Tension pneumocephalus complicating ventriculoperitoneal shunt for cerebrospinal fluid rhinorrhoea: case report

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SUMMARY A case of spontaneous nontraumatic cerebrospinal fluid rhinorrhoea secondary to aqueductal stenosis is reported. The patient required direct repair of the fistula after the insertion of a ventriculoperitoneal shunt for aqueductal stenosis. We emphasise an unusual complication of tension pneumocephalus in a case where the shunt patency had been substantiated. Intracranial pressure fall due to the siphon effect in the ventriculoperitoneal shunt tubing in the erect position might be responsible for ingress of an excessive amount of air.

In the great majority of patients, cerebrospinal fluid rhinorrhoea and pneumocephalus occur after head injuries. These phenomena are attributable to various lesions in the cranial and intracranial structures when trauma is not the cause, and indicate incontrovertibly a fistula connecting the intracranial space with the extracranial. On rare occasions, pneumocephalus may present signs and symptoms of cerebral compression.

Case report

This 22 year old girl was admitted to hospital on 7 May 1975 with a four week history of watery discharge from her left nostril. Her left hand had become clumsy four months before admission, and vision in both eyes had been failing progressively.

EXAMINATION

Neurological findings were normal except for a mild ataxia on the left side on finger-to-nose testing. Ophthalmological examination revealed bilateral optic atrophy and decreased visual acuity (VD=0.04, VS=0.02). There was no apparent weakness, sensory loss, or abnormal reflexes.

Routine laboratory studies were within normal limits. Clear fluid from the nostril contained a cell count of 23 WBC/mm³ with 7% polymorphonuclear and 93% mononuclear cells, 0.294 g/l of protein and 3.4 mmol/l (62 mg/dl) of sugar. Skull radiographs showed an enlarged sella turcica with demineralised dorsum sellae, separation of the coronal sutures, and prominent digital markings. A left common carotid angiogram and a right retrograde brachial angiogram demonstrated marked dilatation of the lateral ventricles. A RISA cisternogram showed no evidence of abnormal downward passage of isotope in the frontobasal area. Ventriculography, which disclosed an intraventricular pressure of well over 200 mmH₂O, demonstrated marked dilatation of the lateral and third ventricles and a porencephalic cyst in the left frontal lobe communicating with the anterior horn of the left lateral ventricle. The aqueduct was completely obliterated near its origin, and a small dip was recognised in the floor of the posterior third ventricle (Fig. 1). No contrast material was found in the posterior cranial fossa.

OPERATION

A right ventriculoperitoneal (VP) shunt was established using a medium pressure Pudenz valve. Postoperative course was uneventful, and cerebrospinal fluid (CSF) rhinorrhoea ceased entirely. Air injected into the lumbar subarachnoid space after VP shunting filled the fourth ventricle and the aqueduct. However, air did not enter the third ventricle (Fig. 2).

SECOND ADMISSION

The patient was readmitted three months later with a motor weakness of the left extremities.
Fig. 1 Air and meglumine iothalamate ventriculogram, lateral view, shows an aqueductal occlusion and the markedly dilated lateral ventricles (arrows).

Fig. 2 Midline tomogram of pneumoencephalography shows obliteration of the aqueduct at its superior part.

Neurological examination revealed a mild spastic hemiparesis with Babinski response on the left side. There was no sensory deficit, and she had no recurrence of CSF rhinorrhea. The Pudenz valve appeared to be functioning normally and there was no evidence of shunt malfunction. Skull radiographs at the second admission showed a massive intracerebral and intraventricular pneumocephalus (Fig. 3). There were no symptoms suggestive of infection, and the routine laboratory studies were
within normal limits. A radioisotope ventriculography, involving 100 μCi RISA injected into the right lateral ventricle by puncturing the Pudenz flushing device, showed enlarged ventricles and extraventricular activity in the left frontal lobe. Radioisotope was washed out in 24 hours. The patient was prescribed complete bedrest and her neurological signs gradually subsided in a week. Repeat skull radiographs at that time showed that the intraventricular air was remarkably reduced.

SECOND OPERATION
A left frontal craniotomy was performed. A plug of protruding brain which had herniated into the frontal sinus was excised after the fistulous tract between the porencephalic cyst and the frontal sinus had been identified. The bony opening was repaired with fascia and tissue adhesives. Postoperative course was uncomplicated and the left hemiparesis disappeared. The patient was discharged from hospital and has been asymptomatic for 24 months.

Discussion
CSF rhinorrhoea occurs most commonly as the result of head injury. Less prevalent nontraumatic CSF rhinorrhoea has also been well described (Ommaya et al., 1968; Schechter et al., 1969; Obrador, 1972). A pneumocele is not an invariable accompaniment of CSF leakage. The present case is uncommon because (1) the CSF rhinorrhoea appeared to be secondary to aqueductal stenosis, (2) CSF diversion might have contributed to the induction of a pneumocephalus, and (3) a tension pneumocephalus occurred in spite of the lack of shunt malfunction.

Nontraumatic CSF rhinorrhoea associated with an aqueductal stenosis is an uncommon phenomenon (Rovit et al., 1969; Schechter et al., 1969; Nishikawa et al., 1972; Little et al., 1975). Little and MacCarty (1976) reported a case similar to the present one which developed a tension pneumocephalus after the insertion of a VP shunt for aqueductal stenosis. Various methods of CSF diversion have been carried out for high pressure rhinorrhoea (Ommaya et al., 1968; Little et al., 1975) as well as for persistent CSF rhinorrhoea (Kaufman, 1968; Greenblatt and Wilson, 1973). Little et al. (1975), however, reported that two out of four cases with CSF rhinorrhoea associated with aqueductal stenosis required direct repairs of the fistulae after shunting procedures. In the present case, repair of the fistula was carried out because the patient developed a pneumocephalus. A CSF fistula secondary to aqueductal stenosis might be less inclined to heal spontaneously for unknown reasons.

Air can gain access to the intracranial cavity only when there is a break in basal structures in
connection with the paranasal sinus, and when the nasal air pressure exceeds the intracranial pressure (Jones, 1970; North, 1971; Magnaes and Nornes, 1972). In the present case, an artificial reduction of the intracranial pressure, as Greenblatt and Wilson (1973) pointed out, appeared to have facilitated the passage of air into the ventricular system through a fistula that connected the frontal lobe which in turn opened to the anterior horn of the lateral ventricle.

Magnaes and Nornes (1972) recorded continuously the intracranial pressure in two patients with traumatic pneumocephalus and found an accumulation of CSF in the period of a closed CSF fistula to be the main cause of an acute tension pneumocephalus. It is of particular interest that, in the present case, tension pneumocephalus occurred presenting signs and symptoms of brain shift even though the shunting system was working well. The results of clinical examinations and radioisotope ventriculography seemed to substantiate the patency of the VP shunting system. The specific mechanism of the tension pneumocephalus in the present case remains unknown. The fact that simple bedrest relieved the pneumocephalus, however, would suggest a possible explanation. The fistula may have acted as a one way valve because the patient showed no evidence of recurrence of CSF rhinorrhea at the second admission. In addition, if a siphon effect had taken place in the VP shunting system, it would seem reasonable to believe that the intracranial pressure could sometimes fall to subatmospheric levels in the erect position, and air might, in the normal course of events, have passed inward in a volume exceeding that of the drained CSF due to an excessive drop of the intracranial pressure. Thus a volume phenomenon from the excessive amount of air which has been drawn inward by an extreme drop in pressure and entrapped by a valvular mechanism might well have contributed to a tension pneumocephalus in the present case.

Marked ventricular dilatation and high intraventricular pressure together with the CSF rhinorrhea prompted us to resort to a shunt operation which, however, did not cure the fistula. Direct surgical repair of the fistula would appear to be the treatment of choice for spontaneous cerebrospinal fluid leakage.

References


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