Letters

and noradrenergic function in the pathogenesis of akathisia. Considering the enormous public health problem of non-compliance with neuroleptic medication and its relationship to akathisia, further research in the direction of a rational pharmacology for this problem is clearly needed.

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We report the first case from India. A 21 year old healthy male suddenly developed pain which started at the nape of the neck and spread to the left upper limb. Within two hours all four limbs had become weak. Retention of urine, diminished sensation below the level of the neck and difficulty in breathing had developed when he was brought to the hospital. There was no preceding history of injury or any other illness. On examination the pulse rate was 108/minute and respiratory rate 30/minute. Respiration was diaphragmatic; he could count up to only six in one breath, chest expansion was less than 1 cm, the trachea was deviated to the right, chest percussion was resonant bilaterally and breath sounds absent over the right hemithorax. He was fully conscious and well oriented but had difficulty in speech due to respiratory distress. All higher functions and cranial nerves were normal. He was quadriplegic with grade zero power in all limbs. The deep tendon reflexes were absent. Abdominal and plantar responses could not be elicited. All cutaneous sensation was absent below C4 level. Position and vibration senses were normal at admission, but were absent later.

Laboratory investigations showed normal haemoglobin, total leucocyte count was 16,000 mm$^3$ with 70% neutrophils, 29% lymphocytes and 1% monocytes but no biochemical abnormalities, and blood culture was sterile. Arterial blood gases showed hypoxaemia with respiratory alkalosis. Chest radiograph showed no pneumonitis and radiographs of the cervical spine were normal. The patient was managed with endotracheal intubation, ambu-bag assisted ventilation and antibiotics. He developed pneumothorax on the right side for which an intercostal drainage was established. His condition progressively deteriorated and he died two days after admission.

Necropsy showed a collapsed right lung, pulmonary oedema, broncho-pneumonia and changes in the spinal cord. The heart, major blood vessels, other extracranial organs and the brain were normal. There was no exudate, haemorrhage or any other abnormality of the surface of the cord. The cut surfaces of serial slices showed focal softening and occasional petechiae in segments C2 to D6. Microscopically, these segments exhibited features of recent infarction affecting the anterior horns, lateral columns and the anterior columns (fig 1). The extent varied in different segments but remained confined to the perfusion areas of anterior spinal artery. The necrotic areas showed loss of details of tissue constituents and cavitation. Mild perivascular leucocytic and macrophage response were present. More peripheral parts showed spongiosis and numerous axonal retraction balls. The major blood vessels outside the parenchyma were normal but several intraparenchymal medium and small arteries were occluded by cartilaginous material (fig 2). These were strongly positive with alcian blue at pH 2.5 and not associated with thrombus. This type of vascular occlusion was observed at several levels of the affected portions of the spinal cord. The veins were not involved. No abnormality was observed in sections of the cord beyond mid-dorsal segments.

The patient’s clinical features are consistent with the infarct shown at necropsy which was the result of occlusion of branches of anterior spinal artery by cartilaginous emboli. They are also similar to those in previous cases$^1$ in some of these involvement of veins has also been noted.$^2$ A prominent feature of all cases has been the dramatic sudden onset. The other features of note include the absence of history of significant trauma, involvement of the cervical and upper dorsal segments of the cord and fatal outcome. The diagnosis has seldom been made during life,$^1$ which may be due to the rarity of such cases and the rather obscure

Cartilage embolism of spinal cord

Sir: Acute spinal cord infarction due to cartilage emboli is rare, with an onset of dramatic suddenness which affects previously healthy individuals. Only 20 cases had been reported up to 1981,$^1$ and the pathogenesis remains unclear.

References


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Fig 1 Section of cervical segment of spinal cord showing multiple foci of myelomalacia. Haematoxylin and eosin (HE). × 6.5.

Fig 2 Section of spinal cord showing cartilage embolus in a medium size artery. (HE), × 120.
mechanism of the condition. There is some agreement that the cartilage originates from the intervertebral disc and various possible mechanisms have been discussed. A sudden increase in disc pressure, leading to injection of disc material into small vessels has been favoured, but the exact nature is not known.

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References


Trigeminal neuralgia associated with contralateral intracranial tumour: a false localising sign caused by vascular compression? Report of two cases

Sir: Trigeminal disturbances clinically resembling “idiopathic” trigeminal neuralgia may rarely occur in association with contralateral intracranial tumours—both supratentorial and infratentorial. Only six cases have been reported previously. We describe two further patients with details of the radiological investigation and operative findings. In one case, our study also provided some insight into the mechanism of contralateral trigeminal involvement.

Case 1: A 44 year old female with a history of headache from the age of 34 presented with a left trigeminal neuralgia (2nd and 3rd branches) of five months duration. Eating and speaking triggered paroxysms of pain and were only partially relieved by carbamazepine. On admission, neurological examination disclosed a mild cerebellar ataxia. A computed tomographic (CT) scan with contrast enhancement showed a large high-density mass on the right posterior fossa, displacing the brainstem contralaterally (fig (a)). Right vertebral angiography showed a very mild contralateral shift of the basilar artery. A right suboccipital craniectomy was carried out and a huge meningoia was excised which was attached to the tentorium and right petrous temporal bone. The postoperative course was uneventful. The trigeminal neuralgia progressively disappeared in the course of five months and treatment with carbamazepine was discontinued. No recurrence of trigeminal neuralgia occurred over a follow-up period of nearly two years.

Case 2: A 55 year old male presented with a right trigeminal neuralgia (3rd branch) of six months duration. He experienced recurrent, excruciating, stabbing pains, triggered by eating, speaking and light touch of the sensitive area. The patient obtained considerable relief in the first two months from carbamazepine; later, the pain became increasingly severe and frequent. Three months before admission, he complained of left facial twitching. Neurological examination revealed a left hemifacial spasm. A CT scan showed a large left petro-clival meningioma (fig (b)). A left vertebral angiography showed considerable contralateral displacement of the basilar artery. During surgery and after total removal of the tumour, it was evident that the basilar artery was displaced against the root entry zone of the contralateral (right) fifth cranial nerve. Immediately after the operation, the patient had left abducens nerve palsy and moderate left facial paresis. These symptoms improved over a six month period but were still present three years later. His trigeminal neuralgia gradually attenuated and disappeared in the course of one year. Treatment with carbamazepine was discontinued. No recurrence of trigeminal neuralgia was reported over a three year follow up period.

The clinical syndrome of contralateral trigeminal nerve dysfunction as a false localising sign in intracranial tumours is known to occur, though rarely. In most cases the tumour responsible is a meningioma, usually infratentorial. Patients with trigeminal false localising symptoms and signs can best be separated into two groups: trigeminal neuralgia (six reported cases) and those with other sensory function disturbances of the fifth cranial nerve (26 reported cases). Our two patients who experienced a typical trigeminal neuralgia contralateral to an infratentorial meningioma were included in the first group.

Some explanations have been put forward to account for the contralateral trigeminal neuralgia. Nevertheless, the mechanism of trigeminal involvement remains speculative since direct observation of the affected trigeminal nerve is rarely possible during surgery to remove the neoplasm. In case 2 of our series, however, the basilar artery was shown to be displaced by the tumour against the opposite trigeminal nerve.

Observations during surgery as well as at

![Fig 1](image1.png) (a) Case 1—CT scan. Huge meningioma of the right posterior fossa attached to retromental portion of the petrous bone and to the tentorium. (b) Case 2—CT scan. Large meningioma of the left petro-clival area.
Cartilage embolism of spinal cord.

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