Two Cases of Delayed Tension Pneumocephalus

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We describe two cases of tension pneumocephalus, one caused by ventriculoperitoneal shunt for communicating hydrocephalus, and the other caused by craniocebral trauma. In the first case report, we examined the relationship between cerebrospinal fluid leakage and delayed onset tension pneumocephalus. The second case report, we addressed issues such as the diagnosis, management, and pathogenesis, as well as computerized tomography (CT) findings.

KEY WORDS: Tension pneumocephalus · Ventriculoperitoneal shunt · Craniocebral trauma.

Introduction

Non-tension pneumocephalus is a common consequence of cranial surgery, and is a relatively benign complication that is usually resolved spontaneously. By contrast, tension pneumocephalus mimics an expanding intracranial space-occupying lesion and may lead to rapid deterioration of the patient, thereby requiring prompt treatment. Symptoms include restlessness, confusion, disorientation, and hiccoughs. Anisocoria, hemiparesis, and signs of meningeal irritation can also be observed.

Henceforth, we describe two cases of tension pneumocephalus, arising from trauma and after ventriculoperitoneal shunt.

Case Report

Case 1

A 64-year-old male patient presented with a confused state at the emergency room. On CT examination subarachnoid hemorrhage was seen in the basal cistern, interhemispheric, and both sylvian fissures (Fig. 1A). Angiography revealed a cerebral aneurysm of the anterior communicating artery, which was treated by cerebral aneurysm clipping (Fig. 2B). Post-operatively, he exhibited a semi-comatose state, and CT revealed intracerebral and intraventricular hemorrhages, for which extraventricular drainage was performed.

Then, his neurological symptoms were gradually improved. However, his mentality was aggravated from lethargy to obtundation in the second post-operative month and CT and radioisotope cisternography were examined (Fig. 2A). He was diagnosed with communicating hydrocephalus type IV and gradually exhibited psychological depression. He underwent a ventriculoperitoneal shunt (Fig. 2B), and became aroused.

On postoperative day 15, he exhibited psychological depression and CT revealed Mt. Fuji sign and severe compression of the bilateral frontal lobes by severe pneumocephalus (Fig. 3A). After two-burr-hole trephination in right frontotemporal area was done to remove air, he became aroused (Fig. 3B). On postoperative day 20, pus was discharged at the site of incision for cerebral aneurysm clipping. Analysis of his cerebrospinal fluid (CSF) showed: WBC 90/ul, RBC 3/ul. Analysis of his blood showed: ESR 96mm/hr and CRP 13.1mg/dl. After onset of a seizure, he fell into a coma.
Delayed Tension Pneumocephalus

Fig. 2. A: Radiosotope cisternogram shows a type IV communicating hydrocephalus. B: Ventriculoperitoneal shunt is performed and postoperative brain computerized tomography shows no accumulation of air.

Fig. 3. A: Fifteen days postoperative brain computerized tomography scan shows prominent accumulation of air in the subdural space. The Mt. Fusi sign is seen. B: Preoperative brain computerized tomography scan shows the drainage tube, which is inserted in right frontal area via an existed Burr hole (arrow).

Reoperation through the previous incision, abscess and inflammatory tissues remained extradurally and the dura was not opened. We did not note the spread of inflammation into the intracranial cavity. The frontal sinus, which had been filled with bone wax during the surgery for the cerebral aneurysm, was reopened, because it was the assumed etiologic site for the inflammation and the tension pneumocephalus.

Inflammatory tissue and the frontal sinus mucosa were removed, and the opened frontal sinus was filled with Methylmetacrylate® (Fig. 5A). Preoperatively, the patient had no abdominal tenderness or abdominal pain. Intraoperatively, no inflammation was noted in his abdominal cavity. Accordingly, the ventriculoperitoneal shunt was maintained. Postoperatively, he showed improvement in his symptoms. At this time, analysis of his CSF showed: WBC 5/ul, RBC 3/ul. The analysis of his blood showed: ESR 10mm/hr, CRP 0.58mg/dl. Based on the follow-up brain CT scan (Fig. 5B), the patient had no notable findings apart from a slight increase in ventricle size, so was then discharged.

Case 2
A 38-year-old female patient had a history of cranial fracture resulting from an accident that occurred 12 years ago, at which she underwent conservative therapy. Since then, she had manifested nasal discharges and intermittent generalized seizure. She visited our outpatient clinic with the primary complaint of persistent generalized seizure, headache and decreased muscle strength on the left side. Brain magnetic resonance imaging (MRI) revealed a low-attenuation balloon-like appearance in the right frontal lobe (Fig. 6).

Intraoperatively, it was noted that a spur was penetrating into the basal portion of the right frontal lobe through the dura mater, and it was removed. The penetrating dura matter was reconstructed with galea and the bone defect of skull basal portion was also covered with galea (Fig. 7), and a catheter was inserted. Postoperatively, she displayed no nasal discharge and pneumocephalus which are manifested preoperatively, and did not develop generalized seizure.

Discussion

Chiari® is credited with the first description of intracranial air, detailed in an autopsy report in 1884; the patient had
ethmoiditis and developed an intraventricular 'air collection' following severe sneezing. With the advent of roentgenography nearly 30 years later, Luckett\textsuperscript{5} first described an intracranial pneumatocele on X-rays of the skull.

'Pneumocephalus' was a term coined by Wolff\textsuperscript{20} in 1914. Dandy's experience with pneumocephalus led to his introduction of pneumoencephalography as a major diagnostic procedure in 1918\textsuperscript{8}. 'Tension pneumocephalus' was first described in 1962 by both Ectors\textsuperscript{5,10} and Kessler and Stern\textsuperscript{13}.

Aside from a diagnostic procedure, pneumocephalus mostly develops due to trauma and accompanies fracture of the frontal and ethmoidal sinuses. In addition to these etiologies, infection\textsuperscript{14}, brain tumor\textsuperscript{2}, anesthesia\textsuperscript{9} and brain surgery\textsuperscript{17} are known causes of pneumocephalus. In most cases, pneumocephalus is cured by conservative treatment.

However, many cases of large tension pneumocephalus in the intracranial cavity, characterized by headache, neurological deterioration and abrupt change of mental status, require surgical treatment\textsuperscript{2,7,9}.

Diagnosis of pneumocephalus requires the use of methods including simple X-ray, brain CT and brain MRI. Using brain CT, niveau formation, a crescent shape, biconvex shape and peaking sign were noted in an asymptomatic form of subdural pneumocephalus\textsuperscript{11,13}; Mt. Fuji sign was also noted in tension pneumocephalus\textsuperscript{2,11,13,9}. Mt. Fuji sign was produced by gradual influx of air between the bilateral frontal lobes and filling the anterior and lateral portions of these two lobes\textsuperscript{11}.

The Mt. Fuji sign has not been noted in some cases of tension pneumocephalus that exhibit niveau formation\textsuperscript{16}. The present case also exhibited tension pneumocephalus with a balloon appearance. Air bubbles had accumulated in the arachnoid cistern, indicating that air transported via the injured arachnoid membrane to several sites within the cerebral cisternae by a patient's head motion, which was more notable in tension pneumocephalus than the asymptomatic form of pneumocephalus\textsuperscript{4,11,16}.

Case 1 exhibited diffuse air bubbles in the cerebral cisternae, although this was not noted in Case 2. This finding indicates that arachnoid injury might not sustained in Case 2. The pathogenesis of tension pneumocephalus is explained by the following four hypotheses: (1) 'Inverted soda pop bottle' phenomenon\textsuperscript{7,18}. That is, continuous leakage of CSF (siphon effect) results in negative intracranial pressure, thereby allowing replacement of lost fluid by air (comparable with emptying a narrow-necked bottle by inversion). (2) 'Ball-valve mechanism'\textsuperscript{2}. That is, air enters the intracranial cavity through a defect whenever extracranial pressure exceeds intracranial pressure (e.g., from the paranasal sinuses during coughing, sneezing, swallowing, straining). Egress of air is then prevented as intracranial tissues under pressure block the site of entry. Such a mechanism may repeat itself until a relatively large volume of air is collected. (3) A third mechanism of tension pneumo-
Pneumocephalus development has been described but it is unique in that it is confined purely to the intraoperative period. Specifically, there is a role played by nitrous oxide (N2O) anesthesia, in particular when administered to a patient with entrapped intracranial air. N2O will diffuse into an air-filled cavity 34 times faster than nitrogen diffuses out. Thus, the volume and pressure of the contained pneumatocele rapidly increases (mimicking brain swelling). (4) Finally, there is a possibility of gas-forming bacteria contributing to the development of pneumocephalus. In Case 1, ventriculoperitoneal shunt was performed for hydrocephalus in the presence of an incompletely closed frontal sinus that had been opened during surgery for cerebral aneurysm. Accordingly, CSF was released into the peritoneal cavity, and the resultant pressure difference caused tension pneumocephalus, based on the 'inverted pop bottle effect'. In Case 2, dural injury, originating from a spur generated by fracture of the cranial base, released CSF, thereby resulting in pneumocephalus based on the 'ball-valve theory'. Delayed treatment for tension pneumocephalus could exert pressure on the brain, thereby resulting in neurological symptoms or intracerebral hemorrhage.

Accordingly, tension pneumocephalus should be managed immediately after being detected. The treatment methods include: (1) simple aspiration through burr holes, (2) use of a closed water-seal drainage system to promptly reduce the intracranial pressure and to maintain the persistent pressure gradient between intracranial and extracranial for closure of the dural fistula, and (3) reconstruction of the injured sites.

In Case 1, tension pneumocephalus was managed through burr hole trephination, but inflammation was noted via the opened frontal sinus. During the revision surgery for removal of inflammatory tissue, granulation tissue filled the site of the former dural suture. Moreover, severe inflammation of galea and temporal muscle was observed. Preoperative enhanced brain computerized tomography revealed the contrast-enhanced lesion, interpreted as intracranial inflammation on previous operation site (Fig. 4). Because of inflammation of galea, we could not use the galea flap to cover the opening of frontal sinus. Accordingly, Methylmetacrylate was used to fill the site of the frontal sinus. In Case 2, the spur was removed from the basal portion of the frontal lobe using a bifrontal approach. Further, a catheter was inserted to lower the intracranial pressure, and the dural injury was repaired using galea.

Conclusion

In the present case report, we describe one case of delayed tension pneumocephalus after ventriculoperitoneal shunt, and the other case of pneumocephalus resulting from trauma that occurred 12 years ago, and also provide a review of the literature. In cases which change in mental status and neurological symptoms are detected, brain CT should be performed to diagnose and enable immediate management of tension pneumocephalus. To prevent secondary infection, the etiologic site should be located and managed.

References

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