Postmastoidectomy Pneumocephalus: Case Report

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ABSTRACT

Chronic otitis media, unlike trauma, seldom leads to the development of tension pneumocephalus. Rarely, it occurs as a complication of mastoid surgery. A Melanesian woman sought treatment for loss of speech, hemiparesis, and headache 4 weeks after undergoing canal-down mastoidectomy for cholesteatoma. The tension pneumocephalus was decompressed urgently by aspirating air via a cannula through the burr hole, and the mastoid cavity was obliterated. She recovered completely. Twelve additional patients with postmastoidectomy pneumocephalus reported in the literature are reviewed.

KEYWORDS: Mastoidectomy, pneumocephalus

Air can accumulate in the intracranial cavity after trauma, bone erosion, or operative injury. The most common cause of such pneumocephalus is trauma. Likewise, pneumocephalus as a complication of chronic otitis media¹ or mastoid surgery is rare. Tension pneumocephalus develops from the continuous accumulation of intracranial air and leads to life threatening complications. Only 12 such cases appear to have been reported in English and non-English literature combined.²⁻¹⁰ This report describes a patient who developed tension pneumocephalus and serious intracranial complications after undergoing canal-down mastoidectomy

for cholesteatoma and reviews the 12 cases reported in the literature.

CASE REPORT

A 65-year-old woman sought treatment in the Emergency Department of the Port Moresby General Hospital complaining of loss of speech, diminished movement of the right side of the body, and 2 weeks of headache. Four weeks earlier she had undergone a canal-down mastoidectomy with wide

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Figure 1 Contrast-enhanced axial CT showing a tension pneumocephalus. Air collection in the left temporo-parietal region is causing subfalcine herniation; the air is surrounded by CSF medially (big arrowhead) and posteriorly (smaller arrowhead).

meatoplasty on the left side for extensive cholesteatoma. One week after the mastoidectomy, the packing was removed from the mastoid cavity and she was discharged in satisfactory condition. The surgical report noted accidental exposure of the dura in the tegmen antri and the use of monopolar cauterization to stop the bleeding from the exposed dura.

Examination of her left ear revealed a watery discharge in the left mastoid cavity that was identified as cerebrospinal fluid (CSF) by a glucose test. Neurological examination revealed expressive aphasia and right-sided hemiparesis. The remainder of her systemic examinations and her hematological tests were within normal limits. Computed tomography (CT) showed air collected in the left Sylvian fissure associated with mass effect, effacement of the left lateral ventricle, and midline shift (Figs. 1 and 2). The third ventricle, suprasellar cistern, and

Figure 2 Sagittal CT reconstruction image showing the location of the air in the left Sylvian fissure.

superficial cortical sulci also were effaced due to increased intracranial pressure (ICP) indicating the presence of tension pneumocephalus.

An emergency burr hole was made and a large amount of air was aspirated from the temporal and parietal lobes via a cannula. A tegmen antri dehiscence (1-cm diameter) was detected. The left mastoid cavity was obliterated with the pedicled temporalis muscle-fascia flap. The left external auditory canal was packed with BIPP (bismuth iodine paraffin paste). Over subsequent weeks, her expressive aphasia and right-sided hemiparesis resolved completely. One year after surgery she had no complaints.

REPORTED CASES

A review of the literature from 1950 to 2001 showed 12 cases of postmastoidectomy pneumocephalus (Table 1). There were eight females and five males. Of the 13 patients (including the present patient)

one was in the second, two were in the fourth, four were in the fifth, three were in the sixth, two were in the seventh, and one was in the eighth decade of life. Headache was the most common clinical symptom followed by altered sensorium, ear discharge, and aphasia. The interval between mastoidectomy and presentation ranged from 1 day to 4 weeks. Intracranial air was present in the parenchyma, ventricle, and subdural and subarachnoid spaces alone or in different combinations. Of these 13 patients, 11 were managed surgically and two were treated conservatively with bed rest and antibiotics. Five patients underwent neurosurgical repair and five underwent combined intracranial and transmastoid approaches. One patient underwent an exclusive transmastoid repair. Full recovery was achieved in all patients except the youngest, a 13-year-old girl, who died 3 months after her hospitalization. This patient's death was possibly caused by a rapid massive reaccumulation of the air within the temporal lobe abscess followed by markedly elevated ICP.

DISCUSSION

Typically, the development of tension pneumocephalus requires a fracture in the bone through which air can enter and a pressure gradient that facilitates the movement of air. Two principal mechanisms can cause such a pressure gradient. First, a "ball-valve" effect is created by intermittent episodes of increased pressure within the middle ear caused by equalization maneuvers, nose blowing, or sneezing.² Such a mechanism would entrap a large amount of intracranial air. The second mechanism is illustrated by the "inverted-bottle" analogy. Just as air replaces fluid leaving an inverted bottle, air replaces leaking CSF in the intracranial compartment.¹¹ This mechanism underlies worsening tension pneumocephalus caused by closed drainage of CSF, as with a subarachnoid catheter, or by a lumbar puncture.⁷ Nitrous oxide anesthesia also can increase the volume of a pneumocephalus intraoperatively.^{11,12}

Horowitz² postulated an additional mechanism for developing negative ICP: a change in head position from horizontal to vertical. The resulting negative pressure pulls air through the ball-valve while a compensatory increase in CSF absorption creates space for the trapped air. Moreover, a small amount of air may be associated with infections involving gas-forming organisms.¹ Our patient had a CSF leak but no features of infection.

Different combinations of these four mechanisms (ball-valve effect, inverted-bottle effect, postural change, and infection by gas-forming organisms) may be responsible for the accumulation of intracranial air leading to the formation of tension pneumocephalus.¹³ ICP increases in response to the mass effect of the air.⁷ If the pressure is not relieved, the situation can rapidly become fatal.⁷ Pneumocephalus produces no significant symptoms unless it is under tension.¹⁴

There may be a latent period before the symptoms of increased ICP manifest with or without the features of meningitis. Our patient had a latent period of 4 weeks before she sought treatment. At the beginning, headache, lethargy, disorientation are common. These unexplained clinical features in a postoperative otologic patient should be viewed with suspicion.

Lumbar puncture or other therapies to lower CSF pressure can be dangerous and should be avoided in the presence of tension pneumocephalus.⁷ More serious clinical manifestations appear as tension increases. The situation may become worse if infection compounds the tension. As little as 0.5 ml of air can be readily identified on CT, allowing rapid and accurate identification of possible catastrophic complications.¹⁵

The development of pneumocephalus after mastoid surgery will most likely be a consequence of the surgery. During the mastoid procedure, a defect in the mastoid bony boundary and, more seriously, in the dural layer may be created and overlooked. In our patient, the use of monopolar cauterization to arrest bleeding from the exposed dural area might have led to necrosis of the meningeal

Table 1 Summary of Reported Patients with Postmastoidectomy Pneumocephalus

Author(s)	Age (yr)/ Sex	Clinical Features and Interval Between Mastoidectomy and Presentation	Location of Pneumocephalus	Surgical Pathology	Treatment/ Follow-up
Horowitz ²	32/F	Headache, neck stiffness 2 weeks	Temporal	Accidental trauma to middle fossa dura; dural exposure in tegmen and sinodural angle with granulation in latter and cranially directed fistulous tract	Transmastoid removal of dural granulation and repair with pedicled temporalis muscle flap. <i>Full recovery</i>
Alonso and Gill ³	73/M	Headache, dysequilibrium, dysphasia, confusion, neck stiffness, +ve Romberg's and Kernig's sign 18 days	Frontal	Dura exposed in tegmen	Conservative. <i>Full recovery</i>
Wortzman and Holgate ⁴	48/F	Headache, nuchal rigidity, upper quadrant hemianopia, bradycardia, hypotension 4 days	Temporal	Area of cerebritis in temporal lobe	Intracranial excision of area of cerebritis and repair. <i>Full recovery</i>
	45/M	Headache, disorientation, drowsiness, nuchal rigidity, fever, CSF otorrhoea 2 weeks	Temporal	Brain herniation into mastoid cavity through dural and tegmen defect	Intracranial repair of dural and tegmen defect with fascial graft. <i>Full recovery</i>
Moore et al. ⁵	59/M	Otalgia, CSF otorrhoea, disorientation, pupils bilaterally constricted and sluggish light reflex, nuchal rigidity 3 weeks	Frontal and parietal	Dural granulation in tegmen; fistula between middle ear and petrous ridge	Transmastoid removal of dural granulation and repair with temporalis muscle fascial graft, mastoid cavity packing by abdominal fat; V-P shunt. <i>Full recovery</i>
Young and Frazee ⁶	39/F	Otalgia, headache, vomiting, nuchal rigidity, hemiparesis, dysphasia, seizure	Temporal	Encapsulated temporal lobe abscess communicating with mastoid cavity through 2-3 mm dural effect	Intracranial excision of abscess and repair of dural defect by pericranial graft. <i>Full recovery</i>

13 F	Otalgia, headache, nausea, vomiting, drowsiness, dilated fixed pupils, bilateral papilledema, flaccid paralysis, respiratory arrest 2 weeks	Temporal	Pus and gas-filled abscess communicating with mastoid cavity through 6–8 mm dural defect	Burr hole and removal of pus and gas; intracranial excision of abscess; repair of dural defect by pericranial graft. <i>Died after 3 months</i>
Matsuba et al. ⁷	Headache, lethargy, disorientation, bizarre behavior, neck rigidity 5 days	Temporal	Accidental dural tear at tegmen tympani	Intracranial decompression of pneumocephalus and sub-dural repair of dural defect. <i>Full recovery</i>
40/F	Headache, lethargy, expressive aphasia 3 days	Temporal	0.4 x 0.5 cm dehiscence of tegmen; temporal lobe abscess	Intracranial excision of abscess and repair of tegmental defect with temporalis muscle fascial graft. <i>Full recovery</i>
Andrews and Canalis				Conservative.
60/F	Otorrhoea instability, memory loss 12 days	Temporal	Accidental dural tear	<i>Full recovery</i>
68/M	Headache, CSF otorrhoea, disorientation 11 days	Temporal and frontal	Tegmen antri dehiscence of 1 cm diameter with dehiscent sinus plate	Trephining and burr hole and closure of fistula by muscle and fibrin glue. <i>Full recovery</i>
Artazkoz del Toro et al. ⁹				
50/M	Headache, somnolence, fever, stupor, neck rigidity, CSF otorrhoea 24 days	Temporal and frontal	Tegmen tympani erosion with dural defect and temporal lobe abscess	<i>Full recovery</i> Craniotomy by middle fossa approach and aspiration of pus; dural and bony defect repaired by lyophilized dura and acrylic resin, respectively; subarachnoid drain inserted; revision mastoidectomy for granulation and mastoid cavity lined by temporalis fascia. <i>Full recovery</i>
Obon et al. ¹⁰				
Present Case	65/F Headache, CSF otorrhoea, aphasia, hemiparesis 4 weeks	Temporal, frontal, and parietal	Accidental exposure of 1 cm diameter of dura at tegmen antri and cautery to stop bleeding from it	Burr hole and decompression of pneumocephalus and obliteration of mastoid cavity by temporalis muscle-fascia flap. <i>Full recovery</i>

layers during the postoperative period. The resulting fistula between the intracranial spaces and the middle ear and mastoid system then permitted the pneumocephalus to form. The dura and arachnoid of the middle cranial fossa are closely adherent, facilitating the creation of a surgical defect through both layers.^{5,16} The underlying subarachnoid space in the middle fossa is also large, and air can flow from the basal cistern into the ventricular system through the foramina of Luschka and Magendie.^{5,16}

There are reports of conservative management with successful results by preventing or containing intracranial infection,^{3,8} but surgical repair is associated with the best outcomes.⁸ Several methods can be used¹⁷: (i) fascial graft of the dural defect and packing of the mastoid cavity with abdominal fat; (ii) radical mastoidectomy with an obliteration procedure; (iii) inferiorly based pedicled tissue flap, if the cellular development of the mastoid cavity is not extensive; or (iv) intracranial closure. If the defect is closed satisfactorily, the CSF leak should cease in the immediate postoperative period. Failure to close the leak not only leads to continued pneumocephalus but also allows a pathway for intracranial infection.⁶

If possible, a dural defect should be closed through an extracranial approach, which is associated with the lowest rates of morbidity and mortality.⁵ Neurosurgical consultation is recommended if intracranial repair is needed.⁵ Neurosurgical intervention may be more advantageous than a simple transmastoid closure of the leak. The former helps to reduce the tension by aspirating air, pus, or both from the accumulated area and to excise the wall of the abscess. Most of the patients reviewed in this article underwent neurosurgical closure or a combination of neurosurgical closure and transmastoid repair.

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Commentary

This article is relevant to the neuro-otologic and neurosurgical community, particularly because this complication is relatively uncommon. The authors elucidate possible pathophysiologic mechanisms and satisfactorily review the cases reported in the literature.

When working on a dural surface, it is essential that only bipolar cauterization be applied

because the dura is often exposed during surgery for chronic otitis media. A violation of the dura needs to be addressed at surgery. At discharge, all patients who have undergone extensive petrous bone microdissection for chronic otitis media with or without cholesteatoma should be educated about the signs and symptoms that can portend the onset of an intracranial complication. The value of this article is underscored by reminding all clinicians that while neurologic sequelae seldom follow ear surgery for chronic infections, they can occur and cause serious complications.

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