Ref: Ro J Neurol. 2024;23(Suppl)
DOI: 10.37897/RJN.2024.S.2

The 22nd National Congress of the Romanian Society of Neurology

POSTERS – ABSTRACTS

(in numerical order)

P001. Subtypes of ischemic stroke in young adults

A.-S. LUPSOIU[1], C. V. ALBU[2], E.-T. TRASCA[1], C. A. SILOSI[1], C. B. ALBU[3], E. A. ALBU[2], D. RADULESCU[1]

[1]"Dr. Stefan Odobleja", Military Emergency Hospital Craiova, Craiova, Romania
[2]Clinic of Neurology, Clinical Hospital of Neuropsychiatry, Craiova, Romania
[3]Emergency County Hospital, Craiova, Romania

Objective. The aim of the study was to evaluate the etiology and subtypes of ischemic stroke (IS) in young (≤55 years) patients, including the occurrence and type of thrombophilia.

Material and methods. We studied 72 patients admitted to the Department of Neurology of Military Emergency Hospital "Dr. Stefan Odobleja", Craiova, between 2020–2023, young adults (≤55 years) with acute ischemic stroke.

Inclusion criteria. Head MRI or CT, electrocardiography, transesophageal echocardiography, and laboratory tests, including coagulation examination, were performed in all patients. Stroke subtype was assessed using the TOAST criteria. Vascular risk factor occurrence was compared in IS patients and in a control group (CG) of 72 age-matched subjects without a history of cerebrovascular events. The results were analyzed by Student's t-test and Chi-Square test.

Results. The following IS types were diagnosed: thromboembolic in 22 (29.8%), cardioembolic in 18 (25%), other determined cause in 25 (35%), undetermined in 7 (10.2%) patients. The following occurrence of risk factors was found in young IS patients versus CG subjects: thrombophilia in 25 (35%) versus 0 (0%) ($p<0.0001$), arterial hypertension in 34 (46.7%) versus 8 (11.1%) ($p<0.001$), hyperlipoproteinemia in 30 (41.7%) versus 15 (20.83%) ($p<0.0001$), diabetes mellitus in 11 (15.27%) versus 3 (4.16%) ($p<0.0001$). Primary antiphospholipid syndrome (APS) was found in 8 (32%), protein C and S deficiency in 6 (24%), antithrombin III deficiency in 4 (16%), factor V Leiden mutation in 3 (12%), and non-specific coagulation abnormalities in 4 (16%) young IS patients with thrombophilia.

Conclusion. Ischemic stroke was associated with a higher occurrence of thrombophilia and arterial hypertension when compared to the CG subjects in our young patients.

Keywords: ischemic stroke, young adults, vascular risk factors, thrombophilia, arterial hypertension

P002. Multiple sclerosis – Pregnancy and its role in female fertility. A multiple sclerosis center analysis

M. M. MARTOIU, S. PETRESCU, C. PROFIRESCU, C. A. PANEA

Neurology Department, Elias Emergency University Hospital, Bucharest, Romania
"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Multiple sclerosis (MS) is a neurological disease that typically affects young women of reproductive age. Fertility and the course of pregnancy are not affected by MS; however, for many years, women with MS have been discouraged from getting pregnant, due to fears of complications for the fetus or mother caused by the disease or ongoing medication.

Aim. We present an analysis of a group of 100 female patients diagnosed with MS.

Methods and results. 37 female patients (37%) are nulliparous, 42 (42%) are primiparous, and 21 (21%) are multiparous. The disease onset occurred during reproductive age in most cases (77% of patients were under 40 at onset). 71 female patients (71%) had a pregnancy, and 65 of them (65%) gave birth. 63 female patients had natural conception, while 2 patients conceived through in vitro fertilization. There were a total of 168 pregnancies, with 86 childbirths (51%) and 81 abortions (49%). 63 were natural childbirths (73%), and 23 were cesarean births (27%). There were 55 abortions on demand (67%) and 27 spontaneous

abortions (33%). Disease-modifying therapies were used by 5 patients (7%) during pregnancy and by 3 patients (4.2%) during breastfeeding.

Conclusion. The present study confirms that fertility does not seem to be impaired in women with MS. Voluntary infertility (abortions on demand) shows a high rate compared to the general population. MS significantly impacts family planning decisions; therefore, all healthcare providers who follow up with multiple sclerosis patients must discuss fertility, pregnancy, and breastfeeding.

Keywords: MS, pregnancy, fertility

P003. Evaluation of early aggressive monitoring in intracerebral hemorrhage

A. NEGRILA[1], R. RADU[1,2], E. TERECOASA[1,2], R. BADEA[1,2], B. CASARU[1], C. TIU[1,2]

[1]Neurology Department, Emergency University Hospital, Bucharest, Romania
[2]Department of Clinical Neurosciences, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Early aggressive medical treatment has been proven to improve outcomes in patients with intracerebral hemorrhage (ICH).

Methods. We prospectively collected data on all patients admitted to the Neurology Department of a Stroke Centre in Bucharest, Romania, with the diagnosis of ICH between January 1, 2023, December 31, 2023. We implemented a local intracerebral hemorrhage monitoring protocol targeting blood pressure management and anticoagulation reversal, in accordance with current guidelines, and evaluated protocol adherence and patients' outcomes.

Results. The study included 131 patients with ICH. The median hemorrhage volume was 14.5 ml (IQR 3.57–35.75). Hypertensive bleeds were the most common cause of ICH in our cohort (54.62%), while in 21.54% of patients, no apparent cause was determined. Early blood pressure control (under 2 hours) was obtained in only 39.39% of cases. Overall mortality in the cohort remained high (50.39%). Patients with early blood pressure control tended to have more favorable outcomes at discharge and lower mortality, but the results did not reach statistical significance (23.08% vs 12.33%, p=0.11; 17.05% vs 33.33%, p=0.17).

Conclusions. This prospective study underscores the challenging nature of treatment in intracerebral hemorrhage, highlighting the need for improved adherence to protocols.

P004. Working memory impairment in multiple sclerosis is associated with the duration of the disease and the quality of life

Simona PETRESCU, Maria Melania MARTOIU, Cristina PANEA

Neurology Department, Elias Emergency University Hospital, Bucharest, Romania
"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

The BICAMS tests (Brief Cognitive Assessment for Multiple Sclerosis) evaluate different cognitive domains affected by the disease, especially the visual and verbal working memory components.

Methods. Thirty-two patients (17 women and 15 men), diagnosed with multiple sclerosis (MS), most with relapsing-remitting MS (85%), completed the quality-of-life questionnaire (MSIS – multiple sclerosis impact scale) and the fatigue questionnaire (MFIS – modified fatigue impact scale). The average age of the group was 38 years, with an average disease duration of 8.3 years. The patients were clinically assessed using the expanded disability status scale (EDSS), as well as cognitive scores: BICAMS and MoCA (Montreal cognitive assessment).

Results. The components of BICAMS: SDMT (symbol digit modalities test), CVLT-II (California verbal learning test-II), and BVMT-R (brief visuospatial memory test-revised)—correlated significantly with the MoCA score, which evaluates global cognitive function ($p = 0.0004$, $p = 0.0000001$, $p = 0.00002$).

The impact of the disease and the level of fatigue reported by patients, measured using the MSIS and MFIS scores, correlated significantly ($p < 0.005$) with the cognitive tests that assess memory impairment: BVMT-R and CVLT-II.

Disease duration also showed a statistically significant correlation with the MoCA score ($p = 0.001$) and the CVLT-II test ($p = 0.003$).

Conclusions. Cognitive impairment is observed in certain phenotypes of multiple sclerosis, with higher frequency in progressive forms. Working memory is one of the most affected cognitive domains and is associated with a decrease in quality of life in patients with MS.

Keywords: working memory impairment, BICAMS, MoCA, MFIS, MSIS

P005. Self-efficacy and quality of life in Romanian stroke patients – A pilot study

Ioan-Alexandru CHIRAP-MITULSCHI, Andreea CHIRAP-MITULSCHI, Doru BALTAG, Sabina ANTONIU

[1]Department of Medicine II/Nursing-Palliative Care, Faculty of Medicine, "Gr. T. Popa" University of Medicine and Pharmacy, Iasi, Romania
[2]Neurology Clinic, Clinical Rehabilitation Hospital, Iasi, Romania
[3]Department of Cardiology, „Sf. Spiridon" County Clinical Emergency Hospital, Iasi, Romania
[4]Department of Medicine II/Nursing-Palliative Care, Faculty of Medicine, "Gr. T. Popa" University of Medicine and Pharmacy, Iasi, Romania

Stroke, a leading cause of long-term disability worldwide, profoundly impacts the quality of life (QoL) of affected individuals. Self-efficacy, a key psychological construct, refers to an individual's belief in their ability to accomplish specific tasks and goals despite challenges. Understanding the role of self-efficacy in the context of stroke rehabilitation and its influence on QoL is vital for developing effective interventions.

We conducted a prospective cohort clinical study for psychometric validation of the Stroke Self-Efficacy Questionnaire (SSEQ) in the Romanian language, to better understand the role of self-efficacy in stroke rehabilitation and its impact on QoL. Patients with stroke undergoing rehabilitation, hospitalized between June 2023 and August 2023 in the neurology department of the clinical rehabilitation hospital of Iași, Romania, were enrolled after signing informed consent and meeting eligibility criteria. The study recorded data on age, gender, comorbidities (hypertension, diabetes, atrial fibrillation), stroke type, stroke duration, nutritional status, QoL, stroke severity scores, functional status, and inflammation status for 50 patients.

The Stroke Self-Efficacy Questionnaire measures stroke survivors' confidence in performing activities essential for their recovery and daily functioning. Validating this instrument in Romanian enables a comprehensive evaluation of self-efficacy beliefs and QoL perceptions among Romanian-speaking stroke patients. The study also presents the outcomes of the validation process, with a focus on the correlations observed between various variables and corresponding scores tracked during the study.

The validation of the stroke self-efficacy questionnaire in Romanian provides a valuable tool for assessing the role of self-efficacy in stroke rehabilitation and its relationship to QoL, contributing to the development of more effective interventions.

Keywords: stroke, self-efficacy, quality of life, rehabilitation, validation

P006. Worn out from wearing out – A comprehensive study of fatigue in multiple sclerosis

O.V. PANAITESCU[1,2], A. PREDA[1,2], O.E. BRISCAN[1,2], A.M. FLOREA[1,2], D.G. LUCA[1], E. I. DAVIDESCU[1,2], B.O. POPESCU[1,2,3]

[1]Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Laboratory of Cell Biology, Neurosciences and Experimental Myology, "Victor Babes" National Institute of Pathology, Bucharest, Romania

Introduction. Fatigue is a prevalent symptom across today's society, with multiple etiological factors and a multifaceted nature—including motor, cognitive, and psychosocial. Common in neurological disorders, fatigue can be a primary symptom or arise secondarily from medication, pain, depression, sleep disorders, and others. In multiple sclerosis (MS), fatigue is one of its most frequent yet underappreciated manifestations.

Methods. We conducted an observational cross-sectional study with a cohort of 40 patients diagnosed with MS. We used a questionnaire designed to assess the relationship between fatigue, multiple sclerosis, and secondary factors that may be associated.

Results. 82.5% (n=33) of the patients reported fatigue, with 70% (n=28) categorizing it as moderate or severe. Moreover, 73% (24/33) of patients with fatigue reported an impact on daily activities and social life, as well as a lack of improvement over the course of the disease. Additionally, 73% (24/33) stated that their fatigue worsens under higher temperatures or in relation to MS flare-ups. No correlation was identified between the progression to severe fatigue and current age, gender distribution, duration since diagnosis, EDSS score, or treatment type. Our analysis of secondary causes revealed that fatigue is more prevalent in patients experiencing pain and in those with numerous secondary factors.

Conclusions. Fatigue represents a concerning, pervasive, and debilitating symptom that could arise at any time during the course of MS and in all its

subtypes. It can lead to significant challenges across personal and socioeconomic spheres, impairing quality of life. Its recognition and management are crucial for patient well-being.

Keywords: fatigue, multiple sclerosis, quality of life

P007. Cognitive impairment and MRI findings in multiple sclerosis – markers of neurodegeneration

Oana UNGUREANU[1], C. DUMEA[1], O. NOEA[1], C. TARNAUCEANU[1], T. TARNAUCEANU[1], V. LESENCIUC[1], C. GROSU[1,2]

[1]Clinical Hospital of Rehabilitation, Iasi, Romania
[2]Department of Neurology, "Gr. T. Popa" University of Medicine and Pharmacy, Iasi, Romania

Multiple Sclerosis (MS) is a chronic, demyelinating disease of the central nervous system involving inflammation and progressive neurodegeneration.

Recent data have confirmed that neurodegeneration is present in all clinical phenotypes, including in early disease stages. Long-term worsening is common in patients with relapsing-remitting MS (RRMS) and is largely independent of relapses or new lesion formation. Thus, insidious progression is present in many patients with RRMS, even before this process is clinically recognized by the patient or clinician.

Cognitive disorders are frequent and disabling manifestations of MS that can be detected from the early stages of the disease, recorded as symptoms of silent progression and neurodegeneration. Recent research shows that cognitive impairment is a measurable sign of neurodegeneration, rather than a simple consequence of inflammatory processes.

Imaging studies show that thalamic atrophy is present from the early stages of the disease and reflects gray matter damage, being correlated with physical and cognitive impairment. Thalamic volume represents an indirect marker of neurodegeneration in MS.

The purpose of this work is to investigate the silent progression and neurodegeneration by correlating cognitive impairment with imaging changes in a group of 50 patients with RRMS, using the SDMT test and MRI image analysis, with a focus on thalamic changes.

Understanding the mechanisms underlying neurodegeneration is fundamental to the development of therapies that can stop this process and prevent the progression of disability.

Keywords: multiple sclerosis, neurodegeneration, silent progression, thalamic atrophy

P008. Cytomegalovirus infection in multiple sclerosis - protective factor?

Tudor-Ioan TARNAUCEANU[1], O. UNGUREANU[1], C. DUMEA[1], O. NOEA[1], C. TARNAUCEANU[1], V. LESENCIUC[1], C. GROSU[1,2]

[1]Clinical Hospital of Rehabilitation, Iasi, Romania
[2]Department of Neurology, "Gr. T. Popa" University of Medicine and Pharmacy, Iasi, Romania

Multiple sclerosis is a chronic demyelinating disease that represents an important cause of disability in young adults. Cytomegalovirus (CMV), a member of the herpesviruses family, causes latent infections in over 70% of the population.

The involvement of CMV in autoimmune diseases such as multiple sclerosis is widely recognized and widespread in the literature. Research on the role of CMV in the pathophysiological mechanisms of multiple sclerosis has conflicting results. While some studies claim that CMV can be a trigger factor in the pathology of multiple sclerosis, recent research supports the protective role of CMV in autoimmune diseases. CMV infection is associated with anti-inflammatory activity that could contribute to a milder evolution of the disease. In addition, CMV encodes numerous factors that initiate immunomodulatory and evasion mechanisms that can reduce the intensity of the immune response in multiple sclerosis.

A negative association between previous CMV infection and the risk of developing MS has been observed in case-control studies. The combined effects of CMV infection with genetic and immune triggers are being investigated in numerous studies.

The purpose of this work is to investigate the prevalence of CMV infection in a group of 60 patients with relapsing-remitting multiple sclerosis from the Neurology clinic of the Clinical Rehabilitation Hospital, Iasi, correlating the seropositivity with the EDSS value and with the MRI evolution.

The final objective of this research is to bring new data that can be useful for understanding the etiology and evolution of multiple sclerosis.

Keywords: CMV, multiple sclerosis

P009. Prevalence and characteristics of urinary complications in multiple sclerosis patients

Angelo VOICU[1], Andreea PLESA[2,3], Ruxandra GEORGESCU[2], Florentina-Cristina PLESA[2,3], Carmen-Adella SIRBU[2,3]

[1]"Prof. Dr. Agrippa Ionescu" Emergency Clinical Hospital, Bucharest, Romania
[2]Department of Neurology, "Dr. Carol Davila" Emergency University Central Military Hospital, Bucharest, Romania
[3]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction/Objective. Multiple sclerosis (MS) patients encounter various complications, notably urinary infections and lower urinary tract damage. This paper aims to identify the prevalence and types of urinary complications among individuals diagnosed with MS. Understanding the frequency and nature of these issues is crucial for comprehensive patient care and management strategies.

Material and methods. We conducted a cross-sectional study spanning from January 1 to February 29, 2024, encompassing 78 patients diagnosed with MS. Among these patients, 70 were diagnosed with the relapsing-remitting form of MS, while 6 manifested secondary progressive MS, and 2 presented with primary progressive MS. The cohort comprised 58 females and 30 males, with ages ranging from 23 to 69 years and Expanded Disability Status Scale (EDSS) scores between 0 and 7.5 points. Utilizing a structured questionnaire comprising six comprehensive inquiries, we assessed parameters such as urinary flow rate, urinary retention, frequency of urinary infections, urgency, and urinary incontinence.

Results/Conclusions. Upon analysis, we noted that 35 patients (50%) reported urinary dysfunction, with the most common complaint being urinary urgency in 28 patients, followed by impaired urinary flow characterized by a weak or interrupted stream, and frequent urinary infections. Furthermore, we observed a correlation between the frequency and severity of urinary complications with higher EDSS scores and longer disease duration. Given the progressive nature of multiple sclerosis, effectively managing urinary complications poses a long-term challenge, significantly affecting patients' quality of life.

Keywords: multiple sclerosis, urinary dysfunction, quality of life

P010. AI contribution to medical registries: Comprehensive solutions of healthcare improvement (RES-Q +)

Bogdan CASARU[1], Diana DAFINESCU[1], Anca NEGRILA[1], Elena TERECOASA[1,2], Ileana VODA[1], Paul CIUCUR[1], Cristina TIU[1,2]

[1]Neurology Department, Emergency University Hospital, Bucharest, Romania
[2]Department of Clinical Neurosciences, "Carol Davila" University of Medicine and Pharmacy Bucharest, Romania

Introduction. Based on the success of the RES-Q Registry, the EU programme HORIZON-HLT-2021-TOOL-06-031101057603-RESQ+ aims to enhance the quality of stroke care using artificial intelligence to automate the filling of medical registries from hospital databases.

Materials and methods. The project is distinguished by its collaborative framework, involving 21 partners from 13 European countries. In Romania, University Hospital of Bucharest is involved in the RES-Q Plus project, contributing to the improvement of stroke care quality through the project's innovative approaches. This includes the use of artificial intelligence and natural language processing to enhance data collection and analysis. Our task as a participating hospital is to extract medical information defined by RES-Q from discharge reports (approximately 1,200) to facilitate the creation of a fully automated tool for obtaining and processing healthcare data. Furthermore, RES-Q+ integrates two novel AI voice assistants: one to assist patients in providing feedback on their health, and another to aid physicians in delivering high-quality care.

Results. A total of 97 patients have been introduced into the registry thus far. Data collection will continue until we reach the target number.

Conclusions. This initiative is designed to create a comprehensive stroke care improvement system that integrates technological advancements to simplify care processes and outcomes.

Keywords: stroke care, RES-Q+, artificial intelligence

P011. Recent advances on the roles of PCSK-9 inhibitors in the management of acute ischemic stroke patients

S. ILUT[1,*], B. O. PIRLOG[1,*], R. PIRLOG[2,*], A. NUTU[2], V. VACARAS[1], S. M. ARMEAN[3]

[1]Department of Neuroscience, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania
[2]Research Center for Functional Genomics, Biomedicine and Translational Medicine, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania
[3]Department of Pharmacology, Toxicology and Clinical Pharmacology, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania
**These authors contributed equally to this work*

Acute ischemic stroke (AIS) represents an important cause of disability and death. Since only a minor percentage of patients with AIS are eligible for acute therapy, the management of risk factors is mandatory. An important risk factor of AIS is hyperlipidemia. The current guidelines recommend a strict correction of it. Statins are recommended as the first-line treatment, while proprotein convertase subtilisin/kexin type 9 (PCSK-9) inhibitors are administered as a second or even third option when the goal for a low-density lipoprotein cholesterol (LDL-C) level is not achieved. PCSK-9 inhibitors effectively decrease the LDL-C levels through the inhibition of PCSK-9-LDL receptor complex formation. The in-depth understanding of the PCSK-9 protein mechanism in the metabolism of LDL-C led to the development of effective targeted approaches. Furthermore, a better understanding of the LDL-C metabolic pathway led to the development of newer approaches, which increased the therapeutic options. This article aims to offer an overview of the PCSK-9 inhibitors and their mechanism in reducing the LDL-C levels. Moreover, we will present the main indications of the current guidelines for patients with hyperlipidemia and for those who have suffered an acute ischemic stroke, as well as the importance of LDL-C reduction in decreasing the rate of recurrence.

Acknowledgment: This work has been published in *Int J Mol Sci.* 2022 Sep;23(18):10221. doi: 10.3390/ijms231810221.

P012. Assessment of depression in a group of 44 patients with amyotrophic lateral sclerosis

Bianca CIUDIN

[1]Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania
[2]Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Targu Mures, Romania

Introduction. Patients with amyotrophic lateral sclerosis (ALS) have a higher risk of developing depression due to impaired quality of life. We present the results of a study conducted on patients with ALS to assess the relationship between this condition and depression. The Beck Depression Inventory was used to quantify the patients' level of depression.

Objectives. The main aims of the study were to identify correlations between the presence of depression, the time interval between onset and diagnosis, the time between onset and initiation of treatment, the severity of functional impairment (assessed by the ALSFRS-R), and the degree of depression.

Methods. A prospective longitudinal study conducted on 44 patients with ALS from the Neurology Clinic I, Targu-Mures.

Results. A statistically significant negative correlation was found between the Beck Depression Scale and the ALSFRS-R ($p < 0.001$). Therefore, the worse the clinical status of the patients, the higher the severity of depression. A statistically significant positive correlation was found between the duration from onset to diagnosis and the Beck Scale ($p = 0.009$), as well as between the duration from onset to treatment initiation and the Beck Scale ($p = 0.013$). Thus, the later the diagnosis and treatment, the higher the severity of depression.

Conclusions. The study suggests that there are correlations between depression and the severity of functional impairment, as well as between the time from onset to diagnosis and treatment and the degree of depression in ALS patients. These findings highlight the importance of early diagnosis and treatment in improving the overall well-being of these patients.

Keywords: ALS, depression

P013. Characterization of non-motor symptoms occurring in patients with amyotrophic lateral sclerosis

Maria VECIUNCA[1], Rodica BALASA[2], Anca MOTATAIANU[2]

[1]Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania.
[2]Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Targu Mures, Romania

Introduction. Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disease of motor neurons with a rapidly progressive course, with death occurring in about 3-5 years. Recent studies recognize ALS as a multisystem disorder, in which, in addition to motor symptoms well described in the literature, non-motor symptoms such as sleep disorders, dysautonomia, and sensory disorders are not as well documented.

Objectives. The aim was to characterize the non-motor symptoms of patients with ALS using the Nonmotor Symptoms Questionnaire (NMSQ), which we divided into 6 domains.

Methods. A prospective longitudinal study was performed on 44 ALS patients at Neurology I, Targu Mures, to whom we applied the NMSQ.

Results. The mean age at disease onset was 58 years. The most common form of disease onset was the spinal form in both genders, with females showing a higher frequency of the bulbar form (31.25%) compared to males (14.28%). Regarding non-motor symptoms: the most common in females was difficulty falling asleep (75%), and in males, unpleasant sensations in the legs (78.57%), with both genders reporting constipation as a common symptom. Cognitive impairment was observed significantly more frequently in females, and anxiety disorders were more common. Males reported sleep disorders (57.14%), sensory disorders (60.71%), cardiovascular disorders (67.85%), genito-urinary disorders (60.71%), and gastrointestinal disorders (39.28%) more frequently than females. Using the ALS-FRS-R, a significant gender difference was observed, indicating a higher percentage of females with ALS-FRS-R ≥30 compared to males.

Conclusions. The present study highlights the importance of non-motor symptoms occurring in patients with ALS, as few studies address this topic. In-depth investigation of these symptoms and their inclusion in patient assessment and diagnosis is essential.

Keywords: ALS, non-motor symptoms

P014. The role of ultrasonography and electromyography in identifying carpal tunnel syndrome among scleroderma patients

N. BLIDARU[1], A. C. TANASOIU[2], D. N. POPESCU[3], A. DIMA[3], D. TULBA[1,2], B.O. POPESCU[1,2,4]

[1]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[2]Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania
[3]Department of Rheumatology, Colentina Clinical Hospital, Bucharest, Romania
[4]Department of Molecular Medicine, "VictorBabes" National institute of Pathology, Bucharest, Romania

Introduction/Objectives. Systemic sclerosis (scleroderma), a connective tissue disease characterized by autoimmunity, alteration of microvascularization, and interstitial fibrosis affecting the skin and internal organs, poses an increased risk for various comorbidities, including carpal tunnel syndrome (CTS). The objectives of our study were to ascertain CTS prevalence in scleroderma patients using nerve conduction studies (NCS), to explore the relationship between clinical and electromyographic findings, and to evaluate the Pain Detect Questionnaire (PD-Q) and ultrasonographic results for CTS diagnosis in this group.

Materials and methods. Our study involved 71 patients diagnosed with scleroderma, assessed at Colentina Clinical Hospital. Participants underwent NCS to diagnose CTS, while the PD-Q evaluated neuropathic pain symptoms, and clinical diagnosis adhered to standard criteria. Ultrasonography was used to measure the median nerve—the maximum area and the area at the entrance into the carpal tunnel. Statistical analysis was conducted to ascertain correlations between diagnostic methods.

Results/Conclusions. We found a 31% prevalence of CTS in our cohort, but no statistical association between PD-Q scores and electromyographic diagnosis, indicating that neuropathic pain symptoms may not accurately reflect electromyographic signs of CTS in these patients. Similarly, clinical diagnosis did not statistically align with electromyographic findings, suggesting limitations in traditional clinical detection of CTS in scleroderma patients. Conversely, ultrasonographic diagnosis correlated with electromyographic results, highlighting ultrasonography's role in accurately diagnosing CTS in scleroderma patients. This research highlights the potential benefits of incorporating ultrasonography into the diagnostic process, potentially enhancing diagnostic precision and refining management approaches.

Keywords: carpal tunnel syndrome, scleroderma, electromyography, Pain Detect Questionnaire, ultrasonography

P015. Predictors of cognitive decline in patients with ischemic stroke

B. CIOPLEIAS[1,2], S. DIACONU[1,2], B. OPRITOIU[2], C. FALUP-PECURARIU[1,2]

[1]*Departmentof Neurology, County Clinic Hospital, Brasov, Romania*
[2]*Facultyof Medicine, Transilvania University Brasov, Romania*

Background. Vascular cognitive impairment (VCI) has a high long-term burden on patients and caregivers. Vascular risk factors and cerebrovascular disease can be used as predictors in assessing the risk of cognitive decline. The aim of the study is to assess the risk factors and prevalence of cognitive decline in patients with ischemic stroke.

Methods. This cross-sectional study included patients one month following ischemic stroke. We evaluated the patients using a standardized protocol, with demographic data, clinical profile, risk factors, neuropsychological, and neuroimaging evaluation obtained during the study.

Results. A total of 107 patients with ischemic stroke were included in the study, 59.81% of whom were male, with a mean age of 62.7 ± 11.4 years. The mean number of years of education was 10.7 ± 7.1 years. Of the patients enrolled, 7.48% were retrospectively diagnosed with a degree of cognitive impairment. The mean MoCA score at the first evaluation was 24.8 ± 2.4 and 22.6 ± 3.4 at follow-up. At the one-month follow-up, 12.14% of the patients fulfilled the criteria for vascular cognitive impairment. Stroke severity, multiple chronic lesions, hypertension, atrial fibrillation, and diabetes were the most frequent factors that increased the risk of cognitive decline.

Conclusions. Cognitive decline is frequently encountered in patients with cerebrovascular events. Lower education, pre-existing cognitive impairment, stroke severity, and multiple vascular risk factors can predict the appearance of vascular cognitive decline.

P016. The influence of vitamin D on the progression of amyotrophic lateral sclerosis

Emanuela SARMASAN[1], Rodica BALASA[1,2], Anca MOTATAIANU[1,2]

[1]*1st Neurology Clinic, Mures County Clinical Emergency Hospital, Targu Mures, Romania*
[2]*Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology of Targu Mures, Targu Mures, Romania*

Introduction. Amyotrophic lateral sclerosis (ALS) is a neurodegenerative condition characterized by the absence of a definitive cure, underscoring the significance of any potential therapeutic interventions. In light of this critical context, research has demonstrated that vitamin D plays a crucial role in promoting the survival of motor neurons and inhibiting death receptor-induced neuronal death. The inadequacy of vitamin D is associated with accelerated symptom progression in ALS and significantly reduced survival rates among affected individuals.

Materials and methods. A prospective cross-sectional single-center study was conducted with the aim of assessing the impact of vitamin D on the progression of ALS. Forty-four ALS patients and 40 age- and sex-matched healthy controls participated, with demographic and anthropometric data collected. Patients underwent clinical neurological and electrophysiological evaluations, including a comprehensive assessment using the ALS Functional Rating Scale-Revised (ALSFRS-R). Serum vitamin D levels were measured for all participants.

Results. Significant positive correlations were observed between vitamin D levels and ALSFRS-R scores, including those specific to respiratory, bulbar, and fine motor functions. Additionally, a positive correlation was noted between vitamin D levels and forced vital capacity. Upon categorizing patients based on the speed of disease progression, those with slower-evolving forms exhibited slightly elevated vitamin D levels.

Conclusions. These findings underscore the potential protective effect of vitamin D on the clinical status of ALS patients, suggesting a role in mitigating disease progression.

Keywords: amyotrophic lateral sclerosis, vitamin D, ALSFRS-R, progression of ALS

P017. Achievement of minimal symptom expression in acetylcholine-receptor antibody-positive participants with generalized myasthenia gravis and effect on disease-specific measures in ADAPT/ ADAPT+ studies

J. F. HOWARD, JR[1], H. MURAI[2], S. MUPPIDI[3], A. WOLLENBERG[4], G. PHILIPS[4],C. QI[4], D. GELINAS[4], E. BRAUER[4], S. ZHAO[4], V. BRIL[5], J. VISSING[6], ADAPT/ADAPT+ study groups

[1]*Department of Neurology, University of North Carolina, Chapel Hill, North Carolina, USA*
[2]*Department of Neurology, School of Medicine, International University of Health and Welfare, Tokyo, Japan*
[3]*Stanford Healthcare, Palo Alto, California, USA*
[4]*argenx, Ghent, Belgium*
[5]*Krembil Neuroscience Centre, University Health Network, Toronto, Ontario, Canada*
[6]*Copenhagen Neuromuscular Center, Copenhagen University Hospital Rigshospitalet, Copenhagen, Denmark*

Introduction. Minimal symptom expression (MSE), defined as Myasthenia Gravis Activities of Daily Living (MG-ADL) total score 0 or 1, is explored as a novel proposed treatment target in generalized MG.

Objective. Assess incidence, characteristics, and changes in other MG-specific scales in participants achieving MSE in ADAPT (phase 3 study of efgartigimod IV) and ADAPT+ (open-label extension).

Methods. Post-hoc analyses of ADAPT (n=129) and ADAPT+ (n=111).

Results. In AChR-Ab+ participants from ADAPT, 44.6% treated with efgartigimod achieved MSE vs 10.9% treated with placebo. In ADAPT+, 40.5% of AChR-Ab+ participants achieved MSE. 81% of efgartigimod-treated participants who achieved MSE in ADAPT continued to achieve MSE during ADAPT+; 23% who had not achieved MSE in ADAPT did so in ADAPT+. Baseline characteristics for efgartigimod-treated participants who achieved MSE during ADAPT were comparable to those who did not. Although mean (SD) baseline MG-ADL score was statistically significantly lower for those achieving MSE vs not (8.2 [1.8] vs 9.7 [2.7]), difference was not clinically meaningful. Achieving MSE was associated with substantial improvements in Quantitative Myasthenia Gravis (QMG) and Myasthenia Gravis Quality of Life (MG-QoL15r) mean scores: 11.4-point and 12.4-point decreases, respectively, from baseline to best score (across all visits). For QMG, the difference was ~4 times the threshold for clinically meaningful improvement (≥3 points). A 28-point increase in EuroQoL-5 Dimensions-5 Levels visual analog score was also seen. MSE achievement resulted in QoL comparable to healthy populations.

Conclusion. Participants who achieved MSE had substantial improvement across multiple disease measures and experienced QoL comparable to healthy populations.

Keywords: myasthenia gravis, minimal symptom expression, efgartigimod, disease-specific measures, ADAPT, ADAPT+

P018. Efgartigimod demonstrates consistent improvements in generalized myasthenia gravis across participant subgroups, including those early in diagnosis

V. BRIL[1], T. VU[2], A. WOLLENBERG[3], E. BRAUER[3], R. KERSTENS[3], J. F. HOWARD JR[4], ADAPT study group

[1]*Krembil Neuroscience Centre, University Health Network, Toronto, Ontario, Canada*
[2]*Department of Neurology, Morsani College of Medicine, University of South Florida, Tampa, Florida, USA*
[3]*argenx, Ghent, Belgium*
[4]*Department of Neurology, University of NorthCarolina, Chapel Hill, North Carolina, USA*

Introduction. In the ADAPT study, treatment with efgartigimod (human IgG1 antibody Fc-fragment that reduces pathogenic autoantibody and total IgG levels through neonatal Fc receptor blockade) resulted in clinically meaningful improvements in acetylcholine receptor autoantibody-positive (AChR-Ab+) participants with generalized myasthenia gravis (gMG).

Objective. Assess efficacy of efgartigimod in subgroups of AChR-Ab+ participants (n=129) with gMG.

Methods. Efgartigimod 10 mg/kg or placebo was administered intravenously in cycles of 4 once-weekly infusions. Efficacy was assessed using Myasthenia Gravis Activities of Daily Living (MG-ADL) and Quantitative Myasthenia Gravis (QMG) scores. Proportion of responders (improvement [MG-ADL: ≥2-point; QMG: ≥3-point] for ≥4 consecutive weeks with first improvement ≤1 week after last infusion) was assessed in participant subgroups.

Results. In participants <3 years from diagnosis, 78.6% of efgartigimod-treated participants (n=11/14) were MG-ADL responders vs 23.5% of placebo-treated participants (n=4/17; between-group difference 55.0%, 95% CI, 25.6–84.5). In non-thymectomized participants, 85.0% of efgartigimod-treated participants (n=17/20) achieved responder status vs 32.4% of placebo-treated participants (n=11/34; between-group difference 52.6%, 95% CI, 30.5–74.8). Proportion of responders among participants receiving only concomitant acetylcholinesterase inhibitors was 84.6% efgartigimod (n=11/13) vs 16.7% placebo (n=1/6; between-group difference 67.9%, 95% CI, 32.3–100.0), and receiving any corticosteroid was 63.0% efgartigimod (n=29/46) vs 29.4% placebo (n=15/51; between-group difference 33.6%, 95% CI, 14.9–52.4). Similar results were observed for other subgroups and QMG responders, consistently favoring efgartigimod. Common adverse events (mostly mild/moderate) across treatment arms were headache, nasopharyngitis, nausea, diarrhea, and upper respiratory/urinary tract infection.

Conclusion. Efgartigimod consistently improved clinical outcomes across subgroups, including participants with early disease, reinforcing efficacy across a broad gMG population.

Keywords: efgartigimod, generalized myasthenia gravis, acetylcholine receptor antibody-positive, ADAPT, subgroups

P019. Vascular risk factors among patients with Parkinson's disease

Ioana STIRBU[1], Antonia-Gabriela DIUDEA[1], Dana Marieta FODOR[1,2], Lacramioara PERJU-DUMBRAVA[1,2]

[1]Department of Neurology, Cluj County Emergency Hospital, Cluj-Napoca, Romania
[2]Department of Neurology, Faculty of Medicine, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

Introduction. Worldwide, cardiovascular diseases are the leading cause of death, and increasing evidence suggests a high prevalence in Parkinson's disease. The present study aimed to assess the relevance of the triglycerides/high-density lipoproteins cholesterol ratio in the development of cardiovascular disease among Parkinson's patients, compared to classical vascular risk factors.

Material and methods. This study was designed as retrospective and analytic, with data collected from patient observation sheets after obtaining approval from the ethics committee. The study group consisted of 193 patients diagnosed with Parkinson's disease. Statistical analysis was performed using IBM SPSS Statistics 26, with Chi-square test, Fisher test, Mann U test, T-test, and univariate and multivariate logistic regression.

Results. A weak correlation was identified between age, triglycerides, systolic and diastolic blood pressure, and the risk of developing cardiovascular diseases. Cholesterol/triglycerides ratio was found to be a protective factor.

Conclusions. The highest prevalence of the disease was found in the age group 61 to 80 years, which is the group most at risk for vascular events. Close monitoring of this group of patients is mandatory.

Keywords: Parkinson's disease, cardiovascular risk factors

P020. Diagnostic and therapeutic approach of a patient with autoimmune encephalitis

A. I. VASILE[1,2], C. NICA[1], A. RATA[1], B. AZAMFIREI[1], C. TIU[1,2]

[1]Department of Neurology, Emergency University Hospital, Bucharest, Romania,
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Encephalitis is a severe inflammatory disorder of the brain that presents as a rapidly progressive encephalopathy. Diagnostic criteria include subacute onset of working memory deficits, seizures, or psychiatric symptoms; bilateral brain abnormalities on T2-weighted fluid-attenuated inversion recovery MRI restricted to the medial temporal lobes; and CSF pleocytosis or EEG showing epileptic or slow-wave activity involving the temporal lobes. Cancer screening should be considered in most patients with autoimmune encephalitis.

Materials and methods. We present a case of a 46-year-old male without previous diseases who was transferred from an infectious diseases hospital with the suspected diagnosis of autoimmune encephalitis. The patient presented with altered mental state, psychomotor agitation, temporal and spatial disorientation, disorientation about himself, and oppositional conduct disorder. Cerebral MRI revealed extensive subcortical areas with swelling of the adjacent cortex in hypersignal T2, with gyriform contrast uptake in the hippocampal, parahippocampal, temporo-basal, and lower temporal areas bilaterally, extending infero-middle on the left side, suggesting autoimmune encephalitis. Lumbar puncture detected anti-GAD antibodies. EEG was not possible due to extreme psychomotor agitation. Screening for malignancies was negative, including negative tumor markers and a normal chest-abdomen-pelvis CT exam.

Results. During hospitalization, the patient was treated with antiviral (Acyclovir), antibacterial (Ceftriaxone), and steroids (Dexamethasone). The treatment for autoimmune encephalitis was initiated with high-dose corticotherapy (Methyl-prednisolone), immunotherapy (Rituximab), intravenous immunoglobulin, and plasmapheresis. The patient showed slow, favorable improvement, with dimensional regression of cerebral lesions on subsequent MRI examinations.

Conclusions. This case highlights the diagnostic and therapeutic approach in a patient with limbic encephalitis and extreme psychomotor agitation. Despite the positive anti-GAD antibodies, no associated malignancy was identified.

Keywords: Autoimmune encephalitis, associated neoplasms, anti-GAD antibodies, psychomotor agitation

P021. Neuro-Behcet's disease: a diagnostic challenge

A. GUTU[1], A. STAN[1,2,3], F. D. MURESANU[1,2,3]

[1]*Neurology Department, Emergency County Hospital, Cluj-Napoca, Romania*
[2]*Department of Neurosciences, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania*
[3]*RoNeuro Institute for Neurological Research and Diagnostic, Cluj-Napoca, Romania*

Introduction. Behcet's disease is a rare systemic disorder of unknown etiology that causes blood vessel inflammation. It is commonly characterized by mucocutaneous lesions and bilateral uveitis. Neurological involvement, known as Neuro-Behcet's disease (NBD), is present in only 5% to 10% of patients, but it is associated with a worse prognosis and can lead to permanent deficits or even death. In this study, we present an interesting case of NBD in which the differential diagnosis was very challenging.

Case report. A 45-year-old male patient with no significant medical history presented with an acute onset of left-sided weakness and headache. The brain CT scan, followed by brain MRI with contrast, showed a right fronto-parietal tumor. The patient was transferred to the Neurosurgical Department where the tumor was removed. However, the result of the histopathological examination came back negative, suggesting ischemic necrosis. Moreover, the one-month postoperative brain MRI with contrast suggested the diagnosis of a pseudotumoral form of multiple sclerosis (MS). Thus, the patient was readmitted for further extensive investigations, including lumbar puncture, which was negative. Repeated brain and cervical spinal cord MRI with contrast and spectroscopy indicated no MS lesions and was also highly suggestive of multiforme glioblastoma. After a more detailed discussion with the patient, we discovered that he had a brother diagnosed with Behcet's disease and a history of oral ulcers, information which led us to the differential diagnosis of NBD. The patient was referred to the Rheumatology Department, where immunosuppressive treatment with glucocorticoids and cyclophosphamide was initiated, leading to favorable clinical and imaging evolution.

Conclusions. Due to its similar presentation with other pathologies such as glioblastoma, stroke, or MS, NBD can be a real challenge for the clinician, emphasizing the importance of a correct differential diagnosis.

P022. Controversies in the diagnosis and management of idiopathic inflammatory myopathies

C. GATCAN, L. CUCU, A. ROTAR, B. E. IGNAT

Clinical Recovery Hospital,"Grigore T. Popa" University of Medicine and Pharmacy Iasi, Romani

Introduction. Idiopathic inflammatory myopathies (IIM) are a group of rare, heterogeneous autoimmune diseases with variable clinical and paraclinic manifestations. Due to the limitations of clinical criteria, they can pose difficulties in diagnosis and management, especially in the setting of overlapping connective tissue disease.

Case presentation. A 69-year-old female patient presented to the Neurology Clinic for progressive weakness with a slow onset, discretely asymmetric, predominantly proximal, later associated with significant dysphagia and dysphonia, along with worsening symptoms and weight loss in the last 2-3 years. The patient also had a history of cardiovascular (acute myocarditis, mitral valve prolapse, extrasystolic arrhythmia), rheumatological (severe polyarticular syndrome), and neurological (two transient ischemic attacks) conditions. The extensive myositis profile was negative, and muscle destruction enzymes were insignificantly elevated. However, further paraclinical investigations, including repeated electrophysiological examination, muscle biopsy (performed twice at a 29-year interval), thigh muscle MRI, and whole exome sequencing (which excluded genetic mutations), suggested IIM. Despite this, the definitive diagnosis remained unclear, and intermittent seropositivity for Rheumatoid Factor, Anti-dsDNA, Anti-Cardiolipin, and Anti-Smith Antibody pointed towards an overlap syndrome. The patient was initially treated with immunosuppressives and corticosteroids without efficacy, but treatment with immunoglobulins improved the patient's symptoms.

Conclusion. IIM overlapping with systemic disease requires an individualized approach in correlation with clinical and paraclinical findings when certain clinical criteria are not met. Identifying characteristic elements is crucial for effective management.

P023. Sporadic Creutzfeldt-Jakob disease: case presentation and update on pathogenesis and diagnostic criteria

Anamaria JURCAU, Vharoon Sharma NUNKOO

[1]*Faculty of Medicine and Pharmacy, University of Oradea, Oradea, Romania*
[2]*Clinical Emergency County Hospital Bihor, Oradea, Romania*

Introduction. Despite being a rare disease, with reported incidence between 1 and 2 cases/1,000,000/year, after the recent pandemic, several published articles have reported cases of sporadic Creutzfeldt-Jakob disease (sCJD) occurring either after SARS-CoV-2 infection or after vaccination.

Material and methods. We present a case of sCJD diagnosed in our hospital, review recent advances in unravelling pathogenesis, diagnostic criteria (with emphasis on imagistic diagnosis), classification, and discuss potential future therapies.

Conclusion. The diagnosis is challenging and requires a high degree of clinical suspicion, but the diagnostic criteria are continuously updated.

Keywords: sporadic Creutzfeldt-Jakob disease, magnetic resonance imaging, diagnostic criteria, therapeutic attempts

P024. Navigating the unusual: a rare case report on ANCA-associated vasculitis unveiling mononeuritis multiplex as the primary presentation

Cristian GHITA[1], Simona PETRESCU[1,2], Ciprian JURCUT[2,3], Ionela CODITA[1], Andrei MIHAILESCU[3], Maria MARTOIU[1], Mihai PERIANU[1], Alina NEDEA[1], Cristina PANEA[1,2]

[1]*Neurology Clinic, Elias University Emergency Hospital, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*Rheumatology Clinic, "Dr. Carol Davila" Central Military Emergency Hospital, Bucharest, Romania*

Introduction. Granulomatosis with polyangiitis (GPA), a rare autoimmune disorder, typically presents with respiratory or renal manifestations. Mononeuritis multiplex, a peripheral neuropathy affecting multiple nerves, is an uncommon debut of GPA, posing diagnostic challenges. We present a case of a 61-year-old female with GPA initially presenting as mononeuritis multiplex.

Methods. In this case report, we detail a rare instance of ANCA-associated vasculitis, specifically GPA, with mononeuritis multiplex as the primary presentation. The patient, with a medical history significant for hypertension and dyslipidemia, presented with distal paresthesias in both legs, progressing over a year. Neurological examination revealed sensory and motor deficits consistent with mononeuritis multiplex, alongside cutaneous manifestations, renal dysfunction, and visual impairment. Notably, these neurological manifestations exhibited asymmetry, significantly affecting her gait, with the left lower limb more impaired. Additionally, weakness in the left brachial region and visual impairment in her right eye were reported.

Results. A comprehensive diagnostic workup ensued to elucidate the underlying etiology of these neurological deficits. Initial imaging studies showed no significant findings. Subsequent investigations, including electrophysiological studies and serum investigations, revealed acute axonal sensory-motor polyneuropathy, inflammatory syndrome, and renal dysfunction. Notably, high c-ANCA titers were observed. Ophthalmologic evaluation revealed ischemic lesions within the right optic nerve. Skin biopsy demonstrated minimal lymphocytic vasculitis lesions. These findings led to the conclusive diagnosis of ANCA-associated vasculitis, specifically granulomatosis with polyangiitis, primarily presenting as mononeuritis multiplex.

Conclusion. This case highlights the importance of considering GPA in neurological presentations and the potential benefits of early aggressive immunosuppressive therapy in managing ANCA-associated vasculitis.

Keywords: granulomatosis with polyangiitis, mononeuritis multiplex, ANCA-associated vasculitis, acute axonal sensory-motor polyneuropathy

P025. Difficulties of diagnosis in immune-mediated necrotizing myopathy overcome by muscle biopsy in an elderly person: A case report

Clara PROFIRESCU, Simona PETRESCU, Maria Melania MARTOIU, Ionela CODITA, Cristina PANEA, Alexandra BASTIAN

Neurology Clinic, Elias University Emergency Hospital, Bucharest, Romania
"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. The ENMC (European Neuromuscular Center) criteria recognize three types of immune-mediated necrotizing myopathy (IMN): myositis with antibodies against SRP (signal recognition particle), myositis with antibodies against HMGCR (hydroxy-3-methylglutaryl-CoA reductase), and seronegative myositis. Muscle histopathological examination allows differentiation between these entities and inflammatory myopathies.

Methods and results. A 77-year-old patient, undergoing statin treatment, was hospitalized for a tetramyelic motor deficit, predominantly proximal, with onset and progressive evolution over two weeks. The patient's symptoms appeared after a probable viral upper airway infection. The neurological clinical examination suggested muscle damage, with predominantly crural proximal motor deficits, possible walking with a frame, and decreased crural osteotendinous reflexes. The patient also had skin lesions on the surfaces of large joints, resembling polymorphous erythema. Biologically, there was an inflammatory syndrome and a significant increase in muscle enzymes, with creatine kinase (CK) elevated x 10,000. Skin biopsy highlighted solar elastosis. Oncological serum markers and chest-abdomen-pelvis computed tomography (CT) with contrast did not reveal any abnormalities. Muscle biopsy established the diagnosis, showing necrosis of muscle fibers in different stages, minimal inflammatory infiltrates, and increased expression of HMGCR. The clinical and biological evolution was slowly favorable under corticosteroid therapy.

Conclusion and discussion. Elderly patients may present with myopathies associated with neoplasia, and skin damage may raise this suspicion. The muscle biopsy result established the diagnosis. The increased expression of HMGCR may be explained by statin use but could also be secondary to a positive status of anti-HMGCR antibodies, a test that was not performed.

Keywords: myopathies, myositis, antibodies against HMGCR

P026. Aseptic meningitis with multiple cranial nerve palsies in a patient with rheumatologic joint pathology

M. C. NITU, R. A. CERCEL, A. ENE

Department of Neurology, Bucharest Emergency University Hospital, Bucharest, Romania

Introduction. Rheumatologic-related meningitis represents an extremely rare manifestation of rheumatoid arthritis with a heterogeneous clinical picture, which may include epileptic seizures, focal neurological deficits, headache, or confusion. The diagnosis is difficult to establish, with few cases reported in the specialized literature, necessitating extensive investigative assessment.

Materials and methods. We present a clinical case of a 38-year-old patient with juvenile idiopathic arthritis diagnosed at the age of 12. The patient was treated with methotrexate and prednisone, under chronic treatment with anti-inflammatory drugs, and presented with symptoms that started a week ago, including bilateral facial paresthesia, facial asymmetry, speech disturbance, horizontal diplopia, and headache.

Results. Neurological clinical examination revealed limited abduction movement in the right eye, right peripheral facial palsy, and right hemiface hyperesthesia. Native cerebral CT scan and contrast-enhanced MRI did not reveal any changes explaining the clinical manifestations. Investigations, including cervical-thoracic MRI, showed no signal abnormalities at the spinal level. Lumbar puncture revealed proteinorachia with albuminorachia and 141 cells/mm^3, predominantly mononuclear. Bacterial and fungal cultures from CSF yielded negative results. Immunologic and infectious assessments were also negative. Pulse therapy with methylprednisolone was initiated, followed by oral corticosteroid therapy, resulting in complete remission of neurological deficits.

Conclusion. Although there is no consensus on the diagnosis and treatment of this pathology due to its low incidence, a differential diagnosis should be considered in any patient with a history of rheumatoid arthritis presenting with neurological or psychiatric manifestations.

P027. Exotic name of an easy-to-miss disease

M. L. FURNICA[1], C. E. MITU[1], B. O. POPESCU[1,2,3]

[1]*Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania*
[2]*Clinical Neuroscience Department, Faculty of Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*Neuroscience and Experimental Myology Laboratory, Laboratory of Cell Biology, "Victor Babes" National Institute of Pathology, Bucharest, Romania*

Introduction. PADMAL (pontine autosomal dominant microangiopathy with leukoencephalopathy) is a recently described, rare form of monogenic cerebral small vessel disease with autosomal dominant transmission associated with mutations in the COL4A gene.

Methods. In this report, we present the case of a 44-year-old patient with no traditional vascular risk factors, who has a history of multiple strokes and associated gait disturbance due to ataxia and spasticity of uncertain onset. Family history includes 2 affected individuals (mother and brother of the patient) with symptoms suggestive of multiple strokes.

Results. Cognitive examination revealed mild impairment (MoCA score of 22). Brain MRI shows multiple subcortical white matter lesions, pontine lacunes, and cerebellar and pontine atrophy. Blood workup was normal, and CSF analysis was nonspecific (slightly elevated protein levels, normal lactic acid levels). Fabry disease testing was negative. We performed genetic testing, which revealed a COL4A1 mutation in the 3'UTR region (c.*32G>A) with pathogenic significance. This specific variant was previously described in 2 families, with the associated phenotypes being multi-infarct dementia and PADMAL, respectively.

Conclusions. Our report confirms the role of mutations in the 3'UTR region of COL4A in the development of PADMAL and contributes to understanding the mechanisms involved in the pathogenesis of this rare and complex condition.

Keywords: PADMAL, stroke, hereditary cerebral microangiopathy

P028. Psychogenic involuntary movements with a treatable cause

I. D. ISACHI[1], D. C. ANGHEL[1,2], I. C. LUPESCU[1,2], M. C. ANGHEL[2], C. V. TOADER[1], A. O. DULAMEA[1,2]

[1]*Fundeni Clinical Institute, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Background. Psychogenic movements are involuntary motor symptoms that develop suddenly and progress rapidly to maximum severity. They have variability over time and may include tremor, dystonia, myoclonus, jerks, and gait disturbances. Distractibility during examination is a very important feature for differential diagnosis with an organic disorder. We present a case of a female with episodic pleiomorphic involuntary movements, voice abnormalities, and alteration of consciousness associated with insulinoma. Insulinomas are small neuroendocrine tumors that are difficult to locate and cause hypoglycemia and neuroglycopenic symptoms such as acute disorders of cognition, consciousness, seizures, and psychosis.

Case report. A 56-year-old female was admitted to the hospital for a prolonged episode of altered consciousness, diaphoresis, and gestural automatisms, which spontaneously resolved after 30 minutes. At admission, she suffered another episode of involuntary psychogenic movements, abnormal behavior, and severe hypoglycemia (23 mg/dl). She was admitted to the ICU and recovered completely during the continuous glucose infusion. Further investigations were started. Abdominal CT scan and 99-m Tc-Tektrotyd SPECT-CT showed pancreatic insulinoma, which was successfully removed by surgery. At two years of follow-up, the patient was asymptomatic.

Conclusion. Hypoglycemia caused by an insulinoma, although not a common disease, should be considered in patients with episodic atypical neuropsychiatric symptoms, as the delay in diagnosis can lead to significant neurological sequelae.

Keywords: hypoglycemia, insulinoma, neuropsychiatric manifestations

P029. Chronic inflammatory demyelinating polyneuropathy as paraneoplastic syndrome – case report

O. NOEA[1], O. UNGUREANU[1], C. DUMEA[1], T. TARNAUCEANU[1], V. LESENCIUC[1], C. GROSU[1,2]

[1]*Neurology Clinic, Clinical Rehabilitation Hospital Iasi, Iasi, Romania*

[2]*"Gr. T. Popa" University of Medicine and Pharmacy, Iasi, Romania*

Introduction. Chronic inflammatory demyelinating polyneuropathy (CIDP) is a rare disease with multiple potential causes. Its association with hematological malignancies, especially multiple myeloma, plasmacytoma, or lymphoma, is well known.

Materials/Methods. We present the case of a 67-year-old man diagnosed with CIDP in December 2022. He was initially treated with intravenous immunoglobulin infusions and corticosteroids, showing a favorable evolution of symptoms. The patient experienced a second relapse in July 2023, which showed a slow but favorable clinical response after the same therapy. In September, he was admitted to the Neurology Clinic of the Clinical Rehabilitation Hospital Iasi with suspicion of a new clinical relapse, presenting flaccid tetraparesis with a predominance of paraparesis. Laboratory studies revealed thrombocytosis and significantly elevated D-Dimer levels. A pulmonary CT angiogram showed an osteolytic tumor of the right seventh rib, with vertebral and pleural invasion. Bone biopsy and immunohistopathology tests confirmed the diagnosis of bone plasmacytoma. Over the following three months, his condition deteriorated with repeated hospitalizations for cardiac and respiratory complications, eventually leading to his death approximately four months after the oncological diagnosis was established.

Conclusions. The particularity of this case lies in the initially favorable clinical evolution under standard treatment, which concealed an underlying malignant hematological condition. We propose considering the etiology as paraneoplastic and emphasize the importance of serum and urine immunoelectrophoresis and/or skeletal bone surveys for patients with CIDP.

Keywords: CIDP, paraneoplastic, plasmacytoma

P030. Cerebral small vessel disease with multiple cerebral hemorrhages in a patient with autosomal dominant polycystic kidney disease

A. I. EVA-MANOLESCU[1], C. BANICA[1], R. CAPSA[2], I. G. LUPESCU[2], A. O. DULAMEA[1]

[1]*Neurology Department, Fundeni Clinical Institute, Bucharest, Romania*

[2]*Radiology and Imaging Department, Fundeni Clinical Institute, Bucharest, Romania*

Introduction and objectives. Autosomal dominant polycystic kidney disease (ADPKD) affects over 12 million people worldwide and is the most common genetic pathology of the kidney. While the association between ADPKD and saccular or fusiform cerebral aneurysms has been known for a long time, recent studies emphasize a link between ADPKD and cerebral small vessel disease independent of cardiovascular risk factors.

Materials and methods. Clinical case presentation based on the study of medical documents and review of current knowledge.

Results/Conclusions. The clinical case presented highlights the complexity of a patient with ADPKD who associates extensive cerebral small vessel disease with multiple cerebral hemorrhages, along with several other complications, mainly of an infectious nature. We will also explore the possible mechanisms by which ciliary dysfunction in PKD1 and PKD2 gene mutations leads to cerebral complications, such as endothelial dysfunction, blood-brain barrier alterations, and disruption of cerebrospinal fluid flow in the glymphatic system.

Keywords: autosomal dominant polycystic kidney disease, cerebral small vessel disease, glymphatic system, cerebral hemorrhages

P031. Cryptogenic brain abscesses with unidentifiable pathogen

T. TRINCA[1], M. C. ZAMFIR[1], C. D. RAGAN[1], C.A PANEA[1,2], F. ANGHEL[1]

[1]*Elias Emergency University Hospital, Bucharest, Romania*

[2]*"Carol Davila", University of Medicine and Pharmacy Bucharest, Romania*

Introduction. A brain abscess is a focal infectious collection in the brain parenchyma that may result as a complication of an infection, secondary to trauma, or cranial surgery. A cryptogenic abscess is diagnosed when no primary source of infection can be identified. We present the case of a male patient with an unremarkable medical condition that could predispose him to develop a brain abscess. His symptoms consisted of meningeal syndrome, fever, altered state of consciousness, epileptic seizures, cranial nerve paresis, and tetraparesis.

Methods. To confirm the diagnosis, we used brain imaging (contrast-enhanced CT and MRI), cerebrospinal fluid examination, and extensive biological and paraclinical infectious screening.

Results. Brain imaging evaluation highlighted the presence of multiple intracranial expansive processes with the appearance of brain abscesses, ventriculitis, and cerebritis. The cerebrospinal fluid examination was suggestive of an infectious process, but no pathogen was identified. The biological and imaging screening refuted the possibility of any neoplastic disease. No infectious focus that could disseminate was found. Neurosurgical intervention could not be performed for technical reasons. The clinical and imaging evolution under broad-spectrum antibiotic and antifungal treatment, administered empirically for a prolonged period, was favorable, with improvement of the patient's clinical condition and regression of the brain lesions.

Conclusions. There are few cases where a brain abscess can develop without any predisposing factor. When the predisposing condition is unknown and neurosurgical procedures cannot be performed, the diagnosis and management of such patients can present a serious challenge.

Keywords: cryptogenic brain abscesses, unidentifiable pathogen

P032. Concomitant Parkinson's disease with familial idiopathic calcifications of the basal ganglia

A. R. BONDALICI[1,2], X. HAMMAD[1,2], D. C. FRATICIU[1,2], R. D. MIHAILESCU[1], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,3]

[1]Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Cell Biology Laboratory, Neurosciences and Experimental Myology, National Institute of Pathology "Victor Babes", Bucharest, Romania

Fahr's disease is a rare hereditary neurological pathology with autosomal dominant transmission, characterized by abnormal calcium deposits in the basal ganglia and cerebral cortex, requiring differential diagnosis with Parkinson's disease and parkinsonian syndromes. We present the case of a 62-year-old man, known for approximately 10 years with major depressive disorder and parkinsonian syndrome, with significant bilateral basal ganglia calcifications evident on imaging. The initial symptoms consisted of bradykinesia, a slow progressive gait disturbance, with inconsistent improvement with dopaminergic agonist administration, and minimal therapeutic response to levodopa-based therapies. A year ago, he was hospitalized for evaluation, presenting freezing of gait, balance disturbance with falls, dysarthria, and non-motor symptoms (muscle cramps, sleep disturbance). The therapeutic regimen was adjusted with levodopa-carbidopa and rotigotine, showing a positive clinical response (notably, significant improvement of hypomimia and global bradykinesia, without rigidity). The apomorphine test was positive, with a recommendation for use during "off-severe" episodes. Currently, he returned due to the progression of motor symptoms and was considered a candidate for advanced therapy with intestinal gel. After titrating the doses dynamically, we observed definite improvement in symptoms (walking speed, bradykinesia). We are most likely witnessing a concomitant pathology of Parkinson's disease and Fahr's disease (given the bilateral basal ganglia calcifications, the variability of therapeutic response, and the very slow progression over 10 years from diagnosis, supporting the probability of parkinsonism within Fahr's disease initially detected). However, the subsequent development of asymmetric symptoms, definite responsiveness to levodopa and apomorphine, the appearance of motor fluctuations (delayed-on/wearing-off), and discrete peak-dose dyskinesias point toward a diagnosis of Parkinson's disease.

P033. An unusual case of leptomeningeal carcinomatosis

T. TRINCA[1], M. C. ZAMFIR[1], C. D. RAGAN[1], C.A. PANEA[1,2]

[1]Elias Emergency University Hospital Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Leptomeningeal carcinomatosis is a rare neuro-oncological disorder with a very low survival rate, rarely exceeding one year. We present the case of a female patient with prior breast cancer, who was diagnosed with leptomeningeal distant metastasis 12 years after the tumour was considered cured, with no other secondary sites found.

Methods. To confirm the diagnosis, we used thoracic, abdomino-pelvic and brain CT scans, contrast-enhanced MRI of the neuroaxis, lumbar puncture and immunohistochemistry of the cerebrospinal fluid.

Results. Thoracic, abdomino-pelvic and brain CT did not detect any pathological modifications which might suggest secondary metastases. Neuroaxis MRI confirmed the site of carcinomatous meningitis. Meningeal relapse with breast cancer cells was confirmed through immunochemistry of cerebrospinal fluid. Diagnosing this disorder is oftentimes challenging, but in this case, the diagnosis was confirmed with relative ease. However, the presentation was atypical, as no other tumour sites or metastases were found, except the one located at the leptomeninges. The patient died 8 months after the confirmed diagnosis.

Conclusions. Carcinomatous meningitis is clinically rare, and its presentation can be atypical. Therefore, failing to find other tumour/metastasis sites in a patient with oncological disease history should not exclude the possibility of this type of cancer.

Key words: breast cancer cells, no breast cancer relapse, no other tumour sites found

P034. Multidisciplinary approach to treating cerebral sinus thrombosis in T-cell acute lymphoblastic leukemia

C. E. VLAICU[1], I. CALOIANU[1,2]

[1]Neurology Department, "Dr. Carol Davila" Central Military Emergency Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. One well-known complication of induction chemotherapy for acute lymphoblastic leukemia (ALL) is venous thrombosis, but the most life-threatening thrombotic event is thrombosis of the cerebral veins. While its incidence during ALL induction therapy is well-recognized, little information is known about its evolution, risk factors, and clinical presentation, making a case like this a unique clinical challenge. We present a case study of a 29-year-old patient diagnosed with T-cell acute lymphoblastic leukemia, who subsequently developed right cerebral transverse and sigmoid sinus thrombosis.

Method. The patient initially presented with symptoms suggestive of a lymphoproliferative disorder and was diagnosed with T-cell acute lymphoblastic leukemia and with a mediastinal tumor, possibly Burkitt or lymphoblastic lymphoma. Following induction chemotherapy, the patient experienced a seizure, prompting further evaluation, which revealed right cerebral transverse and sigmoid sinus thrombosis. The subsequent clinical course was with recurrent seizures, necessitating intensive care management.

Results. Management included anticoagulation therapy along with antiepileptic medications, which led to seizure cessation. The patient demonstrated favorable progress, marked by the absence of further seizures and near-complete remission of hemorrhagic strokes.

Conclusion. This case highlights the importance of recognizing cerebral sinus thrombosis as a potential complication in patients with hematologic malignancies undergoing chemotherapy. Early recognition and multidisciplinary management are crucial for optimizing patient outcomes in such complex scenarios.

Keywords: T-cell acute lymphoblastic leukemia, cerebral sinus thrombosis, chemotherapy, seizure, anticoagulation, multidisciplinary management

P035. Neuromyelitis optica versus multiple sclerosis: a differential diagnosis journey – case report

Maria Ruxandra DOCAN[1], Andreea Raluca HANGANU[1,2], Anca Adriana ARBUNE[1], Alexandra GHIUTA[1], Basel Robert TORON[1], Lorenzo Giovanni ZURINI[1], Adriana Octaviana DULAMEA[1,2]

[1]Fundeni Clinical Institute, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Autoimmune disorders have the potential to impact various systems within the human body, often exhibiting overlapping characteristics across different diseases. We present the case of a 47-year-old male with a lifelong history of autoimmunity, manifested in the neurological area.

The patient experienced early-onset thymoma-associated myasthenia gravis in his twenties, with complete alleviation of symptoms following thymectomy. Over time, he had multiple episodes of visual loss in both eyes, without remission and left untreated. Multiple consecutive MRIs found no cerebral lesions. Thirteen years after the last episode, he developed loss of sensory function and coordination in the upper right limb, with dysesthesias in his right hemithorax. MRI revealed multiple T2/FLAIR hyperintense lesions located both supratentorially and infratentorially, including a cervical C1–C6 lesion, with gadolinium enhancement at the C1–C3 level in the posterior columns. Serum and cerebrospinal fluid laboratory tests revealed negative anti-AQP4 antibodies, a low level of anti-ACh receptor antibodies, oligoclonal bands, and an elevated serum/CSF IgG index, indicating inflammation within the central nervous system. Remission occurred under intravenous methylprednisolone therapy.

Considering the prolonged course over twenty years since the initial episode of optic neuritis, the absence of optic nerve atrophy, the active cervical lesion confined to three vertebral bodies in the posterior columns, the absence of anti-AQP4 antibodies, and the presence of oligoclonal bands, a diagnosis of multiple sclerosis was established and treatment with Ocrelizumab was initiated. Although bilateral optic neuritis and its association with myasthenia gravis are more frequent in neuromyelitis optica, it is imperative to consider the entire clinical picture when reaching a diagnosis.

Keywords: multiple sclerosis, neuromyelitis optica, neuroinflammatory disorders

P036. A case of herpes simplex virus 1 (HSV-1) encephalitis with concomitant syndrome of inappropriate antidiuretic hormone secretion (SIADH) in a patient treated with teriflunomide – can teriflunomide increase the risk of HSV-1 encephalitis in multiple sclerosis?

D. POPESCU[1], I. CALOIANU[1,2]

[1]"Dr. Carol Davila" Central Military Emergency University Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Our aim is to describe an atypical case of HSV-1 encephalitis, with fewer prodromal symptoms, a cerebrospinal fluid (CSF) profile showing low white cell count, and widespread MRI abnormalities, in a patient treated with teriflunomide, and to discuss its possible role as a risk factor.

Materials and methods. A 47-year-old male, diagnosed with relapsing-remitting multiple sclerosis 4 years ago and treated with teriflunomide, with no signs of disease activity at his last check-up and a functional score of 3.5 on the Expanded Disability Status Scale, presented with acute confusional state and slurred speech, symptoms that had started approximately 72 hours prior to admission and showed progressive worsening. Brain MRI revealed bilateral asymmetrical lesions involving the limbic system and the left frontal lobe. The search for HSV-1 DNA in the CSF using qualitative RT-PCR was strongly positive. CSF analysis showed only mild pleocytosis. Blood tests revealed severe hyponatremia with elevated urinary sodium and osmolality, confirming the diagnosis of SIADH. Teriflunomide was discontinued and treatment was initiated with acyclovir alongside electrolyte correction.

Conclusion. To our knowledge, based on a review of the literature, this is the first reported case of HSV-1 encephalitis associated with teriflunomide treatment. We found similar cases related to other disease-modifying therapies such as natalizumab, ocrelizumab, fingolimod, and dimethyl fumarate. To what extent teriflunomide-treated patients might be exposed to a higher risk of viral infections, and whether it implies impaired immunity against viruses, has not been addressed in clinical studies yet.

Keywords: HSV-1 encephalitis, teriflunomide, multiple sclerosis, SIADH

P037. Akinetic mutism following bilateral anterior cerebral artery territory infarction

Valentin MOROSANU, Anca MOTATAIANU, Rodica BALASA

Neurology 1 Clinic, Emergency Clinical County Hospital Mures, Targu Mures, Romania
"George Emil Palade" University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Targu Mures, Romania

Background. Akinetic mutism, also referred to as "abulia major" or "coma vigile", is a rare neuropsychological syndrome characterized by impaired initiation and motivation for behavior, intact consciousness, absence of movement, and lack of speech. This syndrome manifests despite the absence of apparent impairment in language and sensorimotor functions. One of the suggested primary pathophysiological mechanisms is bilateral disconnection of the fronto-subcortical circuits.

Case report. A 77-year-old woman, with a history of myocardial infarction 15 years ago and a bulbar ulcer complicated by hemorrhage 3 years ago, currently on antihypertensive treatment at home, was admitted to our clinic following sudden-onset weakness in her left limbs, predominantly affecting the lower limb, which occurred three days prior to admission. From the onset of hospitalization, the patient displayed apathy, abulia, and exclusively accepted food from her daughter. She exhibited progressive deterioration in her overall condition, manifested by immobility and lack of verbalization.

Subsequent CT scans of the brain did not reveal any lesions that could explain the neurological decline. Cerebral MRI was performed and revealed infarction in the bilateral territory of the anterior cerebral artery. ACA strokes account for approximately 2% of all ischemic strokes, and bilateral ACA infarction is even rarer.

Conclusion. This case emphasizes once again that the main pathogenetic mechanism of this syndrome is alteration of the fronto-subcortical networks. Although akinetic mutism is a rare entity, it represents a major cause of long-term disability. It sometimes requires additional investigations to elucidate the etiology, as was the case presented.

Keywords: akinetic mutism, bilateral anterior cerebral artery territory infarction

P038. Right-sided frontal cerebritis with abscess formation in the setting of disseminated mucormycosis

Ioan-Cristian LUPESCU[1,2],
Maria Speranta IACOB[2,3],
Andreea-Lacramioara MOHOREA-NEATA[4],
Ioana-Gabriela LUPESCU[2,5],
Adriana Octaviana DULAMEA[1,2],
Liliana GHEORGHE[2,3]

[1]*Neurology Department, Fundeni Clinical Institute, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*Gastroenterology and Hepatology Department, Fundeni Clinical Institute, Bucharest, Romania*
[4]*Infectious Diseases, Hematology Department, Fundeni Clinical Institute, Bucharest, Romania*
[5]*Radiology and Medical Imaging Department, Fundeni Clinical Institute, Bucharest, Romania*

Introduction. Mucormycosis is a rare fungal infection usually characterized by rhino-orbital-cerebral and pulmonary involvement. Common risk factors include diabetes mellitus, hematologic malignancies, hematopoietic cell transplantation, and solid organ transplantation.

Material and methods. We present the case of a 54-year-old male with toxic-nutritional liver cirrhosis, for which a liver transplant was performed in 2023. Immunosuppressive therapy with tacrolimus, azathioprine, and corticosteroids was initiated. The patient subsequently developed steroid-induced diabetes mellitus with severe ketoacidosis and sepsis of pulmonary origin. The clinical picture was complicated by the sudden onset of severe headache, right-sided retro-orbital pain with complete ophthalmoplegia and ptosis, right-sided peripheral facial palsy and hypoesthesia, right-sided amblyopia, and paraparesis 4/5 MRC.

Contrast-enhanced head CT scan revealed a hypodense lesion involving the right-sided frontal lobe and bilateral acute sinusitis. Brain MRI highlighted cortico-subcortical lesions with diffusion restriction at the level of the frontal lobe (extending to the genu of the corpus callosum), enhancement of the adjacent meninges, diffusion restriction and enlargement of the right-sided optic nerve in its intraorbital course, thrombosis of the right-sided ophthalmic vein, and pansinusitis with mucosal enhancement. Subsequent brain imaging scans noted the formation of a peripheral ring-like enhancement with perilesional edema at the level of the frontal lobe. Bacteriological exam of nasal samples revealed the presence of *Mucorales rhizopus.*

Conclusions. The patient received antibiotic and antifungal treatment with meropenem, vancomycin, caspofungin, and isavuconazonium. Surgery for the fungal rhinosinusitis was also required and performed. Clinical evolution was favorable, but with persistence of the cranial nerve deficits.

Keywords: mucormycosis, immunosuppression, cerebral abscess, diabetes mellitus

P039. An association between Lyme disease and peripheral facial palsy

Georgiana-ANCAVULPOI[1],
Catalina Elena BISTRICEANU[1,2],
Dan-Iulian CUCIUREANU[1,2]

[1]*Neurology Department, Faculty of Medicine, "Grigore T. Popa" University of Medicine and Pharmacy, Iasi, Romania*
[2]*Neurology Department I, "Prof. Dr. N.Oblu" Emergency Clinical Hospital, Iasi, Romania*

Borrelia burgdorferi causes Lyme disease. After skin erythema, neurological involvement is the second most common clinical manifestation. Approximately 5% of individuals with untreated erythema develop neuroborreliosis. *Borrelia burgdorferi* infections rarely cause hematologic manifestations. In this case report, we describe the case of a 48-year-old patient who presented to the neurology clinic two days after the sudden onset of facial asymmetry.

An objective neurological examination revealed a peripheral right facial palsy. A full neurological assessment showed no additional abnormalities. The general clinical examination revealed diffuse erythematous plaques over the face, trunk, and limbs that did not disappear with vitro pressure. According to the patient's recent history, she also experienced abdominal pain and vomiting.

Biological changes were noted, including an inflammatory syndrome, cholestasis syndrome, and hepatic cytolysis. The patient was referred to the hematology department for further evaluation after a diagnosis of pancytopenia. An iliac crest puncture and peripheral blood smear showed no abnormalities on the medulogram. A brain MRI (1.5T) with contrast was normal.

Laboratory investigations included testing for *Borrelia burgdorferi* antibodies, which showed significantly elevated titers: IgG 232 AU/mL (VN < 10 AU/mL) and IgM 1201 AU/mL (VN < 18 AU/mL). CSF analysis after lumbar puncture revealed no abnormalities.

The patient's condition improved with antibiotics and corticosteroids, with resolution of neurological symptoms, clearance of skin lesions, and normalization of blood counts.

Keywords: Lyme disease, facial palsy, pancytopenia, erythema, antibodies

P040. Acute thoracic spinal cord infarction – causes and the significance of DWI-MRI in diagnosis: a case report

A. ARSENE, S. BOSTAN, M. IORDACHE, S. DUMITRACHE

National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania

Introduction. Spinal cord infarction is a dramatic vascular event, accounting for approximately 1% of all stroke cases and 14–37% of all acute myelopathy cases. Diagnosis is often one of exclusion. These infarctions are frequently associated with thoraco-abdominal aortic repair surgeries, but they may also be secondary to embolism (cardiac, arterio-arterial, fibrocartilaginous), vascular malformations (true venous infarction), or may have an uncertain mechanism. Diffusion-weighted MRI (DWI-MRI) can establish the diagnosis when the clinical examination and conventional MRI sequences are inconclusive.

Methods. A 57-year-old female patient with known risk factors (hypertension, obesity, smoking, diabetes) was admitted to the neurology ward of the National Institute of Neurology and Neurovascular Diseases with sudden-onset flaccid paraplegia, total anesthesia below the T4 spinal level, urinary and bowel incontinence, absent lower limb reflexes, and bilateral Babinski signs. Initially, an inflammatory-infectious myelopathy was suspected based on conventional MRI findings. However, spinal DWI-MRI confirmed the diagnosis of spinal cord infarction. Subsequent investigations did not reveal a definitive cause, but we suspect an arterio-arterial embolism originating from vulnerable atherosclerotic plaques in the ascending aorta.

Conclusions. Spinal cord infarction is a rare but serious cause of stroke. The underlying etiologies are varied, and in some cases, a specific cause cannot be identified. This case highlights the importance of spinal DWI-MRI in the diagnostic workup of acute myelopathy.

Keywords: spinal cord infarction, spinal DWI-MRI, acute myelopathy

P041. Parkinson-plus syndromes – grand challenges in neurology

Emilia FURDU-LUNGUT[1,2], Alexandra-Silvia GIURCA[2], Stefania-Cristiana DERVESTEANU[2]

[1]*Neurology Department, CF2 Hospital, Bucharest, Romania*
[2]*"Titu Maiorescu" University, Bucharest, Romania*

Introduction. Parkinson-plus syndromes are characterized by the core symptoms of Parkinson's disease, such as bradykinesia, ataxia, resting tremor, and rigidity, but also include additional features such as dementia, cognitive deficits, and cranial nerve involvement. Among these chronic, debilitating neurodegenerative conditions is multiple system atrophy (MSA), formerly known as Shy-Drager syndrome. This progressive disorder presents with a combination of symptoms affecting both the central and autonomic nervous systems.

Case presentation. We present the case of a 73-year-old man, obese, with sleep apnea syndrome and cardiac pathology, who was admitted to the neurology department for progressive lower limb disability, gait disturbances, repeated falls without loss of consciousness, and orofacial dyskinesias.

On examination, the patient presented with severe stiffness of the trunk and limbs, a positive Noica sign, a positive Romberg sign with a tendency to fall backward, dysphagia for both liquids and solids, dysarthria, orthostatic hypotension, constipation, and cognitive impairment.

This constellation of symptoms suggested an extrapyramidal syndrome associated with Parkinson's disease; however, the lack of sustained response to levodopa raised the suspicion of a Parkinson-plus syndrome.

Conclusions. Based on the patient's history, clinical and paraclinical findings, and imaging investigations, the diagnosis of multiple system atrophy was established. In this case, the coexistence of both subtypes of multiple system atrophy, combined with significant comorbidities, contributed to the complexity of the clinical picture and posed challenges in therapeutic management.

Keywords: multiple system atrophy, orthostatic hypotension, dysphagia, dysarthria

P042. Central pontine myelinolysis – alternative causes

B-N. TEPOSU[1], P. COLTEA[1], M. MANISOR[1], C. ROMAN-FILIP[1,2]

[1]Department of Neurology, EmergencyClinical County Hospital, Sibiu, Romania
[2]"Lucian Blaga" University, Sibiu, Romania Bucharest, Romania

Introduction. Central pontine myelinolysis represents a subset of osmotic demyelination syndrome, characterized by damage to the white matter, typically in the pons, but potentially also affecting extrapontine regions such as the ventrolateral thalami, caudate nuclei, internal/external/extreme capsule, middle cerebellar peduncles, or the splenium of the corpus callosum. The most frequent cause is rapid correction of hyponatremia, but other etiologies include chronic alcohol use, protein-caloric malnutrition, hepatopathy, head radiotherapy, or hyperemesis gravidarum.

Methods. We present the case of a 41-year-old man with a prior diagnosis of mild mental retardation, left-eye convergent strabismus, generalized tonic-clonic epilepsy, and chronic alcohol use, with poor adherence to treatment. He presented with gait disturbance, impaired coordination, and worsening of his pre-existing left-eye convergent strabismus, with symptom onset three weeks prior to admission. Neurological and psychiatric evaluations were performed, and he was admitted to the neurology department for further investigation, with an initial suspicion of Wernicke encephalopathy.

Symptomatology improved following thiamine administration; however, gadolinium-enhanced brain MRI revealed a T2 and FLAIR hyperintense central pontine lesion without contrast enhancement, suggestive of a subacute metabolic etiology.

Results. With intensive vitamin supplementation, improved nutrition, and cessation of alcohol intake, neurological symptoms gradually improved. The patient was discharged after three weeks with no remaining gait disturbance or ataxia.

Conclusion. This case highlights an alternative cause of central pontine myelinolysis, unrelated to the rapid correction of hyponatremia. In this case, the etiology was likely linked to chronic alcohol use and severe protein-caloric malnutrition.

Keywords: myelinolysis, hyponatremia, alcohol use, malnutrition

P043. Etiological complexity of posterior reversible encephalopathy syndrome (PRES)

Andreea E. LUNGU[1], Ioana R. TEIRAU[1], Raluca CIOCINTA[1,2], Alexandru MICLAUSANU[1], Iulian D. CUCIUREANU[1,2]

[1]"Prof. Dr. Nicolae Oblu" Emergency Clinical Hospital, Iasi, Romania
[2]"Grigore T. Popa" University of Medicine and Pharmacy, Iasi, Romania

Introduction. Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological entity generally characterized by bilateral involvement of the parieto-occipital cortico-subcortical lobes. It is typically manifested by headache, altered mental status, seizures, visual disturbances, and other focal neurological signs, alongside reversible findings on cranial MRI. Multiple etiologies have been described, including chronic kidney disease, malignant hypertension, autoimmune diseases, eclampsia, and preeclampsia. Diabetic ketoacidosis is a rare cause of PRES, more frequently encountered in pediatric cases.

Case presentation. We present the case of a 22-year-old female with a complex medical history, including type 1 diabetes mellitus, stage 5 chronic kidney disease on hemodialysis, and hypertension. The patient suddenly developed an intense diffuse pulsatile headache with photophobia, accompanied by nausea, vomiting, and decreased visual acuity. Subsequently, she experienced a generalized tonic-clonic seizure, followed by intermittent psychomotor agitation and periods of drowsiness. On clinical examination, she had elevated blood pressure, left-sided hemiparesis, and biochemical evidence of diabetic ketoacidosis and severe azotemia.

Cranial MRI revealed multiple hyperintense FLAIR lesions with relatively symmetric distribution, suggestive of PRES. Symptomatic treatment and stabilization of glycemic levels and blood pressure resulted in a favorable clinical outcome.

Conclusion. While the pathogenesis of PRES typically involves endothelial dysfunction due to excessive proinflammatory cytokine activity, the specific mechanism linking diabetic ketoacidosis to PRES remains unclear. It is believed that elevated inflammatory cytokines in ketoacidosis stimulate endothelial cells to produce vasoactive substances, increasing vascular permeability and contributing to the development of PRES.

Given the multitude of risk factors capable of disrupting the cerebrovascular endothelium – including, albeit rarely, diabetic ketoacidosis – early recognition and etiological treatment are critical to improving outcomes in PRES.

Keywords: PRES, ketoacidosis, seizure, diabetes, headache

P044. Atypical meningioma disguising as acute stroke: importance of comprehensive diagnostic approach

C. D. OPREAN[1], G. MARIN[1], M. CRACIUN[1], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,3]

[1]*Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania*
[2]*"Carol Davila"University of Medicine and Pharmacy, Bucharest, Romania*
[3]*"Victor Babes" National Institute of Pathology, Bucharest, Romania*

We present the case of a 78-year-old patient admitted to our clinic with right-sided hemiparesis, deviation of the head and eyes to the right, right central facial palsy, and aphasia with ictal debut—initially suggestive of acute stroke. However, the initial CT scan revealed an intracranial extra-axial parietal expansive mass, without evidence of ischemic or hemorrhagic lesions. The patient experienced focal motor seizures on the right side, with secondary generalization.

Subsequent contrast-enhanced MRI confirmed the presence of the same lesion, suggestive of an atypical meningioma. After multidisciplinary discussion with the neurosurgical team, the patient underwent surgical excision of the lesion through a keyhole craniectomy. Histopathological analysis confirmed the diagnosis of an atypical meningioma.

This case underlines the importance of considering differential diagnoses in patients presenting with stroke-like symptoms. It highlights the critical role of comprehensive diagnostic evaluations, including advanced neuroimaging and electrophysiological studies, in identifying non-vascular etiologies and ensuring appropriate therapeutic interventions.

Keywords: atypical meningioma, stroke, alternative diagnoses

P045. Reversible cerebral vasoconstriction syndrome – diagnostic and etiology

Bogdan POPA-IACOB[1], Florin-Adrian STIUBEA[1], Monica SABAU[1,2]

[1]*Department of Neurology, Neurology 1 Clinic, Bihor Emergency Clinical County Hospital, Oradea, Romania*
[2]*Faculty of Medicine and Farmacy, University of Oradea, Oradea, Romania*

Introduction. Reversible cerebral vasoconstriction syndrome (RCVS) is a rare disorder characterized by reversible vasoconstriction of cerebral arteries that can result in cerebral edema and ischemia, subarachnoid and lobar hemorrhage, clinically manifested by thunderclap headaches, focal neurological deficits, and seizures.

Methods and material. A patient was diagnosed with RCVS (RCVS2 score of 8 points) at the Bihor Emergency County Clinical Hospital in 2021. The patient's gender, symptoms, disease course, and factors predisposing to cerebral vasoconstriction were taken into account.

Results. A 50-year-old patient was transferred from the hematology ward, where she had received repeated transfusions for anemia and severe thrombocytopenia. She had no cardiovascular risk factors. Clinically, she presented with thunderclap headache, left hemiparesis, left homonymous hemianopsia, and confusional state. Brain imaging (CT, ANGIO-CT, MRI, and MRA) revealed right temporo-occipital edema, multiple areas of ischemia with a bilateral borderline character, and multiple pre- and post-polygonal cerebral vessel narrowings. The evolution was favorable with nimodipine administration. Shortly after, within a few days, the motor deficit reappeared. Cranial CT showed hemorrhagic transformation of one of the previously highlighted ischemic lesions, subarachnoid hemorrhage, and on repeat cerebral ANGIO-CT, the cerebral vessels appeared normal.

Conclusions. In the context of thunderclap headache, multiple ischemic lesions, subarachnoid hemorrhage, as well as transient narrowing of cerebral vessels evident on brain imaging, it is interpreted as a reversible cerebral vasoconstriction syndrome most likely triggered by repeated transfusions.

Keywords: reversible cerebral vasoconstriction syndrome, thunderclap headache, transfusion

P046. Management of paradoxical embolism in a young woman with patent foramen ovale: a comprehensive medical approach

C. D. OPREAN[1], G. MARIN[1], A. MUJDABA-ELMI[1], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,3]

[1]Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania
[2]"Carol Davila"University of Medicine and Pharmacy, Bucharest, Romania
[3]"Victor Babes" National Institute of Pathology, Bucharest, Romania

A patent foramen ovale (PFO) is a prevalent condition characterized by a small, flap-like aperture between the atria of the heart that fails to close properly after birth. While many individuals with PFO remain asymptomatic and do not require treatment, others may experience potential health risks. Of notable significance is its link to paradoxical embolism, where a blood clot from elsewhere in the body can traverse the PFO, leading to cerebral embolism and subsequent stroke. PFOs have also been implicated in certain types of migraines and other neurological disorders.

We report the case of an 18-year-old patient who experienced two episodes of transient neurological symptoms associated with headache. The initial episode lasted 25 minutes and presented with right-sided hemiparesis accompanied by unilateral headache. The second episode occurred four years later, featuring motor aphasia accompanied by holocranial headache, with pain remission coinciding with the onset of aphasia, which lasted 4 hours.

Following cerebral imaging via MRI, which returned normal results, and a hereditary thrombophilia work-up with some consequential changes, a microbubble TCD and comprehensive cardiological evaluation were conducted, revealing a tunnel-shaped patent foramen ovale and interatrial septal aneurysm. A multidisciplinary team was assembled, and a minimally invasive procedure was performed to close the patent foramen ovale using an Amplatzer device.

In conclusion, thorough and interdisciplinary evaluation is crucial when assessing young patients presenting with focal neurological deficits.

Keywords: patent foramen ovale, neurological symptoms, paradoxical embolism

P047. Progressive supranuclear palsy – predominant Parkinson syndrome (PSP-P) – diagnosis and future management

Bogdan POPA-IACOB[1], Daiana Alexandra LUPUT[1], Adriana Elena BADEA[1], Monica SABAU[1,2]

[1]Department of Neurology, Neurology 1 Clinic, Bihor Emergency Clinical County Hospital, Oradea, Romania
[2]Faculty of Medicine and Farmacy, University of Oradea, Oradea, Romania

Introduction. Progressive supranuclear palsy (PSP) is a neurodegenerative disease characterized by parkinsonism, supranuclear gaze palsy, recurrent falls, and cognitive decline. Although Richardson syndrome is the classical presentation, the predominantly parkinsonian syndrome (PSP-P) should not be neglected when diagnosing the patient because of the difficulty in differentiating it from Parkinson disease (PD).

Material and methods. One patient was diagnosed with probable PSP-P (according to the Movement Disorder Society criteria for diagnosing PSP-2017) at The Clinical County Emergency Hospital in Bihor, Romania in 2023. The symptoms, course of the disease, cranial imaging, and predominant clinical features were assessed regarding the different PSP phenotypes.

Results. We present a 68-year-old male patient diagnosed with PD and referred to our center for continuous dopaminergic stimulation therapy. Discussion with the patient reveals that he has never had a significant therapeutic benefit from levodopa therapy. Clinically, the patient presents with severe axial akinetic-rigid syndrome, paralysis of combined upright movements, positive retropulsion test, and freezing of gait. MMSE: 19 points. Brain MRI examination shows highly suggestive images for PSP: Humming Bird sign. Thus, the patient meets the diagnostic criteria for PSP-P mentioned above, and therefore, we decided not to pursue advanced therapies.

Conclusion. Continuous dopaminergic stimulation therapy is not recommended for patients with PSP-P because the outcome and prognosis are unfavorable.

Keywords: progressive supranuclear palsy, parkinsonism, advanced aided therapies, diagnosis

P048. Autonomic neuropathy after COVID vaccine

L. E. CUCU[1], C. GATCAN[1], T. G. SCHREINER[2,3], L. BIRSANU1, A. ROTAR[1], B. E. IGNAT[1,2]

[1]*Department of Neurology, Clinical Rehabilitation Hospital, Iasi, Romania*
[2]*Department "Medicale III", Faculty of Medicine, "Gr. T. Popa" University of Medicine and Pharmacy, Iasi, Romania*
[3]*First Neurology Clinic, "Prof. Dr. N. Oblu" Clinical Emergency Hospital, Iasi, Romania*

Introduction. Autonomic neuropathies are a group of pathologies characterized by selective damage to small, poorly myelinated, or unmyelinated autonomic nerve fibers. The clinical picture is diverse, resulting from the involvement of the cardiovascular, gastrointestinal, genitourinary, sudomotor, and pupillomotor systems, with various combinations of symptoms. Although autonomic neuropathies most frequently have a chronic onset, often in the context of diabetes or neurodegenerative diseases, there are also cases with acute or subacute onset secondary to viral infections or, more rarely, vaccines, such as the one against the SARS-CoV-2 virus.

Case presentation. We present the case of a 58-year-old patient with no significant personal history who, two months after receiving the second dose of the SARS-CoV-2 vaccine, developed fatigue, dizziness, anxiety, hypertensive episodes, and multiple hospitalizations in the department of internal medicine for repeated hyposodium. The patient was consulted by specialists in cardiology, endocrinology, rheumatology, and oncology, all of whom excluded any specific cause. Finally, a neurological consultation, electroneuromyographic examination, sympathetic skin response (SSR) tests, and SUDOSCAN established the diagnosis of autonomic neuropathy. The temporal relationship with the administration of the vaccine and the absence of other paraclinical changes suggest that the symptoms could be secondary to sequelae of possibly postvaccinal postradiculoneuritis.

Conclusions. Patients with autonomic neuropathies present a diverse and varied symptomatology, which can raise diagnostic and treatment challenges. Along with classic etiologies, rarer causes, such as post-vaccination reactions, should also be considered.

Keywords: autonomic neuropathy, vaccine, COVID-19, sympathetic skin response, SUDOSCAN

P049. Have we just thrombolysed fever? A step-by-step approach in diagnosis of CNS lupus

A. M. FLOREA[1,2], O. E. BRISCAN[1,2], O. V. PANAITESCU[1,2], A. PREDA[1,2], D. G. LUCA[1], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,3], V. M. POMPILIAN[2,4], S. A. ZURAC[2,5]

[1]*Neurology Department, Colentina Clinical Hospital, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*CellBiology Laboratory, Neurosciences and Experimental Myology, National Institute of Pathology "Victor Babes", Bucharest, Romania*
[4]*3rd Internal Medicine Department, Colentina Clinical Hospital, Bucharest, Romania*
[5]*Pathology Department, Colentina Clinical Hospital, Bucharest, Romania*

Purpose. Systemic lupus erythematosus (SLE), considered to be one of the great masqueraders, is an autoimmune connective tissue disorder that may have multisystemic involvement, including the central nervous system. Aseptic meningoencephalitis as the onset of SLE requires a high index of suspicion.

Material and methods. We present the case of a 66-year-old female patient, admitted to the Dermatology Department for largely distributed erythematous-squamous plaques, who was referred to our department for sudden onset aphasia. Clinical examination revealed a core temperature of 40.2°C, while neurological examination revealed superior limb pronation and aphasia, which persisted despite antipyretic treatment and fever management. In the absence of any contraindications, thrombolytic therapy was administered. Neurological evolution was fluctuating during the first 24 hours post-thrombolysis, outlining global aphasia with anomia elements and verbal paraphasia, while the CT scans excluded any vascular lesions. Consecutively, the differential diagnosis of an inflammatory/infectious process involving the CNS was considered.

Results. Further investigations comprising MRI, EEG, and lumbar puncture were consistent with the diagnosis of aseptic meningoencephalitis. Dynamic monitoring and serial investigations led to fulfillment of the EULAR criteria and the diagnosis of subacute SLE.

Conclusions. This case highlights the time factor, which can be both a blessing and a curse when facing a patient with a newly developed neurological deficit. On one hand, there is the urgency of rapid intervention, and on the other, the necessity for dynamic monitoring, a comprehensive, and multidisciplinary approach to uncover the underlying diagnosis.

Keywords: aphasia, SLE, aseptic meningo-encephalitis

P050. Anti-titin antibodies associated opsoclonus-myoclonus-ataxia syndrome – a case report

S. PETRESCU, D. RUSU , D. MOROSANU, N. GRECU, C. TIU

University Emergency Hospital of Bucharest, Bucharest, Romania

Introduction. Opsoclonus-myoclonus-ataxia syndrome (OMAS) represents a rare neurological condition defined by uncontrolled, arrhythmic, multidirectional eye movements, involuntary muscle jerks, and ataxia. The leading causes of adult-onset OMAS are usually classified as paraneoplastic, postinfectious, toxic-metabolic, or idiopathic; lung cancer, particularly small-cell lung carcinoma, breast, and ovarian cancer are the types most frequently associated with it. While early diagnosis and treatment of the underlying cancer are crucial for the effective management of this syndrome, symptomatic benefit can be obtained through immunosuppressant or immunomodulatory therapy.

Methods. We present the case of a 65-year-old woman with a history of multiple cancers (ovarian 14 years prior, and breast, 2 years before presentation) who developed OMAS, including behavioral changes. Magnetic resonance imaging revealed a non-enhancing cerebellar FLAIR and DWI hyperintensity, with a normal ADC, limited to the nodulus. Diagnostic workup included cerebrospinal fluid analysis, serum antineuronal antibodies, and cancer screening (CT scan of the thorax, abdomen, and pelvis), all within normal range except for the presence of anti-titin antibodies.

Results. The patient received 1g/day of methylprednisolone pulse therapy and 2g/kg of intravenous immunoglobulins, which resulted in significant improvement of symptoms and imaging features.

Conclusion. To our knowledge, this is the first report of anti-titin associated OMAS. Given the known association between breast cancer and anti-titin antibodies, the imaging changes consistent with both the clinical picture and titin expression in the cerebellum, and the response to treatment, we believe that there is a valid association between these antibodies and this rare syndrome.

P051. Vestibular syndrome in a cannabis smoking young patient

Mihaela Corina POSIRCA, Camelia LIPAN, Nicoleta LUPASCU, Rodica GHEORGHE, Titus Mihai VASILE

Neurology Clinic, "Dr. Carol Davila" Central Military Emergency University Hospital, Bucharest, Romania

Introduction. In studies, we found that cannabis users showed a lot of complications, frequently presented high intraocular pressure, longer dark-adaptation periods, reduced color discrimination, increased lacrimation, headache, tinnitus, vertigo, and hearing loss.

Methods. The present work is about a 19-year-old patient, without medical history, cannabis smoker, with a recent respiratory infection, who presented at the emergency room for headache, blurred vision, and dizziness that appeared a few days before. The first neurological examination revealed postural instability and horizontal nystagmus. He was diagnosed and treated as having vestibular syndrome, and the complications of using cannabis were also discussed. 24 hours later, the neurological examination revealed gaze-evoked nystagmus and right hemiparesis.

Results. The results of imagistic evaluation (showed supra- and infratentorial white matter high T2/FLAIR signal abnormalities, involving fronto-temporo-parietal regions, on both sides; the brainstem and midbrain were also involved; none of these lesions showed gadolinium enhancement), neurological examination, blood sample, and lumbar puncture (oligoclonal band negative), and the positive response to corticosteroids led to the diagnosis of acute disseminated encephalomyelitis (ADEM).

Conclusions. ADEM is a rare autoimmune demyelinating disorder that mainly affects the central nervous system and is characterized by an acute inflammatory response targeting the myelin sheath surrounding fibers in the brain and spinal cord. It often appears following a viral or bacterial infection or, rarely, a vaccination. This case report emphasizes that ADEM diagnosis relies on clinical and imaging findings. The early recognition, accurate diagnosis of ADEM, and prompt initiation of appropriate treatment facilitate timely management and reduced neurological damage.

P052. Dorsal medullary glioblastoma associated with secondary cervical syringomyelia: A case report

C. GATCAN, L. CUCU, T. G. SCHREINER, A. ROTAR, B. E. IGNAT

Clinical Recovery Hospital, "Gr. T. Popa" University of Medicine and Pharmacy, Iasi, Romania

Introduction. Among primary tumors of the spinal cord, glioblastoma multiforme (GBM) represents a rare subtype, making up less than 2% of all spinal tumors, with a predilection for the cervical and cervical-dorsal region and a reserved prognosis.

Case presentation. We present the case of a 19-year-old patient who initially experienced dorsalgia followed by a motor deficit in the left lower limb, later also in the right lower limb. After about 2 months, sphincter disturbances appeared, and the patient became paraplegic. Cervical-dorsal spine MRI examination with contrast agent depicted a solid mass at the level of the spinal cord T2-T6 and cervical syringomyelia. He underwent neurosurgical intervention, and the histopathological examination revealed „wild type" glioblastoma, grade IV. The treatment consisted of concurrent radio-chemotherapy, followed by cycles of temozolomide monotherapy in adjusted doses. Even though the control neuroimaging investigations performed at 3 and 6 months postoperatively described a possible relapse and slight progression, the MRI examination at 9 months showed a minimal involution of the tumor lesion in the context of a stationary neurological status.

Conclusions. Although the neurological symptomatology (sensory, motor, sphincteric disturbances) is similar to other spinal pathologies, a cause can also be a rare tumor such as medullary GBM. Multidisciplinary management, supportive and recovery therapy, prevention and management of potential complications are essential and can prolong life expectancy.

Keywords: medullary glioblastoma multiforme, secondary syringomyelia, radio-chemotherapy

P053. An unusual presentation of carotid artery stenosis: Limb-shaking transient ischemic attack

Mihai-Ioan DUMITREASA[1], Sanda PETRU TIU[1], Anca MOTATAIANU[1,2], Rodica BALASA[1,2,3]

[1]Ist Neurology Clinic, Mures County Clinical Emergency Hospital, Targu Mures, Romania
[2]Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania
[3]Doctoral School, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania

Background. Transient ischemic attack (TIA) is an acute temporary focal neurologic dysfunction caused by brain ischemia, which usually presents as motor, sensory, speech, or visual deficit. In rare cases, this pathology can manifest as paroxysmal involuntary movements as a consequence of severe carotid stenosis, often misdiagnosed as focal seizures.

Case report. We present the case of a 71-year-old female with a history of hypertension, diabetes, and atrial fibrillation, who underwent transcatheter aortic valve replacement and presented multiple episodes of clonic involuntary movements of the left limbs. She also reported three episodes of loss of consciousness without convulsions. She was admitted to the hospital without any motor deficit. The patient first underwent a cerebral CT scan, which showed multiple lacunar chronic infarcts. An electroencephalography (EEG) was also performed, which did not show any abnormalities, apart from a slower background rhythm on the right cerebral hemisphere. The carotid Doppler ultrasound was useful for the diagnosis, as severe right carotid artery stenosis (>90%) was observed, and the patient was transferred to the vascular surgery department where she underwent right carotid endarterectomy with favorable evolution afterward.

Conclusion. Limb-shaking transient ischemic attacks are a rare manifestation of severe carotid artery stenosis and should be carefully investigated, as they can often be misdiagnosed as focal seizures. The correct diagnosis is crucial for the prevention of ischemic stroke.

Keywords: limb-shaking, transient ischemic attack, carotid stenosis, ischemic stroke, focal seizures, endarterectomy

P054. Sumatriptan-induced reversible cerebral vasoconstriction syndrome: A case report highlighting diagnostic and management challenges

A. PREDA[1,2], O. E. BRISCAN[1,2], A. M. FLOREA[1,2], O. V. PANAITESCU[1,2], D. G. LUCA[1], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,3]

[1]Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Cell Biology Laboratory, Neurosciences and Experimental Myology, National Institute of Pathology "Victor Babes", Bucharest, Romania

Introduction. Reversible cerebral vasoconstriction syndrome (RCVS) is usually a benign condition characterized by reversible narrowing of the cerebral arteries, often leading to thunderclap headaches and, occasionally, neurological deficits due to brain edema, ischemia, hemorrhage, or seizures. This syndrome is associated with various conditions, including migraine, vasoconstrictive drug use, cervical artery dissection, unruptured saccular aneurysms, and cerebral venous thrombosis, among others.

Methods. We present the case of a 42-year-old female with a history of chronic migraine, who experienced a thunderclap headache and right hemiparesis shortly after receiving her third dose of Sumatriptan within a 24-hour period. The patient arrived at our clinic one hour after symptom onset, qualifying her as a potential candidate for thrombolytic therapy.

Results. Cerebral and supra-aortic computed tomography angiography revealed no signs of acute ischemic or hemorrhagic lesions, cerebral aneurysms, or arterial or venous filling defects. Brain magnetic resonance imaging, including diffusion-weighted imaging and apparent diffusion coefficient sequences obtained two hours after symptom onset, showed no abnormalities. Cerebrospinal fluid analysis yielded normal results. Transcranial Doppler examination demonstrated normal velocities. Management involved supportive measures, resulting in the remission of neurological deficits within hours. The patient was subsequently prescribed Galcanezumab, given the contraindication to use triptans.

Conclusion. This case underscores the importance of considering RCVS in patients presenting with thunderclap headaches and neurological deficits, particularly in the context of recent vasoconstrictive medication use. Normal imaging findings do not exclude the diagnosis, and prompt recognition is crucial for appropriate management.

Keywords: RCVS, Sumatriptan, thunderclap headache

P055. ANCA vasculitis with sensitive and motor polyneuropathy – presentation of 2 clinical cases

Emilia COSOFRET[1], Oana AILIOAIE[2]

[1]SanMed Medical Center, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. ANCA vasculitis is a multisystem disease with renal, pulmonary, cardiac, and neurologic involvement. We present 2 cases of ANCA vasculitis with peripheral polyneuropathy.

Case presentation. A 65-year-old African male with no significant past medical history was admitted for acute renal failure, microscopic hematuria, proteinuria 2 g/g, and bilateral paresthesia of the lower limbs. ANCA PR3 was positive. Renal biopsy showed acute glomerulonephritis with crescents. EMG showed acute sensitive and motor neuropathy compatible with vasculitis. He was treated with cyclophosphamide and corticosteroids, followed by Rituximab. He developed insulin-dependent diabetes mellitus. The clinical course was favorable. ANCA remained positive.

A 56-year-old Caucasian male with no significant medical history presented 3 months ago with acute dyspnea, hemoptysis, acute renal failure (creatinine 6 mg/l), and lower limb paresthesia without motor deficit. ANCA MPO was positive. Pulmonary scan showed bilateral alveolar bleeding. Renal biopsy showed acute extracapillary glomerulonephritis with crescents. EMG showed sensitive and motor neuropathy compatible with vasculitis. He was admitted to Intensive Care for plasmapheresis and hemodialysis, followed by treatment with cyclophosphamide, corticosteroids, and Rituximab. Renal function recovered. ANCA became negative. Repeated EMG showed significant recovery of peripheral neuropathy.

Conclusion. These 2 cases of acute peripheral neuropathy highlight the importance of EMG screening for neurological involvement in ANCA vasculitis.

P056. Ischemic stroke caused by cerebral vasculitis as initial manifestation of systemic lupus erythematosus (SLE) – case presentation

O. B. BALINT[1], C. SALAGEAN[1], R. SZABO[1], M. SABAU[1,2]

[1]Departament of Neurology, Bihor County Emergency Clinical Hospital, Oradea, Romania
[2]Faculty of Medicine and Pharmacy, University of Oradea, Oradea, Romania

Introduction. Cerebral infarction occurs in 19% of patients with SLE, and rarely, it can be the first manifestation of the disease.

Method. We present a young patient with cerebral infarction as the first manifestation of SLE caused by cerebral vasculitis.

Results. A 33-year-old female patient with recurrent episodes of moderate headache for several weeks presented to the Emergency Department with a sudden onset left-sided motor deficit, which had occurred the day before presentation. The onset and nature of the deficit suggested an acute cerebrovascular event. Initial imaging examinations (CT scan and MRI) showed multiple lesions: an ischemic lesion in the right MCA territory and several smaller lesions, the nature of which could not be specified. To elucidate the etiology of the cerebrovascular event, we performed Holter EKG, echocardiography, and carotid Doppler ultrasound, but no pathological findings were detected. MRA suggested cerebral vasculitis. We also performed MRI spectroscopy, which established the ischemic nature of all the lesions. Laboratory examinations confirmed SLE, and immunosuppressive treatment was started, followed by improvement of the neurological deficit.

Conclusion. Cerebral infarction in young people often requires complex investigations and an interdisciplinary approach to elucidate the etiology.

Keywords: systemic lupus erythematosus, ischemic stroke, secondary cerebral vasculitis

P057. Splenic infarction, complication after intravenous immunoglobulin administration: Case presentation

F. A. STIUBEA, M. SABAU, I. B. POPA

Bihor County Emergency Clinical Hospital, Faculty of Medicine and Pharmacy, University of Oradea, Oradea, Romania

Introduction. Adverse effects associated with intravenous immunoglobulin (IVIg) treatment are usually mild and transient. These include changes in blood pressure, tachycardia, mild flu-like symptoms, and headache. Severe side effects are rare and include renal failure, aseptic meningitis, acute anaphylactic reactions, hemolytic anemia, and thromboembolic events.

Method. We present a 44-year-old patient, known to have high blood pressure, type II diabetes mellitus, and grade III obesity, who was diagnosed with acute polyradiculoneuritis and was undergoing IVIg therapy. She presented after initiation of therapy with splenic infarction.

Results. The patient presented to the emergency department with paresthesia followed by motor deficit in the lower limbs, which had been ongoing for approximately two weeks. Upon examination, the diagnosis of polyradiculoneuritis was established. Treatment with IVIg was initiated, and therapeutic management of associated conditions was performed, including antidiabetic, antihypertensive, and low molecular weight heparin therapy. Seventy-two to ninety-six hours after initiation of immunoglobulin treatment, the patient developed pain and tenderness on palpation in the upper abdominal wall, along with nausea and vomiting. Imaging studies revealed mid-segment splenic artery thrombosis and extensive perihilar splenic infarction. Further investigations to determine the etiology of the splenic infarction were negative. The splenic infarction was interpreted as a complication of IVIg treatment.

Conclusions. Thrombotic events may occur in rare cases following IVIg therapy. It is therefore necessary to assess the risk factors for thromboembolic events and to apply prophylactic measures for all patients undergoing IVIg therapy.

Keywords: immunoglobulins, splenic infarction, thrombosis, Guillain-Barré

P058. Percheron artery infarction: A rare cause of acute confusion

O. TOTH-BASA[1], A. CENTEA[2], M. D. OLIVIU[2], L. PERJU-DUMBRAVA[1,2]

[1]Discipline of Neurology and Pediatric Neurology, Neuroscience Department, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

[2]Neurology Department, Emergency County Clinical Hospital Cluj-Napoca, Cluj-Napoca, Romania

Percheron artery infarction can be challenging to diagnose in the emergency room due to its heterogeneous clinical manifestations. It can cause alterations in consciousness, seizures, and confusion, which can easily be mistaken for encephalitis or intoxication.

We report a case of acute confusion caused by Percheron artery occlusion.

A 50-year-old patient, with no known cardiovascular, cerebrovascular, or psychiatric pathology, was brought to the emergency room for hypersomnolence and confusion that began a day before presentation. The patient had normal blood pressure and did not present with fever. The family denied any exposure to toxic agents. Neurological examination revealed a confused, disoriented patient with episodes of psychomotor agitation. No classical cardiovascular risk factors, metabolic abnormalities, or clinical or laboratory signs of infection were found. A native brain CT scan showed small hypodense lesions bilaterally in the thalamus. Magnetic resonance angiography revealed recent bithalamic ischemic lesions corresponding to the Percheron artery territory.

When faced with a patient presenting with acute confusion and small lacunar infarctions, even in the absence of classical cardiovascular risk factors, Percheron artery infarction should be considered as a possible cause of nonspecific symptoms such as confusion and agitation.

P059. Ischemic stroke through large vessel occlusion in the context of antiphospholipid syndrome: case presentation

F.A. STIUBEA, A. COMANESCU, M. SABAU, I. PASCA

Bihor County Emergency Clinical Hospital, Faculty of Medicine and Pharmacy, University of Oradea, Oradea, Romania

Antiphospholipid syndrome is a systemic autoimmune disease characterized by hypercoagulability, which leads to arterial and/or venous thrombosis and obstetric complications. It is caused by the presence of antiphospholipid antibodies such as lupus anticoagulant, anti-β2-glycoprotein I, and/or anticardiolipin antibodies. Neurological events associated with antiphospholipid syndrome are relatively common, but ischemic stroke due to large vessel occlusion is a rare manifestation.

Method. We present a 39-year-old female patient diagnosed with systemic lupus erythematosus, who presented to the emergency department with ischemic stroke caused by large vessel occlusion. The patient's medical history and paraclinical examinations revealed antiphospholipid syndrome as the underlying cause.

Results. Emergency revascularization treatment via mechanical thrombectomy was performed. Cranial contrast-enhanced MRI, 24-hour Holter ECG, transthoracic echocardiography, and carotid ultrasound were conducted. The ANA profile was assessed, along with antibodies for anti-Ku, AMA-M2, anti-nucleosome, anti-P ribosomal (intensely positive), anti-histone (weakly positive), anti-DNA (double-stranded positive), and lupus anticoagulant (positive at three consecutive determinations performed more than 12 weeks apart). The patient also had a history of deep venous thrombosis in the lower limb and an evolving arrested pregnancy one year prior. The patient was prescribed anticoagulant and antiplatelet therapy according to EULAR guidelines.

Conclusion. Following laboratory investigations, imaging, and documentation of the medical history, the EULAR criteria for the classification of antiphospholipid syndrome were met. Lupus anticoagulant is frequently associated with cerebral thrombotic phenomena, ranging from microvascular events to large vessel cerebral thrombosis, although the latter is rare.

Keywords: antiphospholipid syndrome, large vessel, stroke

P060. AQP4-Ab seronegative NMOSD with positive MOG-Ab: case presentation

C. SALAGEAN[1], O. B. BÁLINT[1], A. E. BADEA[1], M. SABAU[1,2]

[1]Department of Neurology, Bihor County Emergency Clinical Hospital, Oradea, Romania
[2]Faculty of Medicine and Pharmacy, University of Oradea, Oradea, Romania

Introduction. Although rarely, anti-myelin oligodendrocyte glycoprotein (anti-MOG) antibodies can be present in seronegative neuromyelitis optica spectrum disorder (NMOSD) patients, and screening could provide an accurate diagnosis and treatment.

Method. A 23-year-old female presented to our hospital with sudden lower limb weakness and urinary retention. Neurological examination revealed MRC 0/5 in the lower limbs, T6 sensory level, and left Babinski sign.

Results. Upon admission, a lumbar puncture was performed, revealing 11 cells/mm^3 in the cerebrospinal fluid, the majority of which were lymphocytes. The Meningitis/Encephalitis (ME) Panel was negative. Magnetic resonance imaging (MRI) with contrast of the cervical and thoracic spine showed hyperintense lesions on T2 at C1-C5 and T1-T6. Brain MRI with contrast showed a non-contrast-enhancing oval lesion on the right frontal-parietal region in T2/FLAIR and mild bilateral optic nerve edema. Blood tests showed neutrophilic leukocytosis, a negative aquaporin-4 IgG test, and positive anti-MOG antibodies (titer 1:80). Using the International Panel for NMO Diagnosis (IPND) criteria from 2015, along with clinical, neuroimaging, and laboratory findings, the final diagnosis was AQP4-Ab seronegative NMOSD with MOG-Ab. The patient received methylprednisolone and physiotherapy and made a full recovery.

Conclusion. We report a rare case of seronegative AQP4-IgG antibody NMOSD with positive anti-MOG antibodies, presenting with extensive transverse myelitis and subclinical bilateral optic neuritis.

Keywords: neuromyelitis optica spectrum disorder, anti-MOG antibodies, lower limb weakness, transverse myelitis, optic neuritis

P061. The correlation between multiple sclerosis and ankylosing spondylitis – challenges in evaluation and management

M. VICOL, R. CRETU, G. APOSTOL, M. HOHAN, B. HODOROGEA, D. CARP, D. ALEXA

Neurology Clinic, Rehabilitation Hospital, Iasi, Romania
"Gr. T. Popa" University of Medicine and Farmacy, Iasi, Romania

Introduction. Multiple sclerosis (MS) is a neurodegenerative disorder affecting the central nervous system. It has been reported to be associated with several autoimmune pathological disorders. There is little documentation regarding the simultaneous occurrence of ankylosing spondylitis and multiple sclerosis in a single patient. Successfully managing these patients constitutes a significant challenge.

Materials and methods. A 38-year-old male patient was diagnosed with ankylosing spondylitis in 2019 following symptoms of back pain and stiffness, as well as a positive laboratory test for HLA B27. The patient received treatment with non-steroidal anti-inflammatory drugs (NSAIDs) prior to the initiation of secukinumab therapy. The primary development of neurological symptoms occurred in 2023, characterized by the presence of numbness and weakness in the left extremities. The brain MRI scan revealed the presence of several demyelinating sites in the periventricular, juxtacortical, and infratentorial regions, which did not exhibit any contrast enhancement. The patient was diagnosed with MS according to the revised McDonald criteria and immediately received treatment with Glatiramer acetate. Throughout a one-year period of treatment, the patient did not experience any relapses or the appearance of new lesions on MRI.

Conclusions. Due to a lack of cases linked to these two diseases, it is essential to properly identify each condition. The simultaneous presence of these factors significantly influences the treatment strategy; nevertheless, there is currently a lack of scientifically supported recommendations for the treatment of these patients.

Keywords: ankylosing spondylitis, multiple sclerosis, secukinumab

P062. Bilateral vocal cord paralysis mistaken as a conversion disorder: A prodrome for multiple system atrophy (MSA) – A case report

S. R. SIMION[1,2], L. I. OLTEANU[1,2], L. M. SELESCU[1], S. N. NICA[1,2]
[1]*Neurology Department, Colentina Clinical Hospital, Bucharest, Romania*
[2]*"Carol Davila", University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Multiple system atrophy (MSA) is a progressive synucleinopathy characterized by a wide range of clinical features, including dysautonomia, parkinsonism, cerebellar ataxia, and pyramidal signs.

Methods. We present a case of a 53-year-old female with major depressive disorder under psychiatric treatment. An episode of acute respiratory failure in 2017, preceded by multiple stridor episodes, led to permanent tracheostomy due to vocal cord adduction paralysis, initially diagnosed as conversion disorder. Subsequent deterioration was attributed to dysautonomia.

Results. Diagnostic workup revealed dysautonomia with severe orthostatic hypotension, cerebellar and pyramidal signs, parkinsonian features, and vocal cord paresis, suggesting a neurodegenerative process. Brain MRI showed hyperintense T2 lesions in the cerebellar peduncle and the pons along with the characteristic "eye of the tiger" sign in the basal ganglia. Vocal cord electromyography demonstrated bilateral abductor vocal fold paralysis. Clinically probable MSA was diagnosed based on specific criteria. We initiated symptomatic treatment for orthostatic hypotension but had a poor response.

Conclusions. MSA's clinical presentation can mimic other neurodegenerative diseases, with special consideration of differential diagnoses. Stridor episodes in the context of cerebellar ataxia or parkinsonism should raise suspicion for MSA.

Keywords: multiple system atrophy (MSA), bilateral vocal cord paralysis, conversion disorder

P063. Stroke concomitant with hidden cerebral metastases

Teodora CIUPAG, Anca MOTATAIANU, Rodica BALASA
[1]*Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania*
[2]*Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania*

Background. Cancer-associated thrombosis is one of the leading causes of morbidity and mortality for cancer patients. Patients can present with venous thromboembolism, like pulmonary embolism or deep venous thrombosis, and less likely as arterial thromboembolism, such as ischemic stroke and myocardial infarction. The majority of deaths in patients with cancer-associated thrombosis are related to arterial thrombosis rather than venous thromboembolism. Thromboembolic disease can be the first manifestation of occult malignancy.

Case presentation. A 62-year-old man with no significant medical history developed sudden motor deficit on the left side of the body. He presented at the emergency department with left central facial palsy, left hemiparesis, and moderate dysarthria. The brain CT examination revealed no lesion or changes. Being within the therapeutic window, he received thrombolytic treatment with Actilyse, which led to slight improvement. In the next 24 hours, he developed motor deficit and contralateral central facial palsy. The brain CT revealed multiple intracerebral hematomas located in the left temporal-parietal and right frontal-parietal regions. Other paraclinical investigations showed iron deficiency anemia, elevated PSA serum levels above normal range, and positive inflammatory tests. A whole-body CT was performed, revealing multiple infarcts at the splenic and renal levels, along with a urinary bladder displaying a thickened wall with heterogeneous contrast enhancement and an enlarged prostate with an irregular structure. Additionally, bone lesions were detected at the vertebral bodies, raising suspicion of secondary bone metastases. Brain MRI imaging performed in the following days revealed bilateral frontal and left temporal brain hematomas, lesions with mass effect, perilesional edema, and gadolinium enhancement with a ring-like pattern, suggestive of secondary hemorrhagic brain metastases.

Conclusions. This case highlights a complex scenario of a patient with no prior medical history or symptoms, presenting with thrombosis associated with cancer, alongside cerebral, spleen, and renal infarcts, concurrently with secondary bone and cerebral metastases.

Keywords: brain metastases, prostate cancer, thrombolysis, associated cancer

P064. Isaacs syndrome in young adult with thymic hyperplasia and LG1 and LRP4 antibodies – a rare clinical case

L. I. OLTEANU[1,2], S. R. SIMION[1,2], L. M. SELESCU[1], S. M. NICA[1,2]

[1]Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]"Carol Davila", University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Isaacs syndrome is a rare condition with genetic, autoimmune, or paraneoplastic causes, characterized by hyperexcitability of peripheral nerves. It can manifest as muscle cramps, hyperhidrosis, muscle fasciculations, and hypertrophy.

Methods. We present the case of a 27-year-old man whose symptoms began in March 2023 with bilateral plantar paresthesia and sweating, followed by generalized spontaneous fasciculations predominantly in the calf muscles and secondary myalgia.

Results. Electromyographic studies revealed spontaneous discharges of motor unit potentials, compatible with myokymic and neuromyotonic patterns. Muscle biopsy showed minimal changes, and calf MRI was normal. Chest CT scan exhibited a thymic mass, and postoperative histopathological examination indicated thymic hyperplasia. A rheumatological autoimmune panel was negative. Serum studies showed high creatine kinase (CK) levels, elevated titers of anti-voltage-gated potassium-channel antibodies (VGKC), anti-leucine-rich-glioma-inactivated-1 (LG1), and low-density-lipoprotein receptor-related-protein-4 (LRP4) antibodies, thus establishing the final diagnosis of Isaacs syndrome. The patient underwent treatment with carbamazepine, intravenous immunoglobulins, and tapered corticosteroid therapy, resulting in remission of muscular cramps but persistence of fasciculations.

Conclusions. Isaacs syndrome could be a neuromuscular manifestation of an exclusively autoimmune cause associated with thymic hyperplasia and not linked with a paraneoplastic syndrome. Even though LRP4 antibodies were positive, the patient did not exhibit any clinical phenotype of myasthenia gravis.

Keywords: Isaacs, neuromyotonia, fasciculations, thymic hyperplasia

P065. Subacute polyneuropathy observed in a patient with microscopic polyangiitis: case presentation

Maria COSOFRET[1], Smaranda MAIER[1,2], Zoltan BAJKO[1,2], Rodica BALASA[1,2]

[1]Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania
[2]Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania

Background. Microscopic polyangiitis is a systemic autoimmune disease primarily affecting the kidneys (>80%), often involving the lungs (30%), nervous system (20-50% peripheral nervous system, 5-15% central nervous system), or skin (10-60%). Characteristic features include focal segmental glomerulonephritis, nongranulomatous necrotizing vasculitis, and positivity for perinuclear antineutrophil cytoplasmic antibodies (p-ANCA) in serum.

Case presentation. We present the case of a 69-year-old female patient with microscopic polyangiitis presenting with polyneuropathy, renal, and pulmonary involvement. Disease onset manifested with pain and sensory symptoms followed by progressive weakness in both legs. Neurological examination revealed flaccid paraparesis, bilateral hypoesthesia, and hypopallesthesia, predominantly distally.

To ascertain the etiology, electroneuromyography revealed subacute motor axonal polyneuropathy; autoimmune screening detected elevated levels of perinuclear antineutrophil cytoplasmic antibodies (p-ANCA) and antinuclear antibodies, along with positive anti-double-stranded DNA (anti-dsDNA) antibodies and anti-nucleosome antibodies in the Antinuclear Antibody Profile Test. Renal involvement was evident through decreased renal function, severe proteinuria, albuminuria, and moderate microcytic normochromic anemia on blood tests. A thoracic CT scan revealed fibrosis, bronchiectasis, and a nodule in the medial right lobe due to recurrent respiratory infections.

The patient received treatment with methylprednisolone during hospitalization, leading to partial remission of symptoms. Methylprednisolone was subsequently combined with azathioprine.

Conclusion. In patients presenting with signs of polyneuropathy alongside renal and pulmonary pathology, consideration should be given to microscopic polyangiitis as a potential cause.

Keywords: microscopic polyangiitis, polyneuropathy, p-ANCA

P066. A surprising etiology of stroke in a patient with atrial fibrillation: Takotsubo cardiomyopathy with ventricular thrombus and multiple cardioembolic complications after COVID-19

N. MARGINEAN[1,2], C. HERGHELEGIU[1,2], I. BADEA-LUPASTEANU[3,4], C. A. BUZEA[3,4], R. MOROTI-CONSTANTINESCU[5,6], L. DUMITRESCU[1,2], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,7]

[1]*Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania*
[2]*Clinical Neuroscience Department, Faculty of Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*Cardiothoracic Cardiology Department, Faculty of Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[4]*Department of Cardiology, Colentina Clinical Hospital, Bucharest, Romania*
[5]*Infectious Diseases Department, "Matei Bals" National Institute of Infectious Diseases, Bucharest, Romania*
[6]*Infectious Diseases Department, Faculty of Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[7]*Laboratory of Cell Biology, Neuroscience and Experimental Myology, "Victor Babes" National Institute of Pathology, Bucharest, Romania*

Introduction. SARS-CoV-2 infection leads to hypercoagulability and predisposition towards thromboembolic events, such as deep vein thrombosis, stroke, and myocardial infarction. Recent studies emphasize the risk of stress cardiomyopathy among COVID-19 patients that can, in rare instances, lead to intracardiac thrombus formation and systemic embolism.

Methods. A 74-year-old male with vascular risk factors (hypertension, type 2 diabetes, hypercholesterolemia, obesity), a history of stroke, and permanent atrial fibrillation was admitted for aphasia with sudden onset. He followed apixaban treatment but was prescribed an inappropriately low dose. Two weeks prior, he tested positive for COVID-19, manifested by fatigue, fever, and cough, which subsided after symptomatic treatment. The brain tomography did not detect acute lesions, the electrocardiogram showed signs of ischemia, and the blood work revealed high inflammatory markers and elevated NT-proBNP. The transthoracic echocardiogram identified a left intraventricular thrombus and low ejection fraction. Anticoagulation with a therapeutic dose of low molecular weight heparin and antibiotherapy were started, with favorable evolution, until seven days later, when he developed acute obstruction of both femoral arteries. The echocardiography showed myocardial normokinesia and absence of the intraventricular thrombus. The brain MRI performed after the successful femoral thrombectomy displayed recent ischemic lesions in the superficial territory of the left middle cerebral artery and in the right cerebellum. The abdominal tomography showed bilateral renal infarction.

Results. This is a case of Takotsubo cardiomyopathy with intraventricular thrombus that caused cerebral, renal, and femoral artery embolism, occurring post-COVID-19 infection, despite anticoagulant treatment. We highlight a rare association of COVID-19 complications.

Keywords: stroke, COVID-19, Takotsubo cardiomyopathy

P067. A rare neurological manifestation of Sjögren's syndrome

A. TONTEA[1], S.MAIER[1,2], Z. BAJKO[1,2], R. BALASA[1,2]

[1]*Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania*
[2]*Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania*

Background. Neurological signs frequently accompany Sjögren's syndrome as a secondary clinical manifestation, often presenting as polyneuropathy. However, damage to the cranial nerves is a rarer occurrence, with a prevalence of 5% in the case of peripheral nervous system involvement in the primary form of the disease.

Case presentation. We present a case of a 52-year-old female patient with a long history of joint pain in the small joints of the hands and feet, myalgias, and paresthesias, who presented suddenly with double vision. The neurological examination revealed left-sided abducens nerve palsy. The cerebral CT and angio CT scan were negative, and cerebral MRI evidenced only small microangiopathic changes. The laboratory investigations showed high levels of inflammatory markers (ESR, CRP), rheumatoid factor, antinuclear antibody, and SS-A and SS-B antibodies. Screening for paraneoplastic and infectious diseases was negative. EMG/ENG examination was negative. Schirmer strip indicated severe dry eyes (5 mm of wetting). Based on the clinical and paraclinical findings, the rheumatological evaluation established the diagnosis of Sjögren's syndrome. Corticotherapy with methylprednisolone was initiated, resulting in symptom improvement during hospitalization. Later, immunosuppressive treatment with azathioprine was added for chronic disease management.

Conclusions. Despite its rarity, rheumatologic pathology should be considered as a potential diagnosis in cases of cranial nerve palsies.

Keywords: Sjögren's syndrome, cranial nerves, abducens nerve palsy, SS-A and SS-B antibodies

P068. Von Hippel Lindau disease, a diagnostic dilemma in a patient with multiorgan tumors and negative genetic test

M. G. I. PROTOSEVICI[1,2], C. L. PROTOSEVICI[2], C. G. BRUMAR[1], V. A. POPESCU[1], A. IVAN[1], M. IOSUB[2]

[1]"Titu Maiorescu" University, Bucharest, Romania
[2]Ilfov County Emergency Clinic Hospital, Ilfov, Romania

Von Hippel Lindau disease is a genetic condition characterized by the lack of suppression of Hypoxia Induced Factor (HIF). This promotes angiogenesis and cell proliferation in the central nervous system and in various other organs. Hemangioblastomas develop in the nervous system and retina, while cysts and tumors appear in other organs.

We present the case of a 46-year-old man who presented to the emergency department with a sensory syndrome. A cerebral CT scan revealed a significant cerebellar lesion, which could not explain the symptomatology. This lack of correlation between the cerebral CT scan and the symptoms prompted further investigations, which discovered additional asymptomatic lesions, including a large renal tumor. The association between cerebellar and renal tumors (cerebellar hemangioblastoma and renal carcinoma) led the radiologist to consider the diagnosis of Von Hippel Lindau disease. Additionally, a vascular subcutaneous retroauricular tumor was found. Finally, spinal MRI demonstrated intraspinal cervical and thoracic lesions responsible for the symptomatology.

Conclusion. The analysis of initial clinical and imaging details led to further investigations and the diagnosis of a rare disease. The patient was referred to a clinic of urology for minimally invasive surgery/nephron-sparing surgery. The possibility of treatment with belzutifan is also being discussed.

P069. Acute motor axonal neuropathy concomitant with SARS-CoV-2 infection: A case report

C. DOBARCEANU[1], A. MOTATAIANU[1,2], R. BALASA[1,2]

[1]Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania
[2]Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania

Introduction. Acute motor axonal neuropathy is a rare form of Guillain-Barré syndrome, characterized by severe motor axonal dysfunction.

Case presentation. We describe a case of acute axonal motor neuropathy arising in the setting of SARS-CoV-2 infection in a 26-year-old male patient, diagnosed with insulin-dependent type 1 diabetes mellitus one year ago, with poor glycemic control. The patient's initial symptoms included fever and coughing, followed by a decrease in strength in the lower limbs. After 12 hours, the motor deficit progressed to the upper limbs, without paresthesias. Upon evaluation in the emergency department, the neurological exam revealed no cranial nerve abnormalities, tetraparesis (2/5 MRC in proximal upper limbs, 1/5 MRC in proximal lower limbs, 0/5 MRC in distal limbs). After 24 hours, he developed tetraplegia. He tested positive for SARS-CoV-2.

On the first day of hospitalization, a diagnostic lumbar puncture was performed. Chemical-physical, virological, and cytological analyses of the cerebrospinal fluid showed mild albuminocytologic dissociation. The electroneurographic examination revealed changes suggestive of acute motor axonal neuropathy. Brain MRI and cervical spine MRI were performed with normal findings. The patient received immunoglobulin treatment for five days without improvement, followed by six sessions of plasmapheresis, with mild improvement of the motor deficit during hospitalization and progressive recovery (approximately 85%) in the first two months after discharge.

Conclusion. This disease is a rare form of acute neuropathy with progressive onset and severe evolution in a young man with important comorbidities, such as diabetes mellitus, during the acute viral infection.

Keywords: axonal neuropathy, SARS-CoV-2, muscle strength, fever, plasmapheresis

P070. Coexistence of two autoimmune pathologies: NMOSD with anti-aquaporin-4 and SLE

A. PALIMARU, C. CROITORU, L. CUCOS

2nd Neurology Clinic, "Prof. Dr. N. Oblu" Emergency Clinical Hospital Iasi, Romania

Introduction. NMOSD is a rare but CNS-impacting inflammatory and demyelinating pathology involving the optic nerves, brainstem, and spinal cord. This disorder, comprising six syndromes, is complexly related to the AQP4-IgG antibody, requiring serologic testing for accurate evaluations. The prevalence of NMOSD is 0.3-4.4 per 100,000 people and typically occurs in females (80%) between the ages of 30 and 40.

SLE is a chronic autoimmune disease that can affect any organ and can coexist with other autoimmune neurological diseases, such as NMOSD.

Case presentation. We present the case of a 31-year-old patient who reported the onset of the disease 6 weeks prior, with nausea, repetitive uncontrollable vomiting. Initially hospitalized in the Department of Internal Medicine, she was later transferred to the Department of General Surgery, where she underwent gastroscopy, abdominal ultrasound, and abdominal-pelvic CT, which established the diagnosis of aorto-mesenteric clamp syndrome, leading to surgical intervention. Given the persistence of post-operative symptoms, a neurological consultation was requested. Upon evaluation, postural instability, ataxia, nystagmus on bilateral gaze, and visual disturbances were noted. These symptoms prompted further investigation through cranio-cerebral and cervical MRI, which revealed an acute demyelinating bulbar lesion, imprecisely delimited with a discretely inhomogeneous structure and minimal diffusion restriction, involving the region of the postrema area. An ophthalmological consultation and OCT revealed optic neuritis, raising suspicion of a pathology from the NMOSD spectrum. The immunological panel and lumbar puncture confirmed the diagnosis of NMOSD with anti-AQP4 antibodies, and SLE was also diagnosed through the presence of double-stranded DNA antibodies and SS-A antibodies. The patient showed favorable evolution under corticotherapy, despite the non-specific clinical picture and the complexity of managing such a case.

Conclusion. NMOSD and SLE are independent autoimmune diseases that can coexist with each other, presenting a real therapeutic challenge for any clinician.

Keywords: NMOSD, anti-AQP4, optic neuritis, area postrema syndrome, SLE

P071. Mysterious headache: a case of spontaneous intracranial hypotension without determined source

Raluca CIOCINTA[1,2],Andreea E. LUNGU[1], Letitia E. VLAD[1], Serban DANCIU[1], Florentina A. DANCIU[1], Dan I. CUCIUREANU[1,2]

[1]"Prof. Dr. N. Oblu" Clinical EmergencyHospital, Iasi, Romania

[2]"Grigore T. Popa" University of Medicine and Pharmacy, Iasi, Romania

Introduction. Spontaneous intracranial hypotension is a condition caused by low cerebrospinal fluid (CSF) pressure and has various etiologies. The dominant clinical manifestation is orthostatic headache. Magnetic resonance imaging (MRI) plays an important role in its diagnosis. Specific neuroimaging changes consist of pachymeningeal enhancement, pituitary enlargement, or subdural collections. Treatment can be conservative, blood patch, or surgery.

Case presentation. A 27-year-old patient with a history of dorso-lumbar trauma is referred for diffuse headache, predominantly nuchal, with recent onset and significant postural character, associated with digestive and cochlear manifestations. Cranial MRI showed quantitative changes (pachymeningeal enhancement, significant cerebral venous distension, descent of the cerebellar tonsils) and qualitative changes: the interpeduncular angle, the mammillo-pontine distance, and the ponto-mesencephalic angle with values below the normal limit. To establish the etiology, 3D myelo-MRI and dorso-lumbar myelo-CT with thecal injection of contrast were performed, which did not identify any etiological source. A favorable clinical evolution was noted, especially after the injection of the intrathecal contrast.

Conclusion. Spontaneous intracranial hypotension is a benign condition that has a good prognosis in the case of an early diagnosis. Often, this diagnosis is missed, and the patient is exposed to an increased risk of important complications that can endanger their life.

Keywords: intracranial hypotension, postural headache, cerebrospinal fluid

P072. The Brownell-Oppenheimer variant of Creutzfeldt-Jakob disease – a case report

Andreea BUTNARU[1],
Thomas Gabriel SCHREINER[2,3],
Mihaela CONSTANTINESCU[1]

[1]Second Neurology Clinic, "Prof. Dr. N. Oblu" Clinical Emergency Hospital, Iasi, Romania
[2]Department "Medicale III", Faculty of Medicine, "Gr. T. Popa" University of Medicine and Pharmacy Iasi, Romania
[3]First Neurology Clinic, "Prof. Dr. N. Oblu" Clinical Emergency Hospital, Iasi, Romania

Introduction. Creutzfeldt-Jakob disease (CJD) is a rare and rapidly progressing neurodegenerative disorder characterized by spongiform changes in brain tissue caused by the abnormal folding of prion proteins. With an incidence of about 1 to 1.5 cases per million people per year worldwide, CJD poses unique diagnostic challenges due to its varied clinical manifestations and the absence of definitive laboratory tests during the early stages.

Case presentation. We present the case of a 62-year-old hypertensive woman who reported the insidious onset of dizziness, balance, and coordination disorders a few months before presentation, with progressive evolution toward severe ataxic tetraparesis. Multiple paraclinical investigations were performed: head CT, abdominal-pelvic CT, electroneuromyography, and ophthalmological exam, all within normal limits. The head MRI highlighted discrete FLAIR hyperintensity of the caudate nuclei bilaterally and symmetrically, suggesting either a metabolic disorder or Creutzfeldt-Jakob disease. Cerebrospinal fluid examination showed the presence of protein 14-3-3, and the EEG revealed theta rhythm with periodic sharp wave complexes, which concluded the diagnosis. Despite various treatments, including corticotherapy, intravenous immunoglobulins, and symptomatic and supportive therapies, the evolution was rapidly progressive with the association of dysarthria, the appearance of myoclonus, and cognitive degradation.

Conclusions. The Brownell-Oppenheimer variant is a rare form of sporadic Creutzfeldt-Jakob disease, characterized by the predominance of cerebellar ataxia in the clinical picture. The presented case report highlights the diagnostic and management challenges of this rare variant, broadening the scarce knowledge on prion diseases and offering a basis for future research directions.

Keywords: Creutzfeldt-Jakob disease, cerebellar ataxia, prion disease, protein 14-3-3, cerebrospinal fluid biomarker

P073. A central variant of PRES associated with dialysis disequilibrium syndrome – case report

M. IORDACHE, S. BOSTAN, A. ARSENE, S. DUMITRACHE

National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania

Introduction. Posterior reversible encephalopathy syndrome (PRES) is a condition that can manifest with headache, altered mental status, epileptic seizures, visual disturbances, and MR imaging showing preferential reversible vasogenic edema within the occipital and parietal lobes. It is often seen in the context of hypertension, lupus, immunosuppressive medications, hemolytic-uremic syndrome, or sepsis. Atypical locations have been described, including the basal ganglia, thalamus, brainstem, and cerebellum.

Methods. We present the case of a 50-year-old hypertensive patient with type I diabetes mellitus and end-stage nephropathy on dialysis, with a recent history of subarachnoid hemorrhage. The patient was hospitalized for altered general condition, dysarthria, and psychomotor agitation that started during the day after a hemodialysis session, presenting with a blood pressure of 230/110 mmHg. Native brain CT depicted hypodensity at the pontine level. Brain MRI revealed hypersignal FLAIR and T2-weighted changes with isosignal T1 at the pontine level, middle cerebellar peduncles, cerebellum, basal ganglia, and bilateral parieto-occipital white matter. There was no contrast enhancement or restricted diffusion, but hemorrhagic stigmata were noted on SWI in addition to the known secondary subarachnoid hemorrhage, suggesting a central variant of PRES. The patient repeated two episodes of restlessness followed by drowsiness shortly after dialysis sessions, clinically classified as dialysis disequilibrium syndrome.

Results. During hospitalization, antihypertensive therapy was adjusted, and nephrology re-evaluation was recommended.

Conclusions. Correlating the clinical manifestations with the imaging data and the patient's medical history, the diagnosis of dialysis disequilibrium syndrome was established, which associates with a central variant of reversible encephalopathy syndrome (PRES).

Keywords: PRES, dialysis disequilibrium syndrome, hypertension

P074. Advanced lung adenocarcinoma presenting with synchronous cranial nerve palsy

Andreea PLESA[1,2], Ruxandra GEORGESCU[1], Angelo VOICU[3], Florentina-Cristina PLESA[1,2], Carmen-Adella SIRBU[1,2]

[1]Department of Neurology, "Dr. Carol Davila" Emergency University Central Military Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]"Prof. Dr. Agrippa Ionescu" Emergency Clinical Hospital, Bucharest, Romania

Introduction/Objective. Adenocarcinoma, the most common type of non-small cell lung cancer (NSCLC), typically presents with symptoms such as cough and dyspnea. Isolated neurological symptoms are rare. This paper presents a case where bulbar cranial nerve involvement was the initial sign of advanced lung cancer.

Material and methods. A 51-year-old non-smoking woman, previously healthy, presented to our hospital ten days after experiencing difficulty pronouncing certain words and swallowing solid foods. Neurological examination revealed paralysis of the right vocal cord, palate, and right side of the tongue, indicating dysfunction of multiple cranial nerves, notably the glossopharyngeal (IX), vagus (X), and hypoglossal (XII) nerves.

Despite undergoing MRI scans of the brain and skull base with contrast administration, no focal lesions compressing these nerves were found. Due to involvement of the recurrent laryngeal nerve, a chest CT scan was conducted, revealing a tumor in the inferior lobe of the right lung. A subsequent lung biopsy confirmed NSCLC. Additional assessment via PET-CT revealed metastases to the liver and bones, categorizing the disease as stage IV.

Given the absence of brain or neck metastasis, we conducted an extensive anti-neuronal antibody profile, encompassing amphiphysin, CV2, PNMA2, Ri, Yo, Hu, Recoverin, and SOX1, along with cerebrospinal fluid analysis. However, all results returned negative. Nevertheless, we continue to regard it as a paraneoplastic manifestation.

Results/Conclusions. This case, a rare instance of advanced NSCLC with initial cranial neuropathies, highlights the importance of comprehensive evaluation in patients with cranial nerve symptoms. Early detection is crucial, as the patient's condition had already progressed significantly, emphasizing the need for timely intervention.

Keywords: paraneoplastic syndromes, advanced NSCLC, cranial neuropathies

P075. A rare cause of intracranial hypertension – A case report

B. O. PIRLOG[1], A. TALOS[1], D. CRISAN[2], S. ILUT[1]

[1]Department of Neurology, Cluj County Emergency Hospital Cluj-Napoca, Romania
[2]Department of Pathology, Cluj County Emergency Hospital Cluj-Napoca, Romania

Background. Hemangiopericytoma is a very rare tumor that derives from the pericytes. Cerebral hemangiopericytomas mostly arise from the meninges or spinal cord. Symptoms may be non-specific and, depending on their size, they can lead to intracranial hypertension syndrome.

Methods. This case report presents a young male patient admitted to our neurology department with an unusual headache.

Results. A 26-year-old male patient with a history of beta thalassemia was admitted to our department for a frontal headache, characterized by a pressure sensation, associated with tinnitus and moderately responsive to NSAIDs. The patient was initially seen by an otolaryngologist for his tinnitus, but the results were negative. Over the past 2-3 weeks, the headache worsened and became insomniac. Due to the persistence of the symptoms, a CT scan was performed, revealing a mass. On neurological examination, no signs of gravity were detected. Brain MRI confirmed the presence of an extra-axial mass localized in the parietal-occipital lobe with vasogenic edema, mass effect, and cerebral herniation. Due to the gravity of the MRI findings, surgery was performed immediately. Post-surgery, the outcome was good, and the post-operative CT scan showed no abnormalities. Pathological results confirmed that the mass was a hemangiopericytoma.

Conclusion. In conclusion, a complete and repeated workup is necessary in cases of unusual headaches, as initial symptoms and signs may be scarce. No correlation between beta thalassemia and this tumor has been found in the literature.

P076. Horton disease with vertebral artery involvement

E.S. BRITA[1], L. POPA[1], H. GOLDSTEIN[1], L. BICA[1], C. A. PANEA[1,2]

[1]Neurology Clinic, Elias Emergency University Hospital Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Horton's disease, also called giant cell arteritis or temporal arteritis, is an idiopathic chronic systemic inflammatory disease that affects the large and medium arteries, primarily the cranial branches of the arteries originating from the aortic arch. It most commonly affects women after the age of 50 and presents numerous complications if left untreated.

Methods and results. We present the case of a 72-year-old female patient who arrived at the emergency room with a severe left temporal headache that had started 2 months ago. It was initially investigated and diagnosed as Arnold's neuralgia, but on the day of arrival, it was associated with nausea, vomiting, and postural instability.

Clinical and neurological examination revealed an indurated aspect of the superficial temporal artery bilaterally, which was sensitive to palpation. Paraclinically, a biological inflammatory syndrome was detected, and cerebrovascular ultrasonography revealed the "halo" sign at the level of the superficial temporal and vertebral arteries bilaterally.

The suspicion of Horton's disease with damage to the left > right vertebral arteries was raised. The patient was referred to a rheumatology service, where a definitive diagnosis was made, and corticosteroid therapy was started, leading to an improvement in symptoms, but with progressive damage to the vertebral arteries. During the angiography-CT examination of the supra-aortic arteries, the occlusion of the right vertebral artery and 50% stenosis of the left vertebral artery were observed.

Conclusions. Horton's disease with bilateral vertebral artery involvement is rare but carries a high mortality risk, which underscores the importance of correct diagnosis and prompt initiation of treatment.

Keywords: Horton's disease, superficial temporal artery, halo sign

P077. Neuromuscular ultrasound helps distinguish multifocal motor neuropathy from amyotrophic lateral sclerosis – a case report

M. PALADE[1], N. GRECU[1,2], A. CRACIUNOIU[1], C. TIU[1,2]

[1]Department of Neurology, Emergency University Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Distinguishing between multifocal motor neuropathy (MMN) and motor neuron disease (MND) can sometimes be challenging, as clinical presentation and electrodiagnostic studies may be similar. Neuromuscular ultrasound is a valuable, non-invasive tool that has been shown to aid in the diagnosis of inflammatory neuropathies.

Materials and methods. We present the case of a 65-year-old man who developed bilateral, slowly progressive, asymmetric, painless upper limb weakness over the course of 1 year, initially labeled as amyotrophic lateral sclerosis (ALS). Clinical examination showed marked atrophy of his upper limb muscles, as well as fasciculations in his deltoids. He also exhibited asymmetric weakness for shoulder abduction, wrist extension, and all intrinsic hand muscles. Sensation was intact, and reflexes were absent.

Electrodiagnostic studies were compatible with an asymmetric motor neuropathy. Needle electromyography showed abnormal spontaneous activity in the arms, as well as chronic denervation in all limb muscles. Spinal MRI was unremarkable. Neuromuscular ultrasound, using a 13-15MHz probe, revealed upper limb muscle atrophy and degeneration, as well as fasciculations. The median and ulnar nerves were hypoechoic, with few enlarged fascicles, and a relatively normal cross-sectional area, suggestive of inflammatory neuropathy.

Diagnostic work-up was supplemented with cerebrospinal fluid analysis, showing elevated protein levels and a normal cell count. Serum antiganglioside antibodies were positive for GM1. Based on these findings, the diagnosis of multifocal motor neuropathy was made, and the patient was started on intravenous immunoglobulins.

Conclusion. While neuromuscular ultrasound does not surpass other diagnostic methods, it can provide valuable information and guide clinicians toward a diagnosis of inflammatory neuropathy.

P078. Clinical cases of bacterial meningoencephalitis during pregnancy

Maria VASILIEVA[1,2,3], Aliona BEJENARI[1,2], Ana BONDARCIUC[1,2], Alexandru GASNAS[1,2,3], Zota EREMEI[1,2,3], Belitei DOINA2, Diana MANEA[2], Stanislav GROPPA[1,2,3]

[1]*DepartmentofNeurologyno.2, "NicolaeTestemitanu" State University of Medicine and Pharmacy, Chisinau, Republic of Moldova*
[2]*Clinical Department of Neurology, Epileptology and Internal Disease, Institute of Emergency Medicine, Chisinau, Republic of Moldova*
[3]*Laboratoryof Neurobiology and Medical Genetics, "NicolaeTestemitanu" State University of Medicine and Pharmacy, Chisinau, Republic of Moldova*

Introduction. The incidence of bacterial meningitis is between 2.6-6 per 100,000 adults, with a mortality rate of up to 30%. During pregnancy, meningoencephalitis is an uncommon disorder, with high mortality for both the mother and child. We aim to evaluate predisposing factors and etiologic agents involved in bacterial meningoencephalitis (BME) during pregnancy.

Material and methods. We present two cases: a 29-year-old pregnant female at 29-30 weeks of gestational age and a 20-year-old pregnant woman at 27-28 weeks of gestational age, both diagnosed with BME. We correlate literature analysis with our cases.

Results and conclusions. The first patient was admitted to the emergency room (ER) with altered consciousness and positive meningeal signs. Brain CT showed diffuse cerebral edema, pneumocephalus in the basal middle cerebral fossa on the right, at the level of the pyramid, and perforated right otitis media. CSF presented with neutrophilic pleocytosis, and she tested positive for COVID-19 infection. Empiric antibacterial medication resulted in clinical improvement within four days.

The second patient was admitted to the ER with psychomotor agitation, RASS +4, and neck rigidity. Brain CT revealed right-sided, acute otomastoiditis. She received empiric antibacterial medication, with clinical improvement in five days. CSF bacteriologic investigation was negative for microorganisms in both cases.

From the literature analysis and our cases, we concluded that BME during pregnancy is an extremely rare pathology, and COVID-19 could also be a predisposing factor. Otitis and sinusitis are important risk factors for developing BME in pregnancy. *S. pneumoniae* and *L. monocytogenes* are the causative pathogens of bacterial meningoencephalitis during pregnancy.

Keywords: BME, pregnancy

P079. Metabolic encephalopathy – nonketotic hyperglycemia associated with basal ganglia anomalies (case report)

A. M. OLARU[1], A. G. BANICA[1], G. VULPE[1], C. A. PANEA[1,2]

[1]*Emergency University Hospital, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Hyperglycemia without ketone bodies and neuroimaging anomalies of the basal ganglia is a rare complication of uncontrolled diabetes mellitus, with an increased mortality rate, occurring more frequently in adults over the age of 50.

Objectives, method, outcome. We present the case of a 73-year-old woman, without significant known medical history or chronic treatment, admitted after loss of consciousness with bilateral motor manifestations on the same day.

The neurological examination noted left brachial dystonic posture with distal pronation, without other movement disorders.

Brain CT scan showed diffuse hyperdense caudate and lenticular nuclei on the right side without pathological contrast enhancement, and a right occipital cortical chronic infarction. Contrast-enhanced brain MRI showed hypersignal in the basal ganglia on the right, with increased paramagnetic susceptibility at this level.

The blood work showed metabolic acidosis (pH = 7.2) with severe hyperglycemia (574 mg/dL), and hemoglobin A1c was 17.7%, leading to the diagnosis of severe unbalanced diabetes mellitus.

Scalp EEG showed no abnormalities.

After fluid and acid-base rebalancing treatment, insulin therapy, dopamine receptor antagonist, and antiepileptic treatment, the patient had a favorable outcome, pending further cognitive assessment.

Conclusions. Metabolic encephalopathy in the context of nonketotic hyperglycemia with basal nuclei imaging changes is a rare complication of unbalanced diabetes. Treatment consists of management of hyperglycemia and vital function support, with antiepileptic drugs having an uncertain role.

P080. Spontaneous intracranial hypotension – case report

D. BUGA, F. FORTIS, M. SABAU

[1]*Department of Neurology, Bihor County Emergency Clinical Hospital, Oradea, Romania*
[2]*Faculty of Medicine and Pharmacy, University of Oradea, Oradea, Romania*

Introduction. Intracranial hypotension is a clinical entity that results from a cerebrospinal fluid (CSF) leak that almost without exception occurs from the spine, either into the epidural space or directly into veins in the setting of CSF-venous fistulas. It usually presents clinically with orthostatic headaches.

Material and methods. We present the case of a 44-year-old woman who complained of orthostatic headache, which was relieved by lying in a recumbent position within around 20 minutes. Painkillers had little to no effect. The persistence of symptoms prevented her from attending work and performing household tasks.

Results. Brain MRI showed pachymeningeal enhancement and tonsillar herniation. Whole-spine MRI later revealed a CSF leak next to the L4 and L5 vertebral bodies. Intravenous fluids were administered, and an epidural blood patch was performed. The headaches resolved the next day.

Conclusions. Although intracranial hypotension often resolves on its own, more severe or persistent cases may require intervention. The epidural blood patch proved effective, with symptoms resolving quickly and completely, thus preventing further complications.

Keywords: intracranial hypotension, headaches, CSF leak, blood patch

P081. Therapy with anti-CD20 monoclonal antibodies can be a therapeutic solution in the case of coexistence between multiple sclerosis and systemic lupus erythematosus – considerations based on a clinical case

Silviu STANCIU[1], Simona PETRESCU[1,2], Luana STANCIU[3], Melania Maria MARTOIU[1], Cristina A. PANEA[1,2]

[1]*Elias University Emergency Hospital, Bucharest, Romania*
2"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
3"Dr. Ion Stoia" Rheumatic Disease Center, Bucharest, Romania

Introduction. Multiple sclerosis (MS) and systemic lupus erythematosus (SLE) are chronic autoimmune diseases that predominantly affect women, with a prevalence of 1–1.2% in various cohorts.

Method and results. A 27-year-old woman was diagnosed with MS (spinal onset) at the age of 23 and initiated immunomodulatory treatment with interferon beta-1a (IFN beta-1a). After two years, she developed erythematous plaques on the palms and soles, followed by arthralgia in the small joints of the wrists. In this context, IFN beta-1a treatment was discontinued. A diagnosis of SLE with cutaneous involvement was established, and treatment with methylprednisolone and hydroxychloroquine was initiated, later continued with hydroxychloroquine alone. Under this regimen, she developed retinal toxicity.

The coexistence of both conditions, along with the adverse reaction to hydroxychloroquine, necessitated a different therapeutic approach. Through a multidisciplinary evaluation, treatment with a fully humanized anti-CD20 monoclonal antibody (ofatumumab) was initiated.

Conclusions and discussions. The coexistence of MS and SLE is rare, with a disease phenotype that is generally not severe for either condition. SLE manifestations in association with MS are typically cutaneous and musculoskeletal. The therapeutic decision should be guided primarily by the severity of the neurological disease.

Keywords: MS, SLE, anti-CD20

P082. Occurrence of amyotrophic lateral sclerosis-like syndrome in a human immunodeficiency virus-infected patient

L. CAULEA[1], A. MOTATAIANU[2], R. BALASA[2]

[1]*Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania*

[2]*Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania*

Background. Neurological complications in HIV infection are presumed to occur by neurotoxic viral proteins or by cytokines and chemokines elaborated as a consequence of the HIV disease. Motor deficit related to the HIV-driven ALS-like syndrome can potentially improve following effective antiretroviral therapy.

Case presentation. We report a case of an HIV-positive 31-year-old female patient diagnosed with amyotrophic lateral sclerosis-like syndrome after giving birth. She was not compliant with her treatment until pregnancy. Despite treatment with emtricitabine/tenofovir and raltegravir and a well-controlled HIV infection (CD4 339 cells/mm^3 and HIV RNA <20 copies/mL), she experienced rapid progression of motor deficits with tetraparesis and severe dysphonia.

The cerebral and cervical spine MRI were normal. Electromyography tests showed neurogenic changes with active and chronic denervation signs (fasciculations and fibrillations) in the median and ulnar nerves bilaterally, with more than 80% drop in CMAP amplitude. In order to exclude a diagnosis of multifocal motor neuropathy with conduction blocks, treatment with intravenous immunoglobulin was administered, without any clinical or electromyographic improvement.

Conclusion. Recognition of an HIV-related motor neuron syndrome is important because it may respond to treatment if the viral plasma levels decrease. Although the patient received treatment with riluzole and antiretroviral medication was adjusted, she continues to present worsening neurological status.

Keywords: HIV, ALS-like syndrome, motor neuron disease, antiretroviral therapy, electromyography

P083. Approach to a case of very early-onset dementia

Ioan-Cristian LUPESCU[1,2], Daniela Cristina ANGHEL[1,2], Andreea GITMAN[1], Adriana Octaviana DULAMEA[1,2]

[1]*Neurology Department, Fundeni Clinical Institute, Bucharest, Romania*

[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Early-onset dementia refers to cases of dementia affecting patients younger than 65 years of age. Emphasis should be placed on excluding potentially reversible causes. Once these are ruled out, the differential diagnosis should consider neurodegenerative, vascular, infectious-inflammatory, and metabolic disorders.

Material and methods. A 27-year-old female, with no significant personal or family history, was admitted in 2018 for behavioral changes (disinhibition, stereotyped behaviors) and difficulties with concentration. Clinically, the patient presented bilateral rigidity and apraxia. Brain MRI showed frontotemporal atrophy. Psychological assessment revealed pronounced hypoprosexia and dyscalculia, without language deficits or visuospatial disorientation. A PET scan demonstrated marked hypometabolism of the frontal lobes.

The patient returned in 2023 with severe global aphasia, overt stereotyped behavior, and utilization behavior. Brain MRI was repeated and revealed marked atrophy of the frontotemporal lobes (sparing the precentral gyri), pons, mesencephalon, and corpus callosum (sparing the splenium).

Blood and CSF laboratory workup excluded causes such as Wilson's disease, thyroid disease, autoimmune (including paraneoplastic) encephalitis, vitamin B12 deficiency, HIV infection, neurosyphilis, autoimmune disorders (lupus, Sjögren syndrome), neurosarcoidosis, neuroborreliosis, Huntington's disease, and Creutzfeldt-Jakob disease. CSF analysis showed normal levels of Tau protein and phosphorylated Tau, but beta-amyloid (1-42) was decreased at one point. Metabolic causes of dementia were also considered and excluded, including metachromatic leukodystrophy, GM1 gangliosidosis, Gaucher disease, Krabbe disease, Niemann-Pick types A/B, and neuronal ceroid lipofuscinosis type 2.

Conclusions. Based on the clinical picture and thorough exclusion of other potential causes, a diagnosis of probable frontotemporal dementia was established.

Keywords: frontotemporal dementia, early-onset dementia

P084. Therapeutic approach in acute ischemic stroke in a patient with solid gynecologic neoplasm

C. A. NEGOITA[1], A. M. MIERCIOIU[1], I. R. NISTOR[1,2], B. DOROBAT[1]

[1]*University Emergency Hospital of Bucharest, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction and objectives. Stroke is a leading cause of disability and death worldwide. Approximately 15% of cancer patients experience cerebrovascular events. Acute ischemic stroke can even be the first manifestation of an underlying malignancy. Patients with gynecological neoplasms are at increased risk of ischemic stroke, and the presence of atrial fibrillation further elevates this risk.

Materials and methods. We present the case of a 77-year-old female patient with relatively few traditional stroke risk factors, such as hypertension and moderate atherosclerosis, but recently diagnosed with atrial fibrillation. She was brought to the emergency department with sudden-onset symptoms, including speech disturbances and left-sided motor impairment. A cerebral computed tomography (CT) scan, both with and without contrast, was performed. A right middle cerebral artery (MCA) occlusion was identified, and the patient underwent diagnostic and therapeutic cerebral angiography, followed by intravenous thrombolysis.

Results. Follow-up non-contrast cerebral CT imaging revealed a hypodense area in the right MCA territory, with signs of confluent petechial hemorrhage post-reperfusion therapy. A comprehensive etiopathogenic workup was performed, and according to the TOAST classification, two or more mechanisms were identified as the cause of the ischemic stroke: atrial fibrillation and a newly diagnosed endometrial neoplasm. The patient had a minimally favorable clinical evolution by discharge and three months later.

Discussion and conclusions. Anticoagulation plays a crucial role in preventing recurrent ischemic strokes in cancer patients with atrial fibrillation. However, patients with malignancy-associated stroke generally have a worse prognosis than those without cancer. An individualized treatment plan and a multidisciplinary approach are essential. In cases of severe malignancy-associated stroke, palliative care may be necessary.

Keywords: ischemic stroke, malignancy-associated stroke, gynecologic cancer, atrial fibrillation, thrombolysis, anticoagulation

P085. Therapeutic approach in acute ischemic stroke associated with traumatic internal carotid artery dissection

C. A. NEGOITA[1], A. M. MIERCIOIU[1], I. R. NISTOR[1,2], B. DOROBAT[1]

[1]*University Emergency Hospital of Bucharest, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction and objectives. The treatment of acute ischemic stroke (AIS) associated with traumatic internal carotid artery (ICA) dissection remains controversial. Although there are no universally accepted guidelines for this combination of pathologies, modern revascularization therapies—either chemical (thrombolysis) or mechanical (thrombectomy)—are increasingly used as preferred interventions in cases of AIS due to large vessel occlusion.

Materials and methods. We present the case of a 59-year-old male patient with a history of chronic ethanol use and smoking. He was brought to the emergency department with sudden-onset symptoms, including speech disturbance and left-sided motor impairment, following a ground-level fall that resulted in right anterior cervical trauma.

The patient underwent diagnostic and therapeutic cerebral angiography. A thrombotic occlusion of the right ICA at the post-bulbar level was identified. Mechanical thrombectomy was performed, during which both thrombus and intimal tissue were extracted. Histopathological examination confirmed the presence of intimal tissue, supporting the diagnosis of arterial dissection with associated thrombosis as the mechanism of stroke.

Stenting was deferred in the acute phase due to prior thrombolysis and the successful reperfusion achieved via mechanical thrombectomy (TICI score III), despite the presence of an irregular vessel caliber and a minor residual dissection.

Results. Dynamic non-contrast CT and CT angiography revealed a hypodense area in the right ICA territory and dissection at the C1 segment with approximately 50% stenosis. Complete etiological investigations indicated an atherothrombotic mechanism secondary to traumatic dissection.

Given the clinical and imaging findings, the patient was started on dual antiplatelet therapy and a high-dose statin. The clinical course was favorable, with near-complete symptom resolution by the time of discharge and sustained improvement at six-month follow-up.

Discussion and conclusions. Current guidelines offer limited direction regarding both acute-phase management and secondary prevention in AIS associated with traumatic ICA dissection. Clinical decisions must be individualized and multidisciplinary in nature. In this case, the benefits of mechanical revascularization outweighed the potential risks, and the decision to delay stenting proved appropriate given the patient's noncompliance and the risk of in-stent restenosis.

P086. Transthyretin-related familial amyloidotic polyneuropathy (FAP ATTR Ile107Val) – Case report

Delia CIOBOTARU[1], Lenuta BIRSANU[1], Emilian Bogdan IGNAT[1,2]

[1]*Rehabilitation Hospital, Iasi, Romania*
[2]*Departamentof Neurology, "Griore.T. Popa" University of Medicine and Pharmacy, Iasi, Romania*

Introduction. Transthyretin familial amyloid polyneuropathy (TTR-FAP) is a severe, disabling, and irreversible neurological disease caused by abnormal deposits of transthyretin fibrils. These deposits are responsible for progressive sensory-motor and autonomic neuropathy.

Case presentation. A 56-year-old male patient presented to our clinic with severe tetramelic paresthesia. The neurological symptoms had evolved over six years, initially manifesting as distal crural paresthesia, which later extended to the upper extremities. Neurological examination revealed decreased superficial sensation in the first, second, and third fingers of both hands, as well as in the lower limbs.

Nerve conduction studies (NCS) showed bilateral carpal tunnel syndrome (CTS) and a length-dependent distal sensorimotor neuropathy, predominantly sensory. Sympathetic skin response (SSR) was absent in the lower limbs but present in the upper limbs. A Sudoscan test demonstrated normal sweat volume in the forearms and nearly absent sweating in the feet.

Genetic analysis of the TTR gene revealed that the patient was heterozygous for a single nucleotide substitution, c.379A>G, which resulted in the replacement of valine with isoleucine at position 107 of the mature protein. The diagnosis of familial amyloid polyneuropathy with transthyretin amyloidosis (ATTR) Ile-107Val was confirmed.

Discussion. The major advantage of genetic testing lies in the early diagnosis of this progressive and potentially devastating disease, allowing for the timely initiation of targeted therapy with transthyretin stabilizers such as tafamidis, which can slow disease progression.

Keywords: amyloidosis, familial amyloid polyneuropathy, NCS, carpal tunnel syndrome, transthyretin, Ile107Val

P087. A case of anti-glutamic acid decarboxylase antibody-associated ataxia: A possible extrahepatic autoimmune manifestation of chronic hepatitis C virus infection

I. ORMENISAN[1], S. MAIER[2], Z. BAJKO[2], R. BALASA[2]

[1]*Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania*
[2]*Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania*

Chronic hepatitis C virus (HCV) infection is associated with multiple extrahepatic manifestations, with numerous reports suggesting its potential association with various autoimmune disorders, often implicating HCV as the triggering factor.

Case presentation. We present the case of a male patient with a medical history notable for psoriasis, who presented with a progressive cerebellar syndrome over several years. Upon referral, blood tests revealed elevated liver enzymes, moderately elevated bilirubin, and mild thrombocytopenia. He tested positive for anti-HCV antibodies, and serum HCV RNA was detected. Given the clinical presentation and cerebral MRI findings showing moderate cerebellar atrophy, our patient was tested for serum anti-glutamic acid decarboxylase (GAD) antibodies, which were positive. We excluded other infectious, autoimmune, endocrine, vitamin deficiencies, or neoplastic disorders. He initiated antiviral therapy with sofosbuvir and velpatasvir and underwent four rounds of plasma exchange, resulting in mild improvement of the neurological deficits.

Conclusion. With the diagnosis of anti-glutamic acid decarboxylase antibody-associated ataxia and the concomitant chronic HCV infection, we suggest that an underlying molecular mimicry mechanism linking the two entities be further investigated, given the established association of HCV infection with several autoimmune disorders.

Keywords: cerebellar ataxia, GAD autoimmunity, HCV infection, molecular mimicry

P088. A rare neurological complication of tuberculosis: longitudinally extensive myelitis

Alina DRAGAN[1], Simona PETRESCU[1,2], Cristian GHITA[1], Maria MARTOIU[1], Monica MARCU[1], Cristina PANEA[1,2]

[1]Elias University Emergency Hospital, Bucharest, Romania
2"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Longitudinally extensive myelitis is a rare inflammatory and demyelinating condition characterized by acute onset and the association of motor, sensory, or autonomic manifestations.

Materials and methods. The present case report details the association of longitudinally extensive myelitis in a patient diagnosed with tuberculous meningitis. The patient, with a history of recurrent tumor formation in the left thigh, initially presented with a confusional syndrome associated with moderate headache that began two weeks prior to presentation and recurrent febrile episodes. The initial neurological examination revealed neck stiffness, temporo-spatial disorientation, and tetraparesis. Subsequently, the clinical course was unfavorable, with the onset of vegetative phenomena such as acute urinary retention, progression of motor deficit to tetraparesis predominantly affecting the paraplegia, tactile anesthesia, and alteration of consciousness up to stupor.

Results. Elucidating the etiology of neurological deficits required comprehensive clinical, laboratory, and imaging evaluation. Biochemical examination of the cerebrospinal fluid showed significant pleocytosis, hypoglycorrhachia, and proteinorrhachia, while microbiological results detected Mycobacterium tuberculosis genetic material via PCR. Imaging studies, including cervical-thoracic and brain MRI, revealed a hyperintense T2 lesion at the thoracic spinal cord level and contrast enhancement involving the spinal meninges and brainstem region. Additionally, investigations were conducted to exclude autoimmune pathologies within the spectrum of neuromyelitis optica potentially responsible for this clinical and imaging presentation, as well as a CT scan of the chest, abdomen, and pelvis to rule out a paraneoplastic syndrome.

Conclusion. In conclusion, this case underscores the rarity of tuberculous meningitis complications and the slow favorable evolution under early instituted antituberculous treatment.

Keywords: longitudinally extensive myelitis, tuberculous meningitis, confusional syndrome, vegetative phenomena, paraplegia, tactile anesthesia, stupor, PCR examination, hyperintense T2 lesion at the thoracic spinal cord level

P089. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): a subdiagnosed pathology?

S. J. KISS[1], A. STOIAN[1,2], R. I. BALASA[1,2]

[1]Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania
[2]Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania

Introduction. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an antibody-mediated inflammatory demyelinating disorder of the nervous system, predominantly targeting the optic nerves and spinal cord. The disease has a predilection for children and young adults.

Case presentation. We are presenting the case of a 47-year-old patient who has been experiencing motor deficit of the lower limbs for 10 years, accompanied by paresthesias more expressed distally. The patient also presented a decrease in visual acuity in both eyes a year ago, with spontaneous remission of symptoms after one week. The patient has performed electroneuromiographic examination, which highlighted a chronic demyelinating polyneuropathy with secondary axonal loss and severe motor impairment of the lower limbs. He has also performed a native brain and spine MRI examination that revealed pontine, left cerebellar hemisphere, and bilateral periventricular demyelinating lesions, as well as cervical medullary cord demyelination adjacent to the vertebral body C3. Laboratory analyses have shown an increased titer of anti-MOG IgG antibodies (1:20) and cerebrospinal fluid oligoclonal banding with questionable results.

Discussion. Given the neurological manifestations similar to those of multiple sclerosis or neuromyelitis optica, clinicians are frequently confronted with the challenge of diagnosing MOGAD. It is estimated that more than half of patients (55.4%) received an alternative primary diagnosis before the final MOGAD diagnosis.

Conclusion. Previously considered as a variant of neuromyelitis optica spectrum disorder, myelin oligodendrocyte glycoprotein antibody-associated disease is now recognized as an autonomous, antibody-mediated inflammatory demyelinating disorder, most frequently with a relapsing course and a variety of clinical manifestations, capable of affecting both the central and peripheral nervous systems.

P090. O cauză rară de crize epileptice

H. GOLDSTEIN[1], L. POPA[1], S. PETRESCU[1], H. NICOLAE[1], V. TIU[1], A. DANEASA[1], M. POPA[1], B. AZAMFIREI[1], L. BICA[1], S. BRITA[1], C. PANEA[1]

[1]*Spitalul Universitar de Urgență „Elias", București, România*

[2]*Universitatea de Medicină și Farmacie „Carol Davila", București, România*

Introducere/Obiectiv. Sindromul Fahr este o entitate clinică rară, cu transmitere genetică autozomal dominantă, care se remarcă prin prezența de calcificări bilaterale simetrice cerebrale, interesând ariile responsabile de controlul motor, în special ganglionii bazali, talamusul, nucleii dințați cerebeloși, substanța albă cerebrală și cerebeloasă. Debutul acestei patologii este mai frecvent între 40–50 de ani. Această boală poate prezenta simptome neuropsihiatrice progresive (precum tulburare neurocognitivă precoce, crize epileptice, cefalee, parkinsonism), dar și endocrine (disfuncție paratiroidiană – hipoparatiroidism în special, deficit de vitamina D).

Material și metode. Prezentăm cazul clinic al unui pacient în vârstă de 67 de ani care a fost diagnosticat cu sindrom Fahr, cu hipoparatiroidism primar și hipocalcemie severă, cu debut al simptomatologiei în mod acut, prin criză de tip epileptic.

Pacientul prezenta atât semne ale hipocalcemiei severe (Chvostek, Trousseau), cât și calcificări extensive cerebrale, de ganglioni bazali, corona radiata și emisfere cerebeloase.

De asemenea, pacientul asociază cardiopatie dilatativă cu disfuncție severă de ventricul stâng, secundară hipocalcemiei cronice. Prezintă și parestezii periorale, fenomene de laringospasm intermitent și insuficiență respiratorie.

Rezultate și concluzii. Hipoparatiroidismul primar poate asocia sindrom Fahr, dar și hipocalcemie cronică – ambele fiind potențiali triggeri ai crizelor epileptice. În cazul nostru, evoluția pacientului este favorabilă odată cu corectarea progresivă a hipocalcemiei. Prin reglarea acestui dezechilibru metabolic putem preveni apariția unor calcificări și mai importante. Acest lucru, împreună cu un tratament antiepileptic corect inițiat și urmat, reduce semnificativ crizele epileptice și, implicit, crește semnificativ calitatea vieții indivizilor afectați de această patologie extrem de rară.

Cuvinte cheie: sindrom Fahr, hipoparatiroidism primar, hipocalcemie, calcificări cerebrale, crize epileptice

P091. A case report of acute onset tetraplegia due to spinal arteriovenous fistula in an elderly patient

D. SPINU[1], S. MAIER[1,2], L. MARGINEAN[3], R. FILEP[3], R. BALASA[1,2]

[1]*Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania*

[2]*Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania*

[3]*Interventional Radiology Department, Radiology and Medical Imaging Laboratory, County Emergency Clinical Hospital of Targu Mures, Targu Mures, Romania*

Introduction. Vascular lesions of the spine are rare, accounting for only 1–2% of vascular neurological pathologies. Of those, 70% are spinal arteriovenous fistulas (AVFs)—vascular malformations characterized by abnormal connections between the arterial and venous systems in the spinal cord. They typically present with vague congestive myelopathy symptoms, and diagnosis can be challenging upon initial presentation. Despite their rarity, AVFs can lead to significant morbidity, with only a few case reports described in the literature, and even fewer presenting with sudden onset symptoms.

Case report. We present a unique case of an 82-year-old male patient who presented with acute onset tetraplegia. Neurological examination and imaging studies, including CT angiography, magnetic resonance imaging, and digital subtraction angiography, revealed a spinal AVF located at the cervical spine level. A multidisciplinary team, including neurologists, neurosurgeons, and interventional neuroradiologists, collaborated to manage the case. Despite the inherent challenges associated with advanced age and frailty, the patient underwent successful endovascular embolization of the AVF. Subsequent neurological evaluation demonstrated gradual improvement in motor function.

Discussion. This case underscores the importance of early recognition and prompt multidisciplinary intervention to prevent irreversible neurological damage and optimize patient outcomes. Furthermore, it highlights the rarity of spinal AVFs in the elderly population and emphasizes the necessity of individualized treatment approaches considering patient-specific factors.

Keywords: spinal arteriovenous fistula, tetraplegia, embolization

P092. Adult-onset metachromatic leukodystrophy – too often too late

I. SOMOIAG, R.S. BADEA, N. GRECU, C. TIU

Department of Neurology, Emergency University Hospital Bucharest, Bucharest, Romania

Introduction. Metachromatic leukodystrophy (MLD) is a genetic disorder characterized by damage to the myelin sheath affecting both the central and the peripheral nervous system. Several gene mutations lead to decreased activity of arylsulfatase A, a lysosomal enzyme, which causes the accumulation of unrecycled cerebroside sulfate. There is a strong correlation between these genetic variants and the severity of the phenotype. The adult form is particularly difficult to recognize because of its presentation with early-onset neurocognitive impairment that is often mistaken for a psychiatric disorder.

Methods. We report the case of a 41-year-old man and his long journey to the final diagnosis of adult-onset metachromatic leukodystrophy.

Results. A 41-year-old patient was hospitalized for a generalized epileptic seizure. His medical history started 15 years earlier with an abnormal CT scan after a traumatic head injury, followed by MRI examination. At that time, the patient already had neurocognitive symptoms and behavioral changes since adolescence. MRI revealed extensive atrophic changes and periventricular bihemispheric white matter lesions with a striped pattern. However, proper screening was delayed several years because of his misleading psychiatric symptoms. Finally, after his presentation in our department, arylsulfatase A activity testing and confirmatory genetic testing were performed, establishing the diagnosis.

Conclusions. With this case presentation, we highlight the importance of early recognition of dysmyelinating MRI patterns. Closely followed by genetic testing and timely diagnosis, this can provide the option of curative or disease-modifying measures.

P093. The importance of differential diagnosis in the evaluation of patients with Miller Fisher syndrome: Case report

R.A. CODREANU-BALABAN[1], D. MUSAT[1], A. AXELERAD[1,2], S.E. ISPAS[1]

[1]Department of Neurology, “Sf. Ap Andrei” Emergency County Clinical Hospital, Constanta
[2]Department of Neurology, General Medicine Faculty, Ovidius University, Constanta, Romania

Introduction. Miller Fisher syndrome is a regional variant of Guillain–Barré syndrome, characterized by cranial nerve involvement and the triad of ataxia, areflexia, and ophthalmoplegia.

Case report. We present the case of a 52-year-old patient who presented with diplopia on forward gaze and gait disturbance. The initial neurological examination revealed bilateral abducens nerve palsy, bilateral horizontal rotary nystagmus, abolished osteotendinous reflexes in the lower limbs, and diminished reflexes in the upper limbs.

Initially, the case was evaluated as myasthenic syndrome; however, the edrophonium test was negative, as were the lumbar puncture, electromyography (repetitive nerve stimulation showed no decrement), anti-acetylcholine receptor antibodies, and anti-MuSK antibodies. Brain and cervico-thoraco-lumbar spine MRI showed no abnormalities.

Corticosteroid therapy was initiated without improvement, and treatment with human immunoglobulin was decided upon, with a favorable response. At the time of discharge, the patient exhibited only mild limitation of eye movements and an ataxic gait. At 2 months post-admission, the patient’s home evolution was remarkable, with a normal neurological examination.

Conclusion. Considering the repeated lumbar puncture and electromyography results, as well as the response to human immunoglobulin treatment, the diagnosis of Miller Fisher syndrome was established. The patient had a favorable course during hospitalization and showed complete recovery at 3 months post-discharge. This case illustrates the importance of thorough evaluation and appropriate management of patients with Miller Fisher syndrome.

Keywords: Miller Fisher syndrome, ataxia, areflexia, ophthalmoplegia, human immunoglobulin, corticosteroid therapy, differential diagnosis

P094. Therapeutic approaches in the case of a patient with multiple pathology – neurological and systemic

Emilia FURDU-LUNGUT[1,2], Stefania-Cristiana DERVESTEANU[2], Alexandra-Silvia GIURCA[2]

[1]*Neurology Department, CF2 Hospital, Bucharest, Romania*

[2]*"TituMaiorescu" University, Bucharest, Romania*

Introduction. Dementia involves the loss of brain functions, usually progressive and irreversible. Pathological change occurs in the brain through the damage and death of neurons. Vascular brain malformations, such as arteriovenous malformations or dural arteriovenous fistulas, can manifest as global brain dysfunction, leading to dementia.

Case presentation. We present the case of a 61-year-old female, whose symptoms started 10 years ago, the diagnosis at that time being that of a major depressive episode. An initial MRI examination revealed multiple demyelinating lesions disseminated above and infratentorially, without clinical accusations. At that time, the patient was investigated for a possible cause of these microlesions, but without a conclusion.

The demyelinating lesions progressed over the years and the last MRI described a possible vascular malformation – a venous angioma with drainage in a periventricular vein.

The dominant mnestic and attention disorders, in correlation with a slight temporal-spatial disorientation, led us to suspect a cognitive disorder. Psychiatric and psychological examinations, in correlation with imaging investigations, proposed the diagnosis of vascular dementia.

During neurological examination, as of now, the patient presents with rotatory vertigo, positive Romberg test with unsystematized oscillations, without other significant pathological changes.

At a subsequent cardiological consultation, the patient was also diagnosed with atrial fibrillation.

Conclusions. The complexity of the patient's diagnoses leads to a difficult therapeutic approach. The dose of anticoagulant required for atrial fibrillation overlaps with the risk of intracerebral hemorrhage determined by the cerebral vascular malformation.

Keywords: vascular malformation, multiple demyelinating lesions, vascular dementia

P095. Burkitt lymphoma with central nervous involvement – case report

A. JOARZA[1], B. MOSCU[1], C. ROMAN-FILIP[1,2]

[1]*Sibiu County Emergency Clinical Hospital, Sibiu, Romania*

[2]*"Lucian Blaga" University, Sibiu, Romania*

Burkitt lymphoma is a variant of B-cell non-Hodgkin's lymphoma. Burkitt lymphoma is classified into three clinical categories: endemic, sporadic, and immunodeficiency-related. The endemic form (average age of onset 6 years) is related to the Epstein-Barr virus or malaria. The immunodeficiency-related form is associated with HIV and, to a lesser extent, with organ transplantation. The average age of diagnosis for the sporadic form is 45 years, with an annual incidence of 2.5/1 million adults.

Material and methods. The present work presents the case of a 47-year-old patient, smoker, chronic ethanol user who progressively developed, over the last 2 months, weight loss, global asymmetric motor deficit, gait and balance disorders, associated with occipital headache, nausea and vomiting.

Cranial CT + CT angio raises the suspicion of secondary intracerebral determinations, associated with leptomeningeal carcinomatosis.

Cerebral MRI: supra- and infratentorial infiltrative areas, with diffuse uptake of contrast, cytotoxic edema of the cerebellar hemispheres, which raises the suspicion of a multifocal lymphoma.

CT-TAP: no pathological changes

CSF examination: leukocytosis 0.078 10^3/µl, protein 1.037 g/l. Immunophenotype compatible with Burkitt lymphoma.

Results. Paraclinical examinations establish the diagnosis of possible Burkitt lymphoma with cerebral localization. By multidisciplinary consensus, specific oncological therapy is initiated for the patient.

Conclusion. We presented a case of Burkitt lymphoma with exclusively cerebral localization, a rare pathology, occurring in an immunocompetent patient, emphasizing the importance of the laboratory examination with flow cytometry in establishing the final diagnosis.

Keywords: Burkitt lymphoma, asymmetric ataxic tetraparesis, leptomeningeal carcinomatosis, immunophenotype

P096. First seizure – diagnosis surprises – a case report

M. MOLNAR[1,2], O. M. VANTTA[1,2], L. PERJU-DUMBRAVA[1,2]

[1]Department of Neurology, Faculty of Medicine, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

[2]Neurology I Department,Cluj County Emergency Clinical Hospital, Cluj-Napoca, Romania

Introduction. An estimated 8 to 10% of people will experience seizures at some point in their lifetime. The main objective when assessing a patient for their first seizure is to determine whether an intrinsic central nervous system dysfunction or a treatable systemic disorder caused it.

Aim. This poster aims to present a complex case of a first episode of consciousness alteration, neurosarcoidosis, and Lyme disease.

Material and methods. A female patient, aged 27 years old, was admitted to the Neurology Department of Cluj County Emergency Clinical Hospital for a seizure, bilateral parotitis, left peripheral facial paresis, and blepharitis. A thorough investigation was conducted by a multidisciplinary team, including infectious and immunological blood tests, native cerebral MRI, electroencephalography (EEG), parotid ultrasound and scintigraphy, thoracic computed tomography, and bronchoscopy with bronchoalveolar lavage.

Results. We found a positive test for acute infection with Borrelia burgdorferi after the investigations. Native brain MRI showed non-specific demyelinating cerebral lesions in the bilateral juxtacortical and deep frontoparietal white matter. The EEG revealed generalized irritative changes triggered by hyperventilation. Imaging investigations of the parotid gland suggested idiopathic bilateral parotitis. The pneumological assessment found imaging signs of bilateral hilar adenopathies that suggested stage II pulmonary sarcoidosis.

Conclusion. Sarcoidosis is a multisystemic granulomatous condition that can rarely present as Heerfordt syndrome. Eye and parotid gland inflammation, chronic fever, and palsy of the facial nerve constitute some of the symptoms, yet an association with seizures is scarcely described. Also, this case emphasizes the importance of a detailed investigation when facing a patient with a first-time seizure.

P097. MS vs stroke: case presentation

A. IACOB, I. MARCU, A. SERBAN, A. POPA, D. NACONECINII

"Prof. Dr. N. Oblu" Clinical Emergency Hospital, Iasi, Romania

Introduction. Although literature data show that multiple sclerosis doubles the risk of ischemic stroke, in our current clinical practice, we rarely encounter MS patients with acute onset of ischemic stroke.

Materials and methods. We present the case of a 56-year-old patient, known to have MS for 16 years, currently on interferon treatment, who presented to the neurology department of a county hospital for symptoms of brainstem distress (vertigo, balance disorders, dysphonia and swallowing disorders) and flaccid left hemiparesis. Cranio-cerebral CT examination revealed no acute brain lesions, and subsequent contrast MRI revealed a bulbar lesion without contrast uptake. These results imply the need for a differential diagnosis between an acute ischemic lesion and a new MS demyelinating lesion. The initial unfavorable evolution required the transfer of the patient to a university clinic where the diagnosis of ischemic stroke was confirmed, slowly evolving favorably under specific treatment.

Conclusion. Considering that the patient's initial condition at admission did not improve under corticosteroid therapy but showed slowly favorable evolution under specific treatment of an acute ischemic stroke, we advocate the diagnosis of ischemic stroke in favor of a new MS lesion as the etiology for the patient's symptoms.

Keywords: multiple sclerosis, stroke, case presentation

P098. Acute myositis – looking for a cause in the haystack. A case presentation

Cristina Aura PANEA, Vlad TIU, Alina STANCA, Bianca CHELARU, Vlad FRUNZA

Elias Emergency University Hospital, Bucharest, Romania

Background. Acute myositis can be a serious complication of numerous infections, with rhabdomyolysis leading to life-threatening consequences. We present a case of acute idiopathic myositis with severe rhabdomyolysis and associated complications following recent digestive symptoms.

Case presentation. A 38-year-old male with a history of treated primary syphilis (2020) presented with abdominal pain and digestive symptoms. The patient's condition rapidly deteriorated, exhibiting subocclusive syndrome, septic shock, and acute renal failure. Exploratory abdominal surgery revealed an entero-parietal adhesion. Extensive infectious work-up identified *Clostridium difficile* and *Blastocystis hominis*. Acute porphyria was suspected, but testing was negative. Antibiotic therapy was initiated, with resolution of infectious symptoms. Following detubation, the patient was quadriplegic, with swallowing dysfunction and myoclonus, with severe rhabdomyolysis.

The patient was transferred under our care. Lab results revealed elevated CPK (48,363 IU/L) and acute kidney injury (creatinine 3.77 mg/dL). The patient underwent five plasma exchange sessions, resulting in CPK reduction but persistent renal dysfunction. Repeated extensive immunological screening revealed only weak positivity for PM-Scl 100 antibodies. Electrophysiological studies suggested a myopathic pattern on EMG, later complicated by an added critical illness neuropathy. Muscle biopsy showed non-specific inflammation. Methylprednisolone pulse therapy was decided, with marked improvement. Following the acute episode, muscle inflammation subsided, and the patient partially recovered to his previous status.

Conclusion. This case highlights the potential severity of infectious myositis and the challenges of diagnosis. Despite extensive investigations, the etiology remained idiopathic. Patient outcomes are usually favorable if acute complications can be overcome, such as was the case for our patient.

P099. Evaluation of risk factors in the prediction of cerebral hyperperfusion syndrome – clinical case

Anca-Rafila STINGACIU[1], Ionut GHINCEA[1], Petru COLTEA[1], Monica MANISOR[1], Corina ROMAN-FILIP[1,2]

[1]*Department of Neurology, Sibiu County Emergency Clinical Hospital, Sibiu, Romania*
[2]*"Victor Papilian" Faculty of Medicine, "Lucian Blaga" University, Sibiu, Romania*

We present the case of an 81-year-old patient with known cardiac and renal pathology who suffered two strokes due to severe stenoses of the bilateral carotid arteries. Consequently, the patient underwent two carotid artery stentings (CAS) within a period of fewer than 30 days periprocedurally. Following the left CAS, the patient presented with elevated blood pressure (SBP >180 mmHg) and, after 7 days post-procedure, developed right-sided hemiplegia (0/5 MRC) and mixed aphasia. Following the administration of corticosteroids, cerebral depletive therapy, and antiepileptic medication, the patient's neurological condition improved, and he was subsequently discharged with mild right hemiparesis (4/5 MRC). At four months post-discharge, the patient remained neurologically intact, motor autonomous, and the follow-up cerebral MRI showed no changes.

Taking into consideration the risk factors used in the HISPANIAS predictive score, the patient had an 85% risk of developing CHS, due to advanced age, chronic kidney disease, and left CAS. Furthermore, he presented with other incriminating risk factors in the literature such as uncontrolled diabetes mellitus, chronic systemic hypertension with post-interventional hypertensive crisis, recent cerebral ischemia, and recent contralateral stenting (22 days).

Early recognition is crucial because delayed diagnosis can lead to a fatal prognosis. Risk factors as well as clinical predictive models based on these factors (HISPANIAS) are valuable tools in achieving this goal.

Keywords: cerebral hyperperfusion syndrome, carotid stenting, predictive factors, HISPANIAS score

P100. Thyrotoxic periodic paralysis: differential diagnosis of sudden-onset muscle weakness in a young patient

Mihai PERIANU[1], Horia NICOLAE[1,2], Roxana URDEA[1], Luminita CIMA[2,3], Ioana TAGA[1], Silviu STANCIU[1], Laurentiu TROFOSILA[1], Cristian GHITA[1], Cristina PANEA[1,2]

[1]*Neurology Clinic, Elias Emergency University Hospital, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*Endocrinology, Diabetes, Nutrition, and Metabolic Diseases Clinic, Elias Emergency University Hospital, Bucharest, Romania*

Introduction. Periodic paralysis (PP) represents a spectrum of channelopathies marked by painless muscle weakness, manifesting through hereditary transmission or acquired factors, presenting as hypokalemic or hyperkalemic states. Moreover, thyrotoxic PP, a subtype of hypokalemic PP, is particularly sporadic, predominantly among Asian males. We present the case of a young patient diagnosed with PP induced by hypokalemia, initially manifesting as sudden-onset tetraparesis.

Methods. This report discusses the differential diagnosis of sudden-onset tetraparesis in a young patient with a normal CT scan. The patient, a 37-year-old male of Asian origin with no medical history, exhibited motor deficits, with tetraparesis graded 3/5 on the BMRC scale in the left arm and both legs, and 4/5 in the right arm, alongside generalized hypotonia.

Results. Following the presentation of neurological deficits, a comprehensive diagnostic investigation was undertaken to identify the etiology. As the initial CT scan showed no abnormal findings, the investigations proceeded with laboratory analysis. The serum tests detected severe hypokalemia with a serum potassium level of 1.7 mmol/L. Corrective treatment with potassium led to subsequent symptom remission the following day. Endocrinological assessment revealed low TSH levels and elevated fT4 levels, leading to the diagnosis of hyperthyroidism. Further investigations through thyroid ultrasound raised suspicion of subacute thyroiditis. The presence of hypokalemia and subsequent recovery with treatment typically indicates hypokalemic PP, ruling out other causes of sudden-onset motor deficits such as vascular events, myelopathies, peripheral neuropathies, myasthenic crises, etc.

Conclusion. This case underlines the importance of a thorough differential diagnosis for sudden-onset muscle weakness, thyrotoxic periodic paralysis being a rare but reversible cause with a favorable outcome.

Keywords: periodic paralysis, tetraparesis, hypokalemia, thyrotoxic periodic paralysis, hyperthyroidism, subacute thyroiditis

P101. Management of immune thrombocytopenia in a multiple sclerosis patient: a case report

R. IATAN[1], C. TIU[1,2]

[1]*Department of Neurology, University Emergency Hospital Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Immune thrombocytopenic purpura (ITP) and multiple sclerosis (MS) are two distinct autoimmune disorders characterized by dysregulation of the immune system, leading to diverse clinical manifestations. While drug-induced thrombocytopenia is a frequent complication of several DMTs for MS, the co-occurrence of spontaneous ITP and MS has been reported, and management requires a tailored approach that considers the interplay between autoimmune processes and the impact of immunomodulatory therapies on both conditions.

Case report. We describe a 56-year-old woman with a longstanding history of relapsing-remitting MS, managed with disease-modifying therapy (DMT), who presented with petechiae and ecchymoses suggestive of ITP. Diagnostic evaluation confirmed severe thrombocytopenia without evidence of alternative etiologies, consistent with a diagnosis of ITP. Corticosteroids were initiated, but due to inadequate response, the patient required romiplostim (a thrombopoietin receptor agonist) as well. At that point, the patient also needed adjustment in her MS treatment, seeing as she recently switched from glatiramer acetate to dimethyl fumarate because of local adverse reactions. However, she had to stop treatment with Tecfidera soon after because of an allergic reaction. Considering the need for a therapy targeting both MS and ITP, B-cell depleting agents emerged as a rational choice. Ofatumumab, a fully-human monoclonal antibody targeting CD20-positive B cells, was selected due to its efficacy in MS and potential benefit in ITP, alongside its favorable safety profile. It was initiated with close surveillance of both MS and ITP manifestations, including neurological status and platelet counts, with no notable side effects. The use of ofatumumab in this case underscores the potential for a targeted approach to address autoimmune comorbidities, leveraging the shared pathogenic role of B cells in MS and ITP.

Conclusion. This case highlights the association of two autoimmune diseases, where anti-CD20 therapy was chosen as a disease-modifying therapy for multiple sclerosis with the hope of it having a favorable clinical response in ITP as well, seeing as CD20 B-cell depleting therapy has shown efficacy in chronic refractory ITP. Further research is warranted to elucidate the underlying immunological mechanisms linking MS and ITP, facilitating the development of targeted therapeutic strategies to address both conditions simultaneously.

P102. Non-Hodgkin lymphoma diagnosed by neurological onset: a case report of secondary cerebral lymphoma

A. PAVEL[1], O. RUSU[1], C. TIU[1,2]

[1]*Department of Neurology, University Emergency Hospital Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Non-Hodgkin's lymphoma may affect the nervous system at any level, through direct invasion or paraneoplastic manifestations. It's rare to diagnose secondary cerebral lymphoma prior to establishing the diagnosis of systemic lymphoma.

Methods. Clinical case of a 64-year-old patient, admitted for postural instability with own height falling, confusional state, and weight loss. Symptoms started 3 months earlier and worsened progressively. He also developed an expanding mandibular mass.

Results. At admission, the patient was confused, hypophonic, presented bradykinesia and postural tremor of the upper limbs, cogwheel phenomenon, tetraparesis with paraparesis predominance, and had postural instability. His clinical condition gradually deteriorated, aggravating motor deficits and tremor, with loss of deep tendon reflexes. Nerve conduction studies revealed axonal sensorimotor polyneuropathy. Mandibular mass biopsy established the diagnosis of non-Hodgkin's lymphoma. The patient's condition continued to decline: he became unable to speak, tremor and tetraparesis worsened, and he developed severe muscle atrophy. Further brain MRI described extensive T2/FLAIR hyperintense lesions in the fronto-parieto-occipital white matter, with invasion of the pontobulbar pyramidal tract, cerebellar peduncles, and cerebellum, causing no mass effect and associated with contrast-enhanced and choline peak in the splenium of the corpus callosum. Subsequent differential diagnosis between secondary lymphoma and PML followed—the latter was excluded by cerebrospinal-fluid-PCR assays.

Conclusion. The case's particularity consists in the fact that the patient did not present with symptoms specific to the underlying condition, but with neurological degradation. Through paraneoplastic effects and direct cerebral invasion, the diagnosis of non-Hodgkin's lymphoma with secondary central and peripheral neurological involvement was established.

Keywords: lymphoma, tetraparesis

P103. When it is not what it seems to be: add some contrast

Alexandra RESIGA[1], Athena RIBIGAN[1,2], Ioana VASILIU[1], Mara MOLDOVAN[1], Sebastian STEFAN[1], Andrei CHESNOIU[1], Raluca BADEA[1,2], Octaviana RUSU[1], Florina ANTOCHI[1], Cristina TIU[1,2]

[1]*University Emergency Hospital Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Leptomeningeal carcinomatosis (LC) is a rare but devastating complication of advanced neoplasia. LC represents a diagnostic challenge due to its diverse neurological manifestations, often mimicking other pathologies.

Materials and methods. We present a case of a 67-year-old female exhibiting progressive symptoms for a month following an upper respiratory tract infection. Neurological examination revealed decreased visual acuity, bilateral peripheral facial palsy, severe dysphagia and dysphonia, tetraparesis (4/5 MRC), distal paresthesias, and right limb hypoesthesia. Initial assessment, according to a nerve conduction study performed prior to the presentation, raised the suspicion of acute polyradiculoneuritis. Brain MRI carried out before hospitalization was suggestive of temporal and occipital subacute strokes.

Results. Nerve conduction studies depicted low motor amplitudes but also low F-wave persistence. Needle examination showed a slightly decreased activation pattern, overall suggestive of central dysfunction. Brain MRI revealed leptomeningeal thickenings with contrast enhancement and two juxtacortical nodular lesions located in the left occipital and temporal regions with diffusion restriction, decreased ADC, and discrete marginal contrast enhancement, suggestive of LC and cerebral metastases. Cerebrospinal fluid analysis showed an increased cell count (cytologic examination revealing rare atypical cells), increased proteins, and low glucose. CT scan of the thorax, abdomen, and pelvis revealed the presence of a colorectal tumor associated with high values of the markers for gastrointestinal tract neoplasia.

Conclusion. This case highlights the importance of a careful clinical exam and appropriate work-up in order to assess the correct diagnosis. LC is a rare finding in the natural invasion-metastasis cascade of colorectal cancer, with a poor prognosis and limited treatment options.

P104. Drug-induced choreoathetosis in a patient with Moyamoya-like vasculopathy and MYH9-related platelet disorder: a case report

Ioana VASILIU[1], Athena RIBIGAN[1,2], Alexandra RESIGA[1], Mara MOLDOVAN[1], Sebastian STEFAN[1], Andrei CHESNOIU[1], Raluca BADEA[1,2], Octaviana RUSU[1], Florina Antochi[1], Cristina TIU[1,2]

[1]*University Emergency Hospital Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Background. Moyamoya-like vasculopathy (MMV) can occur in response to other underlying pathological processes, including hematologic diseases. MYH9-related platelet disorder (MYH9-RPD) is a group of hereditary macrothrombocytopenias characterized by thrombocytopenia, giant platelets, and hearing loss, nephritis, or cataract. The aim of our paper is to present a rare case of a patient with MYH9-RPD and MMV.

Methods. A 33-year-old female was admitted to our department with speech impairment and involuntary movements of the upper limbs (onset two weeks prior to admission). The patient had a medical history of MYH9-RPD, MMV, and focal seizures treated with levetiracetam. Neurological examination revealed involuntary movements suggestive of choreoathetosis of both upper limbs (right > left) and tongue, and hypoacusis.

Results. Computed tomography angiography of the supra-aortic trunks, compared with previous examinations, showed an evolution from stenosis to occlusion of both distal intracranial internal carotid arteries and both proximal middle and anterior cerebral arteries, with engorged pial collateral vessels. Cerebral MRI depicted the presence of multiple T2/FLAIR hyperintensities located in both hemispheres, suggesting ischemic lesions (one of the lesions located in the left head of the caudate nucleus showed signs of petechial hemorrhagic transformation in susceptibility-weighted imaging). Resolution of symptoms was obtained after replacing levetiracetam with lamotrigine in association with clonazepam.

Conclusion. We chose to present this case to raise awareness that movement disorders may be induced by levetiracetam and also to emphasize that various involuntary movements can be seen in patients with MMV, with choreoathetosis being one of the rarest, secondary to basal ganglia lesions or hypoperfusion.

P105. Concomitant atypical PRES and Takotsubo cardiomyopathy – A case report

G. NICOLESCU[1], L. CRACIUN[1], V. M. POMPILIAN[1], B. O. POPESCU[1,2,3]

[1]Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania
[2]Department of Clinical Neurosciences, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Laboratory of Ultrastructural Pathology, "Victor Babes" National Institute of Pathology, Bucharest, Romania

Purpose. To highlight the possible association between atypical posterior reversible encephalopathy syndrome (PRES), Takotsubo cardiomyopathy (TC), and the administration of adrenaline.

Method. We present the case of a 61-year-old female with a history of scleroderma, rheumatoid arthritis, and polymyositis overlap, who developed concomitant atypical PRES and TC after the administration of adrenaline.

Results. PRES is a clinical and neuroradiological condition that could be linked to the administration of certain medications. TC is characterized by reversible left ventricle systolic dysfunction, usually associated with stressors as triggers. Our patient presented to the hospital with significant weight loss and fatigue over the last month. Further evaluation revealed a pulmonary tumor. During bronchoscopy and biopsy, there was significant bleeding, requiring local administration of adrenaline. Subsequently, the patient experienced a generalized tonic-clonic seizure, followed by persistent post-ictal left hemiparesis and chest pain. Diagnostic workup included an MRI that showed T2/FLAIR hyperintense lesions in the right parietal region, without restricted diffusion or contrast enhancement, echocardiography that showed left ventricular hypokinesia, ECG showing ST-T elevation, and increased hs-troponin levels. Thus, a myocardial infarction was suspected, and coronarography was performed, but it was normal. After 24 hours, the cardiac abnormalities disappeared, and the neurological deficit began slowly improving.

Conclusion. Based on MRI findings and the reversible nature of both the cardiac abnormalities and neurological deficits, we concluded that the patient suffered from concomitant atypical PRES and Takotsubo cardiomyopathy. This underlines the similar pathophysiological mechanisms that these rare conditions share.

Keywords: atypical PRES, Takotsubo, adrenaline, bleeding, seizure, reversible neurological deficit

P106. Diagnostic and treatment updates in Weber syndrome (starting from a clinical case)

S. SOCOLOV[1], A. MICLAUSANU[1], T. G. SCHREINER[1,2]

[1]Neurology 1 Clinic, "Prof. Dr. N. Oblu" Clinical Emergency Hospital, Iasi, Romania
[2]Department "Medicale III", Faculty of Medicine, "Gr. T. Popa" University of Medicine and Pharmacy Iasi, Romania

Introduction. Weber syndrome, characterized by hemiparesis and contralateral oculomotor palsy, is one of the main peduncular syndromes. Even though the first case was published in 1863, the rarity of this pathology still leads to difficulties in its diagnosis and treatment.

Case presentation. A 75-year-old left-handed hypertensive patient with a prior diagnosis of regressive left hemiparesis and aphasia due to a right Sylvian stroke in 2017 was admitted to the Emergency Room of „Prof Dr N Oblu" Hospital in Iasi due to a sudden worsening of the motor deficit on his left side and right eyelid ptosis with limited adduction and vertical movements. The emergency CT scan revealed acute hypodensities in the right occipital, thalamic, and superior cerebellar areas, corresponding to the vascular territory of the posterior cerebral artery. An MRI scan later confirmed a lesion in the right mesencephalon, thus establishing the diagnosis of Weber syndrome. We started antihypertensive treatment in combination with antiplatelet therapy, statins, and anti-edematous treatment, with an improvement in the motor deficit.

Conclusion. Weber syndrome remains a rare clinical manifestation in which the semiology can be explained by imaging, while management is adapted to each case's particularities.

Keywords: Weber syndrome, hemiplegia, oculomotor palsy, mesencephalic stroke

P107. Parkinson's disease vs psychogenic parkinsonism – hardware or software problem?

Carmen CROITORU, Madalina LUCA, Sebastian MORCOV, Oana BISOC, Liliana CUCOS

"Prof. Dr. N. Oblu" Clinical Emergency Hospital, Iasi, Romania

Psychogenic parkinsonism is the rarest manifestation of the spectrum of psychogenic motor disorders and represents a challenge from the point of view of diagnosis and therapeutic approach. Although there are elements that suggest a psychogenic substrate—such as the sudden onset, variable clinic over time, pre-existing psychiatric pathology, hypertonia due to voluntary resistance with distractibility, incongruent dyskinesias to levodopa, and normal imaging—differentiation from Parkinson's disease is difficult, especially in advanced cases, with the variability of motor symptoms, secondary manifestations of treatment, and accusations from the psychiatric sphere.

We present the case of a 36-year-old patient, diagnosed with Parkinson's disease at the age of 32, whose atypical evolution under successive treatment schemes raised differential diagnosis problems between advanced Parkinson's disease with rigid off phenomena alternating with episodes of dramatic dyskinesias, and psychogenic parkinsonism, with behavioral disorders and episodes of intense psychomotor agitation. Such cases require a multidisciplinary approach for the correct diagnosis of a potentially curable pathology, but also for the development of an individualized treatment plan.

Keywords: Parkinson's, psychogenic parkinsonism, multidisciplinary

P108. Right middle cerebral artery stroke with contralateral left peripheral facial palsy. A case study

I. PARASCHIV[1], D. TULBA[1,2], B. O.POPESCU[1,2,3]

[1]*Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*Department of Molecular Medicine, "Victor Babes" National Institute of Pathology, Bucharest, Romania*

Introduction. Facial palsy is classified into peripheral and central types, depending on the damaged motor neuron. Central facial palsy (CFP) occurs due to damage of the central motor neuron and typically affects the lower half of the contralateral face because the frontal and orbicularis oculi muscles receive innervation from both cerebral hemispheres. Peripheral facial palsy (PFP) is ipsilateral to the lesion and affects the entire hemiface, including the forehead and orbicularis oculi muscles.

Method. We present a patient with right middle cerebral artery (MCA) stroke manifested with left hemiparesis and left PFP-like symptoms.

Result. A 43-year-old male patient was admitted for sudden onset left hemiparesis (more severe in the upper limb - plegia) and left facial palsy with involvement of the upper face (including frontalis muscle). He also had left tactile and myo-arthro-kinetic hypoesthesia. We performed a brain CT followed by MRI, the latter showing an extensive ischemic area in MCA territory with minimal hemorrhagic transformation. With involvement of the central motor neuron destined to the left hemiface, a CFP would be expected, but our patient also showed involvement of the forehead.

Conclusions. While facial palsy is classically described as central or peripheral depending on the damaged motor neuron, a peripheral-type presentation does not exclude an acute cerebrovascular event in the anterior circulation. Although cortico-nuclear fibers for the upper face usually synapse with both facial nuclei in the pons, a small percentage (approximately 6.6%) of CFPs present with upper face weakness, mimicking a PFP, especially when the right hemisphere is involved.

P109. Cerebrovascular comorbidities exposed by infectious status

Ioana STIRBU[1],Antonia-Gabriela DIUDEA[1], Dana Marieta FODOR[1,2], Lacramioara PERJU-DUMBRAVA[1,2]

[1]Department of Neurology, Cluj County Emergency Hospital, Cluj-Napoca, Romania
[2]Department of Neurology,Faculty of Medicine, "IuliuHatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

This poster aims to present a complex case of cerebrovascular comorbidities exposed by infectious status.

Methods. A 46-year-old patient was presented to our service in 2023 with dysphagia, odynophagia, onset in an infectious context. Neurological examination shows anisocoria, ptosis on the left eye, and left unilateral cranial nerve palsy (IX, X, XI, XII). Brain magnetic resonance imaging (MRI) detects left pontine paramedian vascular malformation, telangiectasia with signs of active bleeding. Investigations were completed with computed tomography (CT) angiography of supra-aortic trunks and cerebral perfusion CT revealing distal left carotid artery dissection from the origin, predominantly extracranial, with false thrombosed lumen, and blood flow in the left hemisphere symmetrical with the right hemisphere.

Results. The patient was treated conservatively due to the presence of telangiectasia with signs of active bleeding. At the three-month evaluation, a reduction in symptomatology and clinical neurological signs was detected.

The peculiarity of the case consists in the onset of symptoms in an infectious context, young age of the patient, and complete remission of symptoms.

Conclusion. Acute infection revealed telangiectasia and internal carotid artery dissection. The presence of pontine microhemorrhage restricted therapy options.

Keywords: anisocoria, ptosis, carotid artery dissection, telangiectasia

P110. Right middle cerebral artery stroke with contralateral left peripheral facial palsy. A case study

C. HERGHELEGIU[1,2], N. MARGINEAN[1,2], L. CHIRITA-CRACIUN[1,2], C. RUSULEANU[3], M. ENACHE[4], I. GELATU[5], L. DUMITRESCU[1,2], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,6]

[1]2nd Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]Clinical Neuroscience Department, Faculty of Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Outpatient Department - Endocrinology, Colentina Clinical Hospital, Bucharest, Romania
[4]1st Internal Medicine Department, Colentina Clinical Hospital, Bucharest, Romania
[5]2nd Hematology Department, Colentina Clinical Hospital, Bucharest, Romania
[6]Neuroscience and Experimental Myology Laboratory, Laboratory of Cell Biology, "Victor Babes" National Institute of Pathology, Bucharest, Romania

Introduction. Severe hypocalcemia may result in encephalopathy and seizures.

Materials and methods. We report the case of a 46-year-old female, without significant medical history, presenting with frequent generalized tonic-clonic seizures with sudden onset 4 months prior, in the absence of an obvious trigger or recent febrile illness. She also complained of cognitive impairment but was able to carry on with her daily activities. Until the seizures became a concern, she consumed around 2.5 liters of sugary caffeinated soft drinks per day, and 1.5 liters of instant coffee with sugar and milk. She had no personal or family history of endocrine or autoimmune disorders and denied any surgical interventions. The neurologic examination was normal except for cognitive impairment with temporal disorientation (MMSE 19 points). The brain CT scan revealed bilateral calcifications of the basal ganglia and insula. The EEG showed no pathological findings. The blood workup revealed severe hypocalcemia, with reference range albumin and magnesium levels, and hyperphosphatemia, inappropriately low levels of parathyroid hormone (at the lower reference limit), very low 25-hydroxyvitamin D levels, moderate microcytic anemia, iron deficiency, and hypovitaminosis B12; the thyroid-stimulating hormone and cortisol levels were within reference range.

Results/Conclusions. The patient was started on levetiracetam and the seizures subsided. A diagnosis of probable autoimmune hypoparathyroidism was made. The hypocalcemia was difficult to correct, but once corrected, the neurocognitive status returned to baseline levels. The potential role of the high quantities of sugary caffeinated drinks in exacerbating hypocalcemia in people with hypoparathyroidism is discussed.

Keywords: generalized tonic-clonic seizures, basal ganglia and insular calcifications, autoimmune hypoparathyroidism, severe hypocalcemia, sugary caffeinated drinks

P111. Clinical and electrophysiological features in a case of multifocal motor neuropathy

Andrei STANESCU, Simona PETRESCU, Ionela CODITA, Cristina A. PANEA

[1]Elias Emergency University Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Multifocal motor neuropathy (MMN) is a rare, immune-mediated neurological condition, with a prevalence of 0.6–2/100,000. It affects males more frequently, with a mean onset ranging from age 30 to 40 years.

Method and results. A 48-year-old male patient was diagnosed with MMN following clinical and paraclinical evaluation, including electrophysiological and biological findings. The patient has a history of progressive disease starting 12 years ago, with asymmetrical, mainly distal involvement of the upper limbs, followed by involvement of the lower limbs. Clinical examination demonstrated significant, mainly distal, amyotrophies in the upper limbs more than the lower limbs, with left-right asymmetry; the left side was more affected.

The electrophysiological exam showed conduction blocks outside of entrapment sites, with multifocal motor involvement, and the absence of sensory abnormalities. Biologically, the patient tested positive for anti-ganglioside M1 (anti-GM1) antibodies.

Conclusion and discussion. The differential diagnosis of asymmetrical motor deficits includes motor neuron disease and a specific form of chronic inflammatory demyelinating polyneuropathy, namely Lewis-Sumner syndrome. The electrophysiological exam (neurography) and the presence of anti-GM1 serum antibodies led to the diagnosis in the presented case.

P112. Aggressive vertebral hemangioma causing spinal cord compression: An unusual etiology of paraparesis

Mara MOLDOVAN[1], Athena RIBIGAN[1,2], Alexandra RESIGA[1], Ioana VASILIU[1], Sebastian STEFAN[1], Andrei CHESNOIU[1], Florina ANTOCHI[1]

[1]University Emergency Hospital Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Vertebral hemangiomas are common benign lesions of the vertebral body. These lesions involve a proliferation of normal capillary and venous structures that may subsequently cause displacement of bone and, in rare cases, extension into the spinal canal with secondary cord compression, leading to neurological symptoms.

We present the case of a 22-year-old patient who presented to our clinic with progressive walking impairment, urinary incontinence, and paresthesia in the lower limbs. The neurological examination showed spastic paraparesis, superficial sensation and joint position sense impairment, and ataxia of the lower limbs. The vertebral-spinal MRI showed a T1-hypointense, T2-hyperintense, enhancing vertebral mass at the T4 level with a mottled appearance. The lesion involved the vertebral pedicles and the left vertebral foramen, showing epidural extension and spinal cord compression. Digital subtraction angiography of the spinal vessels depicted multisegmental feeding branches from bilateral intercostal arteries.

Results. Decompressive laminectomy was performed with anterior resection of the compressive hemangiomatous mass. The patient showed significant clinical improvement. One year later, the MRI revealed a T4 vertebra compression fracture, and vertebroplasty was performed.

Conclusions. We present this case to highlight that aggressive vertebral hemangiomas can, in rare cases, lead to spinal cord symptoms. This case also emphasizes the importance of a multidisciplinary team in the assessment and treatment of this pathology. Vertebroplasty is a valuable option for the treatment of vertebral hemangiomas, often yielding excellent results. the multiple complications that may occur.

P113. Simultaneous arterial and cerebral venous sinus thrombosis in the postpartum period: A rare case report

Mara MOLDOVAN[1], Athena RIBIGAN[1,2], Alida CIOCAN[1], Patricia IOAN[1], Octavian IOGHEN[1], Sebastian STEFAN[1], Florina ANTOCHI[1]

[1]*University Emergency Hospital Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Background. The peripartum period is associated with an increased susceptibility to hypercoagulable states due to changes in the hemodynamic, vascular structural, and coagulation systems that occur during pregnancy and postpartum. We report an uncommon case of concurrent arterial and cerebral venous sinus thrombosis in the postpartum period, which required prompt intervention.

Methods. A 31-year-old patient, who delivered one month prior, presented with a confusional state accompanied by occipital headache. These symptoms emerged a week before presentation. The clinical exam revealed bilateral horizontal-gyratory nystagmus and left hemianopia. The medical history included a prior spontaneous abortion in the first trimester caused by placental infarct and a high titer of lupus anticoagulant antibody found on previous testing.

Results. Initial cerebral magnetic resonance imaging (MRI) demonstrated infarcts in the left cerebellar, right occipital, and left capsulo-thalamic regions. Subsequent MRI with MR angiography and venography revealed multiple ischemic lesions in the vertebrobasilar territory, left vertebral artery thrombosis, and thrombosis in the left sigmoid and transverse sinuses. The cerebrospinal fluid study was non-contributory. Investigations for infections, malignancy, autoimmune disease, thrombophilic mutations, and vasculitis yielded negative results. Treatment with aspirin and anticoagulants (antivitamin K) was initiated, leading to a favorable outcome. Post-discharge MRI showed radiological resolution of the filling defects in the cerebral venous sinuses.

Conclusion. Simultaneous arterial and cerebral venous sinus thrombosis in the postpartum period is a very rare but serious complication. Early recognition, thorough investigation, and appropriate anticoagulation therapy are crucial for successful management and positive patient outcomes.

P114. The way to respond to preventive therapy of migraine episodes with anti-CGRP monoclonal antibody in the first 3 months – A case series

Clara PROFIRESCU, Simona PETRESCU, Maria Melania MARTOIU, Cristina Aura PANEA

[1]*Elias Emergency University Hospital, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Migraine represents the second leading cause of disability after stroke, especially affecting women. Galcanezumab, a monoclonal antibody against CGRP (calcitonin gene-related peptide), demonstrates efficacy in reducing the frequency and severity of migraine attacks.

Methods and results. Four patients—three females and one male, aged between 28 and 47 years—with episodic migraines were followed up. They reported migraine episodes lasting less than 15 days per month. The MIDAS (Migraine Disability Assessment Score) and Montreal Cognitive Assessment (MoCA) tests were applied at baseline and after 3 months of Galcanezumab treatment.

At baseline, the MIDAS score was above 20 points, indicating severe disability, with patients reporting difficulties in concentration and impairment of social activities. MoCA scores at baseline were between 20 and 29 points.

After 3 months, patients reported a significant reduction in episode frequency to 1-2 per month, a MIDAS score of 0 points, and MoCA scores over 26 points, indicating improved cognitive performance. Patients also reported a reduction in migraine episodes related to triggers such as consumption of foods like red wine, cheeses, and the perimenstrual period, but not for weather-related triggers.

Conclusions. Preventive therapy with monoclonal antibodies against CGRP has a rapid and consistent effect on migraine-related disability, as observed in the reported cases. Among the reported triggering factors, weather changes were not influenced by the therapy.

Keywords: Galcanezumab, episodic migraines

P115. Left atrial strain – a helpful method in establishing the etiology of transient ischemic attacks in patients with silent paroxysmal atrial fibrillation

Sergiu Florin ARNAUTU,
Diana Aurora ARNAUTU,
Mirela Cleopatra TOMESCU, Andrei HAJEVSCHI,
Catalin Dragos JIANU

"Victor Babes" University of Medicine and Pharmacy, Timisoara, Romania

Background. Patients with transient ischemic attacks (TIAs) often present asymptomatically and may have paroxysmal atrial fibrillation (AF). Since AF originates in the atria, we aimed to investigate whether abnormalities in left atrial structure and function could help identify the cardioembolic etiology of TIAs in patients with sinus rhythm but documented episodes of paroxysmal AF.

Methods. One hundred ninety patients with TIAs were retrospectively included in the study and divided into two groups, according to the presence (group I) or absence (group II) of documented paroxysmal AF. Documentation of paroxysmal AF was based on medical records. Cardiac ultrasound assessments were performed in sinus rhythm, at least 14 days after the onset of TIA, to avoid assessing atrial stunning.

Results. Group I patients were older, more frequently female, with a history of stroke or TIA, and had a higher CHA2DS2-VASc score. They also presented with larger left atrial volumes, lower left atrial emptying fraction, and significantly impaired left atrial deformation patterns. Multivariate logistic regression identified three variables that were independently associated with paroxysmal AF: age, left atrial reservoir strain, and left atrial emptying fraction ($P<0.0001$). The cut-off levels for these variables were age >55 years, reservoir strain < -17%, and emptying fraction <51%.

Conclusions. This study demonstrates that left atrial strains are independent predictors of paroxysmal AF in TIA patients and could be helpful in identifying the cardioembolic etiology of TIAs.

Keywords: paroxysmal atrial fibrillation, speckle tracking echocardiography, transient ischemic attack

P116. The predictive value of carotid artery strain and strain-rate in assessing the 3-year risk for stroke and acute coronary syndrome in patients with metabolic syndrome

Sergiu Florin ARNAUTU, Andrei HAJEVSCHI,
Catalin Dragos JIANU,
Mirela Cleopatra TOMESCU,
Diana Aurora ARNAUTU

"Victor Babes" University of Medicine and Pharmacy, Timisoara, Romania

Purpose. The goal of this study was to determine whether carotid strain and strain rate can predict major cardiovascular events (MACE) in individuals with metabolic syndrome (MS) over a 3-year period.

Methods. In this prospective observational study, 220 adult MS patients (60.7 ± 7.5 years old, 53% male) were enrolled. Two-dimensional common carotid artery (CCA) speckle-tracking ultrasound was used to measure the peak circumferential strain (CS) and peak circumferential strain rate (CSR). Clinical outcomes were assessed during a three-year follow-up.

Results. After 3 years, 14 (7%) of the patients experienced MACE: 8 (4%) had an atherothrombotic ischemic stroke, 4 (2%) had acute coronary syndrome (ACS), and 2 (1%) were hospitalized for heart failure. Univariate regression analysis of clinical and echocardiographic characteristics revealed that age, systemic hypertension, diabetes mellitus, and CCA circumferential strain and strain rate were significantly associated with the risk of MACE. Multivariate logistic regression identified two independent predictors of MACE in MS patients: CCA-related CS (%) and CSR (1/s), $p < 0.01$. The receiver operating characteristic (ROC) curve analyses of these independent predictors showed good sensitivities and specificities. For CS, the area under the curve (AUC) was 0.806, with a sensitivity of 82.6%, specificity of 79.2%, and $p < 0.0001$; for CSR, AUC was 0.779, sensitivity of 82.6%, specificity of 72.4%, and $p < 0.0001$. The optimal cut-off values were ≤2.9% for carotid CS and ≤0.35 s^{-1} for carotid CSR. Kaplan-Meier survival curves demonstrated that MACE-free survival, ischemic stroke-free survival, and ACS-free survival were significantly lower in MS patients with lower carotid CS and CSR ($p < 0.0001$).

Conclusions. Carotid CS and CSR were independent predictors of major cardiovascular and cerebrovascular events in MS patients without established cardiovascular disease. Carotid deformation could serve as a valuable early prognostic indicator for cardiovascular risk in this population.

Keywords: major cardiovascular events, metabolic syndrome, carotid strain, carotid strain rate, subclinical atherosclerosis

P117. Multiple sclerosis patients self-assessment of quality of life: A real-world data analysis on key aspects

M. NEATU[1,2], A. COVALIU[1,2], A. JUGURT[1,2], I. IONITA[1,2], A. HERA[1,2,3]

[1]Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Cell Biology Laboratory, Neurosciences and Experimental Myology, National Institute of Pathology "Victor Babes", Bucharest, Romania

Purpose. Multiple sclerosis (MS) presents with complex clinical manifestations that significantly affect patients' quality of life. This study aims to identify negative prognostic factors to better understand individual patient experiences, as patient-reported outcomes (PROs) provide insights into the effectiveness of care from the patient's perspective.

Materials and methods. This was a retrospective longitudinal study conducted at the Colentina Clinical Hospital MS Center, involving 48 patients enrolled in the National Health Program and monitored between 01.06.2023 and 31.01.2024. Data were collected through self-administered questionnaires and analyzed using Microsoft Excel 2010. The correlations between specific items were determined using Pearson coefficient, Spearman coefficient, ANOVA, T-test, and Chi-squared test.

Results. The 48 patients had an average age of 48.52 ± 9.77 years and a mean EDSS score of 2.67 ± 1.80. Common disabling symptoms included sleep disorders (68.75%), paresthesia (64.58%), and pain (58.33%). Although the duration of the disease did not significantly influence quality of life, measured by the MSQOL-54 scale ($p=0.23$), it was associated with factors such as disability, perceived pain, energy level, depression, and fatigue ($p<0.01$). Background treatment affected both mobility (T25FW) and perceived fatigue (MFIS scale), which, in turn, impacted the quality of life. Notably, patients who continued their professional activities, despite receiving a disability certificate, had more favorable outcomes ($p=0.0035$).

Conclusions. This analysis highlights the importance of managing MS patients holistically, with a focus on personalized intervention strategies. A tailored approach that addresses individual symptoms, treatment regimens, and lifestyle factors is crucial to improving patient quality of life.

Keywords: multiple sclerosis, quality of life, prognostic factors, disability

P118. Clinical predictors in multiple sclerosis disability: An observational cohort study

Daniel ISIP, Laura BARCUTEAN, Iulian ROMAN, Rodica BALASA

[1]"George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania
[2]Neurology 1 Clinic, Mures County Emergency Clinical Hospital, Targu Mures, Romania

Introduction. The clinical course of multiple sclerosis (MS) is highly variable, and predicting disability based solely on clinical parameters is challenging.

Objective. To assess the time from the onset of symptoms to reaching an Expanded Disability Status Scale (EDSS) score of 4.0 and 6.0 in a cohort of patients with MS (PwMS) who have had the disease for more than 10 years, and who have been treated with interferon beta (IFNbeta).

Materials and methods. We used Kaplan-Meier and Cox analysis to assess the moment and predictors for reaching significant disability in a cohort of 171 patients with a disease duration of more than 10 years.

Results. PwMS reached an EDSS of 4.0 in 28 years and an EDSS of 6.0 in 34 years from the onset of symptoms, with no gender differences ($p= 0.59$; $p=0.28$). The overall median time for reaching an EDSS of 4.0 based on living environment was 29.98 years for patients in urban areas and 27.86 years for those in rural areas ($p=0.65$). For EDSS 6.0, the median time was 33.27 years for urban patients and 34.57 years for rural patients ($p=0.425$). Regarding the onset of symptoms, patients with optic neuritis had the longest median survival time to reach EDSS 4.0 compared to those with a polysymptomatic onset (32 years vs. 7 years, $p=0.01$). A statistically significant relationship was found between the total number of relapses and the likelihood of reaching EDSS 4.0 ($\chi^2 = 4.71$; $p=0.03$), but not 6.0.

Conclusions. There was no statistically significant difference between PwMS in reaching an EDSS of 4.0 or 6.0 based on gender or living environment. The total number of relapses was a determinant for reaching an EDSS of 4.0, but not for 6.0.

P119. Multiple autoimmune syndrome presenting with Lambert-Eaton myasthenic syndrome

D. S. STAN[1], C. MOSTEANU-DIACONU[1], A. MUJDABA-ELMI[1], O. E. MORARI[1], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,3], A. NICOLAU[4]

[1]2[nd] Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]Clinical Neuroscience Department, Faculty of Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Neuroscience and Experimental Myology Laboratory, Laboratory of Cell Biology, "Victor Babes" National Institute of Pathology, Bucharest, Romania
[4]1[st] Internal Medicine Department, Colentina Clinical Hospital, Bucharest, Romania

Lambert-Eaton myasthenic syndrome (LEMS) is a rare disorder that affects the presynaptic end of the neuromuscular junction. It is characterized by an autoimmune response mediated by anti-voltage-gated calcium channel (VGCC) antibodies, leading to reduced acetylcholine release and muscle weakness. The non-paraneoplastic variant often occurs in association with other autoimmune diseases.

We report two cases of multiple autoimmune syndrome presenting with neurological findings consistent with LEMS. Both patients were middle-aged women (56 and 57 years old, respectively) who were admitted to our clinic for progressive proximal leg weakness and bulbar signs and symptoms.

The first patient had a history of smoking and had successfully treated breast carcinoma 13 years prior, while the second patient had no associated diseases or risk factors. Neurological examination revealed ptosis, diplopia, and proximal quadriparesis. Electrodiagnostic studies showed low compound muscle action potential (CMAP) amplitudes with postexercise facilitation and significant decrement with repetitive low-frequency stimulation. Both patients were negative for anti-acetylcholine receptor and anti-MUSK antibodies but had positive titres of anti-VGCC antibodies. Both patients showed partial response to cholinesterase inhibitors. Screening for underlying neoplasia was negative.

Through autoimmune disease screening, the first patient was diagnosed with Sjogren's syndrome, and the second patient was diagnosed with systemic lupus erythematosus. Both patients were also diagnosed with autoimmune thyroiditis.

We report two instances of LEMS coexisting with both organ-specific and systemic autoimmune diseases, highlighting the need for a multidisciplinary approach to management and treatment.

Keywords: Lambert-Eaton myasthenic syndrome, systemic lupus erythematosus, Sjögren's syndrome, autoimmune, neuromuscular junction

P120. Arterial ischemic stroke in young adults and hereditary thrombophilia: a case series and literature review

M. MOISII[1], C. G. CROITORU[1,2], A. CHIRIAC[1], D. HODOROG[1,2], D. I. CUCIUREANU[1,2]

[1]Neurology Clinic, "Prof. Dr. N. Oblu" Emergency Clinical Hospital, Iasi, Romania
[2]"Grigore.T. Popa" University of Medicine and Pharmacy, Iasi, Romania

The association between hereditary thrombophilia and arterial thrombosis, particularly arterial ischemic stroke, remains unclear, despite its established role as a risk factor for venous thromboembolism.

This study aimed to examine the potential link between hereditary thrombophilia – namely factor V Leiden, prothrombin mutation G20210A, MTHFR gene mutations C677T and A1298T, PAI-1 gene mutation, protein C deficiency, protein S deficiency, and antithrombin deficiency – and the occurrence of arterial ischemic stroke. We analyzed a series of 12 consecutive cases of young patients under the age of 50 diagnosed with arterial ischemic stroke, either in the anterior or posterior cerebral vascular systems. These cases were identified at the Neurology Clinic of the Emergency Clinical Hospital "Prof. Dr. N. Oblu" Iasi between January 1, 2023, and March 1, 2024, and were all found to be associated with different types of hereditary thrombophilias. Although all 12 patients had some degree of dyslipidemia, their shared feature was the presence of at least one of the genetic mutations associated with thrombophilia.

However, the detection of hereditary thrombophilia has not yet provided valuable insights into the likelihood of stroke recurrence, nor does it imply an adjustment in the secondary prevention of stroke.

We postulate that the risk associated with the relationship between arterial ischemic stroke and hereditary thrombophilia is underestimated. This hypothesis requires further validation through the implementation of analytical case-control studies.

Keywords: arterial ischemic stroke, young adult, recurrence

P121. Recognizing patterns in autoimmune encephalitis: insights from a tertiary care center in Eastern Europe

S. L. FIANI[1], P. M. CIUCUR[2], C. TIU[1,2]

[1]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[2]Department of Neurology, Emergency University Hospital Bucharest, Bucharest, Romania

Study objective. To retrospectively analyze clinical characteristics, diagnostic findings, treatment modalities, and outcomes of patients with suspected autoimmune encephalitis, aiming to identify presentation patterns, diagnostic challenges, and treatment responses.

Methods. This retrospective study examined medical records of 38 patients suspected of autoimmune encephalitis at a tertiary care clinic (2021-2023), focusing on clinical presentation, immunological markers, neuroimaging findings, and treatment outcomes. Data on demographics, symptoms, laboratory and imaging results, and patient outcomes were analyzed.

Results. The cohort predominantly comprised females (63%), with an average age of 46.5 years. The diagnosis was confirmed in 45% of cases, highlighting GAD (16%) and anti-NMDAr (13%) as prevalent subtypes. Notably, 47% of patients presented with seizures. Imaging revealed FLAIR and T2-weighted hyperintensities in 68% of patients. Preliminary data did not identify imaging features suggestive of limbic encephalitis. Despite various clinical presentations, all patients received standardized treatment with immunotherapy.

Conclusion. Preliminary analysis indicates a significant female prevalence and diverse age distribution in autoimmune encephalitis cases. Key diagnostic markers include seizures and specific MRI findings, with the majority of treatment approaches showing promising standardization. Ongoing research is crucial for deeper insights into disease management and outcome optimization. The complete analysis, aimed at refining diagnostic strategies and treatment protocols, will be published subsequently.

Keywords: autoimmune encephalitis, diagnostic challenges, immunotherapy, outcomes, neuroimaging findings

P122. Clinical evolution of patients with familial multiple sclerosis: an overview of important clinical aspects

A. JUGURT[1,2], A. COVALIU[1,2], I. IONITA[1,2], M. NEATU[1,2], A. HERA[1,2], E. I. DAVIDESCU[1,2], B. O. POPESCU[1,2,3]

[1]Neurology Department, Colentina Clinical Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Neurosciences and Experimental Myology, Cell Biology Laboratory, "Victor Babes" National Institute of Pathology, Bucharest, Romania

Aspects related to the genetic susceptibility of multiple sclerosis (MS) represent an area of interest in medical research, with familial MS currently accounting for approximately 12% of all described cases. The objective of this presentation is to report similar patterns of manifestation and progression within the same family, with a focus on identifying possible correlations between family members, including symptoms, disease severity, and treatment.

Materials and methods. We analyzed a series of 7 pairs of familial MS cases included in the National Health Program. We considered age at onset, disease phenotype, initial symptoms, clinical course assessed by the Expanded Disability Status Scale (EDSS), as well as the number of relapses and type of treatment.

Results. Analyzing the data, we observed that the disease phenotype at onset was similar within 3 families, with 2 pairs presenting with optic onset and one presenting with pyramidal onset. Furthermore, within 2 families, similar onset ages were observed (21 and 23 years, respectively; 36 and 38 years). We note that within the same family, the presence of the same autoimmune comorbidities, such as thyroid or gastrointestinal involvement, may exist. Variability in the EDSS score at diagnosis, predominantly a difference of 0.5 points, was observed within the same family.

Conclusions. The presence of autoimmune disorders in MS patients, as well as having a relative with MS, increases the risk and may contribute to earlier worsening and more severe long-term disability. Prognostic factors may help in developing treatment strategies for these types of MS patients.

Keywords: multiple sclerosis, genetic susceptibility, clinical evolution, prognosis

P123. Stroke in Down syndrome adult patients – A case series

Ruxandra GEORGESCU[1], Alina-Dumitrita JIJIE[1], Andreea PLESA[1,2], Florentina-Cristina PLESA[1,2], Carmen-Adella SIRBU[1,2]

[1]*Department of Neurology, "Dr. Carol Davila" EmergencyUniversity Central Military Hospital, Bucharest, Romania*

[2]*Departmentof Neurology, Faculty of Medicine, "Carol Davila" University, Bucharest, Romania*

Introduction. Trisomy 21 is associated with an increased risk of cerebrovascular disease. The pathophysiologic mechanisms of stroke in this population are extremely complex and intricate. A variety of etiologies have been linked to stroke in Down syndrome patients, including moya-moya syndrome, cervical artery dissection, cardiac malformations, arrhythmias, hematologic disease, and complications related to sleep apnea syndrome.

Methods. We report the cases of two Down syndrome patients who were admitted to the Neurology service for acute ischemic stroke. The first patient is a 26-year-old female with a history of renal transplant and chronic immunosuppressive medication. She presented to the ER with an abrupt onset of left hemiparesis, left homonymous hemianopia, and left inattention. Neuroimaging revealed an ischemic lesion in the middle and posterior right cerebral arteries territories. Cerebral angiography showed right internal carotid occlusion in the C7 segment. The second patient is a 33-year-old female with a history of pentalogy of Fallot and corrective cardiac surgery in childhood. She was admitted for sudden onset of right hemiparesis, right homonymous hemianopia, and global aphasia. CT scan was suggestive of an acute right MCA ischemic stroke. Subsequent investigations disclosed an occlusion of the left internal carotid, raising the suspicion of internal carotid dissection.

Results and conclusions. Ischemic stroke in Down syndrome adult patients can have multiple causes. Therefore, a comprehensive panel of investigations and a multidisciplinary approach is required to establish the correct etiologic diagnosis. The two cases demonstrate the range of potential mechanisms that could lead to cerebral ischemia in this population.

Keywords: Down syndrome, trisomy 21, ischemic stroke

P124. Richardson syndrome variant of progressive supranuclear palsy – Case series

Ruxandra GEORGESCU[1], Nicoleta LUPASCU[1], Ionut CALOIANU[1,2], Andreea PLESA[1,2], Florentina-Cristina PLESA[1,2], Carmen-Adella SIRBU[1,2]

[1]*Department of Neurology, "Dr. Carol Davila" Emergency University Central Military Hospital, Bucharest, Romania*

[2]*Department of Neurology, Faculty of Medicine, "Carol Davila" University, Bucharest, Romania*

Introduction. Progressive supranuclear palsy (PSP) is a rare, chronic neurodegenerative disease with a heterogeneous clinical presentation. It is defined by an abnormal accumulation of 4-repeat tau isoform in the cerebral peduncles, basal ganglia, and other structures. Richardson syndrome (RS) is the first reported variant of PSP. The most consistent clinical manifestations of RS include postural instability, oculomotor disturbances, pseudobulbar dysfunction, and parkinsonism.

Materials and methods. We report two cases of patients with parkinsonian syndrome who were investigated in our clinic and diagnosed with progressive supranuclear palsy.

In the first case, a 65-year-old male patient presented with progressive recurrent unprovoked falls without loss of consciousness and emotional lability, including episodes of uncontrollable crying and laughter. The neurological examination revealed supranuclear vertical gaze palsy, a symmetric parkinsonian syndrome, pseudobulbar palsy with mild dysarthria and dysphonia, and emotional instability. The second patient presented with dysarthria, gait imbalance with repetitive falls, and diplopia, with symptoms of insidious onset and progressive evolution. The neurological exam was positive for symmetric parkinsonian syndrome and a limitation of vertical ocular movement with a present vestibulo-ocular reflex. In both cases, cerebral MRI showed evident atrophy of the midbrain, predominantly on the sagittal planes, with a decreased midbrain-to-pons ratio. According to MDS clinical criteria, both patients met the criteria for probable PSP-RS.

Results and conclusions. This case series illustrates the phenotypic heterogeneity of PSP, highlighting the role of cerebral magnetic resonance imaging in diagnosing and assessing the evolution of the disease.

Keywords: progressive supranuclear palsy, Richardson syndrome, parkinsonism

P125. Pain subtypes and sleep disturbances in Parkinson's disease

S. DIACONU[1,2], I. MURASAN[1], L. UNGUREANU[1,2], L. IRINCU[1,2], I. DESPA[1,2], C. FALUP-PECURARIU[1,2]

[1]*Department of Neurology, Brasov County Clinic Hospital, Brasov, Romania*

[2]*Faculty of Medicine, Transilvania University, Brasov, Romania*

Introduction. Pain is one of the most common complaints in Parkinson's disease (PD), with several subtypes of pain identified and a high prevalence in this group of patients. Associations between pain subtypes and sleep disorders are still poorly characterized to this day.

Objectives. To assess the characteristics and correlating factors of pain and sleep disturbances in a cohort of patients with PD.

Material and method. A cross-sectional study in which we included patients with PD in different stages of the disease. Pain was assessed by the King's Parkinson's Disease Pain Questionnaire (KPPQ) and King's Parkinson's Disease Pain Scale (KPPS). Other assessment tools applied were MDS-UPDRS, REM Sleep Behavior Disorder Screening Questionnaire (RBDSQ), Epworth Sleepiness Scale (ESS), MMSE, MoCA, and Pittsburgh Sleep Quality Index (PSQI).

Results. PD patients enrolled in the study most frequently reported musculoskeletal pain (82.44%), followed by nocturnal pain (58.77%) and radicular pain (55.72%). Weak positive correlations were observed between pain (assessed by the KPPS scale) and PD duration, MDS-UPDRS part III, RBDSQ, and ESS scores, while MMSE and MoCA scores presented a weak negative correlation with pain. PSQI scores showed weak positive correlations with the musculoskeletal, chronic, nocturnal, discoloration/edema, and radicular pain domains.

Conclusion. Pain is a symptom that should be focused on in patients with PD, given its high prevalence and consequences on sleep disturbances.

Keywords: Parkinson's disease, pain, sleep disorders

P126. Excessive daytime sleepiness in Parkinson's disease

S. DIACONU[1,2], I. MURASAN[1], G. PASAT[1], T. DRAGAN[1], L. UNGUREANU[1,2], C. FALUP-PECURARIU[1,2]

[1]*Department of Neurology, Brasov County Clinic Hospital, Brasov, Romania*

[2]*Faculty of Medicine, Transilvania University, Brasov, Romania*

Introduction. Excessive daytime sleepiness may have different etiologies in patients with Parkinson's disease (PD), being the result of the neurodegenerative process, the consequence of antiparkinsonian treatment, or other sleep dysfunctions.

Objectives. To evaluate excessive daytime sleepiness in a group of patients with PD.

Material, method. A cross-sectional study in which 131 patients with PD were enrolled. All participants completed a standardized questionnaire and validated scales and questionnaires, such as the Epworth Sleepiness Scale (ESS) for the assessment of diurnal drowsiness, MDS-UPDRS part III, Pittsburgh Sleep Quality Index (PSQI), Parkinson's Disease Sleep Scale (PDSS-2), and Parkinson's Disease Questionnaire-39 (PDQ-39).

Results. ESS $\geq$ 10 score was used to identify patients with PD and excessive daytime sleepiness (PD + EDS). There were 56 (42.74%) patients with PD and EDS. The age of onset of PD in patients with EDS was higher compared with those without EDS (72.16 ± 9.16 vs. 67.16 ± 10.29 years, $p = 0.05$). Patients with PD + EDS showed higher scores for all individual components of the PDQ-39 scale, with statistical significance in the domains of cognition, ADL, and mobility. Patients with PD + EDS encountered more sleep disorders than those without these symptoms, according to the total mean values of the PDSS-2 scale ($p < 0.001$).

Conclusions. More than one-third of patients with PD experience excessive daytime sleepiness. This symptom negatively affects the quality of life and can be associated with other sleep disorders.

Keywords: Parkinson's disease, excessive daytime sleepiness, sleep disorders

P127. REM sleep behavior disorder in Parkinson's disease

S. DIACONU[1,2], I. MURASAN[1], G. PASAT[1], T. DRAGAN[1], L. UNGUREANU[1,2], R. FILIP[1,2], C. FALUP-PECURARIU[1,2]

[1]*Department of Neurology, Brasov County Clinic Hospital, Brasov, Romania*
[2]*Faculty of Medicine, Transilvania University, Brasov, Romania*

REM sleep behavior disorder (RBD) is an important sleep disturbance in patients with Parkinson's disease (PD), that may occur in all stages of the disease, including the premotor stages.

Objectives. To characterize RBD in patients with PD compared to a control group.

Material, method. A case-control study in which 131 patients with PD and 131 controls were enrolled. Probable RBD (RBDp) was identified by the REM Sleep Behavior Disorder Screening Questionnaire (RBDSQ). Other scales were used to evaluate patients, such as the Epworth Sleepiness Scale (ESS), MMSE, MoCA, Hospital Anxiety and Depression Scale (HADS).

Results. Based on a total score of RBDSQ ≥ 5, seventy three patients with PD (43.51%) were identified as having RBDp, compared to 25 participants in the control group (19.08%). There were no statistically significant differences in patients' age, gender, or LEDD. A greater number of patients with PD + RBDp have cognitive impairment, a wider spectrum of non-motor disorders, and depression than patients in the control group with RBDp.

Conclusions. RBDp is more prevalent in the PD group and is associated with cognitive impairment, non-motor symptoms, and depression.

Keywords: REM sleep behavior disorder, Parkinson's disease, non-motor symptoms

P128. Idiopathic intracranial hypertension – a series of clinical cases

M. M. MARTOIU, S. PETRESCU, C PROFIRESCU, C. A. PANEA

Neurology Department, Elias Emergency and University Hospital, Bucharest, Romania
"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Idiopathic intracranial hypertension (IIH) is a rare disorder of elevated intracranial pressure (ICP) of unknown etiology. It affects mostly obese females of childbearing age, with a mean age of 30 years old.

Objective. We present the cases of two patients diagnosed with IIH.

Case reports. A supraponderal 50-year-old female with a two-month history of severe diffuse headache, presented with sudden onset blurred vision in the right eye. Neurological examination was unremarkable, except for both eyes showing low visual acuity. Fundus examination revealed initially right eye papilledema, and two weeks apart, both eyes showed papilledema. CSF assay showed a markedly increased opening pressure (42.5 cmH_2O) with normal range analysis.

A supraponderal 37-year-old male with recurrent minor headache presented with sudden onset blurred vision in the right eye. Neurological examination was unremarkable, with normal visual acuity. Fundus examination revealed both-eye papilledema, and two months apart, both eyes showed optic nerve atrophy. CSF assay showed a markedly increased opening pressure (49 cmH_2O) with normal range analysis.

For both patients, routine serologic tests, complete blood count, markers of inflammation, electrolytes, and blood chemistry were normal. Gadolinium-enhanced brain and spinal MRI was normal.

The patients were started on acetazolamide 250 mg per os twice daily, under which they progressively improved in headache, but not in visual acuity.

Conclusions. Although IIH typically manifests in overweight, childbearing-aged women, it can also be present in males. These cases highlight the importance of considering this diagnosis even in less commonly affected populations: advanced age and male patients.

Keywords: IIH, ICP

P129. Diplopia in Parkinson's disease – prevalence and correlates

L. UNGUREANU[1,2], S. DIACONU[1,2], L. IRINCU[1,2], R. FILIP[1,2], I. MURASAN[1], C. FALUP-PECURARIU[1,2]

[1]*Department of Neurology, Brasov County Clinic Hospital, Brasov, Romania*
[2]*Faculty of Medicine, Transilvania University, Brasov, Romania*

Introduction. Diplopia is integral to nonmotor (NMS) assessment of PD, a part of the NMS questionnaire (NMSQuest) and the overall spectrum of positive visual symptoms in PD.

Objective. To assess the prevalence of diplopia in Parkinson's disease (PD) patients and the correlation of diplopia with visual hallucinations, cognitive dysfunction, and motor performance.

Methods. 131 consecutive PD patients were included in the study. Diplopia was screened by using the NMSQuest – item 29 and Non-Motor Symptoms Scale for Parkinson's Disease (NMSS) – item 15. Cognitive function was assessed with Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA). Visual hallucinations were measured by Scales for Outcomes in Parkinson's Disease – Psychiatric Complications (SCOPA-PC).

Results. 12.2% had self-declared diplopia (n=16), with 31.2% male. The mean age of patients with diplopia was 76.0 ± 9.0 years. PD patients with diplopia had longer disease duration than those without diplopia and worse cognitive dysfunction, MMSE scores, and MoCA scores respectively. Visual hallucinations were present in 31.2% of PD patients with diplopia compared to only 8.6% of PD patients without diplopia. Motor performance determined by the MDS-UPDRS part III score and Hoehn and Yahr stage did not differ significantly between the groups. The PDQ-39 Summary Index was significantly higher in patients with diplopia compared to patients without diplopia.

Conclusions. Diplopia can occur in over 10% of an unselected cohort of PD cases with longer disease duration, cognitive dysfunction, worse quality of life (QoL), and may include hallucination-related diplopia. It needs to be considered in clinical practice.

Keywords: diplopia, double vision, Parkinson's disease, visual hallucinations

P130. What can hide mixed aphasia with acute onset?

V. SAPIRA, A. TELEHUZ

[1]*Slobozia County Emergency Hospital, Slobozia, Romania*
[2]*Faculty of Medicine and Pharmacy, "Dunarea de Jos" University, Galati, Romania*

Introduction. Hepatic encephalopathy and liver failure have been associated with significant neurologic disturbances, including asterixis, delirium, and coma. However, focal neurological deficits in a specific vascular distribution are rarely attributed to liver dysfunction.

Study presentation. Between December 2022 and December 2023, four patients (three men and one woman) presented in the emergency department with mixed aphasia of sudden onset and facial asymmetry. All four cases arrived at the emergency room more than six hours after the onset of symptoms. The brain CT performed urgently did not show any tomodensitometric changes suggestive of recent onset. Forty-eight hours after admission, a repeat brain CT did not reveal any acute lesions. From the personal medical history, we note that all patients had pre-existing liver damage, with elevated liver transaminases. The serum ammonia level was measured, showing elevated values, which led to the diagnosis of hepatic encephalopathy. Consequently, treatment with rifaximin 1200 mg/day and lactulose was initiated, resulting in significant improvement in symptoms. The evaluation of the patients six months after the acute event highlighted the remission of symptoms in three cases. One patient died 48 hours after discharge.

Conclusions. These cases demonstrate the importance of considering underlying pathophysiological conditions when evaluating patients presenting with ischemic stroke-like symptoms. Clinicians should be aware that patients with hepatic encephalopathy may present with focal neurologic signs. Therefore, in this patient population, we suggest testing for ammonia, liver enzymes, and coagulopathy as part of the acute stroke evaluation without delay, while also considering therapies and interventions for acute stroke.

Keywords: hepatic encephalopathy, mixed aphasia, stroke, serum ammonia

P131. Emergency hospital admission in patients with Parkinson's disease

L. C. PRODAN, L. CUIBUS, C. ROMAN-FILIP, M. PRODAN

[1]*Department of Neurology, Emergency Clinical County Hospital, Sibiu, Romania*
[2]*"Lucian Blaga" University, Sibiu, Romania Bucharest, Romania*

Introduction. Parkinson's disease is a neurodegenerative disorder with a progressive course. In the early stages, patients present fewer complications, while the advanced stages of the disease are associated with many alterations. In the emergency department, patients with Parkinson's disease can present with worsening of movement disorders, dyskinesia, psychosis, confusion, pain, and orthostatic hypotension. Additionally, pulmonary infections, constipation, and occlusive syndrome are other causes for emergency department presentation.

Methods. We included in the analysis all patients with Parkinson's disease evaluated in the emergency department of the Clinical Hospital County Sibiu, between January 2020 and December 2023. The stages of the disease and the causes of admission were observed. We also included the pandemic COVID-19 period in the analysis.

Results. Our cohort comprised 1,129 patients with Parkinson's disease, of which 846 were hospitalized. The causes for admission were: 9.3% direct complications; 27.5% infections; 21.4% trauma; 11.6% cardiovascular emergencies; 11.3% cerebrovascular emergencies; 5.9% gastrointestinal emergencies; 3.6% electrolyte imbalance; 8.9% neuropsychiatric manifestations; and 0.5% other causes.

Conclusions. Patients with Parkinson's disease can present to the emergency department for direct complications, adverse drug effects, neuropsychiatric manifestations, and indirect complications such as infections, trauma, and cardiovascular and cerebrovascular emergencies. There is a high susceptibility to poor quality of life, so medical management is essential for maintaining and improving daily life.

Key words: Parkinson's disease, emergency, medical complications, hospital admission

P132. Charcot-Marie-Tooth disease in Romania: Insights from our centre's experience

G. M. GRECU[1], N. GRECU[1,2], M. T. G. PALADE[1], I. M. VODA[1], A. LEONTE[1], C. TIU[1,2]

[1]Department of Neurology, Emergency University Hospital, Bucharest, Romania
[2]Department of Neuroscience , "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Charcot-Marie-Tooth (CMT) disease is a progressive, hereditary motor and sensory neuropathy, with mutations in over 100 genes related to myelin or axonal proteins having been described. This paper details our centre's experience with CMT patients followed at our institution.

Methods. We conducted a review of patient records from the Neurology Department of the University Emergency Hospital of Bucharest, starting from July 2023. Referrals were made through patient advocacy groups and other neurologists. A comprehensive clinical examination was performed for all patients, supplemented by electrodiagnostic studies (EDX) and genetic tests in some cases.

Results. Fourteen patients from eight families, primarily from Southern Romania, with an average age of 42 years, were included. Of these, 78.57% were female, and 21.43% were male. The time from symptom onset to diagnosis averaged 16.64 years. Gait disturbance was universally reported as the initial symptom. 85.71% of patients exhibited osteoarticular deformities, and 46.15% underwent surgical correction. Hearing impairments and wheelchair dependency were noted in 46.15% and 35.71% of patients, respectively. The median CMTNS2 score was 18, reflecting partial data due to missing EDX examinations (available for 10 patients, all demyelinating). Genetic testing was available for 35.71% of cases; 3 patients had PMP22 duplication, and 2 were homozygous for the c.442C>T mutation in NDRG1. Genealogy analysis showed autosomal dominant and autosomal recessive inheritance in 50% and 42.86% of cases, respectively.

Conclusions. Our findings highlight CMT1A and CMT4D as prevalent types of CMT in our patient population and underscore the significant diagnostic delays caused by limited awareness and resource constraints.

P133. Endovascular treatment – first therapeutic option in carotid dissecting aneurysms: our experience

Andrei-Cristian CHESNOIU-DOBROTA[1], Sebastian STEFAN[1], Anda-Patricia IOAN[1], Alida CIOCAN[1], Cristina DAVIDOIU[1], Bogdan DOROBAT[1], Athena-Cristina RIBIGAN[1,2], Florina-Anca ANTOCHI[1]

[1]Emergency University Hospital, Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Arterial dissection is the result of the separation of the arterial wall layers and the formation of a local hematoma, which may lead to local compression or thrombosis with distal embolization. Dissecting aneurysms (DA), also known as pseudoaneurysms, are a possible complication in such cases. The aim of this study is to present our experience with DAs and their management in a series of five cases.

Methods. Five patients were admitted over the past five years for endovascular treatment of chronic DA, all of them previously diagnosed with internal carotid artery dissection with subsequent pseudoaneurysm. Clinical presentations varied, including Horner's syndrome, lower cranial nerve dysfunction, transient ischemic attack, ischemic stroke, pulsatile tinnitus, and headache, all on the same side as the dissecting lesion. Stent angioplasty was performed in all cases.

Results. The procedure resulted in reduced blood flow inside the pseudoaneurysm and restoration of normal vessel lumen diameter. One patient experienced vertigo, which resolved quickly with treatment, while another presented with headache, confusion, and anomia, which remitted over 24 hours with sustained intravenous hydration and was interpreted as contrast encephalopathy. Follow-up Doppler ultrasonography of cervical arteries showed normal in-stent velocities in all cases.

Conclusion. The management of chronic DAs is a matter of debate, as they may cause vascular compression and serve as a source of distal emboli. Our experience supports endovascular treatment as the first therapeutic option, in combination with antiplatelet therapy, offering minimal perioperative risk, good long-term hemodynamic outcomes, and no subsequent cerebrovascular events.

P134. Plasma exchange in acute attacks of neuromyelitis optica spectrum disorder: a case series

M. ROMAN[1], R. BALASA[1,2]

[1]*Neurology 1 Clinic, Emergency Clinical County Hospital, Targu Mures, Romania*
[2]*Department of Neurology, "George Emil Palade" University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Targu Mures, Romania*

Introduction. Neuromyelitis optica spectrum disorder (NMOSD) is an autoimmune disease characterized by relapsing demyelination primarily affecting the optic nerve and spinal cord, with a mean onset between the ages of 30 and 40. While the main goal in managing NMOSD is to prevent relapses through maintenance therapy (such as mycophenolate mofetil and azathioprine), acute treatment options including high-dose corticosteroids and plasma exchange are essential.

Material and methods. We present a case series of seven female patients diagnosed with NMOSD, all positive for aquaporin-4 antibodies, and treated at the Neurology I Clinic of the Emergency Clinical County Hospital of Targu Mures between 2019 and 2024. A total of eighteen relapses were included, during which patients received plasmapheresis after high-dose intravenous methylprednisolone therapy failed to yield significant clinical improvement. On average, four sessions were performed per treatment course, ranging from two to six.

Results. Plasmapheresis resulted in notable clinical improvement across all cases. Neurological deficits were reduced, and improvements in Expanded Disability Status Scale (EDSS) scores were recorded. The treatment was well tolerated, with minimal adverse reactions reported during sessions, and reflected an overall enhancement in patient outcomes with a high safety profile.

Conclusion. This case series highlights the role of plasma exchange as an effective adjuvant treatment for NMOSD relapses refractory to corticosteroids, supporting its use in improving the clinical outcomes of affected patients.

Keywords: neuromyelitis optica spectrum disorder, plasmapheresis, corticosteroids, aquaporin-4 antibodies, EDSS

P135. Beyond movement: investigating the impact of device-assisted therapies on cardiovascular dysautonomia in Parkinson's disease

A. C. TANASOIU[1], D. TULBA[1,2], B. O. POPESCU[1,2,3]

[1]*Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*
[3]*Department of Cell Biology, Neurosciences and Experimental Myology, "Victor Babes" National Institute of Pathology, Bucharest, Romania*

Autonomic dysfunction (AD) is a frequent non-motor manifestation in Parkinson's disease and may even precede motor symptoms. Cardiovascular dysautonomia contributes to impaired quality of life, increased disability, and reduced life expectancy, and it poses significant therapeutic challenges with current treatments. Cardiovascular AD is subjectively described by patients as light-headedness, dizziness, fatigue, or blurred vision. With the broader use of device-assisted therapies (DATs), there is growing interest in evaluating their impact on cardiovascular profiles.

We present two patients with advanced Parkinson's disease (PD) treated with DATs in our department.

A 64-year-old man, diagnosed with PD in 2016, was started on continuous subcutaneous apomorphine infusion.

A 61-year-old woman, with over 20 years of PD history, was initiated on levodopa/carbidopa/entacapone intestinal infusion.

Initial levodopa equivalent daily dose (LEDD) was 1540 mg for the first patient and 1072 mg for the second. After titration, LEDD increased to 1915 mg and 1141 mg, respectively.

We performed 24-hour blood pressure monitoring before and after DAT initiation, considering the following parameters: mean BP (24 h/day/night), dipper profile, and heart rate (HR) variability using SDNN, SDANN, and RMSSD.

Dysautonomia markers—namely HR variability and dipper profile (non-dipper and reverse-dipper)—were present both before and after DATs. However, an improvement in SDNN was observed in the second patient. Orthostatic hypotension was not recorded. Mean BP values showed no significant changes after DATs.

Device-assisted therapies did not adversely affect the cardiovascular profiles of the two patients, suggesting these interventions may be safe in this context. However, further studies are needed to confirm these findings.

P136. The association between the anti-JCV antibody index in multiple sclerosis patients transitioning from natalizumab treatment to other high-efficacy therapies (HET) and demographic, clinical, and MRI assessments

Irina LATA[1,2], Amanda SCHULDESZ[1,2], Amalia CORNEA[1,2], Geni NICOLA[1,2], Oana CIOBESCU[2], Raul NOSAL[2], Mihaela SIMU[1,2]

[1]*"Victor Babes" University of Medicine and Pharmacy, Timisoara, Romania*

[2]*"Pius Brinzeu" Clinical Emergency County Hospital, Timisoara, Romania*

Background. The risk of developing progressive multifocal leukoencephalopathy (PML) associated with natalizumab treatment is linked to the presence of antibodies against the John Cunningham virus (JCV). This study aims to explore the long-term relationship between anti-JCV antibody serum levels and their association with demographic, clinical, and radiological characteristics in multiple sclerosis (MS) patients who transitioned from natalizumab to other high-efficacy therapies (HET).

Material and methods. We recruited 92 MS patients from the Neurology Clinic "Pius Brinzeu" at the Clinical County Hospital in Timisoara. Inclusion criteria were: diagnosis of MS according to the 2017 McDonald criteria, age between 18 and 55 years, and ongoing treatment with HET.

Results and conclusions. The patient cohort consisted of 91 females (52.7%) with a mean age of 48.43 years (SD 13.9) and 43 males (47.3%) with a mean age of 45.8 years (SD 8.52). Subsequently, we divided the patients into two groups based on their JCV antibody index, with 44 (48.35%) having an index lower than 0.403 (SD 0.465) and 47 (51.66%) showing an index higher than 1.5–1.93 (SD 1.61). There was no significant difference observed between the groups of patients with a higher JCV virus index in terms of the age at onset of the disease (43.55 years with a standard deviation of 1.5 vs. 47.67 years with a standard deviation of 1.8) and treatment duration (7.2 years with a standard deviation of 1.38 vs. 7.5 years with a standard deviation of 1.18), with a p-value of 0.46. The initial evaluation of EDSS showed slightly higher values: 4.68 (SD 0.73) compared to 4.89 (SD 0.47), with a p-value of 0.05, approaching the significance threshold. In our cohort, it appears that patients with elevated JCV indices tend to demonstrate higher Expanded Disability Status Scale (EDSS) scores upon switching to high efficacy therapy (HET), accompanied by an increased number of MRI lesions.

Patients with an anti-JCV index exceeding 1.5 tend to show a higher MRI T2 lesion burden compared to those with lower anti-JCV index levels.

P137. Relationships among lifestyle factors, non-motor symptoms of Parkinson's disease, and cognitive function levels in individuals with advanced PD

Octavia NECOARA[1,2], Romina BALA[2], Radu SOARE[2], Geni NICOLA[1,2], Amalia CORNEA[1,2], Mihaela SIMU[1,2]

[1]*"Victor Babes" University of Medicine and Pharmacy, Timisoara, Romania*

[2]*"Pius Brinzeu" Clinical Emergency County Hospital, Timisoara, Romania*

Background. Parkinson's disease manifests through both motor and non-motor symptoms, the latter being the earliest to appear (premotor) and having a greater impact on patients' quality of life. Among the non-motor symptoms, cognitive decline is directly proportional to a more advanced stage of the disease. Currently, there is discussion about early advanced Parkinson's disease (early APD) and more advanced PD (more advanced APD).

Material and method. We included 20 patients undergoing device-assisted therapy (Duodopa) for Parkinson's disease, with 25% being female and the rest male. The participants had an average age of 64, ranging from 50 to 73 years, and the average duration of the disease was 9 years, with a range of 3 to 26 years. Each patient underwent evaluations including UPDRS, MMSE, EEG, and a Go/No-Go task, all conducted within one clinic visit.

Results and conclusions. Our group consisted of 12 individuals diagnosed with advanced Parkinson's disease and 8 individuals diagnosed with early advanced PD. Regarding the onset pattern, they were divided equally: half had a predominantly tremor-dominant form, and the other half had a hypotonic-hypokinetic form, with an average UPDRS score in the OFF state of 93 points. The majority of patients exhibited cognitive impairment within age-adjusted parameters (MMSE > 24), one patient showed mild cognitive decline (MMSE = 21), and another patient showed moderate cognitive deterioration (MMSE = 13); the mean MMSE score was 26.

Screening of patients with PD is recommended for cognitive decline and non-motor symptoms, as cognitive deterioration worsens with disease progression. Non-motor symptoms and moderate cognitive decline correlate with a subset of more advanced patients who will experience a rapidly progressive course. Cognitive dysfunction associated with PD primarily manifests through visuospatial disturbances (clock test), attentional deficits, executive dysfunction, and memory impairments (MMSE). Although there are few studies that summarize these findings, it is necessary to understand the progression of cognitive decline associated with PD and to identify early the non-motor symptoms associated with it.

P138. The utility of serum neurofilament in the clinical assessment of multiple sclerosis

Geni NICOLA[1,2], Amanda SCHULDESZ[1,2], Romina BALA[2], Gabriel GAMAN[2], Amalia CORNEA[1,2], Mihaela SIMU[1,2]

[1] *"Victor Babes" University of Medicine and Pharmacy, Timisoara, Romania*

[2] *"Pius Brinzeu" Clinical Emergency County Hospital, Timisoara, Romania*

Objectives. We evaluated the correlation between serum neurofilament light chains (NfL) and clinically defined multiple sclerosis (CDMS) as well as McDonald 2017 multiple sclerosis (MS) in individuals who transitioned to or initiated high efficacy therapy (HET).

Material and methods. We performed a retrospective examination of patient data, encompassing those who either transitioned to high efficacy therapy (HET) or initiated HET directly. Our analysis centered on instances of highly active disease, considering factors such as disease and therapy duration, levels of neurofilaments, EDSS at onset, both prior to transitioning and during the current evaluation.

Results and conclusions. Our cohort comprised 14 men (22%) with a disease duration of 32.45 years (SD 7.85) and a treatment duration of 5.98 years (SD 3.36), and 49 women (77.78%) with a mean disease duration of 58.57 years (SD 41.88) and a treatment duration of 7.16 years (SD 4.98).

Within the normal neurofilament group, we assessed 25 patients with a mean age of 49.07 years (SD 1.19), and 38 participants with a mean age of 65.19 years (SD 41.9) in the group with elevated NfL levels.

In both groups, patients with normal levels of NfL and an EDSS <3.0, and those with an EDSS ≥3.0 at the time of therapy transition, showed lower EDSS scores at the second evaluation compared to the group with higher NfL values.

Additionally, the higher NfL group was associated with MRI progression. The utilization of NfL may serve as a dependable marker in the clinical assessment of MS patients, including those in early and advanced disease stages who exhibit clinical and/or MRI activity.

The use of NfL might represent a reliable marker in the clinical evaluation of MS patients with both early and advanced disease who present signs of clinical and/or MRI activity.