optic discs but the nerve fibre layer appeared normal and the discs were flat (Fig 1). Serum haematology and biochemistry were normal with the exception of a raised alkaline phosphatase. Computed tomography (Fig 2) showed a midline cystic tumour with irregular edges and fluid levels which involved sphenoid, ethmoid, and maxillary sinuses. This was felt to be consistent with fibrous dysplasia and sphenoid mucocele.

Surgical decompression was undertaken the same day. A large cavity formed by the expanded sphenoid was entered via a transethmoidal approach, and an ostium fashioned from it into the nasal cavity. Histological examination of the wall of the cavity showed no evidence of malignancy and was characteristic of mucocele.

Ten days after surgery, her vision improved to 3/60 right, 6/12 left, with a centrocecal scotoma in the right eye and a paracentral scotoma in the left eye. One month after surgery her vision was 6/9 in each eye with small paracentral scotomas, and the appearance of the optic discs was unaltered. One year after surgery, vision was 6/9 in each eye.

COMMENT

Sphenoid mucocele may produce clinical syndromes by compression of the optic nerves, chiasm, oculomotor nerves, or pituitary gland. Pain, visual loss, and diplopia are the commonest symptoms.4 Differential diagnoses include pituitary tumour, cranio-pharyngioma, meningioma, sinus neoplasm, clivus chordoma, and internal carotid aneurysm. Computed tomography shows a homogeneous isodense or hyperdense sellar or parasellar lesion, and magnetic resonance imaging a high intensity signal on T1 and T2 weighted images.

Sphenoid mucocele has not previously been reported in Albright's syndrome. Visual loss was, however, ultimately attributed to sphenoid mucocele in one woman age 20 with isolated fibrous dysplasia, in whom an intracranial tumour had initially been diagnosed, but had failed to respond to steroids and radiotherapy.5 Bone biopsy indicated the presence of fibrous dysplasia, and a haemorrhagic sphenoid mucocele found to encircle both optic nerves at craniotomy; however, vision had been no light perception in both eyes for some time before surgery and did not improve following it. This is in marked contrast with the present case in which, despite severe visual loss before surgery, a good visual outcome was obtained because of prompt diagnosis and surgical intervention. These parallels findings in optic nerve compression by dysplastic bone, in which a better visual prognosis is associated with a short duration of visual loss.

It is important that sphenoid mucocele be recognised as a cause of visual loss in patients with Albright's syndrome. Patients are typically young, visual loss may be profound and bilateral, and prompt diagnosis and surgical decompression sight saving. A history of spontaneous fractures, endocrine dysfunction, and characteristic radiological appearances may provide diagnostic clues.


Figure 1 Appearance of optic discs at presentation.

Figure 2 Computed tomography scan showing sphenoid sinus mucocele and bony changes of fibrous dysplasia.
been described. Figure showing the left orbital aerocele and air intracranially in the left parasellar region.

antibiotics were not administered. One week later the left visual acuity was 6/6.

COMMENT

An aerocele (pneumatocele) is a loculated collection of gas under pressure which causes mass effect. This differs from emphysema in which gas is dispersed throughout the tissue. The finding of an intraorbital aerocele has been described twice, following surgery; partial middle turbinectomy complicated by chronic, air filled, epithelial lined sac extending from the ethmoid sinus into the orbit and a subperiosteal collection of air following orbital floor fracture repair.

Our case is the first reported to be caused by non-surgical trauma and to be associated with pneumocephalus (intracranial air) which adds the possible complication of intracranial infection. Pneumocephalus is most commonly caused by head trauma associated with fractures involving parasellar sinuses and resulting in dural tears. It may be the only sign of base of skull fracture which often goes unidentified. The presence of subarachnoid air indicates dural penetration.

In our case no fracture was identified. It is difficult to explain how dural and sinus penetration occurred through a stab wound in this site without causing severe ocular injury which is usual with pneumocephalus secondary to orbital penetration.

The orbital aerocele could be caused by fracture of the lamina papyracea with injury to the optic nerve sheath allowing intracranial communication. The raised intraocular pressure, secondary corneal oedema and proptosis were probably due to the pressure effects of the aerocele causing high intraorbital pressure, as opposed to soft tissue swelling or haematoma. The latter would not be expected to resolve completely in 9 hours, as occurred in this case. Such mass effect might occur if air was trapped, having been drawn or forced into the orbit under pressure at the time of injury. A 'tension aerocele' of the orbit has not been reported before. The rapid resolution of air in this case contrasts with the other cases and reflects the non-encapsulated aerocele, which no longer communicated with a parasellar sinus.

This case illustrates the importance of computed tomography scanning in the investigation of orbital trauma, demonstrating air particularly well and identifying pneumocephalus immediately, thus warning of possible life threatening complications.

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