line therapy for post-anoxic MSE.

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References


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Middle cranial fossa schwannoma of the facial nerve

Sir,

A 24-year-old man presented on 12/05/97 with history of progressive right facial palsy and decreased hearing of one-year duration. On neurological evaluation, he had total infranuclear facial palsy and conductive hearing loss on the right side. His cranial CT scan revealed a homogenously enhancing isodense mass lesion arising from the middle cranial fossa. Surgery could not be done due to financial problems of the family.

On 03/07/2000, he presented again with the additional problem of ataxia. Repeat CT scan showed a significant increase in the size of the tumor with destruction of the underlying petrous bone (Figure 1). He underwent right temporal craniotomy and excision of the tumor. The tumor was approached extradurally and dissected circumferentially and was excised completely. It was arising from the geniculate ganglion of the facial nerve. The tumor-adjoining cranial nerves were saved. The bony defect in the petrous bone was repaired with temporalis fascia. At three years follow-up, the patient showed moderate improvement in facial function.

The facial nerve is the most frequently paralyzed motor nerve, with 95% of infranuclear palsies due to a pathological process within the temporal bone. Neoplasms account for 5% of facial palsies and neurinomas comprise only a small fraction of these. Facial nerve schwannomas are postulated to arise from the nerves intermedius and its connection in the geniculate ganglion. As the geniculate ganglion is anatomically located towards the anterior surface of the pyramid, the schwannomas originating here are partially located in the petrous bone and their bulk is in the middle cranial fossa. Approximately 30 cases of facial nerve schwannomas presenting as middle cranial fossa lesions have been reported in the literature. The clinical features depend upon the site of origin of the tumor on the facial nerve and the direction of its growth. The principal clinical features of facial nerve tumors are progressive facial nerve paresis and hearing loss.

The management strategy for facial neuroma consists of tumor removal and facial nerve reconstruction. The surgical approach to facial neuroma is selected according to the location and extension of the tumor and state of hearing. In these lesions, the facial nerve should be first identified in the fallopian canal, and the nerve can be followed through the tumor while performing decompression and excision. This technique shall probably enhance the chances of facial nerve preservation or reanimation. The greatest determinant of the outcome of facial nerve reconstruction is the duration and severe

Figure 1: Cranial CT scan axial and coronal cuts showing a homogenously enhancing isodense mass lesion arising from the middle cranial fossa with destruction of the underlying petrous bone.
Giant cell arteritis as a cause of jaw claudication

Sir,

Giant cell arteritis (GCA), a systemic panarteritis involving medium and large arteries, has rarely been reported from India. We report an elderly female who presented with fever and jaw claudication.

A 65-year-old housewife was admitted with a history of low-grade pyrexia for 10 weeks. A few days later she started having moderately severe throbbing headache, which was more marked on the right side and was especially in the temporal and occipital regions. In addition, she had jaw claudication, which gradually increased in intensity. All peripheral pulsations were normal except for the right temporal artery which was not palpable and the left temporal artery was feeble. The facial artery on right side was cord-like and no pulsations could be felt. Systemic examination was normal. Temporal artery biopsy (right side) revealed intimal proliferation with medial hypertrophy. Elastic lamini revealed disruption. There was moderate chronic inflammatory infiltrate with a few giant cells (Figure I and 2). The patient was treated with steroids (prednisolone -0.3 mg/kg/body wt.) to which she responded rapidly with disappearance of both headache and fever. Steroids were continued on the same dose for a month after which the dose was tapered.

Giant cell arteritis also known as temporal arteritis is a chronic vasculitis of medium and large-sized arteries. It usually involves the cranial branches of the arteries arising from the aortic arch. It generally manifests as fever of low grade and headache, especially over the temporal and occipital areas. On physical examination the frontal and parietal branches of the superficial temporal artery are tender, thickened, nodular or may be absent. Jaw claudication occurs in half of the cases. The other manifestations are partial or permanent loss of vision, mononeuritis multiplex, peripheral neuropathy and strokes involving the territory of the affected artery.

Laboratory investigations usually reveal elevated ESR and C-reactive protein, anemia, generally normocytic normochro- mic, thrombocytosis and decreased serum albumin. Liver enzymes may be elevated. Temporal artery biopsy is diagnostic. The response to corticosteroids is usually rapid. However, 30-50% patients have spontaneous exacerbations and may re-