ISSN 1392-3064
eISSN 2424-5917

LIETUVOS NEUROLOGŲ ASOCIACIJA
LIETUVOS VAIKŲ NEUROLOGŲ ASOCIACIJA
LIETUVOS NEUROCHIRURGŲ DRAUGIJA

Neurologijos seminarai

The 2nd INTERNATIONAL VILNIUS NEUROSCIENCE CONFERENCE FOR YOUNG RESEARCHERS

ABSTRACT BOOK

APRIL 17–18, 2026

2026, PRIEDAS

ISSN 1392-3064 • eISSN 2424-5917

LIETUVOS NEUROLOGŲ ASOCIACIJA
LIETUVOS VAIKŲ NEUROLOGŲ ASOCIACIJA
LIETUVOS NEUROCHIRURGŲ DRAUGIJA

Neurologijos seminarai

2026, PRIEDAS

VILNIAUS UNIVERSITETO LEIDYKLA
2026

NEUROLOGIJOS SEMINARAI
SEMINARS IN NEUROLOGY

Leidžiamas nuo 1997 m.
Steigėjas: VU MF Neurologijos klinika

Redaktorių kolegijos adresas:
Vilniaus universiteto ligoninė Santaros klinikos
Santariškių g. 2, LT-08661
Tel./faks. (8 5) 236 5220
El. p. ruta.mameniskiene@santa.lt; dalius.jatuzis@santa.lt
https://www.zurnalai.vu.lt/neurologijos_seminarai

Editorial address:
Vilnius Univer sity Hospital Santaros Klinikos
Vilnius Santariškių 2, LT-08661 Vilnius, Lithuania
Tel./fax (370 5) 236 5220
E-mail: ruta.mameniskiene@santa.lt; dalius.jatuzis@santa.lt
https://www.zurnalai.vu.lt/neurologijos_seminarai

„Neurologijos seminarai“ įtraukti į / Indexed in: Scopus, PMC, Dimensions, DOAJ, Google Scholar, JournalTOCs, PubMed, Medline, Index Copernicus, Index Academicus, EBSCO (Academic Search Complete), Microsoft Academic, Scilit, ScienceGate, Scinapse, ROAD.

https://doi.org/10.15388/NS.2026.Supplement

The 2nd INTERNATIONAL VILNIUS NEUROSCIENCE CONFERENCE FOR YOUNG RESEARCHERS

ABSTRACT BOOK

APRIL 17–18, 2026

FACULTY OF MEDICINE,
VILNIUS UNIVERSITY
Vilnius, Lithuania

The conference is organised by researchers of the Faculty of Medicine of Vilnius University and endorsed by the Lithuanian Neurologists' Association, the Lithuanian Society for Epileptology, and the Lithuanian Stroke Association.

Scientific Committee

Prof. **Rūta Mameniškienė**, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Prof. **Saulius Ročka**, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Prof. **Sigita Lesinskienė**, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Prof. **Osvaldas Rukšėnas**, Life Sciences Center, Vilnius University, Vilnius, Lithuania
Prof. **Kęstutis Petrikonis**, Lithuanian University of Health Sciences, Kaunas, Lithuania
Prof. **Makoto Suematsu**, Central Institute for Experimental Medicine and Life Science, Kawasaki, Kanagawa, Japan
Prof. **Natan Bornstein**, Neurology Department, Shaare-Zedek Medical Center, Jerusalem, Israel
Algirdas Dambrava, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Kristijonas Puteikis, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Organising Committee

Prof. **Rūta Mameniškienė**, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Prof. **Dalius Jatužis**, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Kristijonas Puteikis, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Emilijus Žilinskas, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Karolina Podvorskytė, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Aistė Meškėlaitė, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
Marija Jakiševaitė, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Vilnius University Press
9 Saulėtekio Av., III Building, LT-10222 Vilnius
info@leidykla.vu.lt, www.leidykla.vu.lt/en/
Printed by UAB „Baltijos kopija"
Kareivių St. 13B, LT-09108 Vilnius
Print run 100 copies. 2.6 author's sheet

Contents

ADULT NEUROLOGY

PEDIATRIC NEUROLOGY

ADULT PSYCHIATRY

CHILD AND ADOLESCENT PSYCHIATRY

NEUROSURGERY

NEUROSCIENCE

An Autopsy-Based Study of Fatal Stroke Cases and Associated Factors

Milda Saunoriūtė[1], Sigitas Laima[2], Sigitas Chmieliauskas[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Department of Pathology and Forensic Medicine, Institute of Biomedical Sciences, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Despite advances in acute stroke management, a substantial proportion of fatal strokes occur outside healthcare settings, limiting opportunities for timely medical intervention. Autopsy-based studies provide valuable insights into the characteristics and contributing factors of fatal stroke.

Methods. A retrospective analysis of autopsies performed between 2013 and 2024 was conducted. For stroke-related deaths, demographic characteristics, ethanol concentration in biological fluids, stroke type, location, and etiology were analyzed.

Results. The study included 56 subjects (mean age 60±14 years), 21 (37.5%) of whom were women. Hemorrhagic stroke accounted for 94.64% (n=53) of cases, and ischemic stroke for 5.36% (n=3). Stroke localization was most frequent in MCA territory (32.14%, n=18), followed by BA (25%, n=14), PCA (16.07%, n=9), ACA (5.36%, n=3), and unspecified vascular territory (21.43%, n=12). Death occurred at home in 76.79% (n=43) and in a healthcare facility in 23.21% (n=13) of cases. Hemorrhage was in the left cerebral hemisphere in 39.29% (n=22), right hemisphere in 33.93% (n=19), brainstem in 21.43% (n=12), and both hemispheres in 5.36% (n=3) of cases. Etiology of stroke was artery rupture due to atherosclerosis in 30.36% (n=17), aneurysm in 5.36% (n=3), and unknown in 64.29% (n=36) of cases. Patients who died in hospital were older than those that died at home (69±16 vs. 58±12 years, p=0.04). Ethanol concentrations were significantly higher in those who died at home (blood 0.04±0.02‰ vs. 1.32±1.15‰, p=0.01; urine 0.29±0.16‰ vs. 1.75±1.71‰, p=0.02).

Conclusions. Fatal stroke most commonly occurs in patients around 60 years of age and is predominantly hemorrhagic, most often resulting from rupture of an atherosclerotic vessel; however, in more than half of the cases (64.29%), the exact etiology could not be determined. Elevated ethanol concentration is associated with sudden death from stroke outside healthcare settings.

Alcohol Craving Suppression and Recovery in LGI-1 Encephalitis

Odilija Rudytė[1], Jurgita Kuzmickienė[2], Nataša Giedraitienė[2], Mantas Vaišvilas[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Vilnius University, Vilnius, Lithuania

Introduction. LGI-1 antibody mediated autoimmune encephalitis (AE) is characterized by subacute onset of cognitive deficits, working memory impairments and behavioural changes. Due to the rarity of the disease, addictive behaviour is rarely studied in individuals with LGI-1 AE. Therefore, investigating LGI-1 AE provides an opportunity to probe the potential role of working memory dysfunction in the pathogenesis of addiction.

Methods. Single case description of clinical, paraclinical, laboratory, treatment and follow-up features.

Results. A 68-year-old male with a 10 year history of chronic alcohol abuse was diagnosed with LGI-1 AE by commercial cell-based assay and murine brain immunohistochemistry. Following a period of prodromal malaise and fever, the patient developed apathy, severe working memory deficits and abulia. As the disorder progressed, the patient refused any alcohol without intervention. The patient's initial MMSE score was 19 with mild dementia and temporospatial disorientation. Brain MRI revealed symmetrical grade II mesiotemporal atrophy. Immunotherapy, including steroids, plasma exchange, rituximab and cyclophosphamide was administered leading to improved cognitive function (MMSE 26) and re-emergence of the previously absent alcohol craving.

Conclusions. This case demonstrates that LGI-1 AE may suppress addictive behaviours due to mesiotemporal dysfunction affecting the meso-cortico-limbic dopaminergic network. Notably, the loss of alcohol craving represents an atypical behavioural manifestation in the course of the disease while recovery of working memory with treatment implementation may restore the reward seeking behaviour. These findings highlight the role of limbic integrity and cognitive function in addiction related behaviours in AE.

Correction of Motor Deficits in Patients with Relapsing–Remitting Multiple Sclerosis Using Amantadine

Nazarii Kalynovych[1], Kateryna Tarianyk[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Department of Neurology, Faculty of Medicine, Poltava State Medical University, Poltava, Ukraine

Introduction. Multiple sclerosis (MS) is a chronic neurological disease associated with progressive disability and reduced quality of life, particularly in working-age individuals. This study evaluated the effects of amantadine on motor deficits, muscle tone, cognitive function, and quality of life in MS patients.

Methods. Seventy patients with MS (mean age 35.5 ± 11.4 years) undergoing inpatient neurological treatment were examined. Assessments included the Expanded Disability Status Scale (EDSS), neuropsychological testing, and quality of life evaluation using the SF-36 questionnaire. Muscle tone was measured with the Modified Ashworth Scale and the Plastic Hypertonia Severity Scale.

Results. Patients were divided into three groups: Group 1 (n = 25; EDSS 3.0–3.5), Group 2 (n = 25; EDSS 2.0–2.5), and Group 3 (n = 20; EDSS 3.5–5.0). Motor deficits were present in 73% of patients, vestibular disturbances in 70%, sensory deficits in 63%, and neuropsychological impairment in 57%. Spastic hypertonia predominated (Ashworth scores 2–4). Following pulse therapy, all patients received intravenous amantadine for 5 days; Groups 2 and 3 continued oral amantadine (100 mg/day) for 3 months. Significant improvements in motor function and muscle tone were observed after 10 days ($p < 0.05$). At 3 months, Groups 2 and 3 showed further reductions in muscle tone, improved gait, better cognitive performance, and enhanced quality of life.

Conclusions. Amantadine added to standard therapy may improve motor activity by reducing spasticity and may positively influence cognitive function and quality of life in MS patients.

Neurological and Functional Symtom Burden During Head and Neck Radiotherapy: Interim Patient-Reported Outcomes from a Single-Centre Study

Karolina Šimkutė[1], Diana Meilutytė-Lukauskienė[2]

[1] Faculty of Medicine, Lithuanian University of Health Sciences, Kaunas, Lithuania
[2] Laboratory of Hydrology, Lithuanian Energy Institute, Kaunas, Lithuania

Introduction. Radiotherapy (RT) for head and neck cancer (HNC) is associated with acute neurotoxic and functional sequelae affecting peripheral nerves, mucosal sensory pathways, speech, and swallowing mechanisms. Many of these symptoms remain under-recognised in oncological practice. Patient-reported outcome measures (PROMs) enable systematic identification of early neurological and functional impairments that emerge during treatment.

Methods. A prospective observational study was conducted at the Lithuanian University of Health Sciences Oncology Hospital. Adult HNC patients undergoing active RT completed the EORTC QLQ-C30 and the head and neck–specific module (QLQ-H&N35). Clinically relevant symptom burden was defined as Likert-scale responses of 3 or higher. Descriptive statistics were used to determine the prevalence of functional, neurological, and psychological impairments.

Results. Interim analysis included 20 patients (15% female, 85% male). The mean age was 60.5 ± 3.4 years for females and 64.8 ± 7.3 years for males. Clinically relevant emotional dysfunction was reported by 41% of patients, and physical impairment by 40%. The most prevalent systemic symptoms were insomnia (60%), pain (57.5%), and fatigue (51.7%). Neurologically oriented impairments were frequent: chewing difficulties (55%), sticky saliva (50%), neuropathic pain (46.3%), speech disturbances (46%), coughing (45%), and xerostomia (45%). Sensory disturbances were less common (20%). Females tended to report a higher symptom burden across several domains, although the sample size was limited.

Conclusions. Active radiotherapy for head and neck cancer is associated with a substantial early neurological and functional symptom burden. Structured integration of patient-reported outcome measures may enhance detection of treatment-related neurofunctional toxicity and support proactive multidisciplinary management within radiotherapy workflows.

Diagnostic Challenges of Ataxia with Oculomotor Apraxia Type 2 (AOA2): A Case Report

Greta Galinytė[1], Gintarė Baranauskienė[2], Rūta Kaladytė Lokominienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Ataxia with oculomotor apraxia type 2 (AOA2) is an autosomal recessive cerebellar ataxia (ARCA) caused by mutations in the SETX gene. This disorder presents with progressive cerebellar ataxia, sensorimotor neuropathy, tremor and oculomotor abnormalities. Genetic testing of the SETX gene is essential for definitive diagnosis, but inconclusive results often require clinicians to rely on clinical findings such as elevated alpha-fetoprotein (AFP) levels.

Methods. This case report describes a 29-year-old female diagnosed with AOA2. The patient's data was collected from her medical records.

Results. Initial symptoms started in 2012 and included imbalance, gait instability, shaking in her hands and left leg. Brain MRI demonstrated atrophy of the cerebellum and electroneuromyography (ENMG) indicated sensory polyneuropathy of the legs. Genetic testing performed in 2020 identified two SETX variants NM_015046.7:c.675dupC(;)7364A>G, however, a definitive diagnosis could not be made, as one variant was of uncertain significance and the other likely pathogenic. The characteristic AFP elevation was absent at disease onset and significantly increased only after the disease progressed. Moreover, patient's disease progression was unusually aggressive – she had to use a wheelchair within 12 years of symptom onset, which is considerably faster than the typical 20-year timeline. The diagnosis of AOA2 was made based on previous clinical, laboratory, and radiological findings after 10 years of symptom onset.

Conclusions. This case highlights the complexity of AOA2 diagnosis when initial biomarkers are normal and genetic testing does not provide a definitive answer, demonstrating the need for detailed clinical examination and follow-up testing in suspected cases.

Paraneoplastic Encephalomyelitis Progressing to Drug-Resistant Temporal Lobe Epilepsy: A 10-Year Case Analysis

Unė Baranauskaitė[1], Eglė Navickienė[2], Mantas Vaišvilas[3], Arminas Jasionis[3]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Center of Neurology, Vilnius University Hospital Santaros Klinikos, Vilnius, Lithuania
[3] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania.

Introduction. Paraneoplastic neurologic syndromes (PNSs) are immune-mediated disorders linked to an underlying cancer, causing neurological dysfunction without direct tumour invasion or metastasis. While seizures are common in autoimmune encephalitis and often respond to immunotherapy, epilepsy associated with high-risk paraneoplastic antibodies frequently becomes resistant to treatment.

Methods. We present a 10-year longitudinal analysis of a paraneoplastic encephalomyelitis case progressing to drug-resistant temporal lobe epilepsy.

Results. Ten years ago, a previously healthy man in his twenties developed left-sided hemiparesis, hemiataxia, worsened vision and hearing, as well as bulbar symptoms (dysphonia, dysphagia). ENMG demonstrated left peroneal neuropathy, brain MRI and CSF were initially unremarkable. One year later, he experienced his first generalized tonic-clonic seizure, with EEG showing interictal right temporal epileptiform discharges. The following year, a mediastinal yolk sac tumour was detected and successfully treated. In subsequent years, focal seizures with déjà vu aura recurred but seizure control was reached with a combination of three antiepileptic drugs. Repeated MRI demonstrated persistent right amygdala enlargement with T2 hyperintensity, concordant with epileptogenic EEG findings. A decade after symptoms onset, daily focal seizures emerged. Autoimmune testing revealed strongly positive paraneoplastic antibodies in serum and CSF, confirmed by immunohistochemical analysis on rodent tissues. Methylprednisolone pulse therapy was ineffective but treatment with rituximab led to marked EEG improvement and a significant reduction of seizure frequency.

Conclusions. This case highlights the importance of considering PNS in patients with drug-resistant epilepsy and atypical neurological features, even years after tumour treatment. Late intervention with rituximab may still be effective for autoimmune-associated epilepsy.

Hidden Nocturnal Frontal Lobe Epilepsy Presenting as Chronic Insomnia and Affective Symptoms: A Case Report

Tamara Antia[1], Nino Gzirishvili[1,2]

[1] American Hospital Tbilisi, Tbilisi, Georgia
[2] Georgian American University, Tbilisi, Georgia

Introduction. Distinguishing sleep-related parasomnias from nocturnal frontal lobe epilepsy (NFLE) remains a significant diagnostic challenge. Repetitive nighttime motor behaviors are frequently misidentified as primary sleep disturbances, often resulting in inappropriate psychiatric labeling and years of delayed neurological treatment. We present the case of a 22-year-old male whose persistent insomnia and progressive depressive symptoms were long attributed to childhood-onset parasomnia. For nearly a decade, management focused on behavioral and affective complications while the underlying epilepsy remained unrecognized.

Methods. The patient's history included sleepwalking and recurrent nocturnal arousals since early childhood. By 2022, disabling insomnia, anxiety, and depressive features became the primary clinical focus, leading to treatment with escitalopram and hydroxyzine. Due to treatment resistance and the atypical nature of the nocturnal events, the clinical team performed 24-hour video-electroencephalogram (VEEG) and a 3-Tesla brain MRI.

Results. VEEG captured a habitual event 15 minutes after sleep onset during the N3 stage, lasting 46 seconds. The episode featured focal clonic movements of the left upper limb, vocalization, and a sustained gaze, followed by a rapid return to full awareness. The study revealed generalized high-amplitude delta activity associated with the seizure, while the 3-Tesla MRI showed no structural abnormalities.

Conclusions. Symptoms initially interpreted as insomnia with secondary depression were manifestations of epilepsy. This case emphasizes the necessity of prolonged VEEG evaluation for patients with atypical or refractory nocturnal behaviors to prevent the misclassification of neurological disorders as psychiatric or primary sleep conditions.

Association Between FINDRISK Risk Categories and Sensory Neuropathy: The NEUROPA Study

Austėja Šimkutė[1], Irena Žukauskaitė[2], Arūnas Vaitkevičius[3], Antanas Norkus[4,5], Jūratė Gudonytė[6]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Institute of Psychology, Faculty of Philosophy, Vilnius University, Vilnius, Lithuania
[3] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Vilnius University, Vilnius, Lithuania
[4] Public Institution Trakai Primary Health Care Center, Trakai, Lithuania
[5] Public Institution Kaišiadorys District Health Center, Kaišiadorys, Lithuania
[6] Pharmacy and Pharmacology Center, Institute of Biomedical Sciences, Faculty of Medicine, Vilnius University, Vilnius, Lithuania.

Introduction. Type 2 diabetes mellitus (T2DM) and impaired glucose tolerance are major causes of sensory neuropathy (SN), accounting for up to 50% of cases. Neuropathic changes may occur before clinical T2DM onset. The FINDRISK questionnaire estimates 10 year T2DM risk and is widely used for early risk stratification. This study evaluated the significance of FINDRISK risk groups in identifying SN among individuals aged ≥45 years.

Methods. This cross-sectional study included 273 individuals aged ≥45 years without diagnosed T2DM. Based on FINDRISK scores, participants were categorized as ≤11 points (low/slightly elevated risk; probability 1/100-1/25) or ≥12 points (moderate, high and very high risk; probability 1/6, 1/3 and 1/2). Groups were comparable by age and sex. SN was assessed using Neurometer® (2000, 250, 5 Hz), analyzing quantitative sensory thresholds and normal/abnormal values. Pain characteristics were evaluated using the McGill Pain Questionnaire.

Results. The lower risk group demonstrated significantly lower bilateral Neurometer® sensory thresholds, indicating better sensory function ($p<0.05$). Abnormal findings were less frequent, particularly in the right foot at 250 Hz (53.3% vs 68.5%, $p=0.013$) and 5 Hz (44.2% vs 58.3%, $p=0.023$). Neuropathic (stabbing) pain was less common in the lower risk group (12.4% vs 22.6%, $p=0.041$), with no differences in other McGill pain descriptors (e.g., burning, aching). Regarding pain localization, knee pain was reported more often in the higher risk group (19.4% vs 10.2%, $p=0.049$).

Conclusions. Higher FINDRISK scores were associated with greater likelihood of SN even without T2DM. FINDRISK may support early identification for targeted SN screening and prevention.

Prevalence and Determinants of Suicidal Ideation in Adults With Epilepsy: A Large Cross-Sectional Study

Sofiia Martynova[1], Kristijonas Puteikis[1], Gytis Motiejūnas[2], Rūta Mameniškienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. People with epilepsy are at increased risk of suicidal ideation, yet the relative contribution of epilepsy-related characteristics versus psychosocial and psychiatric vulnerability remains debated. Clarifying these relationships is essential for targeted screening and prevention.

Methods. We conducted an anonymous cross-sectional survey of 1,002 adult outpatients with epilepsy at a tertiary neurology centre. Data on sociodemographic variables, epilepsy characteristics, antiseizure medication use, and personal and family psychiatric history were collected. Recent suicidal ideation was assessed using item 9 of the Beck Depression Inventory. Factors associated with suicidal ideation were examined using univariable analyses and multivariable binary logistic regression.

Results. Recent suicidal ideation was reported by 18.5% of participants. In multivariable analysis, suicidal ideation was independently associated with unemployment (OR = 1.935, 95% CI = 1.173 to 3.19, p = 0.010), living in a town rather than a city (OR = 1.722, 95% CI = 1.098 to 2.698, p = 0.018), current clonazepam use (OR = 2.443, 95% CI = 1.244 to 4.794, p = 0.009), a lifetime history of pharmacological treatment for anxiety or depression (OR = 3.050, 95% CI = 1.797 to 5.17, p < 0.001), and, most strongly, past suicidal ideation (OR = 10.024, 95% CI = 6.232 to 16.122, p < 0.001). Epilepsy-related variables, including seizure type, seizure frequency, and epilepsy duration, were not independently associated with recent suicidal ideation.

Conclusions. Our findings indicate that psychiatric history and psychosocial vulnerability play a prominent role in recent suicidal ideation among adults with epilepsy when compared to epilepsy-related clinical factors. These results support the need for routine assessment of psychiatric history and prior suicidality in epilepsy care, beyond a seizure-focused evaluation.

Overlapping Features of Multiple Sclerosis and Suspected Spinocerebellar Ataxia: A Case Report

Arshia Tabassum Abdul Rahman[1], Kristina Grigalionienė[2], Rasa Kizlaitienė[3]

[1] Faculty of medicine, Vilnius University
[2] Center of Medical Genetics, Vilnius University Hospital Santaros Klinikos, Vilnius, Lithuania
[3] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University

Introduction. Differentiating between hereditary neurodegenerative disorders and acquired demyelinating diseases can be challenging when clinical and radiological features overlap. We present a case of suspected spinocerebellar ataxia (SCA) with concurrent multiple sclerosis (MS)-like findings.

Methods. A 38-year-old female presented with gradually progressive gait instability, leg weakness, and imbalance since 2020, worsening over the last year. She underwent neurological examination, brain and spine MRI, cerebrospinal fluid (CSF) analysis, evoked potentials, blood tests, and genetic consultation. Clinical data were collected from medical records.

Results. Examination showed spastic paraparesis, left arm paresis, marked ataxia, dysphonia, and nystagmus. Expanded Disability Status Scale (EDSS) score was 6.0. Brain MRI revealed multiple demyelinating lesions in periventricular, cerebellar, and spinal cord (C1-C2) regions, some with contrast enhancement. Spine MRI showed diffuse spinal cord atrophy (reduced to 4.1 mm) and a dilated central canal (1.7 mm). CSF analysis demonstrated identical oligoclonal bands in CSF and serum, with no intrathecal IgG synthesis. Evoked potentials showed delayed central conduction. Blood tests excluded aquaporin-4 and myelin oligodendrocyte glycoprotein (MOG) antibodies, HIV, syphilis, and hepatitis. The patient received methylprednisolone with minimal improvement, followed by Ocrelizumab with initial stabilization. Genetic testing for SCA types 1,2,3,6,7 performed in February 2026 showed no pathogenic repeat expansions.

Conclusions. This case represents a diagnostic dilemma: radiographic and clinical features suggest MS, but the progressive course, absent intrathecal IgG synthesis, and spinal cord atrophy raised suspicion for an underlying hereditary ataxia. Genetic testing ruled out common SCA subtypes, reinforcing the MS diagnosis while highlighting the importance of broad differential screening in atypical presentations.

Prehospital and Emergency Department Stroke Recognition: Diagnostic Accuracy Across Different Care Pathways

Dovydas Nagelė[1], Paulius Uksas[2], Rytis Masiliūnas[3], Aleksandras Vilionskis[3]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Emergency Medicine, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[3] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Accurate stroke recognition and early activation of code stroke are essential for timely treatment. This study compared the diagnostic accuracy of stroke suspicion between Emergency Medical Services (EMS) and referring physicians and evaluated the performance of FAST in prehospital and emergency department (ED) settings.

Methods. We conducted a retrospective cohort study including all patients with code stroke activated by EMS or in the ED of Republican Vilnius University Hospital between March and June 2025. Stroke was defined by a final diagnosis including ICD-10 codes I60, I61, I63, I64 or G45. Demographics, code stroke initiator, preliminary and final diagnoses, neurological symptoms and FAST assessments were extracted from electronic medical records. FAST sensitivity and specificity were calculated against final diagnosis. Agreement between triage nurse and neurologist FAST assessments was evaluated using Gwet's AC1 and Cohen's kappa.

Results. Among 450 patients, 308 (68.4%) were confirmed strokes. EMS demonstrated higher positive predictive value (PPV) than referring physicians (78.0% vs 45.1%, $p<0.001$). Sensitivity was high in both EMS (84.0%) and referral groups (88.9%). Against final diagnosis, FAST showed high sensitivity (87.3%) and moderate specificity (58.5%). Compared with neurologist examination, nurse FAST assessment demonstrated high sensitivity (90.6%) but low specificity (21.7%), suggesting a tendency toward overtriage. Nurse-neurologist agreement was moderate by AC1 (0.54) but low by kappa (0.15), reflecting prevalence effects.

Conclusions. Both EMS and referring physicians demonstrated high sensitivity in stroke recognition, though EMS achieved higher PPV. FAST is well suited for stroke screening in prehospital and ED settings, where sensitivity is prioritized.

Anti-NMDA and Anti-CASPR2 Overlap Syndrome: Clinical Features and Pathophysiological Mechanisms of Autoimmune Encephalitis. A Case Report

Neda Grikšaitė[1], Kristijonas Puteikis[1], Rūta Mameniškienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Autoimmune encephalitis (AE) is a group of severe, inflammatory, immune-mediated diseases of the central nervous system caused by autoantibodies directed against neuronal surface or synaptic proteins. The occurrence of multiple autoantibodies in AE is a rare phenomenon caused by several possible pathophysiological mechanisms. Dual seropositivity (anti-NMDAR and anti-CASPR2) causes an "overlapping" syndrome, resulting in a complex, atypical clinical and radiological phenotype.

Methods. A clinical case of a 65-year-old man with new-onset seizures is presented alongside a literature review of the etiology, diagnostics and clinical features of multiple auto-antibodies occurrence in autoimmune encephalitis.

Results. The patient arrived for consultation because of a single bilateral tonic-clonic seizure six months prior. Neither the patient nor his family reported subsequent seizures, behavioral changes or other health complaints. Radiological signs of limbic encephalitis were described on brain magnetic resonance tomography (MRI) and dual-positive (anti-NMDA and anti-CASPR2) autoimmune antibodies were detected in serum. The patient was followed by urologists because of a history of urinary bladder carcinoma (pTaN0M0, grade 1) that had been treated with transurethral resection. Videoelectroencephalography (vEEG) and videopolysomnography revealed bilateral interictal discharges in the frontotemporal areas, multiple focal seizures with subtle motor signs emerging from the same regions as well as a severe and predominantly central sleep apnea. Dual-positive autoimmune encephalitis antibodies were confirmed in cerebrospinal fluid. Chest X-ray, computed tomography and abdominal ultrasound revealed no abnormalities. Methylprednisolone pulse therapy led to rapid clinical improvement and resolution of epileptic activity on EEG.

Conclusions. This case reflects a relatively mild clinical presentation of double-positive autoimmune encephalitis accompanied by significant radiological and electrophysiological findings. This emphasizes the importance of a comprehensive evaluation of older patients with new-onset seizures as well as the possibility of subtle symptomatology in rare forms of autoimmune encephalitis.

Sleep Disturbances as a Key Manifestation of LGI-1 Limbic Encephalitis: A Case Report

Justė Rimšienė[1,2], Rūta Mameniškienė[1,2]

[1] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Center of Neurology, Vilnius University Hospital Santaros Klinikos, Vilnius, Lithuania

Introduction. Autoimmune encephalitis (AIE) is increasingly recognized in clinical practice and typically presents with subacute cognitive impairment, psychiatric symptoms, seizures, and autonomic dysfunction. Up to 80% of patients experience sleep disturbances (insomnia, hypersomnia, confusional arousals, various sleep apneas, parasomnias, REM sleep without atonia), which may represent the initial or even sole manifestation.

Case Presentation. We present a 52-year-old male with initial episodes of heat sensation in the lower extremities followed by transient disorientation, during which he repeatedly responded "everything is fine." He was initially misdiagnosed with transient global amnesia and vertebrobasilar transient ischemic attack. Early EEG and neuroimaging were unremarkable. Over the following six months, he developed progressive marked daytime hypersomnolence and nocturnal insomnia, along with recurrent episodes characterized by subjective tremor, piloerection, mydriasis, and facial flushing. A nocturnal generalized tonic–clonic seizure eventually occurred, prompting referral to an epileptologist. MRI showed limbic encephalitis, EEG revealed right temporal epileptiform activity, and LGI1 antibody-associated autoimmune encephalitis was confirmed. Polysomnography demonstrated fragmented sleep, frequent awakenings, obstructive sleep apnea with elevated apnea–hypopnea index (AHI), REM sleep without atonia, reduced REM sleep (4%), and frequent limb movements. After corticosteroid therapy and subsequent relapse, rituximab was initiated. Eighteen months later, the patient is seizure-free, with normalized sleep architecture, reduced AHI, decreased limb movements, and restored REM sleep (12%) with normal atonia.

Conclusions. Sleep disturbances may be the earliest or predominant manifestation of AIE. Although often improving with immunotherapy, they may persist during remission. Adequate sleep is essential for memory consolidation, seizure control, and quality of life. Therefore, AIE should be considered as a potential underlying cause in patients presenting with subacute-onset sleep disturbances.

A Rare Case of Proximal Manifestation of Hirayama Disease

Richard Jehonatan Dudin[1], Daina Pastar[2]

[1] Faculty of Medicine, Rīga Stradiņš University, Riga, Latvia
[2] Department of Neurology, Riga East Clinical University Hospital, Riga, Latvia

Introduction. Hirayama disease is a rare, self-limited cervical flexion–induced myelopathy characterized by selective anterior horn cell involvement and asymmetric upper limb weakness without sensory deficits.

Case report. A 33-year-old patient presented with a 3–5-year history of progressive weakness and discomfort in the left arm. Neurological examination revealed proximal weakness with fasciculations and preserved sensation. No upper motor neuron signs were observed. Over time, symptoms stabilized without spread. During the diagnostic workup, the patient was diagnosed with stage II pulmonary sarcoidosis and thoracic outlet syndrome; however, immunosuppressive therapy did not improve neurological symptoms. Nerve conduction studies showed no sensory abnormalities or conduction block. Electromyography demonstrated chronic denervation changes consistent with a lower motor neuron process. Cervical flexion MRI was unavailable. Diagnosis was established based on clinical course, electrophysiological findings, and exclusion of inflammatory, compressive, and motor neuron disease.

Conclusions. This case highlights the diagnostic challenge of isolated unilateral lower motor neuron weakness in the context of coexisting systemic disease. Hirayama disease should be considered in patients with progressive but self-limiting upper limb weakness without sensory involvement, particularly when electrophysiology excludes conduction block and symptoms stabilize within several years.

Beyond Neuralgia: Identifying the Elongated Styloid Process as a Rare Cause of Cervicofacial Pain

Richard Jehonatan Dudin[1]

[1] Faculty of Medicine, Rīga Stradiņš University, Riga, Latvia

Introduction. Eagle syndrome is a rare condition caused by symptomatic elongation of the styloid process, leading to irritation of adjacent neurovascular structures. Its clinical presentation frequently mimics facial neuralgias, creating substantial diagnostic uncertainty.

Case report. A female patient presented to the Emergency Department of the Department of Neurology at Evangelisches Krankenhaus Unna with suspected trigeminal neuralgia. She reported paroxysmal, stabbing pain affecting the left hemiface with radiation to the left ear, accompanied by a subjective abnormal sensation in the ipsilateral tongue. Marked mechanical allodynia was present; even minimal contact triggered severe pain. However, symptom distribution did not correspond clearly to a single trigeminal nerve branch, and neurological examination showed no focal deficits. Given the atypical features, an extended differential diagnostic evaluation was undertaken. Craniofacial imaging demonstrated elongation of the left styloid process, establishing the diagnosis of Eagle syndrome after interdisciplinary assessment. Pharmacological therapy was initiated, and the patient is currently awaiting local invasive treatment through targeted pain blockade.

Conclusions. Eagle syndrome should be considered in patients with unilateral cervicofacial pain that does not conform to classic trigeminal nerve distribution, particularly when atypical sensory symptoms such as tongue dysesthesia are present. As styloid elongation may be incidental, diagnosis requires careful clinical radiological correlation to avoid prolonged misdiagnosis and unnecessary long term pharmacological treatment.

Genetics of Epilepsy – Literature Review

Laura Micevičiūtė[1], Rūta Mameniškienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Epilepsy is a common neurological disorder with significant etiological heterogeneity. Genetic factors play a major role, particularly in early-onset epilepsies, developmental and epileptic encephalopathies, and drug-resistant cases. Advances in next-generation sequencing have improved the identification of genetic causes and understanding of molecular mechanisms. This study aimed to review the spectrum of genetic epilepsies, assess genetic testing methods and their diagnostic yield, and evaluate the clinical significance of genetic diagnosis, including perspectives of precision medicine and gene therapy.

Methods. A narrative literature review of peer-reviewed publications on genetic epilepsies was conducted, focusing on genetic mechanisms, diagnostic methods, and clinical relevance.

Results. Genetic etiology was addressed in 40 studies, mainly focusing on specific genes and associated phenotypes. The diagnostic value of genetic testing was evaluated in 6 publications, particularly regarding next-generation sequencing methods. Treatment aspects were discussed in 4 studies, with 2 highlighting the impact of genetic findings on treatment decisions. Approximately 5 studies emphasized the role of genetic diagnosis in prognosis and genetic counseling. Overall, genetic etiology was the predominant focus, while treatment-related aspects were less frequently addressed.

Conclusions. Genetic epilepsies constitute a significant proportion of cases and should be considered in selected patients. Modern genomic technologies improve diagnostic accuracy and support personalized treatment strategies, potentially leading to better clinical outcomes.

Nonketotic Hyperglycinemia (NKH): Case Report and Literature Review

Agnė Grigaliūnaitė[1], Rūta Mameniškienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Nonketotic hyperglycinemia (NKH), also known as glycine encephalopathy, is a rare autosomal recessive inborn error of metabolism caused by dysfunction of the glycine cleavage system, leading to glycine accumulation in the central nervous system. The disorder typically presents in the neonatal period with severe neurological manifestations, but attenuated forms may present later and remain unrecognized for many years. The potential impact of early diagnosis and treatment initiation on neurological outcomes remains uncertain.

Methods. A clinical case of NKH was analyzed using anonymized patient data from Vilnius University Hospital Santaros Klinikos, and a literature review was performed in the PubMed database.

Results. A 47-year-old male with lifelong intellectual disability and pharmacoresistant epilepsy of previously unknown etiology underwent metabolic and genetic evaluation. Developmental delay was present from early childhood, and seizures began at four years of age. Brain magnetic resonance imaging showed no structural abnormalities. Plasma amino acid analysis revealed markedly elevated glycine levels (1145 μmol/L). Whole-exome sequencing identified a variant of uncertain significance in the GLDC gene. Based on clinical and biochemical findings, NKH was diagnosed more than four decades after symptom onset.

Conclusions. This case highlights the diagnostic challenges of this rare metabolic disorder and demonstrates that NKH may remain unrecognized for decades when metabolic etiologies are not considered. Early metabolic and genetic evaluation in patients with unexplained intellectual disability and epilepsy may allow earlier diagnosis and timely initiation of symptomatic treatment, although long-term neurological outcomes largely depend on the biological severity of the disease.

Capecitabine-Induced Encephalopathy: A Case Report and Diagnostic Challenges

Agnė Grigaliūnaitė[1], Dovilė Magenytė-Nemčinskė[1], Rūta Mameniškenė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Capecitabine is a fluoropyrimidine chemotherapeutic agent widely used in oncological practice. Although neurotoxicity is rare, it may present as acute or subacute encephalopathy and mimic other neurological conditions, creating significant diagnostic challenges.

Methods. A clinical case was analyzed using anonymized data from Vilnius University Hospital Santaros Klinikos, and a literature review was performed in the PubMed database.

Results. A 73-year-old man, after combined chemoradiotherapy for stage III colorectal cancer treated with the XENOX regimen (oxaliplatin and capecitabine) developed rapidly progressive encephalopathy during the final treatment cycle. Symptoms included cognitive decline, ataxia, and episodic disturbances of consciousness. During hospitalization (July 2025), moderate cognitive impairment was identified (MMSE 15), predominantly affecting orientation and attention. Marked axial ataxia with retropulsion, gait disturbance, and parkinsonism (bradykinesia, hypomimia, rigidity) were observed. EEG showed diffuse encephalopathic changes without epileptiform activity. Brain MRI revealed nonspecific vascular changes and later cortical diffusion restriction in the right temporoparieto-occipital region. Cerebrospinal fluid analysis showed minimal pleocytosis and elevated protein. Anti-Yo antibodies detected in CSF and serum suggested paraneoplastic encephalitis, and treatment with methylprednisolone pulse therapy and plasmapheresis followed by oral prednisone tapper was initiated. However, lack of confirmation and marked clinical improvement after capecitabine discontinuation led to revision of the diagnosis to drug-induced neurotoxicity. Clinical improvement was observed in August–September 2025 (MMSE 15→26), with residual mild cognitive and extrapyramidal symptoms.

Conclusions. Capecitabine-induced neurotoxicity may mimic paraneoplastic encephalitis. Early recognition is essential to avoid unnecessary immunosuppressive treatment and improve outcomes.

Low-Grade Glioma in Children: The Importance of a Multidisciplinary Approach

Radvilė Vaigauskaitė[1], Vilma Rutkauskaitė[2], Jelena Rascon[2,3], Monika Kapitančukė[2,3]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Center for Pediatric Oncology and Hematology, Vilnius University Hospital Santaros Klinikos, Vilnius, Lithuania
[3] Clinic of Children's Diseases, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. The most common central nervous system (CNS) tumor diagnosed in children is low-grade glioma (LGG). The predominant type of LGG is pilocytic astrocytoma (PA). Although complete surgical resection is associated with a favorable prognosis, these tumors may behave aggressively and present significant therapeutic challenges.

Methods. We present the challenging case of an 11-year-old boy who was diagnosed with PA at Vilnius University Hospital Santaros Klinikos.

Results. The patient started complaining of coordination impairment, fatigue, drowsiness, morning vomiting and cognitive regression at age 6. Cerebral magnetic resonance imaging (cMRI) revealed large tumor occupying the third ventricle along with marked periventricular edema and obstructive hydrocephalus. Partial resection was performed, and PA (WHO grade 1) was confirmed (KIAA1549::BRAF fusion positive). Postoperatively, the patient developed panhypopituitarism and cerebral venous sinus thrombosis. Later optic nerve atrophy with vision impairment as well as neurocognitive and speech disorders occurred. Low-molecular-weight heparin, L-thyroxine, hydrocortisone, and desmopressin were prescribed, alongside physiotherapy and close monitoring by a multidisciplinary team (MDT). Additionally, he required subdural peritoneal shunt. Progression of PA was confirmed in cMRI at age 8. Adjuvant chemotherapy with weekly intravenous vinblastine was initiated. PA stabilized during chemotherapy but started to progress at age 10. The boy is currently 11 years old and is receiving continuous targeted therapy with trametinib, with close cMRI scans and surveillance by a MDT.

Conclusions. Pediatric PA is benign CNS tumor with a high long-term survival rate. However, it can negatively impact patients' quality of life and potentially necessitate MDT care.

Medulloblastoma in a Child with Gorlin Syndrome: Calcifications or Metastasis

Greta Ždankutė[1], Elija Jankauskaitė[1], Greta Venckutė[2], Giedrė Rutkauskienė[2]

[1] Faculty of Medicine, Medical Academy, Lithuanian University of Health Sciences, Kaunas, Lithuania
[2] Department of Pediatrics, Medical Academy, Lithuanian University of Health Sciences, Kaunas, Lithuania

Introduction. Gorlin syndrome is a hereditary cancer predisposition disorder caused by germline mutations in the Sonic hedgehog (SHH) pathway, most commonly involving SUFU and PTCH1. SUFU mutations confer high risk of early-onset SHH-activated medulloblastoma. Radiotherapy is often avoided due to secondary malignancy risk, and characteristic intracranial calcifications may complicate imaging and mimic metastases or relapse.

Methods. We report a pediatric case of classic SHH-activated medulloblastoma associated with a germline SUFU mutation.

Results. A 16-month-old boy presented with vomiting, ataxia and abducens nerve palsy. Brain MRI revealed a fourth-ventricle tumor causing obstructive hydrocephalus with cerebellar leptomeningeal dissemination and bilateral calcifications, some demonstrating contrast enhancement; no spinal dissemination was detected. Near-total resection was performed. Histopathology confirmed classic medulloblastoma, SHH-activated, TP53 wildtype. Genetic testing identified a SUFU mutation. The patient received three induction and two maintenance chemotherapy courses. Treatment was complicated by myelosuppression and febrile neutropenia requiring antimicrobial therapy and transfusions. Subsequently, focal seizures and unilateral weakness developed. MRI showed progressive white matter changes consistent with posterior reversible encephalopathy syndrome (PRES) and calcified lesions. EEG was normal, carbamazepine was initiated. Given lesion distribution, temporal evolution, and the underlying SUFU mutation, imaging findings were interpreted as mutation-associated and treatment-related rather than relapse. The patient remains under surveillance without recurrence; his condition has improved.

Conclusions. In SUFU-associated Gorlin syndrome, intracranial calcifications and PRES may mimic medulloblastoma relapse. Accurate interpretation requires integration of genetic background, clinical course, and multidisciplinary imaging review to avoid overtreatment.

Augmentation Strategies in the Treatment of Obsessive-Compulsive Disorder: Antipsychotic Agents, Extended-Release Methylphenidate, and N-Acetylcysteine

Julija Grigaitytė[1], Robertas Strumila[1]

[1] Clinic of Psychiatry, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Obsessive-compulsive disorder (OCD) is a chronic mental disorder characterized by intrusive thoughts and repetitive behaviors that impair daily functioning and quality of life. Although first-line therapies help many patients, a significant number remain treatment-resistant, emphasizing the need for adjunctive interventions. Dysregulation of dopaminergic and glutamatergic systems, as well as oxidative stress, has been implicated in OCD, supporting investigation of pharmacological augmentation strategies, including antipsychotics and emerging agents such as N-acetylcysteine (NAC) and extended-release methylphenidate (MPH-ER).

Methods. A narrative literature review was conducted using PubMed, Embase, EBSCO, and Google Scholar, focusing on studies of pharmacological augmentation in patients insufficiently responsive to SSRIs or cognitive-behavioral therapy (CBT).

Results. Dopamine receptor antagonists, particularly risperidone, and the partial agonist aripiprazole show the most consistent benefit in SSRI-resistant OCD. Olanzapine and quetiapine may help selected patients, though supporting data are limited. Augmentation strategies targeting other pathways, such as NAC and MPH-ER, remain experimental. Preliminary findings suggest potential benefit with promising symptom improvement, but results are heterogeneous and often limited by small sample sizes and methodological variability.

Conclusions. Pharmacological augmentation is a key strategy for treatment-resistant OCD. Antipsychotics have the strongest evidence base, while NAC and MPH-ER show emerging potential but require larger, well-designed trials to clarify efficacy, safety, and patient selection.

The Impact of Psychoemotional Health on Judicial Decision-Making: A Neuroscientific Perspective

Rugilė Kriščiukaitytė[1], Laurynas Bukelskis[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Vilnius University, Vilnius, Lithuania

Introduction. The administration of justice is a cornerstone of democratic governance, and judges' ability to make objective decisions directly affects public trust in the legal system. Although legislation requires decisions to be based solely on the law, judges' decision-making can be influenced by psychoemotional health. Chronic stress, heavy workload, emotional exhaustion, and subclinical symptoms of depression or anxiety may modulate prefrontal cortex functions, executive functions, risk assessment, and emotion regulation, reducing decision-making efficiency and consistency. This study aimed to examine how judges' psychoemotional state, burnout risk, and stress interact with neurocognitive mechanisms underlying decision-making.

Methods. An integrated approach combining literature review, empirical analysis, and systematic evaluation was employed. A literature search was conducted in PubMed and Google Scholar using the keywords: "workplace stress and decision making," "decision making neuroscience," and "burnout syndrome." Eleven publications from 2015–2025 were selected, examining the effects of psychoemotional health, stress, and burnout on neurobiological decision-making processes. Empirical data included annual reports from the National Courts Administration on judges' workload. Systematic analysis reviewed court practice and legislation to identify relationships between workload, psychoemotional state, and decision quality.

Results. High workload and unfavorable psychoemotional conditions were associated with increased cognitive bias and reduced decision consistency. Professional experience often enhanced decision accuracy, but intensive or prolonged workload could negatively affect objectivity.

Conclusions. Psychoemotional state and neurocognitive mechanisms are key factors in judicial decision-making. Chronic stress and emotional exhaustion reduce information processing efficiency and decision consistency. Strengthening judges' psychoemotional well-being, optimizing workload, and mitigating cognitive bias are essential for supporting more deliberate, rational, and objective decisions.

Psychological Well-Being and Lifestyle Changes after Breast Cancer Diagnosis

Agnė Timofejevaitė[1], Smila Šedytė[1], Alvydas Navickas[2], Giedrė Bulotienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Vilnius University, Vilnius, Lithuania

Introduction. A breast cancer diagnosis represents a major life stressor that can profoundly impact psychological well-being and daily functioning. Understanding changes in emotional health, lifestyle interests, and coping behaviours is crucial for developing patient-centered care strategies.

Methods. A literature search was conducted in PubMed and Google Scholar for studies published between 2018 and 2025 on psychological health in women with breast cancer. An anonymous survey was distributed via social media groups for patients, and a supervising physician invited patients to participate via email. The survey assessed psychological symptoms, lifestyle interests, and daily functioning. A total of 153 respondents participated.

Results. Psychological difficulties were common post-diagnosis. Over half of participants reported anxiety and insomnia, while depressive symptoms were less frequent. Demand for mental health support increased, including visits to psychiatrists, psychologists, and psychotherapists, as well as use of anxiolytic medication. No rise in suicidal ideation was noted. Lifestyle changes included increased physical activity, heightened interest in psychology and well-being, and more engagement in leisure activities. Appreciation of life increased, whereas interest in work-related and political matters declined. No participants reported consulting alternative specialists such as astrologers or psychics.

Conclusions. Breast cancer diagnosis is associated with significant psychological distress, particularly anxiety and sleep disturbances, alongside meaningful shifts in lifestyle and coping behaviours. These findings underscore the importance of integrated psychosocial support within oncology care, addressing both emotional well-being and adaptive lifestyle changes.

Impact of Substance Use Disorders on the Clinical Outcomes of Schizophrenia

Agnė Timofejevaitė[1], Algirdas Dambrava[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Substance use is highly prevalent among patients with schizophrenia. Comorbidity between substance use disorders (SUDs) and schizophrenia is associated with an increased risk of relapse, partly due to disrupted medication adherence and worsening of symptoms. This study provides an overview of recent evidence on how tobacco, alcohol, cannabis, and stimulant use disorders affect the clinical outcomes of schizophrenia.

Methods. A literature search was conducted for studies published between 2020 and 2026 using EBSCO, PubMed, and ScienceDirect. Keywords included schizophrenia, substance use, and clinical outcomes. Studies involving adult patients with schizophrenia and addressing SUDs were included.

Results. Recent studies demonstrate that comorbid SUDs are associated with more severe clinical presentations compared to patients without SUDs. Tobacco smoking increases clinical burden, primarily by elevating cardiovascular risk and altering pharmacological management through CYP1A2 induction, which increases the metabolism of several psychotropic medications. Alcohol use is linked to higher rates of hospital admission, worsening of symptoms, increased extrapyramidal side effects, and greater mortality risk. Heavy and chronic cannabis use is associated with more frequent relapses and longer hospitalizations, even among adherent patients. Stimulant use disorders and polysubstance use disorders are associated with psychotic episodes and shorter time to antipsychotic discontinuation. Clinical management involves integrated care addressing both schizophrenia and SUDs, including pharmacological interventions and psychosocial support.

Conclusions. SUDs negatively impact the clinical course of schizophrenia, contributing to increased symptom severity, treatment challenges, and poorer outcomes. Integrated treatment approaches targeting both schizophrenia and SUDs are essential for improving long-term prognosis.

Psychotherapeutic Interventions in Benzodiazepine Deprescription

Eglė Jauniškytė[1], Smila Šedytė[1], Augustinas Žemaitis[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Benzodiazepines are commonly prescribed for anxiety, insomnia, and other psychiatric conditions; however, long-term use is associated with dependence, cognitive impairment, and increased risk of adverse outcomes. As deprescription becomes a clinical priority, psychological interventions such as cognitive behavioral therapy (CBT) and acceptance and commitment therapy (ACT) are increasingly recommended alongside gradual dose-reduction protocols to enhance discontinuation success.

Methods. A narrative literature review was conducted using PubMed and Google Scholar. From 113 screened publications, 25 relevant studies published between 2015 and 2026 were included, focusing on adult populations. Search terms included "benzodiazepine deprescription" AND "behaviour therapy."

Results. Both CBT and ACT improve benzodiazepine discontinuation outcomes, particularly when addressing underlying anxiety and insomnia. CBT-based interventions can increase abstinence success to approximately 70–80 %, whereas tapering guided solely by general practitioners achieves only 25–30 % success, with roughly 7 % maintaining long-term drug-free status. CBT is particularly effective short-term, reducing anxiety and modifying maladaptive beliefs, while ACT, more commonly applied long-term, enhances psychological flexibility and distress tolerance, allowing patients to manage withdrawal discomfort without reverting to medication. Although these therapies require sustained engagement and may involve higher costs, they reduce relapse risk, promote lasting behavioral change, and improve overall quality of life.

Conclusions. Combining gradual tapering with CBT or ACT significantly enhances discontinuation success, alleviates withdrawal symptoms, and decreases the likelihood of future benzodiazepine reuse, making these interventions an essential component of effective deprescription strategies.

A Calendar Effect in Suicidal Self-Poisoning: Sex-Stratified Seasonality Across 7 Years

Liudas Černius[1], Augustinas Žemaitis[2], Robertas Badaras[3]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[3] Clinic of Anaesthesiology and Intensive Care, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Seasonality in suicide is documented internationally and in Lithuania, but less is known about suicidal self-poisoning. This study aimed to assess seasonality patterns of self-poisoning suicide attempts, using data from Vilnius Republican University Hospital Toxicology Centre.

Methods. This study retrospectively analyzed 940 adult cases of self-poisoning suicide attempts. (Vilnius Republican University Hospital, 2018–2024). Data were grouped by month (n=84) and categorized as cold (October-April) or warm (May-September). Monthly average temperatures, precipitation, and sunshine duration were obtained or calculated as deviations from the standard climate norm from the Lithuanian Hydrometeorological Service website. Seasonal differences in counts and rates/day were tested overall and by sex (Welch t-test; Cohen's d), weather correlations assessed through Pearson and partial correlations, and independent effects via Poisson GLMs (log link, offset ln[days]). Analyses used SPSS v27.0.1.0.

Results. The warm season showed a descriptively higher overall burden than the cold season, by monthly counts (12.14 vs 10.47) and rate/day (0.397 vs 0.345). Sex-stratified analyses found no seasonal difference in males (p=0.728), while females had higher warm-season monthly counts (|d|=0.56; 95% CI 0.12–1.00; p=0.013). However, Poisson GLMs with an offset for ln(days) showed no independent associations of season or meteorological covariates in either sex (all p>0.05), with directionally lower cold-season point estimates.

Conclusions. The study demonstrated that seasonal differences in suicide by self-poisoning were small and inconsistent; unadjusted warm-season increases (notably in females) did not persist after adjustment. Neither season nor meteorological variables showed independent associations with suicidal self-poisoning.

Polypharmacy-Induced Delirium: A Case Report

Gabrielė Žilionytė[1], Gabrielė Dargytė[1], Laimutė Stunžėnienė[2]

[1] Institute for Clinical Medicine, Lithuanian University of Health Sciences, Kaunas, Lithuania
[2] Internal Medicine Institute, Hospital of Lithuanian University of Health Sciences, Kaunas, Lithuania

Introduction. Delirium is an acute disturbance of brain function, characterized by confusion, disorientation and hallucinations. In clinical practice, it is more common in older people and often becomes apparent at night. Risk increases when predisposing factors are present, such as cerebrovascular disease (CeVD) and anemia. It is often triggered by precipitating factors, such as medications, hospitalization, drug interactions.

Methods. A clinical case involving an 85-year-old woman is presented. Data were obtained from medical records and inpatient treatment. Medications were reviewed for potential interactions and their link to the observed neuropsychiatric symptoms.

Results. The patient had chronic anemia and a history of CeVD. After a fall, back pain developed. Tramadol was prescribed for persistent pain. She was also taking: amitriptyline, duloxetine, cabamazepine, bromazepam. After starting tramadol, she developed disorientation, drowsiness and incoherent speech. When tramadol is combined with amitriptyline or duloxetine, it may increase adverse drug reactions. On admission, ketoprofen was prescribed for pain control. During the treatment course, fluctuating neuropsychiatric symptoms appeared. At night, she talked to herself, was disoriented and had visual hallucinations. By day, she was oriented and appropriate, but did not recall the night episodes. Given the acute onset, symptomatic treatment with tiapride was initiated, as it is often well tolerated in geriatric patients. Night-time agitation decreased and the hallucinatory symptoms resolved. The patient was discharged home with continuation of the prescribed treatment.

Conclusions. This case shows geriatric delirium in a patient with polypharmacy, older age, anemia and a history of CeVD. A newly prescribed opioid likely triggered acute confusion, disorientation and visual hallucinations. A neuroleptic reduced night-time agitation and resolved hallucinations. Long-term prevention requires addressing precipitating factors and reviewing medications.

Treatment of Adult Attention-Deficit/Hyperactivity Disorder with Stimulants: A Case Report

Eva Matulevičiūtė[1], Algirdas Dambrava[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Vilnius university, Vilnius, Lithuania

Introduction. Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder typically diagnosed in childhood, yet symptoms often persist into adulthood and may significantly impair social, academic, and occupational functioning. Adult ADHD is often underrecognized due to differences in clinical presentation and frequent psychiatric comorbidities. This case report describes a clinical course of ADHD in adulthood.

Methods. A 44-year-old woman presented with lifelong difficulties in attention, organization, and emotional regulation without prominent childhood hyperactivity. These symptoms contributed to poor academic performance and university dropout. She had a history of low self-esteem, depression, and substance use (smoking, alcohol). In her late twenties, following her brother's suicide and a prolonged grief period, her depression worsened while she was in a psychologically abusive relationship. At 27, during psychiatric treatment in the United States, ADHD was suspected based on persistent attentional difficulties. Mixed amphetamine salts were initiated together with antidepressant therapy (escitalopram and bupropion), resulting in significant subjective improvement. After returning to Lithuania, treatment was switched to methylphenidate. Later, during increased occupational stress and perimenopause, cariprazine and occasional alprazolam were added to address mood instability and anxiety.

Results. Stimulant treatment improved attention, cognitive clarity, and daily functioning. The patient reported better concentration and improved ability to perform her duties as a teacher. Some ADHD symptoms persisted but were manageable with pharmacotherapy. Depression and mood instability improved after treatment adjustment.

Conclusions. This case illustrates the diagnostic complexity of adult ADHD and the importance of individualized pharmacological management, which may significantly improve functional outcomes and quality of life.

Beyond the Biological Diagnosis: The Psychological Impact of Infertility on Women's Well-Being – A Literature Review

Vaida Kolbutė[1], Aida Kuznecovaitė[1], Gabija Didžiokaitė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Department of Pathology and Forensic Medicine, Institute of Biomedical Sciences, Faculty of Medicine, Vilnius, Lithuania

Introduction. Infertility constitutes a global public health issue, affecting approximately 17.5% of the population. Researchers traditionally focus on the causes and fertility treatment, while associated psychological strain is often overlooked, despite being a primary reason for treatment discontinuation. This literature review analyzes how a diagnosis of infertility affects women's mental health.

Methods. A literature review of Pubmed, ScienceDirect and Google Scholar databases was conducted for articles published between 2015 and 2025. Search terms included "women infertility", "mental health", "anxiety", "depression", "stress", "sleep disorders", "PTSD".

Results. Recent research consistently shows that infertile women report significantly poorer psychological well-being in multiple domains compared to fertile controls. The prevalence of depressive symptoms ranges from 20% to 40%. Higher level of anxiety and depression is found among women with primary infertility, prior treatment failures, and lower socioeconomic status. Infertility is also linked to higher stress levels, arising from social stigma, continuous treatment, and financial strain. Around one-third of infertile women suffer from sleep disturbances correlated with psychological distress and hormone-related somatic symptoms. Infertility is classified as a potentially traumatic reproductive experience, as many infertile women are reported to exhibit high PTSD symptom scores.

Conclusions. Infertility represents a substantial psychological burden associated with depression, anxiety, chronic stress, sleep disorders, and PTSD symptoms. As these disturbances may impair quality of life and negatively impact treatment outcomes, integrating routine mental health screenings into standard infertility care is essential.

Analysis Of Alcohol Biomarkers in Self-Poisoning Suicide Attempts: A Seven-Year Retrospective Study

Maksim Čistov[1], Augustinas Žemaitis[2], Robertas Badaras[3]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[3] Clinic of Anaesthesiology and Intensive Care, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Alcohol abuse is well known to increase impulsivity, impair judgement and intensify negative effects, thereby heightening the risk of self-harm and suicide attempts, yet objective identification may be limited when direct ethanol testing is unavailable. This study aimed to examine indirect alcohol exposure biomarkers and their objectivity in ethanol self-poisonings.

Methods. This retrospective study (2017-2024) took place at the Republican Vilnius University Hospital. The analysis was conducted on 941 hospitalized adult patients with suicide attempts by self-poisoning. Intoxication was determined objectively via an alcohol test (serum ethanol >0.1 g/l) if available, or determined by physicians (behavioral changes, slurred speech, impaired coordination and other physical indicators) during admission. Indirect alcohol biomarkers (AST, ALT, GGT, MCV) were evaluated.

Results. Alcohol intoxication was observed in 407 cases. Median ethanol concentration was 0.18 g/l. Intoxication groups comparison (Mann-Whitney U) showed a significant difference between indirect biomarker levels ($p<0.05$). Ethanol concentration showed moderate correlation (Spearman's) with ALT, GGT, and MCV ($\rho=0.25$), but weak correlation with AST ($\rho=0.18$) (all $p<0.001$). ROC analysis demonstrated that GGT had the highest discriminatory ability (AUC=0.63, 95% CI 0.58-0.69), followed by MCV (AUC=0.62, 95% CI 0.58-0.69), while ALT, AST and the De Ritis ratio demonstrated poor discriminatory ability, despite a significant association with intoxication status. In multivariable logistic regression analysis ($R^2=0.098$), only GGT (OR=1.009, p=0.002) and MCV (OR=1.046, p=0.008) were independently associated with intoxication status, while AST and ALT were not significant.

Conclusions. Indirect markers of alcohol exposure demonstrate statistical association, but limited utility in detecting acute alcohol intoxication.

Symptom Overlap Between Autism Spectrum Disorder and Schizophrenia Spectrum Disorders: Differential Diagnostic Challenges in Adolescence

Rugilė Kriščiukaitytė[1], Goda Valantiejienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Psychiatry, Institute of Clinical Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Autism spectrum disorder (ASD) and schizophrenia spectrum disorders (SSD) have traditionally been conceptualized as distinct nosological entities; however, an increasing number of studies indicate a significant clinical and etiopathogenetic overlap between them. Difficulties in social communication, altered emotional expression, and behavioral disorganization may occur in both conditions, complicating differential diagnosis. Therefore, the emergence of psychotic symptoms during adolescence in individuals with ASD poses a substantial diagnostic challenge. The aim of this study is to present a clinical case and, based on current scientific literature, to discuss the diagnostic implications of symptom overlap between ASD and SSD.

Methods. A clinical case analysis of a 17-year-old female patient was conducted based on inpatient treatment documentation, psychiatric assessment, and follow-up observation. In addition, a review of recent literature examined the prevalence of comorbidity between ASD and psychotic disorders, their clinical characteristics, and diagnostic criteria.

Results. The patient, with a prior diagnosis of ASD, developed progressively emerging psychotic symptoms during adolescence, including hallucinations, delusion-like experiences, thought disorganization, and affective incongruity. Diagnostic assessment was complicated by partial overlap with previously identified impairments in social communication and behavior. Literature estimates the prevalence of psychotic disorders among individuals with ASD at approximately 3–8%, while autistic traits are reported in 20–30% of patients with SSD, indicating meaningful clinical overlap and possible shared neurodevelopmental mechanisms.

Conclusions. Clinical overlap between ASD and SSD may delay recognition of psychosis, particularly during adolescence. Careful evaluation of symptom dynamics and functional changes beyond a pre-existing ASD diagnosis is essential for timely intervention and improved long-term outcomes.

Complicated Multi-Stage Intervention for HIV-Related Spondylodiscitis and Spinal Candidal Abscess

Vytautas Radavičius[1], Gediminas Lukšys[2]

[1] Faculty of Medicine, University of Vilnius, Vilnius, Lithuania
[2] Centre for Applied Neuroscience, Translational Health Research Institute, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. HIV-related spinal infections often require multidisciplinary approach and surgery for instability, neurological decline, or failed conservative care.

Case report. A 50-year-old patient presented with debilitating symmetrical waist and leg pain, marked by mechanical instability and rapid worsening upon exertion. HIV-positive with a low viral load and a history of L4-L5 spondylodiscitis with candidal urosepsis two-years-prior, treated with IV fluconazole. Repeated MRI showed progressive vertebral destruction, oedema, fluid collection, and complete disc loss at L4-L5. Initial posterior fixation was performed, but the planned anterior corpectomy was aborted due to vascular involvement. After discharge with partial mobility, a revision surgery performed abscess evacuation and circumferential spondylodesis. Postoperative recovery was stable, with regained mobility and diminished pain. Eight days post-revision patient developed lumbar pain, antalgic gait and drain-site exudate. Recovery was complicated by a surgical site infection (MSSA/MRSE), requiring further revision and IV antibiotics.

Discussion. Spondylodiscitis encompasses vertebral osteomyelitis, spondylitis, and discitis as a single disease spectrum. Fungal spinal infections are non-caseating, acid-fast-negative, and primarily opportunistic in immunocompromised hosts. Surgical management in immunocompetent patients often involves a single-stage procedure with posterior stabilization, anterior debridement, and column reconstruction. For high-risk patients, two-stage operations yield better correction and lower implant failure risk by allowing infection control between stages.

Conclusions. Immunocompromised patients with suggestive symptoms require evaluation for spondylodiscitis, including atypical fungal pathogens such as Candida. When antifungal therapy fails, surgical intervention is often necessary.

Decoding the Mind: Algorithms in Brain-Computer Interfaces

Andrius Mašonis[1], Žilvinas Chomanskis[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Department of Neurosurgery, Republican Vilnius University Hospital, Vilnius, Lithuania

Introduction. With the rapid advancement of brain-computer interface (BCI) technology, parsing complex neural instructions for a real-world application remains a critical task. The focus of BCI development is shifting from surgical techniques and hardware towards decoding of raw data recordings to useful real time commands. This study reviews current software decoding algorithms.

Methods. A literature search was conducted using the following keywords: "brain-computer interface", "decoding algorithms", "machine learning" and "deep learning" in the databases PubMed and ScienceDirect. The articles from the last 10 years were read and reviewed.

Results. Recent literature shows that traditional preprocessing and decoding techniques rely heavily on linear algebra and strict mathematical rules to separate neural signals from artifacts, however because these methods use rigid assumption-based filtering they inadvertently risk stripping away task-relevant data. In contrast deep learning (DL) – using advanced convolutional and recurrent neural networks – is highly effective at translating noisy and complex brain signals into actionable commands. By operating on raw or minimally processed neural data DL algorithms learn to extract relevant data features without relying on manual intervention.

Conclusions. Deep learning provides a platform for future brain-computer interface development. By replacing rigid, manual filtering with flexible content aware algorithms, it preserves valuable neural data. This establishes DL as superior technique for decoding complex brain signals into real-world commands.

Straight through the Bone: Cerebellar Haemorrhage Following Cervical Spine Fixation

Karolina Karneckaitė[1], Šarūnas Bogušis[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Department of Neurosurgery, Republican Vilnius University Hospital, Vilnius, Lithuania

Introduction. Intracranial migration of cervical spine instrumentation through the occipital bone is extremely rare, particularly in patients without inflammatory or metabolic bone disease. However, this complication can lead to severe neurological outcomes or become life-threatening.

Methods. Informed consent was obtained from the patient. A literature review was conducted using PubMed and ClinicalKey databases, employing the keywords: cerebellar haemorrhage, cervical fixation, spine hardware migration.

Results. A 60-year-old male presented with a three-day history of headache, lethargy, dysarthria and ataxic wide-based gait with inability to stand unassisted. His medical history was significant for hypertension, lumbar hernia and C1-C3 fixation for a C2 fracture 3 years ago. Cranial CT showed a posterior fossa mass with haemorrhage and obstructive hydrocephalus, initially suggestive of a tumour. After emergent intraventricular drain placement, head MRI revealed a large heterogeneous infratentorial intra-axial lesion (5x4x3cm), consistent with cerebellar haemorrhage, and right occipital bone defect with a metallic rod penetrating intracranially, positioned adjacent to the haemorrhage. The patient underwent the revision of the C1-3 fixation system, including right rod shortening and refixation, and suboccipital craniotomy with cerebellar decompression. Histopathology excluded malignancy and confirmed hemorrhagic changes. Recovery was uncomplicated, routine follow-up revealed significant neurological improvement and radiological regression.

Conclusions. Delayed cerebellar haemorrhage following migration of cervical spine fixation rods and penetration of the occipital bone represents a unique complication in patients without predisposing metabolic bone disease, and, to our knowledge, has not been previously reported. Awareness of this complication is crucial for long-term follow-up and early intervention.

Epilepsy Surgery Outcomes in Drug-Resistant Epilepsy: A 15-Year Single-Center Study

Saulius Ročka[1], Gertrūda Kaubrytė[1], Saulius Ročka[2], Rūta Mameniškienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Neurology and Neurosurgery, Institute of Clinical Medicine, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Despite the availability of multiple antiseizure medications, approximately 1/3 of people with epilepsy develop drug-resistant disease. In such cases, resective and palliative interventions are essential treatment options. The study aimed to evaluate seizure outcomes following epilepsy surgery.

Methods. A retrospective, uncontrolled observational study analyzed all epilepsy surgeries performed in Vilnius, Lithuania, between 2010 and 2025. Postoperative outcomes were assessed using the Engel classification. Data were analysed using the Wilcoxon signed-rank test, and responder rate was defined as a ≥50% reduction in seizure frequency.

Results. In total, 164 patients underwent 173 surgeries, including 54 resective and 119 palliative procedures. Palliative surgeries comprised 16 corpus callosotomies and 103 vagus nerve stimulator (VNS) implantations. Median follow-up was 35 months (IQR 12-60). Resective surgeries involved the temporal lobe (TL) in 86.54%, the frontal lobe (FL) in 11.54%, and the occipital lobe in 1.92% cases. After temporal lobe surgery, 73.33% achieved Engel class I, with a responder rate of 93.3%; median seizure frequency decreased from 4 to 0 ($p<0.001$). Frontal lobe surgery resulted in Engel class I outcomes in 33.3%, with 83.3% responder rate and a median seizure reduction from 4 to 1 ($p<0.001$). Following palliative surgery, responder rates were 46% for VNS implantation and 81.3% for callosotomy, both with significant seizure reduction ($p<0.001$).

Conclusions. All surgical approaches were associated with significant seizure reduction; however, temporal lobe surgery achieved the most favourable seizure outcome. Quantitative long-term seizure assessment provides valuable additional insight into surgical effectiveness.

Fulminant Bilateral Anterior Cerebral Artery Vasospasm Following Resection of a Hypervascular Bifrontal Tumor: A Clinical Case

Rugilė Kriščiukaitytė[1], Indrė Lapinskienė[2]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Clinic of Anaesthesiology and Intensive Care, Institute of Clinical Medicine, Vilnius University, Vilnius, Lithuania

Introduction. Postoperative cerebral vasospasm is a rare but potentially severe complication following intracranial tumor resection. It is most frequently associated with hypervascular tumors and perioperative subarachnoid hemorrhage. Compared to aneurysmal vasospasm, it may develop earlier and follow a less predictable course. Due to its low incidence and nonspecific clinical signs, early recognition can be challenging. This report describes a case of fulminant bilateral anterior cerebral artery (ACA) vasospasm after resection of a hypervascular bifrontal tumor, highlighting its rapid progression and outcome.

Methods. A retrospective review of inpatient medical records was conducted. Clinical data, neurological findings, and multimodal neuroimaging, including CT, CTA, MRI, and digital subtraction angiography, were analyzed. The patient's clinical course, therapeutic interventions, and outcomes were evaluated to assess the development and progression of postoperative vasospasm.

Results. A 32-year-old male was diagnosed with a 58 mm highly vascular bifrontal intra-axial tumor involving ACA branches and associated with arteriovenous shunting. Following bifrontal craniotomy and subtotal tumor resection, early imaging showed minor subarachnoid and subdural hemorrhage without significant mass effect. The patient was initially neurologically stable. On postoperative day two, deterioration occurred with decreased consciousness and right-sided hemiparesis. Angiography revealed severe stenosis (75%) of the left ACA with distal vasospasm. Intra-arterial nimodipine led to only temporary improvement. Vasospasm progressed bilaterally, resulting in extensive ischemia in the frontomedial and supracallosal regions, followed by deep coma and loss of brainstem function. Brain death was confirmed angiographically on postoperative day five.

Conclusions. Postoperative vasospasm after hypervascular tumor resection may be early, aggressive, and resistant to treatment, even in the presence of minimal subarachnoid hemorrhage. Despite prompt recognition and intervention, outcomes may be catastrophic. These findings emphasize the need for vigilant monitoring and suggest that current therapeutic strategies may be insufficient to prevent severe neurological compromise.

Adjunctive Bright Light Therapy to Enhace CPAP Adherence in Patients with Comorbid Major Depressive Disorder and Obstructive Sleep Apnoea Syndrome: Study Protocol for a Randomised Sham-Controlled Trial

Houda El Azzaoui[1-3], Sébastien Catoire[1], Harold Lecharpentier[1], Juliette Felician[1], Olivier Grasset[3], Claude Gronfier[4], William Vallet[1,2], Thierry D'Amato[1,2], Jérôme Brunelin[1,2]

[1] Le Vinatier Hospital Center, Bron, France
[2] Université Claude Bernard Lyon 1, CNRS, INSERM, Centre de recherche en Neurosciences de Lyon CRNL U1028 UMR5292, PSYR2, F-69500, Bron, France
[3] Linde Homecare, 103 Bd Edouard Herriot, CS40249 Viriat, F-01006 Bourg en Bresse Cedex, Bourg en Bresse, France
[4] Université Claude Bernard Lyon 1, CNRS, INSERM, Centre de recherche en Neurosciences de Lyon CRNL U1028 UMR5292, WAKING, F-69500, Bron, France"

Introduction. Obstructive sleep apnoea syndrome (OSAS) co-occurs with major depressive disorder (MDD) in approximately 50% of cases, and this comorbidity is associated with greater severity of depressive symptoms, sleep disturbances and poorer clinical outcomes. Although continuous positive airway pressure (CPAP) therapy is effective in treating OSAS and alleviating MDD symptoms, adherence to treatment during the first few weeks remains low. Bright light therapy has been shown to rapidly improve sleep, wakefulness, cognitive function and mood in patients with MDD. Given these complementary mechanisms, we propose that combining these two treatments may enhance patient adherence.

Methods. In a single-centre, double-blind, sham-controlled study with two parallel arms, 130 patients with both MDD and OSAS requiring CPAP therapy will be randomly assigned to receive either 14 sessions of 30min active bright light therapy (1200 Lux) or 14 sessions of 30min sham bright light therapy (33 Lux) during the first 2 weeks, following CPAP initiation at home. The primary outcome will be adherence to CPAP (in hours per 24 hours during the 14 days of investigation). Secondary clinical outcomes will include changes in depressive and anxiety symptoms and objective sleep parameters

Results. The expected outcomes are that bright light will improve CPAP adherence and reduce depressive symptoms, leading to better sleep and alertness. This treatment could also prevent psychiatric relapses and improve overall long-term health.

Conclusions. This study could pave the way for a new combined therapeutic approach to treat patients with sleep and mood disorders, integrating CPAP and bright light therapy.

The Effect of Tetrahydrocannabivarin (THCV) on Voluntarily Alcohol Drinking In Male Rats

Ceyda Türkcan[1], Ieva Pocevičiūtė[1], Valentina Vengelienė[1]

[1] Department of Neurobiology and Biophysics, Institute of Biosciences, Life Sciences Center, Vilnius University, Vilnius, Lithuania

Introduction. Alcohol use disorder (AUD) is a major public health challenge, and current pharmacological treatments remain limited. Cannabinoid CB1 receptor antagonists reduce alcohol consumption but are associated with adverse psychiatric effects. Δ^9-Tetrahydrocannabivarin (THCV), a phytocannabinoid acting as a neutral CB1 antagonist, may represent a better-tolerated alternative. This study examined the effects of repeated THCV administration on voluntary alcohol intake, drinking microstructure, and locomotor activity in a long-term drinking rat model.

Methods. Male Wistar rats (n = 25) received voluntary two-bottle choice access to alcohol and water for 8–10 months. Following baseline assessment, animals were treated with daily intraperitoneal injections of vehicle or THCV (5 or 20 mg/kg; n = 8–9/group) for three consecutive days. Alcohol and water intake were continuously monitored. Drinking microstructure parameters, including bout number, bout size, bout duration, and licking rate, were quantified using lickometer recordings and a custom Python analysis script. Locomotor activity was assessed to control for nonspecific motor effects.

Results. Repeated THCV administration significantly reduced voluntary alcohol intake without affecting water consumption. THCV decreased alcohol bout size and licking rate, indicating reduced hedonic value ("liking"), and reduced the number of alcohol bouts, suggesting diminished motivational drive ("wanting"). Locomotor activity was unchanged, indicating that reduced drinking was not due to sedation.

Conclusions. THCV selectively reduces alcohol intake and alters both hedonic and motivational components of alcohol consumption in long-term drinking rats without affecting locomotion. Neutral CB1 antagonism by THCV may represent a promising and better-tolerated pharmacological strategy for AUD treatment.

Identification of Potential Glioblastoma Biomarkers in U87, A172, and HS683 Cell Line Spheroid Models

Rima Miknaitė[1], Rytė Rynkevičienė[2], Eglė Strainienė[2,3], Linas Kunigėnas[2], Kęstutis Sužiedėlis[1,2]

[1] Life Sciences Center, Vilnius University, Vilnius, Lithuania
[2] Laboratory of Molecular Oncology, National Cancer Institute, Vilnius, Lithuania
[3] Department of Chemistry and Bioengineering, VILNIUS TECH, Vilnius, Lithuania

Introduction. Glioblastoma is classified by the World Health Organization (WHO) as a grade IV malignant glioma. This highly aggressive tumor carries a poor prognosis, with a median survival of approximately 15 months after diagnosis. Standard treatment includes surgery, followed by radiation therapy and chemotherapy. Without validated biomarkers, it's harder to personalize treatment, identify high-risk patients, and select the right treatment strategy.

Methods. A comprehensive genome-wide study of the U87 cell line identified four principal pathway categories associated with glioblastoma development: metabolism, cell–cell adhesion, oncogenic signaling, and immune-related processes. Gene expression within these functional categories was subsequently evaluated by RT-PCR in three-dimensional spheroid models derived from the U87, A172, and HS683 glioblastoma cell lines.

Results. Gene expression analysis revealed both shared and cell line–specific differences among the spheroids. U87 spheroids showed markedly increased expression of IL1β, PTGS2, and HMGCS1, indicating activated metabolic pathways and a strong stress- and inflammation-like response. A172 spheroids exhibited moderately elevated HMGCS1 and PTGS2, increased COL1A2, and reduced IL1β expression compared with U87, suggesting the absence of a pronounced inflammatory phenotype. HS683 spheroids displayed strongly increased PTGS2 and IL1β levels, partially resembling U87, but also showed elevated CCL20 and FGF7, indicating additional cell line–specific features.

Conclusions. The U87, A172, and HS683 cell lines exhibit different transcriptomic profiles when cultured as 3D spheroids. These variations highlight their utility for modeling glioblastoma heterogeneity and may inform the development of cell-line–tailored, personalized therapeutic approaches.

Effects of Natural Compounds on Functional Recovery Following Spinal Cord Injury in Zebrafish

Viktorija Povilionytė[1], Mindaugas Mitkus[1], Pramuk Keerthisinghe[2], Alexander Tups[2]

[1] Department of Neurobiology and Biophysics, Life Sciences Center, Vilnius University, Vilnius, Lithuania
[2] Department of Physiology, University of Otago, Dunedin, Otago, New Zealand

Introduction. Spinal cord injury (SCI) induces inflammatory and tissue repair responses that critically influence regenerative outcomes. While zebrafish possess a remarkable capacity for spinal cord regeneration, identifying interventions that enhance functional recovery remains an important objective. Natural bioactive compounds, including flavonoids and omega-3 polyunsaturated fatty acids have been reported to modulate inflammatory and tissue repair pathways. This study investigated whether selected natural compounds (designated as A and B) improve recovery following SCI in zebrafish.

Methods. Larval zebrafish underwent a standardized spinal cord transection. Lesion integrity was confirmed using a neuronal fluorescent reporter line to verify disruption at the injury site. Following injury, larvae were treated with compound A, compound B, or a combination of both. Regeneration was initially assessed by imaging of the lesion site, and functional recovery was evaluated using a locomotor assay measuring total distance moved during 40-minute recording period with controlled tapping stimuli. Data was analyzed using repeated measures two-way ANOVA.

Results. Treated groups exhibited increased tissue regrowth at the lesion site compared with untreated controls. Locomotor analysis demonstrated an improvement in distance moved in injured fish receiving compound treatment, with the combination group showing the strongest effect. No significant changes were observed in uninjured fish, indicating that baseline locomotor activity was not altered.

Conclusions. These findings suggest that treatment with natural compounds A and B, particularly in combination, shows positive trend on functional recovery following spinal cord injury in zebrafish. The results support further investigation into the potential role of natural bioactive compounds in modulating regenerative process and promoting neural repair.

Mechanistic Insights into rTMS Response: The Role of miR-16-5p And TNFα in Neural and Metabolic Regulation

Deimilė Asipavičiūtė[1], Aistė Zentelytė[1], Vladas Valiulis[3], Rūta Navakauskienė[1], Giedrė Valiulienė[1]

[1] Institute of Biochemistry, Life Sciences Center, Vilnius University, Vilnius, Lithuania
[2] Republican Vilnius Psychiatric Hospital, Vilnius, Lithuania

Introduction. In our previous studies, we found that lower serum levels of the inflammatory cytokine TNFα in patients with treatment-resistant depression (TRD) were associated with better outcomes following repetitive transcranial magnetic stimulation (rTMS). We also identified miR-16-5p as a potential biomarker of rTMS response, as higher circulating levels predicted improved therapeutic effects. Notably, miR-16-5p and TNFα appear to regulate each other, suggesting that their interaction may contribute to the mechanisms underlying rTMS efficacy.

Methods. To investigate this interaction, SH-SY5Y neuroblastoma cells were differentiated into a mature neuronal phenotype using BrainPhys™ medium supplemented with SM1, NGF (50 ng/ml), retinoic acid (10 μM), and antibiotics. After 7 days, cells were transfected with a miR-16-5p mimic or inhibitor and/or treated with TNFα (10 ng/ml). Transfection efficiency and downstream effects were confirmed by RT-qPCR and western blot, demonstrating modulation of FOXO1 and IRAK1 expression. Cells were then exposed to intermittent (iTBS) or continuous (cTBS) theta burst stimulation.

Results. In this 2D model, cTBS—but not iTBS—increased cleaved caspase-1 levels, suggesting potential cellular stress or damage. In contrast, under miR-16-5p modulation, iTBS enhanced synaptobrevin-2 protein levels, indicating possible synaptic effects. Metabolic analyses using Seahorse ATP Rate, Mito Stress, and Glycolysis Stress assays revealed distinct metabolic responses to iTBS and cTBS depending on TNFα and miR-16-5p activity.

Conclusions. Overall, our findings suggest that miR-16-5p–TNFα interactions may shape neural and metabolic responses to theta burst stimulation, warranting further investigation in more physiologically relevant models.

Multiscale Regulation of Synaptic Plasticity by Coding and Noncoding RNAs during Development

Nemira Žilinskaitė-Tranas[1,2*], Daina Bujanauskienė[2,3*], Monika Kušeliauskytė[2,3], Jaroslav Denkovskij[2,3], Eiva Bernotienė[2,3], Vladimir Benes[2,3], Urtė Neniškytė[2,3]

[1] Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] VU LSC-EMBL Partnership for Genome Editing Technologies, Life Sciences Center, Vilnius University, Vilnius, Lithuania
[3] Institute of Biosciences, Life Sciences Center, Vilnius University, Vilnius, Lithuania
* – equal contributors

Introduction. Critical periods of postnatal development are characterised by large-scale synaptic remodelling that refines neural circuits and establishes mature brain function. While transcriptional programs underlying plasticity have been extensively investigated, the contribution of local synaptic RNA regulation remains poorly defined.

Methods. Here, we combine structural imaging with synaptosome-specific mRNA and miRNA profiling to dissect RNA-level mechanisms of synaptic refinement in the mouse visual cortex across the critical period (P21–P35).

Results. We identify coordinated presynaptic bouton remodelling and transient complement activation at peak plasticity (P28), accompanied by stage-specific synaptic gene expression programs linked to synapse organisation, vesicle trafficking, and translational control. Parallel profiling of synaptosomal miRNAs uncovers temporally restricted clusters aligned with plasticity and consolidation phases. Notably, nearly 70% of synaptic transcripts are predicted targets of detected miRNAs, and network analysis highlights hub genes under convergent post-transcriptional regulation.

Conclusions. Together, these findings reveal a multiscale RNA regulatory architecture in which coding and noncoding RNAs cooperatively shape synaptic plasticity and circuit maturation during cortical development.

Tracking Neural Variability and Inhibitory Control Across the Menstrual Cycle

Gabrielė Kukytė[1], Monika Palaimaitė[1], Ingrida Zelionkaitė[1], Rimantė Gaižauskaitė[1], Lauryna Filatovaitė-Vyčienė[1], Ramunė Grikšienė[1]

[1] Department of Neurobiology and Biophysics, Institute of Biosciences, Life Science Center, Vilnius University, Vilnius, Lithuania

Introduction. A growing body of neuroimaging research indicates that fluctuations in women's sex hormones are linked to changes in neural functioning across the menstrual cycle. Hormonal shifts may affect excitation–inhibition balance, affective processing, and cognitive performance. One proposed mechanism is allopregnanolone, a progesterone metabolite that modulates GABAergic transmission. By enhancing inhibitory neurotransmission and reducing neural excitability, it may contribute to variability in cognitive control. However, there remains a lack of densely sampled longitudinal data to reveal the dynamics of electrophysiological parameters across the menstrual cycle, mainly due to limited temporal sampling and insufficient hormonal confirmation.

Methods. To address this gap, the present study employed an intensive within-person longitudinal case design to examine menstrual phase–specific changes in neural activity and inhibitory control in a healthy adult woman. Data were collected at six time points: the early and late follicular phases, ovulation, and the early, mid, and late luteal phases. Salivary samples were collected at each session to quantify progesterone and improve phase identification. Resting-state EEG (eyes open and closed) was recorded to assess alpha-band activity and the aperiodic slope as markers of global neural activity and excitation–inhibition balance. Inhibitory control and attention were measured using a Go/No-Go task assessing response inhibition and reaction times. The Barratt Impulsiveness Scale (BIS-11) and the Well-Being Questionnaire (WBQ) were administered during early follicular and late luteal phases.

Conclusions. By combining dense temporal sampling, hormonal confirmation, and multimodal neurobehavioral assessment, this study provides a detailed within-subject characterization of menstrual cycle–related variability in neural and cognitive functioning.

Behavioral and EEG Assessment of Emotion Regulation across the Menstrual Cycle

Monika Palaimaitė[1], Gabrielė Kukytė[1], Ingrida Zelionkaitė[1], Rimantė Gaižauskaitė[1], Lauryna Filatovaitė-Vyčienė[1], Ramunė Grikšienė[1]

[1] Department of Neurobiology and Biophysics, Institute of Biosciences, Life Science Center, Vilnius University, Vilnius, Lithuania

Introduction. Emotion regulation capacity in healthy women has been suggested to vary across the menstrual cycle. This pilot study evaluated the feasibility of an experimental paradigm assessing emotional regulation across multiple menstrual cycle phases, using repeated behavioral and neural measures.

Methods. Nine healthy women were recruited and four completed all sessions at the time of abstract preparation (mean age 25.5 ± 2.5 years). Participants completed experimental sessions across early follicular (EF), ovulation (O), and late luteal (LL) phases. During each they performed an emotion regulation (ER) task involving cognitive reappraisal, provided subjective valence and arousal ratings, and completed the Difficulties in Emotion Regulation Scale (DERS) and Emotion Regulation Questionnaire (ERQ). Electroencephalograms (EEG) were recorded to derive event-related potential (ERPs).

Results. Regulation success was quantified as increased valence and decreased arousal ratings after regulation. Mean arousal regulation effects were 21.0% (EF), 18.5% (O), and 17.8% (LL), valence increases were largest during O (46.2%) and lowest during LL (14.1%) phase. These values suggest the smallest regulation effect during LL phase, a pattern consistent with existing menstrual cycle research. ERQ scores showed greater cognitive reappraisal use than suppression across EF and LL phases (reappraisal 5.13 ± 0.44 (EF), 4.96 ± 0.91 (LL); suppression 4.75 ± 0.89 (EF), 4.31 ± 0.66 (LL)). Self-reported emotion regulation difficulties (DERS) indicated generally low-to-moderate difficulties (mean DERS total scores 110.3 (EF); 113.0 (LL)).

Conclusions. Although based on a small sample, descriptively lower regulation success in the LL phase is consistent with literature supporting the feasibility of the paradigm for future biomedical research.

Synbiotic Supplementation Attenuates High-Fat Diet-Induced Metabolic and Behavioral Alteration in Female Mice Modulating Microbiota-Gut-Liver-Brain-Axis

Ashwinipriyadarshini Megur[1], Dominyka Kobeckytė[1], Bablu Kumar[2], Eglė Lastauskienė[3] Aurelijus Burokas[1]

[1] Department of Biological Models, Institute of Biochemistry, Life Sciences Center, Vilnius University, Vilnius, Lithuania
[2] Department of Biosciences, Biotechnology and Environment, University of Bari A. Moro, Bari, Italy
[3] Department of Microbiology and Biotechnology, Institute of Biosciences, Life Sciences Center, Vilnius University, Vilnius, Lithuania.

Introduction. High-fat diet (HFD)–induced metabolic syndrome is closely associated with neurobehavioral disturbances through disruption of the microbiota–gut–liver–brain axis. Females subjects remain underrepresented in preclinical studies addressing these interactions. This study evaluated the long-term effects of a novel probiotic, Lacticaseibacillus paracasei 11W, and a synbiotic combination with galacto-oligosaccharides (GOS) on metabolic, behavioral, inflammatory, and gut microbiota alterations induced by chronic HFD exposure in female C57BL/6J mice.

Methods. 35 Female mice were fed a control diet or HFD for 25 weeks and received oral administration of L. paracasei 11W alone or combined with 5% GOS. Metabolic parameters, glucose tolerance, behavioral outcomes (anxiety-, depression-, and cognition-related tests), colonic and hippocampal inflammatory markers, liver and colon histology, and cecal microbiota composition (16S rRNA gene sequencing) were assessed.

Results. Chronic HFD induced obesity, impaired glucose tolerance, anxiety- and depressive-like behaviors, cognitive deficits, colonic and hippocampal inflammation, hepatic steatosis, and gut microbiota dysbiosis. Probiotic treatment partially improved metabolic and behavioral alterations. The synbiotic intervention significantly reduced fasting glucose, improved glucose tolerance, attenuated anxiety- and depressive-like behaviors, restored recognition memory, reduced pro-inflammatory cytokine expression in colon and hippocampus, improved liver and colon histology, and normalized gut microbiota composition, including increased abundance of Bifidobacterium, Dubosiella, and Lachnospiraceae.

Conclusions. Long-term synbiotic supplementation mitigates HFD-induced metabolic, neurobehavioral, inflammatory, and microbiota disturbances in female mice, supporting its potential as a therapeutic strategy targeting the microbiota–gut–liver–brain axis.

Dietary Prebiotic Intervention Modulates High-Fat Dietassociated Microglial Alterations in the Aged Mice

Urtė Minkevičiūtė[1], Arnas Kunevičius[1], Akshay Kumar Vijaya[1], Aurelijus Burokas[1]

[1] Department of Biological Models, Institute of Biochemistry, Life Sciences Center, Vilnius University, Vilnius, Lithuania

Introduction. Consumption of a chronic high-fat diet (HFD) and ageing are both associated with metabolic dysfunction and increased neuroinflammation. It contributes to the loss of gut microbiota diversity and the development of neurodegenerative diseases. Microglia, the resident immune cells of the central nervous system, are highly sensitive to metabolic and inflammatory signals, and their morphology is closely linked to their functional state. These metabolic and inflammatory molecular signals can be derived from the gut microbiota and influence microglial activation and disease progression. Gut microbiota-focused interventions have the potential to reduce chronic inflammation and have a protective effect on microglia. However, the impact of such interventions in the aged brain remains poorly understood, especially regarding their ability to reverse metabolically primed microglia phenotypes.

Methods. In this study, we examined how prebiotic supplementation with galactooligosaccharides and fructooligosaccharides (GOS+FOS) modulates microglial morphology in aged mice exposed to long-term HFD. The C57BL/6J mice were assigned to four dietary conditions for 18 months: control diet (CD), CD - GOS+FOS, HFD, and HFD - GOS+FOS. We utilised immunohistochemical staining to quantify three-dimensional branching architecture and twodimensional shape descriptors.

Results. Compared with CD, microglia in HFD-fed conditions exhibited quantifiable morphological change consistent with a metabolically primed microglial phenotype: increased total branch number and length, greater slab voxel count, triplejunction frequency, enlarged projected cell area and perimeter. HFD supplementation with GOS+FOS reduced several of these HFD-induced structural alterations: average branch length decreased towards CD levels, projected soma area and perimeter were significantly smaller than in HFD, indicating partial structural recovery. Importantly, in the CD - GOS+FOS group, microglial morphology was similar to that in the CD group, indicating that the effects of GOS+FOS are specific to reducing HFD-associated microglial alterations rather than modifying baseline microglial structure.

Conclusions. Collectively, these findings suggest that prebiotic supplementation with GOS+FOS can modulate microglial structural remodelling in the aged brain under metabolic challenge, highlighting a protective role for gut microbiotatargeted interventions in diet-induced neuroinflammatory conditions.