclerosis (RRMS) was established. MRI of the brain and cervical spine demonstrated ≥ 9 T2/FLAIR lesions. The Expanded Disability Status Scale (EDSS) score was 3. Patient was started on Ofatumumab as first-line treatment for patients with RRMS and high risk of progression of neurological disability.

Conclusion
This case presents the complexity of diagnosing MS and SLE due to radiological, clinical and immunological overlap. A multidisciplinary approach and long-term follow-up are essential to establish an accurate diagnosis and ensure appropriate management.

Keywords:
Autoimmune Diseases; Magnetic Resonance Imaging; Multiple Sclerosis; Lupus Erythematosus, Systemic

FROM NEUROLOGICAL SYMPTOMS TO RECEPTOR SHIFTING BRAIN-ONLY METASTASIS: AN UNUSUAL CNS RESTRICTED TUMOR EVOLUTION

Lori Lovrenčić1, Sara Martinović1, Petar Suton2, Irma Stilinović2, Martina Bašić-Koretić2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 University Hospital Dubrava, Division of Oncology and Radiotherapy, Zagreb, Croatia
Presenting author: Lori Lovrenčić, lorilovrencic@yahoo.com

Introduction
Brain metastases occur in subset of breast cancer patients and represent a therapeutic challenge arising from the physiology of the central nervous system (CNS), as tumor cells and neural tissue interaction fosters resistance to systemic therapies. Treatment selection remains complex. This unique case illustrates how neurological presentation, neuroimaging, tumor molecular dynamics and CNS-specific therapeutic decisions converge in long-term patient management towards prolonging survival.

Case Report
We present a case of a 63-year old woman diagnosed in 2013 with invasive ductal carcinoma (NST), grade III, Luminal B, HER2-negative, initially treated with radical mastectomy, chemotherapy, radiotherapy and anastrozole. In 2017. she presented with focal neurological defects, speech difficulties and visual disturbances. A solitary intracranial metastasis in left temporal lobe, four years after primary diagnosis, was confirmed by neuroimaging. Patient underwent neurosurgical resection, fractionated radiotherapy and hormone therapy with tamoxifen.
In 2019. control magnetic resonance imaging (MRI) showed same site lesion recurrence and is treated with radiosurgery. In 2022 histopathological examination (HPE) after reoperation due to recurrence confirmed HER2-positive conversion. Systemic restaging repeatedly demonstrated no extracranial disease progression. The patient received docetaxel with pertuzumab/trastuzumab, achieving partial intracranial regression.
Between 2023 and 2025 MRI showed fluctuating intracranial activity between partial regression and renewed progression, development of cystic components, increased perfusion, and vasogenic edema, requiring two additional neurosurgical resections. HER2 status was shifting with striking receptor instability. In March 2025 confirmed another shift to HER2-negative disease, only to show a clear HER2 gene amplification in July 2025.
Throughout the treatment, all body imaging remained stable, confirming CNS-only progression and stable neurological status. The patient commenced trastuzumab deruxtecan and as of February 2026 MRI confirmed reduction of lesion thickness with no signs of active systemic disease.
Conclusion
This case reinforces the need for tailored CNS surveillance and repeated pathological assessment for changing biomarker profiles. Considering the limitations of systemic therapy penetration across the blood–brain barrier, understanding rare patterns of isolated intracranial evolution may guide improved strategies for long-term control of brain metastases and personalised treatment.

Keywords:
Central Nervous System Neoplasms; Neoplasm Recurrence; Magnetic Resonance Imaging; HER2 Gene Amplification;

POSTPARTUM ACUTE MULTIFOCAL CEREBRAL ISCHEMIA IN A YOUNG WOMAN: A CASE REPORT

Dorja Drenjančević1, Marija Kišmartin1, Dijana Rutnik Fot1,2, Eleonora Strujić1,2
1 Faculty of Medicine, J. J. Strossmayer University of Osijek, Osijek, Croatia
2 University Hospital Osijek, Department of Neurology, Osijek, Croatia
Presenting author: Dorja Drenjančević, dordrenjan@gmail.com

Introduction

Hereditary thrombophilia and patent foramen ovale (PFO) are recognized risk factors for stroke in young individuals, most commonly through paradoxical embolism. Transient prothrombotic states, such as pregnancy further increase the risk of acute cerebral ischemia.

Case Report

A 29-year-old woman presented with sudden-onset paresthesia involving the fingertips and palm of the left hand and left foot, accompanied by left-hand clumsiness. On neurological examination, left-sided hemiparesis was noted. Past medical history was significant for preeclampsia, with delivery by cesarean section one month prior to presentation. Magnetic resonance imaging revealed multifocal acute ischemic lesions involving right precentral and postcentral gyri, situated in the watershed region between the anterior and middle cerebral arteries, as well as smaller lesions in the right basal ganglia and the left frontal corticosubcortical area, suggestive of an embolic etiology. Computed tomography angiography of head and neck arteries showed no evidence of occlusion or dissection. Transesophageal echocardiography, including a bubble study, demonstrated a PFO measuring less than 1 mm in diameter. Given its very small size, which alone would be less likely to explain the multifocal embolic pattern, an underlying prothrombotic condition was suspected. Consequently, a thrombophilia gene panel was performed and identified a Factor V Leiden mutation, which together with the postpartum procoagulant state, suggested a possible multifactorial etiology for her cerebral ischemia. The patient was treated with rivaroxaban and acetylsalicylic acid, and PFO closure was advised. Although paradoxical embolism was the presumed mechanism, short-term dual therapy was considered justified in the setting of a postpartum prothrombotic state and a multifocal embolic pattern. On follow-up, she showed marked neurological improvement with functional recovery.

Conclusion

This case highlights the multifactorial etiology of acute multifocal cerebral ischemia in a young postpartum woman. Early recognition, comprehensive diagnostic work-up, timely management, and awareness of these risk factors in young women are essential to improve outcomes.

Keywords:
Ischemic Stroke; Patent Foramen Ovale; Postpartum Period; Preeclampsia; Thrombophilia, hereditary

CAN WE FORESEE THE FUTURE? MARKERS IN STROKE RECOVERY AND REHABILITATION

Lucija Falamić1, Sara Bonet2, Anamarija Soldo Koruga3,4
1 Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Osijek, Croatia
2 Primary Health Care Center of Osijek-Baranja County, Osijek, Croatia
3 Department of Neurology, University Hospital Center Osijek, Osijek, Croatia
4 Department of Educational Rehabilitation and Speech and Language Pathology, Faculty of Education J. J. Strossmayer University of Osijek, Osijek, Croatia
Presenting author: Lucija Falamić, lucija.falamic00@gmail.com

Introduction
Stroke is a major cause of disability, with rehabilitation outcomes being heterogeneous and difficult to predict. Early recognition of rehabilitation potential and concordant categorization of patients for individualized therapy relies on the development of a reliable set of markers. The goal of this review was to identify and categorize markers used in the context of post-stroke recovery and rehabilitation.

Materials and Methods
A systematic PubMed search was conducted using keywords “stroke", "markers", “prognosis” and “rehabilitation”. Full-text, English-only publications published within the last 5 years were included. A total of 73 publications were deemed eligible and screened for markers which were then sorted into following categories: biomarkers, radiological, functional, neuropsychological markers, or other; as well as recovery prognostic, rehabilitation prognostic or other, based on clinical purpose.

Literature Review
Out of 73 publications, biomarkers made up 43.5% of the total markers identified. Inflammatory derived biomarkers (CRP, IL-6, albumin) represent the most frequently investigated molecular predictors, along with mi-RNA, and metabolic markers (FT4/FT3 ratio, homocysteine, vitamin B12). Radiological markers constituted a substantial proportion of the reported predictors - 25%, predominantly featuring CSVD (Cerebral Small Vessel Disease) burden, and rs-fMRI (resting-state functional magnetic resonance imaging) connectivity metrics. Functional markers (15.22%) comprised commonly used motor and mobility scales, including Fugl-Meyer Assessment, gait speed, Berg Balance Scale, and electromyography-derived synergy indices. Among other, functional scales were also used as outcome measures. Neuropsychological markers, primarily cognitive assessments, were infrequently reported. Markers were used for rehabilitation prognosis in only 27% of instances, compared to 52% in case of acute recovery prognosis.

Conclusion
Despite the significant potential of markers for individualizing post-stroke rehabilitation, there’s a lack of standardized multimodal prognostic frameworks and very few studies address this area. Rehabilitation-focused prognostic research is essential, as it guides early initiation of the optimal rehabilitation approach, which is crucial for long-term outcomes.

Keywords:
Biomarkers; Neurorehabilitation; Prognosis; Recovery of Function; Stroke;

THE DIAGNOSTIC LABYRINTH OF INFLAMMATORY MYOPATHIES: ONE ELECTROMYOGRAPHIC PATTERN, MULTIPLE CAUSES

Nikolina Pilj[1], Lea Radić[1], Barbara Ravlić[1], Tihana Gilman Kuric[1,2]
[1]Josip Juraj Strossmayer University of Osijek, Faculty of Medicine, Osijek, Croatia
[2]Clinical Hospital Center Osijek, Department of Neurology, Osijek, Croatia
Presenting author: Nikolina Pilj, nikolina.pilj@gmail.com

Introduction

Inflammatory myopathies may show similar clinical and electrophysiological findings despite distinct causes. They typically affect proximal shoulder and pelvic muscles. Diagnosis relies on clinical, laboratory, electrophysiological, and sometimes histopathological findings.

Case Report

At the neurology department, two female patients were admitted simultaneously with a similar clinical presentation of proximal muscle weakness. An 80-year-old woman reported 12 months of difficulty performing daily activities, such as rising from a chair and climbing stairs, due to pelvic and proximal muscle weakness. She had well-controlled diabetes and hypertension. A 34-year-old woman reported three weeks of progressive proximal muscle weakness accompanied by dysphagia. Neither patient had muscle atrophy or sensory deficits, but muscles were tender on palpation. Fatigability was present, without fasciculations or oscillations. Neuromuscular junction tests were negative, and the neurological examination was unremarkable. Laboratory tests showed elevated creatine kinase, lactate dehydrogenase, and myoglobin in both patients. Sensory and motor nerve conduction studies were normal, excluding peripheral neuropathy or focal mononeuropathy as the cause of weakness. Needle electromyography demonstrated a myopathic pattern with short-duration, low-amplitude, polyphasic motor units and early recruitment, consistent with an inflammatory myopathy. Serology revealed recent Epstein-Barr virus infection in the younger patient, suggesting parainfectious myositis, a typically short-lasting and reversible condition that explains her rapid improvement with supportive care. Due to microcytic anemia and unintentional weight loss of 12 kilograms over one year, the older patient underwent abdominal evaluation, which revealed renal carcinoma, consistent with a paraneoplastic inflammatory myopathy associated with solid tumors. Her myositis improved following treatment of underlying malignancy with adjunctive corticosteroids.

Conclusion

Identical clinical and electromyographic features of inflammatory myopathy do not define a single entity; they may represent secondary inflammatory myopathies, from self-limited parainfectious to paraneoplastic processes requiring urgent oncologic evaluation. Electromyographic and neurological findings alone may not be sufficient to establish the underlying etiology, requiring further investigations.

Keywords:
Carcinoma, Renal Cell; Electromyography; Epstein-Barr Virus infections; Muscle Weakness; Myositis

HEADACHES AND NAUSEA IN PREGNANCY – PREGNANCY OR CEREBRAL VENOUS SINUS THROMBOSIS?

I**rma Nina Orlandić1,** Anđela Jurić1, Dora Komić1, Lucija Kovačić1, doc.dr.sc. Krešimir Čaljkušić dr. med.1,2
1 University of Split, School of Medicine, Split, Croatia
2 University Hospital Split, Department of Neurology, Split, Croatia
Presenting author: Irma Nina Orlandić, Irma.orlandic@gmail.com

Introduction

Cerebral venous sinus thrombosis (CVST) is a rare, life-threatening disorder where a thrombus forms in the dural venous sinuses. There is a risk of misdiagnosis due to an often subacute presentation. The symptoms can mimic other neurological diseases, such as migraine or subarachnoid haemorrhage headaches, as well as acute ischemic strokes.

Case Report

A 26-year-old, 9-weeks-pregnant woman presented to the emergency department with paresthesia and heaviness in her right arm. The symptoms began on the morning of admission and gradually progressed to her right leg. Two weeks prior, she had visited the gynaecology emergency department for severe vomiting, and again two days before admission for vomiting and headaches. She had also recently recovered from an influenza infection, a potential risk factor. The initial blood work showed elevated D-dimer levels (6.27 mg/l), suggesting a clot. The patient experienced two partial epileptic seizures, one of which progressed to a generalised seizure. An urgent brain magnetic resonance imaging (MRI) scan was performed. It showed areas of signal hyperintensity in multiple dural venous sinuses consistent with CVST. Levetiracetam and Heparin were administered. Further laboratory tests for haematological and rheumatological disorders, as well as serology, were all negative. A follow-up MRI scan showed regression of previously seen pathologies. The neurological deficits improved, but a slight right-sided hemiparesis remains. She was discharged after 18 days with Heparin and Levetiracetam with instructions to monitor blood pressure and to do a repeat brain MRI scan and MR venography in 2 months.

Conclusion

This case highlights that CVST, though rare in the first trimester of pregnancy, mustn't be overlooked when a pregnant patient presents with neurological symptoms. Pregnancy increases the risk of thrombosis and blurs the lines between physiological and pathological symptoms. The goal is to minimise complications such as death or disability.

Keywords:
Anticoagulants; Paresthesia; Pregnancy; Sinus Thrombosis, Intracranial; Venous Thrombosis

TWO IN ONE: A CASE REPORT ON COEXISTING MULTIPLE SCLEROSIS AND MYASTHENIA GRAVIS

Iva Kristić1, Marija Kišmartin1, Dorja Drenjančević1, Tea Mirošević Zubonja2
1Faculty of Medicine Osijek, J. J. Strossmayer University of Osijek, Croatia
2 Department of Neurology, University Hospital Centre Osijek, Croatia
Presenting author: Iva Kristić, iva.kristic2@gmail.com

Introduction
The overlap of multiple sclerosis (MS) and myasthenia gravis (MG) is rare. MS is a central nervous system autoimmune disorder and MG is an autoimmune disorder of the neuromuscular junction. MS affects approximately 35 per 100,000 people worldwide, while MG occurs in 200 per million individuals. The simultaneous appearance of these diseases could point to their shared autoimmune mechanisms, possibly related to common genetic or environmental factors. This could have an impact on therapeutic approaches.

Case Report
Our patient is a 56-year-old male with neurological history starting in 2003 with left-sided paresthesias and weakness. The symptoms resolved spontaneously and no definitive diagnosis was made. Ten years later similar symptoms reappeared. Brain magnetic resonance imaging (MRI) showed a hyperintense area interpreted as an old vascular lesion. In 2024, the patient was hospitalized with diplopia and left-sided ptosis. The diagnosis of ocular myasthenia gravis was suspected. Clinical improvement after administration of an acetylcholinesterase inhibitor confirmed the diagnosis. Negative finding of anti-AChR and anti-MuSK antibodies in the patients serum defined the diagnosis as seronegative MG. On neurological examination the patient showed abnormal reflexes leading to another brain MRI. This MRI revealed multiple demyelinating lesions defined as a central demyelinating process. Cerebrospinal fluid analysis showed positive oligoclonal bands supporting the diagnosis of MS. The patient received Mestinon and plasmapheresis for MG and pulse corticosteroid therapy was planned for MS. Cladribine, an immunomodulatory drug, was also initiated. It targets immune cells responsible for pathogenic antibody production, potentially modulating both autoimmune disorders. At present, the patient reports clinical improvement.

Conclusion:
Diagnosing a single autoimmune disease is challenging and identifying two in the same patient requires a thorough investigation. The coexistence of MS and MG should be considered in patients with atypical or fluctuating weakness. Early clinical suspicion followed by timely diagnostic work-up and treatment selection may improve patient outcomes.

Keywords:
Autoimmune Diseases; Immunology; Magnetic Resonance Imaging; Multiple Sclerosis; Myasthenia Gravis

AUTONOMIC DYSFUNCTION IN HYPERMOBILE EHLERS-DANLOS SYNDROME PRESENTING AS POTS; CASE REPORT

Maritea Arelić1, Rea Divjanović1, Valentino Rački2,3
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Department of Neurology, Clinical Hospital Centre Rijeka, Rijeka, Croatia
3Department of Neurology, Faculty of Medicine, University of Rijeka, Rijeka, Croatia
Presenting author: Maritea Arelić, marelic@student.uniri.hr

Introduction

Hypermobile Ehlers–Danlos syndrome (hEDS) is a hereditary connective tissue disorder characterized by generalized joint hypermobility, chronic musculoskeletal pain, and systemic manifestations. Connective tissue laxity affects vascular tone and baroreceptor function, contributing to autonomic dysregulation and predisposing patients to postural orthostatic tachycardia syndrome (POTS). POTS frequently coexists with hEDS and may remain unrecognized for years due to nonspecific symptoms despite normal findings on standard cardiovascular workups. Early recognition is essential to reduce diagnostic delay and functional impairment.

Case Report

A 29-year-old patient reported recurrent palpitations since 2018 and was initially evaluated for suspected arrhythmia. Following COVID-19 infection in 2022, the patient developed orthostatic intolerance with recurrent syncope, severe fatigue, gastrointestinal symptoms, and chronic pain. Tilt-table testing confirmed POTS with concomitant vasovagal syncope.
Further evaluation fulfilled clinical criteria for hEDS, a diagnosis based on clinical findings due to the absence of a known genetic marker. Family history was notable for syncope in first-degree relatives. The patient underwent multiple specialist evaluations, including cardiology, gastroenterology, and psychiatric assessment.
Physical examination showed generalized joint hypermobility (Beighton score 6/9). Active standing test demonstrated heart rate increase from 86/min supine to 128/min upright without significant blood pressure drop. Continuous monitoring revealed variability between 46 and 158 bpm.
Non-pharmacological treatment (increased fluid (2–3 L/day) and salt intake (8–10 g/day), compression garments, graded exercise, and smoking cessation) improved heart rate variability (48–103 bpm) and symptoms, without the need for pharmacological therapy.

Conclusion

This case highlights the systemic nature of hEDS and its association with autonomic dysfunction. POTS should be considered in patients with joint hypermobility and unexplained orthostatic symptoms. Early recognition, clinical criteria, and multidisciplinary management improve outcomes. Greater awareness is needed to avoid misdiagnosis, as patients with hEDS and POTS often undergo extensive evaluation and may have symptoms misattributed to psychiatric conditions due to their nonspecific presentation.

Keywords: Ehlers-Danlos Syndrome; Postural Orthostatic Tachycardia Syndrome; autonomic Nervous System Diseases; Joint Hypermobility; Syncope

GRAY MATTER HETEROTOPIA WITHOUT SEIZURES: CLINICAL DILEMMAS IN ANTICONVULSANT THERAPY DECISION-MAKING

Sara Mrkonjić1, Sonja Palija1, Igor Prpić2, Franka Kovačević2
1Department of Pediatrics, Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2Clinical Hospital Center Rijeka, Rijeka, Croatia
Presenting author: Sara Mrkonjić, mrkonjicsara@gmail.com

Introduction

Gray matter heterotopia (GMH) is a malformation of cortical development characterized by ectopic neuronal clusters, with epilepsy occurring in 69–82% of patients, while 19% remain seizure-free. This creates a clinical dilemma: whether to initiate or continue antiseizure medication (ASM) in patients with structural lesions who have never had seizures or who achieve long-term seizure freedom. This review examines available evidence through two pediatric cases.

Materials and Methods

Two patients with right frontal GMH were reviewed. Patient 1 had five seizures between ages 13 and 14; after starting lamotrigine, he achieved 10 years of seizure freedom, raising the question of therapy continuation. Patient 2 was diagnosed incidentally at age 10 on magnetic resonance imaging (MRI) for headache; he had focal electroencephalography (EEG) abnormalities but no seizures. Levetiracetam was initiated but led to anxiety-depressive symptoms. A literature search was conducted using PubMed and guideline databases. Inclusion criteria were English-language studies, clinical guidelines, and systematic reviews on ASM use in pediatric patients with structural brain lesions or epilepsy. Exclusion criteria included adult-only populations, non-peer-reviewed sources, and case reports. A total of 14 studies and guideline documents were reviewed.

Literature Review

Guidelines from the American Academy of Neurology and Italian League Against Epilepsy do not recommend prophylactic ASM in patients with structural lesions without seizures, while withdrawal may be considered after ≥2 years of seizure freedom. Structural lesions and EEG abnormalities increase relapse risk but do not justify treatment in asymptomatic patients. Levetiracetam is associated with behavioral adverse effects in ~37.6% of pediatric patients, limiting long-term use.

Conclusion

Evidence-based guidelines for prophylactic therapy in seizure-free patients are lacking, and current recommendations are extrapolated from brain tumor literature and have limited applicability. The significance of isolated EEG abnormalities remains uncertain, and whether focal epileptiform changes alone justify treatment is debated. Clinicians must balance seizure prevention against long-term ASM adverse effects, particularly psychiatric symptoms with levetiracetam in adolescents. Uncertainty also persists regarding ASM withdrawal after prolonged seizure freedom in patients with structural lesions. Finally, GMH heterogeneity supports individualized rather than uniform management.

Keywords: Anticonvulsants; Electroencephalography; Epilepsy; Gray matter; Heterotopia

THC EXPOSURE AND FOCAL STATUS EPILEPTICUS IN STRUCTURAL EPILEPSY

Dorotea Živalj1, Gabrijela Šoštarić1, Lana Oštro2, Petra Črnac2
1 University of Rijeka, Faculty of Medicine, Rijeka, Croatia.
2 Clinical Hospital "Sveti Duh", Department of Neurology, Zagreb, Croatia
Presenting author: Dorotea Živalj, dorotea.zivalj@gmail.com

Introduction
Structural focal epilepsy carries an increased risk of seizure clustering and status epilepticus, particularly in the presence of precipitating factors. Aim of this case report is to emphasize recreational marijuana use, especially THC-dominant exposure, as a potential seizure trigger and contribute to destabilization of seizure control in patients with structural brain lesions.

Case Report
We present a 41-year-old male with structural focal epilepsy secondary to severe traumatic brain injury sustained 10 years earlier, treated with decompressive craniectomy and evacuation of an intracranial hematoma. His habitual seizures were focal with impaired awareness, occasionally progressing to bilateral tonic–clonic seizures. He was treated with oxcarbazepine (Trileptal), although adherence to therapy was irregular. The patient reported intermittent recreational marijuana use. He was admitted due to a cluster of 2–4 seizures on the day of admission, including focal impaired awareness seizures and bilateral tonic–clonic seizures. The seizures were terminated with intravenous diazepam, followed by postictal confusion. On admission, he was somnolent but arousable, partially oriented, and without focal neurological deficits. Electroencephalography demonstrated left frontotemporal epileptiform activity with secondary generalization, consistent with focal status epilepticus. Neuroimaging revealed no new structural lesions. Laboratory evaluation showed marked elevation of creatine kinase, consistent with seizure-related muscle injury, as well as mild transient elevation of liver enzymes and reduced folate levels. A mild inflammatory response was also present. Toxicological screening was positive for tetrahydrocannabinol (THC) and benzodiazepines (administered during treatment). No other substances were detected. Levetiracetam was introduced in addition to oxcarbazepine, leading to clinical stabilization. The patient was discharged in stable condition with dual antiepileptic therapy and strong recommendations for strict medication adherence and avoidance of marijuana use.

Conclusion
In this patient with structural epilepsy, seizure clustering and focal status epilepticus occurred in the context of irregular antiepileptic therapy and confirmed THC exposure, without evidence of new structural or metabolic triggers. Although a direct causal relationship cannot be definitively established, this case suggests that recreational marijuana use may act as a potential modifiable risk factor contributing to seizure destabilization in susceptible individuals.

Keywords:
Epilepsy, Focal; Status Epilepticus; Tetrahydrocannabinol; Drug Interactions; Medication Adherence

CEREBRAL VENOUS SINUS THROMBOSIS IN AN INTRAVENOUS DRUG USER WITH HEMORRHAGIC COMPLICATIONS

Andrijana Andrešić1*, Luka Barać1, Ana Bardić1, Tihana Majetić1, Petra Pupić-Bakrać2
1 Faculty of Medicine, University of Rijeka, Rijeka, Republic of Croatia
2 General Hospital Zadar, Department of Neurology, Zadar, Republic of Croatia
***Presenting author:** Andrijana Andrešić, andrijana.andresic@gmail.com

Introduction

Cerebral venous sinus thrombosis (CVST) is a rare cause of stroke, accounting for less than 1% of all cerebrovascular events. Clinical presentation is variable and may include seizures, focal neurological deficits, or altered consciousness. Venous infarction is often complicated by intracerebral hemorrhage, creating a therapeutic dilemma regarding anticoagulation. Early recognition is crucial, as timely initiation of anticoagulant therapy improves outcomes, even in the presence of hemorrhage.

Case Report

A 42-year-old man was brought to the emergency department after a witnessed fall followed by generalized tonic–clonic seizures. On arrival, he was somnolent, with oxygen saturation of 87%. A second seizure occurred during transport and was treated with intravenous diazepam. Neurological examination revealed impaired consciousness, right gaze deviation, left-sided weakness, and hemispatial neglect. Emergency brain computed tomography (CT) revealed a right occipital intracerebral hematoma with surrounding edema and suspected sulcal subarachnoid hemorrhage. CT venography showed thrombosis involving the right transverse sinus, sigmoid sinus, and internal jugular vein, while arterial imaging was normal. The left-sided weakness resolved within hours, consistent with Todd's paralysis. The patient was admitted to the intensive care unit due to respiratory insufficiency, metabolic acidosis, hyperammonemia, and positive toxicology for cocaine and amphetamines. These substances are known to promote a prothrombotic state through endothelial dysfunction and platelet activation, likely contributing to the development of CVST. Therapeutic low-molecular-weight heparin and antiepileptic therapy were initiated, and neurosurgical treatment was not indicated.Further evaluation demonstrated heterozygosity for the prothrombin G20210A mutation and plasminogen activator inhibitor-1 (PAI-1) polymorphism, both associated with increased thrombotic risk and likely acting synergistically with substance abuse in the pathogenesis of CVST. Infective endocarditis was excluded by negative blood cultures and normal transesophageal echocardiography. Follow-up imaging demonstrated regression of the hemorrhage with persistent venous thrombosis.
After clinical stabilization, the patient was discharged without neurological deficits on oral anticoagulant and antiepileptic therapy, although he declined complete diagnostic workup and follow-up.

Conclusion

CVST should be considered in patients presenting with seizures and intracerebral hemorrhage, especially when imaging is atypical for primary hypertensive bleeding. Anticoagulation remains the cornerstone of treatment despite intracranial hemorrhage and should not be delayed once the diagnosis is confirmed. Identification of underlying risk factors, including thrombophilia and substance abuse, is essential for guiding long-term management and preventing recurrence.

Keywords:
Anticoagulants; Cerebral Hemorrhage; Intracranial Thrombosis; Sinus Thrombosis, Intracranial; Venous Thrombosis

ANTI-NMDAR ENCEPHALITIS

Dora Komić1, Anđela Jurić1, Angela Kalpić1, Irma Nina Orlandić1, prim. Eugenija Marušić, dr.med.1,2
1 - University of Split, School of Medicine, Split, Croatia
2 - University Hospital of Split, Department of Pediatrics – Institute of Neurology, Split, Croatia
Presenting author: Dora Komić, dorakomic@gmail.com

Introduction

Autoimmune encephalitis refers to acute to subacute, progressive inflammation of the brain associated with antibodies against neuronal cell surface and synaptic proteins, most commonly being anti-NMDAR encephalitis. Complex neuropsychiatric symptoms are the most common presenting features. Early diagnosis and prompt immunotherapy treatment can be beneficial for the outcome.

Case Report

A seventeen-year-old girl was hospitalized for dysarthria and skin paresthesia. Upon admission, an extensive diagnostic evaluation was performed. Neurological examination revealed cortical language processing impairment, cognitive dysfunction, comprehension difficulties, palmar hyperhidrosis, and reduced gross muscle strength. Psychiatric manifestations or epileptic seizures were not present. The EEG was low-voltage and dysrhythmic, without epileptogenic elements, and the fundus was normal. Laboratory findings were normal except for decreased vitamin B12 and folate levels, which were corrected with supplementation. Cerebrospinal fluid analysis revealed pleocytosis, type 2 oligoclonal IgG bands, and negative multiplex PCR for common encephalitis and meningitis pathogens. Brain MRI revealed bilateral occipital peritrigonal hyperintensities suggestive of possible glial lesions. Serologically, the finding of intrathecal synthesis of IgG on EBV was also observed, but the clinical significance was assessed as questionable. Diagnostic work-up has been extended to rule out paraneoplastic syndrome. Finally, anti-NMDAR antibodies were detected in serum and cerebrospinal fluid, which confirmed the diagnosis of autoimmune anti-NMDAR encephalitis. Treatment was initiated with pulsed corticosteroid therapy, followed by oral corticosteroids and IVIG. Due to neurological deterioration, plasmapheresis with repeated IVIG was initiated, resulting in significant clinical improvement.

Conclusion

This case stands out because it presents anti-NMDAR encephalitis with an atypical presentation without dominant psychiatric symptoms and epileptic seizures. The importance of timely recognition of the disease and the need for detailed and extensive diagnostic examination is emphasized in order to prove the correct diagnosis and apply therapy for a clinically better outcome.

Keywords

Encephalitis, Anti-N-Methyl-D-Aspartate Receptor Encephalitis, Dysarthria, Paresthesia, Immunotherapy

NEUROSURGERY

24th APRIL - 26th APRIL 2026

PTERIONAL CRANIOTOMY IN THE TREATMENT OF AN UNRUPTURED MIDDLE CEREBRAL ARTERY ANEURYSM - A CASE REPORT

Karla Kleščić*1, Loreta Horvatek1, Lucija Mitrović1, Vinka Cvetković1, Hrvoje Šimić (mentor)1,2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Centre Rijeka, Department of Neurosurgery, Rijeka, Croatia
Presenting author: Karla Kleščić, karla.klescic2@gmail.com

Introduction

Intracranial aneurysms occur in approximately 2–3% of the general population and are most commonly located in the anterior cerebral circulation. Middle cerebral artery (MCA) aneurysms account for about 20–30% of all intracranial aneurysms. Although small unruptured aneurysms are frequently asymptomatic, their rupture may lead to subarachnoid hemorrhage with significant mortality and morbidity. Treatment decisions are based on aneurysm size, morphology, location, and individual patient risk factors. Microsurgical clipping via a pterional craniotomy remains a standard treatment option for MCA aneurysms due to excellent exposure and reliable neck occlusion.

Case Report

A 68-years old female patient with a history of arterial hypertension, obesity, and smoking was referred for evaluation of a confirmed left MCA aneurysm. MSCT angiography demonstrated an aneurysm located at the M2 segment of the left MCA measuring 2.2 mm with a 1.3 mm neck, oriented laterally. Elective surgical treatment was indicated. Under general endotracheal anesthesia, an elective pterional craniotomy with microsurgical aneurysm clipping was performed. The intraoperative course was uneventful. Postoperatively, the patient was admitted to the intensive care unit, initially sedated and mechanically ventilated. She remained hemodynamically stable without vasopressor support. Neurological examination showed isochoric and reactive pupils with no new neurological deficit. The postoperative course was without complications.

Conclusion

Microsurgical clipping via pterional craniotomy represents a safe and effective treatment option for MCA aneurysms, particularly in morphologically suitable lesions. Timely elective treatment of unruptured aneurysms may prevent potentially fatal complications and ensure a favorable outcome.

Keywords:
Intracranial Aneurysm; Middle Cerebral Artery; Craniotomy; Microsurgery; Neurosurgical Procedures

CEREBRAL CONTUSIONS AND MIDFACE TRAUMA IN THE ELDERLY: MULTIDISCIPLINARY CONSERVATIVE CARE WITH PROPHYLAXIS

Lana Paulić1*, Klara Pintarić1, Laura Novak1, Iva Paldi Marijetić2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Department of Anesthesiology, University Hospital Sveti Duh, Zagreb, Croatia
***Presenting author:** Lana Paulić, lana.paulic0@gmail.com

Introduction

Multiple viscerocranial fractures are often associated with traumatic brain injury (TBI), especially in elderly patients, where cerebral atrophy and comorbidities increase the risk of delayed deterioration. In the absence of surgical indications, management is usually conservative. Due to the proximity of paranasal sinuses to the anterior skull base, coordinated care between neurosurgeons and maxillofacial surgeons is essential, while the indication and duration of antibiotic prophylaxis in sinus fractures remain controversial.

Case Report

63-year-old female patient presented after a fall down the stairs. On admission, she was alert (Glasgow Coma Scale (GCS) 15), hemodynamically stable, and denied loss of consciousness. Radiological assessment (X-ray and multislice computed tomography) revealed multifragmentary distal radius fracture, nasal bone fractures, maxillary and sphenoid sinus fractures, and cerebral contusions without mass effect or midline shift. Despite these intracranial findings, the patient exhibited no neurological deficit, and ophthalmological evaluation showed no visual impairment, indicating no need for immediate neurosurgical or ophthalmic intervention. Given the involvement of the paranasal sinuses and potential intracranial proximity, antibiotic prophylaxis was initiated with clindamycin (3 × 600 mg IV) due to penicillin allergy, balancing the risk of infection against the lack of overt contamination. The patient's clinical and neurological course remained stable, and she was discharged with instructions for follow-up by maxillofacial surgery.

Conclusion

In elderly patients with multiple viscerocranial fractures and concomitant TBI, infections remain a significant concern. While systemic antibiotic prophylaxis can reduce infection risk, the optimal indication and duration—particularly in closed sinus fractures—remain debated. This case demonstrates that, when neurological status is stable and GCS is high, conservative management of cerebral contusions is safe. It also highlights the need to balance prophylactic benefits against the risk of antimicrobial resistance, emphasizing individualized selection of antibiotic type and therapy duration within a multidisciplinary care framework.

Keywords:
Antibiotic Prophylaxis; Conservative Treatment; Craniocerebral Trauma; Multidisciplinary Approach; Paranasal Sinuses

SMALL IMPACT, MAJOR CONSEQUENCE: PROGRESSIVE INTRACRANIAL HEMORRHAGE AFTER MINOR TRAUMA IN THE ELDERLY

Loreta Horvatek1, Veronika Hanić1, Karla Kleščić1, Hrvoje Šimić1,2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Centre Rijeka, Department of Neurosurgery, Rijeka, Croatia
Presenting author: Loreta Horvatek, horvatekloreta@gmail.com

Introduction

Head trauma in elderly patients presents a significant clinical challenge due to brain atrophy, vascular fragility, and multiple comorbidities. Even minor trauma without loss of consciousness may result in substantial intracranial hemorrhage. Traumatic subdural hematoma (SDH) and subarachnoid hemorrhage (SAH) are associated with high morbidity and mortality in this population and often require urgent neurosurgical evaluation.

Case Report

A 95-year-old woman was admitted to the emergency department after experiencing dizziness while standing and striking the occipital region of her head against a door. She denied loss of consciousness, nausea, vomiting, or falling. On admission, she was hypertensive but hemodynamically stable, fully conscious, oriented, with a Glasgow Coma Scale (GCS) score of 15. Physical examination revealed an actively bleeding T-shaped occipital scalp laceration with a surrounding hematoma. Laboratory analysis showed anemia with normal coagulation parameters. Non-contrast brain computer tomography revealed a left supratentorial extra-axial crescent-shaped hematoma, consistent with acute exacerbation of a subacute SDH. A small interhemispheric acute SDH and minimal SAH in the left Sylvian fissure were also present, causing mild mass effect without herniation. The scalp wound was sutured, and the patient was admitted for neurosurgical monitoring. Follow-up CT revealed progression of the SDH, while the SAH remained stable. Neurological status subsequently deteriorated with reduced responsiveness. Due to progression and clinical worsening, urgent neurosurgical intervention included evacuation of the SDH, while the traumatic SAH was managed conservatively. The patient was classified as IV E according to American Society of Anesthesiologists (ASA) Physical Status Classification.

Conclusion

This case demonstrates that even trivial head trauma in elderly patients can result in clinically silent yet progressive intracranial hemorrhage, despite a normal initial neurological examination (GCS 15). The observed acute-on-subacute subdural hematoma with delayed deterioration emphasizes that a reassuring initial presentation does not exclude subsequent worsening. Therefore, early CT imaging must be complemented by routine follow-up neuroimaging and close clinical observation. This case highlights the need for a low threshold for hospital admission and timely neurosurgical involvement, as recognition of hematoma progression directly impacts patient outcome.

Keywords:
Aged; Craniocerebral Trauma; Emergency Treatment; Hematoma, Subdural; Neurosurgery; Subarachnoid Hemorrhage

THE IMPACT OF INTRAOPERATIVE INDOCYANINE GREEN VIDEO-ANGIOGRAPHY IN CEREBROVASCULAR NEUROSURGERY – A SCOPING REVIEW

Tomislava Tretinjak1, Lara Uglik1, Marko Roso1, Marija Kišmartin1, Lara Rončević1
1 Faculty of Medicine Osijek, University of J. J. Strossmayer Osijek, Osijek, Croatia
Presenting author: Tomislava Tretinjak, tomislava.tretinjak@gmail.com

Introduction
Indocyanine green video-angiography (ICG-VA) is a simple intraoperative tool that provides real-time vascular imaging. This review aims to evaluate the impact of ICG-VA on intraoperative decision-making and outcomes in cerebrovascular neurosurgery.

Materials and Methods
This scoping review was conducted following the PRISMA-ScR guidelines. PubMed/MEDLINE database was searched for retrospective observational studies published in the last decade. Included studies evaluated ICG-VA's effect on intraoperative modification of surgical strategy and postoperative outcomes. 10 relevant articles fit the inclusion criteria, and data on 433 procedures were synthesized descriptively.

Literature Review
Intraoperative ICG-VA influenced surgical strategy and suboptimal results correction in multiple neurovascular indications. In patients with World Federation of Neurosurgical Societies (WFNS) grade I-II subarachnoid hemorrhage undergoing middle cerebral artery (MCA) aneurysm clipping, ICG-VA-assisted clip positioning was associated with reduced postoperative morbidity by identifying vessel kinking and compromised branch perfusion following the first clip placement. In arteriovenous malformation surgery, ICG-VA visualized superficial feeders and draining veins, although visualization was limited for deep-seated lesions due to restricted fluorescence penetration. Detected venous flow at the presumed end of the procedure accurately indicated a hidden arterial feeder. During extracranial-intracranial and intracranial-intracranial bypass procedures, ICG-VA enabled intraoperative graft patency assessment followed by the same-act revision of insufficient anastomoses, prolonging overall operative time by less than 10 minutes. Additionally, ICG-VA assisted in identifying occult feeders in hemangiomas. For the posterior approach to intramedullary spinal cord tumors, ICG-VA differentiated the posterior spinal arteries from veins, demarking the pial venous plexus and optimizing the complex vascular identification.

Conclusion
This review illustrates how ICG-VA provided real-time intraoperative assessment of vascular flow, followed by clip repositioning, detection of residual feeders, and verification of bypass graft patency, supporting its association with improved procedural safety and postoperative outcomes.

Keywords:
Aneurysm; Angiography; Arteriovenous Malformations; Indocyanine Green; Neurosurgery

A CASE REPORT OF TETRAPLEGIA FOLLOWING UNRECOGNIZED SPINAL CANAL STENOSIS

Leon Davidović1, Matias Andjal1, Filip Ovničević1, Jakov Kovačević1, Ivana Haršanji Drenjančević1,2
1 Faculty of Medicine, Josip Juraj Strossmayer University of Osijek, Croatia
2 Department of Anesthesiology, Resuscitation, and Intensive Care, University Hospital Center Osijek, Croatia
Presenting author: Leon Davidović, leon.davidovic2001@gmail.com

Introduction
Cervical spinal canal stenosis is the narrowing of the spinal canal in the neck, most commonly due to age-related degeneration, which may compress the spinal cord and predispose to severe neurological injury even after minor trauma. Objective of this case report is to highlight the potential for catastrophic spinal cord injury following seemingly minor cervical trauma in patients with unrecognized cervical canal stenosis and to raise awareness of risks associated with low-energy whiplash injuries during virtual reality gaming.

Case Report
We report a 62-year-old male with previously unrecognized cervical spinal canal stenosis who developed tetraplegia following a low-energy whiplash injury. He fell backwards while playing a video game using virtual reality equipment and struck his head, resulting in sudden hyperflexion–hyperextension of the cervical spine. He had not previously reported typical symptoms of cervical stenosis, such as cervicalgia, incoordination, or limb weakness. On the afternoon of the injury, he noticed neck pain, followed by paresthesia and weakness in his arms, and presented to the emergency department that evening. Computed tomography of the brain and spine revealed no fractures, while emergency cervical magnetic resonance imaging demonstrated spinal cord edema at C3/4 and C4/5 and marked stenosis from C3 to C7. Despite conservative management, his neurological status deteriorated, leading to surgical decompression. Postoperatively, patient developed respiratory failure requiring tracheostomy and ventilator-associated pneumonia due to multidrug-resistant pathogens, resulting in profound disability.

Conclusion
This case illustrates how occult cervical spinal canal stenosis can transform seemingly minor cervical trauma into catastrophic spinal cord injury. Cervical stenosis often remains unrecognized because early symptoms such as neck pain, hand clumsiness, gait imbalance, or paresthesias are mild or attributed to normal aging. Proactive assessment of these warning signs, cervical imaging in at-risk patients, and counseling about high-risk activities like virtual reality gaming may reduce irreversible neurological disability after low-energy cervical injury.

Keywords: Cervical Vertebrae; Spinal Cord Injuries; Spinal Stenosis; Tetraplegia; Virtual Reality

MINIMALLY INVASIVE, MAXIMALLY EFFECTIVE: ENDOSCOPIC BIPORTAL UNILATERAL DISCECTOMY IN L5/S1 RADICULOPATHY

Rajna Međimurec1*, Dora Mladenić2, Hrvoje Šimić3
1,2 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
3 Clinical Hospital Center Rijeka, Department of Neurosurgery, Rijeka, Croatia
Presenting author: Rajna Međimurec1, rajna.medimurec@gmail.com

Introduction
Lumbar disc herniation is one of the most common causes of radiculopathy and lumbosciatic pain. When conservative therapy fails, surgical decompression is indicated. Although conventional open microdiscectomy remains the standard procedure, minimally invasive techniques have become very popular. Endoscopic biportal unilateral discectomy (EBUD) is a minimally invasive approach that uses an arthroscopic system. Recent studies suggest that this approach is associated with reduced postoperative pain, less tissue damage, shorter hospitalization and improved postoperative recovery compared to standard techniques.

Case Report
A 36-year-old female patient was admitted for surgical treatment due to persistent right-sided lumboischialgia lasting four months, refractory to conservative management. The pain radiated along the right lower extremity. Magnetic resonance imaging (MRI) of the lumbosacral spine revealed a right-sided L5/S1 disc extrusion compressing the corresponding nerve root. Neurological assessment showed signs of right L5 radiculopathy, with preserved global muscle strength and intact sphincter control. Due to clinical and radiological correlation, surgery was indicated. In February 2026, the patient underwent EBUD at the L5/S1 level under general anesthesia. An endoscopic interlaminar approach was used to perform decompression and remove the herniated disc fragment. This technique uses 2 independent channel endoscopies which provide a clear and magnified surgical field that improves operational flexibility, enabling precise removal of herniated disc fragments while preserving surrounding anatomical structures. The postoperative course was without complications and primary wound healing. Early mobilization with lumbosacral orthosis and physical therapy was initiated. The patient reported significant pain reduction and was discharged home in stable condition with recommendations for activity modification, analgesics as needed, orthosis use during verticalization, and suture removal 12 days postoperatively.

Conclusion
EBUD proved to be a safe and effective treatment for this case of lumbar disc herniation with radiculopathy. This minimally invasive approach resulted in rapid postoperative recovery, absence of complications and promising early outcomes in this patient.

Keywords:
Diskectomy, Endoscopy, Intervertebral Disc Disease, Radiculopathy

CONGENITAL FIBROSARCOMA OF THE CRANIOFACIAL REGION: A NEONATAL CASE REPORT

Eric Kovačina1*, Ana Hrkać Pustahija2
1 School of Medicine, University of Zagreb, Zagreb, Croatia.
2 Department of Diagnostic Neuroradiology, University Hospital Centre Zagreb, Zagreb, Croatia; School of Medicine, University of Zagreb, Zagreb, Croatia.
Presenting author: Eric Kovačina, email: ekovacina@gmail.com

Introduction
Infantile fibrosarcoma (IFS) is a rare soft tissue malignancy of early childhood, presenting as a rapidly enlarging mass with locally aggressive growth and low metastatic potential. We present a case of a newborn with rapidly enlarging mass located in the right temporozygomatic region. Radiological work-up including ultrasound and MRI depicted intracranial and extracranial heterogeneous mass and suggested sarcoma as the most probable preoperative diagnosis. Integrated pathohistological diagnosis obtained after surgical resection was infantile fibrosarcoma with *ETV6-NTRK3* gene fusion.

Case Report
A male newborn presented with a painless 2 cm swelling over the right zygomatic bone. The mass was progressively growing, and by the day 13 of life measured up to 3.5 × 3.5 cm. Ultrasound showed a well-circumscribed, heterogeneously structured and vascularized solid mass. MRI revealed a large, aggressive tumour of the anterior and middle skull base with significant intra- and extracranial extension. The lesion was hypercellular, characterized by T2-hypointensity and low ADC values, and haemorrhagic, showing intense contrast enhancement. There was marked mass effect, including extensive perilesional oedema, midline shift, and obstructive hydrocephalus. Locally, the tumour caused widespread bone destruction and extended into the temporal and infratemporal regions, with no evidence of orbital invasion. Neurosurgical resection was performed, and final integrated pathohistological diagnosis was infantile fibrosarcoma with *ETV6-NTRK3* gene fusion.

Conclusion
This case highlights the importance of early multidisciplinary evaluation with advanced imaging, neurosurgical resection and comprehensive molecular testing in neonates with rapidly progressive masses. Accurate differentiation from other high-grade paediatric sarcomas is critical, as prognosis and therapeutic strategies differ significantly. Timely radical surgical resection enabled effective cerebral decompression and local disease control, while molecular confirmation provided a basis for risk stratification and guidance of further oncologic management, including consideration of targeted therapies.

Keywords:
Brain Neoplasms; ETV6-NTRK3 Fusion Protein; Fibrosarcoma; Infant; Neurosurgical Procedures

RIGHT MESIAL TEMPORAL LOBE EPILEPSY: OUTCOME AFTER SELECTIVE AMYGDALOHIPPOCAMPECTOMY

Klara Grgorinić1, Lukas Grbac-Lacković1, Željka Petelin Gadže2, Andreja Bujan Kovač2
1 University of Zagreb, School of Medicine, Zagreb, Croatia
2 Department of Neurology, University Hospital Centre Zagreb, University of Zagreb, Zagreb, Croatia
Presenting author: Klara Grgorinić1, klaraygrgorinic@gmail.com

Introduction

Mesial temporal lobe epilepsy (mTLE) associated with hippocampal sclerosis (HS) represents one of the most frequent indications for surgical intervention in patients with drug-resistant epilepsy. Clinical decision-making regarding surgical resection and the subsequent titration of anti-seizure medication remains challenging, particularly in patients presenting with complex medical and surgical comorbidities. This case analyzes the diagnostic pathway and successful surgical outcome of a patient with right-sided mTLE who achieved long-term seizure freedom following selective amygdalohippocampectomy.

Case Report

We present a case of a 35-year-old female who suffered a gunshot injury at the age of 20 requiring multiple abdominal and thoracic surgeries and prolonged intensive care unit (ICU) treatment. Two years post-trauma, she began experiencing focal impaired-awareness seizures characterized by an epigastric aura, déjà-vu, past recollections, sensory hypersensitivity and speech arrest, notably without postictal confusion. A formal diagnosis was established seven years after symptom onset when EEG revealed right frontotemporal abnormalities. Initially, carbamazepine (CBZ) provided control; however, seizures progressed to a drug-resistant state despite trials of CBZ, valproate, oxcarbazepine (OXC) and levetiracetam. 3 Tesla MRI demonstrated right mesial temporal sclerosis and superficial hemosiderosis, likely as a sequela of prior trauma and prolonged ICU treatment. PET-CT imaging showed right parieto-temporal hypometabolism, while prolonged video-EEG monitoring captured typical focal seizures with right frontotemporal onset. In October 2022, she underwent a right selective amygdalohippocampectomy via a transsylvian microsurgical approach. Neuropsychological assessment before surgery showed average verbal and non-verbal abilities with mild deficits in attention and processing speed, which remained unchanged postoperatively. She has remained seizure-free on OXC monotherapy, with residual right temporal interictal EEG abnormalities and no evidence of new cognitive decline.

Conclusion

This case illustrates that selective amygdalohippocampectomy can provide durable seizure control without inducing new cognitive or neurological deficits. Successful epilepsy surgery can significantly improve clinical stability and daily functioning, even in patients with long-term drug-resistant epilepsy.

Keywords:
Carbamazepine; Drug Resistant Epilepsy; Epilepsy, Temporal Lobe; Hippocampal Sclerosis; Neurosurgery

NEUROPLASTICITY AND FUNCTIONAL RECOVERY FOLLOWING RESECTION OF PAPILLARY GLIONEURONAL TUMOR: A CASE REPORT

Lucija Gazić1, Marina Raguž 1,2
1 Faculty of Medicine, Catholic University of Croatia, Zagreb, Croatia
2 Department of Neurosurgery, University Hospital Dubrava, Zagreb, Croatia
Presenting author: Lucija Gazić, lucija.gazic@gmail.com

Introduction

Papillary glioneuronal tumor (PGNT) is a WHO Grade 1 neoplasm with a low incidence rate. This report describes an unusual case of complete motor recovery following severe postoperative hemiparesis, highlighting the role of neuroplasticity and early rehabilitation in achieving functional recovery after periventricular tumor resection.

Case Report

A 37-year-old female presented with vertigo and gait instability. MRI revealed a lesion at the trigone and posterior body of the left lateral ventricle extending into the parietal lobe. The periventricular location placed the tumor in proximity to corticospinal fibers of the corona radiata, which may explain the postoperative motor deficit. She underwent neuronavigation-guided biparietal osteoplastic craniotomy with complete tumor resection and ventricular drain placement.

Histopathology showed a moderately cellular glial neoplasm with absent mitotic activity and a Ki-67 index of ~1%. Tumor cells were positive for GFAP and synaptophysin, indicating mixed glioneuronal lineage and favoring PGNT. DNET was considered due to similar morphology and low Ki-67, but was less consistent with synaptophysin positivity and periventricular location. Postoperatively, severe right-sided hemiparesis developed, consistent with resection within a functionally eloquent periventricular region, exacerbated by reactive edema. Follow-up MRI excluded ischemia, showing perifocal edema and subdural collections. Perfusion imaging demonstrated segmental neovascularization without permanent vascular compromise, suggesting transient white matter disruption rather than irreversible injury. Intensive inpatient rehabilitation was initiated early. By discharge, the patient regained symmetrical anti-gravity motor strength in all extremities, with residual mild right peroneal weakness requiring an ankle-foot orthosis.

Conclusion

Severe postoperative deficits following low-grade periventricular tumor resection can be reversible in the absence of ischemia. Consequently, early neuroimaging to differentiate edema from ischemic injury is critical, as it directly guides rehabilitation decisions and recovery expectations. This case reinforces that when neuroplasticity is met with early, structured rehabilitation, even severe motor deficits can resolve into meaningful functional recovery.

Keywords:
Brain Neoplasms; Neuronal Plasticity; Parietal Lobe; Neurological Rehabilitation; Paresis

USE OF HEMADSORPTION IN TICAGRELOR-ASSOCIATED BLEEDING

Hana Šimić1*, Filip Šimunić1, Franka Šindler1, Luka Škaro1, Vasilije Stambolija1, 2
1Faculty of Medicine, University of Zagreb, Zagreb, Croatia
2Department of Anesthesiology, Reanimatology, Intensive Care Medicine and Pain Therapy, Neurosurgical Intensive Care Unit, KBC Zagreb, Zagreb, Croatia
***Presenting author**: Hana Šimić, hana.simic123@gmail.com

Introduction
Ticagrelor is a reversible P2Y12 receptor antagonist used in acute coronary syndromes, but as a potent platelet aggregation inhibitor, it poses a significant bleeding risk in cases of overdose or intoxication. Ticagrelor is not dialyzable and platelet transfusions are ineffective due to circulating drug binding to freshly transfused platelets. Hemadsorption using sorbent-based devices such as CytoSorb has demonstrated >99% efficiency in removing ticagrelor from human blood and has been successfully employed. Bentracimab is an antidote that could be used in such cases although the European Medicines Agency has not approved it.

Case report
A 56-year-old male presents to the emergency department due to a headache with photophobia, denying previous head trauma. The patient was disoriented, hypotensive and bradycardic. Past medical history included a myocardial infarction 15 months ago with consequent antiplatelet therapy, including acetylsalicylic acid and ticagrelor. The initial multislice computed tomography (MSCT) scan showed an acute subdural hematoma with signs of raised intracranial pressure, such as the brain midline shift to the right, subfalcine and uncal herniations. During initial examination, the patient was intubated because his condition worsened (Glasgow Coma Scale 5), which indicated a decompressive craniotomy. Postoperatively, neuromonitoring revealed a persistently increased intracranial pressure (ICP) of 36 mmHg. Control MSCT scan indicated a newly formed epidural hematoma, prompting emergent repeat craniotomy and its evacuation. Due to significant blood loss with signs of hemorrhagic shock caused by ticagrelor-induced hemostasis impairment, transfusion of thrombocytes and blood was necessary. Postoperative hemadsorption with CytoSorb was initiated as an alternative drug reversal therapy and demonstrated a favorable outcome. During further hospitalization, the clinical course was characterized by regression of the hematoma, stable ICP and improvement in consciousness. The patient was extubated and showed mild neurological impairment.

Conclusion
Ticagrelor is an effective antiplatelet therapy, but it carries significant risks for hemorrhagic events. Although antidotes are not available yet, administration of CytoSorb hemadsorption can be a life-saving alternative.

Keywords:
Platelet aggregation inhibitor; Antidote; Hemadsorption; Neuromonitoring; Subdural hematoma; Epidural hematoma

MULTIDRUG-RESISTANT POSTOPERATIVE MENINGITIS FOLLOWING AN ELECTRIC SCOOTER ACCIDENT: A CASE REPORT

Lucija Tomić1, Laura Trbušić1, Branimir Gjurašin1,2
1 University of Zagreb, School of medicine, Zagreb, Croatia
2 University Hospital for Infectious Diseases, Zagreb, Croatia
Presenting author: Lucija Tomić, lucija.tomi@gmail.com

Introduction

Cervical trauma may result in nerve root avulsion and disruption of meningeal structures, leading to pseudomeningocele formation and cerebrospinal fluid (CSF) leakage. Trauma is a frequent cause of CSF leakage in adults, associated with a high risk of meningitis, especially when the leakage is prolonged.

Case Report

A 28-year-old man presented to emergency department with acute alcohol intoxication and multiple traumatic injuries from electric scooter accident, including left arm paresis, brachial plexus damage, cervical and thoracic spine fractures. He was treated conservatively and discharged after five days. Two months later, he underwent two surgeries for post-traumatic cervical CSF fistula and left-sided pseudomeningocele. One month later, follow-up MRI demonstrated pseudomeningocele progression and persistent CSF fistula, requiring additional surgery. The day after discharge, he presented to emergency department with headache and left-sided neck swelling due to MRI-confirmed pseudomeningocele progression and persistent fistula. Elevated inflammatory markers in blood and pleocytosis confirmed meningitis, while CSF cultures were sterile. External lumbar drainage was placed and empirical therapy with vancomycin and meropenem initiated, resulting in cytologic CSF normalization. On the 15th day of therapy, pseudomeningocele and fistula were treated with drainage and epidural blood patch. On day 17, clinical worsening and pleocytosis prompted modification of therapy to cefepime and linezolid, which was changed on 24th day to ceftazidime-avibactam and linezolid after isolation of multidrug-resistant Klebsiella pneumoniae OXA-48 in CSF. Colistin was added to therapy on the 35th day due to fever and elevated inflammatory markers. Another pseudomeningocele surgery was performed. From day 53 onward, meningitis was managed with ceftazidime-avibactam and colistin, leading to clinical improvement. He was discharged after 69 days of hospitalization, with left arm paresis and scheduled neurosurgical follow-up.

Conclusion

This case highlights the risk of severe infectious complications following cervical trauma and emphasizes the importance of timely diagnosis, multidisciplinary management, surgical consultation and targeted antimicrobial therapy.

Keywords:
Meningitis; Klebsiella pneumoniae; Cerebrospinal fluid leak; Blood patch, epidural

SEVERE TRAUMATIC BRAIN INJURY WITH MULTIFOCAL INTRACRANIAL HEMORRHAGE AND DECOMPRESSIVE CRANIECTOMY IN A THREE-MONTH-OLD INFANT

Dora Mladenić1, Hrvoje Šimić2, Rajna Međimurec3
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Center Rijeka, Department of Neurosurgery, Rijeka, Croatia
3 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
Presenting author: Dora Mladenić1, doramladenic@gmail.com

Introduction

Severe traumatic brain injury (TBI) in early childhood is a critical condition associated with high mortality due to the vulnerability of the developing brain. Complex skull fractures with subdural (SDH) and subarachnoid hemorrhage (SAH), cerebral contusions, and secondary herniation pose significant risks of prolonged coma, respiratory failure, and neurodevelopmental impairment. Early neuroimaging, intensive care unit management, and timely neurosurgical intervention are essential to optimize outcome.

Case Report

A 3-month-old girl was admitted to the hospital following severe head trauma of unclear mechanism, with clinical suspicion of abusive head trauma. Initial evaluation revealed left parietal soft tissue swelling and hematomas on the forehead, nasal bridge, and under the lower eyelids. A CT scan revealed a multifragmentary fracture of the left parietal-occipital bone with extracranial herniation and soft tissue hematoma, and multiple intracranial hemorrhages (SDH, SAH, focal contusions) with subsequent neurological deterioration. She was intubated and mechanically ventilated.
Initial conservative management included sedation, intracranial pressure monitoring, and seizure prophylaxis. Due to worsening herniation, an emergency left parietooccipital osteoclastic craniectomy with SDH evacuation was performed. Early postoperative imaging confirmed adequate decompression, while subsequent imaging demonstrated stabilization and gradual resolution of intracranial hemorrhages without development of hydrocephalus. Despite the severity of injury, gradual neurological improvement was observed, including return of primitive reflexes, spontaneous movements, and increasing motor activity. After 33 days of intensive care, the infant was extubated. Serial imaging showed resolution of the hematoma and absence of hydrocephalus. At discharge, the infant was hemodynamically stable, with spontaneous eye opening, crying, and partial recovery of age-appropriate motor function. Physical therapy was initiated to support neurodevelopment. Given the unclear mechanism of injury, child protection services were involved early in the hospital course. This case highlights the importance of timely neurosurgical intervention and multidisciplinary care in improving outcomes in severe infant traumatic brain injury

Conclusion

Severe traumatic brain injury in infancy requires rapid diagnosis and urgent multidisciplinary management. Timely decompressive craniectomy and comprehensive intensive care can significantly improve survival and neurological recovery, although careful long-term follow-up remains essential

Keywords:
Brain injury; Decompressive craniectomy; Herniation; Infant;
Subdural hematoma

LONG-TERM SURVIVAL IN HER2-POSITIVE BREAST CANCER WITH BRAIN METASTASES: A MULTIMODAL TREATMENT APPROACH

Ivo Todorić1, Marta Lugarić1, Luka Trkmić1, Sofija Tomas1, Tajana Silovski1,2
1 School of Medicine, University of Zagreb
2 Department of oncology, University hospital Zagreb
Presenting author: Ivo Todorić, ivotodoric@gmail.com

Introduction
Brain metastases from breast cancer occur in 15–30% of patients with metastatic breast cancer (BC), with the highest incidence in human epidermal growth factor receptor-positive (HER2+) and triple-negative subtypes. Treatment depends on the number, size, and location of metastases, as well as tumor subtype and patient performance status. Treatment options include stereotactic radiosurgery (SRS) for patients with limited brain metastases, surgery for accessible and symptomatic lesions and whole-brain radiotherapy (WBRT) for multiple brain metastases, as well as systemic therapy. Prognosis remains poor, with overall survival from 6 to 11 months.

Case Report
Two years after the diagnosis of early HER2+ BC and initial systemic treatment based on chemotherapy, endocrine therapy and anti-HER2 therapy trastuzumab, a 35-year-old patient with history of Leydig cell tumor presents with multiple brain metastases, initially treated with SRS. Although the patient responded well, quite soon she developed metastases in the lungs and liver. At the time of extracranial progression, systemic treatment with pertuzumab, trastuzumab and taxane-based chemotherapy was introduced. Eight months after brain metastases occurrence, a further progression in the central nervous system was revealed. Therefore the patient underwent a right-sided craniotomy and resection. Four months later, due to further progression, another neurosurgery was performed, removing another brain metastasis. The patient continued systemic therapy alongside WBRT. After 15 months, progression of another brain metastasis occurred which was treated by stereotactic body radiation therapy. Due to central progression instead of dual anti-HER2 treatment, systemic treatment with neratinib was commenced. Despite initial good control of disease centrally, progression occurred and patient died 4 years after the occurrence of metastatic BC with brain metastases.

Conclusion
Although brain metastases tend to have a poor prognosis, this case shows an exceptional patient that lived 4 years after the detection of brain metastasis due to teamwork and combination of various therapeutic options.

Keywords:
Breast Carcinoma; Brain Metastases; Oncogene Protein HER-2; Stereotactic Radiosurgery; Disease Progression
Clarify the rationale for each treatment switch (e.g., transition to WBRT and later to neratinib) to better demonstrate decision-making and guideline alignment.

DOMINANT POLYCISTIC KIDNEY DISEASE & SACCULAR INTRACRANIAL ANEURYSMS

Luka Matakovićl *, Hrvoje Šimić2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Center Rijeka, Department of Neurosurgery, Rijeka, Croatia
Presenting author: Luka Matakovićl, matakovic.luka@gmail.com

Introduction
Autosomal dominant polycystic kidney disease (ADPKD) is a hereditary disorder caused by pathogenic mutations in the PKD genes. Besides renal enlargement and cystic destruction, the disease also affects the liver, pancreas, and seminal vesicles. Approximately 25% of affected individuals develop mitral valve prolapse, while up to 15% present with saccular aneurysms of the Willis' circle. Hypertension is a frequent comorbidity which increases the likelihood of aneurysmal rupture, potentially resulting in fatal subarachnoid haemorrhage. This clinical case underscores the importance of early recognition and management of all potential manifestations of ADPKD, particularly those with the most severe neurological consequences, such as saccular intracranial aneurysms.

Case Report
The patient was transported to the emergency department by ambulance as a co-participant in a motor vehicle accident. She was found unconscious and apnoeic. Upon arrival, she underwent emergent orotracheal intubation and was placed on mechanical ventilation. Bronchoscopy was performed due to suspected foreign-body aspiration, while other vital parameters remained normal. She was subsequently referred for whole-body, brain, and cervical spine CT imaging. Neuroimaging revealed extensive subarachnoid haemorrhage involving both cerebral hemispheres, basal cisterns, and Sylvian fissures. CT angiography demonstrated two 6mm saccular aneurysms, one at the M1/M2 junction of the left MCA and the other in the pars communicans of the right ICA. She underwent urgent neurosurgical intervention with successful placement of an external ventricular drain, reducing intracranial pressure. Transfer to KBC Zagreb had been planned, but the patient passed away the following day.
The patient had been diagnosed with ADPKD in 2022. Despite a positive maternal family history, she hadn't undergone earlier genetic testing. Since 2011 she had been diagnosed with chronic headaches and hypertension – nonspecific manifestations of the ADPKD.

Conclusion
In conclusion, early genetic testing is essential for individuals with hereditary risk, enabling timely diagnosis, evaluation of complications, and improved long-term outcomes.

Keywords:
ADPKD, headache, hypertension, saccular aneurysm, SAH

INTRAVENTRICULAR TUMOR OF UNCERTAIN HISTOPATHOLOGY – A CASE REPORT

Lea Radić1, Nikolina Pilj1, Barbara Ravlić1, Dario Mužević2
1 Josip Juraj Strossmayer University of Osijek, Faculty of Medicine, Osijek, Croatia
2 Clinical Hospital Center Osijek, Department of Neurosurgery, Osijek, Croatia
Presenting author: Lea Radić, learadic510@gmail.com

Introduction
Intraventricular tumors are rare lesions, accounting for approximately 1–10% of all intracranial neoplasms. Pilocytic astrocytoma is a World Health Organization grade 1 benign tumor usually cured by surgical resection alone, with excellent prognosis after gross total removal. Ependymoma is a glial tumor originating from ventricular and spinal canal cells. Unlike pilocytic astrocytoma, treatment usually requires adjuvant radiotherapy and strict follow-up, especially after subtotal resection or in higher-grade tumors. Therefore, a precise histopathological diagnosis is necessary. However, neuropathology sometimes faces overlapping tissue features, complicating diagnosis.

Case Report
A 49-year-old female patient presented with headaches and psycho-organic changes. Her past medical history was unremarkable. Magnetic resonance imaging revealed a tumor mass in both lateral ventricles with intraventricular foramen obstruction and chronic hydrocephalus. The patient underwent right frontal craniotomy with planned subtotal resection and placement of temporary external ventricular drainage. Histopathology showed a hypocellular glial tumor of pilocytic appearance with numerous Rosenthal fibers and calcifications. The immunohistochemical profile was glial fibrillary acidic protein+, oligodendrocyte lineage transcription factor+, isocitrate dehydrogenase-, Ki-67 protein 1.5%. Despite thorough histopathological and immunohistochemical analysis, distinction between a WHO grade I pilocytic astrocytoma and a WHO grade I subependymoma with concomitant piloid gliosis could not be established. The patient fully recovered following surgery and did not require permanent ventriculoperitoneal shunt placement. Postoperative MRI confirmed residual tumor in the left lateral ventricle, consistent with the planned subtotal resection. Due to diagnostic uncertainty, management is guided by tumor behavior and clinical course, with close radiological surveillance and adjuvant therapy reserved for progression.

Conclusion
This case illustrates the limitations of histopathology in diagnosing intraventricular tumors and highlights the importance of an integrated approach, combining histology, molecular profiling, and radiology. Indeterminate histology complicates treatment planning, follow-up, and patient counseling, emphasizing the need for individualized management and careful radiological surveillance.

Keywords:
Astrocytoma; Brain Neoplasms; Cerebral Ventricle Neoplasms; Ependymoma; Hydrocephalus

RAPID PROGRESSION OF ACUTE SUBDURAL HEMATOMA: THE IMPORTANCE OF INTENSIVE MONITORING DESPITE STABLE INITIAL PRESENTATION

Matija Peić1, Matias Andjal1, Jakov Kovačević1 Leon Davidović1 Nenad Koruga1,2
1 Faculty of Medicine, Josip Juraj Strossmayer University of Osijek, Croatia
2 Department of Neurosuregry, University Hospital Center Osijek, Croatia
Presenting author: Matija Peić, peicmatija4@gmail.com

Introduction

Rapid progression of acute subdural hematoma in initially neurologically stable patients remains a relatively rare and underestimated complication. This case emphasizes the critical importance of intensive neuroradiological monitoring even in patients with initially stable neurological status (Glasgow Coma Scale, GCS 15), as rapid hemodynamic progression may require urgent neurosurgical intervention.

Case Report

A 51-year-old male with chronic alcoholism was admitted after his first generalized tonic-clonic seizure with a fall and head trauma. Neurological examination revealed GCS 15, the patient was oriented with normal pupillary reactions and no motor deficits. Emergency brain computed tomography (CT) verified acute subdural hematoma in the left frontotemporal-parietal region with minimal midline shift, linear temporooccipital fracture on the right, traumatic SAH, and multiple contusions.

Within 13 hours, sudden deterioration of neurological status occurred. Follow-up CT showed significant hematoma progression with massive edema and midline shift of 22 mm. Emergency decompressive craniectomy with hematoma evacuation was performed. Postoperative course was complicated by mechanical ventilation, bilateral pleuropneumonia, left pneumothorax requiring chest tube insertion, and respiratory insufficiency. Tracheostomy was performed, and microbiological cultures identified vancomycin-resistant *Enterococcus*.

Serial CT findings showed gradual regression of edema and midline shift from 22 mm to 7 mm. The patient was successfully weaned from the ventilator, enteral feeding via percutaneous endoscopic gastrostomy was initiated, and cranioplasty was performed. He was discharged conscious with residual tetraparesis and referred for intensive neurorehabilitation with antiepileptic therapy (Keppra 500 mg twice daily).

Conclusion

In cases of rapid deterioration and development of subdural hematoma in initially neurologically stable patients, close clinical monitoring and repeat neuroimaging are essential, as early recognition of progression and timely surgical intervention have a significant impact on functional outcome.

Keywords: Hematoma; Epilepsy; Craniocerebral Trauma; Decompressive Craniectomy; Brain Edema

TRANSFORAMINAL ENDOSCOPIC DISCECTOMY FOR UP-MIGRATING L5/S1 DISC HERNIATION: A CASE REPORT

***Teodora Pilat1**, Matea Cica1, Vita Pervan1, Karlo Houra2,3
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Aksis hospital, Zagreb, Croatia
3 University North, Varaždin, Croatia
***Presenting author:** Teodora Pilat, pilatteodora1@gmail.com

Introduction

Lumbar disc herniation is a common cause of radicular pain and neurological deficit due to compression of spinal nerve roots. While conventional management has typically involved open approaches, minimally invasive endoscopic techniques have gained increasing acceptance in recent years. These techniques enable targeted decompression of neural structures while minimizing tissue trauma and facilitating faster postoperative recovery.

Case Report

The patient presented with a four-month history of progressive right-sided radicular pain radiating along the L5 and S1 dermatomes, accompanied by intermittent paresthesia in the right leg. On clinical examination at presentation, the Lasegue sign was positive at 50°, and dorsiflexion of the right foot was graded 4/5. Hyperesthesia was noted in the L5 dermatome and, to a lesser extent, in S1. MRI scans of the LS spine showed an upward-migrating right-sided disc extrusion at the L5/S1 level with compression to the dural sack and nerve root. Surgery was performed under local anesthesia via a right-sided transforaminal endoscopic approach to the L5-S1 disc. Endoscopic visualization confirmed extruded disc material migrating cranially and compressing the S1 nerve root. A selective discectomy was performed to remove the extruded fragment, followed by foraminotomy to decompress the L5 nerve root. Following removal of the disc fragment and widening of the foramen, the nerves were adequately decompressed. Postoperatively, the patient reported regression of pain, and the Lasegue test was negative. The patient was discharged the same day from the hospital with detailed instructions for postoperative care.

Conclusion

This case supports the transforaminal endoscopic approach under local anesthesia as a minimally invasive strategy for the treatment of superiorly migrated L5-S1 disc herniation with combined L5 and S1 nerve root involvement. This approach allows direct decompression of affected nerve roots while minimizing surgical trauma and excellent rapid recovery.

Keywords:
anesthesia, local; decompression, surgical; endoscopy; foraminotomy; intervertebral disc displacement

PSYCHIATRY

24th APRIL - 26th APRIL 2026

FRAGMENTATION OF THE SELF OR ADAPTIVE COMPROMISE? A DYNAMIC ANALYSIS OF DISSOCIATIVE EXPERIENCES

Leonarda Novoselec1,3, Petra Jurković1,3, Ana Šestanj1, Andrijana Šantić1,2
1 Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
2 Psychiatric Clinic Osijek, Clinical Hospital Centre in Osijek, Osijek, Croatia
3 Section of Neuroscience Osijek (SenzOs), Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek Croatia
Presenting author: Leonarda Novoselec, leonarda.novoselec@gmail.com

Introduction
Dissociation is a complex psychological phenomenon characterized by discontinuity and disruption in person's sense of identity, memory and perception of self. Those experiences are often explained as defense mechanisms due to repeated trauma. As this phenomenon can appear as a symptom of multiple mental health conditions, the need for personalized approach to an individual patient is pronounced.

Case Report
We present the clinical case of a 20-year-old male student admitted to a psychiatric clinic due to a deterioration in mental health characterized by marked anxiety, affective liability, impulsivity, obsessive-compulsive symptoms and fragmentation of self through internal female-identifying identities. The patient comes from a conservative family with a pronounced symbiotic attachment to his mother and non-acceptance of his homosexual tendencies, which, from a dynamic perspective, is associated with feminized internal identities functioning as adaptive defense mechanisms. Psychiatric and psychological evaluation indicated an immature personality structure with borderline traits and transient dissociative phenomena that emerge under stress, without psychotic decompensation. Pharmacotherapy was initiated with fluvoxamine to control obsessive-compulsive symptoms and anxiety, along with alprazolam as needed. Following assessment of daily mood fluctuations and affective liability, lamotrigine was added as a mood stabilizer. Psychotherapeutic interventions combined supportive and dynamic approaches, emphasizing emotional regulation, integration of internal identities, development of autonomy, and reflection on defense mechanisms. During hospitalization, the patient participated in group and individual therapy sessions, achieved impulse and anxiety control, and enhanced social interactions.

Conclusion
This case illustrates the importance of individualized pharmacotherapy and a supportive-dynamic psychotherapeutic approach in patients with dissociative experiences within a borderline personality structure. The question of "fragmentation of self or adaptive compromise" highlights the dynamic dilemma and underscores the necessity of understanding dissociative phenomena not only as symptomatic manifestations but also as psychologically adaptive strategies for affect regulation and coping with stress.

Keywords:
Dissociation; Defense Mechanisms; Lamotrigine; Fluvoxamine; Borderline Personality Disorder

DIFFICULTIES OF PARKINSON'S TREATMENT IN PATIENTS WITH SCHIZOPHRENIA: A CASE REPORT

Petra Sučić1, Ema Šepl1, Lira Strenja1, Branka Aukst Margetić1,2(mentor)
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Clinical hospital Dubrava, Department of Psychiatry, Zagreb, Croatia
Presenting author: Petra Sučić, petra.sucic3@gmail.com

Introduction
Parkinson's disease (PD) is a progressive neurodegenerative disorder caused by dopaminergic neuron degeneration in the substantia nigra. It is usually well managed with dopamine agonists. However, when paired with a psychotic disorder such as Schizophrenia, which is characterized by hyperdopaminergia in the associative striatum, and whose treatment can worsen extrapyramidal symptoms, the problem becomes more complex.

Case Report
A seventy-two-year-old female patient treated for Schizophrenia for over twenty years, on stable therapy of Clozapine (50mg /day) and Risperidone (1 mg/day), gradually developed resting tremor. Clonazepam was introduced to suppress tremors; however, symptoms progressed with unilateral extremity rigidity, hypomimia, and bradykinesia. Family history was significant for Parkinson's disease. The patient was advised to be examined by a neurologist. Given that the extrapyramidal symptoms were asymmetric and occurred on low doses of antipsychotics, the neurologist introduced Levodopa (125 mg) under suspicion of PD. At the follow-up visit a month later, the extrapyramidal symptoms had regressed; however, the patient developed psychotic decompensation, and Levodopa was discontinued. The patient was hospitalized in the psychiatric department, and the dosage of Clozapine was increased to 125mg/day. Because the patient decompensated on a low dose of Levodopa, its reinitiation was not recommended. As other antiparkinsonian drugs may cause more severe psychiatric side effects, the patient was left on revised antipsychotic therapy and Clonazepam.

Conclusion
PD and Schizophrenia have opposing pathophysiology, which makes it seem as if the two disorders cannot coexist. However, they do occur in comorbidity and pose a serious problem when it comes to treatment. Antiparkinsonian drugs may exacerbate psychiatric symptoms, and antipsychotics can aggravate extrapyramidal symptoms. Benzodiazepines (Clonazepam) may relieve tremors, but due to their sedative effects, they may not be suitable for geriatric patients. There is no singular approach to such patients, and treatment must be titrated individually.

Keywords:
Antiparkinson Agents; Antipsychotic Agents; Parkinson Disease; Schizophrenia

HIKIKMORI: ADOLESCENCE WITHOUT AN END

Ena Ivanković1, Dora Gašparović1, Antonia Mišić1, Tamara Stanojlović1, Vlatka Kovač1,2
1 Faculty of Medicine, J.J. Strossmayer University of Osijek, Osijek, Croatia
2 Clinical Hospital Centre Osijek , Department of Child and Adolescent Psychiatry, Osijek, Croatia
Presenting author: Ena Ivanković, enaena89@gmail.com

Introduction
Originating in Japan in the late 20th century, hikikomori is a social phenomenon marked by prolonged social isolation, disrupted communication, and avoidance of work or school for at least six months. Hikikomori, considered a syndrome rather than a diagnosis, involves persistent social withdrawal, unlike fear-based Social Anxiety Disorder or Major Depressive Disorder, and is increasingly reported outside Japan.

Case Report
We present a case of a 19-year-old girl who has been receiving outpatient treatment since 2019 due to sudden mood changes and anxiety disturbances. At the beginning of high school, she developed a school phobia and noticed rapid heartbeat, palmar sweating, appetite and sleep disorders, and intense fear of human contact. She spent the third grade isolated in her room playing computer games. Sertraline (25 mg) therapy was initiated, which she received between 2020 and 2022, however, at that time, she showed no interest in psychotherapeutic treatment. After a more stable period, clinical deterioration occurred in 2023, and fluvoxamine (50 mg) was introduced, later replaced by fluoxetine (20 mg) in November. In cooperation with the school's professional support service, she took qualifying exams. Since January 2024 until today, she's been involved in multiple cycles of the day hospital therapeutic program. She participated in individual and group psychotherapy, biofeedback treatment, and exposure therapy. Following the applied interventions, improvements were observed in psychological functioning, emotional regulation, and self-concept. She learned to apply breathing, relaxation, and fear-coping strategies. Over time, she started leaving the house and returned to school. Considering the achieved improvement, further outpatient treatment is recommended.

Conclusion
With appropriate interventions, most patients can gradually reintegrate into society and regain functional social skills. Prolonged isolation, however, may result in significant deficits in social functioning and increase vulnerability to mental health disorders, highlighting the importance of early detection, continuous support, and strategies to preserve psychological well-being**.**

Keywords:
Anxiety Disorders; Psychotherapy, Group; Psychological Well-Being; Sertraline; Social Isolation

A LONG ROAD TO DIAGNOSIS: DIAGNOSTIC CHALLENGES IN A CASE OF AUTISM SPECTRUM DISORDER AND ADHD

Vinka Cvetković1*, Natalija Grdanjski[1], Veronika Hanić[1], Lucija Mitrović[1], Ana Kaštelan (mentor)1,2
1 Faculty of Medicine, University of Rijeka, Croatia
2 Department of Child and Adolescent Psychiatry, Psychiatry Clinic, CHC Rijeka, Croatia
Presenting author: Vinka Cvetković, vinka.cvetkovic02@gmail.com

Introduction

Neurodevelopmental disorders such as Attention Deficit Disorder with Hyperactivity (ADHD) and Autism Spectrum Disorder (ASD) may remain unrecognized until late adolescence or adulthood, particularly when symptoms are partially masked by anxiety or depressive features and cognitive functioning is preserved. This case report illustrates diagnostic challenges in a young adult whose clinical picture evolved over time and whose care was provided by multiple clinicians.

Case Report

A male in his mid-twenties had been involved in discontinuous psychiatric care since high school. Over a six-year period he was evaluated by six different psychiatrists and received several diagnoses, including emotional disorders of childhood, social anxiety disorder, depressive disorder, impulse control disorder, behavioral addiction, and acute psychotic disorder. Pharmacological treatments varied and adherence was inconsistent.
Repeated psychological assessments demonstrated above-average intellectual functioning with a markedly uneven cognitive profile, including impaired executive functions and persistent socio-communicative difficulties. Developmental history revealed early hyperactivity, long-standing attentional deficits since primary school and difficulties in peer relationships. Based on the integration of developmental history, psychological testing and clinical evaluation, the final diagnoses of ADHD and Autism Spectrum Disorder were established.
At the most recent follow-up the patient reported subjective improvement in functioning and continued working in a low-stress environment. Mental status examination showed a calmer presentation and improved motivation. Treatment included methylphenidate, propranolol, and intermittent anxiolytics.
Further follow-up was planned, although regular attendance remained inconsistent.

Conclusion

This case illustrates how changing clinicians, multiple provisional diagnoses and discontinuous follow-up may delay recognition of underlying neurodevelopmental disorders.
Careful developmental history-taking and comprehensive diagnostic assessment are essential for timely diagnosis.
Early recognition allows more appropriate treatment planning and may improve functional adjustment and quality of life in young adults.

Keywords:
Anxiety Disorders; Attention Deficit Disorder with Hyperactivity; Autism Spectrum Disorder; Diagnostic Errors; Neurodevelopmental Disorders

THE STRUCTURE AND PERSISTENCE OF DELUSIONAL BELIEFS IN CHRONIC SCHIZOPHRENIA: A CASE REPORT

Matias Andjal1, Leon Davidović1, Filip Ovničeić1, Matija Peić1 , Dunja Degmečić1,2
1 Faculty of Medicine, Josip Juraj Strossmayer University of Osijek, Croatia
2 Department of Psychiatry, University Hospital Center Osijek, Croatia
Presenting author: Matias Andjal, andjalmatias@gmail.com

Introduction
Schizophrenia is a chronic mental disorder characterized by disturbances in perception, thought processes, and reality testing, including hallucinations and delusions. Persistent delusions strongly affect insight, functioning, and treatment adherence, often leading to nonadherence and relapses. This case highlights the clinical relevance of the relationship between delusions and medication adherence in long-term management.

Case Report
We report a 48-year-old male with a long-standing diagnosis of schizophrenia, with symptom onset in early childhood and multiple hospitalizations due to recurrent psychotic exacerbations. Relapses were consistently associated with antipsychotic discontinuation. Delusions were the dominant psychopathological feature, primarily religious content. The patient described divine signs, including visual and tactile experiences interpreted as communication from God, and intrusive self-harm thoughts perceived as spiritual trials. Although suicidal ideation was intermittently present, it was inhibited by fear of divine punishment. He also reported “spiritual battles” and occasional beliefs of being specially chosen, contributing to poor insight, repeated non-adherence, retreat into a fantasy world. The patient was treated with antipsychotics, mood stabilizers, and anxiolytic/sedative medications. Adherence was inconsistent and strongly influenced by his delusional belief system, leading to repeated discontinuation of therapy during religious ideation, followed by clinical deterioration and rehospitalization. Over time, a cognitive shift occurred within the same religious framework: the patient began to interpret treatment as compatible with his faith and sanctioned by God. This reinterpretation was associated with improved adherence, fewer relapses, and greater clinical stability. He remains adherent to antipsychotic treatment with sustained stabilization.

Conclusion
This case highlights the role of the patient’s delusional belief system in shaping treatment adherence in chronic schizophrenia. Religious delusions contributed to poor insight and repeated medication discontinuation, affecting relapse risk. A shift within the same framework—reframing treatment as divinely sanctioned—led to improved adherence and clinical stability, emphasizing the importance of working within the patient’s belief system to enhance outcomes.

Keywords:
Patient Compliance; Delusions; Hallucinations; Religion and Psychology; Schizophrenia

WHEN ANTIDEPRESSANTS INTERACT WITH ONCOLOGICAL THERAPY: A CASE REPORT

Lara Žubrinić1, Natalija Volgemut1, Ivana Ljubičić Bistrović,2,3
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinic for Psychiatry, Clinical Hospital Centre Rijeka, Rijeka, Croatia
3 Department of Psychiatry and Psychological Medicine, Faculty of Medicine, University of Rijeka
Presenting author: Lara Žubrinić, zubrinic.lara@gmail.com

Introduction
Tamoxifen is a cornerstone therapy in hormone-receptor–positive breast cancer. However, its clinical efficacy depends on metabolic activation via the cytochrome P450 2D6 (CYP2D6) enzyme system. Several commonly prescribed antidepressants inhibit CYP2D6 and may reduce the conversion of tamoxifen into its active metabolite, potentially diminishing therapeutic benefit. Consequently, antidepressant selection represents an important consideration in psycho-oncological practice, where effective treatment of depressive and anxiety symptoms must be balanced with oncological safety.

Case Report
G.Š., a 51-year-old female, diagnosed with breast cancer two years prior. She was treated with combined surgical and oncological therapy, and is currently maintained on adjuvant tamoxifen. Over the same period, she experienced multiple psychosocial stressors, including her daughter's diagnosis of diabetes, her sister's malignancy and significant marital difficulties. Gradually, she developed persistent anxiety, frequent crying episodes, excessive fear that negative events would occur and pronounced health-related worries affecting both herself and her family. She also reported loss of motivation as well as reduced interest in previously enjoyable activities and social withdrawal. Considering the need to avoid significant CYP2D6 inhibition during tamoxifen therapy, and in accordance with current evidence indicating that venlafaxine exerts minimal inhibitory effect on this enzyme compared to several first-line antidepressants, psychopharmacological treatment was initiated with venlafaxine 37.5 mg daily and titrated to 75 mg daily. The medication was well tolerated and on follow-up evaluation, after three months, she demonstrated significant improvement in depressive symptoms, decreased anxiety intensity and improved daily functioning without adverse effects or interference with oncological management.

Conclusion
This case highlights the clinical importance of careful antidepressant selection in patients receiving tamoxifen therapy. Awareness of CYP2D6-mediated drug interactions is essential in psycho-oncology, as inappropriate pharmacotherapy may compromise oncological efficacy. Venlafaxine represents a suitable and effective option for treating depressive and anxiety disorders in breast cancer patients on tamoxifen therapy.

Keywords:
Antidepressive Agents; Anxiety; Breast Neoplasm; Depression; Tamoxifen

CONFABULATIONS IN DELIRIUM OR/ AND DENIAL FOLLOWING POLYTRAUMA IN A TRAFFIC ACCIDENT

Adriana Klobučarić1, Luka Kaštelan1, Ivan Knežević1, Jelena Rebić, MD, PhD 2, 3 (mentor)
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Clinical Hospital Center of Rijeka, Clinic of Psychiatry, Rijeka, Croatia
3 Faculty of Medicine, Department of Psychiatry and Psychological Medicine, University of Rijeka, Rijeka, Croatia
Presenting author: Adriana Klobučarić, adriana.klobucaric02@gmail.com

Introduction

Denial is a powerful, immature, psychotic defense mechanism used to reject awareness of painful reality and to avoid distressing emotions, allowing time for adaptation. Delirium is an acute, usually reversible organic mental disorder; among alterations of many mental functions, individuals in delirium often display confabulations – unconscious filling in of memory gaps with fabricated memories.

Case Report

A 48-year-old man was brought to the emergency hospital department with cardiopulmonary resuscitation (CPR) in progress (total duration 8 minutes) after a traffic accident resulting in bilateral traumatic lower-leg amputations. CT scan showed no brain traumatic lesions. In the ICU he was treated for hemorrhagic shock, acute renal and hepatic failure, with several surgical interventions. During hospitalization he was repeatedly examined by a psychiatrist. On the 22nd hospitalization day, he manifested psychomotor agitation, confusion, disorientation, visual hallucinations and lack of insight - he asked to borrow a car, stated that he just returned from a walk, that he needs to go back to work (car mechanic) - in reality, that was the first day he was seated with assistance of the staff. A diagnosis of delirium was assigned and psychotropic medication introduced. 2 days after, clinical presentation remained similar - amnestic for trauma, displayed confabulations, denied any injuries despite seeing his amputated limbs (uncovered). Psychopharmacotherapy was revised, diagnosis of acute reaction to stress added. 17 days after, follow-up noted resolution of previous psychopathology.

Conclusion

This case demonstrates delirium following polytrauma, prolonged CPR, multiple physical disorders and operations. The gradual resolution of delirium with stabilization of physical condition and psychiatric care highlights the importance of timely delirium recognition and management. Here, the content described as confabulation could also be interpreted as psychological denial of an extreme traumatic experience.

Keywords:
Amputation; Cardiopulmonary resuscitation; Delirium; Denial; Multiple trauma

The Importance of Therapeutic Communities in the Treatment of Polysubstance Addiction

Natalija Grdanjski1, Lucija Mitrović1, Vinka Cvetković1, Loreta Horvatek1, Tanja Grahovac Juretić1,2
1 Faculty of Medicine, University of Rijeka, Republic of Croatia
2 Department of Psychiatry and Psychological Medicine, Faculty of Medicine, University of Rijeka, Clinic of Psychiatry, Clinical Hospital Center Rijeka, Republic of Croatia
Presenting author: Natalija Grdanjski, natalija.grdanjski@gmail.com

Introduction
Polysubstance use disorder (politoxication) is a psychiatric condition that significantly impairs an individual's mental health, behavior, and social functioning. Its treatment is complex and demanding, involving not only pharmacotherapy but also sociotherapeutic and psychotherapeutic approaches. Therapeutic communities play an important role in the psychiatric management of addiction, providing a structured and controlled environment suitable for long-term rehabilitation and patient stabilization.

Case Report
The patient is a 30-year-old physically healthy male with a positive family history of substance use disorder; his father was an opioid addict who died from an overdose. He had a long history of polysubstance abuse, including amphetamines, ecstasy, lysergic acid diethylamide, methylenedioxymethamphetamine, cocaine, ketamine, and tetrahydrocannabinol. Due to this, he spent one and a half years in a therapeutic community, where he participated in occupational therapy, self-help groups, and psychosocial support programs. He was highly motivated for abstinence and did not receive psychopharmacological treatment during this period. He does not consume alcohol but smokes up to two packs of cigarettes daily. After discharge, psychiatric evaluation confirmed good functioning and continued abstinence. He was advised to attend weekly sociotherapy, which he accepted. Due to mild mood fluctuations, treatment with bupropion (150 mg) and diazepam (5 mg) was initiated. Later, carbamazepine (up to 400 mg) and quetiapine (200 mg) were added, resulting in improved mood and reduced tension. During long-term follow-up, with regular psychiatric care, medication adherence, and continued group therapy, the patient maintained stable mental health, reported no cravings, and remained future-oriented.

Conclusion
Polysubstance use disorder is more complex to treat than single-substance addiction due to overlapping drug effects. Treatment often requires combined pharmacotherapy and psychosocial interventions. Therapeutic communities represent an essential component of long-term rehabilitation and stabilization.

Keywords:
Community Psychiatry; Psychoactive Drugs; Psychotherapy; Therapeutic Community

CLOSE TO DEATH, AWAY FROM PEACE: PTSD AFTER LIFE-THREATENING MEDICAL EMERGENCIES

Izak Feher1, Marina Letica Crepulja, MD1.2
1 Faculty of Medicine, University of Rijeka, Rijeka, Croatia
2 Department of Psychiatry, Clinical Hospital Centre of Rijeka, Rijeka, Croatia
Presenting author: Izak Feher, ifeher@uniri.hr

Introduction

Life-saving medical care can itself become a traumatic exposure. While PTSD is classically associated with violence or accidents, repeated near-death medical emergencies and invasive procedures may generate persistent fear conditioning, bodily hypervigilance, and avoidance-yet this pathway remains under-recognized in routine specialist follow-up.

Case Report

A middle-aged man underwent craniotomy and clipping for an unruptured anterior communicating artery aneurysm in 2018. In the months after surgery, he developed recurrent panic attacks characterized by deathly fear, triggered by uncomfortable head sensations, anxiety, insomnia, and progressive avoidance, prompting psychiatric consultation. He was diagnosed with adjustment and panic disorders and prescribed an SSRI and PRN anxiolytic, but discontinued follow-up after two visits. In 2021 he suffered an acute ST-elevation myocardial infarction requiring urgent coronary angiography and stenting of the occluded right coronary artery. Following the incident, symptoms intensified and became consistent with DSM-5 PTSD criteria: persistent re-experiencing (Criterion B), avoidance (Criterion C), negative alterations in cognition and mood (Criterion D) with depression and social withdrawal and pronounced hyperarousal (Criterion E). PCL-5 was 58/80, well above the cutoff, indicating high severity. Pharmacotherapy with an SSRI, gabapentinoid anxiolytic, and PRN anxiolytic produced partial relief; he attended psychiatric controls every 2–3 months but initially declined psychotherapy. After further worsening, he began weekly psychotherapy in 2024, gradually improving emotional regulation, social engagement, and coping. Therapy incorporated ESTAIR, a phase-based program teaching affective and interpersonal regulation skills, with trauma-focused work in the final phase. By late 2025, functional improvement was noted, though residual PTSD symptoms persisted.

Conclusion

Life-threatening medical emergencies may meet DSM-5 Criterion A and, in some cases, lead to clinically significant PTSD symptoms. These may be reinforced by ongoing somatic sensations that act as trauma reminders and contribute to avoidance of care. Given associations with poorer somatic outcomes, early recognition is important. Routine PTSD screening after such events may be beneficial, with referral for specialist assessment and trauma-focused care when indicated, potentially reducing chronicity and improving outcomes.

Keywords:
Mental Health; Panic Disorder; Post-Traumatic; Psychiatric Somatic Therapies; Stress Disorders

POLYSUBSTANCE DEPENDENCE IN A FORMER INMATE UNDER COMPLEX PHARMACOTHERAPY: A CASE REPORT

Stjepan Škudar1, Miroslav Hanževački1,2
1Zagreb West Health Center, Croatia
2Department of Family Medicine, School of Medicine, University of Zagreb, Croatia
Presenting author: Stjepan Škudar, stjepan.skudar@gmail.com

Introduction

Recently released inmates with polysubstance use disorder often present with persistent craving, withdrawal-related symptoms, and personality pathology. Treatment is further complicated by high-risk behaviors and misuse of prescribed medications. This case demonstrates successful outpatient stabilization in a complex clinical profile and underscores the clinical relevance of coordinated pharmacological and psychosocial interventions during the vulnerable post-release period, when relapse risk and functional impairment are particularly pronounced.

Case Report

A 34-year-old male with a history of polysubstance use disorder involving heroin and cocaine and comorbid personality disorder presented voluntarily for psychiatric evaluation one year after release from a correctional facility. He was receiving opioid substitution therapy with buprenorphine due to prior heroin use. Although he denied recent illicit substance use, he reported persistent abstinence-related symptoms, including craving, anxiety, sleep disturbances, and emotional dysregulation. He also reported heavy tobacco use, approximately 40 cigarettes daily. A history of supratherapeutic intake of pregabalin was noted, necessitating close monitoring and clearly defined treatment boundaries. Mental status examination revealed full orientation, mildly depressed mood, reduced psychomotor activity, preserved cognitive functions, and partial insight into addictive behavior. No psychotic symptoms, suicidal ideation, or heteroaggressive tendencies were observed. Pharmacological treatment included buprenorphine and adjunctive therapy with pregabalin, diazepam, and quetiapine, prescribed under strict supervision due to prior non-medical use of medications. Despite multiple risk factors, including recent incarceration, persistent craving, and personality pathology, the patient demonstrated good treatment adherence, motivation for recovery, and engagement in outpatient psychosocial interventions, while living in stable housing with family support. During a six-month follow-up period, withdrawal symptoms and craving decreased without evidence of relapse or intoxication.

Conclusion

This case highlights that individualized, closely monitored polypharmacotherapy combined with psychosocial support can achieve stabilization even in high-risk former inmates with polysubstance use disorder and personality disorder. Therapeutic alliance and continuous risk assessment remain essential for successful outpatient management.

Keywords:
Buprenorphine; Polypharmacy; Substance-Related Disorders; Psychosocial Support Systems; Nicotine

THE METABOLIC MIMIC: HYPONATREMIA PRESENTING AS PSYCHIATRIC DISTURBANCE

Petra Jurković1,3, Leonarda Novoselec1,3, Valentin Kordić2
1 Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
2 Psychiatric Clinic Osijek, Clinical Hospital Centre in Osijek, Osijek, Croatia
3 Section of Neuroscience Osijek (SenzOs), Faculty of Medicine Osijek, Josip Juraj Strossmayer University of Osijek, Osijek Croatia
Presenting author: Petra Jurković, petrajura28@gmail.com

Introduction
Acute disturbance of consciousness may closely mimic primary psychiatric disorders, posing a major diagnostic challenge in emergency neurology. Hyponatremia is a common yet potentially life-threatening metabolic condition that can present with behavioral changes, confusion, and impaired awareness, thereby mimicking psychiatric disorders. Chronic alcohol abuse further increases susceptibility through malnutrition, liver dysfunction, endocrine disturbances, and low-solute intake states such as beer potomania. This case highlights the importance of recognizing reversible metabolic and endocrine causes in presentations mimicking psychiatric disorders.

Case Report
A middle-aged woman was admitted immobile, non-communicative, and negativistic, in a moderately impaired general condition. Acute cerebrovascular events, central nervous system (CNS) infection, and structural neurological pathology were excluded. Initial laboratory analyses revealed hypoosmolality with significant hyponatremia and hypokalemia (K 3.3 mmol/L; Na 112 mmol/L). After partial correction, sodium increased to 135 mmol/L. Her history included structural epilepsy, alcohol dependence, alcoholic liver disease, and a positive psychiatric family history. Initial diagnoses included stupor and mental disorder due to brain dysfunction. Differential diagnoses included non-convulsive status epilepticus, toxic–metabolic encephalopathy, and primary psychiatric catatonia. Despite intravenous fluids, diazepam, and lamotrigine, consciousness deteriorated, prompting multidisciplinary evaluation. A key diagnostic turning point was the identification of adrenal insufficiency and a pituitary microadenoma contributing to persistent electrolyte imbalance. Additional findings included toxic sensory polyneuropathy and sideropenic anemia. The disturbance of consciousness was ultimately attributed to metabolic encephalopathy secondary to hyponatremia in the context of chronic alcohol abuse. Targeted treatment with hydrocortisone and electrolyte correction led to gradual clinical improvement.

Conclusion
This case emphasizes the importance of considering endocrine causes of hyponatremia in patients with chronic alcohol abuse and altered mental status. Early recognition of adrenal insufficiency is critical, as prompt corticosteroid replacement may be lifesaving. Integrated addiction treatment, psychiatric support, and preventive strategies are essential to mitigate the severe neurological and systemic consequences of chronic alcohol use.

Keywords:
Alcoholism; Adrenal Insufficiency; Diazepam; Hypokalemia; Hyponatremia; Mental Disorders

ANTIDEPRESSIVE EFFECTS OF EXERGAMING IN OLDER ADULTS – A CASE REPORT

Marjeta Linarić Lipnjak1, Iskra Šimpraga2, Franka Kovačić3, Dubravka Kalinić4
1 Primary Health Care Center Zagreb-East, Zagreb, Croatia
2 Specialist Family Medicine Practice, Vlatka Topolovec Nižetić, MD, Zagreb, Croatia
3 Healthcare institution "Srna", Zagreb, Croatia
4 University Psychiatric Hospital Vrapče, Department for Biological Psychiatry and Psychogeriatrics, Zagreb, Croatia
Presenting author: Marjeta Linarić Lipnjak, marjeta.linaric@gmail.com

Introduction
Older adults show an increased prevalence of pharmacoresistance and polypharmacy, often accompanied by heightened susceptibility to adverse drug reactions, which limits the effectiveness of antidepressant pharmacotherapy and highlights the need for non-pharmacological interventions. The Dividat Senso system represents a novel, technology-driven exergaming platform, designed to deliver cognitive-motor training (CMT). CMT may promote neuroplasticity by simultaneously engaging cognitive and motor processes.

Case Report
A 70-year-old woman with secondary education and a diagnosis of recurrent depressive disorder, currently in a moderate episode, had been undergoing psychiatric treatment for three years. Despite stable antidepressant dosing during the previous six months, pronounced sensitivity to medication side effects prompted the use of CMT as an adjunctive intervention. The patient also demonstrated reduced engagement in conventional socio-occupational therapies due to their repetitive nature. Baseline depressive symptom severity, assessed using the Montgomery–Åsberg Depression Rating Scale (MADRS), was 33 points. CMT was delivered by an exergaming-based dual-task protocol combining goal-directed motor activity with concurrent cognitive load. Sessions were conducted four times a week over a two-month period. Post-intervention assessment revealed a reduction in MADRS score to 26 points, indicating a rapid and clinically meaningful improvement.

Conclusion
This case report supports the feasibility, tolerability and potential antidepressant efficacy of an innovative exergaming-based CMT as an adjunctive intervention in late-life depression. The observed rapid symptom reduction suggests modulation of neurobiological mechanisms relevant to affective regulation, particularly in patients with limited pharmacological treatment options. Further research is needed to confirm these effects in larger samples and to explore the long-term benefits of CMT in older adults with depressive disorders.

Keywords:
Depression; Depressive Disorder; Exergaming; Cognitive Training; Neuronal Plasticity

GENDER DYSPHORIA – THERAPEUTIC TENDENCIES IN HELP-SEEKING ADOLESCENT POPULATION IN CHILDREN'S CLINICAL UNIVERSITY HOSPITAL, RIGA, LATVIA

Sofija Liskova1, Ņikita Bezborodovs1,2
1 Rīga Stradiņš University, Riga, Latvia
2 Children's Clinical University Hospital, Child psychiatry department, Riga, Latvia
Presenting author: Sofija Liskova, sofijaliskova@gmail.com

Introduction

As the gender identity–related concerns are becoming more recognizable in the last decades, and the need for treatment continues to rise, it is crucial to explore gender-affirming care options in Latvia. The aim of this study was to investigate treatment options provided of gender identity disorders in adolescents seeking help at the Children's Clinical University Hospital.

Materials and Methods

Retrospective medical documentation study - outpatient and inpatient episodes of patients diagnosed with gender identity disorder (ICD-10, F64) or other sexual disorders (ICD-10, F66) from 2021 to 2025 were investigated, data collection and selection, and analysis were performed using Microsoft 365 Excel and IBM SPSS Statistics Data editor. All the data was coded before the analysis. Inclusion criteria include: diagnosis F64 and/or F66 mentioned in the medical documentation; at least one of the following symptoms that persists over time (recorded in the medical documentation): persistent distress related to biological sex; desire/belief that one is a member of the opposite sex; persistent rejection of one's body/social norms of gender; discomfort affects functioning. Exclusion criteria: diagnosis code F64 and/or F66 mentioned <2 times; presence of the diagnosis code, but at least one of the aforementioned symptoms is not mentioned in the medical documentation; subcodes of the diagnosis code F66 that are not related to gender identity disorders, and at least one of the aforementioned symptoms is not mentioned in the medical documentation. The study realization was approved by Riga Stradiņš University Scientific Ethics Committee.

Results

During the period from 2021 to 2025 included, 51 patients have been newly diagnosed with gender identity or other sexual disorders, from whom 41% (N=21) received gender-affirming therapy, meaning psychoeducation and social transition averagely at 15 years of age (SD=1,18); 28,6% of those patients (N=6) followed with voice therapy, 15% of FAAB (female assigned at birth) used COCP (combined oral contraceptive pill) to stop/control their menstruation. From known information, 14,3% (N=3) started cross-hormonal treatment after the age of 18, receiving with the diagnosis or transsexualism (F64.0) provided by a multidisciplionary consilium and under supervision of the endocrinologist.

Conclusion

According to existing guidelines1,2, treatment of gender dysphoria should include an individualized approach with different options provided by a multidisciplinary team. All of the patients going through gender-affirming care in Children's Clinical University Hospital during 2021-2025 period received psychological support and started social transition under the care of specialists, a little more than a quarter of patients followed with voice therapy. Other chosen treatment options were COCP for menstruation cycle control, and cross-hormonal therapy.

Keywords:
Adolescent psychiatry; Child psychiatry; Gender dysphoria; Gender-affirming care; Sexual and gender disorders

BALANCING LEVODOPA AND ANTIPSYCHOTIC THERAPY IN PARKINSON DISEASE WITH PARANOID PSYCHOSIS: A CASE REPORT

Ivan Udiljak1, Ivana Vranjković1, Ana Vlatković1, Editha Vučić2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Neuropsychiatric Hospital Dr. Ivan Barbot, Popovača, Croatia
Presenting author: Ivan Udiljak, ivanudiljakk@gmail.com

Introduction

The coexistence of Parkinson's disease (PD) and chronic paranoid psychosis poses a significant therapeutic challenge, as management requires simultaneous control of motor symptoms and prevention of psychiatric relapse. Medication changes may easily destabilize this balance because of the complex interaction between dopaminergic and antipsychotic treatment.

Case Report

A 57-year-old man with an established history of paranoid psychosis was admitted for urgent psychiatric treatment several weeks after clozapine and valproate had been discontinued. After war, he had developed auditory hallucinations, persecutory delusions, and heteroaggressive behaviour. Although initially considered within the spectrum of post-traumatic symptomatology, the clinical picture was later recognized as paranoid psychosis, and antipsychotic and mood-stabilizing treatment was introduced. Later, he developed progressive motor symptoms, including resting tremor, bradykinesia, and reduced muscle strength. Further neurological assessment confirmed Parkinson's disease, and he was started on antiparkinsonian medication.

Following the withdrawal of clozapine and valproate, the patient experienced psychiatric deterioration manifested by paranoid delusions, apathy, anxiety, restless legs, and muscle spasms. The combination of antipsychotic withdrawal and ongoing levodopa treatment likely contributed to dopaminergic imbalance and clinical decompensation. During the acute phase, priority was given to psychiatric stabilization through careful reintroduction and adjustment of antipsychotic therapy, with close monitoring of adverse effects, particularly hypersalivation. After improvement of acute psychiatric symptoms, antiparkinsonian treatment was gradually reintroduced and titrated according to neurological status. The final therapeutic strategy consisted of stepwise optimization of both antipsychotic and antiparkinsonian therapy to maintain psychiatric stability without significant worsening of motor symptoms. The patient was discharged in a clinically stable condition with planned outpatient psychiatric and neurological follow-up.

Conclusion

This case highlights the importance of a staged and multidisciplinary treatment approach in patients with comorbid paranoid psychosis and Parkinson's disease. Careful sequencing and titration of therapy are essential to restore clinical stability and achieve a sustainable balance between psychiatric and motor outcomes.

Keywords:
Antipsychotic Agents; Drug Interactions; Levodopa; Parkinson Disease; Schizophrenia, Paranoid

SERONEGATIVE AUTOIMMUNE LIMBIC ENCEPHALITIS WITH RESIDUAL COGNITIVE DEFICITS: REPORT OF A UNIQUE CASE

Laura Čajo1, Ana Šmit2 , Suzan Kudlek-Mikulić(mentor) 3
1 – Faculty of Medicine, University of J.J. Strossmayer, Osijek, Croatia
2 – Faculty of Medicine, University of Rijeka, Rijeka, Croatia
3– Department of Psychiatry, University Hospital Center Zagreb, Zagreb, Croatia
Presenting author: Laura Čajo1, cajolaura@gmail.com

INTRODUCTION: Autoimmune limbic encephalitis (ALE) is an inflammatory disease of the central nervous system that affects the structures of the limbic system and is clinically manifested by the subacute development of memory disorders and epileptic seizures. The purpose of this report is to emphasize the importance of early recognition of ALE, timely therapeutic escalation, and early psychological and neurocognitive rehabilitation to preserve the patient's functional ability and quality of life.

CASE REPORT: We present a 23-year-old male patient who suffered from serous meningoencephalitis of unknown etiology in 2013. After complete recovery, he achieved full functional recovery. In 2022, he presented with febrile illness, photophobia, and sudden onset of anterograde amnesia and hallucinations. The diagnosis of seronegative ALE was established based on Graus' criteria, following the subacute onset of symptoms, MRI evidence of bilateral temporal lobe involvement, and the exclusion of infectious, toxic, or neoplastic causes. Due to prodromal symptoms, empirical antimicrobial (ceftriaxone) and antiviral (acyclovir) therapy was initiated**.** Electroencephalography registered epileptiform activity. Extensive serological work-up and cerebrospinal fluid analysis for specific onconeural antibodies were negative. Pulse corticosteroid therapy (methylprednisolone) and intravenous immunoglobulins were administered, but no significant improvement in memory was observed. Follow-up MRI showed significant regression of edema, while clinical signs persisted. Currently, the patient is disoriented, anxious, and dependent on his family for daily functioning.

CONCLUSION: This case demonstrates that in ALE, despite timely diagnostic work-up and regression of neuroradiological signs, significant cognitive deficits may persist, resulting in permanent functional limitations and highlighting the unpredictable outcomes of treatment. Such patients require a multimodal and multidisciplinary treatment approach, with continuous stimulation and encouragement to support rehabilitation and progress.

KEYWORDS:
Amnesia, Anterograde; Autoimmune Diseases of the Nervous System; Electroencephalography; Immunoglobulins, Intravenous; Limbic System

LATE DIAGNOSIS OF ATTENTION DEFICIT DISORDER- A CASE REPORT

Sofija Tomas1, Marta Lugarić1, Ivo Todorić1**,** Fran Tomica1 Maja Bajs Janović1,2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 University Hospital Centre Zagreb, Department of Psychiatry, Zagreb, Croatia
Presenting author: Sofija Tomas, sofija.tomas99@gmail.com

Introduction
Attention-deficit disorder (ADD) is a neurodevelopmental condition that could go unrecognized in childhood, adolescence, and adulthood. It is characterized by dysregulation of dopaminergic neurotransmission affecting attention, executive functions, and reward processing, presenting as inattention, reduced motivation, and impaired task initiation. In adults, diagnosis requires a minimum of 5 symptoms of inattention, present for at least six months, that result in clinically significant functional impairment. Neuropsychological testing, while not required, may aid in complex cases.

Case Report
A 19-year-old male presented with social anxiety, hypobulia, diminished motivation, emotional dysregulation, insomnia, low self-esteem, depressive and passive suicidal ideation, and recurrent self-harm, without history of severe trauma. He had finished high school, lived with his parents, was unemployed, and lacked clear goals. Psychiatric evaluation and psychological testing concluded major depression with suspected borderline personality disorder. Treatment included an antidepressant (SSRI), an anxiolytic, and both group and individual psychotherapy over two years, resulting in improvement in depressive symptoms, anxiety, and resolution of suicidality, with partial functional recovery. Focus stayed on the residual symptoms: lack of motivation, sense of exhaustion and energy loss with consequent impaired engagement in education or employment. Later in treatment, the patient revealed specific rituals that were helping in everyday accommodations. Medication was changed to an SNRI antidepressant without improvement. Neuropsychological testing revealed impaired sustained attention and abstract reasoning, indicating executive dysfunction. The patient suspected ADD and completed the online screening. Further psychiatric treatment allows for a period of observation, including monotherapy methylphenidate, as depressive symptoms were absent. Psychotherapy is ongoing with a modified approach.

Conclusion
This case highlights the importance of recognizing motivational impairment as a potential manifestation of dopaminergic dysfunction in the late-diagnosed adult ADD. Comorbid depression and anxiety with insomnia and problems of functioning could be masking an underlying neurodevelopmental condition, leading to delayed diagnosis and treatment.

Keywords:
Attention Deficit Disorder; Borderline Personality disorder; Dopamine; Major depressive disorder; Motivation

EARLY-ONSET FRONTOTEMPORAL DEMENTIA PRESENTING AS ACUTE PSYCHOSIS IN A YOUNG ADULT

Marija Bunić1, Dora Gašparović1, Ivana Ivoš1, Antonia Mišić1, Valentin Kordić1,2
1 Faculty of Medicine Osijek, University of J. J. Strossmayer, Osijek, Croatia
2 Psychiatry Clinic, Clinical Hospital Centre Osijek, Osijek, Croatia
Presenting author: Marija Bunić, mbunic33@gmail.com

Introduction

Frontotemporal dementia (FTD) is rare in young adults and may initially mimic primary psychiatric disorders. Prominent behavioural changes, cognitive decline, and negative symptoms can lead to misdiagnosis, delayed recognition, and prolonged exposure to psychiatric stigma. This case highlights the importance of thoroughly excluding organic causes before attributing symptoms to a primary psychiatric condition.

Case Report

A 24-year-old woman with normal early development who had completed higher education, and had no family history of psychiatric or neurological disorders, presented with progressive behavioural and cognitive changes over a year, following a psychosocial stressor. Early symptoms included emotional blunting, social withdrawal, regression to childlike behaviours, impulsive stealing, stereotyped movements, and functional decline. Neuropsychological assessment revealed a marked decline from presumed premorbid functioning, with severe deficits in attention, executive functions, verbal learning, and memory, with high suggestibility and confabulation. Personality evaluation indicated pronounced defensiveness, denial, and dissimulation. Preserved premorbid cognitive abilities and cognitive reserve likely masked early disease manifestations, contributing to delayed recognition. Initially, the patient was diagnosed with an acute and transient psychotic disorder (ICD-10 F23.9) and treated with multiple antipsychotics, with minimal clinical improvement. This lack of response to psychiatric treatment represented a critical clinical indicator that should have prompted earlier neuroimaging. Due to atypical progression and treatment resistance, a comprehensive somatic and neurological evaluation was undertaken. Brain magnetic resonance imaging revealed marked bilateral frontal and temporal lobe atrophy, involving hippocampi, corpus callosum, and caudate nuclei, consistent with a neurodegenerative process. Extensive cerebrospinal fluid, autoimmune, metabolic, and genetic testing excluded secondary causes. The final diagnosis was early-onset frontotemporal dementia.

Conclusion

This case highlights the importance of systematically excluding organic and neurodegenerative causes in young patients presenting with atypical psychosis and behavioural changes. Premature psychiatric labelling may delay accurate diagnosis and expose patients to unnecessary treatment and stigma.

Keywords:
Frontotemporal Dementia; Psychotic Disorders; Neurodegenerative Diseases; Magnetic Resonance Imaging

MILD COGNITIVE DEFICITS AND BILATERAL PARIETAL HYPOPERFUSION IN A YOUNG PATIENT WITH CHRONIC CANNABIS USE

Fran Tomica1, Sofija Tomas1, Nataša Klepac1,2Maja Bajs Janović1,2
1 School of Medicine, University of Zagreb, Zagreb, Croatia
2 Clinical Center Zagreb, Clinic of Psychiatry, Zagreb, Croatia
Presenting author: Fran Tomica, frantomica85@gmail.com

Introduction

Cannabis modulates neurotransmission via CB1 receptors within cortical and subcortical networks implicated in attention and working memory. Chronic exposure has been associated with alterations in synaptic plasticity and regional cerebral perfusion. Brain SPECT using Tc-99m HMPAO allows assessment of functional perfusion patterns in patients presenting with subtle cognitive complaints.

Case Report

A 31-year-old man was referred for neurological and psychiatric evaluation due to progressive subjective difficulties with concentration, forgetfulness, and reduced efficiency in acquiring new information. Emotional dysregulation with mood swings, irritability, impulsivity, and attention disturbance was also observed. He reported long-standing daily use of large amounts of cannabis. His medical history was negative for traumatic brain injury, epilepsy, or other neurological disorders.

Neurocognitive testing showed largely preserved cognitive functioning, including intact attention, processing speed, and executive functions, without evidence of frontal dysfunction. Memory was generally preserved, with mild encoding difficulty and a slight reduction in working memory performance on longer sequences, suggesting borderline attentional capacity.

Brain SPECT (Tc-99m HMPAO) demonstrated mildly reduced tracer uptake bilaterally in the parietal cortices, without significant lateralization. No focal areas of hypoperfusion or hyperperfusion were identified. Perfusion of the frontal and temporal lobes, basal ganglia, thalami, cerebellum, and brainstem was preserved.

Conclusion

This case demonstrates mild cognitive deficits predominantly affecting attention and working memory, accompanied by bilateral parietal hypoperfusion. The perfusion pattern is not characteristic of ADHD, given the absence of frontal hypoperfusion and preserved executive functioning. While the findings may be consistent with functional alterations within parietal associative networks, a causal relationship with chronic cannabis use cannot be established based on a single case. The findings are nonspecific and warrant longitudinal follow-up with reassessment after abstinence to further clarify their clinical significance and potential reversibility.

Keywords:
Attention; Brain; Cannabis; Cognitive Dysfunction; Single-Photon Emission Computed Tomography

PSYCHOTIC DECOMPENSATION IN THE CONTEXT OF SUBSTANCE USE: DOPAMINERGIC AND IMMUNE DYSREGULATION AFTER PROLONGED ABSTINENCE

Ana Šestanj1; Leonarda Novoselec1; Anamarija Bećarević1; Ema Purgar1; Andrijana Šantić (Mentor)1,2
1 Faculty of Medicine Osijek, University of J. J. Strossmayer, Osijek, Croatia
2 Department of Psychiatry, Clinical Hospital Centre Osijek, Osijek, Croatia
Presenting author: Ana Šestanj, ana.sestanj31@gmail.com

Introduction

Psychotic disorders associated with psychoactive substance use represent a complex clinical entity in which neurobiological vulnerability and psychosocial factors interact. Although commonly linked to acute intoxication, psychotic symptoms may persist or even emerge after prolonged abstinence. This case is notable for progressive psychotic decompensation despite sustained abstinence, with partially preserved insight, raising important diagnostic and therapeutic challenges.

Case Report

We present a 37-year-old female patient with borderline personality disorder and long-standing polysubstance dependence, primarily opioids, treated with buprenorphine (6–8 mg/day). Since her first hospitalization in 2011, she has been continuously treated with pharmacotherapy, psychoeducation, and addiction monitoring, with occasional relapses accompanied by transient psychotic symptoms. Over time, however, psychotic phenomena became increasingly pronounced and less temporally related to substance use. In early 2025, after several months of maintained abstinence, the patient developed progressive anxiety, insomnia, persecutory ideation, and auditory disturbances, leading to admission to a day hospital, where risperidone was introduced with partial response. Over the following two months, further deterioration occurred, including intensification of nihilistic thoughts, fear of being followed, and perceptual disturbances, requiring inpatient hospitalization. Limited response to risperidone (up to 6 mg/day) prompted a switch to fluphenazine and the introduction of valproate as a mood stabilizer, alongside continued buprenorphine therapy. This resulted in partial stabilization; however, residual psychotic symptoms persisted despite sustained abstinence and preserved insight. Prolonged substance exposure may contribute to this vulnerability through dopaminergic sensitization and low-grade immune activation. Psychodynamically, symptoms may reflect regression to more primitive levels of self-experience, manifested through somatic and nihilistic themes.

Conclusion

This case emphasizes that psychotic symptoms in substance use disorders may persist independently of intoxication, complicating the distinction between substance-induced and primary psychosis. It highlights the need for careful longitudinal assessment and individualized treatment approaches in patients with enduring vulnerability.

Keywords:
Dopamine; Immune system; Personality disorders; Psychotic disorders; Substance-related disorders

BEYOND THE SCALPEL: SOMATIC-TYPE DELUSIONAL DISORDER DRIVING REPEATED SURGICAL AND DIAGNOSTIC INTERVENTIONS

Evelin Abdić1, Marina Letica Crepulja1,2
1 University of Rijeka, Faculty of Medicine, Rijeka, Croatia
2 Clinical Hospital Centre Rijeka, Department of Psychiatry, Rijeka, Croatia

Presenting author: Evelin Abdić, evelin.abdic@student.uniri.hr

Introduction

Somatic type delusional disorder is a chronic psychotic condition characterized by fixed false beliefs of having a medical illness despite repeated negative evaluations and reassurance. The disorder often presents with persistent somatic complaints that can mimic “actual” somatic disease, posing a significant diagnostic challenge in clinical practice.

Case Report

A 40-year-old male patient presented to the Emergency Department of Clinical Hospital Centre Rijeka with polymorphic symptoms in the chin region, describing recurrent “excess tissue” that appeared and disappeared. He had previously received a dermal filler after cervical schwannoma surgery and believed his current symptoms indicated tumor recurrence. Sixteen of his teeth were extracted because of persistent dental complaints. Excision of a chin tumefaction showed normal histopathology. Two months later he again requested surgery by a maxillofacial surgeon. Over the next two years, he underwent numerous specialist consultations, including rheumatology, immunology, plastic surgery, oral surgery, neurosurgery, otorhinolaryngology, and infectious diseases. Extensive diagnostics included computed tomography of the viscerocranium and brain, skeletal scintigraphy, magnetic resonance imaging, and multiple ultrasound examinations.After his seventeenth visit to the maxillofacial surgeon within two years, psychiatric referral was made. Medical documentation described persistent distress focused on perceived bodily symptoms, accompanied by a fixed belief in a serious medical condition despite repeated medical reassurance and lack of objective findings. The patient demonstrated marked distrust of physicians and medical explanations, resulting in poor treatment cooperation and functional impairment in daily life. These features led to the diagnosis of somatic-type delusional disorder. The patient did not attend the recommended follow-up psychiatric evaluation.

Conclusion

Somatic type delusional disorder imposes a significant burden not only on the affected individual but also on his surroundings. This case demonstrates how a psychiatrically undiagnosed patient can substantially impact healthcare resources, underscoring the importance of early diagnosis, appropriate referral, and timely treatment.

Keywords:
Delusional Disorder; Health Resources Utilization; Psychotic Disorders; Referral and Consultation; Somatoform Disorders

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