results. It is often noted that patients do not do very well on B-lactam antibiotics despite bacteria being sensitive to them in cultures. The classic treatment of NF would be a combination of benzyl penicillin and clindamycin. In our case the isolated *Streptococcus pyogenes* was sensitive to penicillin. The *Staphylococcus aureus* was sensitive to chloramphenicol, but resistant to both penicillin and erythromycin, so clindamycin was not a first choice antibiotic, due to the phenomenon of dissociated resistance with erythromycin, and chloramphenicol was used instead. Chloramphenicol also gives good cover for anaerobes; hence metronidazole was stopped.

Antibiotics alone are unable to penetrate necrotic tissues and have limited effect until extensive fasciotomy and excision of all necrotic tissue is done. After surgery the involved areas should be watched carefully for expansion of necrosis and repeated debridement should be performed if necessary. The skin defect is initially allowed to granulate and secondary reconstruction with skin grafts should be delayed for 3–6 months unless there is corneal exposure; then reconstruction should be performed as soon as the infection has resolved.

Good nutritional support, aggressive fluid resuscitation and analgesia are important measures of treatment. The use of hyperbaric oxygen has been suggested but its efficacy is not known.

NF, if not treated adequately, can lead to ophthalmic artery occlusion, severe skin loss with lagophthalmos, ectropion, epicanthus and significant cosmetic disfigurement. NF involving the neck area carries a higher mortality rate as it tends to spread caudally to the chest and mediastinum, often resulting in pulmonary complications and death. This stresses the importance of early recognition and proper treatment of the disease.

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Sir,

Traumatic prolapse of the globe into the maxillary sinus diagnosed as traumatic enucleation of the globe

A pure blow-out fracture is a fracture of one or more of the orbital walls without involvement of the orbital margin.[1] To our knowledge there have been only a few documented cases of blow-out fracture with prolapse of the entire eyeball into the maxillary sinus.[2–4]

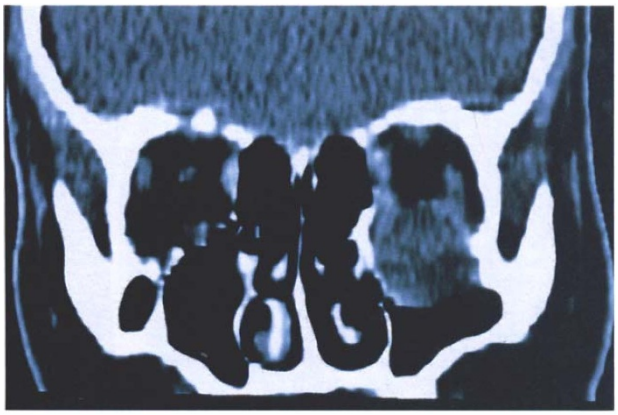
CT scan is diagnostic and is helpful for subsequent management.[5] In our patient the condition was initially misdiagnosed as traumatic enucleation of the globe. Two months later a CT scan of the orbits and paranasal sinuses showed the prolapsed eyeball in the maxillary sinus.

Case report

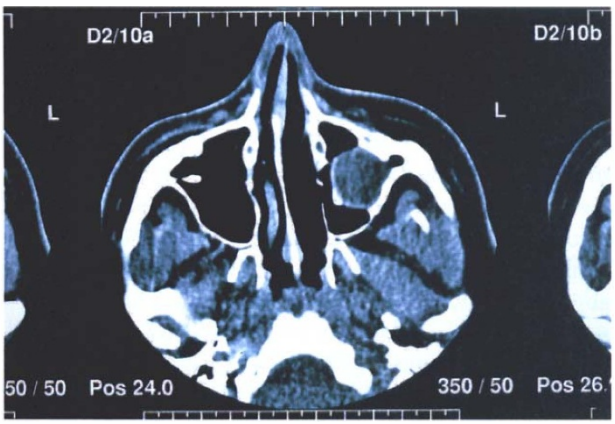
A 29-year-old man presented to the eye casualty department with severe pain in the left orbit. He had been the victim of an assult in which he had suffered blunt trauma to the left orbit. The patient could not recall the exact circumstances of the accident because he was under the influence of alcohol. Following the trauma he had immediately lost all sight in the left eye and experienced neurosensory loss in the distribution of the infraorbital nerve. On examination he had no light perception on the left side and there was marked left periorbital oedema.

The eyelids were opened with a speculum and the globe appeared to be absent. On examination under general anaesthesia the globe, extraocular muscles and optic nerve were not found. A diagnosis of traumatic enucleation of the globe was made. A central conjunctival defect was sutured. The patient was subsequently followed up in the eye clinic and 2 months later was seen in the oculoplastic clinic for consideration of secondary orbital reconstructive surgery. A pre-operative CT scan showed a huge pure blow-out fracture of the orbital floor with prolapse of the entire globe into the maxillary antrum (Fig. 1).

The patient was subsequently referred to the oculoplastic service at Manchester Royal Eye Hospital. The patient had developed a marked socket contracture (Fig. 2). A surgical exploration of the socket and of the blow-out fracture was undertaken. The globe was firmly adherent to the walls of the maxillary antrum and could not be reposited into the contracted socket (Fig. 3). The globe was enucleated. Of the extraocular muscles only the horizontal recti could be identified. A scleral wrapped 18 mm hydroxyapatite implant was placed



(a)



(b)

Fig. 1. *Pre-operative CT scans. (a) Coronal view showing the left eyeball prolapsed through the fractured orbital floor into the maxillary antrum. (b) Axial view showing the displaced left eyeball within the maxillary antrum.*

along with a labial mucous membrane graft. The orbital floor defect was reconstructed using a titanium microplate anchored by screws to the infraorbital margin and a Medpor sheet implant. Post-operatively the implant was noted to be low within the socket. A further labial mucous membrane graft was placed into the inferior fornix. An ocular prosthesis was fitted 4 months after the enucleation.

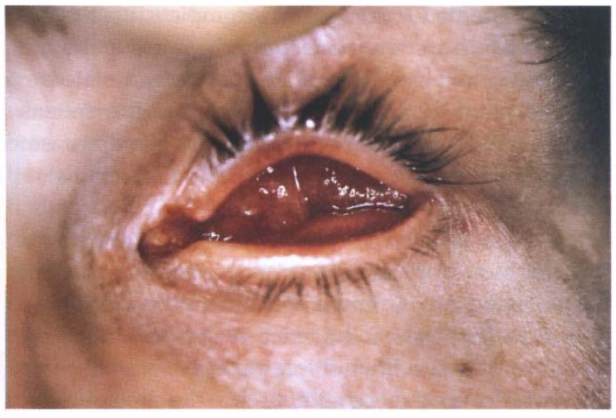


Fig. 2. *A photograph of the left eye showing a contracted socket and giving the appearance of post-enucleation socket syndrome.*

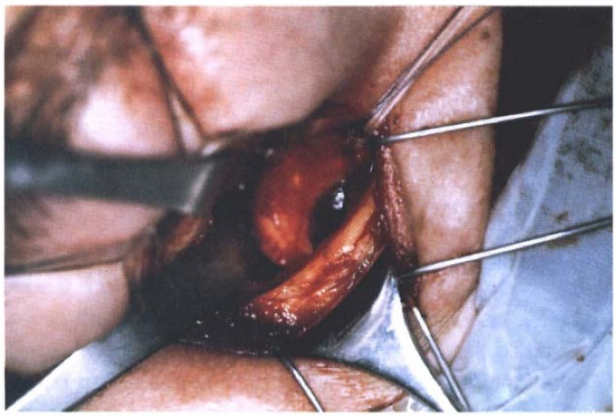


Fig. 3. *An intraoperative photograph showing the left eyeball clearly visible through the deficient orbital floor.*

Comment

An orbital blow-out fracture usually results from a blunt trauma by an object larger than the orbital opening.[6] The medial wall and the orbital floor, either singly or in combination, are most frequently involved. Structures in the orbit can herniate through the fracture into the paranasal sinuses.

The globe is suspended in the orbit by the suspensory ligament of Lockwood, and by the medial and lateral check ligaments. The globe is also supported by the extraocular muscles and orbital fat.[7] The globe does not normally sink significantly after removal of the maxilla and orbital floor.[8] Many of the supporting structures of the globe must be ruptured or severely damaged before the globe can be displaced inferiorly into the maxillary sinus.

Displacement of the globe into paranasal sinuses should be suspected in cases with blunt trauma in which the globe cannot be seen within the orbit. A CT scan should be performed early in all such cases. The surgical management of such a case is much simpler if undertaken early. The late management is much more difficult as illustrated by our case.

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Sir,

A case of trigeminal schwannoma presenting as Raeder's syndrome in a child

Raeder's paratrigeminal syndrome consists of Horner's syndrome in association with headache and trigeminal dysfunction. Raeder's syndrome is perhaps now more often associated with unilateral cluster headache and atypical migraine. The original description was of five cases of painful post-ganglionic Horner's syndrome, considered to be due to lesions arising from the middle cranial fossa. There are a multiplicity of other causes for this combination of signs, including tumours arising from the trigeminal nerve. We describe such a patient.

Case report

A 10-year-old girl presented with an 18 month history of painless blurring of vision in her right eye. She had been diagnosed as suffering from allergic keratoconjunctivitis and treated with topical steroid and sodium cromoglycate. Her eye had failed to respond to this treatment and the diagnosis had been revised to one of floppy eyelid syndrome. All topical therapy was stopped for a period but there was no improvement in her condition.

She was referred to the oculoplastic service at Manchester Royal Eye Hospital. Her examination findings were as follows: Her corrected visual acuity was 6/24 right and 6/5 left. The right cornea showed punctate epithelial erosions, anterior stromal scarring and peripheral neovascularisation. She had a right 3 mm ptosis. The right pupil was miosed, but reacted normally to light and near. Her ocular motility was normal; neurological examination showed reduced soft touch sensation on the right side of her face and anaesthesia of the right cornea. There was wasting of the temporalis and masseter muscles, and weakness of the pterygoids. Her examination findings were otherwise normal. In particular, there were no clinical features of neurofibromatosis and she had no other abnormal neurological findings.

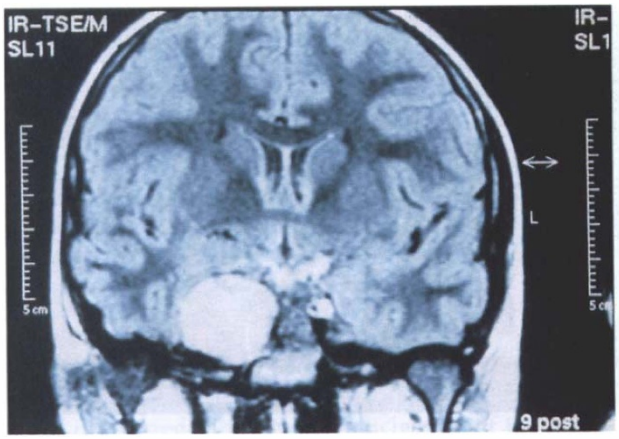

Fig. 1. *Coronal T2-weighted MRI scan showing the 5 cm trigeminal schwannoma.*

The findings of a right Horner's syndrome combined with signs of ipsilateral trigeminal dysfunction suggested a lesion of the floor of the middle cranial fossa. An MRI scan showed a large (5 cm diameter) tumour arising from the right trigeminal nerve (Fig. 1). She underwent neurosurgical resection of the lesion. Histological examination showed a tumour composed of spindle cells staining strongly for vimentin and S100, and negative for neurone-specific enolase and smooth muscle actin. The diagnosis was therefore a schwannoma of the trigeminal nerve.

Her post-operative course was complicated by the development of a large central neurotrophic ulcer in the right cornea (Fig. 2). This was treated initially with topical antibiotics and lubricants followed by botulinum-toxin-induced ptosis. As a definitive protective procedure, she later required a lateral tarsorrhaphy, which allowed the cornea to heal completely. Her corrected visual acuity was reduced to counting fingers secondary to corneal scarring. A permanent central tarsorrhaphy was required for corneal protection. This had obvious cosmetic implications and therefore following careful discussion with the parents she underwent an enucleation with primary hydroxyapatite implant wrapped in autologous fascia lata.

Comment

Raeder first described his paratrigeminal syndrome in 1924. He presented four cases of incomplete, post-ganglionic Horner's syndrome with trigeminal and other cranial nerve involvement and one case with isolated trigeminal neuralgia.[1] In each of the original descriptions there was either facial or periorbital pain. His first case was found to have an endothelioma of the trigeminal nerve arising from the Gasserian ganglion at post-mortem. Ford and Walsh[2] discussed a group of patients with what is now recognised as cluster headaches, all of which were benign. Grimson and Thompson,[3] in their excellent review, classified Raeder's syndrome into three main groups: those with multiple parasellar cranial nerve involvement and, of those without this, the classic cluster headache group and the atypical headaches group (in which there may be associated systemic or local disease,