

## Infections and AIDS

### Neurological manifestations of systemic diseases

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#### Pattern of clinical manifestations in early and late neuroborreliosis

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**Objective** To identify the possible range of nervous system injuries in Lyme borreliosis depending on the duration of disease and differential diagnosis.

**Methods** The study was done in the Scientific and Practical Centre of Neurology and Neurosurgery from the Republic of Moldova on a sample size of 57 patients with average age of 42 years (ranging 16–79 years) selected throughout 1997–2001. The diagnosis was made based upon history data of a tick bite and/or the presence of a migrating erythema and identification of specific antibodies in the serum and CSF whereby indirect immune fluorescence test, ELISA test and Western Blot technique.

**Results** The tick bite was recalled by 36.8% of patients. The migrating erythema was recorded in 28% cases. Cranial mono- and multi-neuritis (43.7%) or peripheral radiculo-neuritis (28.5%) dominated in early neuroborreliosis. The facial nerve was the most often injured one: 49% of all neurological manifestations of Lyme disease and 93% of all cranial nerve impairment. In 12 patients, Bell's palsy was associated with concurrent signs of trigeminal involvement. Axonal distal chronic polyneuropathy and Lyme encephalopathy with 17% each were the most often encountered neurological manifestation in late borreliosis.

**Conclusions** *Borrelia burgdorferi* infection entails multiple lesions at the central and peripheral nervous system level and a differential diagnosis should be made with acute and chronic neurological conditions. Neuroborreliosis might be the first and the only manifestation of the Lyme disease.

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#### Effect of acetyl-L-carnitine on painful neuropathy; HIV related

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**Introduction** Distal sensitive polyneuropathy (DSP) is one common neurological complication during HIV infection. Possible pathogenetic hypothesis are the role of TNF-alpha and the reduced blood levels of endogenous acetylcarnitine. Acetyl-

L-Carnitine (ALC) is a safe and well-tolerated drug in the treatment of DSP in HIV+ patients. ALC supplement may represent a valid therapeutic option.

**Method** Twenty HIV- positive patients affected by neuropathic pain with EMG-evidence of DSP were enrolled. Patients with mini-mental test score, less than 24, acute cytomegalovirus infection, demyelinating neuropathies were excluded. All patients were treated with ALC at the dose of 1 gr t.i.d. for 4 weeks. A visual analogue scale (VAS) was used to evaluate characteristics of patient's pain before, during, and after treatment. EMG assessment was performed before and after treatment.

To evaluate changes in VAS score, we used non-parametric Friedman's test (F). Wilcoxon's test (W) was performed to time the appearance of pain improvement and to evaluate neurophysiological data.

**Results** The changes in VAS score were statistically significant during ALC treatment (mean score: before  $6.7 \pm 2.1$  - after treatment  $5.0 \pm 2.1$ ) ( $F = P < 0.001$ ). The appearance of therapeutical effect was reached during the first week ( $W = P < 0.03$ ) and during the fourth week ( $W = P < 0.05$ ) of treatment.

Neurophysiological data showed a statistically significant improvement of peroneal nerve motor parameters ( $W = P < 0.02$ ).

**Conclusions** Our data show the efficacy of ALC in the treatment of neuropathic pain. The analgesic effects appear during the first week of treatment according with pre-clinical data in different experimental models of pain.

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#### Electromyography characterization of polyneuropathy in the first found tuberculosis

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Affection of nervous system in tuberculosis is now widely known.

The aim of this study was the investigation of the clinical electromyographical features of polyneuropathy in tuberculosis.

615 men with first found and untreated tuberculosis were examined. Among them 107 people (mean age 37) suffered from polyneuropathy. The patients were subjected to neurological, immunological and electromyography examinations. Level of antibodies to the basic protein myelin was determined using immunoenzymatic method. Electromyographical examining consisted of the defining of the velocity of impulse conducting along motor and sensitive nerve, the value of sensory and muscular answers. The control group consisted of 20 people of the same sex and age.

The neurological examining showed the following forms of polyneuropathy: Sensitive (84%), motor (5%) and mixed (11%). Electromyography revealed the decrease of the velocity of impulse conducting along sensitive nerve fibre in all cases ( $32.7 \pm 1.35$  m/s) that reliably differs from results in control groups ( $56.2 \pm 4.8$  m/s,  $p < 0.001$ ). The size of M-answer and sensory were the same as in control group. The delay of impulse conducting is considered to be a sign of demyelination. High level of antibodies to the basic protein myelin is evidence of demyelination pattern of affection in peripheral nerves. That level in main group was a  $0.199 \pm 0.002$  unit of optical density (UOD), and in the control group -  $0.077 \pm 0.01$  UOD.

Therefore, the study showed presence of polyneuropathy in 17.4% of cases in first found tuberculosis. Most of them were manifested in form of sensory polyneuropathy with demyelination character.

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**Role of antibodies to myeloperoxidase in pathogenesis of secondary cerebral vasculitis**

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Secondary cerebral vasculitis can arise in various allergic conditions caused by inflammatory process, drug effect, parasitic infection, paraneoplastic syndrome, etc.

Clinically cerebral vasculitis is manifested in both acute and chronic cerebral insufficiency. However, cerebral vasculitis is rarely diagnosed due to neurologist's little knowledge of this pathology. Antibodies to myeloperoxidase (AM) are a reliable marker of primary systemic vasculitis. The aim of this study was revealing the role of AM in cerebral vasculitis dynamics.

45 patients with secondary cerebral vasculitis were monitored. Of these, the discussed pathology developed on the background: in 15 cases of tuberculosis of the lungs, in 20 cases of drug disease, in 10 cases of a chronic infection of otolaryngologic pathology.

Clinically secondary cerebral vasculitis manifested in the dispersed cerebral microsymptomatology syndrome, focal symptomatology and neurangiosis. In 30% cases ophthalmoscopy showed retinal vasculitis which occurred in presence of "muffs" and oedemas along the vessels. Besides, cerebral vasculitis was combined with general symptoms: asthenia, sweating, sub febrile status.

Study of the level of AM showed its reliable ( $p < 0.001$ ) increase compared to the control group (correspondingly  $2.8 \pm 0.2$  UOD and  $0.08 \pm 0.02$  UOD). AM were determined with the help of immunoenzymatic analysis (testing systems of CLARC company, USA). At that, the degree of neurological symptomatology expressiveness correlated with the level of rising of AM ( $Q = +0.7 \pm 0.02$ ).

Thus, high titers to myeloperoxidase in secondary cerebral vasculitis are evidence of presence of immune inflammation in small vessels including cerebral vessels. The obtained data prove that AM can serve as a secondary cerebral vasculitis marker.

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**Cognitive impairment, depression and quality of life – comparison between coeliac disease patients and reflux disease patients**

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**Background** Association of coeliac disease and dementia has been described. In this study, we evaluated cognitive symptoms, depression, and quality of life among well-treated CD patients on gluten-free diet compared to patients with gastroesophageal reflux disease.

**Methods** The study group consisted of twenty-eight patients with diagnosis of CD and the control group of twenty-seven patients with gastroesophageal reflux disease. All patients were asked for neurological examination. Cognitive status was evaluated by using MMSE and 3MSE scale. Depressive symptoms were measured with Beck depression inventory and general well being with PGWB index.

**Results** 3MSE scale showed cognitive impairment (3MSE < 90) altogether in five (17.9%) out of 28 CD patients and one (3.7%) out of 27 reflux disease patient ( $p = n.s.$ ). MMSE scale showed cognitive impairment (MMSE  $\leq 27$ ) in six (28%) CD patients

and one reflux disease patient ( $p = 0.0485$ ). Correlation between MMSE and 3MSE test results was 0.85 ( $p < 0.001$ ).

Seven (25%) CD and twelve (44%) reflux disease patients were depressive according to Beck inventory, ( $n.s.$ ). Mean of overall general well-being score was 102 with CD patients and 93.7 with reflux disease patients, ( $p = 0.0095$ ).

**Conclusion** CD patients do have increased risk for cognitive impairment compared to reflux disease patients. CD patients do not suffer from depressive symptoms more often than patients with reflux disease. Quality of life with CD patients on gluten free diet is normal.

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**Neurological complications during dialysis**G. Mihailescu<sup>1</sup>, C. Mihailescu<sup>1</sup>, M. Ticmeanu<sup>1</sup>, I. Cojocaru<sup>1</sup>, I. Monda<sup>2</sup><sup>1</sup>UMF Carol Davila, Bucharest, ROMANIA, <sup>2</sup>Colentina Hospital, Bucharest, ROMANIA

Patients with end-stage renal disease eventually require haemodialysis, peritoneal dialysis and/or renal transplantation. Apart from removing unwanted substances from the blood while adding desirable components, using the available dialysis techniques can also induce new neurological conditions.

We performed a retrospective study on 91 patients; neurologically normal at the different start dates of dialysis, receiving haemodialysis (HD) or continuous ambulatory peritoneal dialysis (CAPD). From this group, 31 patients (18 male, 13 female) with the age ranging from 30 to 79 years developed neurological complications in one-year duration. More than one neurological complication occurred in 16% of these patients. In the 10 patients receiving CAPD, the following conditions occurred: cerebral vascular accidents (CVA's) – 6 cases, the majority recognizing an ischemic mechanism, polyneuropathy – 4 cases, uremic encephalopathy – 1 case. In the group receiving HD (21 patients), there have been 4 cases with CVA's, 8 cases with polyneuropathy, encephalopathies (hypertensive – 5 cases, uremic – 1 case, Binswanger – 2 cases, hydro electrolytic disturbances – 2 cases), 2 cases with carpal tunnel syndrome and 2 cases with cerebral atrophy.

Most of the encephalopathies developed in the early years after the start of dialysis, whereas CVA's, polyneuropathies and cerebral atrophy occurred mostly later. A close neurological follow-up of the patients receiving dialysis is therefore justified, the clinical and paraclinical investigations being aimed to avoid irreversible neurological complications and to help the nephrologists move from one form of therapy to another as indicated by the degree of success and incidence of complications with each.

P 1070

**Intramedullary sarcoidosis: two atypical cases**S. Perdigão<sup>1</sup>, S. Pereira<sup>1</sup>, J. Resende<sup>2</sup>, M. Costa<sup>1</sup><sup>1</sup>Neurology Service, Hospital Pedro Hispano, Matosinhos, PORTUGAL, <sup>2</sup>Neuroradiology Service, Hospital Pedro Hispano, Matosinhos, PORTUGAL

**Introduction** Neurological involvement in sarcoidosis appears in 5 to 15% of the cases, and medullary involvement is uncommon. We report two cases of intramedullary sarcoidosis. In the first case, there was no systemic involvement and in the second, ocular and skin involvement accompanied the neurological picture.

**Case Reports** A 41 year-old man presented with a 4-month history of lumbar pain and gait and sphincter disturbances; examination revealed a spastic paraparesis, hyperreflexia, and a sensory level by D4–D6. Serological studies were normal, including ACE. MRI showed diffuse spinal cord enlargement with hyper intense T2 signal between D4–D5 and D10–D11, with contrast enhancement. Spinal biopsy was consistent with sarcoidosis. 2. A 36 year-old man presented with a 2-month history of gait difficulties and two episodes of blurred vision on his left eye during the last year, which improved with topical therapy. Examination revealed lupus pernio in the ears, and a spastic paraparesis with hyperreflexia. Laboratory evaluation detected increased ACE. MRI showed focal spinal cord areas of high signal intensity at cervico-dorsal levels with contrast enhancement. Ear biopsy was consistent with sarcoidosis. The clinical course under corticosteroids was favourable in both cases.

**Conclusions** Intramedullary lesions are infrequently the first manifestation of sarcoidosis, especially when there is no evidence of systemic involvement (case 1). It is also rare in association with lupus pernio and ocular involvement (case 2). The ACE is raised only in case 2, in which systemic manifestations are present. Biopsy is relatively safe and very useful for early diagnosis.

#### P 1071

##### **Investigation of TNF-alpha, IL-6, IL-8 and of procalcitonin in patients with neurological complications in sepsis**

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**Background** Some mediators of inflammation are associated with sepsis, involving nervous system.

**Objective** Investigations of proinflammatory cytokines of TNF- $\alpha$ , IL-6, and IL-8, and of procalcitonin (PCT), proinflammatory protein, in patients with neurological complications in sepsis.

**Material and methods** TNF- $\alpha$ , IL-6, IL-8, and PCT were prospectively investigated in 62 patients with neurological complications in sepsis. TNF- $\alpha$  and IL-6 were studied both in serum as in the CSF, IL-8 and PCT were studied only in serum. TNF- $\alpha$ , IL-6, and IL-8 were studied by ELISA (R & D Systems), and the PCT by immunoluminometric assay (BRAHMS).

**Results** Mean value of TNF- $\alpha$  in serum was  $578 \pm 214$  pg/ml, and in CSF was  $458 \pm 167$  pg/ml ( $p < 0.01$ ). Mean value of IL-6 in serum was  $749 \pm 213$  pg/ml, and in CSF was  $617.5 \pm 182$  pg/ml ( $p < 0.01$ ). Mean value of IL-8 in serum was  $332 \pm 196$  pg/ml ( $p < 0.01$ ). Mean value of PCT in serum was  $80 \pm 16$  ng/ml ( $p < 0.01$ ).

**Conclusions** The investigated parameters do not permit the identifying of cases with neurological complications. The increased correlation coefficient between cytokines in serum and in CSF suggests the damage of the blood-brain barrier. The raise of PCT in serum, induced by TNF- $\alpha$  and IL-6, is an argument of the severity of sepsis.

#### P 1072

##### **Therapeutical particularities in patients with cerebral abscess**

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**Background** Modern investigations facilities permit a faster diagnostic of cerebral abscess and the appropriate management.

**Objective** To observe the particularities of some cases with cerebral abscess.

**Material and methods** Retrospective study of 32 cases with cerebral abscess.

**Results** The pathogenesis of the cerebral abscess was: haematogenic—12 cases, during cyanogenic cardiac diseases—7 cases, otogenic—4 cases, synusal—2 cases, post cranio-cerebral trauma—1 case, unknown—6 cases. The organisms were identified in 17 cases. The topography of the abscess was: supratentorial—25 cases, (basal ganglia—7 cases), subtentorial—4 cases, and mixed—3 cases. The number of abscesses was: single—26 cases, multiple—6 cases. The management was neurosurgical approach in 22 cases (excision—7 cases, stereotaxic aspiration—15 cases) and only antibiotic therapy in 10 cases. Evolution: remission—19 cases, 7 with neurological sequelae of medium severity, 6 deaths. The factors associated with high mortality were: coma at the admission, multiple abscesses, and profound abscesses with intraventricular rupture.

**Conclusion** Modern imaging and modern antibiotic therapy allows the abscess cure without neurosurgical intervention in the small abscesses, less than 1 cm. The abscesses with a diameter of 3.5–4 cm, the profound abscesses and those multiple of severe prognosis with high mortality involve stereotaxic attitude and antibiotic therapy.

#### P 1073

##### **Lyme borreliosis (neuroborreliosis) -consideration on clinical and diagnostic problems**

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**Introduction:** Lyme borreliosis (Neuroborreliosis) is most commonly reported tick disease in Europe and America. Infection can be sub clinical or have a range of clinical presentation. The diagnosis should be made after careful evaluation of the clinical history, physical findings, laboratory evidence and exposure risk evaluation.

**Objective** The main goal of the study was to correlate the clinical manifestation of neuroborreliosis and to the other neurological diseases with similar clinical, neuroimaging findings, particularly problems appear in cases with negative serology

**Methods** We present two patients with definite diagnosis of Neuroborreliosis who appeared with atypical clinical manifestation. The other 2 patients who had fulfilled some criteria of MS, but as well with some clinical symptoms of borreliosis, positive oligoclonal bands in CSF, MR with some atypical demyelinating plaque, and negative ELISA. They had a history of being in endemic tick area. Therapy with high doses of methylprednisolone was without significant benefits. We provided therapy with doxycycline and observed that the main neurological deficits were withdrawn.

**Results** Two patients with definite diagnosis of neuroborreliosis, serology positive and other 3 patients with probably diagnosis as well had good response on antibiotic therapy.

**Conclusion** A positive serology is indicative of exposure to the organism and should not be considered proof of active infection. Serological test (ELISA) is further confounded by the evidence of seronegative Lyme cases. The CSF is typically normal in chronic Lyme diseases particularly in polyradiculoneuropathy and encephalopathy-encephalomyelitis. These findings make a differential diagnosis of neuroborreliosis more difficult.

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**Demyelinating encephalopathy in Lyme disease**

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**Introduction** Lyme disease (LD) is an endemic infectious poly-systemic disease. Its causative agent is *B. burgdorferi* Spirochaeta transmitted by ixodes ticks. Neurological manifestations include aseptic meningitis, encephalitis, cerebellar ataxia, neuritis, plexites, and polyradiculoneuritis.

**Methods** We have observed 9 patients with diagnosis of Lyme disease, neuroborreliosis, and demyelinating encephalopathy. The following methods were used: clinical, neurophysiological (EEG, EMG, evoked potentials), MRI, electron microscopy, antibody titres, DNA analysis by PCR method.

**Results** The neurological symptomatology was represented by meningoencephalitis in combination with neuritis of cranial nerves and radiculoneuropathies. The MRI of the brain revealed in the deep compartments of the white matter bilateral hypo intensive foci that was regarded as the demyelinating process. Seven patients had history of migrating erythema, 5 patients had arthritis, all the patients lived in the endemic regions, and therefore in all the patients titers of antibodies of *Borrelia burgdorferi* in the blood serum and liquor were studied. The levels of the specific antibodies in the blood serum were high in all patients and those in the liquor in 5 patients. The DNA assay of both the serum and liquor in all 9 patients yielded positive results for borrelia. The electron microscopy of the muscular tissue found borrelia in 5 patients. The study of evoked potentials (visual, brainstem, somatosensory and cognitive) showed rough changes, mostly at the basal ganglia-cortex level of the cerebral hemispheres. EMG revealed in 8 patients the myopathy-like syndrome.

**Conclusion** Demyelinating encephalopathy is encountered in Lyme disease. High titres of antibodies, detection of borrelia by electron microscopy confirm this.

P 1075

**Atypical form of tick-borne encephalitis: A case report**

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**Background** Tick-Borne Encephalitis (TBE) is a zoonotic arbovirus infection endemic to many European countries and Russia. There are three main clinical forms: febrile, aseptic meningitis and focal neurological form.

**Case history** A 44-year-old male, non-vaccinated, living in endemic area had used unpasteurized milk in May. After 4 days,

he developed fever, deterioration of consciousness, seizures. Neurological examination revealed signs of meningeal irritation, nystagmus, facial (right) palsy, bulbar symptoms, asymmetric quadriplegia, loss of tendon reflexes, respiratory impairment and bladder sphincter involvement.

He had normal routine laboratory investigation. Serological tests were negative for lues, VIH and for *Borrelia burgdorferi*. There were high levels of IgM and IgG antibodies against TBE in serum. CSF study showed 224 cells (neutrophils 16%, lymphocytes 84%). The CSF total protein content was 1,25 gr/dl. The patient was investigated by MRI scan of the brain and EEG. The displayed changes had a non-specific character (moderate hydrocephaly, diffuse disrhythmia). EMG disclosed an axonal sensorimotor polyneuropathy.

Because of the finding of positive serological test for TBE, combined treatment was started. The adjuvant intramuscular immunoglobulin against TBE, plasmapheresis, high-dose (IV) methylprednisolone, intravenous immunoglobulin and respiratory reanimation formed the basis of therapy in an intensive care unit. The patient improved after 3 weeks. He could walk with support 8 months later.

**Conclusion** TBE can have a total form as meningoencephalomyelopolyradiculoneuropathy.

P 1076

**Neurological complication in children with parenteral and perinatal HIV-infection**

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The neurological disorders of HIV-infection is a clinical syndrome complex manifested by varying and sometimes discrepant degrees of cognitive, motor, behavioural impairment.

We studied 125 parenteral and 31 perinatal HIV-infected children.

**Methods** clinical and neurological data, results of electroencephalography, magnetic resonance imaging.

**Results** 76 parenteral HIV-infection children in stage AIDS have AIDS-dementia complex; 31 – decrease memory and attention, 18 – distal polyneuropathy. All perinatal HIV-infected infants retarded of psychomotor development, 8 patients have a progressive encephalopathy.

**Conclusion** Progressive AIDS-dementia-complex – is a more frequent neurological syndrome in children.

P 1077

**Functional state of the blood-brain barrier in patients with herpes zoster ganglionitis**

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Herpes zoster virus persists in cranial and spinal ganglia and under unfavourable conditions reactivates and causes ganglionitis.

We studied 62 patients with Herpes zoster ganglionitis aged 17-84 years with 36 females among them. Cerebrospinal fluid (CSF) was examined in 34 (54.8%) patients. The functional state of the blood-brain barrier (BBB) was assessed using albumin index (AI):  $AI = A_{CSF} / A_{blood} * 1000$ .

15 patients (24.2%) had signs of general infection in the prodromal period, 30 patients (48.4%) had pain syndrome and 17 patients (27.4%) had skin manifestations. The patients were divided into 2 groups subject to the BBB functional state. The

first group included 12 patients (35.3%), average age 65 years, with the moderate impairment of BBB permeability (8–25 units). The prevailing concurrent diseases in this group were coronary heart disease, arterial hypertension, and diabetes mellitus. CSF total protein level was in the range of 0.6–1.2 g/l ( $X=0.83\pm 0.06$  g/l), cytosis was 2–330 cells/l ( $X=35.4\pm 10.5$  cells/l). It was increased in 7 (58.3%) patients. 5 patients (41.6%) developed complications: keratouveitis, postherpetic neuralgia. BBB function (3.6–7.8 units) was preserved in 22 patients (64.7%), average age 42 years, from the second group. CSF total protein level was in the range of 0.18–0.6 g/l ( $X=0.35\pm 0.02$  g/l). Cytosis was increased in 12 patients (54.5%),  $X=72.9\pm 28.6$  cells/l. Benign course of the disease, complete recovery and no complications were typical for the patients from this group.

The severity of herpes zoster ganglionitis is subject to the patient's age, concurrent cardio-vascular disease and BBB functional state.

#### P 1078

##### Rare case of recurrent narcotising myelitis episodes from viral infection

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The rate of onset an inflammatory damage in spinal cord may be acute or sub acute and the process often ascends or spreads transversely, and acute or sub acute cord lesions may occur that exactly mimic transverse myelitis. Usually the spinal lesion does not repair, but the distinction may not be easy in the acute stage.

A 40-year-old female patient developed severe inflammation of the thoracic cord with severe pain, areflexic paraplegia and loss of sphincter control

The spinal fluid showed two cells. The total protein proportion <20%. Oligoclonal bands normal. Glucose concentration normal. Magnetic resonance imaging: thoracic cord swelling, a large area (T3–T9) necrosis.

Complete recovery occurred after treatment with high dose intravenous methyl-prednisolone and acyclovir.

Two years later, she developed the same neurological symptoms 15 days after the cutaneous rash of herpes zoster. MRI: spinal demyelination (T7). Complete recovery occurred after the same treatment.

The diagnosis of transverse myelitis is often made by exclusion and the precipitating cause is often not identified. In our case, the cutaneous rash and the similar clinical and pathological findings during the two episodes signify that it is about herpes zoster myelitis.

#### P 1079

##### Splenic lymphoma presenting as progressive multifocal leukoencephalopathy (PML)

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**Introduction** Until the appearance of AIDS, PML was considered a rare disease occurring in middle-aged or elderly persons as a terminal complication of chronic lymphatic leukaemia or other lymphoproliferative diseases.

**Case report** A 51 year-old woman presented with a six-month history of humour and personality changes, followed by progressive visual loss. On admission she was alert, oriented and without mental impairment. She was unable to establish visual contact and could only vaguely distinguish movement and light. The pupils were midsize, equal and slowly reactive to light. No other neurological signs were found. General physical examination was normal. The brain MR showed bilateral and confluent hyper intense lesions on T2-weighted images at both parieto-occipital areas. The CSF cytochemical profile was normal, but PCR for the JC virus was positive. HIV infection was ruled out. Subsequent investigation with thoraco-abdominal CT, lymphocyte immunophenotyping, immunoglobulin pattern and bone marrow biopsy, led to the diagnosis of splenic lymphoma with bone-marrow invasion and severe cellular and humoral immunosuppression. She was readmitted two weeks later with a respiratory infection, severe anaemia and cognitive impairment. She died soon after, eight months after the first symptoms.

**Conclusions** This woman's six months progressive disease and brain MR abnormalities were very suggestive of PML. The CSF PCR for JC virus is highly specific and establishes the diagnosis, nowadays rarely dissociated with HIV infection. This is a peculiar form of presentation of a relatively indolent lymphoproliferative malignancy. The literature is scarce regarding the management of non-HIV associated PML.

#### P 1080

##### Cerebral Whipple's disease without gastrointestinal symptoms

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**Background** Whipple's disease (WD) is a rare systemic bacterial infection caused by *Tropheryma whippelii*. Brain manifestations comprise slowly progressive dementia, supranuclear ophthalmoplegia, ataxia, hypothalamic dysfunction and oculomasticatoryskeletal myorhythmia. Cerebral WD treatment should be performed with antibiotics that reach the central nervous system (CNS) for at the least one-year.

**Clinical case** A 40-year-old male started in 1999 a slowly progressive encephalopathic clinical picture with abulia, mild to moderate cognitive dysfunction, bilateral ophthalmoplegia, dysarthria, dysphonia, facial and superior limbs refractory myorhythmia, mild ataxia and loss of weight (although no intestinal symptoms nor clinical malabsorption syndrome), and lately rhythmic adducting ocular movements. Copper metabolism and CSF study were normal (14.3.3 protein was negative). EEG showed non-specific slowing and sometimes-periodic complexes. Brain magnetic resonance showed non-specific lesions mainly on the ganglionic region. Muscle biopsy with respiratory chain study was normal, as was the genetic study for dominant ataxias. Diagnosis of WD was made 2.5 years after the first complaints, with a duodenal biopsy disclosing numerous PAS-positive foamy macrophages. The patient was started on intravenous ceftriaxone for 15 days followed by a long-term treatment with oral trimethoprim-sulfamethoxazole, but has only shown a partial regression of the symptoms.

**Conclusion** WD is rare but even rarer is the CNS involvement without gastrointestinal symptoms. The clinical presentation and the findings encountered in the duodenal biopsy suggest

that this entity. This case alerts for the earlier recognition of WD in patients with exclusively neurological manifestations because it is a potential treatable disease.

P 1081

**Pathogenic mechanism of neurosensory bradyacusia in tuberculosis**

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Patients suffering from lung tuberculosis can develop neurosensory bradyacusia whose cause is commonly regarded as ototoxic effect of antituberculosis drugs. However, in some cases this lesion can occur in patients with first found lung tuberculosis and cannot be explained by drug ototoxicity. In this study, we analyse pathogenic mechanisms of neurosensory bradyacusia in first found tuberculosis.

107 patients with first found and untreated tuberculosis aging from 20 to 51 (mean age—37) were monitored. All patients were subjected to detailed neurological and otoneurological examination. Besides, basophile specific damaging response (BSDR) and antibodies to myeloperoxidase were determined. The control group consisted of 20 persons.

Neurosensory bradyacusia of different degrees of severity was observed in 29 (27.1%) cases. All patients showed multiple cerebral and focal neurological symptomatology, neuroangiomas, and polyneuritis. Immune tests showed sensibilisation to tubercle bacillus. Thus, tuberculin BSDR in the main group was  $18.3 \pm 0.67\%$  compared to  $5.2 \pm 0.63$  in the control group ( $p < 0.001$ ). At the same time, the degree of hearing impairment correlated with the level of tuberculin allergization ( $R = 0.66 \pm 0.002$ ). High level of antibodies to myeloperoxidase was found in all cases ( $1.3 \pm 0.02$  ODU compared to  $0.08 \pm 0.01$  in the control group,  $p < 0.001$ ). This evidences of vasculitis with affection of small vessels. Thus, bradyacusia in cases of first found tuberculosis was caused by an allergic factor as a result of sensibilisation to tubercle bacillus. Bradyacusia in tuberculosis may be caused by vasculitis, for vessels and connecting tissues are involved in the allergic process.

P 1082

**The affection of nervous system in tuberculosis**

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Presently we are observing considerable increase of nervous system affection in tuberculosis. However, the pathogenesis of neurological disorders has not yet been subjected to a thorough analysis. The aim of this study was to investigate the pathogenic mechanisms of neurological disorders in tuberculosis.

260 patients with primary pulmonary tuberculosis were examined. Along with clinical and neurological examination we determined level of antibodies to myeloperoxidase, anti-DNA, antibodies to basic protein myelin, performed the test of specific alteration of basophiles with tuberculin.

The following neurological syndromes were observed: focal lesion (8%), disseminated microsymptoms (43%), vegetative dystonia (31%) and polyneuropathy (18%). In all patients we observed positive test of specific alteration of basophiles with tuberculin— $16.47 \pm 0.36$ , in control group— $5.21 \pm 0.63$  ( $p < 0.001$ ). The level of antibodies to myeloperoxidase was  $1.93 \pm 0.084$ , in control group— $0.62 \pm 0.03$  ( $p < 0.001$ ), anti-DNA was  $0.321 \pm 0.048$ , in control group— $0.196 \pm 0.008$  ( $p < 0.001$ ), the level of antibodies to basic myelin protein was  $0.221 \pm 0.01$ , in control group— $0.077 \pm 0.01$  ( $p < 0.001$ ).

There was correlation between the degree of nervous system affection and the increase of titre of test of specific alteration of basophiles with tuberculin and rest of the tests.

The obtained data evidence that the patients with tuberculosis and pathology of nervous system have allergic status. The clinical picture of neural pathology in tuberculosis presents itself in forms of allergic cerebral vasculitis with focal lesions, disseminated microsymptoms and primary symptoms of insufficiency of cerebral haemodynamics, polyneuropathy, which is caused by vasculitis of peripheral vessels.

P1083

**Peduncular hallucinosis in a patient with brain metastases**

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**Introduction** Peduncular hallucinosis represents a rare cause of complex visual hallucinations. The content of these hallucinations is striking and fairly stereotyped, involving animals and human figures in bright colours and dramatic settings. Peduncular hallucinosis manifests itself usually in lesions of vascular nature, which are commonly localized in the rostral brainstem. These lesions are thought to impair the fidelity of the retino-geniculo-cortical transmission by affecting the ascending reticular formation. To our knowledge, the characteristics of peduncular hallucinosis have not been previously described as the presenting feature of metastatic carcinoma of the brain.

**Patient and methods** A previously healthy 53-year old man developed over a period of two weeks complex visual hallucinations; consisting of human figures in vivid colours, cartoon animals moving near the bed of the patient and flowers of outstanding beauty. Insight was preserved. The patient developed ataxia of gait a few days later. A computed tomography of the brain demonstrated multiple lesions in the cerebral hemispheres and the posterior cranial fossa, while brainstem structures were compressed by the mass effect produced by a large lesion in the cerebellar vermis. Following treatment with dexamethasone the hallucinations subsided. A biopsy specimen performed from a parietal lesion showed malignant infiltration with histological features of an underlying large cell carcinoma of the lung.

**Conclusion** Whereas prior cases of peduncular hallucinosis have been described in association with brainstem compression by an extrinsic source, our case was significant because metastatic carcinoma of the lung has not been previously reported as a cause of this syndrome.

P 1084

**Astrocytoma gemistocyticum in common variable immunodeficiency**

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Common variable immunodeficiency (CVID) is a rare acquired disease of unknown cause. Immunodeficiency syndrome is characterized by low levels of most or all immunoglobulin classes. The major clinical features associated with CVID are: recurrent infections, autoimmune inflammatory diseases and malignancy. Such neoplasms as: lymphomas, adenocarcinoma of the gastrointestinal tract and ovary were found with increased frequency in patients with CVID. To our knowledge, there was **no report** on patient with CVID and primary neoplasm of CNS.

32-years old men had medical history with recurrent infection, tuberculosis and hepatitis C. None of the immunoglobulins IgA and only trace amounts of IgG and IgM were found in his

serum. He was treated with the i.v. infusion of immunoglobulin concentrates in a dose of 400 mg/kg, once every three weeks for four years. The patient had two-month history of progressive right hemiparesis, dysarthria and dysphagia. MRI examination revealed high signal intensity lesion on T2-weighted images in periventricular area on the left side and low signal intensity on T1-weighted images in the same area. Stereotactic biopsy and histopathological examination revealed *Astrocytoma gemistocyticum II*<sup>o</sup> WHO. The patient was treated with interstitial brachytherapy of CNS, HDR mode (Ir<sup>192</sup> in total dose 1500 cGy/isodose included the tumour in 10 fractions). In addition, he received an adjuvant telebrachytherapy.

#### P 1085

##### **Cerebellar ataxia and emotional instability due to anti-glutamate decarboxylase-antibodies (anti-GAD) of 65 kD**

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**Introduction** Antibodies against glutamate decarboxylase were originally assigned to the pathophysiology of stiff-man syndrome. Recently, a strong association with other progressive neurological diseases was affirmed and immunological treatment is under discussion. We report on a case of progressive cerebellar ataxia and emotional instability due to anti-GAD successfully stabilized by immunoglobulins.

**Case report** A 52 year-old woman presented cerebellar ataxia with emotional instability and intermittent confusion of insidious onset and subacute progression. Diagnostic work-up including magnetic resonance imaging, neurophysiology, vascular exams, tumour search, mammography and extensive laboratory parameters were normal but for slight hyperthyreosis and anti-GAD 65 with a titre of 61800 U/ml. Treatment with immunoglobulins was initiated and led to an impressive stabilisation of gait and stance as well as diminished melokinetic ataxia and dysarthria. Affect became stable. In a two-year's course, there was a further slight amelioration of the psychopathology more than motricity, and no tumour was detected at follow-up.

**Discussion** Anti-GAD autoantibodies are found to participate in an increasing number of neurological disorders as in this case of cerebellar ataxia. The psychiatric disturbance might have been due to either an encephalopathic affection due to anti-GAD or being constituted by the cerebellar affection itself. As this represents a treatable condition, search for this class of antibodies seems to become mandatory in progressive neurological disease despite the lack of specificity regarding the clinical syndrome.

#### P 1086

##### **Acute primary iliopsoas and adductor magnus abscess presented with proximal leg muscle weakness**

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Pyomyositis is an infection of the striated muscle seen frequently in Africa, but rarely in Western countries with a temperate climate.

We report the case of a 77 year-old woman who presented frequent falls, low back pain, left thigh and groin pain, but she was afebrile. The neurological examination showed a left proximal leg muscle weakness without sensory disturbance and reflex involvement. The investigations of the nervous system were negative. The serum electrophoresis, ESR and CRP were

abnormal, while the blood cultures were negative. A computed tomography of the pelvic region showed a 4x10 cm left iliopsoas abscess and a 5x5 cm homolateral adductor magnus abscess. No obvious source of infection was seen. The abscess was caused by *Staphylococcus aureus*. Our patient did not respond to systemic antibiotic therapy, but she recovered completely with surgical resection of the abscess.

#### P 1087

##### **Meningococcal meningitis presented as sudden coma**

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**Introduction** Vieusseux described Meningococcal meningitis for the first time in 1805. It affects infants in particular but also adolescents and young adults. It is still a serious illness even after the introduction of antibiotics. Meningococcal disease can have atypical presentations ranging from acute fulminating sepsis to prolonged febrile states or isolated psychiatric symptoms.

**Case report** A previously healthy 23 year-old female was found unconscious on her bed, one hour after complaining of feeling unwell, without vomiting, headaches or fever. She was admitted within the following hour, and presented in coma with spontaneous breathing, midsize fixed and dilated pupils, absent oculocephalic reflexes, no pain response and no neck stiffness. Her temperature and blood pressure were normal. There was no rash. Laboratory studies showed leucocytosis (19.510/ $\mu$ l with 90% neutrophils) and hypernatremia (150 mEq/l), with normal chemistry panel and clotting times, negative blood cultures and toxic screening. CT scan showed diffuse brain swelling. She progressed to respiratory failure and hypotension over the next hour. Lumbar puncture (performed after brain death tests) revealed WBC 4474 with 98% neutrophils, high protein (118 mg/dL), and low glucose content (4.89 mg/dL). A CSF culture produced *C* group *Neisseria meningitidis*.

**Conclusion** This patient presented with a fulminant meningitis evolving to brain death in approximately two hours, without any of the classical symptoms. Meningococcal disease presenting as a rapid onset sepsis is relatively frequent, but fulminating meningitis is rare. No references were found in the literature to any case with such a rapid and atypical course.