Peripheral nerve disorders 2

PP2232
Prophylactic anticoagulation in Guillain-Barré syndrome: too much of a good thing?
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Objectives: Venous thromboembolic complications are common during recovery from Guillain-Barré Syndrome (GBS). The use of prophylactic anticoagulation to reduce this risk is accepted as good practice although can be associated with a risk of haemorrhagic complications. We examined the current practice of prophylactic anticoagulation in patients with GBS admitted to a tertiary neurosciences centre. The frequency of venous thromboembolism and haemorrhagic complications were also recorded.

Methods: A retrospective notes review of 50 consecutive patients admitted with GBS to the Greater Manchester Neurosciences Centre between 2008 and 2013 was performed. Disease severity, prophylactic anticoagulation type, dose and duration, and the frequency and timing of haemorrhagic and thromboembolic complications were recorded and analysed.

Results: Details of prophylactic anticoagulation prescription were obtained for 42 of 50 patients. All non-ambulant patients (95%, 40/42) received low molecular weight heparin (LMWH) at any dose at some point during a mean inpatient stay of 64 days. 14 haemorrhagic complications occurred in 10 patients. 7 of these coincided with the use of 'treatment (high) dose' LMWH. A bleeding tracheostomy site contributed to the death of 1 patient.

1 thrombotic event was observed: a portal vein thrombosis. No deep vein thrombosis or pulmonary emboli occurred.

Conclusions: Thromboembolic complications were infrequent in this population. However, a relatively high frequency of haemorrhagic complications were observed and these appeared to correlate with the use of 'treatment (high) dose' LMWH.

Systematic work is required to define the optimal prophylactic anticoagulation strategy in patients with GBS to ensure that the benefits outweigh risks.

Disclosure: Nothing to disclose

PP2233
Sensitivities of the different electrodiagnostic criteria in chronic inflammatory demyelinating polyneuropathy
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Introduction: Chronic inflammatory demyelinating polyneuropathy (CIDP) is an autoimmune disease characterized by progressive or relapsing course lasting more than 8 weeks. Because of its highly variable clinical features and nonspecific laboratory findings, several diagnostic criteria have been proposed. In this study, we reviewed the nerve conduction study (NCS) findings of patients with CIDP in order to examine the sensitivity of various electrodiagnostic criteria.

Methods: In this study, 31 cases were recruited from seven tertiary referral centers in Southeast Korea. The distribution of four types of abnormalities suggesting demyelination of peripheral nerve was examined, and six different sets of electrodiagnostic criteria for the CIDP were applied to the individual cases.

Results: The four types of abnormalities suggesting demyelination showed relatively even distribution among four motor nerves tested. Among them, the F-wave abnormality was most frequent and the conduction blocks were least common. Each set of criteria showed sensitivity ranging from 74.2 to 96.8%. The INCAT criteria and Nicholas’ criteria showed highest sensitivity (96.8%), while the Albers & Kelly’s criteria were least sensitive (74.2%).

Conclusion: In comparison to the previous reports, this study showed relatively higher frequency of abnormalities including conduction blocks, and thus, each diagnostic criterion showed higher sensitivity than other studies. Such discrepancies likely came from differences in the ethnic background, clinical subtypes of the patients groups, and normal limits used. Since we have chosen only the classic cases of CIDP, the selection bias cannot be excluded.

Disclosure: Nothing to disclose
**PP2234**

**Unusual and disabling neurological presentation of ANCA-positive small vessel vasculitis**

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**Introduction:** PNS symptoms are usual on the course of ANCA-positive small vessel vasculitis. The commonest type of neuropathy is multiple mononeuropathy. A purely sensory axonal distal symmetric polyneuropathy is very rarely described.

**Methods:** We observed and followed a patient whose clinical case is described below. Furthermore, we reviewed the current scientific evidence and similar published clinical cases.

**Results:** Male, 54yo, who begins with paroxysms of dysesthesias on both legs and constitutional symptoms - anorexia, asthenia, weight loss, nocturnal sweating and fever. He progressively deteriorated along ~3mo. When observed on the ED by a Neurologist, his general status was visibly deteriorated; he was unable to stand up because of the excruciating pain on both soles. He was admitted and an extensive study excluded tumoral and infectious causes and revealed N/N anemia, elevated ESR, nephritic syndrome and positive ANA, ANCA anti-MPO, anti-SSa and anti-SSb. A skin biopsy demonstrated necrotizing angiitis and a kidney biopsy showed necrotizing and crescentic glomerulonephritis. An EMG confirmed a purely sensory axonal polyneuropathy. He started immunosuppressive therapy with methylprednisolone 1g/day for 3 days and afterward switched to oral prednisolone 1mg/kg/day, with marked improvement of the neurological symptoms. Later on, cyclophosphamide was associated with good tolerance and additional relief of all symptoms.

**Conclusions:** The type of neuropathy observed on this case is rare among cases of systemic vasculitis. As described in the literature, neurological symptoms resolved promptly with the beginning of immunosuppressive therapy. An early onset of therapy is essential in order to prevent possible sequelae.

**Disclosure:** Nothing to disclose

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**PP2235**

**Characteristics of polyradiculoneuropathy patients - single center 10 years retrospective analysis**

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**Introduction:** Polyradiculoneuropathy is a post infectious or immune mediated peripheral nerve disorder. It is a clinically heterogeneous group with either acute or chronic course, axonal or demyelinating, motor or sensory nerve damage. Acute forms are known under eponym Guillain-Barré syndrome (GBS). Relapsing or slowly progressive course is a feature of chronic inflammatory demyelinating polyneuropathy (CIDP). The aim of our study was to analyze clinical features, laboratory characteristics (cerebrospinal fluid, antiganglioside antibodies), electrophysiological findings and patients’ outcome.

**Methods:** Our hospital database was searched from 2002 to 2012 for patients with GBS and CIDP and was analyzed according to our aim.

**Results:** Ninety-eight patients were included in our study, 76 (78%) with GBS and 22 (22%) with CIDP. Major characteristics are shown in table 1. Sensory symptoms and pain were equally present in both groups; weakness and disability were more prominent in GBS patients. Six (6%) patients had Miller-Fischer syndrome; 5 were tested for anti GQ1b and 4 were positive. Presence of antiganglioside antibodies was not related to bad outcome after 18 months (Modified Rankin Scale 4-6). Age ≥65 years and axonal damage were predictors of worse outcome after 18 months (p=0.026 and p=0.04 respectively). A need for ventilatory support was not related to bad outcome.

**Conclusions:** Higher age and signs of axonal damage are bad prognostic factors in GBS. Antiganglioside antibodies screening might have some diagnostic but no prognostic value in GBS patients. Proper intensive care is needed to prevent additional mortality from complications of severe initial disability.

**Disclosure:** Nothing to disclose
PP2236
Capillary tolerance test in diabetic neuropathy diagnosis
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Introduction: In Belarus diabetes affects 3,900 of every 100,000 people. Diabetic Distal Sensory Polyneuropathy (DDSPN) is a common diabetes complication. In 5 years after diabetes manifests, 12.5-14.5% of patients are diagnosed with DDSPN (FIELD Study, 2012). Electroneuromyography (ENMG) is the gold standard for the clinical stage of DDSPN.

Methods: In 2010-2012, 249 patients were examined (Male: 179, Female: 70, average age: 38±13) with diabetes 2 (82%) and 1 (18%). Vibrational, tactile, temperature, and pain legs sensitivity were defined using ENMG. There was picked a group of patients (116 people) without clinical manifestations and without ENMG criteria of DDSPN (M-response n.Suralis 21 mV (±3). Capillary tolerance test (CTT) was performed before using ENMG. The test involved M-response registration in 30 minutes after 2,0 ml of Xanthinol Nicotinate injected intramuscularly.

Results: After the CTT performed, 73 patients had M-response drop to 13 mV (±3) - (p<0.03) which is typical for DDSPN (San Antonio single ENMG program, 1988). 12 months later, ENMG was repeated. 56 patients showed typical DDSPN signs without using CTT. 24 months later, 36 patients were diagnosed with DDSPN, which proves high sensitivity and specificity of CTT (95 and 75% correspondingly (p<0.05)).

Conclusions: CTT improves the ENMG diagnosing on the preclinical stage of DDSPN.

Disclosure: Capillary tolerance test improves the ENMG diagnosing on the preclinical stage of DDSPN

PP2237
Abstract withdrawn

PP2238
The importance of early diagnosis and therapy with intravenous immunoglobulins in patients with multifocal motor neuropathy
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Introduction: The aim of the study was to evaluate the effect of intravenous immunoglobulins (IVIG) treatment on disease progression and quality of life in multifocal motor neuropathy (MMN) patients.

Methods: This study included patients with MMN who were diagnosed and treated in the period from 2000 to 2010. Initially IVIG therapy is administered at a dose of 0.4g/kg for five consecutive days and then at 6-8 weeks in dose of 0.5g/kg - 1g/kg. After a period of at least 2 years, check-ups were carried out including: the assessment of muscle strength (MRCscor), electromyography (EMG) and assessment of quality of life (QoL) using the SF36 and INQoL questionnaire.

Results: Twenty patients with MMN were included, 14 men and 6 women. The mean age at onset was 39.5±11.9 years and the mean duration of disease 9.3±5.7 years. The delay of diagnosis was 2.9±2.7 years. The mean duration of IVIG therapy was 5.0±4.8 years. All patients had a significant reduction in MRC score at the control neurological examination in relation to the period of diagnosis but without statistical significance (70.54±4.87 vs. 63.38±13.94, p>0.05). In EMG examination, higher axonal degeneration was observed as a sign of progression of the disease (p<0.05). QoL was not in association with disease duration, clinical and EMG parameters (p>0.05).

Conclusion: The basic preconditions for a favorable outcome of the clinical course of MMN are early diagnosis and initiation of IVIG therapy immediately after making a diagnosis.

Disclosure: Nothing to disclose
**PP2239**

**Tafamidis treatment in a patient with transthyretin amyloidosis due to domino liver transplantation**

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Domino liver transplantation (DLT) increase the number of grafts available to treat patients with liver disease. But, this procedure has a risk of transmission of systemic transthyretin (TTR) amyloidosis. A 69-year-old FAP male patient whose complaints started 8 years after DLT was evaluated because of numbness and burning sensation of feet and hands, walking difficulty, dizziness, diarrhea, dry mouth, urinary retention. His neurological examination showed distal weakness of all limbs with bilateral steppage gait, stocking and glove type hypoesthesia and hypalgesia, diminished vibration sensation and absent tendon reflexes. According to these findings his clinical disease stage was classified as I and his neurological disability score (NDS) 68. EMG showed findings consistent with distal sensory motor axonal polyneuropathy accompanied by autonomic involvement. His sural nerve biopsy disclosed severe axon loss with amyloid deposition. He did not have any cardiac, renal or eye involvement due to amyloidosis. He refused to undergo a new liver transplantation and was put under treatment with Tafamidis Meglumine (Vyndaqel). He is still receiving the treatment that started seven months ago. He became stable and his sensory symptoms showed slight improvement.

Although estimated time of de novo amyloidosis transfer risk is expected to be minimum 20 years, according to the literature patients can become symptomatic earlier than expected. Our patient’s signs and symptoms started 7 years after transplantation. We do not yet know the effects of the Tafamidis treatment in these patients.

**Disclosure:** Nothing to disclose

**PP2240**

**Acute bilateral non-traumatic radial nerve palsy - rare presentation, uncertain etiology**

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**Introduction:** Acute transient radial nerve palsies usually have a traumatic/compressive etiology. Occasionally, no such mechanism of lesion is verified and a bilateral presentation is even rarer.

**Methods:** We present a case of a 63-year-old female patient, observed at emergency department. Three days before, she had noticed left hand extension weakness, followed, in the next day, by right hand extension weakness. The establishment time in each side was brief (hours). She had no other motor or sensory complaint. She had controlled hypertension and hypothyroidism, and used to have frequent artistic hobbies using lead-containing dyes and ceramics.

**Results:** Apart from bilateral wrist and fingers extension paresis (MRC 1-2/5), her neurological examination was unremarkable. Blood analysis, immunological studies, serologies, C-reactive protein and sedimentation-rate were normal or negative. The serum-lead levels were within normal limits; however urinary-lead levels were 91.73 micromol/day (normal 0.00-0.39). She performed neurophysiological tests, 15 days after palsy-onset: i)nerve conduction studies were normal, including motor and sensory radial potentials; ii)needle EMG revealed fibrillations and positive-sharp waves, at rest, and reduced number of motor unit potentials, during voluntary activity, in common extensor digitorum, brachioradialis and triceps muscles, bilaterally. She has avoided contact with lead-containing substances. Five months later, without any specific treatment, she was fully recovered.

**Conclusions:** This presentation is rarely described in the literature. Although serum-lead level was negative, the high urinary-lead level could suggest some pathophysiologic involvement of this metal, in this case. Additionally, an underlying genetic susceptibility or immunological condition cannot be excluded.

**Disclosure:** Nothing to disclose
PP2241
Abstract withdrawn

PP2242
Subcutaneous is better tolerated than intravenous immunoglobulin treatment: the experience with two CIDP patients
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Introduction: Treatment with intravenous immunoglobulin (IVIg) is commonly used in patients with autoimmune neuropathy with good results but may show adverse effects partially limiting its use. Here we report our experience with two patients showing chronic inflammatory demyelinating polyneuropathy (CIDP) treated with IVIg who needed to shift to subcutaneous immunoglobulin (SCIG) because of considerable adverse events.

Methods: The first patient was a 48-year-old man who started after CIDP diagnosis a 5 days of high dosage IVIg (0.4 mg/Kg/die) and a maintenance IVIg cycle (3 days) every 4-5 months. The second patient was a 70-year-old man affected by CIDP starting IVIg at high dosage (0.4 mg/Kg/die for 5 days). After the treatment neurological examination showed a motor and sensory improvement in both patients.

Results: IVIg although producing a significant improvement of neurological symptoms was stopped in both patients because of a disabling symptoms such as a "tension-type headache" limiting work activities in the first patients or a lower limb venous thrombosis with subsequent pulmonary embolism after the first IVIg cycle in the second patient. The shift to SCIG (Hizentra: 8 gr/week) produced no change at the neurological examination compared to IVIg without any side effects after 8-12 months. Importantly after SCIG they reported an improvement of quality of life as evaluated by a short Form Health Survey scale (SF-36).

Conclusions: SCIG treatment was effective as such as IVIg but it was better tolerate without any relevant side effects and with higher quality of life.

Disclosure: Nothing to disclose
PP2243

Posterior interosseous neuropathy – a diagnostic challenge

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Introduction: Posterior interosseous neuropathy (PIN) is most commonly caused by posterior interosseous nerve compression (by space-occupying lesions/Frohse’s arcade tendinous hypertrophy) or trauma. Neuralgic amyotrophy (NA), multifocal motor neuropathy (MMN) and inflammatory/idiopathic etiologies are considered rare.

Case report: 63-year-old male, retired banker, right-handed, complained of sudden-onset weakness of right thumb extension/abduction. He denied trauma history, repetitive supination-pronation movements, local/cervical pain and previous vaccination/infection. Medical history of Bell paralysis.

Objectively: Without wrist radial deviation or pain on elbow/forearm palpation.

Neurologic examination: paresis of right pollicis abduction/extension and index finger extension at metacarpophalangeal-joint (grade 3 and 4, respectively). Normal osteotendinous reflexes. Without atrophy/sensitive abnormalities.

Laboratory screening: normal. EMG: right radial compound-motor action potential (CMAP) amplitude (recorded in extensor indicis proprius) was reduced. Without conduction blocks. Positive sharp-waves/fibrillations in extensor pollicis brevis (EPB), longus (EPL) and EIP, and incomplete recruitment-pattern on voluntary activation. Extensor digitorum communis was normal. Right-forearm MRI: Abductor pollicis longus (APL) and EPL hiperintensity, without nerve compression signs. He received analgesic/anti-inflammatory medication and physical therapy, recovering in three months.

Discussion: We present a rare case of PIN involving the lateral branch that innervates EPB/EPL/APl muscles. A compressive/traumatic etiology was excluded. NA seems unlikely (painless, without atrophy or previous vaccination/infection), as well as MMN (without clinical/neurophysiological evidence of other conduction blocks, spontaneous remission). So, inflammatory/idiopathic appears as the most probable etiology. Hashizume et al. found three similar simple nerve paralysis in thirty-one non-traumatic PIN cases. Peripheral mononeuropathies might be challenging, considering the complex clinical evaluation and diversity of differential diagnosis.

Disclosure: Nothing to disclose

PP2244

Search for autoantibodies targeting the nodes of Ranvier in chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)

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Introduction: Nodal, paranodal and juxtaparanodal proteins have been the focus of ongoing research as potential antigens in both central and peripheral demyelinating disorders. Antibodies against contactin-2, which assists in the formation of axonal connections, have been detected in multiple sclerosis patients (Derfuss, PNAS 2009; 106, 8302) and polymorphisms in the gene encoding contactin-2 may influence treatment response in CIDP (Iijima, Neurology 2009; 73, 1348). Cx32, which comprises gap junctions of the paranode, is also a potential target antigen in PNS demyelination as mutations in Cx32 cause Charcot-Marie-Tooth disease. Our objective was to examine whether CIDP patients harbour antibodies against antigens expressed at the nodes of Ranvier such as contactin-2/TAG1 and Cx32.

Methods: Sera from 45 patients with CIDP (with paired CSF samples from two), 5 with multifocal motor neuropathy (MMN), and 4 with combined CIDP and central demyelination (with paired CSF from one) were examined. We established a cell-based assay (CBA), in which human embryonic kidney cells were transfected with cDNA clones encoding either the FNIII or the IgC2 domains of TAG-1 or Cx32, all tagged with eGFP. Antibodies against IgC2 and Cx32 were used as positive controls. Antibody binding was visualized using an anti-human fluorescent secondary antibody.

Results: No positive staining was detected in any of the patients with autoimmune peripheral neuropathies for both antigens.

Conclusions: Contactin-2 and Cx32, which is responsible for a genetic demyelinating neuropathy, are not autoantibody targets in acquired autoimmune demyelinating neuropathies. Reactivity to other nodal, paranodal or juxtaparanodal antigens is currently explored.

Disclosure: Nothing to disclose
PP2245

Sensory mononeuritis: differences between pure neural leprosy and non systemic vasculitic neuropathy

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Introduction: Our aim was to determine the distinguishing features of two cases of sensory mononeuritis who presented with similar clinical, electrophysiological and past medical history features.

Methods: We systematically reviewed the clinical features, laboratory studies, neurophysiologic findings, and histopathological changes of two patients with sensory mononeuritis. In one, the final diagnosis was pure neural leprosy (PNL) and the other non-systemic vasculitic neuropathy (NSVN).

Results: Our patients were females who had resided in areas endemic for leprosy (Brazil). They both developed a progressive, purely sensory, painful mononeuritis distally in the lower limbs followed, in patient 1, by asymmetric ankle edema and nodular induration without skin changes. In both cases, sensory nerve potentials were asymmetrically reduced in amplitude, and sural nerve biopsy revealed nonspecific inflammatory infiltration of the vasa nervosum in the epi- and perineurium. An axonal neuropathy, granulomas with epithelioid cells and caseous necrosis were observed in patient 1 confirming paucibacillary PNL; a skin punch biopsy revealed similar changes. Multifocal axonodemyelinating changes in patient 2 were compatible with NSVN. Both patients improved following targeted treatment (rifampicin and dapsone in case 1 and rituximab in case 2).

Conclusions: Both patients were surprisingly homogeneous in their clinical and electrophysiological manifestations. Late appearance of edema and nodular induration in the vicinity of affected nerves, as well as, distinct pathological features with granulomas and caseous necrosis in skin or nerve biopsies appeared to be the cardinal features distinguishing PNL from NSVN.

Disclosure: Nothing to disclose

PP2246

Guillain-Barré syndrome with acute psychotic disorder during therapy with PEG-IFN and Ribavirin for chronic hepatitis C – a case report

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Introduction: The first line treatment for chronic hepatitis C is the combination of PEG-IFN and Ribavirin. There are a few reports of acute inflammatory demyelinating polyneuropathy (AIDP) as a neurological side effect of this therapy. Psychiatric side effects such as depressive disorders, panic attacks and mental status abnormalities are likely to appear during immunomodulating therapy but also in the course of AIDP.

Results: We report the case of a 59-years-old woman treated with PEG-IFN and Ribavirin for 21 weeks for a chronic HCV infection that developed Guillain-Barré Syndrome (GBS) and acute psychosis. The patient was admitted in our department with distal rapid progressive paresthesias in her arms and feet, associating weakness, numbness, as well as intense pain in her low thoracic spine region. The normal MRI scan of the spine, the modified conduction studies and the albuminocytological dissociation of the CSF were consistent for AIDP. The Pegasis and Ribavirin treatment was interrupted and the patient received intravenous Immunoglobulin G. In the first days of the treatment the neurological status aggravated to tetraparesis with bilateral Bell’s palsy. More to that point she became agitated and experienced vivid dream and visual hallucinations. The neurological symptomatology improved in the following days and the mental status abnormalities resolved after neuroleptic treatment. She recovered partially, after 6 weeks of intense neurological rehabilitation.

Conclusions: We should acknowledge that the course of Guillain-Barré Syndrome could be atypical, especially during immunomodulating therapy for chronic HCV infection.

Disclosure: Nothing to disclose
PP2247

Bi-brachial palsy (man-in-a-barrel syndrome) from acute demyelinating polyneuropathy associated with sarcoidosis

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Introduction: 5% of sarcoidosis has neurosarcoidosis. Peripheral nerves are involved in 20% of neurosarcoidosis. We present a case of acute demyelinating polyneuropathy associated with neurosarcoidosis resulting in bi-brachial weakness.

Case report: A 26-year-old man developed rapid onset of bi-brachial pain and weakness over 4 days. He could barely move his upper arms, forearms, and wrists, with minimal strength in the fingers (man-in-a-barrel). He had no other neurologic findings. The following were normal: CBC, serum chemistry, CK, viral hepatitis screen, heavy metals, ANA, RPR, HIV, lyme titer, and ACE. CSF showed glucose = 68 mg/dl, protein = 77 mg/dl, 1 WBC, 81 RBC; Albumin, IgG, and IgG index were marginally high, and no oligoclonal bands. NCV studies showed conduction slowing (26 to 42 m/s) and blocks in the upper-limb motor nerves with relative sparing of sensory nerves. He was treated with 5 days of IVIG with no improvement. Chest CT scans showed lymphadenopathies and biopsy revealed non-caseating granulomas and negative AFB consistent with sarcoidosis. He improved on high-dose prednisone and was able to raise both arms in one week, and in three months had 4/5 strength in the proximal muscles and 5/5 in the distal ones. Repeat NCV studies showed reversal of conduction blocks, although CVs remained slow.

Discussion and conclusions: This is an unusual case of neurosarcoidosis presenting like Guillain-Barre syndrome (acute demyelinating polyneuropathy) based on the clinical, electrophysiological, and CSF features. Except that it did not respond with IVIG treatment and improved dramatically with high-dose corticosteroids.

Disclosure: Nothing to disclose

PP2248

A rare Guillain-Barré syndrome variant: facial diplegia paresthesia

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PP2249

Delayed diagnosis of neurogenic thoracic outlet syndrome: a case report

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PP2250

Mekersson-Rosenthal syndrome – a case report

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PP2251

Clinical and nerve conduction studies of paraneoplastic polyneuropathies

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PP2252

Bickerstaff’s encephalitis – a case report of a rare clinical variant

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**PP2253**

**Guillain-Barré syndrome without compatible electromyography findings, presenting with bilateral facial paralysis**

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**PP2254**

**Demyelinating polyneuropathy with axonal loss and spastic paraparesis as a neurological manifestation of ovarian cancer – a case report**

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**PP2255**

**Heterogeneity of peripheral neuropathy in chronic lymphocytic leukemia**

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