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Mastoiditis with Concomitant Pneumocephalus, Meningitis, and Frontoparietal Ischemic Infarction

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Abstract

Otogenic pneumocephalus describes intracranial free air as a consequence of mastoid effusion secondary to complicated otitis media. While the incidence of intracranial complications related to middle ear disease is low, the associated mortality rates remain particularly high. Clinical manifestations vary from asymptomatic to an acute neurosurgical emergency. Otogenic pneumocephalus is a rare condition and as a consequence lacks a well-defined clinical presentation.

We report a case of a 51-year-old female with a weeklong history of otitis media, who presented with an altered mental status and quickly deteriorated clinically. A head computed tomography study revealed a unilateral mastoid effusion and pneumocephalus, and a lumbar puncture identified pneumococcal meningitis. This case portrays a clinical assessment that is scarce in the relevant literature.

Keywords: Otogenic Pneumocephalus; Otitis Media; Otomastoiditis; Mastoiditis; Mastoid Effusion; Frontoparietal infarction.

Case presentation

A 51-year-old FM with a past medical history of IDA, HTN, morbid obesity, and a weeklong otitis media, presented with an AMS. On arrival, she was unresponsive to all stimuli, with a Glasgow coma scale of 3/15. Physical examination yielded intact cranial nerve reflexes, positive Bilateral (bl) oculocephalic reflex, bl corneal reflex, increased muscle tone in the right leg, no spontaneous movements, normal reflexes, and a negative Babinski reflex. She was intubated, sedated, and remained on mechanical ventilation.

Initial workup yielded leukocytosis (18.4/mm^3), bacterial meningitis (positive lumbar puncture), sepsis due to Streptococcus pneumonia bacteremia, otomastoiditis, and a positive influenza B infection. IV Ceftriaxone, Levofloxacin, Vancomycin and a course of Tamiflu were started. She received Zofran after an episode of bilious vomiting. Labetalol was provided for blood pressure support. Nasogastric tube feeding was delivered at a goal rate of 60 mL/h.

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The computed tomography (CT)-head with contrast (Figure 1) showed mucosal thickening within the paranasal sinuses. Mild pneumocephalus is noted with the involvement of the subdural space at the left convexity. There is left frontoparietal edema with effacement of the sulci. We appreciated partial opacification of the left mastoid air cells and the left middle ear, consistent with Otomastoiditis (Figure 2). This finding is considered the most consistent etiology for pneumocephalus.

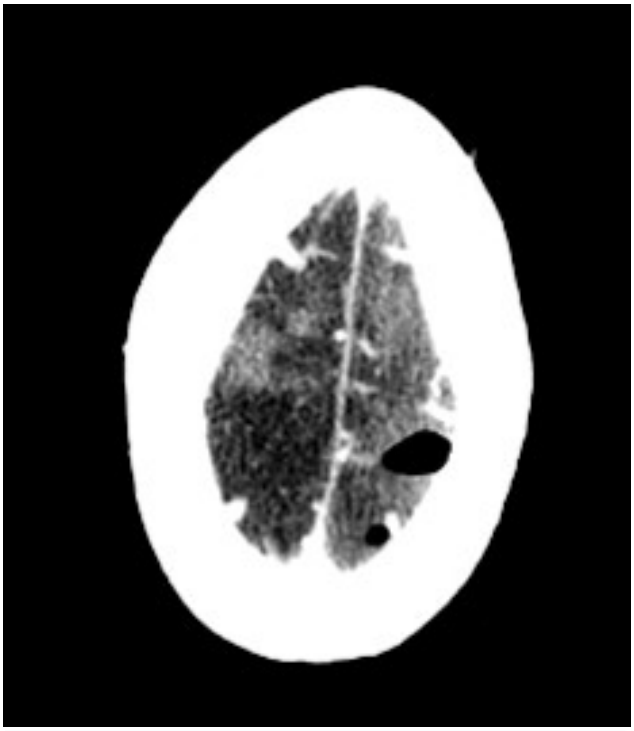

Figure 1: Axial CT-Head with contrast showing pneumocephalus.

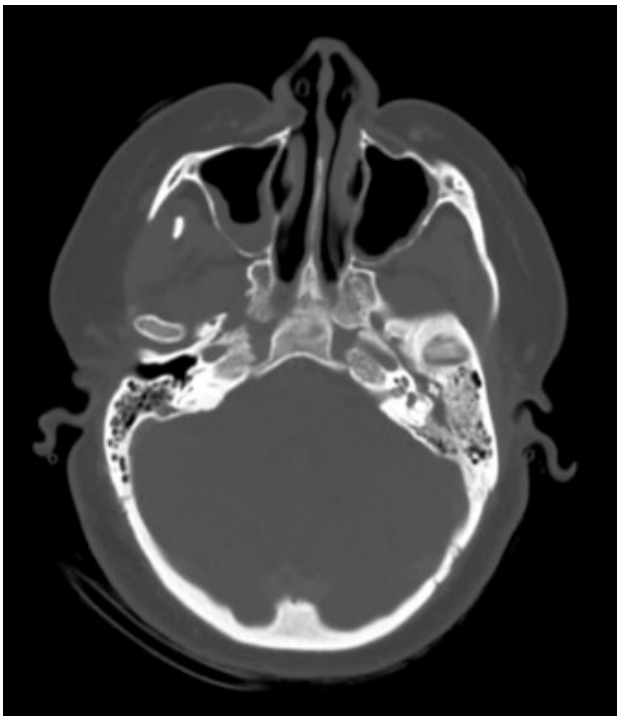

Figure 2: Axial CT showing partial opacification of the left mastoid air cells and left middle ear.

At this time the patient exhibited a Generalized Tonic-Clonic (GTC) seizure with right arm jerking, followed by right-sided hemiparesis. Video-EEG confirmed the seizure. For further evaluation, a contrast-enhanced magentic resonance imaging (MRI)- head was completed. In addition, MRI was used to correlate a diffusion weighted image (DWI) with an Apparent Diffusion Coefficient image (Figure 3a,3b). The cerebral MRI confirmed an increased signal at the left frontoparietal lobe with diffusion-weighted image. This finding was interrelated with the low signal Apparent Diffusion Coefficient image to endorse restricted diffuse as a result of acute infarction. Finally, the MR sequence underscored localized leptomeningeal and dural-enhancement along the left cerebral convexity (Figure 4). These findings are consistent with inflammatory changes that justify the clinical description of meningitis.

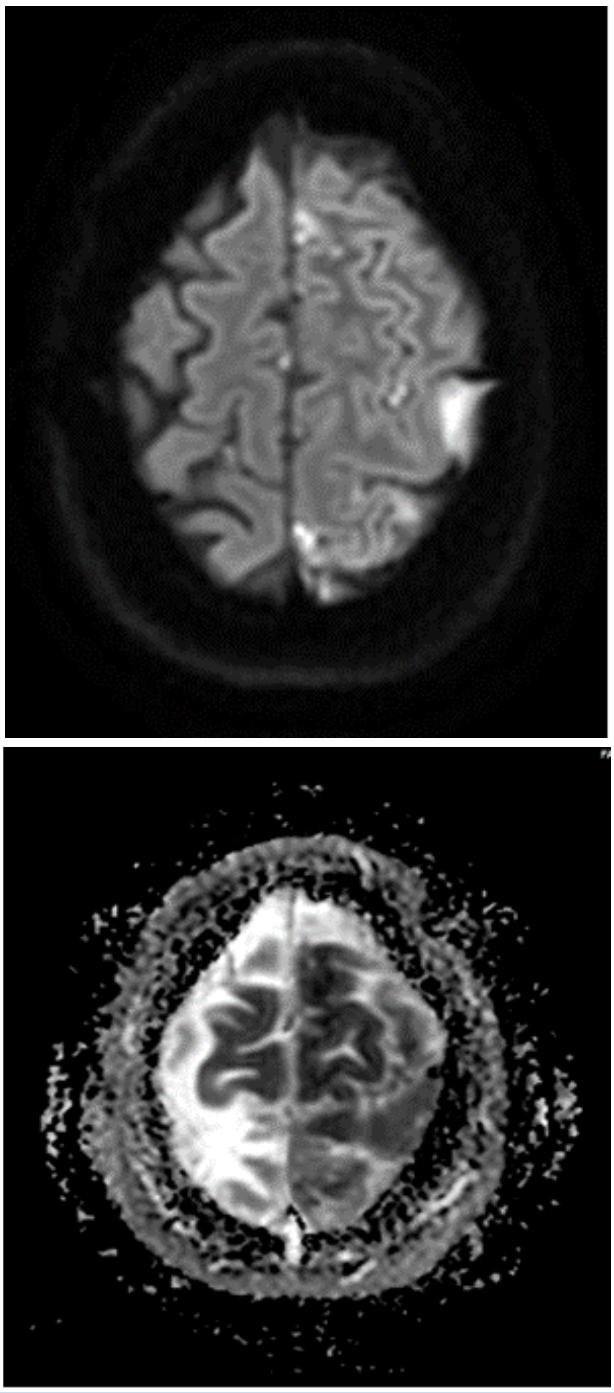

Figure 3: Axial MRI with DWI shows signal intensity.

In the left frontoparietal lobe. Axial MRI with ADC mapping shows a low restricted diffusion.

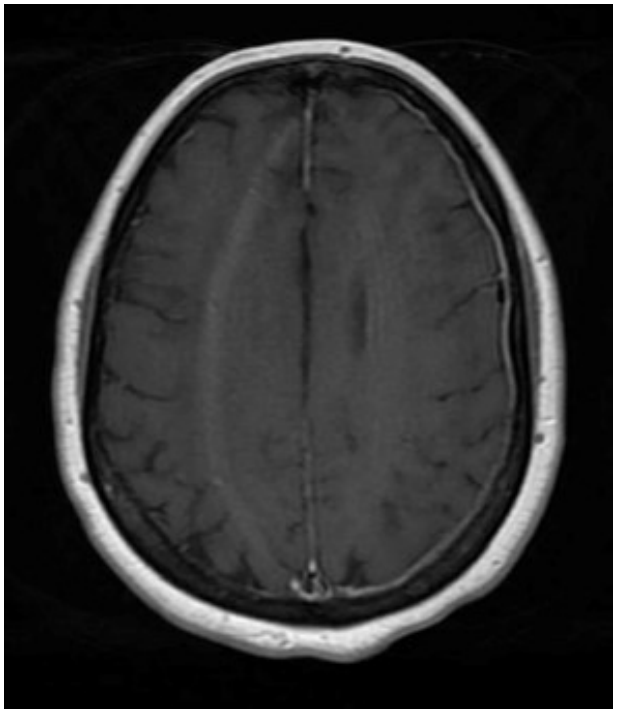

Figure 4: Axial T1 weighted MRI showing localized leptomeningeal and Dural-enhancement along the left cerebral convexity.

The patient was started on Depakote and Keppra to control generalized tonic-clonic seizures. A final CT Head with contrast was completed to evaluate the left temporal bone changes. The findings revealed marked left tympanomastoid inflammatory changes with no evidence of soft tissue abscess formation (Figure 5). Left frontoparietal edema with an enhancement of the sulci remained stable on thin- slice brain CT (Figure 6).

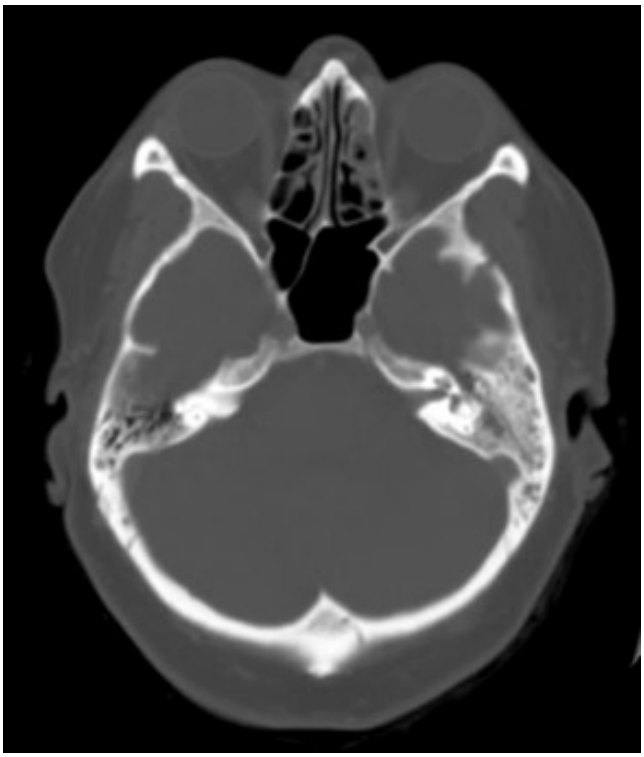

Figure 5: Axial CT showing left tympanomastoid inflammatory changes.

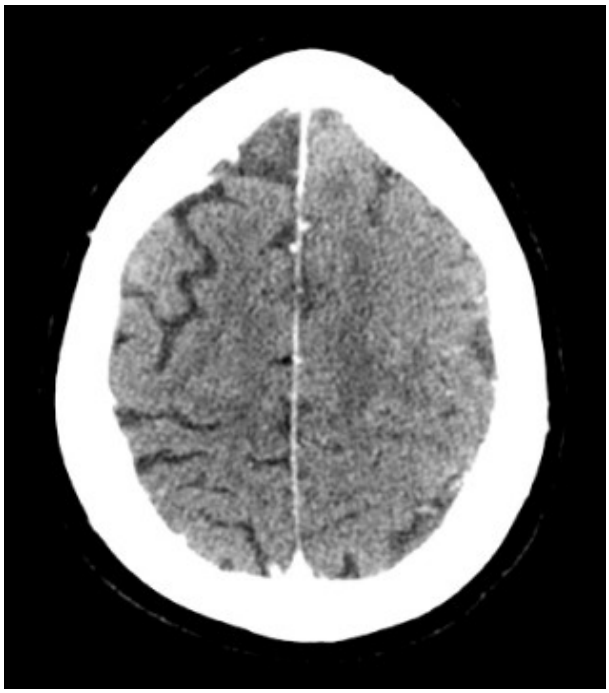

Figure 6: Axial Thin- slice Brain CT showing left frontoparietal edema with an enhancement of the sulci.

Neurosurgical intervention was not recommended, as the patient was subsequently transferred to the ICU for further management. After a gradual sedation vacation, she was awake and alert but displayed marked difficulty keeping her eyes open. She regained motor function on all extremities. She was later discharged on Ceftriaxone and Vancomycin to complete a 21 days course.

Discussion

Irrespective of medical advancement, intracranial complications of otitis media [1] continue to cause life-threatening conditions. Chronic otitis media typically results from inappropriate or incomplete antibiotic treatment. When complications arise, a multidisciplinary approach for care is highly recommended [2]. Air in the cranium, also termed Pneumocephalus, is typically reported following craniofacial trauma, neurosurgery, and atypically reported post-radiation therapy of an intrasellar tumor (air enters through sphenoid sinus and erodes cellar floor) [3]. It can also present secondarily to mastoiditis or pneumococcal meningitis, however there is a scarcity of such cases in the literature.

The physiopathological mechanism resulting in Otogenic pneumocephalus includes a pathological increase of middle ear pressure above intracranial pressure causing a breach through the posterior cranial fossa [2,4]. Intracranial infections can also yield gas as a byproduct of their metabolism [4].

Presently, the rates of Otogenic pneumocephalus remain at 0.5%-4% with a mortality rate between 5% and 5% [1]. Cranial CT without contrast is the standard diagnostic test. Cranial MRI is fundamental to evaluate the ear bone architecture to determine if mastoid effusion or epidural abscesses are present [4]. Pneumocephalus is usually asymptomatic and found incidentally on imaging. The most common clinical sequelae associated with pneumocephalus include an altered mental status, headache, hemiplegia, and seizures [2,4]. The most daunting sequelae include increased intracranial pressure with brain stem herniation. Clinical management aims to control symptoms and prevent complications but is typically dictated by the etiology.

Conservative management includes using antibiotics, while a surgical repair is reserved cases with elevated intracranial pressure [4]. The intracranial air will be absorbed within a week in 85% of patients [3,5]. However, when associated with meningitis, pneumocephalus usually has a fatal outcome [3].

Recommended antibiotic coverage for Otogenic meningitis with pneumocephalus includes the use of third generation Cephalosporin and Vancomycin for broader coverage [2]. Additionally, intravenous steroids have shown to reduce neurological sequelae by 50%, excluding hearing deficits [2].

Conclusion

Although pneumocephalus is a rare complication of untreated otitis media, and meningitis, it remains clinically elusive with predominately asymptomatic patients. Promote identification is imperative since it can progress to life-threatening sequelae. Leveraging clinical suspicion with radiological inquiry will guide the clinician to act promptly.

Consent

The patient provided verbal consent for publication of this case report and for any use of accompanying images.

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