Ref:Ro J Neurol. 2020;XIX(Suppl)
DOI: 10.37897/RJN.2020.S.2

The 18th National Congress of the Romanian Society of Neurology PROCEEDINGS

POSTERS – ABSTRACTS

(in alphabetical order of the first author)

NEUROIMUNOLOGIE / *NEUROIMMUNOLOGY*

Small discoveries can have great consequences – a peculiar case of neuro-Behçet

F. ANTONESCU[1,2], F. IVANCIU[1], E. MIHAI[1], N. ANGHEL[1], S. TUȚĂ

[1]*National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania*
[2]*"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Behçet disease is a chronic, inflammatory condition, characterized by oral and genital ulcerative lesions, generally associated with systemic manifestations. We will be presenting the case of a patient in which the changes on MRI were not suggestive, but a careful clinical examination was key to the diagnosis.

Objective and methods. We will be presenting the case of a patient admitted to our clinic with acute transient focal neurological symptoms, suggesting a transient ischemic attack, followed by progressive confusional state, cognitive impairment and fever. At first, we suspected an infectious meningoencephalitis due to changes on MRI, but the CSF examination disproved this diagnosis. The patient also presented with pulmonary manifestations, as well as deep vein thrombosis on the lower right limb which extended to inferior vena cava. A careful clinical reexamination showed the presence of oral ulcerative lesions, a major criterion for diagnosis of Behçet disease. The initial immunosuppressive treatment was cortisone, to which we added azathioprine, with a slowly favorable evolution with marked improvement of the clinical and imaging parameters.

Conclusions. Our case is particular in the way that the first symptom was a stroke mimic, a phenomenon also encountered in other types of autoimmune encephalopthies. Currently, there are no investigation methods available which can confirm with certainty the diagnosis, this remains a diagnosis of exclusion in which the clinical findings (oral and genital ulcers, uveitis) remain paramount.

Chitinase 3-like-1 – a potential biomarker for benign multiple sclerosis

Laura BARCUTEAN, Smaranda MAIER, Rodica BALASA

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

Introduction. Chitinase 3-like-1(CHI3L1) is a serum protein secreted by the immune cells and its presence in the inflammatory lesions suggest that it may be an essential player in the astrocytic response in modulating the central nervous system inflammation. Benign multiple sclerosis (B-MS) is a controversial diagnosis that has no definite consensus criteria.

Material and method. We selected 20 MS patients with an EDSS score ≤2.0, treated continuously with Interferon beta-1b (IFNβ1b) for at least 10 years and 20 healthy controls (HC). Venous blood samples were harvested from both groups and underwent serum CHI3L1 analysis. Both clinical and demographical data were assessed in the chosen B-MS lot, including evolution of the disease before and after the treatment was started, type of onset, cognitive and ambulatory assessment.

Results. The mean age of B-MS patients was 47.75±7.07, and of HC was 39.8±9.42. The B-MS patients had an average of 18.1 years of evolution, being under active IFNβ1b treatment for more than 14 years, and with a median EDSS of 1.5. No statistical significance was found when comparing the median level of CHI3L1 levels in B-MS lot with HC (p=0.085).

Conclusion. A long disease evolution and a low disability score carries a good prognosis, but it is uncertain if this follows the natural trend of the disease or we are faced with a homogenous lot of perfect IFNβ1b responders. The lack of statistical significance in comparing the CHI3L1 levels between B-MS patients and HC indicates that the MS patients have a similar peripheral immune reaction as normal population.

Is there any use for event-related auditory evoked potentials in multiple sclerosis? – A preliminary study

S.M. BULAI[1], A. LEONTE[1], D.A. SPÎNU[1], E.B. IGNAT[1,2]

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

Cognitive impairment affects patients suffering from multiple sclerosis and even though it was believed to be characteristic to late stages of the disease, it is now known to be frequent, sometimes early, and an important cause of disability. Recent recommendations include cognitive screening of MS patients and annual re-assessment. For patients showing cognitive decline on minimal evaluation, there are other validated evaluation tools that can be used by clinicians for a more extensive assessment.

We present a preliminary report regarding our experience in the Hospital of Rehabilitation in Iasi, following a 3 year period of cognitive assessment of MS patients, using electrophysiological evaluation along with other clinical and imaging screening tools. As a neurophysiological indicator of cognitive function we used the P300 wave, a late component of the event related potential. It is elicited by distinct (target) stimuli delivered among a series of standard, non-target stimuli, thus depending on the subject's attention and working memory.

We present the correlations emerged while also discussing the importance and utility of the selected tests.

An atypical case of multiple cranial neuropathies in Sjögren's syndrome

A.M. DOBRI[1], G.M. MOCI[1], L Nistorec[1], I.A. OLARU[1], L. PINTE1, G.A. SÎRBU[3], M.M. MANEA[1,2], C. BAICUS, S. TUȚĂ[1,2]

[1] National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania
[2] "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3] Diagnostic Center Medinst, Bucharest, Romania
[4] Internal Medicine Department, Colentina Clinical Hospital, Bucharest, Romania

Objectives. Primary Sjögren's syndrome (SS) is an autoimmune inflammatory disease, mainly affecting the salivary and lacrimal glands, but may be complicated by neurological manifestations, involvement of cranial nerves being the rarest.

Methods. A 65-year-old patient, known with compensated essential hypertension, is admitted for sudden onset binocular diplopia. The neurological examination revealed horizontal diplopia and convergent strabismus of the left eye (left abducens nerve palsy), bilateral abolished corneal reflex (bilateral trigeminal nerve impairment), slight peripheral right facial palsy, otherwise normal clinical examination.

Results. Cerebral MRI scan with contrast revealed enhancing lesions at the level of the intralabyrinthine segment of the right facial nerve, the maxillary and mandibular branches of the bilateral trigeminal nerves and at the level of the retroclival meninges; without any lesions of the cerebral parenchyma. CSF biochemistry and cytology analysis was normal. Bioumoral tests excluded other infectious diseases (TB, *Borrelia burgdoferi,* HIV, syphilis, hepatitis B or C infection). Angiotensin convertase was negative. Other connective tissue diseases were excluded, with the exception of SS antibodies (antiLa antibodies, anti-Ro antibodies, positive ANA antibodies) and the ultrasound imaging which revealed suggestive changes of SS in the parotid glands. One month after the onset of neurological symptoms, the patient developed xerostomia and dry eye syndrome, meeting the diagnostic criteria.

Conclusions. Neurological manifestations may precede the clinical onset of SS, therefore, in patients with apparently isolated involvement of the cranial nerves, a correct diagnosis of the underlying SS may often be delayed.

Clinical and paraclinical features of posterior reversible encephalopathy syndrome (PRES) in systemic lupus erythematosus (SLE)

Mustafa FILIS-ELIF[1], Corina GATU[1], Simona PETRESCU[1,2], Cristina Aura PANEA[1,2]

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

Cerebral autoregulation is a homeostatic process that regulates and maintains cerebral blood flow constant across a range of blood pressures, with limits ranging from 50-150 mmHg cerebral perfusion pressure (CPP) or 60-160 mmHg mean arterial pressure (MAP). This mechanism ensures an increase in resistance in the small cerebral arteries as CPP or MAP increases.

Objectives. The symptoms of RPES syndrome evolve rapidly, being characterized by headaches, altered con-

sciousness, visual disturbances and seizures. Neuroimaging reveals bilateral edema of the white matter in the posterior cerebral hemisphere, especially in the parieto-occipital region.

Methods. A 33-year-old woman, with history of SLE with hematological, articular and renal determinations, in treatment with corticosteroids and hydroxychloroquine, was admitted for severe occipital headache with recent onset and high blood pressure. At the neurological examination, she had temporal and spatial disorientation, bradypsychia, bradylalia, blinked inconsistently at visual threats in both visual hemifields, weakly reactive bilateral miosis, oculomotor disorder with bilateral abduction limitation, plantar cutaneous reflex in extension bilaterally. Biologically, she had mild leukocytosis with neutrophilia and minimal lymphopenia, bicytopenia, moderate inflammatory syndrome, significant nitrogen retention syndrome, high D-dimers.

Results. Native MRI examination revealed diffuse bilateral lesions with hyperintensity on T2 and Flair, predominantly at the level of the structures irrigated by the posterior circulation (bilateral occipital and cerebellar), with superficial and deep topography.

Conclusions. The occurrence of PRES syndrome associated with SLE was mainly caused by hypertension induced by renal failure and the antihypertension treatment was the most important to reverse the RPES lesion.

Keywords: posterior reversible encephalopathy syndrome (PRES), systemic lupus erythematosus (SLE)

CLIPPERS syndrome: A rare cause of gait impairment – case report

E. FINNA[1], S. MAIER[1,2], Z. BAJKÓ[1,2], A. MOȚĂȚĂIANU[1,2], R. BĂLAȘA[1,2]

[1] Neurology I Department, Emergency Clinical County Hospital, Targu-Mures, Romania
[2] Discipline of Neurology, UMFST, Targu-Mures, Romania

Introduction. Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS)-syndrome is an inflammatory disease of CNS with distinct clinical and radiological features, representing a rare cause of uncoordinated gait amongst other specific CNS symptoms. An important aspect of the diagnosis is significant clinical and radiological responsiveness to glucocorticosteroid (GCS)-based immunosuppression.

Case report. A 45-year-old male patient with history of electric shock, with no prior medical history, presented with progressive gait instability, developing progressive weakness and pain in lower limbs, with marked improvement under administration of GCS shortly after onset of symptoms. After two years the patient presented with recurrence of uncoordinated gait, lower limbs weakness, facial paresthesia, diplopia and dizziness. Neurological examination revealed Lhermitte's sign, grade 4/5 paraparesis, palmomental reflex, exaggerated deep tendon reflexes in lower limbs and bilateral Babinski sign. Investigations including routine bloodwork, autoimmune-bloodwork, infectious markers, Borrelia burgdorferi antibodies, anti-aquaporin-4 antibodies, angiotensin converting enzyme, chest x-ray, ECG, electromyoneurography revealed no abnormality. Cerebrospinal fluid examination showed absence of oligoclonal bands. Brain MRI scan showed a bulky pons with mildly hypointense lesions on T1-weighted images, being hyperintense on T2 and FLAIR image, with gadolinium contrast enhancement giving a typical 'peppering of pons' appearance. MRI scan of the spinal cord also revealed hyperintense T2-weighted lesions in the cervico-dorsal region. After intravenous corticotherapy continued by 4-month-long oral GCS treatment, regression of symptoms occured and at 6-month follow-up, MRI imaging showed resolution of lesions.

Conclusions. After excluding pathological conditions that may result in similar clinical and radiological features and taking into consideration the success of GCS therapy, the case was interpreted as CLIPPERS-syndrome. The core of early diagnosis and adecvate management is emphasising the importance to consider CLIPPERS in young patients with progressive gait impairment accompanied by other brainstem signs, therefore leading to a good prognosis as making this yet not fully discovered entity curable.

Natalizumab and persistent leukocytosis treatment considerations in a group of patients with relapsing-remitting multiple sclerosis

C. GROSU[1,2], M. STOICA[1], A. PLESCA[1], O. UNGUREANU[1]

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

Natalizumab (Tysabri) is a recombinant humanized α4-integrin antibody on the surface leukocytes, available in Romania since 2006, approved for the treatment of multiple sclerosis very active recurrent remiss form, proving its effectiveness by decreasing the number of

relapses and slowing the progression of disability. The literature considers the occurrence of leukocytosis in patients treated with Natalizumab as a haematological adverse effect, but there are also studies that conclude that it may be a biomarker of therapeutic efficacy.

We analyzed a group of 19 patients, registered in the Neurology Clinic, the Clinical Hospital of Recovery Iasi, diagnosed with recurrent remiss multiple sclerosis that are being treated with Natalizumab for at least 1 year. The average age of the study group was 36.7 years, 4 men and 15 women. The values of leukocytes, lymphocytes, neutrophils and were analyzed of ESR for each patient, for 1 year, monthly. Thus, the mean value of leukocytes was 9107 leukocytes / mm 3, of which 34% were increased. Also 19% of the values neutrophils and 69.7% of lymphocyte values were increased, ESR showing increased by 7.5%. We mention that no patient presented a burst during the period analyzed.

Large observational studies with standardized data collected from real experience are needed to provide additional information about the Natalizumab profile, to improve intake decisions and optimization of treatment management.

Keywords: Natalizumab, leukocytosis, multiple sclerosis

Electrophysiologic abnormalities in patients with Miller-Fisher syndrome

R. GURGU[1], V. MIHAI[1], F. MUSTAFA[1], M. MOLDOVAN[1], I. CODITA[1], C. PANEA[1,2]

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

Background. Miller-Fisher syndrome is a rare type of acute poliradiculoneuritis, with a typical clinical picture including ophtalmoparesis, ataxia and areflexia. It can also associate other clinical signs indicating involvement of other cranial nerves (facial paresis, swallowing difficulties, etc) as well as sensory disturbances in the limbs.

Objectives. Characterization of electrophysiological changes in patients with Miller Fisher syndrome.

Materials and method. We identified all patients discharged between 2007-2019 from the Neurology Department of Elias University Hospital with a diagnosis of acute poliradiculoneuritis - Miller Fisher variant. For all identified patients, we collected demographical data and the results of the nerve conduction studies performed during the admission.

Results and discussions. Between 2007 and 2019 there were 13 patients discharged from the Neurology Department of Elias University Hospital with a diagnosis of Miller Fisher syndrome. Complete results of the electrodiagnostic testing were available for only 9 patients. The assessed patients were 25 to 65 years old. Sex distribution was 5 males and 4 females. In total, the electrodiagnostic testing included 58 motor nerves (including evaluation of minimum latency of the F wave in 49 motor nerves) and 59 sensitive nerves. Only one patient had normal electrodiagnostic testing. In order of frequency, we observed the following changes of electrodiagnostic parameters: reduced SNAP amplitude (21 sensitive nerves in 6 patients), increased peak distal sensitive latencies (15 sensitive nerves in 6 patients), reduced SCVs (5 sensitive nerves in 4 patients), reduced distal CMAP amplitude (6 motor nerves in 3 patients), increased distal motor latency outside usual entrapment sites (3 motor nerves in one patient), reduced segmentary MCVs (4 motor nerves in one patient), possible conduction blocks (2 motor nerves in one patient) and increased minimum F wave latency (2 motor nerves in one patient). All changes described above (except reduced SNAP amplitude and reduced SVCs in sensitive nerves) were minimal and the electrophysiological criteria for acute demyelinating poliradiculoneuritis were not fulfilled.

Conclusion. The main electrophysiological changes in Miller-Fisher syndrome are usually found in sensitive nerves and are frequetly severe, but unspecific, for demyelinating disorder.

Keywords: Miller Fisher syndrome, electrophysiology

Diagnostic difficulties in atypical demyelinating lesion with pseudotumoral form. Case report

Raluca-Andreea IGNAT[1], Diana ELISEI[1], Liviu PENDEFUNDA[1], Anca SAVA[1,2], Gabriela Florenta DUMITRESCU[3]

[1] Neurology Departament II, "Prof. Dr. Nicolae Oblu" Emergency Clinical Hospital, Iasi, Romania
[2] Morphofunctional Science Department I, "Grigore T. Popa" University of Medicine and Pharmacy, Iasi
[3] Anatomical Pathology Departament, "Prof. Dr. Nicolae Oblu" Emergency Clinical Hospital, Iasi, Romania

Multiple sclerosis is the most common chronic disabilitating disease of the central nervous system in young adults. It is characterized by the presence of multiple inflammatory demyelinating lesions, disseminated in the CNS.

The pseudotumoral form of multiple sclerosis is considered a rare variant of the disease. The frequency is uncertain, but a prevalence of 3 cases per million inhabitants per year has been estimated, being described more frequently in women, in the second and third decade of life. The definition of pseudotumoral form is based on image explorations. However, the lesions have not yet been given an exact description (1).

There may be difficulties in establishing the diagnosis. The differential diagnosis should include tumors, abscesses and acute disseminated encephalomyelitis when the lesions are multiple.

Clinical evolution, cerebrospinal fluid analysis, new advanced MRI techniques, and response to steroid treatment could guide the clinician to diagnosis (2).

Due to their rarity, clinical behavior and exact lesion management are partially known to clinicians.

We present the case of a 24 year old patient, without any special medical history, who presents in the neurology department for vertigo aggravated by head mobilization, static and dynamic balance disorders. MRI examination that detects multiple demyelinating lesions (subtentorial and supratentorial, some with pseudotumoral cystic appearance with maximum diameter ranging from 12 to 24 mm) raises differential diagnostic problems; additional clinical and paraclinical examinations, including brain biopsy, guide the etiologic diagnosis and therapeutic behaviour.

Keywords: demielinizing, pseudotumoral

References

1. Mauri-Fábrega L et al. Pseudotumoral Forms of Multiple Sclerosis: Report of 14 Cases and Review of the Literature. Eur Neurol. 2014;72:72-78.
2. Totaro R et al. Tumefactive demyelinating lesions: spectrum of disease, diagnosis and Treatment. Curr Neurobio. 2016;7(1):21-26.

Patient associating multiple sclerosis, epilepsy and intolerance to methylprednisolone – a difficult therapeutic approach

D.M. ILIE[1], G. MIHĂILESCU[1,2]

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

Introduction. Multiple sclerosis (MS) can associate epileptic seizures, most of them with focal onset, but it can also be associated with epilepsy as a prior disease. Even if under diagnosed the incidence of epilepsy in MS patients is statistically 3 to 6 times higher in both sexes, compared to the general population. Treating patients with MS and epilepsy may be challenging.

Case report. We present the case of a 41 years old woman, with personal history of meningitis, diagnosed with generalized tonic-clonic epilepsy at the age of 35, treated with Levetiracetam and Lamotrigine and diagnosed at the age of 39, on clinical (sensory and motor symptoms) and imagistic criteria (MRI), with relapsing remitting MS. Glatiramer acetate was added, knowing that beta interferons could induce seizures. Two relapses were treated with intravenous methyprednisolone, but every administration induced severe generalized tonic-clonic seizures, treated with emergency treatment. Headache and several hours of language disturbances (speaking in foreign languages) suggesting a temporal status epilepticus followed the seizures.

Conclusions. MS treatment should be changed, due to disease activity and methylprednisolone intolerance but in accordance with the patient's status. Teriflunomide and Cladribine were taken into account and plasma exchange for relapses. White and grey matter MS lesions can increase epileptogenesis. Language dysfunctions may occur due to demyelinating lesions, but also as epileptic ictal or postictal manifestations. Limited literature data regarding the association of epilepsy and MS, drug interactions for the two diseases and methylprednisolone intolerance need a special consideration in the treatment decision.

Rare infratentorial demyelinating lesion associated with mediastinal mass

I. IONIȚĂ[1], G. VULPE[1], A. DRUȚĂ[2], A.C. PANEA[1]

[1] *Elias University Emergency Hospital, Bucharest, Romania*
[2] *Rahova Clinic, Bucharest, Romania*

Introduction. Infratentorial lesions can have a wide range of aspects on brain imagery, often not very specific for a certain diagnosis, therefore making the case rather challenging for the medical team.

Case presentation. It is the case of a 50 year old hypertensive, obese, non-smoker female without other significant comorbidities, who presents in the neurology unit for vertigo and imbalance. Upon clinical evaluation we found a discrete left palpebral ptosis, diminished left pupillary reflex, torsional nystagmus at lateral view, axial and left arm and leg ataxia and absence of plantar reflex on the left side. The MRI shows a large, infratentorial lesion of demyelinating aspect, affecting the brainstem and the cerebellum, mostly on the left side. She under-

went a full body CT (thorax, abdomen and pelvis) to investigate a possible paraneoplastic syndrome; the result was a mass in the superior mediastinum, compressing the thyroid and the aortic arch. As for the laboratory findings, mild pleocytosis in the cerebrospinal fluid was noted and high values of angiotensin-converting enzyme. Therefore, a first question is raised – which is the aetiology of the brain mass and of the mediastinal mass? An excisional biopsy is performed in order to answer the question.

Conclusion. The case explores the presence of two different masses, a brain mass and a mediastinal mass and the possible correlation between them, considering as well the high levels of angiotensin-converting enzyme and the role it might play.

Morbid associations in a case of multiple sclerosis

Emilia FURDU LUNGUȚ[1,2],
Iustinian PORFIR FURDU[3], Silviu LUNGUȚ[4]

[1] Neurology, CF2 Hospital, Bucharest, Romania
[2] "Titu Maiorescu" University, Bucharest, Romania
[3] Radiology, CF2 Hospital, Bucharest, Romania
[4] "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Introduction. Multiple sclerosis is a chronic disease, which is characterized clinically by episodes with focal deficits of the optic nerves, brain and spinal cord, which are remeasured to varying degrees, presenting recurrences over the years and usually progressively.

Characteristic, demyelinating lesions may vary in diameter, mainly affecting the white matter of the brain and spinal cord, and do not extend beyond the entrance areas of the cranial or spinal nerves.

There are situations in which demyelinating disease is associated with other conditions, which complicates both its clinical manifestations and its evolution over time.

Case presentation. We present the case of a 43-year-old male patient with recurrent remissive MS at the age of 30, who was treated with Betaferon until 2016, when he discontinued it on his own initiative.

In 2011, he was also diagnosed with HTAE, dyslipidemia, spinal cord compression syndrome; In 2012 she suffered a lower myocardial infarction followed by triple stent coronary angioplasty.

In this context, the demyelinating disease was manifested with frequent outbreaks, then it became secondary progressive with major aggravation of neurological disorders: accentuation of the motor deficit (tetraparesis with the predominance of paraparesis), astazo-abasis, balance disorders, repeated falls, persistent dizziness.

The treatment of pathologies associated with the specific treatment of MS, has led to a stagnation of evolution.

Conclusions. Combining multiple sclerosis with other systemic disorders, complicates the clinical picture, can lead to diagnostic confusion and unexpected aggravation of symptoms.

Early diagnosis and proper treatment are the key to success in such situations.

Natalizumab-induced peripheral immune profile changes correlated with the clinical activity of the disease

Smaranda MAIER[1,2], Mihaela SIMU[3,4],
Laura BĂRCUȚEAN[1], Zoltan BAJKO[1],
Anca MOȚĂȚĂIANU[1], Adina HUȚANU[5],
Septimiu VOIDĂZAN[6], Rodica BĂLAȘA[1,2]

[1] Neurology, "G.E. Palade" UMFST, Tg. Mures, Romania
[2] Mures County Clinical Emergency Hospital, Tg. Mures, Romania
[3] "Victor Babes" University of Medicine and Pharmacy, , Timisoara, Romania
[4] Department of Neurology, "Pius Brinzeu" County Emergency Clinical Hospital, Timisoara, Romania
[5] Laboratory Medicine, CCAMF, "G.E. Palade" UMFST, Tg. Mures, Romania
[6] Epidemiology, "G.E. Palade" UMFST, Tg. Mures, Romania

Introduction. Natalizumab (NAT) was the first disease-modifying treatment used for the treatment of multiple sclerosis with a targeted mechanism of action to block the penetration of activated T-cell lymphocytes through the blood-brain barrier.

The aim of this study is to characterize the effects of NAT on the production of cytokines in the periphery and to find correlations between changes in serum levels of cytokines, induced by NAT, and disease activity.

Materials and methods. The study included 60 patients with multiple sclerosis registered in the regional centers of Timisoara and Targu Mures and a number of 33 healthy controls. Demographic and disease-related data of patients were obtained by medical history and medical records. Using the Bio-plex Pro Human Th17 Cytokine Panel kit, serum levels of 15 cytokines were assessed simultaneously at both the beginning and the end of the study.

Results. A significant decrease in IL-17, IL-33 and IL-31 serum levels was observed after approximately 6 months of NAT treatment in naïve patients. The number of relapses during the study correlated positively with the initial serum levels of IL-17 and negatively with IL-33. Patients who did not have relapses during the study had lower baseline serum levels of IL-17 compared with those with relapses. Patients in whom the EDSS score increased during the study had lower baseline serum levels of IL-33 and IL-17 compared to those in whom the EDSS score decreased.

Conclusions. The results of our study demonstrate once again that the mechanism of action of NAT is not as simple as it was created, NAT treatment causes changes in the inflammatory mechanism in the periphery. IL-17 can be used both as a biomarker of disease activity and progression in patients treated with NAT.

Neuronal surface autoantibodies anti-VGKC complex in thyroid antibody-positive and negative limbic encephalitis – case report

M.M. MANEA[1,2], G.M. MOCI[1], A.M. DOBRI[1], G.A. SÎRBU[3], L. NISTOREC[1], I.A. OLARU[1], S. TUȚĂ[1,2]

[1] *National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania*

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

[3] *Medinst Diagnostic Center, Bucharest, Romania*

Introduction. Thyroid peroxidase (TPO) antibodies present in Hashimoto's encephalopathy (HE) may be elevated in other cases of autoimmune encephalitis, often in combination with antibodies to specific antigens. A large proportion of HEs are actually encephalitis with voltage-gated potassium channel antibodies (VGKCs), targeting the extracellular domains of leucine-inactivated glioma 1 (LGI1) and contactin-associated protein-like 2 (CASPR2). Dystonic facio-brachial epileptic seizures (FBDS) reported a high incidence in encephalitis with anti-VGKCs / LGI1 antibodies.

Methods. A 71-year-old patient diagnosed 3 months prior with focal seizures presented to our clinic with increased seizure frequency. The neurologic examination revealed temporo-spatial disorientation, behavioral, mnesic symptoms and faciobrachial dystonic seizures, without other pathological findings.

Results. The cerebral MRI scan revealed a pseudofocal area with hyperintense signal in T2, FLAIR, DWI B1000 and T1 images, at the level of the right striatal nuclei, without contrast enhancement or any hemorrhagic signs on SWI). EEG showed no epileptiform changes during DFBS and CSF biochemical test was normal. The TPO antibody titer was increased (245 IU / ml) and the anti-VGKC antibodies strongly positive (897 pmol / l). Other causes of infectious / autoimmune encephalopathies / alterations in the context of nonketotic hyperglycemia were excluded. Following immunomodulatory, the evolution was favorable.

Conclusions. HE patients should be monitored for anti-VGKC antibodies in the presence of FBDS.

Causal lesions in patients with FBDS appear in the basal ganglia and do not affect the EEG pattern, suggesting that FBDS may be associated with these lesions.

Executive disfunction and its correlation with quality of life at patients with multipe sclerosis

Maria M. MARTOIU[1], Simona PETRESCU[1,2], Cristina A. PANEA[1,2]

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

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

Introduction. Motor disability, cognitive dysfunction and neuropsychiatric symptoms are common in multiple sclerosis (MS). The common cognitive symptoms include deficits in executive functioning, attention, information processing and long-term memory. These deficits affect the quality of life of the patient.

Aim. The paper aims to analyze the degree of executive disfunction and to identify the relationship between it and the quality of life for patients with multiple sclerosis.

Methods. 97 patients diagnosed with MS have been clinically evaluated using Expanded Disability Status Scale (EDSS). We applied cognitive tests: MMSE (Mini Mental State Examination), MoCA (Montreal Cognitive Assessment) and executive function test: Trail Making Test Part A and B (TMT). Quality of life was measured by the self-reporting scale: EuroQol 5-Dimension, with EQ-5D-index and EQ-Visual Analogue Scale (EQ-VAS). Statistical correlations were performed.

Results. Over 50% of patients have mild executive disfunction with score values between average and deficient on TMT Part A and B examination. Executive function test TMT Part A and B corelated statistically significant with EDSS score (p_A = 0,002 < 0.01 and p_B=0,0004 < 0,001), and self-reported scale EQ-5D correlates statistically significant with all applied cognitive scale including executive function test TMT Part A and B (p< 0,001).

Conclusions. The quality of life in patients with multiple sclerosis is greatly influenced by cognitive impairment, especially by executive disfunction. Patients with executive dysfunction were more likely to have a higher degree of physical disability.

Keywords: multiple sclerosis, executive dysfunction, quality of life

Cognitive impairment and depression in multiple sclerosis pacients undergoing Natalizumab treatment in a romanian population: The role of education, physical disability and relapse rate

A. MOLDOVAN[1], S. MAIER[1,2], A. MOTATAIANU[1,2], Z. BAJKO[1,2], L. BARCUTEAN[1,2], R. BALASA[1,2]

[1] Neurology I, Emergency Clinical County Hospital, Tg. Mures, Romania
[2] Neurology, UMFST, Tg. Mures, Romania

Background. Recently, studies have described cognitive impairment as having major effect on the disability of patients with multiple sclerosis (MS). Depression is a frequent part of the clinical presentation of patients with MS and influences their quality of life, compromising cognitive function, leading to suicide attempts and reducing treatment compliance. Monoclonal antibody Natalizumab has been shown to improve these parameters at 1 and 2 years of treatment.

Objective. The purpose of this study is to identify patients with MS who are treated with Natalizumab and have cognitive impairment and depression, to monitor the evolution of neurocognitive and depression aspects, to identify the main factors, while assessing the impact of MS on quality of life.

Material and methods. A prospective case-control study was performed aiming to analyze patients (n = 28, 8M / 20F) diagnosed with MS according to McDonald's criteria from the records of the Neurology I Department of Târgu-Mureș County Emergency Clinical Hospital who are following Natalizumab treatment. Control subjects (n = 100, 42M / 59F) were recruited to analyze cognition and depression between the two groups. EDSS (Expanded Disability Status Scale), Beck depression inventory (BDI-II) and Symbol Digit Modality Test (SDMT) were applied.

Results. We found a positive association between EDSS and BDI-II - p 0.010, as well as EDSS and SDMT - p 0.048; statistically significant correlation was found between education and BDI-II (p 0.001) and SDMT (p 0.0040); the relapse rate was not playing a significant role in cognitive deterioration or depression.

Conclusions. Cognitive impairment and depression have an increased prevalence among patients with MS. EDSS correlates with cognitive dysfunction and depression. The level of education of patients can contribute to a better cooperation and treatment compliance, thus with a favorable prognosis. Natalizumab demonstrated a decrease in annual recurrence rate our results were not statistically significant.

Multiple sclerosis Balo like form wit a good response to plasmapheresis

B. MOSCU[1], C. ROMAN-FILIP[1,2], M. HANEA[1]

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

Balo concentric sclerosis (BCS) is considered a variant of multiple sclerosis. It occurs predominantly in young adults, it is a rare and relatively acute condition. Attacks can progress quickly for weeks or months without remission, leading to death or severe disability. Most cases have a more benign, self-limiting course, with spontaneous remission. The progression of the disease is monophasic, with a rare frequency of recurrences. MRI is essential in establishing the diagnosis, it can highlight lesions in the cerebral hemispheres, optic chiasm, brainstem, cerebellum and spinal cord. Intensive immunosuppression treatment may be indicated in patients with more aggressive form.

We present the case of a 30 year old patient, who presented during the year of 2016 a right sided paresthetic syndrome and in January 2019 an episode of retrobulbar optic neuritis of the right eye.

Cerebral MRI (February 2019) highlights 2 non-specific frontal subcortical punctual lesions.

In 18.10. 2019 he has a sudden motor deficit of the right upper and lower limb.

Cerebral MRI (18.10.2019) shows a concentric active demyelinating nodular lesion in the left semioval center.

Under treatment with methylprednisolone, symptom relief was achieved.

In 15.11.2019 the patient returns, for an aggravated motor deficit at the level of the upper and lower right limb.

Cerebral MRI (15.11.2019) shows an active concentric nodular demyelinating lesion in the left semioval center, increasing in size, with oedema in the adjacent white matter.

The symptomatology improved after the administration of methylprednisolone 5 days and 5 sessions of plasmapheresis performed at 2 days interval.

The link between neuroinflammation and neurodegeneration in amyotrophic lateral sclerosis

Anca MOȚĂȚĂIANU

Neurology, „George Emil Palade" UMFST, Tg. Mures, Romania

Amyotrophic lateral sclerosis (ALS) is a neurodegenerative multifactorial disease of the central nervous system (CNS), characterized by selective destruction of motoneurons in the spinal cord, brain stem and primary motor cortex.

Neuroinflammation is now accepted as a key modulator factor of disease progression in ALS. Innate and adaptive immunity have an complex interrelationship in neurodegenerative process in ALS. As a part of innate immune system, CD4+T (TCD4+) cells under the influence of transcription factors and of cytokines differentiated into T effector cells (Teff). This Teffs cells have different immunological functions: Th1 and Th17 have an proinflammatory role, Th2 has an antiinflammatory effect, while regulatory T cells (Treg) are involved in immune suppresion. Th1 and Th17 are the most frequent TCD4+ cells infiltrating the CNS in ALS and by secreting proinflammatory cytokines influences the function of microglia and switch the phenotype and function, from an protective type (M2) to a neurodegenerative (M1). Treg are potent immune modulators wich supress the neuroinflammation by inhibiting the proliferaration of Th1 and Th17, by down-regulation of proinflammatory cytokines (IL-17, Il-6, IL-21, IL-22, TNF-α) and by secreting antiinflamatory types of cytokines (IL4, IL10, IL13).

Recent data from experiments in animal models and in ALS patients have shown that the main effectors of neuroinflammation (reactive microglial and astrocytes) actively can display, in a time dependent and context dependent manner, both neuroprotective and neurodegenerative properties, depending on the stage of disease progression and cytokine secretion. This data demonstrated the pivotal role of T cells and microglia in disease progression, mediating the neuroinflammatory processes of ALS.

Understanding of the biological processes of the neuroinflammation in ALS will increase our understanding of motoneuron degeneration and the ability to target new perspective for treatment intervention in this devastating disease.

Acknowledgment. This work was supported by funding number 293/5/14.01.2020 from University of Medicine, Pharmacy, Science and Technology George Emil Palade Tg. Mureș.

Association between systemic lupus erythematosus and neuromyelitis optica

I.M. OLARU[1], M. SPOIALĂ[1], M.L. HOHAN[1], D. ALEXA[1,3], E. REZUS[2,3]

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

[2] *Rheumatology Department, Clinical Rehabilitation Hospital, Iasi, Romania*

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

Introduction. Systemic lupus erythematosus (SLE) is an autoimmune disease that can affect any component of the central or peripheral nervous system, and its manifestations can range from headaches, mood disorders, to stroke, optic neuropathy or myelopathy. Lupus mielopathy may have as pathogenic mechanism the inflammation mediated by anti-aquaporin-4 (AQ-4) antibodies and is associated with neuromyelitis optica (NMO) or non-AQP4-mediated inflammation or vascular occlusion associated with antiphospholipid antibodies.

Case study. We present the case of a 37-year-old patient, known with SLE with neurological and immunological manifestations, who suddenly presents in December 2019 cervical pain with irradiation in the right upper limb accompanied by motor deficit and paresthesias in the lower limbs and right upper limb. The examination of cervico-dorsal MRI with contrast substance showed extensive longitudinal transverse myelitis, and biologically AQ-4 were in high titer. Subsequent development under immunosuppressive therapy was slightly favorable with amelioration of motor deficit and sensitivity disorder.

Discussion. The association between SLE and NMO may involve a common immune mechanism and an increased susceptibility to antibody-mediated autoimmune diseases. In patients diagnosed with SLE it is necessary to establish the pathogenic mechanism of myelopathy. The presence of AQ-4 implies an increased risk of relapse, and for this purpose aggressive long-term immunosuppressive therapy is used.

Conclusions. In the case of lupus myelopathy, the detection of the presence of AQ-4 antibodies requires repeated tests, especially during relapses, being absent during remission periods.

Keywords: systemic lupus erythematosus (SLE), optic neuromyelitis (NMO), anti-aquaporin-4 antibodies (AQ-4)

Morbidity and mortality risk factors in type 2 diabetes mellitus patients with acute ischemic stroke-immunological mechanisms

L. POPA[1], S. PETRESCU[1,2], C. PANEA[1,2]

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

Background and aims. Type 2 diabetes mellitus (T2DM) is a well-established risk factor for ischemic stroke. We aimed to assess the impact of T2DM on morbidity and mortality in patients with acute ischemic stroke, as well as associated factors that could lead to a bad prognosis.

Methods. We conducted a study on 194 patients with T2DM and acute ischemic stroke. The information was collected retrospectively, using patient electronic charts and analyzed using SPSS v20 – through logistic regression, Chi-square and Pearson bivariate correlation test.

We used clinical, etiological and imagistic scales for categorizing the severity and subtypes of ischemic stroke (modified Rankin Score-mRS, TOAST Classification, Oxford Stroke Classification). We used glycosylated hemoglobin (HbA1c) level, glycaemia level at admission and triglycerides level as indicators for the metabolic control of diabetes.

Results. The considered indicators of poor metabolic control - HbA1c (either for a threshold of 8%, or for 10%), triglycerides level (>200mg/dl) and glycaemia levels at admission (either for a threshold of 180 mg/dl, or for 210 mg/dl) - were not associated with a worse outcome.

We noticed a lower risk of death in men (p = 0.015, OR = 3.425, CI 95% (1.26, 9.2)). Patients older than 60yo appear to have a worse outcome (mRS > 3) (p = 0.035, OR = 3.823, CI 95%(1.1; 13.25)).

We observed an increased risk of mortality in patients that presented atrial fibrillation (p < 0.001, OR = 9.67, CI 95% (3.34;27.97)), cardioembolic stroke (p < 0.001, OR = 8.83, CI 95% (3.06;25.48)), cardiac failure (p = 0.006, OR = 4.01, CI 95% (1.5;10.73)) and healthcare associated infections (p < 0.001, OR = 11.308, CI 95% (4.16;30.69)).

Conclusions. Immune dysfunction in T2DM predisposes patients to health-care associated infections, also linked to higher morbidity and mortality. T2DM also leads to increased mortality due to chronic cardiac complications. There was no direct causal link between high level of HbA1c, glycaemia, or triglycerides at admission and mortality.

Keywords: stroke, type 2 diabetes, mortality

TULBURĂRI ALE SOMNULUI / SLEEP DISORDERS

Sleep quality in patients with minor stroke

R. GURGU[1], V. MIHAI[1], F. MUSTAFA[1], O. DRUGA[2], C. PANEA[1,3]

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

Background. Sleep disorders are frequent in patients with acute stroke and are associated with a poor outcome, on short- and medium-term. On the other hand, recent studies show that persons with a poor quality of sleep develop cardiovascular diseases (coronary ischemic disease and/or stroke) more frequently than persons with a good quality of sleep.

Objectives. Analysis of quality of sleep before the acute clinical event in acute stroke patients.

Materials and method. We identified patients discharged in March 2020 from the Neurology Department of Elias University Emergency Hospital with a diagnosis of acute stroke (ischemic/haemorrhagic) who also underwent neurocognitive testing and were also administered the self-assessment quality of sleep scale PSQI (Pittsburgh Sleep Quality Index) on admission. For all patients identified, we collected demographic data, concomitant disease data, PSQI score (total score and subcomponents scores) on admission.

Results and discussions. We identified 14 patients (9 males and 4 females), with ages between 48 and 82 years, without major cognitive impairment (MMSE >=26) and without other significant neurological disabilities who also completed the sleep quality questionnaire administered at admission. The PSQI scores (N = 14) were between 3 and 17 (mean total PSQI score 7.71). 50 percent of all patients reported a poor quality of sleep (total PSQI score >=6) in the 4 weeks preceding the acute vascular event. Patients with a poor quality of sleep were younger than patients with a good quality of sleep (mean age 62 years old vs. 68.57 years old) and were more frequently associated with atherothrombotic is-

chemic stroke. There were no significant differences between the two groups regarding type or location of acute stroke or regarding the presence of other vascular risk factors.

Conclusions. The proportion of patients with acute stroke in this small case series that report poor quality of sleep before the vascular event is similar to the proportion of patients presented in literature that report sleep disorders after the occurrence of acute stroke. Sleep disorders could represent risk factors for acute stroke, and not just complications.

Keywords: quality of sleep, stroke, vascular risk factors

VARIA

Therapeutic plasma exchange in acute motor axonal neuropathy (AMAN) – case report

Sebastian ANDONE[1], Adina STOIAN[1,2], Anca MOȚĂȚĂIANU[1,3], Zoltan BAJKO[1,3], Smaranda MAIER[1,3], Laura BĂRCUȚEAN[3], Rodica BĂLAȘA[1,3]

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

Guillain-Barré syndrome (GBS) is an acute disease with immune-mediated mechanisms and represents a neurological emergency. Acute motor axonal neuropathy (AMAN) is a rare GBS subtype with poor outcome and is treated with intravenous immunoglobulins and therapeutic plasma exchange (TPE).

We present the case of a 27-year-old male patient that had a mild upper respiratory tract infection 1 week before the onset of the neurological symptoms. The onset of the disease was 2 days prior to his admission, the patient developing paresthesia in his lower, with ascending character towards his upper limbs, followed by progressive motor deficit. At admission, the patient had already a moderate respiratory dysfunction. The neurological exam at admission showed grade 2/5 MRC tetraparesis, abolished deep tendon reflexes, without any pyramidal signs, and later developed urinary retention.

Nerve conduction studies were performed showing low amplitude of compound muscle action potential (CMAP) which is characteristic for axonal loss.

We performed a total of 4 TPE sessions, being able to replace 4 plasma volumes. Between sessions, a number of seric parameters were followed: calcium, magnesium, fibrinogen, total proteins, potassium, platelets, hematocrit. In our case, there were no significant changes.

The neurological exam after the last TPE session, revealed motor deficit improvement up to grade 4/5 tetraparesis, no respiratory symptoms, no urinary retention.

Conclusions. TPE can be considered as a first-line treatment for AMAN. Performing TPE as soon as possible can save further axonal damage and increases the chances for a good outcome, but it requires specific equipment and trained staff.

Probable early-onset Alzheimer's disease – case presentation

Ramona-Andreea ANDREI[1], Raluca Andreea MOLDOVAN[1], Cristina-Aura PANEA[1,2]

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

Introduction. Alzheimer's disease is a neurocognitive disorder with insidious onset and gradual progression of impairment in one or more cognitive domains, without extended plateaus, excluding other etiologies. Onset age is usually after the age of 65 years old, but some patients may preset with early-onset forms in the fourth or fifth decade of life.

Case presentation. We present a 52-years old, male patient without a significant medical family history, occasional ethanol consumption in moderate quantities, with cardiovascular risk factors, with memory and behavioral impairment. The symptoms were noticed by the patient and the family 5 months ago, and had a progressive evolution since.

Management and results. The clinical, neurological and psychological evaluation showed that the patient had partial temporal-spatial disorientation, a depressive mood, a diminished spontaneous speech with difficulty in finding words and names, attention deficits, difficulty in retaining new information and processing mathematical calculation, and biography errors. Cognitive tests: letter verbal fluency = 2 words/minute; semantic group verbal fluency = 3 words/minute; MMSE= 18/30 points, Clock test = 5/10 points.

Lab results showed elevated levels of glycemia, mixed dyslipidemia, while the cerebrospinal fluid analysis showed low levels of β-amyloid 42, β-amyloid 42/β-amyloid 40 < 0.1 and intermediate levels of tau protein.

Brain tomography and magnetic resonance showed a few small vascular-degenerative lesions and uncharacteristic diffuse cerebral and cerebellum atrophy.

Conclusions. From the patient history, clinical examination, paraclinical and imagistic investigation, we conclude the diagnosis of a major neurocognitive disorder of neurodegenerative etiology, probable Alzheimer's disease. The case's particularity is the early-onset of the disease without a family history of neurocognitive disorders.

Keywords: Alzheimer's disease, young onset

Case series depicting carotid artery dissection – outcomes, particularities

A. BĂNICĂ[1], S. PETRESCU[1,2], C. PANEA[1,2]

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

Introduction. Carotid artery dissection is a significant cause of ischemic stroke in young and middle-aged patients. It is generally considered to be provoked by minor trauma or to occur spontaneously, most likely due to genetic disorders.

Objectives, method and results. We studied the cases of 18 patients diagnosed with carotid artery dissection in our clinic over a 5-year period, presenting with various underlying or associated conditions.

Men were twice more affected than women, with a similar mean age, most patients not undergoing previous treatment.

NIHS score upon admission was under 8 points for 6 patients, whereas 7 patients scored over 16 points. Mean hospitalization duration was 13 days, only 7 patients needing under 7 days of medical care.

Initial imaging studies consisted of Doppler examination of the cervical and cerebral arteries (raising diagnostic suspicion), completed by cerebral angiography in 7 patients or CTA (9 patients)/MRA (2 patients).

Regarding therapy, 2 patients received intravenous thrombolysis with rtPA; all patients received aspirin, 3 of which also received clopidogrel, while for 10 patients low molecular weight heparin was associated, and other 3 patients also received unfractionated heparin.

There were 4 deaths, mean Rankin score for the rest of the discharged patients was 2,8, while 8 patients remained with moderate or severe disability (mRS > 2).

We noticed underlying or associated conditions in 11 patients (cardiovascular, haematological, respiratory systems), and for 1 case we recorded minor trauma as trigger factor (parachute jump).

Conclusion. Carotid artery dissection, generally considered to have a benign clinical outcome, may prove itself a severely debilitating condition for young patients, while we are still figuring out the complexity of predisposing factors.

Keywords: stroke, carotid artery dissection

Infective endocarditis and stroke – case series

A. BĂNICĂ[1], S. PETRESCU[1,3], A. RACHIERU[2], A. POPESCU[2,3], C. PANEA[1,3]

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

Background. Infective endocarditis is a disorder of the cardiac valves with systemic repercussions and which mimics multiple pathologies, creating diagnostic difficulties. The neurologic complications are the most frequent extracardiac manifestation of the disease, with the highest mortality rate.

Objective. Upon facing a clinical stroke-like onset, brain imaging raises the suspicion of possible cardioembolic etiology. Specialized investigations confirm the primary diagnosis of left-sided infective endocarditis, with cerebral embolization.

Method and results. We studied the cases of 9 patients diagnosed with infective endocarditis in our clinic during 2016-2019. Mean age of the patients was over 80 years, 6 of them were previously independent or had slight disability and most patients had significant valvulopathies or valvular prosthesis of the left heart.

Clinical examination upon admission revealed altered state of consciousness or focal neurologic deficits, with a NIHS score > 16 for 6 patients. Cerebral CT scans showed multiple ischemic strokes in different vascular territories, some with haemorrhagic transformation, and in some cases, concomitant multifocal haemorrhagic lesions, raising suspicion of infective endocarditis.

Progressive deterioration of clinical and biologic state (obtundation, progression of neurologic deficits,

fever, inflammatory syndrome) requires cardiologic evaluation with transthoracic echocardiography, completed by transesophageal echocardiography for 4 patients, which visualises vegetations attached to the mitral and/or aortic valve.

Blood culture samples confirm the infection with *Staphylococcus aureus* in 5 cases. Despite adequate antibiotic treatment, clinical evolution is fatal, resulting in 8 deaths (3 of which occurred within 7 days of admission).

Conclusions. *Staphylococcus aureus* infective endocarditis associating neurologic complications has the highest mortality rate. However, prompt adequate antibiotic therapy is frequently delayed faced with the neurologic diagnostic trap.

Keywords: endocarditis, stroke, mortality

Diagnostic problems in inflammatory myopathies

Lenuța BÎRSANU, Raluca ASAFTEI,
Dan Iulian CUCIUREANU

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

Inflammatory myopathies are a group of conditions characterized by muscle weakness, elevated CK levels, and inflammatory infiltrate on muscle biopsy.

Inflammatory myopathies can be classified into 4 groups: idiopathic myopathies- whose aetiology is unknown; myositis associated with connective tissue disease (CTD) and autoimune disease; myositis associated with neoplasm; myositis in infections. The group of idiopathic inflammatory myopathies includes: dermatomyositis, polymyositis, myositis with inclusion bodies and necrotizing autoimmune myopathy.

Overlap syndrome refers to dermatomyositis, polymyositis and necrotizing myopathy in association with other connective tissue disorders: systemic lupus erythematosus, sleroderma, rheumatoid arthritis and Sjogren syndrome.

The annual incidence of inflammatory myopathies is about 1:100,000.

In the following we discuss two clinical cases of patients with polymyositis and myositis with inclusion bodies.

Keywords: myopathies, inflamation

Posterior reversible encefalopathy syndrome: A neurological and radiological challenge

A.M. BORUZ[1], A. VELICU[2], S. DUMITRACHE[1]

[1] National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania
[2] University Emergency Hospital, Bucharest, Romania·

Introduction. Posterior reversible encefalopathy syndrome (PRES) is a neurological condition caused by neurotoxic status secundary to the dysfunction of posterior self-regulation in response to high blood pressure (BP). Typically PRES is caused by damage to the blood-brain barrier by hyperperfusion causing vasogenic edema most commonly located in posterior territories (parieto-occipital), atypically these lesions can occur in the basal ganglia, frontal lobe brainstem and cerebellum.

Case presentation. We present an atypical case of PRES of a 40-year-old male, known with severe kidney disease and hypertension, who presents for intense headache, neck stiffness and emetic syndrome, BP = 240 / 120 mmHg and normal neurological examination. Biologically there is uremic syndrome (creatinine 6 mg/dl and urea 104 mg/dl), without large variations of electrolytes. Initial imaging: head CT reveals ponto-mesencephalic and left cerebellar hypoattenuation as well as cerebellar cortical hemoragic foci raising the suspicion of stroke in the vertebro-basilar territory, but a possible PRES, vasculitis, hypoglycemic crisis, cerebral gliomatosis, hypoxic-ischemic encephalopathy or cerebral venous thrombosis can not be excluded.

It is decided to perform a rapid head MRI-DWI sequence that excludes ischemic lesions and after the rest of the MRI sequences plus angio-MRI that reveals confluent signal changes of supra / infratentorial with confluent appearance, without diffusion restriction, in the context of higher blood pressure values at the time of presentation we raise the suspicion of PRES. Given the creatinine values, no contrast could be administrated.

Conclusions. Corroborating imaging, clinical and severe hypertension its raises the suspicion of PRES confirmed by clinical improvement after administration of antihypertensives and control MRI performed at one month.

A pathology that should not be overlooked in patients with subarachnoid haemorrhage – spontaneous intracranial carotid artery dissection

I-F. BRATU[1], A.C. RIBIGAN[1], R.S. BADEA[1], C. LAZA[1], O. RUSU[1], B. DOROBAT[2], A. DIMITRIADE[2], A. CARP[2], O.A. BAJENARU[1], F.A. ANTOCHI[1]

[1] Department of Neurology, University Emergency Hospital, Bucharest, Romania
[2] Department of Interventional Neuroradiology, University Emergency Hospital, Bucharest, Romania

Introduction. Intracranial carotid artery dissection (ICAD) represents an uncommon cause of subarachnoid haemorrhage (SAH), the mortality in patients with this association being approximately 50%. This case was chosen to be reported as to its particularities of presentation.

Case presentation. We present the case of a 66-year-old female with medical history of arterial hypertension and right lenticular haemorrhagic stroke a few years back, who was transferred to the Neurology Department for subsequent management of a recent SAH (Hunt-Hess grade 2). The patient had been admitted to our hospital for intense occipital headache, dizziness, emesis and sleepiness of abrupt onset. The neurosurgical examination established the presence of nuchal rigidity and right oculomotor nerve palsy. A cerebral computed tomography (CCT) scan was performed and it revealed the presence of a peribulbar, prepontine, interpeduncular cisterns as well as forth and lateral ventricles hyperdensity (suggestive for SAH), diffuse cerebral oedema and hydrocephalus. The 4-vessel digital subtraction angiography excluded a vascular malformation or an aneurysm and showed complete occlusion of the left common carotid artery and multiple hemodynamically significant stenoses (including of the contralateral internal carotid artery (ICA)). The patient received symptomatic treatment and was transferred to our department. Cerebral and cervical region MRI scan was performed and revealed a right intracranial ICA dissection. Successive CCT scans established the resorption of the SAH with the CCT scan taken at one month showing the absence of the SAH. The patient received antiplatelet therapy.

Conclusions. This case report pinpoints the importance of taking into consideration ICAD as a potential cause of SAH.

Personalized treatment of the patient with ischemic stroke and severe systemic atherosclerosis

Constantin BUNEA, O. IOGHEN, D. DUMITRASCU, F.A. ANTOCHI

Department of Neurology, University Emergency Hospital, Bucharest, Romania

Introduction. Personalized treatment of secondary prevention of ischemic stroke by revascularization procedures (carotid endarterectomy / percutaneous angioplasty with carotid stent) is indicated for symptomatic segmental stenosis, hemodynamically significant (> 70%) located at the carotid level in the extracranial segment. Patients candidates for revascularization therapy should be evaluated to detect other sites of systemic atherosclerosis and associated cardiovascular diseases in order to prevent peri- or post-procedural complications.

Case presentation. We present the case of a 75-year old man, smoker (50 packs of cigarettes), with stage II of hypertension , with non-insulin-dependent diabetes mellitus, who presented an ischemic stroke in the territory of the left middle cerebral artery. Doppler ultrasound evaluation of cervical vessels showed moderate segmental stenosis (~ 60%) in left ICA (clinically symptomatic) and severe segmental stenosis (~ 90%) in right ICA (clinically asymptomatic).

The patient performed cerebral angiography "4 vessels", which showed a segmental stenosis of ~ 50% in the left ICA origin, as well as a contralateral stenosis of 90% in the extracranial segment. Regarding the presence of cardiovascular risk factors, a coronary angiography was requested, although there are no clinical symptoms of affecting the coronary territory. This highlighted the occlusion of the right coronary artery and the subocclusive stenosis of the circumflex artery (ACx), for which it was decided to mount 2 pharmacologically active DES stents. After 6 months, the patient was reevaluated neurologically and the stationary appearance of the degree of bilateral carotid stenosis was underlined, as well as the stable appearance of atheroma plaques, with the absence of microembolism in the cerebral circulation when monitoring TCD. It was decided to continue the maximal drug therapy and periodic neurological and imaging reassessment.

Conclusions. The case presented above shows the importance of performing coronary angiography before taking a personalized decision of secondary prevention of ischemic stroke in patients with severe systemic ath-

erosclerosis. Another particularity of the case is that the patient suffered an ischemic stroke in the distribution territory of the left ICA, which had a moderate carotid segmental stenosis extra-cranial and in the asymptomatic territory had a severe carotid segmental stenosis, for which it will be considered personalized revascularization decision as primary prevention.

A case report of Alzheimer disease associated with cerebral amyloid angiopathy

Dr. Bogdan CĂȘARU, Dr. Raluca BADEA, Dr. Florina ANTOCHI

Department of Neurology, University Emergency Hospital, Bucharest, Romania

The accumulation of beta amyloid peptide within the blood vessels of the brain, which is the hallmark of the cerebral amyloid angiopathy, can occur as a sporadic event or it can be associated with Alzheimer disease. However, even in the sporadic cases the vascular amyloid deposits are biochemically similar to the senile plaques described in Alzheimer disease. While most of the patients remain asymptomatic, this pathological process has been recognized to increase the risk of intracerebral haemorrhage.

Hereby we would like to present the case of a 70-year-old man with a medical history of arterial hypertension, who was prompted to have a cerebral magnetic resonance imaging (MRI) after complaining of memory problems, especially short-term memory deficits. Given the fact that the MRI result described multiple areas of lobar microbleeds and cortico-meningeal hemosiderin deposits, the suspicion of Alzheimer's disease was raised. This was confirmed by the lumbar puncture which revealed low beta42-amyloid level with high tau and phosphorylated-tau protein level in the cerebro-spinal fluid.

Incidental findings of microbleeds and hemosiderosis on MRI suggestive of cerebral amyloid angiopathy should prompt us to look further for the presence of cognitive impairment and eventually for the diagnosis of Alzheimer disease.

Posttraumatic right sided direct carotid-cavernous fistula with bilateral carotid artery dissection – case report

A. CIOBAN[1], A. MOȚĂȚĂIANU[1], Z. BAJKO[1,2], L. MĂRGINEAN[3], R. BĂLAȘA[1,2]

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

[2] Neurology Department, "G.E. Palade" University of Medicine, Pharmacy, Science and Technology, Targu-Mures, Romania

[3] Interventional Radiology Department, County Emergency Clinical Hospital, Targu-Mures, Romania

Carotid artery dissection may arise as a complication of blunt head or cervical trauma, especially after car accidents. High flow carotid cavernous fistula is a direct connection between the cavernous sinus and the intracavernous carotid artery and occur most often after a traumatic injury.

We report the case of a 21-year-old male who suffered a polytrauma caused by a car accident. The patient sustained a bilateral hip dislocation, a trochanteric femoral right fracture and a right knee injury. He was admitted in the Orthopedic Clinic for specialized treatment and after four days he presented bruit, headache, diplopia, pulsatile proptosis and chemosis of the right eye associated with ocular pain. Neurologic examination showed abducens nerve palsy. The cerebral angiography demonstrated a right sided direct carotid-cavernous fistula with venous drainage through ipsilateral superior ophthalmic vein, facial vein, superior and inferior petrosal sinus with a complete flow steal from the right internal carotid artery. Furthermore, the cerebral angiography revealed the dissection of both carotid arteries with the vascular supply to the right cerebral hemisphere from the left carotid artery through anterior communicating artery.

The aim of the present report is to communicate this rare posttraumatic association between direct carotid cavernous fistula and bilateral carotid artery dissection and to examine the proper management and the suitable treatments.

The pharyngeal-cervical-brachial variant (PCB) of Guillain-Barré syndrome (GBS) associated to a cytomegalovirus infection in an immunocompetent patient – case report

R. CIOCAN, O.C. DAN

Neurology Department, Nord Franche-Comté Hospital, France

Introduction. The pharyngeal-cervical-brachial (PCB) variant of Guillain-Barré syndrome is defined by rapidly progressive oropharyngeal and cervicobrachial weakness associated with areflexia in the upper limbs. The neurologic signs are preceded by signs of upper respiratory tract infection in 56-76% of the patients. The most common pathogens are *Campylobacter jejuni, Haemophilus influenzae, Mycoplasma pneumoniae* and cytomegalovirus. The onset is typically acute, beginning with neurologic symptoms approximately 8–10 days following the antecedent illness. The disease then progresses until a clinical nadir is reached approximately a week after the initial neurologic symptoms. Serum antiganglioside antibodies are positive in 85% of patients.

Case report. A 56 years old woman with a history of polytrauma was hospitalized for cervicalgia followed by dysphagia to both solids and liquids associated with progressive weakness first of the neck and then in the upper limbs with numbness in the fingers and then hands. A progressive deterioration occurs in the patient's neurological status: worsening dysphagia with dysphonia and dysarthria, bilateral facial nerve palsy, numbness in the face, weakness in the upper limb. Three weeks before admission, she presented an episode of viral rhinopharyngitis. At admission, neurological examination showed bilateral facial nerve palsy, bilateral hypoesthesia and numbness in the maxillary and mandibular divisions of the trigeminal nerve, severe dysphagia with left absent pharyngeal reflex, dysphonia, left tongue weakness, loss of deep tendon reflexes in lower limbs, mild proximal limb muscles fatigability and bilateral paresthesias of both hands. The patient need to have a nasogastric intubation pour feeding.

Complementary tests highlighted albumino-cytological dissociation in the cerebrospinal fluid with no detected PCR DNA for CMV, Epstein-Bar and herpes virus. Neuroimaging tests (brain, cervical and thoracolumbar MRI scans) showed no morphological changes or signal alterations in the brain, brainstem, spinal cord or cauda equina in T-1, T-2 or FLAIR weighted sequences. The electromyogram of the upper limb (deltoid, biceps and extensor digitorum muscles) revealed a neurogenic pattern. The seroimmunological study of antiglycolipid antibodies conducted by enzyme immunoassay (ELISA) were negative (anti-GT1a, GT1b, GQ1b, GD1a, GD1b, GD2, GD3, GM1, GM2, GM3, GM4 and anti-sulfatides antibodies), also the serologic tests (HVB, HVC, HIV, syphilis, Lyme, Campylobacter, Mycoplasma), excepting CMV, with both IgG and IgM elevated. Our conclusion was a seronegative PCB following a CMV infection.

Neurological improvement was observed after intravenous gamma globulin and follow-up examinations showed a continuous clinical amelioration with no more need of the nasogastric intubation folowed by normal refeeding.

Conclusions. When one sees acute onset bulbar palsy and limb muscle weakness, the possibility of PCB should be considered, thus compelling the need for serum anti-ganglioside antibody measurement. CMV can also induce the production of anti-GT1a antibody, thereby resulting in PCB. This case suggests the need for all ganglioside antibodies to be tested in suspected variant Guillain-Barré syndromes.

Keywords: Guillain-Barré syndrome, cytomegalovirus, anti-ganglioside antibody

Therapeutic challenge in hyperacute simultaneous cardiocerebral infarction – case report

R. CIOCAN, O.C. DAN

Neurology Department, Nord Franche-Comté Hospital, France

Introduction. Both acute ischemic stroke and acute myocardial infarction are medical emergency conditions, which require timely diagnosis and management. Simultaneous acute ischemic stroke and acute myocardial infarction is described as "hyperacute simultaneous cardiocerebral infarction" and concerns patients with simultaneous cardiocerebral infarction who arrived at the hospital within 4.5 h of the thrombolytic therapeutic window.

Case report. We present a case of a 80-year-old woman, who suffered from an acute ischaemic stroke (AIS) and ST-elevation myocardial infarction (STEMI) concurrently. She was admitted at emergency department due to acute onset of right-sided body weakness with slurred speech and clinical findings suggestive of a stroke with National Institute of Health Stroke Scale (NIHSS) of 11. MRI revealed acute ischaemic stroke in the left middle cerebral artery (MCA) territory with

proximal thrombus in M1 segment and ECG showed atrial fibrillation and acute inferolateral ST-elevation. The first decision was to proceed with thrombolysis. Intravenous recombinant tissue plasminogen activator (rtPA) was administered with standard dose of 0.9 mg/kg at 2 h 50 min after onset with the improvement in NIHSS score to 5 after 1 hour post-thrombolysis. A second ECG made after the thrombolysis and the kinetics of cardiac troponins were indicating one more time the acute STEMI. Upon multidisciplinary collegial discussion (neurologists, cardiologists, neuroradiologists) and due to the occlusion at proximal M1 segment of the left-middle cerebral artery we decided to perform mechanical thrombectomy after intravenous thrombolytic. In second time, the emergency coronary angiography was performed and revealed acute occlusion at bifurcation of the circumflex artery and the first lateral artery. Percutaneous coronary intervention (PCI) with drug eluting stent placement was performed. The patient's neurological evolution was favorable with the progressive regression of the deficit, NIHSS to 1. The CT-scan 24 hours later was not complicated by hemorrhagic transformation.

Conclusions. Due to the rarity of the condition, the management of these patients with a concurrent AIS and STEMI is very challenging and there is no ideal recommendation. Decision to treat AIS should be individualized based on the risk and benefit of the treatment. Balanced management should be a trade-off between early rescuing the brain or the heart. Mechanical thrombectomy should be performed as soon as possible after symptom onset. High age alone should not be considered as a contraindication for mechanical thrombectomy. Re-organization of stroke care systems is needed to provide rapid access to endovascular therapy equitably to all eligible patients.

Keywords: hyperacute simultaneous cardiocerebral infarction, stroke, STEMI, thrombolysis, thrombectomy

POEMS syndrome – a challenging diagnosis

D.I. CUCIUREANU[1,2], L. CUCOS[2], V. CONSTANTINESCU[1]

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

Background. POEMS represents a rare paraneoplastic syndrome caused by a clone of aberrant plasma cells. It is the acronym for polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy and skin changes. Both clinical suspicion and diagnosis of POEMS syndrome can be challenging.

Case presentation. We report on a patient presenting with a neuropathic syndrome and generalized adenopathy secondary to monoclonal gammopathy and also outline considerations that can impact diagnostic decision-making. Clinical examination, serum protein electrophoresis followed by bone-marrow aspiration and PET-CT were performed.

A 64 years old male patient with a positive history of progressive, generalized adenopathy for one year was addressed to our center for lower limb paresthesia. He was diagnosed with monoclonal gammopathy, light chain Kappa subtype. He developed sclerotic bone lesions and enlarged liver and spleen. He associated capillary permeabilization syndrome with generalized edema and pleurisy. The endocrine evaluation revealed hypothyroidism. Moreover, the patient associated sclerodermic-like skin changes. POEMS syndrome is therefore established and the characteristics of paraproteinemic neuropathy are discussed.

Conclusions. Failure to recognize the patient's signs and symptoms as attributable to POEMS syndrome is unsurprising given the rarity of the entity. Furthermore, it can also mimic other disorders. Without treatment progress is rapid, mandating early diagnosis.

Keywords: POEMS syndrome, polyneuropathy, monoclonal gammopathy, organomegaly, endocrinopathy

Stroke and hypercortisolism – a bidirectional relationship

V. CONSTANTINESCU, G. CONSTANTINESCU, D.I. CUCIUREANU

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

Background. Neurovascular events may trigger hypothalamic-pituitary-adrenal axis activation determining an adrenal glucocorticoid stress response. Post-stroke cortisol concentration may reflect the severity as well as predict outcomes. Prolonged exposure to cortisol can also lead to several metabolic complications such as diabetes or hypertension, raising the risk of cardiovascular events. Cyclic hypercortisolism is a rare disorder characterized by episodes of cortisol excess, followed by periods of normal secretion. It can occur regularly or irregularly with intercyclic phases ranging from days to years.

Case presentation. A 52-year old female patient was referred to our clinic for neurological evaluation after right middle cerebral artery ischemic stroke. For about 20 years she was diagnosed with cyclic Cushing. She had developed secondary hypertension, diabetes mellitus, depression and osteoporosis. A postero-lateral pituitary microadenoma was identified but technical difficulties made surgery impossible. Left adrenalectomy was performed for a macronodular aspect at the age of 39. Prior hospitalization to our center, she presented a myocardial infarction, ventricular fibrillation and a concomitant ischemic stroke. She did not present any clinical signs typical of Cushing's with the exception of distal muscular atrophy. Biologically, there was mild hypercortisolism.

Conclusion. Excessive cortisol can be deleterious to cerebrovascular structures, not only inducing acute vascular events but also complicating the outcome. Several studies identified elevated cortisol levels post-stroke.

Keywords: ischemic stroke, hypercortisolism, diabetes, hypertension

Repetitive vertebro-basilar transient ischemic attacks secondary to giant cell arteritis – Case report

R. COTOI[1], Z. BAJKO[1,2], S. MAIER[1,2], A. MOȚĂȚĂIANU[1,2], A. STOIAN[1,2], L. BĂRCUȚEAN[1,2], R. BĂLAȘA[1,2]

[1] Emergency Clinical County Hospital, Targu Mures, Romania
[2] "G.E. Palade" University of Medicine Pharmacy Science and Technology, Targu Mures, Romania

Introduction. Giant cell arteritis (GCA) is a chronic inflammation of the medium and large vessels, which can lead to multiple complications. The main symptoms are: headache, visual disturbances, laterocervical and facial pain.

Objective. The aim of this paper is to emphasize how important is a correct diagnosis in a patient with craniocervical algic symptoms.

Case report. We present a case of a 74-years old male,hypertensive, diabetic, with occipital headache and right shoulder pain which radiates to the laterocervical area. At the consultations made prior to hospitalization, the symptoms were interpreted in the context of a cervical spinal syndrome, migraine, vertebrobasilar insufficiency syndrome. After six months, the symptoms got worse despite antalgic treatment and the patient experienced brief episodes of vertigo, nausea and balance disturbances.

Clinically, the temporal superficial arteries are thicker, with no pulse. On neurologic examination, the patient have: bilateral neurosensory hearing loss, no deep tendon reflexes of the lower limbs, Babinski sign present bilateral. Cranial computed tomography is negative. The inflammation markers are positive (reactive protein C, erythrocyte sedimentation rate 46 mm/h, fibrinogen 460 mg/dl). Head, neck and transcranial Doppler ultrasound findings are: 20-25% right carotid artery stenosis, occlusion of the left vertebral artery, hypointense thickening of the right vertebral artery wall, superficial temporal arteries with compression sign positive, significant segmental stenosis, hypoechoic halo sign suggestive for arteritis. The symptoms improved after therapy with methylprednisolone.

The patient refused temporal artery biopsy, but the diagnosis is sustained by clinical and ultrasound findings, the inflammatory syndrome and therapeutic response.

Conclusions. GCA, misdiagnosed or untreated, can lead to serious complications like vertebrobasilar transient ischemic attacks even in patients with no visual disturbances.

Subacute axonal polyneuropathy after bariatric surgery

I.A. DRĂGHICI[1], A. ALEXANDRESCU[1], A. MOȚĂȚĂIANU[1,2], Z. BAJKO[1,2], S. MAIER[1,2], A. STOIAN[1,2], L. BĂRCUȚEAN[2], R. BĂLAȘA[1,2]

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

Introduction. Nutrition-related neuropathies are defined by a progressive loss of function in the peripheral nerves, that result from either vitamin or mineral deficiency, related to acquired factors such as malabsorption due to bariatric surgery. The most common type of pathological reaction is a length-dependent axonal degeneration. The aim of this presentation is to highlight the major neurological complications that might occur after bariatric surgery.

Case presentation. We present the case of a 33-year-old female, who underwent gastric sleeve surgery in October 2019 with favorable postoperative evolution and a total weight loss of 30 kilograms. The onset of symptoms occurred two months after surgery, the patient describing a slow progressive distal upper and lower limb muscle weakness, accompanied by paresthesia.

It is important to mention that the substitutive vitamin B complex treatment has been initiated after the onset of symptoms and before she was admitted into our clinic The most suggestive laboratory findings are: vitamin B1 86.3 µg/l (28-85), vitamin B6 97.83 µg/l (8.7-27.2), vitamin B9 2.12 ng/ml (3-17), 25-hidroxi-vitamin D 6.7 µg/l (30-100), seric copper, selenium and vitamin B12-normal levels. Lumbar puncture was perfomed describing mild protein elevation 455.7 mg/l (155-400) and normal level of glucose in CSF. MRI of cervical spine described no abnormalities. The electroneurography/electromyography revealed the diagnosis: subacute axonal polyneuropathy.

Conclusions. We concluded that this is a case of nutritional subacute axonal polyneuropathy occured as a complication of the bariatric surgery the patient suffered. We observed serum deficite of vitamin B9, coupled with the suspicion of a serum deficite of the B12 vitamin. The latter was addresed with a tardy administration of vitamin B12 – all before getting admitted into our clinic.

Keywords: bariatric surgery, subacute axonal polyneuropathy, vitamins

The challenging diagnosis of a tumefactive brainstem lesion – a case report

Diana DUMITRASCU[1], R. BADEA[1], A. RIBIGAN[1], F. ANTOCHI[1], O. BAJENARU[1,2]

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

Background. Tumefactive multiple sclerosis is a rare form of multiple sclerosis. This type of multiple sclerosis with brainstem involvement is sometimes indistinguishable from neuro-Behçet disease, glial neoplasm, lymphoma or abcesses.

Case presentation. We report on a 24-year-old male who was admitted for difficulty in swallowing liquids, blurred vision in the right visual field, loss of coordination and involuntary movements affecting the left arm and leg, symptoms that progressed during a 4 year period as repeated episodes with complete or partial remission, that lasted for one to 3-4 days. Clinical examination revealed multiple brainstem signs. The brain MRI exam performed revealed numerous T2-FLAIR hyperintense and T1 hipointense disseminated supratentorial white matter lesions and a 2.3 cm round shaped hyperintense T2-FLAIR, DWI, Gd+ brainstem lesion with perilesional edema involving the left side of the pons and the midbrain. The cervical medular MRI showed a hyperintense STIR C3 posterior columns lesion. The laboratory tests indicated positive HLA B51 and also positive cerebrospinal fluid oligoclonal bands. The diagnoses of multiple sclerosis, neuro-Behçet disease and glial neoplasm were taken into consideration.

Conclusions. Although cases of tumefactive multiple sclerosis with brainstem involvement are rare, they pose a challenge in term of diagnosis and treatment. This statement is also valid in our case as, for the time being, further investigations (3T MRI exam, proton MR spectroscopy, rheumathology evaluation) are needed to establish an accurate overview of the pacient's state and a correct subsequent therapeutical approach.

Clinical implications of anatomical variations of circle of Willis identified in an autopsy study

A.M. DUMITRESCU[1], D.I. CUCIUREANU[2], A. SAVA[1]

[1] Department of Anatomy and Embriology, "Gr.T. Popa" University of Medicine and Pharmacy, Iasi, Romania
[2] Department of Neurology, "Gr.T. Popa" University of Medicine and Pharmacy, Iasi, Romania

Introduction. Autopsy or imaging studies that have been conducted so far showed that a normal circle of Willis (CW) occurs in less than half of the population. Our aim was to identify the clinical importance of the anatomical variations of CW in certain forms of cerebro-vascular diseases.

Materials and methods. The study analyses all the autopsies that have been performed over a period of 30 months, in "Prof. Dr. N. Oblu" Emergency Clinical Hospital, Iași, Romania. The demographic data of the deceased, the macroscopic appearance of the CW and the causes of death were taken from the Necropsy Register of the Laboratory of Pathology of the same hospital.

Results. Out of 96 available human circles of Willis, 28 cases (29.17%) were classified as „anatomical variants". The average age of the group was 61.78 years. The Male:Female ratio was 1.1. 60.71% of patients with anatomical variants of CW had cerebro-vascular disease as the cause of death, among which 32.14% died from ischemic stroke and 21.42% from hemorrhagic strokes. In 10.71% of all performed autopsies, the cause of death was an aneurysm of the anterior communicating artery. 39.29% of autopsies revealed some other causes of death: acute myocardial infarction (17.85%), pneumonia with pleurisy (7.14%), multiple vertebral and liver metastases from lung cancer (7.14%), metabolic encephalopathy (3.57%) and tuberculous meningitis with intracerebral tuberculomas (3.57%).

Conclusions. The anatomical variants of CW may be a risk factor for stroke, especially the ischemic ones, in cases with comorbidities (hypertension, cerebral atherosclerosis or diabetes).

A case of late-onset chorea

C. DUMITRESCU, O. ARAMA, N. IONITA, G. MIHAILESCU, S.M. NICA

Colentina Clinical Hospital, Bucharest, Romania

Introduction. Chorea is an abnormal involuntary movement that is characterized by brief, abrupt, irregular, unpredictable, non-stereotyped movements. There are numerous causes of late-onset chorea, including drugs, medications, cerebrovascular disease, genetic and sporadic neurodegenerations, immune disorders and metabolic disorders.

Case report. We report the case of a 75-year old woman who was admitted to our clinic for generalised involuntary movements, balance problems and reccurent falls. About 2 years ago, she first noticed the involuntary movements that aggravated progressively. She had no family history of movement disorders.

Neurological examination showed generalised chorea, with orofacial involvement and asymmetric involvement of the limbs.

Laboratory evaluation, including complete blood cell count, peripheral blood smear, routine chemistry, thyroid and parathyroid function, coagulation profile, tumor markers, autoimmune studies, was normal. A brain MRI was performed and showed only diffuse brain atrophy.

Genetic testing for HD revealed 35+/-1 CAG repeats on one allele and 15+/-1 repeats on the other.

Discussion. Late onset HD patients present more frequently with gait and balance problems and with no family history of movement disorders. Repeat size is significantly lower in the late-onset compared to common-onset.

Camptocormia – a challenging diagnosis

G. FRUNZĂ, M. BACIU, D.F. MUREȘANU

Department of Clinical Neurosciences, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

County Emergency Hospital, Cluj- Napoca, Romania

Introduction. Camptocormia may be present in a variety of pathologies but when there are insufficient resources to exclude all the rare causes, we are faced with a wide range of diagnostic possibilities.

Case presentation. A 60 year old patient with mild hypertension was admitted in the Neurology Department at the County Emergency Hospital in Cluj-Napoca accusing sudden onset lower back pain after physical exertion. The neurological examination revealed a painful trunk anteflexion and a right extrapyramidal syndrome. He received treatment with a dopaminergic agonist and had a favorable evolution. However after a short while the parameters changed. The patient was no longer able to suppress the trunk anteflexion, did not tolerate levodopa treatment and did not have the specific evolution of Parkinson's disease over time. A diagnostic reassessment was mandatory in this case and we also included an exhaustive research of all the medical literature published about similar cases diagnosed with camptocormia.

Conclusion. The pathology is quite common but fairly new without having a well-developed diagnostic guide. We only found systematic analyzes that try to integrate the symptomatology of patients as efficiently as possible.

Particularities. The disease had a sudden onset with symptoms mainly in the lumbar spine. Also it was quite similar to Parkinson's disease at first and could only be excluded by the disease evolution.

The utility of semiology in etiological disclosure. Incomplete Villaret's syndrome. Case review

I. GEORGESCU[1], M. ȚARNĂ[1], L.A. GOGU[1], D.A. PÂRVU[2], R.D. MIHĂILESCU[1], B.O. POPESCU[1,3]

[1] Neurology Department, Colentina Clinical Hospital, Bucharest, Romania

[2] Affidea Hiperdia Colentina Clinic, Bucharest, Romania

[3] Neurology Department, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Villaret's syndrome consists of ipsilateral paralysis of the last four cranial nerves (sometimes also the 7th cranial nerve) and the cervical sympathetic fibers. It is caused by a lesion in the posterior retroparotid space.

We present the case of a 37 year old woman, with a history of bilateral polychondritis, who came in for mixed dysphagia, dysarthria and sensation of swollen tongue, with relatively acute onset two days prior. She was also complaining of intense constrictive unilateral left headache, worsening in the evenings. In addition, she mentioned facial asymmetry (dropped left oral commissure and left lagophtalmia) that lasted for 2 days, ca.

one month before. Neurological examination: mixed dysphagia, loss of gag reflex on the left side, dysphonia, dysarthria, left deviation of the tongue in protrusion and no other significant abnormalities. Biological tests: minimal inflammatory syndrome. Differential diagnosis: bulbar stroke, vertebral artery dissection, retropharyngeal or retroparotidian tumor, glomus jugulare tumor, infectious, inflammatory, granulomatous, carcinomatous or vasculitic lesion. Brain CT – angiography was normal. Brain MRI showed left retropharyngeal tumor, with diffuse contrast, involving the left internal carotid artery and partially the left internal jugular vein, with left mastoiditis. Corticosteroids and antibiotics were initiated, with minimal benefit on dysphagia and with complete remission of headache. She was referred to an otolaryngologist for biopsy.

This particular case emphasizes the importance of clinical neurologic examination in diagnostic process.

RWD 2019: Alteplase administration in Neurology Department of Colentina Clinical Hospital

I. GEORGESCU[1], R.D. MIHĂILESCU[1], A.M. VLADILA[1], D.G. LUCA[1], E. ROSIANU[1], I.E. DAVIDESCU[1,2], B.O. POPESCU[1,2]

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

Objectives. We present the evolution of thrombolytic therapy in stroke, in the Neurology Department of Colentina Clinical Hospital, in 2019.

Materials and methods. Retrospective analysis of internal data, National Register and Res.Q data regarding total number of patients, key procedural times, scales, tests and investigations, treatment results and discharge recommendation. We compared this data with that reported in previous years, national reported results and international recommendations.

Results. Total number of patients and addressability for intravenous thrombolysis were higher than previous years, 90 patients in 2019 (as opposed to 38 patients in 2018 and 18 patients in 2017), covering a 120 km area. Average door-to-needle time was 40 minutes (best one being 5 minutes). NIHSS dropped from an average of 13 to 5.2 points. There were 16.6% deaths reported and 15.5% hemorrhagic transformations (none of them being cause of death). Also, there were 2 thrombolysed patients with stroke mimics, without adverse reactions.

Conclusions. We observe a significant increase in total number of patients treated with alteplase administration and an important improvement of key procedural times. Only 55.5% of patients were able to benefit from echocardiography during their admission. Screening of dysphagia, taking into account interventional treatment, calculating NIHSS, monitoring ECG and smoking cessation should be extended to all stroke patients. Unfortunately, there are still unjustified delays in pre-hospital phase and also a shortage in interventional therapy specialists.

The many faces of infective endocarditis

Cristina GHIȚĂ[1], Anca NICULESCU[1], Elena TERECOASĂ[1,2], Cristina TIU[1,2]

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

Background. Neurologic complications in infective endocarditis (IE) are frequently encountered and directly influence the short- and long-term outcome. Acute ischemic or hemorrhagic stroke, subarachnoid hemorrhage, mycotic aneurisms, spondylodiscitis, encephalopathy, meningitis and brain abscesses are the most common neurologic entities that may arise during the course of this disease.

Case report. We present the case of a 74 year old patient who was initially admitted in our department due to confusion, fever and aphasia. Cerebral MRI showed multiple bilateral milimmetric lesions with restricted diffusion suggestive for acute ischemic strokes of cardioembolic ethiology. He was at that time diagnosed with infective endocarditis of unknown etiology, based on a highly suggestive image of mitral valve vegetation, severe mitral valve regurgitation and 3 sets of negative blood cultures. He was administered antibiotic treatment and had a favourable outcome with complete remission of symptoms. 4 months later he was admitted again in our department with left sided hemiparesis, confusion and fever and was diagnosed with acute right parietal hemorrhagic stroke and subarachnoid hemorrhage. Taking into consideration the positive blood cultures with *Staphylococcus aureus*, a myxomatous degeneration of the mitral valve found on transthoracic echocardiography, as well as the Janeway lesions observed on both hands, the vascular manifestations (right parietal hemorrhagic stroke, subarachnoid hemorrhage) and the immune complications (glomerulonephritis) the

diagnosis of recurrent IE was established. Beside the aforementioned complications, the patient also developed spondylodiscitis during hospitalization. After receiving prolonged specific antibiotic therapy, the symptoms remitted entirely and the patient was referred to the Department of Cardiac Surgery.

Conclusion. The heterogenic symptoms of onset of IE should not be disregarded, as early initiation of proper antibiotic therapy greatly improves its outcome. IE must always be considered in patients with ischemic or hemorrhagic stroke of unclear etiology, especially if they have specific brain imaging findings and significant heart valve disease.

Spontaneous multiple cervical artery dissection in a young patient during marathon

A. IAROVOI[2], Z. BAJKO[1,2], L. TOMA[2], A. MOȚĂȚĂIANU[1,2], S. MAIER[1,2], A. STOIAN[1,2], L. BĂRCUȚEAN[1], R. BĂLAȘA[1,2]

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

Cervical artery dissection (CAD) is a relatively unusual entity within cerebrovascular events, but represents approximately 20% of ischemic strokes in patients between 30 to 45 years old. Management options include thrombolysis, antiplatelet or anticoagulation therapy, and endovascular or surgical intervention.

We present a case of a 38-year-old male smoker who presented left neck pain, amaurosis fugax and motor aphasia during a marathon after rapid flexion and extension of the head. Symptoms disappeared after 20 min. In the emergency room, the neurological examination showed no neurological deficit. CT-brain was interpreted as no acute intracranial abnormality. CT-angiography revealed left internal carotid artery dissection from its origin to cavernous segment with subocclusive stenosis. Doppler ultrasound demonstrated left internal carotid artery dissection with severe stenosis and also a dissection of right vertebral artery in V1-V2 segments. The patient was admitted to the hospital and started treatment with heparin and aspirin. He was evaluated by interventional radiology which recommended conservative medical management. On the 13th day of hospitalization, before discharge, Doppler revaluation showed left internal carotid and right vertebral arteries occlusion secondary to the fold dissection. He was discharged home on the 14th day of hospitalization on antiplatelet and anticoagulant agents, with no neurological deficit. Six months later Doppler control showed a total recanalization of the cervical arteries, the patient being asymptomatic.

Conclusion. CAD is a major cause of cerebrovascular diseases in young people and most of them can be safely and conservatively managed, with generally good clinical and imagistic outcomes.

Rapid progressive dementia syndrome. Diagnosis by anatomopatologic post-mortem examination

Raluca-Andreea IGNAT[1], Diana HALIT[1], Ion ROTARU[1], Anca SAVA[2,3], Gabriela Florenta DUMITRESCU[2]

[1] Neurology Departament II, "Prof. Dr. Nicolae Oblu" Emergency Clinical Hospital, Iasi, Romania
[2] Anatomical Pathology, "Prof. Dr. Nicolae Oblu" Emergency Clinical Hospital, Iasi, Romania
[3] Morphofunctional Science Department I, "Gr.T. Popa" University of Medicine and Pharmacy, Iasi, Romania

It is argued that most dementias develop slowly. A careful anamnesis usually detects dementia secondary to drugs or depression, and lab tests help eliminate metabolic disorders that can cause dementia. Most slow progressive dementias are secondary to Alzheimer's disease (AD).

Rapidly progressive dementia (RPD) is a neurological condition that develops subacute, within weeks or months, or less frequently within a few days.

It is important to have an organized and systematic approach to differential diagnosis, especially in identifying DPRs that are treatable and potentially reversible.

DPR prototype are considered by some clinicians, prion diseases, such as Jakob-Creutzfeldt disease (1).

The most commonly used clinical criteria do not allow the early diagnosis of CJD and use auxiliary tests with a sensitivity and specificity considered by some clinicians to be difficult to use in current practice (2).

We present the case of a 70-year-old patient who addresses the neurology service for mental confusion, temporal-spatial disorientation, with sudden onset of the condition. A neoplastic, vascular disease (through repeated imaging scans), an infectious disease, a toxic/metabolic cause (through laboratory tests), and an autoimmune encephalitis with positive auto-antibodies (antibody panel testing) are excluded. The neurological evolution is initially stationary, later with neurological aggravation, with the alteration of the conscious state, with the installation of a right motor focal epileptic sta-

tus, later with death. The positive 14-3-3 protein and the post-mortem anatomopathological result are determinant for our etiological diagnosis.

Keywords: prion, dementia, rapid progressive

References

1. Geschwind MD. Rapidly Progressive Dementia. Continuum (Minneap Minn). 2016;22(2):510-537.
2. Geschwind MD. Prion Diseases. Continuum (Minneap Minn). 2015;21(6 Neuroinfectious Disease):1612-38.

A clinical case of hemodynamic stroke during carotid artery stenting

Octavian IOGHEN, Patricia IOAN, Raluca BADEA, Constantin BUNEA, Diana DUMITRASCU, Alida CIOCAN, Florina ANTOCHI, Ovidiu BAJENARU

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

Carotid artery stenting (CAS) represents a revascularization procedure for symptomatic and hemodynamically significant carotid stenosis. It comes with the advantage of being minimally invasive, but it can lead to various peri- and post-procedural complications.

We present the case of a 76-year-old patient with generalised atheromatosis. Within the last 4 months, he suffered 2 ischemic strokes in the left internal carotid artery teritory, with mixed aphasia and mild right hemiparesis as post-stroke neurologic sequelae. „Four-vessel" cerebral angiography was performed and a suboclusive segmental stenosis was observed at the internal carotid artery origin. An autoexpandable stent was mounted, followed by balloon dilatation. A significant stenosis of the M1 segment of the left middle cerebral artery was observed. During the procedure, hemodynamic depression with severe bradycardia and hypotension occured, which improved after atropine administration. Subsequently, the patient presented confusion, right homonymous hemianopia and worsening of the right arm motor deficit. A subsequent cerebral imaging revealed the formation of a left temporo-parieto-occipital ischemic stroke. Considering the hemodynamic depression episode, there is a high chance that the respective episode together with the alteration of the collateral circulation by the MCA stenosis caused the stroke. Subsequently, hemorrhagic transformation was identified, without worsening in the neurological condition of the patient.

Hemodynamic instability represents a complication of CAS that should always be taken into account. It is produced by the excessive carotid sinus baroceptor stimulation during stent mounting. Thus, monitoring the blood presssure and heart rate during and after the procedure is of utmost importance for prevention and rapid correction.

Tolosa-Hunt syndrome in a patient with hypersensitivity reaction to systemic corticosteroids

Octavian IOGHEN, Patricia IOAN, Raluca BADEA, Florina ANTOCHI, Ovidiu BAJENARU

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

Tolosa-Hunt syndrome is an inflammatory idiopathic disease. It consists of cavernous sinus noncaseating granulomas formation. The diagnosis is made by the presence of the typical clinical picture- periorbital pain and unilateral paresis of the oculomotor, trochlear or abducens nerves- and by the detection of the granulomatous inflammation, either using cerebral imaging or histopathological examination. Other diseases that can affect the cavernous sinus should be excluded.

We present the clinical case of an otherwise healthy 50-year-old female patient. She presented to the hospital with severe left periorbital pain and diplopia. Neurological examination revealed left exophtalmos, impaired left visual acuity, diplopia caused by trochlear and abducens paresis, left periorbital hypoesthesia. Cerebral MRI revealed T2 hyperintense gadolinophilc material in the cavernous sinus, which extends to the superior orbital fissure, orbital apex and optic canal, surrounding the optic nerve. Biopsy was performed and the histopathological examination reveled nonspecific granulomatous inflammation. Subsequent paraclinical tests excluded other cavernous sinus syndrome causes. Treatment with oral metilprednisolone was initiated, which led to the rapid alleviation of the periorbital pain. Nevertheless, the medication was discontinued due to a hypersensitivity reaction. As an alternative, the patient was started on an immunosupresive treatment, such as azathioprine. At 6-month follow-up, the brain MRI revealed a reduction of the inflammatory lesion.

In conclusion, Tolosa-Hunt syndrome is a rare disease that should be taken into account when painful ophtalmoplegia is found. Even though systemic cortico-

therapy is the prefered treatment, immunosupression might as well be used in case of corticoid intolerance.

Differential diagnosis and unusually long survival in a patient with recurrent glioblastoma

A. LEFTER[1], C. LUPU[1], I. GOBEJ[2], A. BASTIAN[3,4], V. IONESCU[5], L. DUMITRESCU[1,4,6], B.O. POPESCU[1,4,7]

[1] Department of Neurology, Colentina Clinical Hospital, Bucharest, Romania
[2] Department of Neurosurgery, Colentina Clinical Hospital, Bucharest, Romania
[3] Department of Anatomic Pathology, Colentina Clinical Hospital, Bucharest, Romania
[4] "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[5] Department of Radiology, Monza Metropolitan Hospital, Bucharest, Romania
[6] Department of Neurology, Research Division, Colentina Clinical Hospital, Bucharest, Romania
[7] Laboratory of Molecular Medicine, "Victor Babeș" National Institute of Pathology, Bucharest, Romania

Introduction. Glioblastoma is the most common primary malignancy of the central nervous system. It is highly aggressive and bears a poor prognosis despite multimodal treatment, with a mean overall survival rate of about 12 to 15 months.

Case report. We report the case of a 46-year old woman who presented at 42 years with headache and focal seizures over the course of 2 weeks, which prompted admission to an infectious disease ward on suspicion of infectious encephalitis. Brain magnetic resonance imaging (MRI) showed a right frontal mass with heterogeneous enhancement, vasogenic oedema and bilateral fronto-insular grey matter changes potentially suggestive for paraneoplastic encephalitis. The patient underwent virtually complete surgical resection of the right frontal mass and histopathological evaluation revealed glioblastoma grade IV WHO. Radiotherapy and chemotherapy with temozolomide were indicated and followed. Over the next 8 months she had a recurrence which was completely excised. At age 46 she had a left frontal relapse, which was surgically removed, yet 4 months later she presented with aphasia, dysphagia and status epilepticus, and brain MRI revealed a left contrast-enhancing temporo-parietal relapse, which was inoperable. The family declined continuing chemotherapy. Thus, palliative care measures were further indicated.

Conclusions. This case initially implied a diagnostic challenge prior to histopathological assessment and the differential diagnosis is further discussed here comprehensively. Management of glioblastoma requires a functional multidisciplinary team despite the unfavourable outcome, with a poor survival rate. Our case is also remarkable in that the survival rate of over 4 years greatly surpasses the median overall reported rate.

Intracerebral calcifications – Fahr disease or Fahr syndrome?

L.D. MAKO[1], N. ASAFTI[1], A.C. BELDEAN[1], A.V. BESNEAG[1,2]

[1] Neuroloy II Clinic, Municipal Hospital, Cluj-Napoca, Romania
[2] Department of Neurosciences, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

Introduction. Fahr disease or Fahr syndrome are characterized by intracerebral calcifications in the basal ganglia, which according to their location can determine a various symptoms: parkinsonism, involuntary movements, cognitive dysfunction and rarely seizures. The etiology of Fahr disease is genetic, whereas Fahr syndrome occurs secondary to disturbances in calcium metabolism.

Case presentation. 81 year old male presents in the Neurology department for dizzines and balance issues. His family also reports moments of compulsive eating, emesis and loss of consciousness with generalized tonic aspect. Our patient is diabetic and hypertensive, both pathologies controlled with oral medication. The neurological examination revealed an asymmetric parkinsonism, postural tremor in the upper limbs, diminished osteotendinous reflexes in the lower limbs, independent walk with small steps and mild cognitive dysfunction.

In order to establish the aethioogical diagnosis we collected blood samples for hormonal investigations, results which showed primary hypoparathiroidism: PTH level extremely low, hypocalcemia, phosphate and magnesium within normal range. An endocrinology consult confirmed the diagnosis. An electroencephalography showed no pathological graph elements which could suggest epileptiform discharges. Head CT showed intracerebral calcifications in the basal ganglia (caudate nucleus), thalamus, cerebellous bilateral, subcortical in the white matter of the occipital lobe and centrum semiovale bilaterally, with a symmetrical aspect.

The patient was diagnosed with Fahr syndrome, secondary to primary hipoparathyroidism and recieved substitutive treatment with calcium, vitamin D and anticonvulsivant (Levetiracetam).

Distinctivness of the case. Late diagnosis, episodes of loss of consciousness with generalized tonic aspect after reaching a normal calcemia, EEG without pathological graph elements.

Confusional state and somnolence in a 52-year old male with hairy cell leukemia and quick worsening of consciousness – a clinical case

C. MARGARIT, I. GANDABESCU, O.A. BAJENARU

University Emergency Hospital, Bucharest, Romania

Introduction. Hairy cell leukemia (HCL) is an uncommon lymphoid neoplasm characterized by the reduction in the production of normal red blood cells, platelets and monocytes. Among its complications there are anemia, bleeding and infection.

Case report. This is the case of a 52-year old male patient diagnosed with HCL four months ago (undergoing chemotherapy), secondary anemia, diabetes, hypertension, sepsis in recent history, hospitalized for confusion and somnolence in the last day. A week ago he had fever that passed without autoadministered medication. At clinical examination the patient is somnolent, confused, central left facial palsy, left hemiparesis 4/5MRC.

The head computed tomography shows no recent or sequelar lesions. Lumbar puncture cannot be performed as the ophthalmologic examination describes bilateral papillary edema. Biologically there is pancytopenia, mild anemia, spontaneous high INR, hyposodemia, hypopotassemia, negative tumoral markers. Electrocardiogram shows sinus rhythm. The patient's situation worsens very fast, becoming very somnolent and with very low cooperation. At many times repeated ophthalmologic exam the aspect remains the same. The electroencephalography and echocardiography are normal. The MRI head scan describes right temporo-insular and capsulo-lenticular ischemic stroke. The patient has fever. Hemoculture sets and urine culture results are negative, no inflammatory syndrome, with hepatic cytolysis and renal deficiency. The patient's situation continues to worsen, unjustified by the MRI scan and he is transferred in the intensive care unit, where he died soon after.

Conclusions. This case was challenging concerning the diagnostic. Cumulating all the information and taking into consideration the history of sepsis, the patient most probably had sepsis again determined by HCL, the highest risk of infection in these patients occuring in the first years after the diagnostic was established.

The evolution of a 32-year old female patient with recurrent remissive multiple sclerosis noncompliant to the treatment – a case presentation

C. MARGARIT, I. GANDABESCU, O.A. BAJENARU

University Emergency Hospital, Bucharest, Romania

Introduction. Multiple sclerosis (MS) is the most common immune-mediated inflammatory demyelinating disease of the central nervous system that predominantly affects young adults.

Case report. This is the case of a 32-year old female patient diagnosed with recurrent remissive multiple sclerosis in 2013, hospitalized for motor deficit worsening in the lower limbs. In 2010 she had an episode of optic neuritis. She enrolled in the National Programme for Multiple Sclerosis, received treatment with interferon beta-1a and then with Natalizumab. As the patient says, she suddenly stopped the treatment two years ago. She was proposed for Ocrelizumab therapy but did not take it. EDSS was first 3,5p, then 4,5p two years ago when she stopped the treatment. When she came to hospital, she has right central facial palsy, horizontal nystagmus at left gaze and when looking up, spastic tetraparesis predominantly crural and in the left limbs, dysmetria, exaggerated osteotendinous reflexes, bilateral achillean clonus, moderate-severe dysarthria – EDSS = 8.5 p.

The head and spine MRI shows in T2 hypersignal/fluid-attenuated inversion recovery (FLAIR) and T1 hyposignal over 40 ovalar supra- and infratentorial bilateral lesions in the middle cerebellar peduncles, pons, midbrain, subcortical, periventricular, fronto-temporo-parietal bilateral, intramedullary C2-T2, T6-T8, T10-T11, with over 10 enhanced-gadolinium lesions. The patient received treatment for the exacerbation with favorable evolution.

Conclusions. This case represents a red flag for patients with multiple sclerosis to take the treatment, without (sudden) discontinuation in order to prevent a rapid evolution of the pathology and its accompanied psycho-social consequences. The patient now has progressed to secondary progressive MS, immobilized in bed, with a chronic urinary catheter.

The case of a 63-year old patient with aphasia and a temporal ischemic hipodensity on CT scan

C. MARGARIT, I. GANDABESCU, O.A. BAJENARU
University Emergency Hospital, Bucharest, Romania

We are presenting the case of a 63-year old female patient, smoker, with diabetes, dyslipidemia, hypertension, overweighted hospitalized for predominant expressive aphasia noticed suddenly in the morning of the day of coming to hospital.

The head computed tomography (CT) examination shows a recent ischemic hypodensity in the left temporal lobe. The patient receives antiplatelet therapy under which the patient's situation evolves rapidly very well, with the almost complete remission of symptomatology, from having alexia, being unable to read to having difficulties in reading the first letter of the word. Taking into consideration that the hypodensity aspect on CT scan was atypical, round-ovoid shaped, inhomogeneous, without respecting a specific vascular territory it is decided to make an MRI head scan. This describes in the left temporo-parietal region in T2 hypersignal / Fluid-Attenuated Inversion Recovery (FLAIR) and T1 hyposignal a cortico-subcortical zone with associated left temporal hematic effusion, suggestive for hemorrhagic venous infarction. The anticoagulant treatment is started. The evolution is favorable and when leaving the patient's neurologic examination is normal. Thrombophilic antibodies were taken with the results in progress.

Conclusions. It is extremely important to investigate in detail every clue that raises a question mark in a patient. The particularity in this case is the unusual image on CT scan together with the symptomatology and rapidly favorable evolution of the patient.

Atypical excessive somnolence in a 62-year old female with left hemiparesis – a clinical case

C. MARGARIT, I. GANDABESCU, O.A. BAJENARU
University Emergency Hospital, Bucharest, Romania

Introduction. Somnolence is a vague, nonspecific symptom with a variable etiology. In some cases it can become a problem in diagnosing.

Case report. This is the case of a 62-year old female patient, known with hypertension and chronic alcoholism, hospitalized for left hemiparesis and mild somnolence since one day. At clinical examination the patient is somnolent, with left ataxic hemiparesis 4/5MRC, dysarthry, normal temperature, accusing intense vertigo. At head computed tomography there are two small sequelar hypodensities in the left occipital region and right parietal region. During the hospitalization the somnolence progressively becomes more intense. Biologically there is an inflammatory syndrome and hyperglycemia with high value of glycosylate hemoglobin. Electrocardiography, electroencephalography, ophthalmologic exam, chest radiography and Holter examination/24 h results are normal.

Transthoracic echography shows nodular, hyperechogenic forms attached to the posterior mitral and aortic valves. Hemoculture sets were negative. Transesophageal echography shows small subvalvular aortic forms, nonsuggestive for vegetations. Head MRI shows multiple regions with restriction of diffusion in cerebral peduncles, pons, right thalamus, occipital bilateral, right cerebellum peduncle, suggestive for multiple embolic strokes. New hemoculture sets are positive for Enterococcus spp. The patient received antibiotic therapy with favorable outcome.

Conclusions. What is striking at this case is the excessive atypical somnolence, which is evolving. Besides, the normal temperature, the transesophageal echography not suggestive for vegetations, the first hemocultures sets that were negative, the Holter examination result made it difficult to establish a diagnostic. The head MRI and the result of the last hemoculture sets helped us conclude that the patient suffered multiple vertebro-basilar cardioembolic strokes through septic dissemination.

Etiopathogenic and therapeutic considerations based on a clinical case of paroxysmal cerebellar ataxia

Maria M. MARTOIU[1], Carmen RAGAN[1], Cristina A. PANEA[1,2]
[1] *Neurology Department, Elias University Hospital, Bucharest, Romania*
[2] *"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Introduction. Episodic ataxia known as paroxysmal cerebellar ataxia is a heterogeneous group of disorders that are characterized by recurrent episodes of ataxia and incoordination, lasting minutes to hours.

Objective. We present the case of a young patient with a form of paroxysmal cerebellar ataxia including clinical features of episodic ataxic type 2 (EA2), which posed diagnosis difficulties, and was responsive to Acetazolamide and Levetiracetam.

Case report. 23 years old male with recurrent episodes of dysarthria, nystagmus, incoordination and walking disorder, with variable duration – minutes to hours, with frequency and intensity that had increased over the last few months.

The onset of the symptoms was around the age of seven, when the episodes were classified as epileptic seizures, especially as treatment with valproate controlled the symptomatology at that time.

Family anamnesis revealed fetal distress at birth, delayed motor and cognitiv development.

The neurologic exam between episodes revealed a right cerebellar syndrome, nystagmus and mild dysartria. The brain MRI showed mild atrophy of the cerebellar vermis. The EEG revealed bihemispheric epileptiform discharges. The EMG and ionogram during episodes were normal.

Acetazolamide was associated to Levetiracetam therapy.

Conclusions. The non-progressive course of the disease, the cerebral MRI and the other paraclinical exams did not bring arguments for infantile encephalopathy or a hereditary metabolic disorder.

Although the EEG revealed epileptiform discharges, the clinical episodes aspect and duration were not typical for epileptic seizures.

Taking into account the paroxysmal aspect of the symptomatology it was taking into discussion an episodic ataxia type 2, a channelopathy of the calcium channel. Genetic testing was not possible.

Keywords: paroxysmal cerebellar ataxia, channelopathies

A rare cause of ischemic stroke in vertebro-basilar territory: Rotational vertebral artery occlusion (Bow-Hunter's syndrome)

O. MOSORA[1], A. MOȚĂȚĂIANU[1,2], Z. BAJKO[1,2], S. MAIER[1,2], A. STOIAN[1,2], L. BĂRCUȚEAN[1], R. BĂLAȘA[1,2]

[1] Neurology I Clinic, Emergency Clinical County Hospital, Targu Mures, Romania
[2] Discipline of Neurology, "G.E. Palade" University of Medicine, Pharmacy, Science and Technology, Targu Mures, Romania

Introduction. Bow Hunter's syndrome is a rare vascular pathology that can be manifested by transient ischemia as a result of compression on the vertebral artery at the time of rotation of the head.

Case report. We present the case of a 35-year-old patient who suddenly presents with balance disorders, nausea and vomiting, reporting a similar episode in history. A cranial CT scan is performed that does not show acute heterodense lesions, an angio CT scans that do not show thrombosis or aneurysms in the arteries afferent or efferent to the Willis polygon, homogeneous opacified cerebral venous sinuses and carotid Doppler ultrasound that didn't show any modifications. The patient returns to the Neurology Clinic for reassessment. The Doppler ultrasound is repeated in left lateral rotational position of the cephalic extremity that highlights the disappearance of blood flow in the right vertebral artery, raising the suspicion of mechanical compression at V3 or V4 level. Angiography CT is performed at the level of the cervical vessels in the lateral rotation position of the cephalic extremity, highlighting a stenosis of 90% at the level of the C2 segmental vertebral artery.

The patient received treatment with Plavix, Sortis, Enap, Metoprolol, Metoclopramide, Betaserc, vitamin B1, vitamin B6 and the patient presented a favorable evolution with regression of symptoms.

Conclusions. The case was interpreted as an acute central vestibular syndrome that arose as a result of rotational occlusion of the right vertebral artery. This case highlights a rare cause of cerebral ischemia in young patients. It is important to monitor these patients to reduce the appearance of symptoms due to circulatory dysfunction.

The attention deficit in Parkinson's disease

H. NICOLAE[1,2], I. IONIȚĂ[1], I. BARBU-RĂDULESCU[2], C. PANEA[1,2]

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

Introduction. Parkinson's disease is the second most frequent neurodegenerative disorder in the world, after Alzheimer's disease, with cognitive impairment present from the beginning of the disease. Out of the six cognitive domains in DSM-5, the study we conducted analyzed the presence of the attention deficit in patients with Parkinson's disease.

Material and methods. We conducted a study on 130 patients with Parkinson's disease in evidence at the department of Neurology of Elias University Emergency Hospital. We identified the presence of attention and memory deficit through both subjective methods –

Parkinson's well-being map – and objective methods – MMSE score. We evaluated sleep quality through Parkinson's disease sleep scale. We performed a statistical analysis on the data collected.

Results. We identified a deficit of attention in 66 patients, 32 did not have neurocognitive disorder, 24 had mild cognitive impairment and 10 had a moderate form of major neurocognitive disorder. The attention deficit existed in 50.76% of the patients with Parkinson's disease, affecting almost equally patients with and without neurocognitive disorder. An association was found between sleep and the frequency of attention and memory deficit symptoms. The attention deficit existed either alone, or associated with memory loss.

Conclusions. The attention deficit is important in patients with Parkinson's disease, affecting both patients with and without neurocognitive impairment. There is a correlation between the frequency of attention deficit symptoms and sleep disturbances.

Keywords: Parkinson`s disease, attention deficit

Increased risk of hypoxia-induced seizure recurrence in a young patient with autoimmune encephalitis and severe secondary tracheal stenosis

I. ODAJIU[1], L. CRĂCIUN[1], I. DAVIDESCU[1,2]

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

Background and aims. Anti-GABA-A receptor encephalitis is an immune-mediated inflammatory condition of the brain, with a rapidly progressive course manifesting with refractory seizures, status epilepticus and/or epilepsia partialis continua. Tracheal stenosis can determine variable degrees of hypoxia, therefore serving as an epileptogenic trigger. Our aim was to present the case of a patient with anti-GABA-A receptor encephalitis that developed recurrent seizures following a severe secondary tracheal stenosis.

Case report. We present the case of a 22-years old female patient, with no remarkable personal of family medical history, who developed anti-GABA-A receptor encephalitis manifested initially by convulsive status epilepticus with a prolonged course of protective orotracheal intubation. She underwent treatment with methylprednisolon pulse-therapy, plasma exchange and intravenous immunoglobulins along with antiepiletic medication, with a remarkable clinical outcome without any MRI specific lesions afterwards. Yet, she developed a severe upper tracheal stenosis with stridor and constant dyspnea for which a tracheal dilation was performed. Two weeks later, new repetitive focal motor seizures appeared and the stridor with inspiratory dyspnea recurred. The patient had constant reduced oxygen pressure levels and was referred for to the ENT department.

Conclusions. The peculiarity of the case relies in the fact that anti-GABA-A receptor encephalitis patients have an increased risk of recurrent seizures, but additional factors such as hypoxia can further alter the function of the GABAergic system and serve as a serious epileptogenic factor especially in such patients.

Real world data – Teriflunomide experience in Colentina Clinical Hospital MS Center

I. ODAJIU[1], D. SANDU[1], I. DAVIDESCU[1,2]

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

Background and aims. Teriflunomide, an immunomodulatory disease modifying therapy (DMT) administered in relapsing-remitting multiple sclerosis, is the single oral drug currently available in Romania since 2017. Our aim was to present the real world data regarding patients undergoing treatment with Teriflunomide in Colentina Clinical Hospital.

Methods. Non-interventional retrospective study. Data gathered and processed in Microsoft Excel.

Results. Out of 820 patients in the Multiple Sclerosis Program in our center, 16.46% (135) administer Teriflunomide 14 mg once daily. The majority of them are women (65.93%) from urban areas (79.25%) with a median age of 41.26 years (80% range between 35 and 59)**.** The median EDSS score was 2.7 and disease evolution time of 7.8 years, values comparable to other studies. Naïve patients (42.22%) in contrast to patients who switched from other DMTs to Teriflunomide (57,78%) had a shorter disease duration of 5.5 years versus 9.43 and a better EDSS score of 2.0 versus 3.0. The majority of patients switched from Interferon ß1-a (66%) followed by Glatiramer acetate (26%) and for 12 patients it was the second or even third switch, the primary determinant being lack of tolerance regarding injectables. As regards Teriflunomide's efficacy, there were registered no relapses or EDSS progression since treatment initiation.

Adverse events, namely hair loss or thinning, occurred in 8.88% cases (12 patients).

Conclusions. The real world data gathered in our center proves that Teriflunomide appears to be one of the best tolerated DMTs with a satisfactory safety and effectiveness profile in carefully selected patients which ensures a stable disease course.

Good outcome of an acute severe sensory-motor axonal neuropathy after infection with AH1N1

R.B. OLARIU-ARITON[1], A. MOȚĂȚĂIANU[1,2], Z. BAJKO[1,2], S. MAIER[1,2], A. STOIAN[1,2], L. BĂRCUȚEAN[2], R. BĂLAȘA[1,2]

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

Introduction. Axonal acute sensory-motor polyradiculoneuropathy is a variant of Guillain-Barré syndrome, the defining characteristics being represented by electrically inexcitable motor nerves, signs of extended denervation and also by a possible complete recovery. The most common cause of this pathology is a respiratory infection.

Case report. We present the case of a 56-year-old patient who, after a respiratory tract infection with influenza AH1N1 virus a month before hospitalization, suddenly presents, in the morning, paresthesias in the distal lower and upper limbs, later, the patient develops motor deficit at this ascending level. Less than 24 hours after appearance, the patient becomes tetraplegic, with severe acute respiratory failure, requiring orotracheal intubation and mechanical ventilation. Four plasmapheresis sessions were performed with a stationary evolution in the Neurology Department of the Sfântu Gheorghe County Emergency Hospital, later she was transferred to the Neurology I Clinic within Mures County Emergency Clinical Hospital for the treatment with intravenous immunoglobulins.

The electroneuromyographic examination performed on the median, ulnar, bilateral common peroneal nerves does not show motor / sensory response, at supramaximal stimulation the motor and sensory nerves are inexcitable, in the vast lateral and right deltoid muscles show acute neurogenic pathway with active denervation.

Examination of cerebrospinal fluid reveals albuminocytological dissociation.

During hospitalization, the patient required advanced life support, mechanical ventilation for four months, then after multiple attempts at ventilatory withdrawal, it shows effective spontaneous breathing and succeeds in suppressing the tracheostomy cannula. After about five months, the patient is discharged with endoscopic percutaneous gastrostomy and tetraplegic.

Conclusions. The acute axonal form of Guillain-Barré syndrome is an important, rapidly evolving pathology that can occur after a respiratory infection. Establishing treatment on time increases the prognosis of recovery.

"Thunderclap" headache in pituitary apoplexy with aseptic meningitis clinical case

Cristina PATRASCU, Vlad GHIMPU, Stefan DUMITRACHE

National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania

Introduction. "Thunderclap" is a headache of maximum intensity, which may suggest a varied neurological pathology, such as SAH, cerebral venous thrombosis, reversible cerebral vasoconstriction syndrome, meningoencephalitis or pituitary apoplexy.

Case report. T.C.A, female, 19 years old, without medical history, smoker, postpartum, without medication at home, is admitted for persistent, intense headache, reaching the maximum threshold quickly, nausea and vomiting, symptoms that suddenly started, which did not respond to any NSAIDs.

Neurological examination reveals stiff neck. Laboratory tests show mild microcytic hypochromic anemia, mild leukocytosis with neutrophilia. Native brain CT is normal. Lumbar puncture shows increased cellularity (lymphocytes and neutrophils), but with proteins and glucose within normal limits, negative Pandy reaction, IgG and oligoclonal bands normal and negative cultures. The suspicion of encephalitis is raised so that an EEG is performed, but with normal results and brain MRI with contrast and angio-MRI, made to exclude venous thrombosis as a differential diagnosis, given the postpartum status, reveals constitutional stenosis in the transverse and sigmoid sinuses and a non-secreting hemorrhagic pituitary microadenoma (normal hormonal profile) without any characteristics of encephalitis.

Conclusions. Following the investigations, subarachnoid hemorrhage (cerebral CT and LP), venous thrombosis (cerebral MRI and venous angio-MRI), as well as meningoencephalitis (MRI and EEG) were excluded. The particularity of the case is the onset of pituitary microadenoma bleeding with "thunderclap" headache

accompanied by meningism, and lumbar puncture with the appearance of aseptic meningitis.

Keywords: "thunderclap" headache, meningism, pituitary apoplexy, aseptic meningitis

Atypical imaging after pontine ischemic stroke

A.M. PARTOACA, E.I. IOACHIM, A. SIRBU, A. POPESCU

National Institute of Neurology and Neurovascular Diseases, Bucharest, Romania

Introduction. Wallerian degeneration is the process of degeneration and disintegration of axons and their accompanying myelin sheaths secondary to axonal proximal lesions or neurons that can affect both the peripheral and central nervous system. In the case of brain injuries, this process frequently affects the cortico-spinal tract but also the optic tract, the fibers of the corpus callosum, the fornix and the pontocerebellar tracts. The investigation that can objectify these changes, in case of damage to a large number of fibers, is MRI.

Case report. The authors present the case of a 60-year-old patient with cardiovascular risk factors and medical history of vertebro-basilar ischemic stroke treated with thrombolytic therapy (with favorable evolution, Rankin score = 0 points), who presented to our clinic for neurological reassessment. We illustrate the imaging changes at a distance of 12 months after cerebral ischemia.

Brain MRI shows a right paramedian pontine lacunar lession and T2/FLAIR hypersignal with relatively symmetrical T1 hyposignal, at the level of the middle cerebellar peduncles in the context of Wallerian degeneration.

Conclusions. The distribution of Wallerian degeneration depends on the relation of the brain lesion to the efferent axonal tracts. The most common pattern seen in the brain is degeneration of the corticospinal tract in the context of lesions of the precentral gyrus. The peculiarity of the case is the highlighting of this process at the level of the middle cerebellar peduncles, by degeneration of the ponto-cerebellar tracts, a less common location.

Progressive multifocal leukoencephalopathy after non-Hodgkin's lymphoma treatment – Case presentation

C.M. PETREA[2], A. ALEXANDRESCU[2], A. MOȚĂTĂIANU[1,2], A. STOIAN[1,2], S. MAIER[1,2], L. BĂRCUȚEAN[1], Z. BAJKO[1,2], R. BĂLAȘA[1,2]

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

Introduction. Progressive multifocal leukoencephalopathy (PML) is a rare demyelinating disease of the central nervous system and results from the reactivation of a latent JC polyomavirus. It is frequently diagnosed in HIV-positive patients, but recently, the attention has been focused on the association between immunosuppressive therapy and PML.

Objective. The purpose of this presentation is to raise the suspicion of PML when a patient meets the anamnestic, clinical, imaging and laboratory criteria.

Case presentation. We would like to present the case of a 27-year-old patient diagnosed with T-cell lymphoblastic lymphoma, treated with polychemiotherapy and autologous stem cell transplantation. After one year, he presents in the emergency department accusing dizziness, nausea, vomiting and vision disorders. At the neurological examination were detected: diplopia in the lateral gaze to the left, limitation of abduction of the left eyeball, non-systematic positive Romberg test, gait ataxia. Serum biochemistry revealed a hepatic cytolysis syndrome, anti-HIV serological tests, VDRL, HBs antigen, anti-HCV antibodies were negative and cerebral CT/AngioCT examinations were normal. Cerebral MRI examination revealed pontine and left cerebellar white matter hyperintense lesions on T2/FLAIR and the CSF biochemical examination revealed an increased proteinuria of 1,949.6 mg/dl, with albuminocytological dissociation. With the suspicion of PML, serum samples were collected for JC virus antibodies, which were positive. Isolation of viral DNA from the CSF was not possible, so we can only very likely suspect PML.

Conclusions. This case draws attention to neurological symptoms during or after immunosuppressive treatment and aims to include PML in the differential diagnosis series. Early diagnosis also implies a better therapeutic attitude.

Keywords: progressive multifocal leukoencephalopathy, immunosuppressive therapy, JC virus

Amyloid angiopathy and concomitant atrial fibrillation – challenges regarding long-term treatment

C. POSTOLE, A.M. NICULESCU, N. GRECU, C. TIU

Neurology Department, University Emergency Hospital, Bucharest, Romania

Background. Cerebral amyloid angiopathy is a degenerative condition with an increased risk of intracerebral hemorrhages, most commonly encountered in the elderly. A major problem is the antithrombotic treatment in this setting in this age group, as these patients frequently have associated disorders with an increased risk of stroke.

Case report. An 85-year-old patient, with a history of atrial fibrillation (for which she received apixaban 2.5 mg bid), hypertension, hypercholesterolemia, and diabetes mellitus, was admitted for facial asymmetry and moderate dysarthria.

Non-contrast-enhanced cerebral CT scan showed a small hyperdensity of 91 Hounsfield units (HU) located in the left frontal cortex. Cerebral MRI showed multiple cortical and subarachnoid hypointense T2* lesions, suggestive of cerebral amyloid angiopathy (CAA).

She subsequently developed facial motor seizures with secondary generalization, with good response to therapy with levetiracetam. Cognitive evaluation showed a severe impairment. Serum analysis was significant for reduced B12 vitamin levels.

Final treatment recommendation, taking into consideration the increased risk for cardioembolic events, was the continuation of the patient's ongoing therapy as this provides a superior safety profile compared to other drugs.

Conclusions. Conventional cerebral CT examination cannot precisely determine the etiology of lesions less than 100HU. MRI, with the aid of the Boston criteria, can aid the diagnosis of CAA. While CAA can also manifest as dementia it is advisable to perform a complete standard evaluation for other treatable causes of cognitive impairment. Oral anticoagulation reduces the global risk of cerebrovascular events in patients with CAA and atrial fibrillation.

PRES, psychotic disorder and eclampsia in a woman with twin pregnancy and major thrombophilia

L. PRODAN[1,2], C. ROMAN-FILIP[1,2], A. TANASE[1]

[1] *Department of Neurology, Emergency County Hospital, Sibiu, Romania*

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

Background. Posterior reversible encephalopathy syndrome (PRES) is a rare clinical and neuro-radiological condition, often associated with hypertensive encephalopathy, preeclampsia-eclampsia, immunosuppressive agents.

Objective. This is the case of a patient with twin pregnancy, known with major thrombophilia, complicated with preeclampsia-eclampsia and PRES.

Case report. The 33 years old pregnant woman, known with major thrombophilia treated with Clexane, who presented at 35 weeks twin pregnancy lower extremity swelling and discrete global headache. Her blood pressure was 196/138 mmHg. The patient presents a first generalised tonic-clonic seizure, then a second generalized tonic-clonic seizure. An emergency cesarean section was performed using general anesthesia.

Postoperative neurological examination revealed: spatial and temporal disorientation, heteroaggressivity and self-defense at minor stimulation, prosopagnosia and an episode of absence preceded by psychomotor agitation. Pregnancy succeeded with two alive fetuses with dynamics in normal limits.

The MRI examination initially reveals multiple areas with hypersignal T2 and FLAIR, hyposignal T1 with subcortical distribution and cortical extension, located at the temporo-occipital level and the parietal lobe, with complete resolution of the lesions in dynamics.

The patient was evaluated periodically, neurologically and psychiatricly with favorable evolution. She has not presented anymore epileptic seizures, false recognitions, visual hallucinations. Her blood pressure was normal and also the brain MRI control.

Conclusions. This is the case of a patient known with major thrombophilia, twin pregnancy, who presented eclampsia and PRES. She presents prosopagnosia, delirium and hallucinatory syndrome, drug controlled and clinical remission associated with remission of neuroimaging signs.

Sturge-Weber syndrome and hemiplegic migraine – case report

L. PURCARU[1], I. TUDOR[1], E. TERECOASA[1,2], N. GRECU[1], C. TIU[1,2]

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

Introduction. Sturge-Weber syndrome is a sporadic neurocutaneous disorder which associates epileptic seizures and migraine, the latter in about 28% of patients.

Case presentation. A 28-years-old female, with type II Sturge-Weber syndrome and epilepsy (treated with levetiracetam, with lowered doses during pregnancy), developed right hemicrania and left hemiparesis 2 days after a caesarean section; she also developed recurrent seizures with left hemifacial onset and secondary generalization. Her medical history was also significant for two other episodes of right-sided hemicranias associating left hemiparesis, which lasted for about 7 days. A cerebral MRI, excluded a cerebral venous thrombosis, but was consistent with her prior diagnosis, showing volume asymmetry between the cerebral hemispheres, with no leptomeningeal angiomatosis. Her clinical evolution was good, with remission of neurologic deficits in 10 days. Taking into consideration her history, the diagnosis of associated sporadic hemiplegic migraine was taken into consideration.

Conclusions. The absence of leptomeningeal angiomatosis as well as the absence of significant cortical structures modifications and EEG alterations raises the issue of the etiology of the migraine and epileptic seizures in this case. Hemiplegic migraine can trigger epileptic seizures, both during the attack as well as intercritical.

Depression in ALS patients with a minumum one year since diagnosis in a Romanian population: Interraction of physical function, vegetative symptoms and progresion rate

C. RADU[1], S. MAIER[1,2], Z. BAJKO[1,2], A. MOȚĂȚĂIANU[1,2], R. BĂLAŞA[1,2]

[1] *Neurology I Department, Emergency Clinical County Hospital, Targu-Mures, Romania*
[2] *Discipline of Neurology, „G.E. Palade" University of Medicine Pharmacy Science and Technology, Targu Mures, Romania*

Introduction. Until recently, amyotrophic lateral sclerosis was perceived as a pure motor neuron disease and the involvement of cognitive impairment was not properly studied. Depression is an underdiagnosed comorbidity associated with ALS.

Material and method. 33 patients with the diagnosis of amyotrophic lateral sclerosis, who attended the Neurology I Department of Mures *County Clinical* Emergency *Hospital, were included. All participants underwent a neurological evaluation and were evaluated using Beck Depression Inventory and* ALS-FRSr scale. BDI-I is often denounced for overestimating depression in disabled persons because of its somatic items, but we created a sub-score of vegetative symptoms measured within the BDI-I. Five questions were selected to describe vegetative symptoms: loss of interest in sex, sleep disturbance, fatigue, appetite and weight loss.

Results. 36% of patients were diagnosed with depressive disorder according to BDI-I. The total score of the vegetative symptoms is correlated with depression ($p = 0.001$, OR 35.20, CI 95% 3.6-344.18), as well as particular items: appetite and weight loss ($p = 0.028$, OR 10.0, CI 95% 0.96-103.77) and libido loss ($p = 0.002$, OR 13.3, CI 95% 2.08-84.98). Also, we found significant positive correlation between the BDI-I score and respiratory impairment (($p = 0.008$, OR 14.28, CI 95% 1.41-144.37). Patients affected by salivary dysfunction ($p = 0.001$, OR 4.000, CI 95% 2.106-7.598) and speech disturbance ($p = 0.03$, OR 0.147, CI 95% 0.023-0.942) have more severe symptoms of depression. Increased depressive symptoms were associated with disease progression, and a positive correlation is established $p = 0.001$, $R = 0.558$.

Conclusions. Our results suggest that there are significant correlations between depression and physical function, vegetative symptoms and progression rate. Depression may occur in patients with ALS, but is not an inevitable consequence of the progression of this disease.

Parkinsonism – a complex approach

P. ROȘU[1], B. AZAMFIREI[1], A.V. BLESNEAG[1,2]

[1] *Neurology II Clinic, County Emergency Hospital, Cluj-Napoca, Romania*
[2] *Neuroscience Department, „Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania*

Introduction. Parkinsonism is frequently enclosed in Parkinson's disease, but many other signs and symptoms of the clinical picture are falsely interpreted or overlooked.

Case presentation. A 79 year old female patient, diagnosed with Parkinson's disease for 5 years, is admit-

ted to our clinic for postural instability, frequent falls, worsening gait, confusion and apathy. Initially the patient had a favorable outcome under treatment with Levodopa+Carbidopa, however gradually she presented cognitive and motor function decline. The neurological exam reveals temporo-spatial disorientation, asymmetrical hypertonic-hypokinetic syndrome, bradylalia, eupraxia, insomnia and the psychiatric evaluation exposed visual and auditory hallucinations, especially during the night accompanied by psychomotor agitation. Cerebral MRI showed symmetrical cortico-subcortical atrophy. The detailed heteroanamnestic data revealed that the cognitive decline and the postural instability had an earlier onset than the motor dysfunction. This fact alongside the presence of the hallucinations lead to the final diagnosis of Lewy Body dementia.

Conclusion. The patient exhibiting parkinsonism requires a multidisciplinary approach and monitorization. The differential diagnosis of the Parkinson-plus syndromes should not be overlooked.

Particularities. The early onset of the cognitive disfunction, the poor response to treatment and the presence of hallucinations lead to the final diagnosis of Lewy Body dementia, confirmed solely post-mortem.

Can we safely use non-vitamin K antagonist oral anticoagulants in patients with myasthenia gravis? Case report

V. SAPIRA[1,2], A. TELEHUZ[3], A. NECHIFOR[2], A. MOCANU[1], I.M. COJOCARU[4], L. CHIPER[1], M. LUNGU[1,2]

[1] "Sf. Ap. Andrei" Emergency Clinical County Hospital, Galati, Romania
[2] "Dunarea de Jos" University, Galati, Romania
[3] Emergency County Hospital, Slobozia, Romania
[4] Colentina Clinical Hospital, Bucharest, Romania

Introduction. There are several factors that influence cholinergic transmission, among them medication, temperature and emotional state play a key role. There are some classes of medications that can provoke exacerbations, therefore, carefully obtaining a medication history is of vital importance. In the literature there is little or no data whatsoever that concerns the use of non-vitamin K antagonist oral anticoagulants (NOAC) in patients with myasthenia gravis. What NOAC should we chose? Is there a difference between different types of NOAC?

Case report. We present the case of a 70 year old woman that has been diagnosed with myasthenia gravis 9 months ago. She was on pyridostigmine 240 mg/daily in all this time. Now she was admitted in our Neurology department for palpebral ptosis dysphagia, frequent cough, that she developed about 2 weeks ago. In the last 3 days the symptoms started to get worse. Her medical evolution gets worse and she develops acute respiratory failure that requires the use of endotracheal intubation and mechanical ventilation. She undergoes four sessions of plasmapheresis with a slow improvement of medical condition. There had been performed multiple electrocardiograms that demonstrated that the patient had paroxysmal atrial fibrillation with a CHA_2DS_2VASc score of 4 that needed oral anticoagulation. Therefore we revised the case reports in the international literature and chose to use Apixaban as the NOAC. Under treatment with Apixaban there was no signs of exacerbation.

Conclusions. There are multifactorial mechanisms implicated in the exacerbation of myasthenia gravis, this fact reflects the complex dynamics of the neuromuscular junction system. The use of NOAC in patients with myasthenia gravis that have atrial fibrillation is for the moment at the beginning and that is why patients that take oral anticoagulants should be very carefully monitored.

Keywords: myasthenia gravis, NOAC, neuromuscular junction, atrial fibrillation

Levodopa-Carbidopa Intestinal Gel impact on long term QoL and safety in advanced Parkinson disease patients: Romanian results from GLORIA registry

J. SZASZ[1,2], M. SIMU[3,4], L. PERJU-DUMBRAVA[5], L. BERGMANN[6], D. POPESCU[7], O.A. BAJENARU[8,9]

[1] Department of Neurology, Emergency Clinical County Hospital Mures,
[2] "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Targu-Mures, Romania
[3] Department of Neurology, Clinical Emergency County Hospital,
[4] "Victor Babes" University of Medicine and Pharmacy, Timisoara, Romania
[5] "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania
[6] AbbVie, Inc., North Chicago, USA
[7] AbbVie SRL, Bucharest, Romania
[8] Department of Neurology, University Emergency Hospital,
[9] "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Background. Levodopa-Carbidopa Intestinal Gel (LCIG) treatment aims to reduce motor fluctuations by

providing stable plasma levodopa concentrations in advanced Parkinson's disease (APD) patients.

Objective. GLORIA registry evaluated LCIG efficacy, safety and effect on quality of life (QoL). We present the results for the enrolled Romanian patients.

Methods. GLORIA was an international, multicenter, observational registry. In Romania, 39 APD patients were enrolled and followed for up to 24 months. Changes in complications of therapy and activities of daily living (ADL) were evaluated by Unified Parkinson's Disease Rating Scale (UPDRS). QoL was assessed by the Disease-specific 8-item Parkinson's Disease Questionnaire (PDQ-8) and the EuroQoL-5 Dimensions questionnaire (EQ-5D). Adverse Drug Reactions (ADRs) were collected from baseline up to 28 days after the end of the follow-up.

Results. Over 24-month LCIG treatment period there was a significant and clinically relevant improvement in "Off" time, "On" time with dyskinesia, and ADL. PDQ-8 total score decreased significantly up to 12 months post LCIG therapy initiation and maintained numerically lower than baseline up to study end. EQ-5D usual activities item score improved significantly up to 18 months and remained numerically lower than baseline up to 24 months. During the permanent tube phase was reported at least 1 ADR for 62.9% of the patients. In 1 patient the ADR (polyneuropathy) led to LCIG discontinuation. The most frequently reported ADRs were decrease of body weight and polyneuropathy.

Conclusions. LCIG treatment showed sustained improvements in ADL and QoL in APD patients and confirmed the established safety profile.

A hypoechogenic left atrial appendage formation – cause of ischemic stroke in a young patient

Daniela STEFAN[1], Bogdan CASARU[1], Athena RIBIGAN[1,2], Ovidiu BAJENARU[1,2]

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

Background and aims. Although cardioembolic strokes account for an important proportion of ischemic strokes, left atrial appendage thrombi and myxomas are rare causes of embolism.

Methods. We present the case of a 51-year-old man, known with intellectual disability, who was admitted for an acute ischemic stroke in the territory of the left internal carotid artery, for which he undergone intravenous thrombolysis and endovascular mechanical thrombectomy. The EKG performed at admission and the 24 hours EKG monitoring showed sinus rhythm. The carotid Doppler ultrasonography did not reveal stenoses or atheromas. No significant changes were found at transthoracic echocardiography. Follow up cerebral MRI revealed an acute ischemic stroke in the same territory, despite antithrombotic treatment. On further evaluation, transcranial Doppler monitoring for emboli detection showed the presence of one microembolic signal. Transesophageal echocardiography revealed a hypoechogenic formation in the left atrial appendage, attached to the lateral wall of the left atrium, mobile, with irregular margins (0.9/0.4 cm). The hypotheses of an intracardiac thrombus or myxoma were placed. We also performed a cardiac MRI, with an aspect more likely suggestive of a thrombus.

Results. We initiated anticoagulant treatment. Follow up transesophageal echocardiography after 1 month showed a minimal decrease in the size of the left atrial appendage formation. At this moment, there is a high probability of the formation being a thrombus, but we can not certainly exclude a myxoma.

Conclusion. Although left atrial appendage thrombi and myxomas are rare causes of stroke, they should not be overlooked in young patients with no other apparent cause of the stroke.

What does a half swollen tongue hide?

I. TUDOR[1], L.I. PURCARU[1], E.O. TERECOASA[1,3], R.A. RADU[2], B. DOROBAT[2], C. TIU[1,3]

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

Introduction. We present the case of a 44 years old female patient, without any other associated pathology, except for a cervical trauma 10 years before, admitted in our department for paresthesias with tingling on the right side of the tongue and mild dysarthria.

Case presentation. At the moment of admission the neurological examination was normal. The patient affirmed a feeling of swollen tongue and minimal dysarthria. Next day, the neurological examination revealed tongue deviated to the right side in protrusion. Diagnosis: right hypoglossal nerve palsy. Brain magnetic resonance imaging revealed a 10/5 mm mass with marked

contrast enhancement located anterior to the right internal carotid artery, imprinting the right hypoglossal nerve channel and a T2/FLAIR hyperintensity of the right half of the tongue. Cerebral angiography revealed 2 sacciform aneurysms of the right internal carotid artery mirrorly located (most likely secondary to a post-traumatic dissection), and a small caliber of the artery, suggestive of vasospasm. A second angiography, performed with the purpose of placing a stent graft, showed the presence of a single aneurysm of reduced size compared with the first examination and a loop made by the right internal carotid artery. During the procedure the patient developed severe vasospasm, so the procedure was stopped, without placing the stent. The electrophysiological examination was normal and the dysarthria and the swollen tongue meanwhile remitted. She was discharged with the recommendation of antiplatelet treatment with Aspirin.

Conclusions. The hypoglossal nerve palsy was caused by ischemic nerve compression due to an internal carotid artery aneurysm.

Blindness – a diagnostic challenge in neurology

I.M. VĂDAN[1,2], I. PĂCURAR[1], A.V. BLESNEAG[1,2]

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

A 49-year-old patient with a smoking history and a traumatic amputation of the right lower leg presents himself to the Neurology Department for progressive loss of vision for approximately 18 months. The previous ophthalmology consultations revealed an optic atrophy with mild progression.

The neurological examination showed low visual acuity in both eyes, right areactive mydriasis, abolished right photomotor reflex, convergence paralysis of the right eye, normal oculomotricity, diminished deep tendon reflexes of the left lower limb, absent left plantar reflex. The laboratory tests showed mild leukocytosis, positive qualitative VDRL test, positive TPHA test, quantitative VDRL test 1/32 0/2, negative ELISA IgM test, negative HIV test. Magnetic-resonance angiography examination of the head and neck vessels showed few nonspecific white matter lesions. Electroneurography was normal. CSF examination showed positive TPHA test, positive qualitative and quantitative VDRL test. Mental status examination was normal.

After the dermatology consultation treatment was started with benzylpenicillin 7,200,000 IU, administered IM in 3 doses at 1 week interval.

Familial investigation showed that patient's wife was also positive for syphilis, but asymptomatic.

Ocular syphilis, a type of neurosyphilis, must always be taken into consideration when diagnosing young apparently healthy individuals that present for progressive loss of vision, even though it is acknowledged to be more common in HIV positive patients.