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The 22nd National Congress of the Romanian Society of Neurology

ORAL PRESENTATIONS – ABSTRACTS

(in alphabetical order of the first author)

CO01. Identification of early transition biomarkers from RR to progressive multiple sclerosis

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Multiple sclerosis (MS) is a chronic, immune-mediated, inflammatory, demyelinating, and neurodegenerative disease of the central nervous system (CNS). Recently, it has been shown that the pathological processes are a continuum of injuries involving different players, in varying proportions depending on each patient, but with the final aim of producing irreversible degenerative damage to CNS structures. In MS, an ideal biomarker should have prognostic value, correlate with specific MS activity such as relapse or progression, and respond to or indicate the most effective treatment.

Biomarkers with greater prognostic value in detecting early transition to progression in MS are now considered to be serum neurofilaments (NfL). However, NfL levels are not 100% specific to processes within MS pathology and are influenced by many confounding variables such as age, blood volume, and body mass index, which can make them difficult to use when clinically evaluating individual MS patients.

This presentation aims to provide an overview of diverse biomarkers—such as NfL, proteins, immune cells, transcriptomics, extracellular vesicles, and metabolites—that can predict MS disease progression. In addition, the experience of our center on this subject will be presented.

CO02. Multiple sclerosis and microbiota

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Background. Dysbiosis has been proposed as a potential risk factor for developing multiple sclerosis (MS). The composition of the gut microbiota presents significant interindividual variability, and the quantification of microbiome metabolites poses significant challenges. Short-chain fatty acids (SCFAs) and medium-chain fatty acids (MCFAs) are produced by commensal bacteria and have the capability of crossing the blood-brain barrier, exerting immunomodulatory effects within the central nervous system. SCFAs, such as butyric acid (BA), have anti-inflammatory roles, while MCFAs, like caproic acid, promote inflammatory processes.

Objective. To assess neuroinflammation across four dimensions: microbial fermentation by-products (SCFAs, MCFAs), the gut-brain connection, blood-brain barrier permeability, and inflammatory biomarkers in MS patients treated with interferon beta.

Material and methods. We implemented and validated a bioanalytical method using high-performance liquid chromatography coupled with mass spectrometry (LC-MS/MS), which allowed the analysis of SCFAs and MCFAs from human serum.

Conclusions. SCFAs and MCFAs play significant regulatory roles in immunomodulation. Quantification of these analytes and multifactorial analysis from the perspective of neuroinflammation in MS patients offers new insights into MS pathophysiology, possibly triggered by microbiome dysfunction.

CO03. Progressive spastic paraparesis – a systematic approach

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Introduction. Progressive spastic paraparesis is caused by lesions affecting both corticospinal tracts anywhere along their course, from the frontoparietal cortices to the anterior horns of the spinal cord. Whereas spastic paraparesis is usually a straightforward clinical diagnosis, determining its etiology might require a systematic approach.

Methods. We herein present three patients with progressive spastic paraparesis and our approach to the etiological diagnoses. A 56-year-old male presented to our clinic with progressive gait disturbances that had started several years earlier. Brain MRI revealed lesions compatible with multiple sclerosis, and an extensive work-up consisting of blood and cerebrospinal fluid tests was otherwise unremarkable. Considering the clinical course, primary progressive multiple sclerosis was diagnosed. A 66-year-old female was admitted for a 2-year history of progressive gait disturbances and urinary incontinence. Subsequently, she developed subtle upper motor neuron signs in the upper limbs. After excluding other etiologies, we established the diagnosis of primary lateral sclerosis. Another female, aged 23, with an unremarkable family history, was evaluated for progressive paraparesis, gait disturbances, and muscle spasms, first noticed 2 years prior. Brain and spinal MRI images were suggestive of hereditary spastic paraparesis, which was later confirmed by genetic testing.

Conclusions. Progressive spastic paraparesis encompasses a myriad of etiological diagnoses; therefore, we emphasize the need for a systematic approach, thorough examination, and extensive work-up. Furthermore, patients should be thoroughly screened for reversible causes and benefit from appropriate etiological and symptomatic treatment.

Keywords: progressive spastic paraparesis, differential diagnosis, case series

CO04. Stroke and epilepsy

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Stroke is the leading cause of seizures and epilepsy among older adults, with approximately 6% of the 3 to 6 million individuals who experience a stroke each year developing post-stroke epilepsy. Seizures after stroke are defined as early, occurring fewer than 7 days after the insult, or late, occurring more than 7 days after.

Patients who have larger and more severe strokes involving the cortex, are younger, and have early seizures and intracerebral hemorrhage are at the highest risk of developing post-stroke epilepsy. Prognostic models, including the SeLECT and CAVE scores, help assess the risk of epileptogenesis both in the short and long term.

Seizures can have a detrimental impact on recovery and quality of life after a stroke, as well as increase the cost of in-hospital care. Seizures occurring shortly after a stroke can exacerbate metabolic stress and cellular death, leading to larger infarct sizes, higher mortality rates, and poorer functional outcomes. Meanwhile, recurrent seizures can result in injuries, cognitive impairment, and reduced ability to work or drive, ultimately lowering quality of life. Thus, selecting the right treatment promptly is essential.

The approach to managing acute and remote symptomatic seizures post-stroke varies significantly. In choosing the most suitable antiseizure medication, it is important to consider not just its effectiveness but also its side effects and how its pharmacodynamics may be altered in older adults. The potential for drug-drug interactions, especially between antiseizure drugs and anticoagulants or antiplatelet agents, is also a critical factor in making treatment decisions.

CO05. Migraine comorbidities

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Among young adults, especially women, migraine is a primary headache disorder associated with high prevalence and morbidity. Over the past few decades, novel knowledge has emerged regarding the relationships between migraine and epilepsy, migraine and mood disorders, and migraine and irritable bowel syndrome. Several factors are involved in migraine pathophysiology, including the trigeminovascular system. One hypothesis suggests that migraines may be caused by altered sensory input processing in the brainstem. CGRP release is increased during migraine attacks, and the CGRP pathway has become better understood through current migraine treatments.

The association between epilepsy and migraine has been documented over time. In other words, seizures can trigger migraines, and migraine attacks can be epileptic. Migraine and epilepsy are also connected through antiseizure drugs (ASDs), which can prevent migraine attacks. Additionally, migraines and anxiety disorders are bidirectionally related, each increasing the likelihood of the other.

Apart from its function as a vasodilator, CGRP also plays a critical role in transmitting nociception. This is associated with frequent migraine attacks, stress, and anxiety. In addition to limbic system dysfunction, serotonergic dysfunction is closely linked to migraines and anxiety. Dysbiosis can affect the gut-brain axis, contributing to chronic migraines. Dysbiosis also increases calcitonin gene-related peptide (CGRP) production, which may be linked to migraines.

Our purpose is to review migraine pathophysiology and the latest theories regarding migraine comorbidities. These comorbid migraine conditions, with similar pathophysiological mechanisms, are likely to influence treatment choices significantly, providing clues for future therapies.

Keywords: migraine, epilepsy, anxiety and mood disorders, CGRP, gut-brain axis, microbiome

CO06. Sphingosine 1-phosphate receptor modulator molecule as new therapeutic option in the treatment of multiple sclerosis

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The emergence of new molecules from the sphingosine 1-phosphate receptor modulator series in the already existing arsenal for multiple sclerosis (MS) therapy expands the range of available molecules, offering the possibility of increasingly personalized therapies.

The new concepts of Relapse-Associated Worsening of disease (RAW) and Progression Independent of Relapse Activity (PIRA) are currently important contributors to the accumulation of disability and are to be taken into account even from the initial stages of the disease, as they are important factors in long-term cumulative impairment.

The pivotal SUNBEAM and RADIANCE trials that led to the registration of ozanimod for the treatment of relapsing-remitting MS are reviewed, with inclusion criteria, design, and outcomes of primary and secondary endpoints established. Cumulative safety data and the most common possible adverse events are presented.

Also, information is provided from the OLEDAYBREAK multicenter, open-label extension study, which evaluated the long-term safety, tolerability, and efficacy of ozanimod treatment through data obtained after 3.5 years of exposure to this therapy. It was well tolerated, with no new safety risks identified.

The administration with titration of the initial doses, as well as information for the preparation and monitoring of patients, are presented.

CO07. Updating vaccination strategies in multiple sclerosis, considering highly effective therapies

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Multiple sclerosis (MS) is the most common neurological disease with onset in young adults, causing di-

sability and representing a lifelong diagnosis. The emergence of newly developed, highly effective molecules has led to a paradigm shift in global MS therapy.

Vaccination is an essential element in the risk management strategy for MS patients. However, there are challenges in clinical practice related to vaccination in patients treated with different disease-modifying therapies—such as the timing of certain vaccines, the immune response elicited, and the individualization of therapies through the lens of the benefit/risk balance—in order to mitigate risks.

The main directions established by the ECTRIMS/EAN consensus developed by a group of experts in 2023 are presented. Thus, data on the safety and efficacy of vaccines, strategies for sequenced immunization according to the therapy used, and general recommendations are provided. At the same time, issues related to special population groups are addressed, such as the elderly—where the phenomena of immunosenescence and inflammaging interfere with the vaccine immune response—or pregnant women with MS, for whom specific recommendations exist. Additionally, for patients who wish to travel to endemic areas for certain infectious diseases, specific vaccination indications have been established.

It is preferable that vaccination is carried out at the time of MS diagnosis or in the early stages of the disease, so that when the use of highly effective therapies is required, the initiation is no longer postponed due to pending vaccination.

Keywords: multiple sclerosis, vaccination, immune response, highly effective therapies

CO08. Nervous system involvement in Fabry disease: analysis of a Romanian cohort

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Background. Fabry disease (FD) is an inherited lysosomal storage disorder caused by mutations in the GLA gene (Xq21.3–q22), leading to the absence or deficiency of alpha-galactosidase A (α-Gal A), resulting in impaired metabolism and the subsequent accumulation of neutral glycosphingolipids and globotriaosylceramide (Gb3) in the plasma and cellular lysosomes of various tissues and organs such as the heart, kidneys, central and peripheral nervous system, skin, and eyes.

Material and methods. This was a retrospective study of a cohort of patients diagnosed and treated at Fundeni Clinical Institute. The patients were diagnosed using enzymatic and genetic testing, and some patients underwent renal biopsy.

Results. The cohort included 68 patients, with a median age of 44 years, 56% men and 44% women, of whom 22 patients were treated with enzyme replacement therapy: 13 with agalsidase beta and 9 with agalsidase alfa. Seventy-seven percent of patients exhibited acroparesthesia, 59% had polyneuropathy, 74% had cerebrovascular involvement, 19% had hypoacusia, and 88% presented with tinnitus.

Conclusion. Central and peripheral nervous system involvement is frequent in Fabry disease, and both men and women are affected.

CO09. Neurological features of tuberous sclerosis complex: findings from a Romanian tertiary hospital

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Background. Tuberous sclerosis complex (TSC) is a rare multisystem genetic disorder characterized by the involvement of a wide range of organs, including the brain, skin, kidneys, lungs, and eyes. Central nervous system–related findings in TSC include structural brain lesions such as cortical dysplasias (cortical tubers and cerebral white matter radial migration lines), subependymal nodules (SEN), subependymal giant cell astrocytomas (SEGA), epilepsy, and TSC-associated neuropsychiatric disorders.

Material and methods. This is a retrospective, single-center study of adult patients with TSC who were treated and followed from 2015 to 2020 in the Neurology and Nephrology Clinics of Fundeni Clinical Institute, Bucharest, Romania.

Results. The study included 32 patients, 19 female (59.4%) and 13 male (40.6%), with a mean age at diagnosis of 11 years. Cortical tubers on neuroimaging appeared to be associated with a diagnosis of epilepsy, while subependymal nodules represented a risk factor for intellectual impairment. Males exhibited a greater variety of dermatological lesions, especially café-au-lait patches. A statistically significant positive association was found between epilepsy and the presence of cutaneous angiofibromas, as well as overall dermatological involvement. Females had significantly higher Charlson Comorbidity Index scores, indicating a greater disease burden. Everolimus appeared to be a well-tolerated treatment and showed promising results in controlling epileptic seizures in two patients.

Conclusions. TSC patients should be managed by a multidisciplinary team, and complex antiepileptic therapy combined with mTOR inhibitor treatment should be considered for seizure control.

CO10. Oculomotricity tests – video head impulse test (vHIT)

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The vestibulo-ocular reflex can be examined using tests that assess the response to stimulation of the semicircular canals, namely the video head impulse test (vHIT), caloric tests, and the rotational chair. vHIT stands for video monitoring of the head impulse test and evaluates all six semicircular canals by applying high-frequency stimulation. The advantage of vHIT is its ability to identify covert saccades, which occur during head movement and are not visible to the naked eye, unlike the traditional head impulse test (HIT), where only saccades occurring after the movement stops, called "overt saccades", can be identified.

The aim of this paper is to study the physical principles of vHIT, its clinical use in the positive and differential diagnosis of neurotological pathologies, its application in examining vestibular pathology in pediatric patients, and to present relevant case studies.

Keywords: vertigo, neurotology, video head impulse test

CO11. Conjoint rarities: a case of fibromuscular dysplasia with unexpected vascular hyperreactivity

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Introduction. Fibromuscular dysplasia (FMD) causes non-atherosclerotic, non-inflammatory architectural changes in the musculature of medium or small arteries, resulting primarily in stenosis. Of particular note is its potential to unexpectedly affect vessel reactivity, leading to cerebrovascular damage.

Materials and methods. A 55-year-old female with recently discovered and treated hypertension suddenly developed recurrent thunderclap headaches over the course of several days and speech problems on the day of admission. Initially, the patient had mild focal signs upon examination, but her right hemiparesis and aphasia gradually worsened. Cerebral CT and MRI showed an acute frontoparietal lobar hematoma with associated subarachnoid hemorrhage (SAH). 3D TOF MRA revealed an irregular contour of both internal carotid arteries and a mild focal stenosis of the left middle cerebral artery (M2 segment). FMD was initially excluded by cerebral angiography, which showed extensive multivessel vasospasm, confirming reversible cerebral vasoconstriction syndrome (RCVS). Repeated cerebral angiography, after the vasospasm resolved, revealed the characteristic string-of-beads appearance in both internal carotid arteries, an aspect initially masked by the severe vasospasm. The patient was therefore diagnosed with multifocal FMD. The coronary and renal arteries were not affected, as shown by subsequent tests.

Results/Conclusions. SAH, RCVS, and FMD set the stage for a constellation of catecholamine discharge and irregular vascular response. Synergistic mechanisms resulted in this patient's multiple cerebrovascular complications. The relationship between RCVS and FMD is a topic of interest for future research.

Keywords: fibromuscular dysplasia, RCVS, SAH, thunderclap headache

CO12. Cardiovascular dysautonomia in Parkinson's disease

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The dysfunction of the autonomic nervous system affects a large number of patients with Parkinson's disease (PD) and other synucleinopathies, such as multiple system atrophy (MSA), Lewy body dementia (LBD), and pure autonomic failure (PAF), with cardiovascular dysautonomia being a common occurrence.

The most frequent symptom of cardiovascular dysautonomia is neurogenic orthostatic hypotension (nOH), which can vary in intensity from general weakness to syncope and is reported by many patients with PD. Certain factors such as stress, temperature, dehydration, reduced blood volume, food intake, and the resulting postprandial hypotension can exacerbate an asymptomatic nOH. Postprandial hypotension can trigger symptomatic nOH, as can prolonged standing or walking. Increased vasodilation due to heat, alcohol, or other factors may also cause nOH symptoms to emerge.

In PD, cardiovascular dysautonomia results from cardiac sympathetic denervation, inadequate activation of norepinephrine (NE) pathways, and baroreflex failure. Central structures, particularly the dorsal motor nucleus of the vagus, are also affected in PD and are involved in orthostatic hypotension.

Standard tests such as 24-hour ambulatory blood pressure monitoring (ABPM) can identify significant autonomic failure requiring treatment by measuring blood pressure values during normal daily activities and providing information about circadian variations.

In patients with confirmed neurogenic OH, neurogenic supine hypertension (SH) is defined as a systolic BP of ≥140 mmHg and/or a diastolic BP of ≥90 mmHg, measured after at least 5 minutes of rest in the supine position.

This paper discusses the dipper and non-dipper profiles of patients with PD.

Keywords: Parkinson's disease, cardiovascular dysautonomia, assessment

CO13. Hereditary motor and sensory neuropathy – from a historical perspective to current challenges

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Over 130 years have passed since the first description of hereditary motor and sensory neuropathy by Jean-Martin Charcot, Pierre Marie, and Howard Henry Tooth, and while many advances have been made in understanding the underlying genetic and pathophysiological basis, treatment remains, for the moment, elusive. While some of the typical symptoms are well known, a degree of phenotypical variability and symptom overlap with other hereditary and degenerative neurological disorders sometimes makes clinical diagnosis difficult. Genetic testing has the ability to establish a definitive diagnosis and, with the help of national registries, prepare these patients for potential future clinical trials and therapies.

The Romanian Registry for Hereditary Neuropathies, a joint project of adult and pediatric neurology departments as well as patient advocacy groups, is an endeavor to provide a real-life picture of the prevalence, phenotypes, and genotypes of these neuropathies, currently focusing on Charcot-Marie-Tooth patients.

CO14. The role of ultrasonography in the diagnosis of giant cell arteritis

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Giant cell arteritis (GCA) is a primary autoimmune vasculitis that specifically affects medium-sized extracranial arteries, such as the superficial temporal arteries (TAs). The most important data to be considered for the ultrasound (US) diagnosis of temporal arteritis are stenosis, acute occlusions, and the "dark halo" sign, which represents edema of the vascular wall. Vessel wall thickening of large arteries in GCA can be detected by US, which has high sensitivity and is easy to use.

Ocular complications of GCA are common and consist primarily of anterior ischemic optic neuropathy or central retinal artery occlusion, presenting with sudden and painless loss of vision in the affected eye. Color Doppler imaging (CDI) of the orbital vessels (showing low end-diastolic velocities and a high resistance index) is essential to quickly differentiate the mechanism of ocular involvement (arteritic versus non-arteritic), since the characteristics of TAs on US do not correlate with ocular involvement in GCA.

Keywords: giant cell arteritis (GCA), "dark halo" sign, color Doppler imaging (CDI) of the orbital vessels

CO15. Update on amyotrophic lateral sclerosis therapies

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Amyotrophic lateral sclerosis (ALS) remains a devastating neurodegenerative disease with limited treatment options, necessitating ongoing research to develop effective therapies. ALS is the most common neurodegenerative disorder of young and middle-aged adults, with unknown etiology and a heterogeneous clinical phenotype, and has a median survival of 36 months. This presentation provides an update on ALS therapies, encompassing pharmacological, non-pharmacological, and emerging treatment modalities.

Pharmacological interventions, including riluzole and edaravone, continue to be the mainstay of ALS management, albeit with modest efficacy. Recent advancements in understanding ALS pathophysiology have led to the exploration of novel therapeutic targets, such as RNA-based therapies and sodium phenylbutyrate–taurursodiol. Tofersen represents a novel approach to treating ALS by targeting the mutated SOD1 gene, a known genetic cause of familial ALS. Clinical trials have demonstrated promising results, suggesting that tofersen may slow disease progression by reducing the production of toxic SOD1 protein aggregates.

Non-pharmacological interventions, including multidisciplinary care, respiratory support, and nutritional optimization, play a crucial role in improving quality of life and prolonging survival in ALS patients. Moreover, the integration of telemedicine and assistive technologies has enhanced accessibility to care and support services for individuals with ALS. Despite these advancements, challenges persist in translating promising preclinical findings into clinically meaningful therapies. The heterogeneity of ALS phenotypes underscores the need for personalized treatment approaches and the development of precision medicine strategies tailored to individual patient profiles.

Keywords: ALS, riluzole, edaravone, tofersen

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CO16. Early management of post-stroke spasticity movement disorder

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Post-stroke spastic movement disorder (PS-SMD) is one of the main causes of severe disability in the chronic phase after stroke. The prevalence of SMD increases over time, affecting more than 28% of patients in the chronic phase. Its secondary complications – such as contracture, abnormal postures and/or movement patterns, and spasticity-associated pain – also increase when physical and medical management of PS-SMD is delayed in the early stroke phase.

Spasticity is a serious disorder requiring a structured management and treatment plan. Early treatment of PS-SMD in the subacute phase following stroke is recommended to avoid or reduce the development of post-stroke disability and to improve rehabilitation outcomes. A number of treatments are available for managing spasticity, including physical therapy, oral agents, botulinum toxin type A, casting, and splinting.

The efficacy of treatment with botulinum toxin type A, administered via eco/EMG-guided injection in muscles affected by spasticity, is typically observed within 7–10 days, with a maximum effect in 4–6 weeks. To optimize therapeutic effectiveness, it is recommended to combine injections with physical therapy as part of a comprehensive rehabilitation program.

In conclusion, stroke represents a major public health problem worldwide and a significant cause of long-term disability, with spasticity being among the most frequent complications. The coordinated involvement of a multidisciplinary team early after stroke can improve function and quality of life.

Keywords: post-stroke spasticity, disability, early treatment, multidisciplinary team, botulinum toxin type A

CO17. Therapeutic strategies for chronic inflammatory demyelinating polyradiculopathy (CIDP)

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Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an inflammatory neuropathy with a relapsing or progressive course, characterized by diverse phenotypes and including both typical and variant forms. CIDP is an acquired immune-mediated neuropathy typically characterized by macrophage-induced demyelination, and affects approximately 2.81 per 100,000 people. Diagnosis of CIDP relies on a combination of clinical, electrophysiological, imaging, and laboratory features, along with the exclusion of other disorders that share similar symptoms.

First-line treatments, as recommended by international guidelines, include immunoglobulins (Ig), corticosteroids, and plasma exchanges. Corticosteroids may be chosen as first-line therapy in cases where IV Ig is not available or is contraindicated. Plasma exchange requires good vascular access and specialized equipment.

We discussed treating CIDP with immunoglobulins (Ig) at the diagnostic, induction, and follow-up stages, including the management of Ig dependence. IV Ig induction phase therapy should only be initiated in patients with moderate or severe disability that impacts daily life. The rationale for maintenance therapy should ideally be based on objective measurements demonstrating immunoglobulin dependence. Dependence is defined by a positive response to treatment and a significant relapse when the treatment is stopped. Maintenance therapy should offer immunoglobulins to patients, either via the intravenous or subcutaneous route. The current practice is to periodically reduce the dose or increase the interval between treatment courses while monitoring the response using validated outcome measures.

Patients should be followed regularly by a neurologist, as well as by physical and occupational therapists. CIDP may impact patients' ability to work and perform daily activities.

Keywords: CIDP, treatment, induction, maintenance, IV Ig

CO18. Intracerebral hemorrhage. What is new?

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Intracerebral hemorrhage is a significant neurological emergency associated with a high mortality rate, despite current therapeutic efforts. The most clinically expressed category of intracerebral hemorrhage is intraparenchymal hemorrhage, which can be caused by several factors, including the administration of antithrombotic treatment (antiplatelet and/or anticoagulant agents).

Intraparenchymal hemorrhage related to anticoagulant administration is a potentially lethal complication, with mortality rates reaching as high as 85–90%. Adverse effects of antiplatelet or anticoagulant drugs affect an increasingly large population segment, and alongside an increase in life expectancy, this has led to an increased incidence of intraparenchymal hemorrhage.

Surgical intervention (minimally invasive neurosurgical procedures) in cases of intraparenchymal hemorrhage has been proven, in recent clinical studies, to yield superior outcomes compared to conservative treatment, but it is reserved for selectively identified cases, based on specific criteria related to hemorrhage size, its location, and the patient's comorbidities.

In instances of intraparenchymal hemorrhages with distinct etiology stemming from anticoagulant therapies involving direct action on coagulation factors, there is currently a therapeutic arsenal consisting of biological therapies: a monoclonal antibody (idarucizumab) and an active biological agent (andexanet).

Andexanet is a specific reversal agent for factor Xa inhibitors—apixaban, edoxaban, and rivaroxaban—while idarucizumab is the specific antidote for dabigatran, a direct thrombin inhibitor.

In the present work, we will discuss the methods of administration of antidotes for the most commonly used direct anticoagulants in current medical practice.

Keywords: anticoagulant, intraparenchymal hemorrhage, antidote

CO19. Pitfalls of central vestibular syndrome

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Central vestibular syndrome is a common neurological disorder that clinically manifests as vertigo, postural instability, nystagmus, and autonomic dysfunction. It poses a challenge in current medical practice and sees a high degree of consultation both in outpatient clinics (primary care physicians, ENT specialists/neurologists) and in emergency departments.

In central vestibular syndrome, the brain's ability to integrate information from the vestibular system (responsible for maintaining balance) is compromised. This condition occurs due to inflammatory, neurodegenerative, or vascular lesions in the central nervous system, primarily located in the infratentorial region, but not exclusively.

Rapid identification of the topographical lesion is necessary, and it is essential to differentiate it from peripheral vestibular syndrome, with which it frequently shares clinical features. Although the clinical presentation may suggest a peripheral cause of the vestibular syndrome, the lesion that determines the severity of this condition can be located in the central nervous system.

Central vestibular syndrome may be part of the clinical manifestations of cerebrovascular disease (the classic Wallenberg syndrome involving the brainstem), may coexist within the constellation of demyelinating diseases (such as multiple sclerosis), or could be a clinical expression of brain tumors.

The diagnosis of central vestibular syndrome relies on accurate medical history, detailed clinical examination—including specific vestibular function tests—and cerebral imaging, with magnetic resonance imaging (MRI) being the primary indication.

Therapeutic management of central vestibular syndrome entails an etiopathogenic approach while also addressing symptomatology (vertigo/postural instability) and subsequently incorporating vestibular rehabilitation therapy.

In the present work, we we describe a series of cases where the initial clinical expression (temporally incomplete) may have led to diagnostic confusion.

Keywords: central vertigo, diagnosis, clinical presentation

CO20. Utilization, effectiveness, safety, and tolerability of adjunctive brivaracetam in patients with focal-onset seizures in mid-European countries and the Romanian subpopulation: interim real-world data from the BRIVA-REG study

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**Susanne Müller-Scholtz was an employee of UCB Pharma at the time of study commencement and is now working as an independent consultant.*

Introduction/Objective. The BRIVAracetamREGistry (BRIVA-REG/EP0099) is an ongoing, prospective, non-interventional, post-marketing study exploring the long-term utilization of adjunctive brivaracetam (BRV) treatment for focal-onset seizures (FOS) in a clinical practice setting.

Materials and methods. This is a planned interim analysis from BRIVA-REG (snapshot: 01-Jun-2023) assessing patients aged ≥4 years with FOS, with or without focal to bilateral tonic-clonic seizures, in six mid-European countries.

Results/Conclusions. In total, 683 patients had received ≥1 BRV dose at the snapshot date (Safety Set/SS; 387 in the Per Protocol Set/PPS). Median age (SS/PPS) was 40.0/41.0 years; 54.8%/55.3% were female. Mean time since epilepsy diagnosis was 18.65/18.61 years (n = 679/384). Median baseline FOS frequency per 28 days was 3.0/3.0 (n = 614/348). The median number of concomitant antiseizure medications (ASMs) at BRV initiation was 2.0/2.0 (n = 660/387). The 6-month BRV retention rate was 90.9%/90.0% (n = 483/279); 30.4%/27.2% of patients were seizure-free from baseline to 6 months (n = 289/173). Treatment-emergent adverse events (TEAEs) were reported in 11.6%/12.7% of patients; 2.3%/2.1% discontinued due to TEAEs.

In the Romanian subpopulation, the SS and PPS included 130 and 68 patients, respectively. Median age (SS/PPS) was 42.5/42.0 years (n = 126/67); 56.2%/57.4% were female. Mean time since epilepsy diagnosis was 18.13/18.25 years. Median baseline FOS frequency per 28 days was 2.00/2.67 (n = 121/65). The median number of concomitant ASMs at BRV initiation was 2.0/2.0 (n = 127/68). The 6-month BRV retention rate was 89.4% (n = 47); 36.0% of patients were seizure-free from baseline to 6 months (n = 25) (PPS). TEAEs (SS/PPS) were reported in 6.9%/10.3%; 1.5%/1.5% discontinued due to TEAEs.

Adjunctive BRV was well tolerated and effective in patients with FOS in routine clinical practice in mid-European countries and in the Romanian subpopulation.

Keywords: antiseizure medication, brivaracetam, effectiveness, epilepsy, focal-onset seizures, non-interventional study, prospective, post-marketing study, Romania, tolerability

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CO21. Super-refractory status epilepticus and uncontrolled arterial hypertension

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Introduction. Super-refractory status epilepticus (SRSE) is a life-threatening neurological emergency with high morbidity and mortality. It is defined as status epilepticus that continues or recurs 24 hours or more despite anesthetic treatment. Any pathology that can trigger an acute symptomatic seizure can cause status epilepticus, which may progress to SRSE. This includes neurological and systemic infections, acute vascular events, traumatic brain injury, and immune, metabolic, or toxic encephalopathies, including PRES.

Objectives. In this case presentation, we aim to exemplify the diverse acute systemic consequences of uncontrolled arterial hypertension.

Material and method. We present the case of a 77-year-old woman who was brought to the emergency room for altered state of consciousness, involuntary movements of the right limbs, and increased blood pressure values.

History was positive for uncontrolled hypertension and smoking.

Extensive workup was done, consisting of repeated brain imaging, repeat EEG monitoring, lumbar puncture, and extensive blood work.

Results. Continuous monitoring of blood pressure showed increased values despite aggressive treatment. Serial EEG was indicative of focal status epilepticus, which proved to be refractory to standard therapy. Brain MRI revealed posterior left white matter edema and small bilateral watershed strokes.

Conclusions. After the complete workup, a diagnosis of PRES with SRSE was reached, which, in the context of acute uncontrolled hypertension, explains the complex clinical picture.

CO22. End of the road in Parkinson's disease. Dispelling a myth

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Parkinson's disease (PD) itself is not a direct cause of death. Many PD patients can enjoy an almost normal life. However, data from life expectancy and mortality studies in PD, despite modern treatment, show that PD patients may live somewhat less than people in the same age group. Even if these studies included selected patient populations, their results allow the clinician to be guided in assessing, step by step, the individual patient's prognosis.

It has been proven that the evolution of PD patients over time is strongly influenced by the motor and non-motor complications determined by disease progression. These are closely related to the complex pathophysiology of PD and to the variety of phenotypic expression. Young age at onset, male sex, non-tremor dominant phenotype with more severe motor impairment, early autonomic dysfunction, and the presence of psychotic symptoms and dementia predict a decrease in life expectancy in PD patients. Cardiovascular comorbidities, traumatic events, and recurrent infections also influence mortality.

Early diagnosis of PD, correct monitoring of disease progression, and appropriate treatment of comorbidities, psychotic symptoms, and dementia using a multidisciplinary approach are important ways to help patients live a life as close to normal as possible.

Keywords: Parkinson's disease, life expectancy, mortality

CO23. Gepants in migraine therapy

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CGRP is a 37-amino acid neuropeptide derived from the gene encoding calcitonin. It is a potent vasodilator and also functions as a messenger in nerve cells, with an important role in migraine pathophysiology.

CGRP (calcitonin gene-related peptide) and its receptors are the targets for new therapies for the prevention and acute treatment of migraine.

Initially, research focused on the development of small-molecule drugs designed to block CGRP receptors.

However, while these were effective in the acute treatment of migraine, there were a number of issues, including signs of liver toxicity with some first-generation gepants.

Despite initial disappointment with small-molecule CGRP antagonists, further research has led to the development and approval of three agents with reassuring safety profiles, approved by the FDA:

- Ubrogepant (Ubrelvy) is approved for the acute treatment of migraine.
- Rimegepant (Nurtec ODT/Vydura) is approved for both acute and preventive treatment of migraine.
- Atogepant (Qulipta) is approved for migraine prevention.

CO24. Alpha avalglucosidase in the treatment of late-onset Pompe disease – efficacy and safety data

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Background. Pompe disease is a rare, progressive neuromuscular disorder caused by a deficiency of acid

α-glucosidase (GAA) and the accumulation of lysosomal glycogen.

Material and methods. Avalglucosidase alfa, a new recombinant human GAA enzyme replacement therapy specifically designed for enhanced mannose-6-phosphate receptor targeting and enzyme uptake aimed at increased glycogen clearance, has been compared with the currently approved standard of care, alglucosidase alfa, in patients with late-onset Pompe disease in a study registered with ClinicalTrials.gov (NCT02782741), conducted between Nov 2, 2016, and March 29, 2019. This presentation will discuss the study and its outcomes in terms of efficacy and safety.

Results. This was a randomized, double-blind, phase 3 trial conducted at 55 sites across 20 countries. It enrolled 100 individuals (aged ≥3 years) with enzymatically confirmed late-onset Pompe disease who had never received treatment and were randomly allocated to receive either avalglucosidase alfa or alglucosidase alfa. The primary outcome measure (non-inferiority) was the change from baseline to week 49 in upright forced vital capacity percent (FVC%) predicted. The key secondary objective was the effect on functional endurance, measured by the 6-minute walk test (6MWT). Safety was assessed, including treatment-emergent adverse events and infusion-associated reactions. The modified intent-to-treat population was the primary analysis population for all efficacy analyses.

Conclusion. The study provides evidence of clinically meaningful improvement with avalglucosidase alfa therapy over alglucosidase alfa in respiratory function, ambulation, and functional endurance, with no new safety signals reported. An open-label extended treatment period is ongoing to confirm the long-term safety and efficacy of avalglucosidase alfa, with the aim of establishing this therapy as the new standard treatment for late-onset Pompe disease.

CO25. Autoimmune encephalitis – from guidelines to clinical practice

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Background. Autoimmune encephalitis represents the generic definition of a syndrome characterized by rapidly progressive encephalopathy, memory deficits, altered mental status, and/or psychiatric symptoms.

Material and methods. Syndrome-based criteria for autoimmune encephalitis (AE) were proposed as part of a diagnostic algorithm to help clinicians navigate differential diagnoses while awaiting antibody results. Failure to adhere to these criteria contributes to misdiagnosis, leads to morbidity from unnecessary immunotherapies, and delays treatment of the correct diagnosis. However, some patients with neural antibody-associated AE do not meet possible AE criteria, indicating suboptimal sensitivity.

Results. Neural antibody-based diagnostic criteria could capture such patients and complement the syndrome-based algorithm to improve sensitivity while maintaining high specificity. However, there may be concerns that neural antibody testing could contribute to increased testing in low-probability scenarios, decreasing positive predictive value and increasing the proportion of false-positive antibody results, which can lead to inappropriate immunotherapy and patient harm. The development of neural antibody-based diagnostic criteria aligns with the definite AE classification assigned by the syndrome-based diagnostic algorithm when a particular neural antibody is present.

Conclusion. In the present work, we present the current theoretical status with clinical application, presenting cases admitted during 2020–2024 to our clinic, from clinical diagnosis to ancillary testing and therapeutic response.

CO26. The profile of the patient eligible for continuous administration of levodopa/carbidopa treatment

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Background. Identifying advanced Parkinson's disease is still a challenge for many neurologists, due to the progressive character of the disease and also to the specific clinical picture and individual evolution for each patient. The combinations of treatments for Parkinson's disease should be tailored to each person's unique symptoms, depending on age, patient preferences, mental and cognitive status, duration of illness, physical and social activity, and presence of a caregiver. In the advanced Parkinson's disease stage, oral medications are less effective and/or their side effects outweigh their benefits, so a non-oral (continuous dopaminergic stimulation – CDS) approach is recommended.

Material and methods. The MANAGE-PD tool is a modern, validated clinical tool to identify uncontrolled PD patients, as well as those eligible for device-aided therapies. There are device-assisted therapies (DATs) providing CDS: infusion therapies with levodopa/carbidopa ± entacapone, as intestinal gel, or continuous administration of apomorphine, as well as deep brain stimulation.

Results. Levodopa remains the gold standard in PD treatment for all disease stages. In this respect, the "newcomer" is levodopa as continuous dopaminergic stimulation (foslevodopa/foscarbidopa subcutaneous infusion). Foslevodopa/foscarbidopa subcutaneous infusion addresses advanced Parkinson's disease with severe motor fluctuations and/or hyper/dyskinesia when available combinations of therapies did not offer satisfactory results.

Conclusion. The current presentation approaches the clinical profile of the eligible patient for CDS with levodopa/carbidopa, either as intestinal gel administered through percutaneous endoscopic gastrostomy (PEG) or by less-invasive subcutaneous infusion as foslevodopa/foscarbidopa.

CO27. Treatment by terminal complement inhibition in gMG – the Romanian experience with ravulizumab in early access program

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Background. Myasthenia gravis (MG) is a rare, chronic autoimmune disease characterized by fluctuating and fatigable skeletal muscle weakness resulting from impaired neuromuscular transmission. Uncontrolled disease increases the risk of clinical deterioration, including life-threatening myasthenic crises requiring intensive care and respiratory support.

A fundamental pathogenic mechanism of MG is the aberrant complement activation by autoantibodies against the acetylcholine receptor (AbAchR), causing the most common subtype of the disease. Activation of the classical complement pathway leads, in a successive, cascade-wise protein lysis, to the cleavage of C5 into C5a and C5b, with C5b leading to the assembly of the membrane attack complex (MAC).

The MAC induces cell lysis and death via disruption of the target cell membrane and damages the postsynaptic membrane at the neuromuscular junction (NMJ), reducing the membrane surface area and the number of acetylcholine receptors and voltage-gated sodium ion channels.

Material and methods. Complement 5 inhibitors bind specifically with high affinity to the human terminal complement protein C5, blocking its cleavage and thus preventing the cascade of events that leads to MAC-mediated architectural destruction of the NMJ postsynaptic membrane.

Immunosuppressive therapies used to treat gMG suppress cellular mediators of the immune response, but most have a slow onset of action and do not directly target the complement system.

Results. Ravulizumab represents the second generation of complement inhibitors and was engineered from eculizumab to have an extended half-life and longer duration of action, maintaining therapeutic serum concentrations over an 8-week dosing interval.

Conclusion. Ravulizumab is indicated for the treatment of generalized MG (gMG) in adult patients who are AbAchR-positive.

CO28. Post-COVID-19 syndrome and neurological involvement

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According to the WHO definition, post-COVID-19 syndrome (also referred to as chronic COVID syndrome, persistent COVID-19, or long COVID) manifests in individuals diagnosed with SARS-CoV-2 infection at least 3 months prior, presenting as a combination of neurological sequelae from the acute phase or new-onset symptoms that emerge after the resolution of the infection. These symptoms persist for a minimum of 2 months, without another explanation.

The clinical presentation encompasses a range of nonspecific symptoms affecting all organ systems, with a predominance in females. The neurological involvement is termed neuroCOVID and often presents as disabling symptoms, including fatigue, generalized musculoskeletal pain, peripheral neurological disor-

ders (such as paresthesia, neuropathies and plexopathies), persistent disturbances in smell and taste, brain fog, concentration and memory difficulties, vertigo, depression/anxiety, sleep disturbances, headaches, and autonomic dysregulation. Additionally, rare cases of autoimmune encephalitis, myelitis, and encephalomyelitis have been reported. There is also an increased risk of cerebrovascular disease within the first year following SARS-CoV-2 infection.

The primary pathophysiological mechanisms implicated in neurological dysfunction include direct viral invasion, the systemic inflammatory response, and a prothrombotic state. The suggested mechanisms for neuroinvasion are: (1) direct viral infection of the CNS via the olfactory bulb, (2) trans-synaptic transmission, (3) neuroinfection through the bloodstream, and (4) the dissemination of the infected immune cells through the nervous system. Viral infections have been associated with triggering autoimmune responses, often in conjunction with genetic predisposition and environmental factors.

The disruption of the blood-brain barrier is exacerbated in the presence of various comorbidities, leading to increased astroglial reactivity and triggering a neuroinflammatory cascade that may induce para-infectious autoimmunity. The primary mechanisms contributing to viral-induced autoimmunity include molecular mimicry, bystander activation, and epitope spreading.

The diagnosis of post-COVID-19 syndrome requires a multidisciplinary approach, and treatment is primarily symptomatic or immunomodulatory therapy in case of immune involvement.

CO29. Evaluating residual stroke risk for patients with atrial fibrillation

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Background. Atrial fibrillation (AF) is a major risk factor for stroke, and oral anticoagulation (OAC) is the cornerstone of stroke prevention. However, a number of patients experience stroke despite OAC therapy, highlighting residual stroke risk.

Objective and methods. This presentation will offer a review of current data regarding residual stroke risk in AF patients treated with OAC. Studies investigating risk factors, clinical characteristics, and potential modifiable targets will be presented, focusing on practical aspects.

Results. Despite OAC, several factors contribute to residual stroke risk. These include advanced age, hypertension, diabetes, prior stroke or transient ischemic attack (TIA), and persistent AF. Additionally, suboptimal management of modifiable risk factors and medication adherence play a role.

Conclusion. Residual stroke risk in AF patients on OAC is a significant clinical challenge. Neurologists can play a crucial role by identifying high-risk patients, optimizing modifiable risk factors, and ensuring adherence to OAC therapy. Future research should investigate personalized risk stratification and potential therapeutic strategies to further reduce residual stroke risk in this population.

CO30. Bruton's tyrosine kinase inhibitors

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Background. Multiple sclerosis (MS) is a chronic, immune-mediated disease of the central nervous system, where both inflammation and neurodegeneration play key roles. Bruton's tyrosine kinase (BTK), a critical enzyme mediating B-cell and myeloid cell function, has become a compelling therapeutic target. BTK inhibitors (BTKIs) offer a novel approach to modulate both the adaptive and innate immune systems.

Objective and methods. This review explores the development of BTKIs, their specific mechanisms of action, and the current body of evidence supporting their use.

Results. BTKIs target both peripheral immune responses and microglial activation in the CNS. Many experts believe they might hold the key to tackling smoldering MS, but the clinical impact of their use will become clear once data from ongoing large clinical trials becomes available.

Conclusion. BTK inhibitors represent a promising class of disease-modifying therapies in MS. Their potential to address chronically active lesions in MS may change the therapeutic landscape of tomorrow. Understanding their full role in managing MS will be key to optimizing patient outcomes.

CO31. Neurological manifestations of antiphospholipid syndrome

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Neurological manifestations of antiphospholipid syndrome (APS) are heterogeneous and sometimes difficult to address. In the recently updated classification criteria for APS (i.e., 2023 ACR/EULAR Antiphospholipid Syndrome Classification Criteria), two clinical criteria have been assigned to cerebral thrombotic events, namely Domain 1 – macrovascular (i.e., venous thromboembolism, that also includes cerebral venous thrombosis) and Domain 2 – macrovascular (i.e., arterial thrombosis, that includes stroke). Apart from the well-known cerebral thrombotic events, neurological non-thrombotic manifestations might also occur in APS (non-criteria manifestations). Migraine, seizures/epilepsy, movement disorders, multiple sclerosis-like disease, transverse myelitis, cognitive impairment/dementia, psychiatric manifestations, peripheral neuropathy, and dysautonomia have been referenced as frequent comorbid conditions in APS and/or manifestations of APS or antiphospholipid antibody positivity. The pathogenesis of non-thrombotic manifestations in APS is complex and presumably relates to microthrombosis and immune-mediated neuronal damage, the latter elicited by diffuse endothelial dysfunction and disrupted blood-brain barrier, with antiphospholipid antibodies targeting nervous system antigens. Atypical epileptic seizures in young patients with multiple cerebral lesions of unidentified cause, refractory migraine, and atypical presentations in multiple sclerosis should raise suspicion of APS. A trial of antithrombotic or anticoagulant therapy might be attempted in certain clinical settings, after weighing potential risks.

CO32. Diagnosis and treatment of impulse control disorder in patients with Parkinson's disease

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Introduction. Impulse control disorders (ICDs), such as compulsive gambling, buying, sexual, and eating behaviors, are a serious complication in Parkinson's disease (PD). Related behaviors include punding (stereotyped, repetitive, purposeless behaviors), dopamine dysregulation syndrome (DDS) (compulsive medication overuse), and hobbyism (e.g., compulsive internet use, artistic endeavors, and writing). These occur in up to 20% of PD patients over the course of the illness and have a significant impact on quality of life and patient function, strain interpersonal relationships, and are associated with significant psychiatric comorbidity.

Methods. In this presentation, we aim to discuss the frequency and types of ICDs, risk factors, and the types of antiparkinsonian medications that can induce them. We will also analyze the main mechanisms behind ICD development in PD, diagnostic tools, and potential therapeutic resources for ICDs.

Conclusions. ICDs are challenging to discern in the context of routine visits. Patients may be ashamed to report them, may think these symptoms are irrelevant to their PD, or may lack insight. The primary treatment of ICDs in PD is discontinuation of the offending treatment, but not all patients can tolerate this due to worsening motor symptoms or dopamine agonist (DA) withdrawal syndrome. There is limited empirical evidence for psychiatric medications and psychosocial treatments in PD, so it is critical for patients to be monitored closely for ICDs from disease onset and throughout its course. A multidisciplinary approach is recommended, including aggressive patient and caregiver education, vigilant monitoring for ICDs, and related impulsive–compulsive behaviors.

Keywords: impulse control disorders, mechanisms, diagnosis, treatment

CO33. Redefining generalized myasthenia gravis treatment

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Generalized myasthenia gravis (gMG) is a rare, chronic, IgG-mediated, neuromuscular autoimmune disease that causes debilitating and potentially life-threatening muscle weakness. Most patients with MG first present with only ocular symptoms, but up to 80% progress to gMG within 2 years of initial disease onset. Due to a lack of consensus in assessment practices, the extent of disability and disease burden may not be fully appreciated. Muscle weakness in gMG results from impaired transmission at the NMJ mediated by IgG autoantibodies. Treatment response varies among patients with gMG and often depends on serotype and disease status. Specific autoantibodies are associated with treatment response, so their presence may impact treatment decisions. Along with serotype, onset of disease and comorbidities should inform treatment approach at diagnosis. Patients who are refractory to cholinesterase inhibitors or immunosuppressive agents have traditionally had limited treatment options. Treatment individualization in gMG requires an understanding of the relationship between a multitude of factors. MG mortality has decreased over time, leading to a shift in treatment goals toward managing disability. Traditional therapies are associated with a range of treatment-specific hurdles. New targeted treatments inhibit the mechanisms of FcRn, IL-6, and complement to reduce damage induced by these mediators. With the development of new targeted therapies, a shift in the focus of treatment goals toward achieving remission and a good quality of life (QoL) may be possible.

CO34. Secondary-progressive multiple sclerosis – Is early diagnosis possible?

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Materials and methods. We conducted a unicentric, case-control, cross-sectional observational/non-interventional study, composed of two study arms: patients diagnosed with RRMS (study group) and patients diagnosed with SPMS (control group). In addition to taking a detailed history, the participants were assessed clinically and through specific tests (EDSS plus) and questionnaires (LMSQoL, HADS). Using binary logistic regression tests, a probability score was developed.

Results/Conclusions. A higher prevalence of MS was observed in female patients. The majority of SPMS patients were retired, and they reported more previous relapses than RRMS patients. SPMS patients had significantly higher scores in both the total EDSS and its individual domains, as well as greater cognitive impairment and elevated levels of depression and anxiety. A predictive model that can discern the likelihood of SPMS diagnosis based on several key variables was developed.

Keywords: multiple sclerosis, secondary-progressive multiple sclerosis, risk factors, risk score