Danazol, prednisolone, insulin and physiotherapy were prescribed. At discharge his platelet count was 1,20,000/cumm and perception of sensations and muscle power in both upper limbs was improving. At follow-up after one and a half months, the power also improved, but he required prednisolone and danazol for maintaining a good platelet count.

“Kaleidoscopic autoimmunity” has been reported with various diseases. Our patient developed transient DIC, diabetes mellitus and multiple plexopathy following splenectomy. Multiple plexopathy has a variable presentation. One of the forms is neuralgic amyotrophy (NA) or acute brachial neuritis (ABN) which usually presents as severe pain in the shoulder followed by weakness of shoulder girdle muscles. Moore et al reported a case of lumbosacral plexopathy in a woman with CREST syndrome and vasculitis.7 The most common mechanism is thought to be viral etiology or immune-mediated. Blood lymphocytes are known to get sensitized to branchial plexus nerves in patients with neuralgic amyotrophy.8 It is usually unilateral with rare bilateral asymmetrical findings. Sensory symptoms are rare. Sometimes patchy sensory loss may be present. Electrophysiological studies might demonstrate sub-clinical involvement in asymptomatic limbs in up to 25% of patients and very rarely there is a mild lymphocytic pleocytosis or a rise in protein in the CSF.9 In our patient there was bilateral asymmetrical involvement which is a rare presentation along with weakness without any pain. Our patient also had right common peroneal nerve involvement demonstrated by electrophysiological studies. Prognosis of NA is good with full recovery of strength in 90% of the patients by three years.10 Plexopathy in our patient was probably immune-mediated. The cause for DIC in our patient was most probably immune-mediated though surgical trauma or diabetic ketoacidosis with severe dehydration contributing to DIC could not be ruled out.

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References

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Multiple sclerosis in a patient with chronic ulcerative colitis

Sir,

We report a patient with chronic ulcerative colitis who developed multiple sclerosis after 5 years of remission of ulcerative colitis.

A 34-year-old lady had recurrent bouts of loose stools mixed with blood and mucus for 8 years and a non-healing ulcer on the lateral aspect of left leg. Investigations revealed ulcerative colitis. Skin biopsy revealed features of pyoderma gangrenosum. She was treated with prednisolone and 5-aminosalicylate following which there was improvement in colonic symptoms and the ulcer over the leg healed. Five years after the treatment the patient complained of diminution of vision in both eyes and pain in the orbit on eye movements for 5 days. She was treated with oral prednisolone and showed significant recovery. Three months later, she developed weakness in both lower limbs over 6 days. She was treated with oral steroids and her symptoms ameliorated in 2 weeks. One month after recovery, she was readmitted with complaints of progressive weakness and heaviness in the left upper and lower limb for 15 days. Neurological examination revealed a visual acuity of 6/9 in both eyes and a relative afferent pupillary defect in the left eye. Pronator drift could be elicited in the left upper limb; in the left lower limb the power was Grade 4/5. The deep tendon jerks were Grade 3 in the upper limbs and Grade 4 in the lower limbs bilaterally. Spasticity was demonstrable in the left side with extensor plantar response. MRI brain showed white matter lesions (Figures 1a and 1b). Visual evoked potential was abnormal in both sides. Cerebrospinal fluid (CSF) findings were as follows: cells were absent; protein was raised (74 mg/dl) with normal sugar. CSF albumin 2 mg/dl (13.6 – 34.6 mg/dl), IgG 19.6 mg/dl (1.7-4.4 mg/dl), serum IgG 1722 mg/dl (1000 – 2000 mg/dl) and serum albumin 3.8 gms/dl (3.5 – 8 gm/dl). The CSF IgG level was greater than 12% of the total protein (26.4%). CSF electrophoresis revealed decreased albumin, 35.57% (53.5 –
Letter to Editor

Acute inflammatory demyelinating polyneuropathy in patients with pregnancy

Sir

Acute inflammatory demyelinating polyneuropathy (AIDP) is an acute monophasic type of demyelinating neuropathy, with symmetrical muscle weakness areflexia and ascending paralysis. AIDP has been reported during all the three trimesters of pregnancy and in the post-partum period. It is known to worsen during the post-partum period due to a rapid increase in delayed type hypersensitivity during this period. Relapse during successive pregnancies has been reported. Though the incidence of AIDP in pregnancy is similar to that in the normal population, only 50 cases of AIDP during pregnancy have been reported. The occurrence of the disease in the third trimester presents a high maternal risk because of respiratory complications and risk of premature delivery. We successfully managed a patient of AIDP during pregnancy who showed remarkable recovery following delivery.

A 25-year-old primigravida with 26 weeks of pregnancy presented with acute onset progressive weakness of all four limbs for 10 days and diarrhea with mild fever 2 weeks prior to limb weakness.

References


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70.2%) and β1 globulins - 1.89% (5.1-11.4%). She received a course of methyl prednisolone followed by oral prednisolone for 21 days. At 2 years follow-up the patient’s visual acuity was normal and there was marked improvement in the power of all the four limbs.

The various neurological manifestations of ulcerative colitis reported in the literature are myelopathy, peripheral neuropathy, myopathy, cerebral venous thrombosis, cerebrovascular disease and myasthenia gravis. There are reports about the association between multiple sclerosis and inflammatory bowel disease. Twenty-seven instances of familial concurrence were identified from British Columbia. In another series from Alberta, 17 instances of familial concurrence of both disorders were ascertained. The family history in our patient didn’t reveal any such familial association of two disorders. Familial concurrence of inflammatory bowel disease and multiple sclerosis may represent shared genetic or environmental causes.

Sadovnick and his co-investigators postulated that one or more loci contributing specifically to inflammatory bowel disease may also determine susceptibility to multiple sclerosis. Multiple sclerosis and inflammatory bowel disease share common predisposing factors, but not enough information is available to speculate about possible mechanisms.