
Severe Tension Pneumocephalus Complicating Frontal Sinus Osteoma

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ABSTRACT: Background: Tension pneumocephalus, the accumulation of intracranial gas under pressure, is a rare but potentially life-threatening condition which can complicate craniofacial surgery, trauma, or cranial tumor. It presents as an acute or subacute expanding mass lesion. **Case Report:** We present a case of a 40-year-old male who developed tension pneumocephalus as a consequence of a previously detected but untreated frontal sinus osteoma. Despite prompt decompression and repair of the fistulous connection between the sinus and the intracranial compartment, the patient suffered permanent frontal lobe damage with significant neurocognitive sequelae and seizures. **Conclusions:** This case illustrates that tension pneumocephalus can be a dangerous entity with potential for early mortality and long-term morbidity. We recommend, therefore, early treatment and close follow up of destructive lesions involving the posterior frontal sinus wall.

RÉSUMÉ: Pneumo-encéphale sévère compliquant un ostéome du sinus frontal: à propos d'un cas. Introduction: Le pneumo-encéphale sous tension, une accumulation de gazes sous pression, est une pathologie rare mais potentiellement mortelle qui peut compliquer une chirurgie crâniofaciale, un traumatisme ou une tumeur crânienne. Il se présente comme une lésion expansive aiguë ou subaiguë. **Histoire de cas:** Nous présentons le cas d'un homme de 40 ans qui a développé un pneumo-encéphale sous tension à la suite d'un ostéome du sinus frontal déjà connu mais non traité. Malgré une décompression rapide et une réparation de la fistule entre le sinus et le compartiment intracrânien, le patient a subi un dommage permanent au lobe frontal avec des séquelles neurocognitives importantes et des crises d'épilepsie. **Conclusion:** Ce cas illustre que le pneumo-encéphale sous tension peut être une condition dangereuse avec un potentiel de mortalité précoce et de morbidité à long terme. Nous recommandons donc un traitement précoce et un suivi serré des lésions destructrices impliquant la paroi postérieure du sinus frontal.

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Pneumocephalus is defined as an intracranial collection of gas in subdural, subarachnoid, intraventricular, extradural or intracerebral compartments. Tension pneumocephalus, or the accumulation of intracranial gas under pressure, has numerous etiologies, and can present with neurologic symptoms that mimic other more common space-occupying lesions. We describe a patient who presented with symptoms related to tension pneumocephalus secondary to an osteoma of the frontal-ethmoid sinus. We discuss the implications for sinus osteoma management and emphasize the potential for long-term morbidity from this life-threatening complication.

CASE PRESENTATION

A 40-year-old male was incidentally found to have a right ethmoid sinus tumor, felt to most likely represent an osteoma. He refused surgical intervention. Approximately ten years later, he presented to the Emergency Department with a four day history of bifrontal headache, confusion and behaviour changes. The patient reported sneezing four days prior and immediately thereafter experienced a severe headache. He had continued to work, but co-workers noted occasional incoherent speech and prolonged lapses in attention. He was brought to hospital when he was found lost in a friend's backyard.

There was no history of fever, stiff neck, nausea or vomiting, bladder or bowel disturbance, seizure, gait disturbance or recent trauma. There was no history of drug or alcohol abuse.

On examination, the patient was alert but disoriented to person, time and place, and attention was impaired. He was unable to recall recent world events. Fundi were normal. Neurological examination was otherwise unremarkable.

CT scan of the head showed an enormous left frontal pneumocephalus compressing the left lateral ventricle with posterior displacement of the left frontal lobe. There was a calcified lesion in the right ethmoid sinus penetrating the cribriform plate with extension to the floor and posterior wall of the right frontal sinus (Figure).

The patient was admitted to the neurosurgical service. A Richmond screw was placed to evacuate the intracranial air and he subsequently became more lucid. He stated that several days prior to the onset of this headache he had noted a watery nasal discharge. He underwent removal of the sinus lesion through a frontal craniotomy. Intraoperative findings included a breach of the posterior wall of the right frontal sinus, with an associated dural defect. An osteoma with a base in the anterior right ethmoid labyrinth extending into the frontal sinus was incompletely removed. The dural defect was repaired and overlaid with fascia lata. The frontal sinus was packed with fat.

The post-operative course was marked by difficulty with concentration, preventing his return to work. In formal psychometric testing and

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neurologic follow-up two years later, he showed decreased psychomotor speed and mental flexibility on the trail-making test, poor attention, reduced fluency, and mild right sided arm and leg weakness. These findings reflected the persisting left frontal lobe damage. Concomitant impairment of left manual dexterity, spatial planning and visuospatial problem-solving suggested right frontal lobe dysfunction as well. Follow-up imaging by MRI two-and-a-half years after presentation demonstrated extensive left frontal encephalomalacia (Figure D). SPECT scanning showed decreased perfusion of the left frontal, basal ganglia and temporal areas and a more subtle defect in the right frontal lobe. The patient remained unable to work because of his cognitive difficulties.

The patient developed complex partial and secondarily generalized seizures seven months after presentation, which have been difficult to control fully on dual therapy with Epival and Tegretol at therapeutic doses. Repeated electroencephalograms demonstrated left temporal slowing.

DISCUSSION

We feel that two useful points can be made with this case. First, while tension pneumocephalus is widely recognized as a neurosurgical emergency, little attention is paid to the long term neurologic sequelae which can be disabling. Secondly, such disability may be preventable by early treatment and regular follow

up of high risk lesions involving the posterior frontal sinus wall.

Tension pneumocephalus may present with severe symptoms in less than 24 hours, or symptoms slowly progressive over weeks or months. Symptoms reflect mass effect and increased intracranial pressure.

A communication between the environment and intracranial cavity, and a pressure gradient favouring the inflow of air are the necessary preconditions for the development of tension pneumocephalus. Craniofacial surgery, trauma and a destructive tumor, cause the vast majority of cranial defects, almost always in the paranasal sinus or pneumatized petrous temporal bone regions.^{1,2} CSF leakage, including CSF shunting and/or increased intranasal pressure in sneezing, coughing or straining provide the pressure gradient. Accumulation under pressure occurs presumably because of a ball-valve effect of tissue adjacent to the defect.^{3,4}

In a review of 295 cases of pneumocephalus, 75% of the cases followed craniofacial surgery and trauma, and 13% were attributable to tumors. Osteoma of the frontal and/or ethmoid sinuses accounted for the majority of the tumors.⁵ Over the past 12 years, 11 case reports of pneumocephalus complicating sinus osteoma have appeared in the literature. Rare causes are

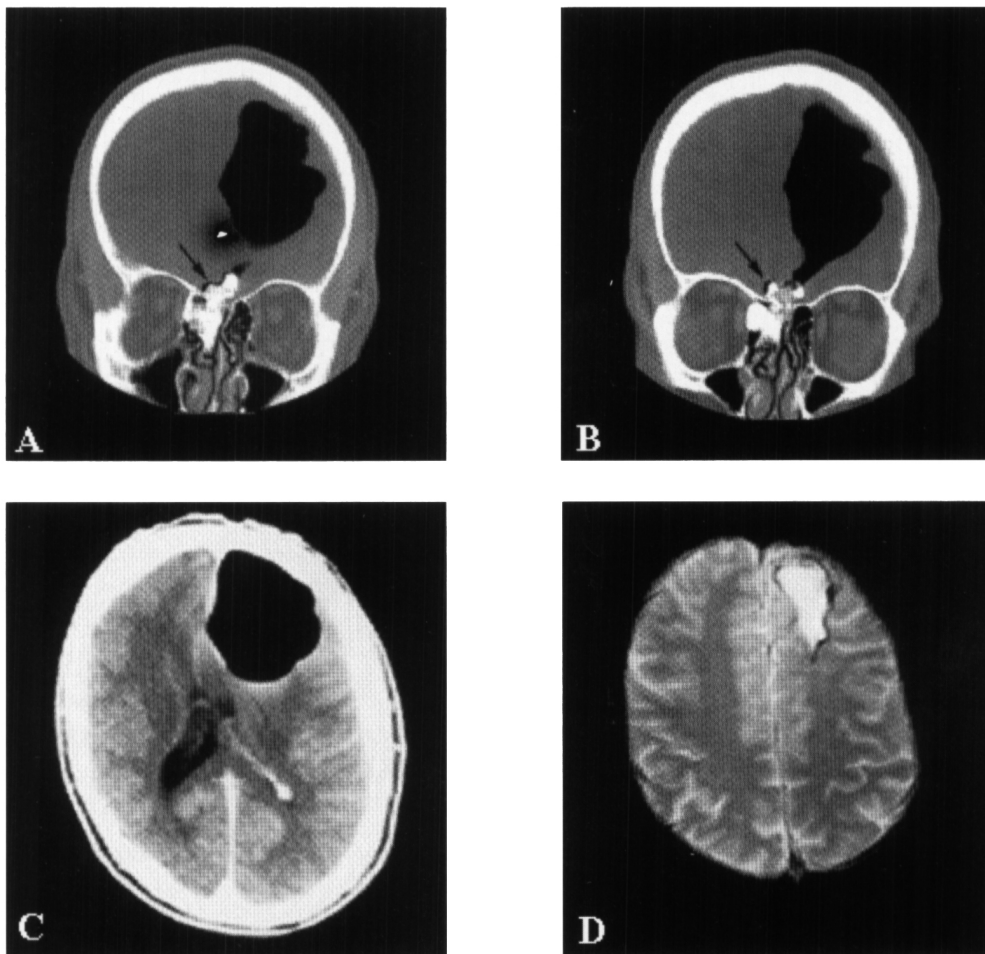


Figure: A+B) Coronal images demonstrates the osteoma in right frontal-ethmoid sinus (arrows) with intracranial extension to the left of midline contiguous with the intracranial air. C) Axial non-contrast CT head at presentation showing tension pneumocephalus with displacement of left frontal lobe. D) T2-weighted MR axial image demonstrating left frontal encephalomalacia at 2½ years follow up.

barotrauma,⁶ bronchopleural-subarachnoid fistula following thoracotomy,⁷ encephalocele rupture,⁴ nasal intubation⁸ and epidural catheter placement.⁹ In patients with cranial defects from surgery or trauma, CSF shunting,^{10,11} nitrous oxide anaesthesia or nasal CPAP¹² may produce tension pneumocephalus.

Management of asymptomatic pneumocephalus not causing mass effect may be conservative. Most post-traumatic and post-operative pneumocephalus resolves without specific treatment. Tension pneumocephalus presenting as an expanding space-occupying lesion, with patient deterioration, is treated with high-flow, high concentration oxygen by mask to decrease volume acutely by expediting nitrogen diffusion from the aerocele. This method of management can only be employed for about 48 hours because of the risk of oxygen toxicity. Emergency surgical decompression is the treatment of choice if long term neurological sequelae are to be avoided. Needle decompression is effective but tension pneumocephalus may quickly recur. A closed water seal drainage system for four to six days, as described by Arbit et al., can keep the pneumocephalus safely decompressed.¹³ We concur that closed drainage of air is an ideal immediate measure. Definitive surgery including removal of skull base lesions, as well as dural repair may then be deferred briefly to a more appropriate time. Use of the Richmond screw (Codman and Shurtleff Inc., Randolph MA) facilitates connection to a closed drainage system and has not, to our knowledge, been previously described. Closed drainage of pneumocephalus via a well placed Richmond screw appears to be an ideal method of achieving an immediate decompression.

The distribution of intracranial air in the present case deserves comment. Pneumocephalus was contralateral to the sinus osteoma because its intracranial extension projected to the left side of the midline, eroding the dura to create a defect on the left side of the crista galli, seen at operation. It is also of interest that the distribution of intracranial air was circumscribed (Figure A) rather than being widely distributed in the subdural space. This probably implies subdural adhesions resulting from either previous episodes of local bleeding or infection which prevented wide distribution of air within the subdural space.

Acute complications include venous air embolism and death from brainstem herniation. There have been two case reports of air embolus in this setting in the past ten years.^{14,15} As illustrated by this case, tension pneumocephalus can result in long term morbidity related to adjacent infarction and later seizure disorder. This stresses the need for prompt recognition and treatment of this disorder, and close observation of patients with small amounts of intracranial air.

Osteomas of the paranasal sinuses are relatively common neoplasms that are uncommonly symptomatic. Childrey (1939) retrospectively reviewed 3510 consecutive sinus x-rays and found an incidence of 0.43%.¹⁶ Complications of sinus osteoma include ostial obstruction resulting in infection or mucocele, and dural erosion creating a fistula with risk of pneumocephalus or meningitis.

Osteomas grow very slowly, over years to decades. Most authors do not advocate removal of asymptomatic osteomas,

although it has been suggested that osteomas growing rapidly with the likelihood of producing symptoms in the near future, should be removed.¹⁷ In addition, Rappaport et al.³ have suggested that osteomas arising from the posterior frontal sinus should be removed at an early stage because of their potential to cause intracranial complications. We suggest that any posterior frontal sinus involvement, regardless of point of origin, be an indication for early removal given the potential for tension pneumocephalus as a rare but serious complication with potential for acute mortality and long-term morbidity. Often, complete removal of osteomas is not possible and the potential for regrowth is present, necessitating continued follow up.

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