Subdural tension pneumocephalus

Report of two cases

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Two patients developed subdural tension pneumocephalus after undergoing posterior fossa surgery performed in the sitting position. The mechanism for entry of air into the intracranial compartment is analogous to the entry of air into an inverted soda-pop bottle. As the fluid pours out, air bubbles to the top of the container. We have thus referred to this as the “inverted pop-bottle syndrome.” Computerized tomography provided prompt diagnosis and confirmed brain displacement. Twist-drill aspiration of the air resulted in improvement in both patients, although one patient subsequently died from an intracerebellar hemorrhage. Tension pneumocephalus appears to be another potential complication of posterior fossa surgery in the sitting position. This condition is easily diagnosed and treated, and should be considered whenever a patient fails to recover as expected following posterior fossa surgery.

KEY WORDS □ pneumocephalus □ posterior fossa surgery □ surgery in sitting position □ computerized tomography

DETECTION of intracranial gas or pneumocephalus is usually of pathological significance only when no previous diagnostic or surgical procedures have been performed.6,8-12 If present in sufficient volume and under pressure, subdural intracranial gas may produce mass effect and neurological deficit (tension pneumocephalus).3 We recently encountered two patients who developed tension pneumocephalus after undergoing posterior fossa surgery performed in the sitting position. Computerized tomography (CT) was used to detect the presence of tension pneumocephalus. Neurological improvement followed prompt twist-drill aspiration of air from the subdural space.

Case Reports

Case 1

This 69-year-old right-handed woman was admitted for treatment of debilitating glossopharyngeal neuralgia. Past history disclosed evidence of chronic obstructive lung disease and healed peptic ulcer disease. She had previously undergone cholecystectomy and excision of a lymphoid cyst at the base of the left epiglottis. Blood pressure and general physical examination were normal. The neurological examination revealed a rapid, fine tremor of all extremities at rest. No cranial nerve deficit was detected. Otolaryngological evaluation, skull x-ray films, and computerized tomography (CT) were normal.

The patient underwent a right retromastoid craniectomy in the sitting position. Complete transection of the ninth and partial section of the tenth cranial nerves were performed. Asymptomatic air embolism occurred during the procedure, and was treated by intermittent withdrawal of 40 cc of air through a catheter previously positioned in the right atrium. Anesthesia consisted of a balanced technique of barbiturate, narcotic, muscle relaxant, and 50% nitrous oxide.

After surgery the patient awakened from anesthesia. One hour postoperatively she became lethargic and unresponsive to verbal commands. No lateralizing neurological deficit was noted. A CT scan disclosed massive bifrontal subdural air (Fig. 1 left), and a right frontal twist-drill hole was immediately placed. Upon penetration of the dura, air escaped under pressure. A flexible polyethylene catheter was introduced, and in fractional amounts, a total of 260 cc of air was evacuated. Repeat CT showed re-expansion of the lateral ventricles, and reduced subdural air
FIG. 1. Case 1. Appearance of the brain on computerized tomography scanning before (left) and after (right) twist-drill aspiration of 260 cc air from the right frontal subdural space. Note the "re-expansion" of the cerebral hemispheres following treatment of tension pneumocephalus.

No intracranial hemorrhage was seen. Immediate neurological improvement followed. The patient responded to verbal commands and other than depressed sensorium, no focal deficit was present.

Fifteen hours after surgery, however, her blood pressure rapidly increased to 180/100 mm Hg, and she became deeply comatose with bilateral decerebrate posturing and fixed miotic pupils. Immediate posterior fossa exploration was performed and a 2 x 2½ cm intracerebellar hematoma was evacuated. No neurological improvement followed surgery and she died 48 hours later. At postmortem examination, massive cerebral edema, cerebellar tonsillar herniation, and a right hemorrhagic cerebellar infarct were noted. In addition, an unsuspected and unexplained acute left temporo-occipital intracerebral hematoma was discovered.

Case 2

This 56-year-old right-handed man was admitted for treatment of trigeminal neuralgia uncontrolled by medical therapy. Past history disclosed insulin-dependent diabetes mellitus and Type IV hyperlipidemia. General physical examination revealed labile hypertension and mild obesity. The neurological examination was completely normal. Skull x-ray films and a CT scan disclosed no abnormality.

With the patient in the sitting position, a right retromastoid craniectomy and microvascular decompression of the fifth cranial nerve was performed. Anesthesia consisted of a balanced technique, using barbiturate, narcotic, muscle relaxant, and 50% nitrous oxide. Asymptomatic air embolism was detected by Doppler monitoring intraoperatively and treated by aspiration of 7 cc of air through a right atrial catheter.

Two hours postoperatively, the patient was noted to be lethargic and not easily arousable. Lateral gaze paresis in both eyes, anisocoria, left hyperreflexia, and a left Babinski response were noted. Immediate CT scanning disclosed a massive bifrontal subdural collection of air (Fig. 2 left). Six hours after surgery, the patient became unresponsive and developed Cheyne-Stokes respiration and a marked left hemiparesis. Immediate bifrontal twist-drill holes were made. Air escaped under pressure when the dura was penetrated on the right side. Approximately 40 cc of air was then aspirated from the left side. Repeat CT scanning disclosed a slight reduction of the subdural air (Fig. 2 right). Within 4 hours he was more alert and followed simple commands. At 24 hours, a left hemiparesis, left lateral gaze paresis, and anomic aphasia remained. Serial lateral skull radiography performed over the next 3 days revealed disappearance of the pneumocephalus. No recurrence of his facial pain has been noted. He was discharged 7 days postoperatively in a stable and normal neurological condition.

Discussion

Chiari is generally credited with the original descriptions of intracranial pneumocephalus in 1884, when he demonstrated a fistulous connection between the ethmoid air cells and the frontal lobe. Lecat actually reported the first case of cranial "aerocele" in 1741. In 1913, Luckett was the first to demonstrate post-traumatic intracranial air radiographically. Since then the diagnostic importance of intracranial gas has been stressed by many reviewers. Pneumocephalus results from trauma, infection due to...
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gas-forming organisms, tumors eroding the skull base, and congenital defects of the skull; it is also reported following air encephalography, lumbar puncture, and intraventricular drainage.\textsuperscript{6,8,12} Pneumocephalus may be classified as subarachnoid, intracerebral, intraventricular, extradural, or subdural.\textsuperscript{6,12}

Intracranial air is diagnosed readily by plain skull radiography; however, even amounts of air as small as 0.5 cc can be detected by CT.\textsuperscript{11} Because of its extremely low attenuation coefficient (\textasciitilde1000 Hounsfield), air appears as a region of very low density with a white rim surrounding it.\textsuperscript{4,11} Computerized tomography has been particularly useful in detecting pneumocephalus following trauma.\textsuperscript{4,8} Osborn, et al.,\textsuperscript{31} have described the radiographic appearance of subdural air.

In this report, two patients developed progressive neurological deterioration after undergoing posterior fossa surgery performed in the sitting position. As cerebrospinal fluid is evacuated intraoperatively, air enters the potential subdural space. This effect may be likened to inverting a soda-pop bottle. Air bubbles to the top of the pop bottle, just as air rises to the top of the subdural space when the patient is in the upright sitting position. With closure of the operative wound, air is loculated intracranially, and eventually dissected frontally due to the usual supine position of the patient following surgery. A CT scan reveals mass effect and severe anteroposterior cerebral distortion. Such distortion may result in focal deficit or generalized neurological deterioration associated with increased intracranial pressure.\textsuperscript{4} Nitrous oxide will rapidly diffuse into a cavity to equilibrate the partial pressure of the nitrous oxide in the cavity with that in the blood.\textsuperscript{13,14} Thus, nitrous oxide anesthesia may potentiate tension pneumocephalus by increasing the volume of the intracranial gas. Other hazards of posterior fossa surgery performed with the patient in the sitting position have been detailed by Albin, et al.\textsuperscript{1}

Frontal twist-drill holes revealed air under pressure in both patients. Upon penetration of the dura, air escaped as if a balloon were deflated. Prompt aspiration of air was followed by immediate improvement in the CT appearance of the brain, and gradual neurological improvement. Unfortunately, the first patient subsequently died from the effects of a hypertensive cerebellar hemorrhage which occurred hours after surgery. After treatment of the pneumocephalus, repeat CT disclosed no hemorrhage. The left temporooccipital hemorrhage was an unexpected and unexplained autopsy finding, remote from both the original operative site and the subsequent twist-drill hole. Five other supratentorial intracerebral hemorrhages following posterior fossa surgery have been described by Haines, et al.\textsuperscript{8} As in this case, the hemorrhage was remote from the operative site. The etiology remains obscure, although transient hypertension may be implicated. Possibly the marked cerebral distortion associated with tension pneumocephalus might produce this type of hemorrhage. The second patient made a complete neurological recovery within 5 days postoperatively.

Subdural intracranial air under pressure may produce severe neurological deficit by distortion and displacement of the normal brain. A rapid diagnosis is possible with CT. Prompt reduction of pressure and evacuation of the air may result in a gratifying neurological recovery, providing no further complications arise. Tension pneumocephalus may occur in the presence of symmetrical, bilateral subdural gas accumulation, and may be potentiated by the use of nitrous oxide anesthesia. Tension pneumocephalus should be considered whenever a patient deteriorates after posterior fossa surgery has been performed in the sitting position.

References


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