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Trigeminal neurinoma presenting with trismus

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Abstract
We describe a patient with trigeminal neurinoma whose main presenting symptom was trismus. This has not previously been reported in the literature. We review the previously described symptoms and signs of trigeminal neurinoma.

Key words: Neurinoma; Trigeminal nerve; Trismus

Introduction

Trigeminal neurinomas account for about 0.2 per cent of all intracranial tumours (Schisano and Olivecrano, 1960) and between two and three per cent of intracranial neurinomas (Nager, 1984). The initial symptoms are usually an expression of trigeminal nerve dysfunction, most commonly irritation or changes in facial sensation (Cohen, 1933). We describe what we believe to be a unique case of a trigeminal neuroma with trismus as the main presenting feature.

Case report

A 59-year-old male presented in August 1997 to the oral surgery department with trismus and parasthaesia in the distribution of the left maxillary and mandibular divisions of the trigeminal nerve. He had previously suffered with left fronto-temporal headaches and pain in the lower jaw, which had settled. No dental pathology was found but it was noted at this time that he had previously suffered with nose and sinus problems and had undergone septoplasty, trimming of turbinates and polypectomy. He was taking no medication and had no known allergies.

Magnetic resonance imaging (MRI) showed an altered signal in the muscles of mastication on the left and a lesion in the foramen oval. Examination under anaesthetic and biopsy of the pterygoids showed changes most consistent with a chronic inflammatory myopathy.

The patient was referred to the Oxford Skull Base Clinic where a repeat scan of the trigeminal ganglion showed a high signal mass lesion filling Meckel's cave, contiguous with the trigeminal nerve and with all the characteristics of a trigeminal neuroma (Figures 1 and 2).

Discussion

Trigeminal neurinomas originate from neurilemmal cells in the distal portion of the nerve, almost exclusively from the sensory fibres. They may arise in any segment of the nerve but the majority develop at the gasserian ganglion.

Most intracranial neurinomas arise from the VIIIth cranial nerve, with trigeminal tumours following in frequency at two to three per cent. There is no sex difference, with the peak age for presentation being between 38 and 40 years. Two thirds of affected patients have initial symptoms of trigeminal nerve dysfunction, primarily irritation and changes in facial sensation. Pain may be present although it is not a constant feature. Further investigation may disclose hypaesthesia or hypalgesia in one or all divisions of the nerve and decreased or absent corneal sensation. Weakness of the masticatory muscles may be present (Table I).

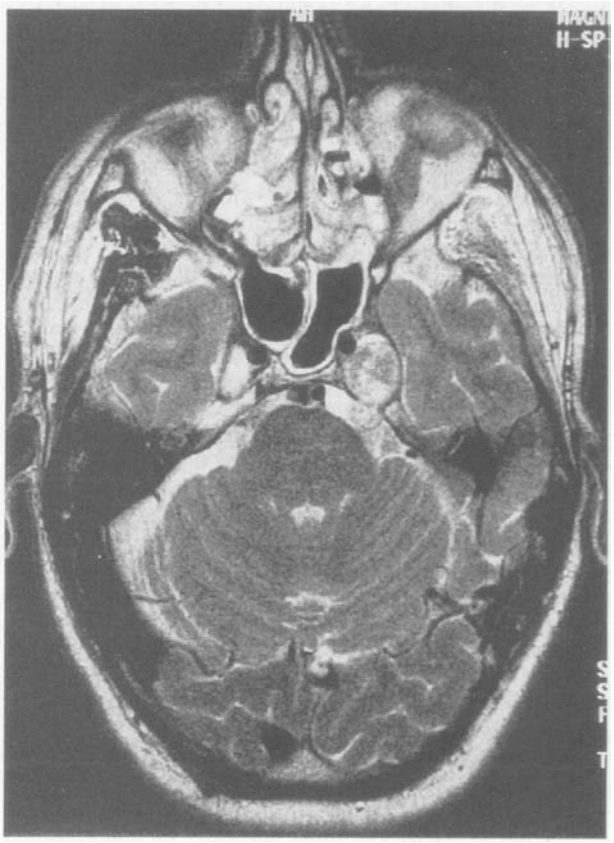

Fig. 1
Axial MRI showing left trigeminal neurinoma in Meckel's cave.

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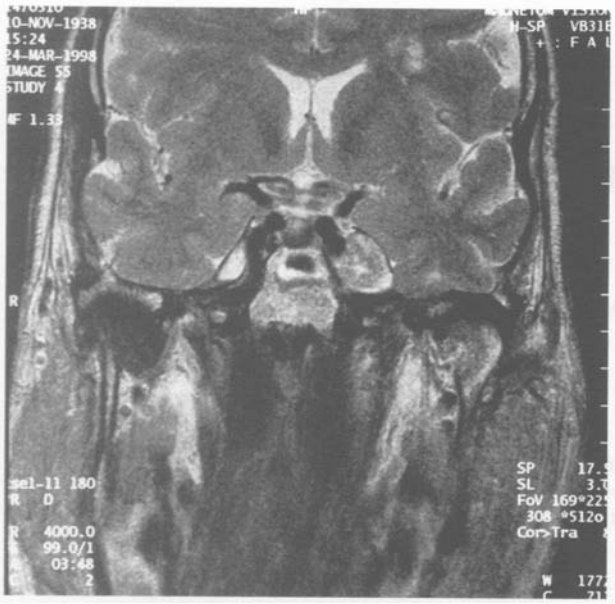

FIG. 2
Coronal MRI showing left trigeminal neurinoma.

The degree of trigeminal dysfunction as well as the relationship between impairment of sensation and motor function may vary considerably (Nager, 1984). Jefferson attributed this to the fact that neurinomas involve only part of the nerve fibres and the tumour in its subsequent development does not affect the root of the nerve (Arseni *et al.*, 1975).

In general, trismus is thought to be result of a forced closure of the mouth caused by persistent spasm of the jaw-closing muscles. It is useful to differentiate between painful and painless causes. In the case of painful arrest, a pathological process in the jaw or the mouth is probable, such as inflammation or traumatic lesion of the temporo-mandibular joint or parapharangeal abscess. Painless trismus can be more difficult to differentiate. The most common form is tetanus, which is usually easily identified by the other symptoms of tetanus (although in localized cephalic tetanus, the trismus may be the only sign). Mechanical changes such as shortening, induration and reduced elasticity of the temporal muscle or lesions of the temporo-mandibular joint will also result in trismus (Schwerdtfeger and Jelasic, 1985). Apart from tetanus, neurogenic trismus has been described and has been attributed not to spasm of the jaw-closing muscles, but to a paradoxical activity of these muscles during jaw opening secondary to disturbed programming and co-ordination of the masticatory muscles within the mesencephalic nucleus (Jelasic and Freitag, 1978). This hypothesis has been further expanded with EMG studies of patients suffering from trismus whose identified pathological processes included meningioma of the cerebello-pontine angle and trigeminal root section (Schwerdtfeger and Jelasic, 1985).

TABLE I
PRESENTING SYMPTOMS IN TRIGEMINAL NEURINOMA (DECREASING ORDER OF FREQUENCY)

Trigeminal nerve dysfunction	– numbness – pain – paraesthesia
Headache	
Diplopia	
Hearing loss I tinnitus	
Visual loss	
Ear pain	
Subarachnoid haemorrhage	
Vertigo	
Seizure	
Exopthalmos	
Gait abnormality	

(McCormick *et al.*, 1988)

The actual cause of trismus in this patient is not clear but seems likely to have a mechanical background with disuse atrophy secondary to weakness, given the results of the biopsies.

This case underlines the insidious onset, frequently atypical sequence of clinical manifestations and protracted course that account for the frequently observed delays in recognizing trigeminal neuroma.

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