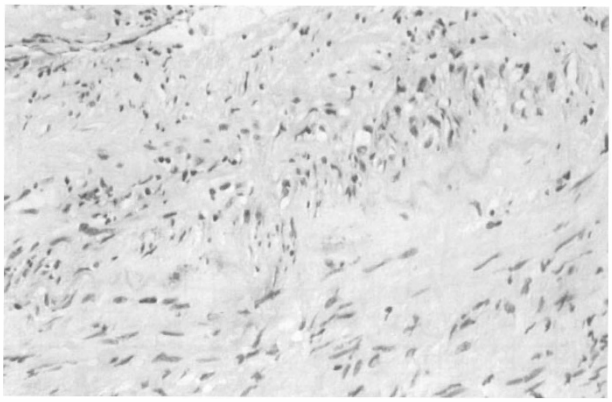

Fig. 2. *Temporal artery biopsy demonstrating a mixed inflammatory infiltrate of the vessel wall.*

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Sir,

Management of orbital emphysema

Orbital emphysema is a well-known clinical and radiological finding in cases of orbital trauma with fracture.[1] The condition occurs when a fracture of the orbital wall is associated with a laceration of the adjacent sinus mucosa that allows air to enter the orbit. The vast majority of these patients are asymptomatic and the air spontaneously resolves without adverse sequelae.[2] Rarely severe visual loss from optic nerve or retinal ischaemia can occur due to the intraorbital air mass causing a compressive orbital compartment syndrome.[3,4] Transient reduction of the ocular circulation is not necessarily followed by permanent visual loss[5] and therefore prompt diagnosis and management of these cases is essential.

Case report

A 76-year-old man presented with a 1 day history of a swollen, red, painful left eye with reduced vision. He had fallen 4 days previously suffering blunt facial trauma, and a fractured nasal bone had been diagnosed clinically; radiological investigations were not deemed necessary. There was no significant past ophthalmic history. Regular medications included nifedipine for hypertension and aspirin following a transient ischaemic attack.

On examination he was apyrexial with a pulse rate of 80 beats/min and blood pressure 150/90 mmHg. Visual acuity was 6/5 right eye and count fingers left eye, with a left afferent pupillary defect. There was a 4 mm left-sided ptosis with a haemorrhagic, chemosed conjunctiva (no lid crepitus was palpable). The left eye was proptosed 3 mm, displaced downwards 5 mm and ductions were restricted in all directions of gaze. The intraocular pressure was 20 mmHg left (16 mmHg right) and fundoscopy, including the retinal vasculature, was normal in both eyes.

A clinical diagnosis of optic nerve compression secondary to orbital emphysema was made. An urgent computed tomogram (CT scan) demonstrated a fracture in the lateral wall of the left maxillary antrum with involvement of the orbital floor. There was a marked left-sided proptosis with a large air collection situated laterally in the orbit and retrobulbar space (Fig. 1). With the patient supine a 25 G needle attached to a syringe was passed into the lateral orbit under topical anaesthesia and approximately 5 ml of air aspirated. The proptosis reduced and the patient's discomfort improved immediately. Oral and topical antibiotics were commenced.

On review 12 h later, the left visual acuity was 6/18 with normal pupillary responses. There was a substantial improvement in the ptosis, hypoglobus and proptosis with less restriction of the ocular movements. A repeat CT scan (Fig. 2) demonstrated a small residual amount of intraorbital air. One week later the patient was asymptomatic and the ocular examination, including visual acuity, colour vision and visual fields, was normal.

Comment

The development of orbital emphysema is thought to involve the forceful passage of air through a sino-orbital communication. The pressure gradient required is most likely a consequence of sneezing or nose-blowing. Positive orbital pressures are probably maintained by either a hinged fragment of bone acting as a one-way valve, or orbital fat functioning as a ball-valve.[3] Confinement by the osseous walls, periorbita and septum restricts spontaneous decompression of the

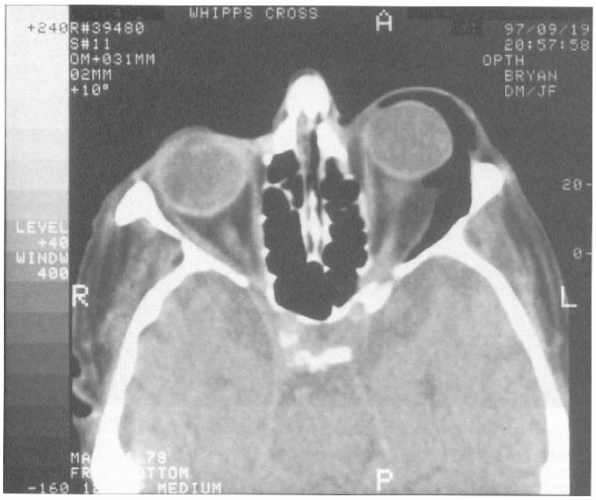

Fig. 1. *Axial CT scan through the orbits showing left-sided proptosis with elongation of the optic nerve, intraorbital emphysema and fracture of the left nasal bone.*

orbital compartment to forward movement of the globe. As the limits of spontaneous decompression are surpassed, the pressure is transmitted directly to the orbital structures. The small vessels supplying the optic nerve are more sensitive to orbital pressure than the larger central retinal artery, but may withstand longer compression without permanent visual loss.[6] This mechanism probably accounts for the reduced vision in our case, where no signs of retinal ischaemia were observed.

Management of cases with significant discomfort or visual impairment includes an urgent orbital CT scan both to confirm the diagnosis and to localise the air mass. The air does not stay predictably at any single site within the orbit, but its position does correlate well with the location of the orbital fracture.[7] Air that enters the orbit but remains subperiosteal can have a more transient effect on vision, most likely due to the lack of ball-valve effect from the orbital fat.[8]

Fig. 2. *Axial CT scan at the same level 10 h later showing reduced proptosis and air mass.*

Systemic absorption of an intraorbital air mass can take up to 7 days[9] and patients with impaired visual function therefore require immediate orbital decompression. Treatment using a needle-coupled syringe is effective and relatively safe. A modification to this technique, however, is the addition of normal saline to an open syringe which then allows the monitoring of air bubbles released and avoids potential suction of orbital structures into the needle tip.[7] In cases with severe visual loss a lateral canthotomy and cantholysis may be appropriate.[4]

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Sir,

Successful treatment of saccular endophthalmitis with clarithromycin

A 78-year-old patient underwent extracapsular cataract extraction in 1995 when an intra-ocular lens (6.5 mm PMMA optic and PMMA type of haptic) was implanted. There were no post-operative complications and visual acuity was maintained at 6/6.

A trabeculectomy was performed in that eye in November 1997 for open angle glaucoma as the intraocular pressure could not be controlled with dipivefrine and timolol drops. The anterior chamber had become flat post-operatively and this was managed with a pressure pad. On the fifth post-operative day the anterior chamber was still shallow and there was a suspicion of uveal tissue seen through the thin superficial