Clinical Records

An unusual case of otogenic pneumocephalus

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Abstract

Otogenic pneumocephalus is a rare entity usually caused by temporal bone trauma. This paper describes a case of otogenic pneumocephalus of traumatic origin, in which the type of the fracture (a bony spicula was detached from the mastoid) and the location (Trautmann's triangle) were uncommon.

Key words: Pneumocephalus; Temporal bone; Injuries, non-penetrating

Introduction

The development of pneumocephalus generally requires a break in the cranial cavity which allows air to enter. In an otogenic condition this usually means a communication between the temporal bone and either the middle or posterior cranial fossa. In 1986 Andrews and Canalis analyzed 59 cases of otogenic pneumocephalus and reported as aetiological factors: trauma in 36 per cent of the cases, otitis media in 30 per cent, otological surgery in 30 per cent and congenital defects in four per cent of the cases.

Despite its rarity, pneumocephalus has to be considered whenever the dura is violated, especially if associated with a cerebro-spinal fluid (CSF) leak.

Case report

A 62-year-old Caucasian male presented to casualty, with a three-day history of otorrhoea and fullness in his right ear after sustaining head trauma resulting from a fall



FIG. 1a Axial CT scan showing a detached bony spicula of the petrous bone (arrow).

in the bath-tub. Otoscopy revealed a pulsating watery discharge coming through a small central perforation of the pars tensa of the drum. The left ear was normal. No nystagmus was noted. Audiometry showed a 35 dB conductive hearing loss in the right ear. The neurological examination was normal as was the remainder of the physical examination.

Computed tomography (CT) scan showed a detached fracture of the posterior-inferior surface of the petrous bone. A bony spicula was torn and a few bullae of air were noted to be communicating between the mastoid and the endocranium (Figures 1a and b). The patient was managed conservatively (bedrest, head-up position, antibiotic course). The day after his admission, he blew his nose hard although he had been advised not to. Immediately after this, he complained of severe headache requiring strong medication to calm. His neurological examination remained nonfocal. He was afebrile, awake and alert. Repeat CT scan in combination with magnetic resonance imaging (MRI) revealed wide pneumatization of the



FIG. 1b Coronal CT scan showing a few air bullae in the temporal region (arrow).

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Fig. 2

CT scan picture showing wide pneumatization of cerebral ventricles, after nose blowing.

cerebral ventricles (Figure 2). The patient underwent a mastoidectomy approach which revealed the fractured point at the posterior dural plate in Trautmann's triangle. The spicula was removed and the defect repaired with a temporal fascia sandwich graft across it, held in place with fibrin glue. The mastoid cavity was obliterated by fat. The patient's symptoms resolved two days later. The otoscopic examination, 10 days later, showed a dry central perforation of the tympanic membrane. It healed in three weeks. The patient has reported no problems since. A CT scan, one month later showed the mastoid obliteration and the healing of pneumocephalus.

Discussion

Pneumocephalus, or aerocoele, is defined as the presence of air within the cranial cavity. Temporal bone fracture is the most common cause of otogenic pneumocephalus. The pathophysiology involves the presence of a craniodural fistula through which the air can enter, and a pressure gradient facilitating the movement of air. There are two principal mechanisms that may cause such a pressure gradient: The 'ball valve' effect (Bhimani et al., 1985) and the 'inverted-bottle' effect (Lunsford et al., 1979). The first mechanism assumes that air is forced through the cranial opening by a variety of measures that act to increase nasopharyngeal pressure, such as nose blowing, straining or coughing. The intracranial pressure increases, forcing the brain and dura over the fistula and trapping the air within. The second mechanism requires the presence of a profuse CSF leak which results in the development of negative intracranial pressure allowing the lost fluid to be replaced by air.

This case is unusual in the type and location of the fracture as well as the severity of air pressure developed within the middle ear cleft. A bony spicula from the mastoid was detached and the meninges of the posterior cranial fossa (Trautmann's triangle) was torn, in close proximity to the cerebellomedullary cistern. This resulted in persistent CSF otorrhoea, which is more often found after injury of the anterior cranial fossa. The injury of the posterior cranial fossa rarely causes the development of pneumocephalus.

The predisposition in this case was that the patient was a 'vigorous nose blower' and that there was direct communication between the mastoid and the cistern. The violent nose blowing allowed air to pass through the Eustachian tube to the middle ear and, via the mastoid defect, directly to the cistern filling up the ventricles with air via the foramina of Luschka and Magendie. The mechanism is of the 'ball-valve effect'. The air pressure gradient in this case must have been very high, considering that passage of air through the Eustachian tube (even with a Valsalva manoeuvre) is difficult and especially because, in this case, the air was able to escape from the perforation of the tympanic membrane.

Signs and symptoms of pneumocephalus may be quite minor and nonspecific (Orebaugh and Margolis, 1990). The diagnosis is usually established by CT scan. This imaging modality is extremely sensitive and can identify as little as 0.5 ml of air in the intracranial spaces (Osborne *et al.*, 1978). The treatment of pneumocephalus is dependent on its aetiology and whether or not it is symptomatic. Aerocoele after trauma is often associated with a CSF leak. The therapeutic approach to traumatic pneumocephalus is similar to that for a CSF fistula. Initial therapy is usually conservative. Surgical intervention is indicated for continued CSF leakage, progression of the pneumocephalus, or if a tension condition exists (Andrews and Canalis, 1986).

In the above case, the continued CSF leakage and the progress of the pneumocephalus were the factors that indicated surgical intervention.

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