Neurological manifestation of systemic diseases

P1871

The effects of oral benfotiamine on peripheral nerve function and inflammatory markers in type-1 diabetes: a 24-month, double-blind, randomised, placebo-controlled trial

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Introduction: Short-term studies (3-6 weeks) in humans have suggested that high-dose benfotiamine (up to 600mg/day) can improve symptomatic scores in diabetic polyneuropathy. To assess the efficacy of long-term supplementation, we have carried out a 24-month, randomised, double-blind, placebo-controlled study to evaluate whether 300mg/day benfotiamine supplementation can influence peripheral nerve function, soluble markers of inflammation and other biochemical variables in patients with type-1 diabetes.

Methods: 67 patients with type-1 diabetes of more than 15 years duration (mean 31 years) were randomly assigned to receive 24-months benfotiamine (300mg/day) or placebo supplementation. Peripheral nerve function and levels of soluble inflammatory variables were assessed at baseline and after 24 months. Tests of peripheral nerve function included nerve conduction studies (including F-waves) of peroneal, tibial, and sural nerves, as well as heart rate response to deep breathing.

Results: 59 patients completed the study. Marked increases in whole-blood concentrations of thiamine and thiamine diphosphate were found in the benfotiamine group (both p<0.001 as compared to placebo). However, no significant differences in changes between the groups in either peripheral nerve function or soluble inflammatory biomarkers were observed.

Conclusion: Our findings suggest that high-dose benfotiamine (300mg/day) supplementation over 24 months has no significant effects upon peripheral nerve function or soluble markers of inflammation in patients with type-1 diabetes.

P1872

Rendu-Osler-Weber disease with hepatic systemic shunting may lead to cerebral manganese accumulation: clinical, neuropsychological and radiological findings

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Introduction: Rendu-Osler-Weber (ROW), or hereditary haemorrhagic telangiectasia (HHT) is associated with arteriovenous malformations (AVM) in various organs, including porto-systemic shunting with neurological signs and manganese accumulation in the basal ganglia. There are hardly any reports on the relationship between neurological symptoms with cognitive changes and neuroradiological findings of manganese accumulation in ROW.

Methods: 2 patients with previously diagnosed ROW were tested on tremor, myoclonus, dystonia, hypo- and bradykinesia, rigidity, speech and walking difficulties. Extensive neuropsychological testing included Stroop colour and Word test, subtests of Wechsler Adult Intelligence Scale-III, Number connecting test A and B, Wisconsin card-sorting test and Rey-Osterrieth complex figure. Blood chemistry, CT imaging of abdomen and MRI of brain were performed.

Results: Patient #1 showed an abdominal AVM at the inferior mesenterical vein, and patient #2 had an intrahepatic AVM with enlarged portal veins. Detailed neurological examination revealed no abnormalities in #1; #2 showed a mild postural tremor and discrete facial hypokinesia. Neuropsychological testing showed moderate attention dysfunction in #1 and normal values in #2. Hepatic function was normal in both patients. MRI markedly showed hyperintense T1 signal at the globus pallidus in both patients suggesting manganese accumulation; the pallidal index was 140 and 166, in #1 and #2, respectively.

Conclusion: Hepatic AVM leading to cerebral accumulation of manganese results in a hyperintense T1 signal at the basal ganglia with a variable severity of extrapyramidal, cognitive or behavioural abnormalities. The underlying pathophysiological mechanism of manganese storage in the brain and the effects on neurotransmitter function will be discussed.
P1873

Drosophila Parkinson’s mutant displays olfactory impairment both to natural and synthetic compounds

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Introduction: Parkinson’s disease (PD) is the most common degenerative disorder characterized by the clinical triad: tremor, akinesia and rigidity. Recently, several studies suggested that PD patients, at their very first onset, show disturbance of olfaction which, since not restrained to PD, has recently received attention in several studies. The fruit fly is a powerful model organism for studying neuronal dysfunction and loss that proceeds from neurodegenerative diseases. The Drosophila genetic model of PD (Dmel/Pink1 B9) displays the two most important diagnostic criteria of the disease: rigidity and akinetic behaviour.

In this study the olfactory sensitivity of the PD flies has been investigated for the first time.

Methods: Electrophysiological recordings have been obtained in response to natural volatiles and synthetic compounds from groups of PD flies in their early stage of their life cycle (3-10 days) and in a group at the end of it (15-30 days). The results have been compared with the same age-groups of wild type flies.

Results: In both age-groups of PD flies, a decrease in olfactory response has been measured in respect to the wild type flies tested to natural substances and to Hexanol and α-pinene among the synthetic volatiles. Morphological, an analysis of neuro-anatomical correlates is in progress.

Conclusions: Our result demonstrates that PD flies show olfactory impairment and suggest a good tool for a new approach for studying Parkinson’s disease and may greatly contributes to better underline the development of preclinical strategies to treat it.

P1874

MRI findings and outcome of neuro-Behçet’s disease: the predictive factors

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Introduction: Initial MRI findings of patients with neuro-Behçet’s disease can predict unfavourable outcomes and course of disease in these patients.

Methods: All the consecutive patients referred from 2002 to 2009 to Behçet Clinic at Nemazee Hospital, Shiraz, Iran, who fulfilled ISG criteria for Behçet’s disease and diagnosed as neuro-Behçet’s disease, were enrolled in this study. Characteristics of initial brain MRI were studied in patients with different courses of neuro-Behçet’s disease.

Results: Initial MRIs of 58 patients (31 women) with a mean ±SD age of 38.9±9.7 years were reviewed. 49 (84%) patients had parenchymal and 9 (16%) had non-parenchymal neuro-Behçet’s disease. Of those patients with parenchymal neuro-Behçet’s disease, 15 (31%) had monophasic, 13 (27%) polyphasic, and 10 (20%) had progressive courses; 11 (22%) had only headache attributed to Behçet’s disease. The most common sites of involvement in patients with parenchymal neuro-Behçet’s disease were periventricular and superficial cerebral white matter, midbrain and pons, respectively. Of those with parenchymal involvement, 12 (24%) had extension of lesions, 7 (14%) had contrast enhancement, 12 (24%) had black holes and 5 (10%) had brainstem atrophy. Patients with the progressive course had a significantly (p=0.017; OR=18, 95% CI: 1.7-19.1) higher rate of brainstem atrophy than those with non-progressive course (monophasic or polyphasic).

Conclusion: Presence of brainstem atrophy in the initial MRIs may predict a progressive course in patients with neuro-Behçet’s disease.
**P1875**

**Thymoma-associated cerebellitis: the early stage of a paraneoplastic cerebellar degeneration?**

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**Background:** Thymoma are infrequent causes of paraneoplastic cerebellar degeneration (PCD). In PCD, an initial oedematous phase has rarely been observed. We report a case of subacute cerebellitis associated with a thymoma.

**Case report:** A 34-year-old woman was admitted following a 1-month history of mild fever, headache, dizziness, vomiting and progressive gait imbalance. She was confused, with a horizontal nystagmus in all gaze positions and mild limb but severe truncal and gait ataxia. A traumatic lumbar puncture showed 249 cells/mm³ (predominantly lymphocytes) and 117mg/dL proteins. Brain MRI showed diffuse symmetrical swelling of the cerebellum, with cortical-pial enhancement and compression of the fourth ventricle. Acyclovir was administered, but there was progressive clinical deterioration. PCR analysis for neurotrophic virus in CSF was negative. Serum viral serologies were unremarkable. Body CT scan revealed an anterior mediastinal mass, which proved to be a thymoma after biopsy. An extensive screening for anti-neuronal antibodies was negative. The patient underwent surgical removal of grade II thymoma, with a striking clinical improvement in the following days. A follow-up MRI documented radiological resolution. A second lumbar puncture revealed 30 cells/mm³.

**Discussion:** We report a case of cerebellitis associated with a thymoma. The impressive clinical and radiological improvement immediately after tumour removal, and an extensive investigation negative for infectious causes being consistent with an immunological paraneoplastic response. The aspect of oedematous cerebellitis possibly corresponds to an early phase of PCD, previously rarely observed. Moreover, this patient evolution suggests that this may be a still reversible stage of PCD.

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**P1876**

**Consequences of an impaired cholinergic system in the CNS and at the periphery**

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**Introduction:** The cholinergic system in brain is believed to be in control of some major physiological functions and its dysfunction leads to severe pathologies, e.g., Alzheimer’s disease, Parkinson’s disease and Huntington’s chorea. Moreover, a mechanism by which cholinesterase (ChE) inhibitors improve cognition in dementia is explained by enhancement of cholinergic transmission in the brain. Here we present our recent results from genetic studies in mice that contrast with recent views on the cholinergic system in the brain.

**Methods:** Genetically modified mouse models were used in which acetylcholinesterase is absent in the whole animal (AChE-/-), only soluble monomer AChE is present (E5,6-/-), or ChE are locally omitted predominantly in CNS (PRiMA-/-) or at the neuromuscular junction (ColQ-/-). Histochemical and biochemical analyses of cholinergic markers were performed. Phenotype and behavioural test results were compared with wild-type littermates.

**Results:** In AChE-/-, PRiMA-/- and E5,6-/- mice, we confirmed high levels of acetylcholine in brain resulting from similar acetylcholinesterase deficiency in the CNS. This was accompanied with severe changes in the number of acetylcholine receptors. AChE-/- mice developed a dramatically changed phenotype (including motor tremor, lower body weight, seizures and impaired thermoregulation). E5,6-/- mice had a similar but less profound appearance. However, PRiMA-/- mice were indistinguishable from the wild-type littermates. On the other hand, changes in the phenotype of ColQ-/- mice were severe and resembled the phenotype of AChE-/- and E5,6-/- mice.

**Conclusion:** Our results suggest that some actions of the cholinergic system may arise from the periphery, rather than from the CNS as believed nowadays.
P1877
Central nervous system involvement in primary Sjögren’s syndrome

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Introduction: Primary Sjögren’s syndrome (pSS) is a systemic autoimmune disease characterized by salivary/lacrimal glands lymphocytic infiltration and auto-antibody secretion, excluding other disorders. The burden of central nervous system (CNS) involvement is controversial. We performed an observational retrospective cross-sectional case-control study to evaluate prevalence, clinical patterns and outcomes of CNS involvement in a cohort of pSS patients followed in our centre.

Methods: We studied the CNS involvement in 91 pSS patients (88 females, 3 male). Mean age was 47.6 years. Diagnosis was established according to the criteria of the American-European Consensus Group. Demographic, clinical, immunological data were assessed.

Results: Nervous system involvement was detected in 24 patients (26.4%), of which 12 (13%) had CNS involvement. All were women with mean age at disease onset and neurological onset of 40 and 44 years, respectively. Neurological syndromes found were: seizures in 2 patients, motor and sensory deficits in 2, movement disorders in 2, chronic progressive myelitis in 1, chronic progressive myelitis and dementia in 1, aseptic meningitis in 1, and headache and dementia in 1. From the 24 patients with neurological involvement, 12 had headaches: 9 migraine and 3 tension type headache. From those, only two with magnetic resonance abnormalities compatible with inflammatory disease were included. Neurological involvement was the initial manifestation in 8 (75%). The CNS involvement outcome was good in 10 (83%).

Conclusion: Our study showed great heterogeneity of CNS involvement in pSS which managed to be frequently the initial manifestation of pSS, as had been found in previous studies. The prevalence was much higher than the majority of the series in the literature.

P1878
A retrospective study of neuro-Behçet’s disease

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Introduction: Neurological manifestations in Behçet’s disease represent between 4 to 49% of systemic manifestations and remain the leading cause of morbidity and mortality.

Aim of study: This report describes clinical features, therapeutic aspects and follow-up of neuro-Behçet’s disease.

Methods: Retrospective series of 56 neuro-Behçet cases fulfilling the International Study Group criteria for Behçet’s disease were consecutively recruited over a period from June 2004 to December 2010. All patients had clinical and ophthalmologic examinations; they underwent laboratory and imaging investigations. Patients with severe conditions (parenchymal involvement and cerebral deep venous thrombosis) received cyclophosphamide and corticosteroids. The other patients received only corticosteroids. Anticoagulant therapy was given to patients with cerebral venous thrombosis. Patients’ follow-up and tolerance to treatment were analyzed.

Results: The average age at diagnosis was 34±12 years, with a sex ratio of 1.15. The clinical presentation was dominated by meningo-encephalitis (50.9%), cerebral deep venous thrombosis (43.4%) and myelopathy (5.7%). Of the 56, 16 patients were treated by corticosteroids and 40 patients received cyclophosphamide associated with corticosteroids. All patients, despite two aggravated cases, evolved positively with clinical improvement and good tolerance.

Conclusion: The demographic and clinical aspects of our series are similar to those reported in the literature. In contrast to previously reported cases of a poor prognosis in severe neuro-Behçet’s disease, our study suggests that immediate and aggressive treatment by cyclophosphamide may ameliorate the prognosis. However, a multicenter study is needed to confirm the possible efficacy of cyclophosphamide and to further assess the long-term tolerance.
P1879

Astrocytes are sensitive indicators of hyperthermia-induced brain oedema in normal and in Cu nanoparticles treated rats

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Whole body hyperthermia (WBH) induces breakdown of the blood-brain barrier (BBB), oedema formation and brain pathology. However, hyperthermia induced damage to glial cells are still not well documented. The glial cells actively regulate the brain fluid micro-environment and maintain the BBB function. Thus, it is likely that hyperthermia will influence astrocytic reaction in WBH. Previous studies in a rat model of WBH exhibited profound BBB disruption, oedema formation and brain pathology. In the present investigation, we examined the role of astrocytic activation following WBH in rats using glial fibrillary acidic protein (GFAP) immunoreactivity. Furthermore, influence of copper (Cu) nanoparticles (CuNPs 50-60nm) on WBH induced GFAP immunoreactivity was also investigated. Rats subjected to WBH (38°C for 4h) resulted in massive increase in GFAP immunoreactivity in the thalamus followed by hypothalamus, pons, brain stem, cerebellum, hippocampus, caudate nucleus and cerebral cortex. Pre-treatment with CuNPs (50mg/kg, i.p. for 1 week) exacerbated GFAP immunoreactivity in all the brain areas examined. This activation of astrocytes in WBH was largely seen in areas exhibiting BBB disruption and brain oedema formation in normal or nanoparticle treated rats. These observations suggest that astrocytes are sensitive indicators of thermal brain injury, not reported earlier.

P1880

Neurological status assessment in patients after orthotopic liver transplantation

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Background: Neurological disorders are the commonest reason of life quality decreasing in patients after orthotopic liver transplantation (OLT). The actuality of the problem depends on the increase of the amount of OLT in Belarus.

Objective: To estimate the neurological disorders intensity in patients after OLT.

Methods: 20 patients after OLT were investigated, treated in RSPC “Organs and Tissue Transplantation”, 10 men and 10 women, mean age 39.6±12 (20-59 years), Body mass Index (BMI) - 23.1±4.2kgm², basic disease duration 3.4±4.8 years; the period they were listed 4±9 months; the follow up - 12±11 months.

Neurophysiological testing: Mini-Mental State Examination (MMSE), Frontal Assessment Battery (FAB), Hospital Anxiety and Depression Scale (HADS); Paced Auditory Serial Addition Test 3” (PASAT 3”).

The neuropathy intensity was assessed by Neuropathy Disability Score (NDS).

Results: The mean indicator on NDS was 6.4±3.7. The mild cognitive disorders were revealed in 6 patients on MMSE 24.7±1.8, on FAB 14.4±1.5. The operative memory decrease (PASAT 3” 27.5±14.1) presented in 8 patients. Depression and anxiety presented in 13 patients (HADS 10.1±4.5). The correlation between neurological disorders intensity and depression-anxiety manifestations (p<0.05), aged >40 y.o., gender (mostly women), BMI >25kg/m², basic disease duration >5 years, the period they were listed >6 months, the follow-up >5 months were registered.

Conclusions: In neurophysiological testing prominent operative memory decreasing, depression and anxiety presence was revealed. In neurological status the distal symmetrical sensorimotor polyneuropathy of moderate stage was present. The obtained results may prove the neurological disorder progression.
P1881
Reversible injury of the corticospinal tract with decreased perfusion-weighted magnetic resonance imaging in a patient with transient hypoglycaemic hemiparesis
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Profound hypoglycaemia has diverse neurological manifestations from headache to coma and death. Transient hypoglycaemic hemiparesis (THH) is the rarely reported complication of hypoglycaemia, usually misdiagnosed as ischemic stroke. We report a patient with THH whose diffusion-weighted image (DWI) and apparent diffusion co-efficient (ADC) values showed reversible lesions in the corticospinal tract from the internal capsule to the midbrain. A 75-year-old man with diabetes mellitus and hypertension was admitted to our hospital complaining of right-sided weakness and dysarthria. He was diagnosed with type-2 diabetes 10 years ago and took metformin, voglibose and glimepiride. He was slightly drowsy but communicable. Other neurologic examinations showed right facial palsy and hemiparesis. Initial blood glucose was 34mg/dl. Intravenous dextrose was given to him. Routine laboratory tests were all within normal range. Initial DWI performed at 1.5 hour after symptom onset showed high signal intensity lesions in the left internal capsule and midbrain compatible with the corticospinal tract. ADC values reduced in the same area. Perfusion-weighted image disclosed delayed mean transit time in the posterior limb of the left internal capsule. MR angiography showed no intracranial artery stenosis. Blood sugar was increased to 155mg/dl and his neurologic deficits were fully recovered after MRI. DWI, ADC values and FLAIR image taken 5 days after symptom onset revealed completely resolved lesions. In conclusion, THH seems to be the sequela of reversible cytotoxic oedema. Physicians should consider the possibility of THH when MRI reveals the lesion suggesting ischemic stroke in a patient with hypoglycaemia.

P1882
Use of body-CT and PET-CT in the investigation of paraneoplastic neurological syndromes: retrospective audit of practice in a regional neuroscience unit in the UK
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Background: Paraneoplastic neurological syndromes (PNS) are characterised by progressive neurological dysfunction resulting from non-metastatic effects of cancer. Positron emission tomography (PET) has emerged as an effective tool for diagnosis of PNS. An advantage of PET over conventional imaging modalities such as body-CT includes demonstration of metabolically active disease in anatomically normal appearing structures.

Aims: To analyse and improve use of whole-body CT and PET-CT in the investigation of PNS in a regional neuroscience unit.

Methods: Retrospective review of 42 patients with suspected PNS referred for imaging between April 2007 and March 2008 was conducted. The data was presented locally followed by recommendations that a consultant neurologist with interest in PNS reviews; each case before referral and PET-CT is considered, in cases where body-CT is negative. We re-audited 44 patients referred between July 2008 and June 2009 and identified each patient’s final diagnosis along with any additional investigations undertaken.

Results: In the first cycle, 42 patients underwent whole-body CT for investigation of PNS. 4 scans were positive for malignancy and 38 were negative, of which only one was followed up with PET-CT. In the second cycle, whole-body CT was performed in 44 patients, of which 11 subsequently proceeded to PET-CT. 5/44 patients had a final diagnosis of PNS, of which only 3 had undergone PET-CT.

Conclusion: Early PET scanning in clinically suspected PNS may prevent expensive and often unnecessary investigations. Trust guidelines now advise use of PET-CT rather than conventional body-CT in clinically suspected PNS. We aim to re-audit our practice in due course.
P1883
A rare case of embolic stroke due to uterine leiomyosarcoma treated with mechanical thrombectomy

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Introduction: Stroke as a direct consequence of tumoral emboli is rare and mainly described in primary cardiac or lung cancer.

Methods: We present the case of a 59-year-old woman with past medical history of obesity and hypothyroidism who presented with dysarthria, left hemianopsia and left-sided hemiparesis, (NIHSS 10). A CT scan showed no abnormalities and the angioCT demonstrated a right distal internal carotid artery occlusion (TICI 0). Lab test results were normal apart from mild leucocytosis. A mechanical thrombectomy with a Solitaire device was attempted after unsuccessful intravenous thrombolysis. Recanalization of the internal carotid artery was obtained while the middle cerebral artery remained partially occluded (TICI 1).

Results: The histopathological examination of the thrombus was compatible with a mesenchymal sarcomatous tumour. A transoesophageal echocardiography showed a superior right pulmonary vein mass protruding to the left atrium and a toracoabdominal CT showed a heterogeneous, cystic and large (13cm) uterine mass with calcified regions as well as disseminated lung nodules compatible with a metastasic leiomyosarcoma. The MRI showed flux absence in the right middle cerebral artery as well as deep mild hemorrhagic transformation. No clinical changes were seen at the time of discharge (NIHSS 10, mRS 4).

Conclusions: Stroke due to tumour emboli is a very rare complication of systemic cancer. To our knowledge this is the first reported case of a leiomyosarcoma presenting as a cerebrovascular tumoral emboli.

P1884
Marchiafava-Bignami disease, Wernicke's encephalopathy or metronidazole toxicity in a non-alcoholic patient: a diagnostic challenge

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Introduction: Marchiafava-Bignami disease (MBD) is a rare entity, usually associated with chronic alcoholism, characterized by demyelination and necrosis of the corpus callosum. It has been sporadically reported in non-alcoholic patients, especially in the context of malnutrition or cancer, and was also reported in patients with concomitant Wernicke’s encephalopathy (WE). Metronidazole is metabolized into an unstable analogous of thiamine, and can induce an WE-like syndrome.

Case report: 57-year-old female patient, with mucinous cystadenocarcinoma of the appendix previously submitted to right hemicolecotomy and small bowel resection. The patient was under prolonged parenteral nutrition due to surgical complications and chronically treated with high doses of metronidazole. She presented with acute onset of spontaneous vertical nystagmus and dysarthria followed by rapid neurological deterioration, with somnolence, apathy, disorientation, nystagmus in all directions of gaze, paresis of left eye adduction, bilateral pyramidal signs, ataxia and limb hypoesthesia in a stocking-sleeve pattern. T2-weighted MRI showed bilateral symmetric hyperintense lesions in corpus callosum (mainly splenium), deep cerebellar white matter, pons, optic chiasm and optic tracts. EMG showed severe axonal sensory peripheral neuropathy. Replacement therapy with thiamine and folate was started, with prominent clinical and imagiological improvement.

Conclusions: In this case, the clinical picture and MRI findings combine different distinctive features of WE, MBD and Metronidazole toxicity. In selected patients with malabsorption syndromes, WE and MBD may share similar pathogenic pathways to metronidazole toxicity. Given the potential dismal prognosis (especially for MBD), prompt replacement treatment with thiamine should be initiated.
P1885

Intracranial plasmacytoma mimicking meningioma

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Objective: Meningioma is the most common dural neoplasm. However, a variety of tumour entities may present clinically as meningiomas. Even though a significant number of patients with solitary plasmacytomas eventually develop multiple myeloma, intracranial plasmacytoma is exceptional and has been rarely described as primary manifestation of multiple myeloma.

Methods: A 61-year-old man presented with a left occipital skull protrusion. MRI of the head showed a lesion suggestive for intra-osseous meningioma. Intraoperatively, the tumour imposed as meningioma. Histologically, a tumour of high cellularity was encountered. Based on typical findings, a neuropathological diagnosis of plasmacytoma was established. This diagnosis prompted complete skeletal radiographs, which revealed an osteolytic lesion of the eighth rib. Before chemotherapy could be initiated, the patient died of aspergillus pneumonia.

Results: Intracranial plasmacytomas are rare and infrequently diagnosed by imaging due to their resemblance to meningiomas. On MRI, plasmacytomas are often indistinguishable from meningioma, with intermediate signal on T-weighted sequences and isointense signal to grey matter on proton density and T2-weighted sequences. The tumour showed typical histological and immunohistochemical staining of plasmacytoma with positivity for the plasma cell marker CD138 and strong positivity for kappa light chain. Since additionally CD20-expressing lymphocytic tumour cells were also encountered, a diagnosis of “lymphocytic plasmacytoma” was established, which is very rare.

Conclusions: In conclusion, solitary intramedullary plasmacytoma should be considered in the differential diagnosis of meningioma. Conversely, in dural based plasmacytoma lymphocyte rich meningioma must also be considered as a differential diagnosis. In some cases differentiation may be difficult.

P1886

Sjögren’s syndrome and mixed connective tissue disease: a case report and a review of literature

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The occurrence of neurological or psychiatric disturbances in association with rheumatic diseases has been an area of considerable interest for many years. Mixed connective tissue disease (MCTD) is classically considered as an “overlap” of three diseases, systemic lupus erythematosus, scleroderma, and polymyositis that typically have high quantities of antinuclear antibodies (ANAs) and antibodies to ribonucleoprotein (anti-RNP). Sjögren’s Syndrome (SS) is rarely reported in MCTD. We report the case of a 53-year-old woman with no medical history who presented relapsing remitting neurological manifestations for 20 years. The patient presented with memory impairment, gait disorder, paresthesias, arthralgias and worsening of general state. On examination, the patient showed features of cognitive impairment, frontal lobe syndrome, ataxic and spastic gait and left hemiparesis. MRI showed multiple hyperintense lesions of supra and infra tentorial white and grey matter and subcortical atrophy. Cerebrospinal fluid studies showed a mild elevation of protein level. Salivary gland biopsy, salivary gland scintigraphy and ophthalmologic examination showed features consistent with SS. ANAs and anti Scl 70 antibodies were increased whereas anti SSa/SSb, rheumatoid factor and anti DNA were negative. MCTD with predominating features of SS was diagnosed. The patient received methylprednisone intravenously for three days and cyclophosphamide monthly. A mild improvement in cognitive and motor performances was noticed. MCTD is a rare autoimmune disorder and cause of “treatable dementia”. SS is rarely reported in MCTD. Neuropsychiatric features may be the first manifestations of the disease. Symptoms eventually evolve to become dominated by features of one of the component illnesses.
P1887
Catastrophic anti-phospholipid syndrome: analysis of a clinical and morphological case
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Introduction: Catastrophic APS - a rare form of primary anti-phospholipid syndrome (APS) is associated with autoimmune thrombocytopenies. There are multiple thromboses of arteries and veins of different localization.

Materials and methods: 43 patients with primary APS (26 female and 17 male, mean age 38.7±9.7) were observed at the neurological department Hospital № 5 from 2001 to 2011. Diagnosis was confirmed accordingly with universally adopted International Criteria (Sydney, 2006). Macro- and microscopic examination was performed in case of autopsy.

Results: The main manifestation of primary APS was acute vascular lesions of the brain caused by thrombosis. It is displayed as stroke (73.1%). In case of a female, 36 years old, with an obstetric history (miscarriage) we observed catastrophic APS, which was affected by multiple thromboses: skin lesions (bed sores, haemorrhage), lung (heart attacks in both lungs), heart (mitral valve vegetation in the diffuse cardio), oesophagus (multiple erosions and ulcers over), colon (paresis), brain (haemorrhage, and multifocal infarcts in both hemispheres), which ended lethally. Microvascular thrombi of varying degree of limitation were found on microscopic examination, a part of recanalization without inflammatory infiltration of vessel walls.

Conclusion: Catastrophic APS develops acutely, manifests multiple systemic thromboses and has a poor prognosis. Macro- and microscopic results are crucial in its diagnosis.

P1888
Clinical manifestations and imaging findings in posterior reversible encephalopathy syndrome
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Objectives: Posterior reversible encephalopathy syndrome (PRES) is typically characterized as symmetric and reversible oedema of the posterior cerebral hemisphere with variable symptoms. This study determines the incidence of clinical and MRI imaging manifestations of PRES.

Materials and methods: We included 16 patients who were diagnosed with PRES, from October 1, 2003, through February 28, 2012, based on clinical manifestations and MRI findings. We performed a review of clinical information, including cause of PRES, presenting symptoms, and imaging findings. The lesions were recorded on the basis of FLAIR findings. As atypical imaging findings, we included gadolinium enhancement, restricted diffusion, haemorrhage, and unilateral or irreversible lesions.

Results: The aetiologies of PRES were hypertension, 43.8%; chronic kidney disease, 25.0%; eclampsia, 12.5%; multiple organ dysfunction, 6.2%; and unclear cause, 12.5%. The main presenting symptoms were seizure, 43.8%; encephalopathy, 68.8%; headache, 31.2%; dizziness, 18.8%; and visual disturbance, 18.8%. The incidence of the regions of involvement was parieto-occipital, 87.5%; posterior frontal, 62.5%; temporal, 50.0%; brainstem, 37.5%; cerebellar, 43.8%; thalamus, 37.5%; basal ganglia, 31.2%; and corpus callosum 18.8%. The incidence of atypical MRI manifestations were gadolinium enhancement, 50.0%; restricted diffusion, 31.3%; haemorrhage, 12.5%; unilateral lesion, 0%; and irreversible lesion, 41.7%.

Conclusions: Even though it is somewhat hard to accept the result of this study as an established fact because of the small number of cases, the result suggests that atypical MRI manifestations of PRES, contrast enhancement, were more frequent than that commonly perceived in the previous literature.
P1889

Neurological complications of Celiac disease: a report on 4 patients

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Introduction: Celiac disease (CD) is an auto-immune gastro-intestinal disorder occurring in genetically susceptible individuals triggered by the ingestion of gluten and characterized by the presence of anti-transglutaminase 2 and anti-gliadin antibodies. Clinical manifestations are various. Neurological involvements of CD are rare. The objective was to determine the clinical, para clinical, therapeutic and the outcome of patients with CD with neurological involvements.

Methods: A 19-year retrospective study, including all patients followed at the neurology departments in Sfax, Tunisia for CD with neurological involvements.

Results: We collected 4 patients with a female predominance (sex ratio: 0.3). The average age of onset of neurological involvements was 20 years (range 7 to 36 years). Neurological manifestation was inaugural in one case. It was peripheral nerve damage in one case and central nervous system (CNS) in 3 patients (cerebellar ataxia, seizures and cerebral venous thrombosis). All patients received a gluten-free diet (GFD) with a favourable outcome.

Conclusion: Neurological complications of CD are various and can especially be revealing CD in adults. Their pathophysiological mechanisms are still uncertain. They may affect the CNS (often cerebellar ataxia and/or seizures). Peripheral neuropathy is common. On brain imaging, calcification and white matter signal abnormalities are most often described. The introduction of a gluten-free diet often improves clinical symptoms.

P1890

A case of flaccid paraparesis caused by intestinal Taeniasis

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Introduction: Taeniasis is an uncommon parasitic infection in Romania. It usually has unspecific symptoms: abdominal colic, constipation or diarrhoea, anal itching, headache and nervous hyper excitability.

Objective: We present a case with paraparesis diagnosed as nutrition-related myelopathy and polyneuropathy in a nutritional deficiency syndrome caused by intestinal Taenia infection.

Case presentation: A 62-year-old man reports a long standing diarrhoea of more than 1 year duration, following a vegetarian and hypocaloric nutrition regimen. Marked asthenia, as well as pain and numbness in the extremities developed in the following months, while reduction of the muscle strength and gait disturbance occurred in the last 4-5 weeks. Serial and complex medical examination gave unremarkable findings. Pallor of the skin and of the mucous membranes, with furfuraceous desquamation was found on physical examination. The gait was unsteady and of steppage type, as if „walking on pillows”. Impairment of all types of sensibility, distally distributed in both upper and lower limbs, Lhermitte’s sign, absence of deep tendon reflexes and Babinski sign were found, too. Laboratory findings disclosed a mixed type anaemia, an increased number of eosinophiles, and decreased serum levels of iron and B12 vitamin. Repetitive parasitological examination of the stool was negative for several days, but showed Taenia solium infestation after ten successive examinations. Niclosamide, B12 vitamin, folic acid, iron and a well balanced nutrition were started, with rapid improvement of the symptoms.

Conclusion: Malnutrition due to taeniasis resulted in severe anaemia, nutritional deficiency polyneuropathy and myelopathy.
P1891

**Chronic inflammatory demyelinating polyradiculoneuropathy and systemic lupus erythematosus**

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**Introduction:** Chronic inflammatory demyelinating polyradiculoneuropathy is often idiopathic. It is presumed to be caused by an antibody-mediated immunologic reaction and may be associated with other systemic diseases such as systemic lupus erythematosus. A possible causal link between these dysimmune diseases is still a matter of debate.

**Methods:** A woman affected by a chronic inflammatory demyelinating polyradiculoneuropathy associated with systemic lupus erythematosus was selected.

**Results:** We report the case of a 24-year-old woman who developed within a few days a bilateral brachial weakness with paraesthesia in upper limbs. Neurological examination found a bilateral weakness with distal sensory loss predominantly in upper limbs and essentially in the proximal region, associated to areflexia. The CSF contained one white cell with hyperproteinorrachia (1.84g/l). Nerve conduction studies found a demyelinating neuropathy. The diagnosis of a CIDP was retained because the patient was fulfilling the criteria of the American Academy of Neurology. During her hospitalization, malar erythema appeared with lymphopenia and positive tests for antinuclear antibody and antinuclear anti-DNA. The diagnosis of systemic lupus erythematosus was retained on the basis of five criteria of the American College of Rheumatology. The patient had full recovery under steroid therapy.

**Conclusion:** We should consider the diagnosis of systemic lupus erythematosus in case of chronic polyradiculoneuropathy and perform specific immune tests. This case report is atypical because of the clinical presentation with brachial weakness, which is a rare condition.

P1892

**Complicated pyloric stenosis with Wernicke’s encephalopathy**

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**Introduction:** Wernicke’s encephalopathy (WE) is a serious but curable neurologic disease caused by thiamine deficiency. The classical clinical triad of ocular abnormalities, gait ataxia and mental status changes is present in only a subset of patients. WE is commonly associated with alcohol consumption, but any condition (repeated vomiting, prolonged fasting, etc) that induces thiamine deficiency lasting 2 to 3 weeks may lead to WE. We present a case of WE due to pyloric stenosis.

**Methods:** A 55-year-old man developed mental status changes, including drowsiness, confusion and dizziness within four days. The family denied history of alcohol use, but he had a pyloric stenosis. In the last month, the patient complained of gastric pain with vomiting and fasting. Consequently, total parenteral nutrition was required. At neurological examination, he had a vertical nystagmus, VI cranial nerve palsy, signs of cerebellar dysfunction, lethargy and general muscle weakness.

**Results:** Laboratory values were unremarkable. Brain magnetic resonance imaging showed hyperintensive signals in periventricular areas, thalamus and mamillary bodies. Even though the patient’s initial thiamine levels were not determined, WE was diagnosed. Thiamine 300mg daily intravenous was started. After 7 days, his symptoms slowly began to improve, with correction of dizziness and alterations in consciousness. Nystagmus and ophthalmoplegia improved but were not reversed completely.

**Conclusion:** The recognition that WE can occur in non-alcoholic patients and not always being manifested by the classical clinical triad is sometimes forgotten. WE is a neurological emergency and treatment should be initiated promptly to limit morbidity and mortality.
P1893

Septo-optic dysplasia and schizencephaly: a case report
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Introduction: Septo-optic dysplasia (SOD) is an uncommon developmental disorder involving variable midline brain structures, characterized by optic nerve hypoplasia, dysgenesis of septum pellucidum and pituitary-hypothalamic dysfunction with consequent endocrine deficits. The association of septo-optic dysplasia and cortical dysplasia is described as septo-optic dysplasia-plus. Reports on patients with septo-optic dysplasia-plus have been rare. Other distinct features, which occur especially when cerebral cortical abnormalities are also present (SOD-plus), consist of significant generalized developmental delay and/or spastic motor deficits.

Methods: We report a 10-year-old boy with septo-optic dysplasia-plus syndrome, characterized by septo-optic dysplasia with schizencephaly, significant generalized developmental delay, spastic motor deficits and seizure intractable. Extraction of DNA for gene EMX2 is in the process of reporting.

Results: Neuroimaging studies revealed schizencephaly with enormous ex vacuo dilation of the occipital horns of the lateral ventricles that involves medium cells up to the vertex of the brain; the residual frontal cortex of both sides develop a pachigiric aspect, especially in parasagittal regions; the corpus callosum is represented in all its sections but slightly hypoplastic at the rostrum and the splenium. There are also dislocated left femoral head, optic atrophy, deficiency determination and alternating exotropia bilaterally.

Conclusion: Schizencephaly is a distinct congenital cerebral malformation presenting as transcerebral mantle cleft between lateral ventricle and pial surface. Nearly half of the patients with SOD have schizencephaly. When the SOD is associated to open-lip schizencephaly, the subjects have a poor prognosis with respect to psychomotor development and complicating intractable epilepsy.

P1894

The challenges in establishing a positive diagnosis of a seronegative Wegener’s granulomatosis with unusual clinical presentation: a case report
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Wegener’s granulomatosis/GPA (granulomatosis with polyangiitis) is a small vessel pauci-immune necrotizing vasculitis with granulomatous inflammation that classically involves the upper respiratory tract, lungs and kidneys and rarely the nervous system, gastrointestinal tract and heart. A 39-year-old woman known with diabetes and obesity was referred to our department because of a right peripheral facial nerve palsy associated with a peripheral right vestibular syndrome, hearing loss and acute otitis media. After a careful assessment of her pathological history we discovered: a pseudotumor pancreatitis two years before (chronic pancreatic inflammation with no necrosis in the histological exam), a complete heart block requiring a permanent pacing eighteen months before (considered cardiac complication of a Lyme disease after an extended assessment with normal auto-immune serology but positive Lyme serology) and no pulmonary or renal pathology. She presented feverish, painful strawberry gums, saddle-nose deformity, raised dark spots on the ankle and halux, arthralgia and weight loss. Laboratory showed anaemia, leukocytosis, elevated ESR and repeated negative ANCA tests. Histological examination of the mastoid biopsy showed a central necrotic granulomatous process with multinucleated giant cells and necrotizing vasculitis. A final diagnosis of GPA was retained and immunosuppressive therapy with cyclophosphamide and steroids was started. Three months later she developed oligoanuric acute renal failure and died two days later. This uncommon GPA presentation, diagnosed after cranial neuropathy, can result in diagnostic difficulty and may allow a potentially poor outcome. Cranial neuropathies in GPA occur later in severe systemic forms. A negative ANCA test does not eliminate a well-diagnosed GPA.
P1895

Sensorineural hearing loss - an auto-immune sign

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Introduction: Sensorineural hearing loss (SNHL) is an unusual neurological symptom. An auto-immune etiology is rare and its association with systemic auto-immune diseases is neurologically relevant. We describe a patient whose diagnosis of multiple sclerosis (MS) was reviewed after the onset of episodes of hearing loss, leading to the diagnosis of Sjögren’s Syndrome (SS).

Case report: A 59-year-old woman who was diagnosed with MS at 47, after an episode of vertigo/imbalance and brain-MRI revealing supratentorial demyelinating lesions with cerebral atrophy. Interferon-beta1a was started. She remained clinically and imagiologically stable until, at 55, relatively sudden episodes of hearing loss began, first unilateral, then bilateral, initially with spontaneous recovery, and then only with partial response to steroids. Neurological examination was otherwise unremarkable. Audiogram showed a moderate SNHL. CSF study was unremarkable (negative oligoclonal bands) and brain and spinal MRI were unchanged. At 56, she first noticed a dry mouth and “red eyes”. Schirmer test was subnormal. Retinal angiography was normal. Salivary gland scintigraphy showed bilateral enhancement, and minor salivary gland biopsy, although atypical, showed a lymphocytic infiltrate. Immunological study was negative, including anti-SSa/SSb antibodies. After exclusion of secondary causes and a rheumatologist consultation, SS was assumed, and azathioprine plus corticosteroids initiated.

Conclusion: SNHL may be associated with auto-immune disorders of CNS. In the presented case, the recurrent episodes of NSHL became the dominant symptom and thus, a red-flag that led to a diagnosis review. Based on clinical and imagiiological features, SS was assumed, a known MS mimic but with a different therapeutic approach.

P1896

Abstract cancelled

P1897

Acute-onset of critical illness: polyneuropathy and myopathy as a severe complication of percutaneous nephrostolithotomy: a case report

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P1898

Convulsive status epilepticus due to hypoglycaemia as the initial presentation of primary hepatic carcinoma

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P1899

Reversible posterior leuko-encephalopathy syndrome and bilateral renal fibromuscular dysplasia

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Abstract cancelled

P1901

Cerebral venous thrombosis as the initial presentation of Behçet’s disease: a case report

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P1902
Reversible hepatocerebral degeneration due to portovenous shunts: a case report
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P1903
Posterior reversible encephalopathy syndrome as presenting form of systemic sclerosis
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P1904
Central pontine and extrapontine myelinolysis: a case report with assessment of cognitive function
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P1905
Fahr's syndrome supports the involvement of basal ganglia in the pathogenesis of Schizophrenia: a case report
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P1906
Restless legs syndrome in haemodialysis patients: association with anxiety and sexual life
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P1907
Neuromyelitis optica associated with primary Sjögren's syndrome
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P1908
Acute onset cervical myelopathy as first manifestation of late onset SLE (systemic lupus erythematosus)
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P1909
Gluten sensitivity and ataxia
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Abstract cancelled

P1911
Wernicke's encephalopathy following total gastrectomy for gastric neoplasm
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P1912
A neurological presentation of Prinzmetal (variant) angina
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P1913

The clinical significance of brain angioarchitectonic features in Alzheimer's disease

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P1914

Polyradiculoneuritis revealing Behçet's disease

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P1915

Analytic tetraparesis

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P1916

Central and peripheral involvement in a neurosarcoidosis case

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Intracranial vasoreactivity alterations in portal-systemic encephalopathy

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