tradural extension was seen in two patients.

On MRI about 75% of ganglioneuromas are isointense and 25% are hypointense on T1 images. Most of them are hyperintense on T2 images. The non-homogeneous appearance corresponds to areas of cystic degeneration, hemorrhage or necrotic degeneration.[4,5]

Ganglioneuromas are well encapsulated tumors and can be completely excised. Even when they are intradural, the tumor could be removed without cord injury because they are not adherent to the spinal cord.[1] This and previously reported cases indicate that spinal ganglioneuromas could be completely removed and cured.

Danilo V. Radulovi, D. Branislav, Milica K. Skender-Gazibara,*Nikoli M. Igor
Institute for Neurosurgery, Belgrade, Serbia & Montenegro and
*Institute for Pathology, Medical faculty University of Belgrade,
Serbia & Montenegro
E-mail: imnik@eunet.yu

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Multicentric glioma presenting as man-in-the-barrel syndrome

Sir,

Primary motor cortex is somatotopically organized, and the motor representation in the precentral gyrus forms a motor homunculus – the leg and perineum is represented over the medial aspect of the motor strip, and the arm and the hand over the convexity. It is well known that precise and circumscribed weakness may affect one limb only if the appropriate area of the motor cortex or its projection pathway is selectively damaged.[1]

Bilateral upper limb weakness with relative sparing of lower limbs is usually seen in lesions involving the medullary decussation of the pyramidal tracts, or cervical spinal cord. Such a clinical syndrome due to lesions occurring bilaterally in the motor cortex is a rare event. These bilateral cortical lesions producing brachial diplegia are usually infarcts secondary to cerebral hypoperfusion following shock or aortic surgery. Cerebral tumor causing such paralysis is extremely rare.

A 37-year-old Nepalese national was admitted with seven-week history of gradually progressive worsening weakness of both arms. Weakness involved predominantly the shoulders, elbows and to a lesser extent, the wrist movements. Hands were relatively spared. He was unable to raise his arms or flex his elbows. He had remained ambulatory, continent, seizure-free, with no visual or gait disturbances. For two days prior to admission, he had complained of dull, generalized headache accompanied by one episode of vomiting.

Clinical examination revealed well-built and nourished normotensive male, with no abnormality of higher mental functions. Funduscopy revealed early bilateral papilledema. There was no nystagmus or involvement of facial or of lower cranial nerves. Motor system examination revealed power in both deltoids to be grade 0/5, that in elbow flexors 1/5 with wasting of deltoids. Tone was increased in both upper limbs with brisk biceps and triceps jerks. No fasciculations were observed. There was no sensory impairment. MRI brain showed bilateral frontal convexity space occupying lesions with surrounding edema [Figure 1].

After initial treatment with cerebral decongestants and dexamethasone, the right sided tumor was excised by craniotomy, while the left sided tumor was biopsied stereotactically at a later date. Histopathology of the excised specimen confirmed both the tumors to be glioblastoma multiforme.

The syndrome of disproportionate weakness of the upper
limbs versus the lower extremities (eruciate palsy, brachial diplegia) is seen in the traumatic central cord syndrome or cervical spondylotic myelopathy of elderly patients. Brachial diplegia due to pyramidal tract involvement was first described by Mohr[3], while the term man-in-the-barrel syndrome (MIBS) was coined by Sage and Van Clitert[4] to describe the clinical aspect of the patient with disproportionate weakness of both arms, while maintaining mobility of face and lower limbs (as though the trunk of the patient is stuck on a barrel). The term eruciate palsy is best used for lesion of corticospinal tracts in the medulla, while exclusive use of the term MIBS for bilateral frontal lobar lesions as in the original description would provide more clarity to the terminology[5]. MIBS is seen commonly after cardiac tamponade, aortic surgery[5], systemic hypoperfusion and hypovolaemic shock[6], head injury[7], anoxic damage to the cortex[8] in the area of somatotopic representation of the arms. There is only one report of tumors being responsible for MIBS – that due to cerebral metastases from undifferentiated carcinoma lung[9]. Multicentric glioma presenting as MIBS has not been reported earlier.

Harjinder Singh Bhatoe
Dept of Neurosurgery Army Hospital (R & R) Delhi Cantt, Delhi 110010, India, E-mail: hsbhatoe@indiatimes.com

Reference


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Letter to Editor

Acute painful peripheral neuropathy due to metronidazole

Sir,

Metronidazole is a 5-nitroimidazole compound that has potent activity against anaerobic bacteria and several protozoa. Peripheral neuropathy is its rare side effect. We report an unusually rapid development of peripheral neuropathy due to metronidazole. A 45-year-old lady received oral metronidazole for the treatment of vaginitis. After 3 days of treatment, after receiving 3.6 g of metronidazole, she developed severe burning pain in both the feet and aching pain in the muscles of the thighs and calves. On the seventh day she developed burning pain in the hands and fingers and severe aching pain in the muscles of forearms and arms. There were no history of diabetes mellitus, chronic alcohol intake, renal failure, occupational toxin exposure or chronic diarrhoea. She consumed wholesome non-vegetarian diet. There was no history of arthritis, skin rash, recurrent oral ulcers, uveitis, xerostomia or xerophthalmia.

Examination revealed a well-nourished middle-aged lady. There was no pallor, hyper-pigmentation, and evidence of nutritional deficiencies or hypopigmented skin lesions. She weighed 65 kg and her height was 154 cm. Her blood pressure was 120/70 mm Hg. She had no organomegaly. Detailed neurological examination was normal. Her hemogram, peripheral smear, fasting blood sugar, serum creatinine and serum vitamin B12 level were normal. Antinuclear antibodies were negative.

Electrophysiological studies done on the 10th day of illness revealed prolonged distal motor latency (6.5 ms for a distance of 90mm) of posterior tibial nerve, prolonged distal motor latency (5.3 ms for a distance of 90mm) and mildly reduced compound muscle action potential amplitude (3.1mV) of the peroneal nerve and decreased sensory nerve action potential amplitude (2.0 mV) of the posterior tibial nerve. Nerve conduction studies of median, ulnar and sural nerves were normal. Metronidazole was discontinued and symptomatic therapy (Carbamazepine and Gabapentin) was given. She found significant relief of symptoms. After 3 months, she continued to take carbamazepine and gabapentin for symptomatic relief. A repeat nerve conduction study at 3 months did not show any improvement in the abnormalities. Applying the Naranjo’s algorithm,[2] our patient’s neuropathy could be considered a “probable (score +5)” adverse effect of metronidazole.

The cumulative neurotoxic dose of metronidazole in the lit-