Otogenic tension pneumocephalus caused by therapeutic lumbar CSF drainage for post-traumatic hydrocephalus: A case report

Edwin K. Chan, MD; Lawrence Z. Meiteles, MD

Abstract

Tension pneumocephalus occurs when a continuous flow of air accumulates in the intracranial cavity and produces a mass effect on the brain. We describe a case in which tension pneumocephalus was caused by the performance of continuous lumbar CSF drainage in a middle-aged man who had experienced a temporal bone fracture. Continuous lumbar CSF drainage is commonly performed in patients with temporal bone or basilar skull fractures to treat concomitant post-traumatic CSF rhinorrhea, CSF otorrhea, and/or hydrocephalus. However, to the best of our knowledge, there has been no previously reported case of tension pneumocephalus occurring as a complication of this procedure in a patient with a temporal bone fracture.

Introduction

Tension pneumocephalus secondary to head trauma is an uncommon but serious condition in which air steadily accumulates in the intracranial cavity and produces a mass effect on the surrounding brain parenchyma. This condition is potentially life-threatening, and it should be included in the differential diagnosis of a patient whose functional mental status has deteriorated following head trauma. Basilar skull fractures, in particular, may be complicated by pneumocephalus. Pneumocephalus is said to be of otogenic origin when a head trauma involving the temporal bone results in the creation of a fistulous communication between the middle or posterior cranial fossa and the pneumatized temporal bone.

Case report

A 43-year-old man experienced severe blunt trauma to the head when his all-terrain vehicle rolled over; he had not been wearing a helmet. He arrived at the emergency room intubated and responsive to pain. On physical examination, he exhibited raccoon eyes, significant edema and ecchymosis over the left parietal scalp, and Battle’s sign over the left mastoid area. Otologic examination revealed a tympanic membrane perforation with active drainage of clear discharge. No evidence of rhinorrhea was noted on rhinoscopy, and facial nerve function was found to be grossly symmetrical on grimacing during painful stimuli. The initial computed tomography (CT) scans showed a left longitudinal temporal bone fracture and developing post-traumatic hydrocephalus (figure 1).

The patient underwent placement of a continuous lumbar CSF drain for treatment of both the post-traumatic hydrocephalus and the left CSF otorrhea. His mental status improved dramatically and the CSF otorrhea resolved, but he subsequently became increasingly obtunded and
somnolent over the next 2 days. Postcisternography CT of the head detected significant pneumocephalus originating in the area of the temporal bone fracture (figure 2, A). The patient was transferred to the neurosurgical intensive care unit. Serial CTs showed that the pneumocephalus was progressing rapidly and that it had extended into the ventricles and caused cerebral compression (figure 2, B). A diagnosis of tension pneumocephalus was made, and the lumbar drain was clamped. A bur hole was made to accommodate placement of an emergent ventriculotomy drain to decompress the tension pneumocephalus.

In light of the left temporal bone fracture and CSF otorrhea, we presumed that the entry point for the air into the cranial cavity was the mastoid bone, probably through defects in the tegmen tympani and dura. The patient was brought back to the operating room for an exploratory mastoidectomy and exploration of the middle cranial fossa. Using the middle cranial fossa approach, we found a 3 ×
2-cm tear in the dura overlying the tegmen tympani and a comparably sized bony defect in the tegmen tympani itself. The dura was repaired with a fascia lata graft and fibrin glue, and the tegmen defect was covered from above with a split calvarial bone graft.

Examination from below through the mastoidectomy revealed comminution of the tegmen tympani fracture with multiple subcentimeter openings in the bone in addition to the dominant bone defect. The decision was made to repair the remaining tegmen defects with hydroxyapatite bone cement. Most of the mastoid cavity was also obliterated with the cement, with care taken to preserve the middle ear cleft.

Postoperatively, the ventriculotomy drain was clamped, and serial CT scans revealed that the pneumocephalus had resolved. The patient was restarted on continuous lumbar CSF drainage for hydrocephalus, with close monitoring of the drainage rate. The lumbar drain was subsequently replaced by a lumbarperitoneal shunt for long-term management of the hydrocephalus. No further complications occurred, and the patient’s neurologic status improved progressively.

Discussion

When head trauma cases are narrowed down to basilar skull injuries only, the finding of pneumocephalus increases tenfold. Markham’s review of pneumocephalus cases revealed that trauma was overwhelmingly the leading cause (73.9% of cases), with tumor (12.9%), infection (8.8%), surgery (3.7%), and unknown etiology (0.7%) accounting for the remainder of the cases. Andrews and Canalis found that trauma was also the most common cause of otogenic pneumocephalus (36% of cases), followed by otitis media (31%), otologic surgery (31%), and congenital defects (2%).

As for the cause of pneumocephalus, two primary mechanisms have been proposed: the ball-valve effect and the inverted-soda-bottle effect:

• The ball-valve effect occurs when air is forced from an air-containing extracranial space (e.g., the nasopharynx or the middle ear and mastoid air cell system) into an intracranial cavity through a fistulous connection. An increase in pressure from the extracranial space may occur as a result of a Valsalva maneuver, sneezing, a straining cough, or nose blowing. The air travels in only one direction (extracranially to intracranially), and as air enters the cranial cavity, the intracranial pressure rises. The pressure gradient is therefore reduced, and the brain and dura tamponade against the fistulous connection, trapping the air intracranially. If subarachnoid adhesions are present, air may travel intracerebrally and create a pneumocele, or it may travel through the foramina of Luschka and Magendie into the ventricular system.

• The inverted-soda-bottle effect occurs when negative intracranial pressure is induced by a loss of CSF through a leak or iatrogenically via a lumbar drain. As a result of the negative pressure gradient from the extra- to the intracranial cavity, air is drawn in through a bony or dural defect in the skull base. The air replaces the CSF as the pressure in the two cavities equalizes. This phenomenon is analogous to what occurs when a soda bottle is inverted; when air enters the bottle, bubbles form as the soda drains out. This was the effect that was responsible for the development of tension pneumocephalus with intraventricular extension in our patient.

The symptoms of tension pneumocephalus are the same as those of increased intracranial pressure; they include headache, confusion, lethargy, nausea, and vomiting. Other neurologic signs—such as visual-field deficits, seizures, and behavioral changes—have also been documented. Because these symptoms are so vague, pneumocephalus is often unsuspected. However, because pneumocephalus is a potentially lethal condition, it is important to consider it in the differential diagnosis of any head trauma patient with deteriorating mental status. The diagnosis can be readily established by CT, which is an extremely sensitive diagnostic tool because it can detect as little as 0.5 ml of air in the intracranial cavity.

The treatment of tension pneumocephalus secondary to excessive CSF drainage includes interruption of the inverted-soda-bottle mechanism, which in our case meant simply clamping the continuous lumbar CSF drain. Surgical intervention to relieve the increase in intracranial pressure induced by pneumocephalus must be undertaken promptly. Creating an exit route for the intraventricular air in an acute setting can be quickly accomplished by performing a ventriculotomy and placing a ventriculotomy drain. Next, the fistulous connection between the air in the extracranial cavity and the intracranial space must be identified. (In our case, the defect was revealed via an exploratory mastoidectomy and middle cranial fossa exploration.) The defect, which can involve either bone or bone and dura, can then be sealed with any of a number of autologous materials (e.g., fascia, muscle, fat, or bone) or with various synthetic sealants and cements.

Tension pneumocephalus secondary to continuous CSF drainage can be prevented by closely monitoring the rate of drainage and avoiding overaggressive drainage:

• A passive gravity-dependent drainage system allows for fluctuations in the rate of drainage whenever there is any change in the position of the drain chamber in relation to the position of the patient. Such a change can result in rapid drainage, so vigilant monitoring is imperative.

• Overdrainage can be avoided by keeping the rate of CSF drainage less than the rate of CSF production. CSF is

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