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Surgical Neurology International

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SNI: General Neurosurgery

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Case Report

Pneumocephalus causing oculomotor nerve palsy: A case report

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Received: 04 October 2019 Accepted: 22 May 2020 Published: 25 September 2020

DOI:

10.25259/SNI_503_2019



ABSTRACT

Background: Pneumocephalus, the presence of gas or air within the intracranial cavity, is a common finding after cranial procedures, though patients often remain asymptomatic. Rare cases of cranial nerve palsies in patients with pneumocephalus have been previously reported. However, only two prior reports document direct unilateral compression of the third cranial nerve secondary to pneumocephalus, resulting in an isolated deficit.

Case Description: A 26-year-old male developed a unilateral oculomotor (III) nerve palsy after repair of a cerebrospinal fluid leak. The pneumocephalus was treated with a combination of an epidural drain, external ventricular drain (EVD), and high-flow oxygen. Following treatment, repeat computed tomography imaging of the head demonstrated that the pneumocephalus was progressively resorbed and the patient's deficit resolved.

Conclusion: In rare cases, isolated cranial nerve palsies, specifically of the third cranial nerve, can result from pneumocephalus following cranial procedures. Acute cranial nerve palsy secondary to pneumocephalus will often resolve without intervention as the air is resorbed, but direct decompression with an epidural drain and an EVD may expedite the resolution of deficits.

Keywords: Cranial nerve, Oculomotor, Pneumocephalus

INTRODUCTION

Pneumocephalus by definition is the presence of gas or air within the intracranial cavity. This collection may be localized to various compartments and potential spaces, including epidural, subdural, subarachnoid, intracerebral, or intraventricular. Although commonly found postoperatively in craniectomy or craniotomy surgical procedures, pneumocephalus is a rare finding outside of surgery, often caused by trauma (75%), infection (9%, most commonly chronic otitis media), or tumors with involvement of the intracranial space, sinuses, orbit, or nasal passages. [1] About 0.5–1% of all head traumas result in pneumocephalus. [3] Computed tomography (CT) can demonstrate as little as 0.5 mL of air compared to plain radiographic films which require nearly 4 times the volume. [7,10] There are multiple pathophysiologic mechanisms leading to the development of pneumocephalus. In postoperative cases, the leakage of excessive cerebrospinal

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fluid (CSF) causes negative intracranial pressure to draw air into the cranium.[3] Commonly, patients are asymptomatic but may develop severe headache, rhinorrhea, seizures, or, on rare occasions, focal neurologic deficits.

A review of the literature highlights a shortage of reports on isolated cranial nerve deficits related to pneumocephalus.^[6] We present a case of a patient who underwent a frontoparietal craniotomy for a postoperative CSF leak and subsequently developed a unilateral oculomotor nerve palsy as a direct result of compression from pneumocephalus.

CASE PRESENTATION

A 26-year-old male with a history of the right temporal chondrosarcoma resected in 2010 with subsequent selflimiting CSF rhinorrhea presented 8 years later with newonset left temporal headaches. MRI of his brain at this time showed a right temporal contrast enhancing lesion suspicious for recurrent intracranial chondrosarcoma measuring 2.1 cm × 1.9 cm. He was monitored at 3 month intervals with repeat MRIs which showed stability of the mass. Approximately 1 year later, he presented with new-onset seizures and MRI findings that demonstrated an increase in size to 2.4 cm × 2.4 cm with evidence of encephalomalacia of the right temporal lobe. He subsequently underwent a repeat right craniotomy for resection. Pathology confirmed the lesion was World Health Organization Grade 1 chondrosarcoma. Several weeks later, he presented with a pseudomeningocele and a history of fevers. Seventy milliliters of subgaleal fluid were drained followed by a reexploration of his cranial incision and a wound washout. At this time, a small defect was noted in the frontal bone near the frontal sinus. The mucosa of the defect was obliterated with electrocautery, packed with muscle graft, then covered with Duragen® (Integra LifeSciences) and sealed with DuraSeal® (Integra LifeSciences). A subgaleal drain was placed and discontinued 2 days postoperatively. Cultures from CSF and pseudomeningocele specimens grew MSSA and he was started on appropriate antibiotics. Two months later, he presented with positional headaches and intermittent fluid leaking from his nose that started 2-3 weeks after his wound washout. A fluid sample was collected and was positive for β_2 -transferrin. He was admitted from the clinic and a lumbar drain was placed for CSF diversion. Shortly after, he complained of severe nuchal rigidity and headaches. CT head without contrast was performed [Figure 1], which showed diffuse pneumocephalus.

The lumbar drain was clamped and the patient subsequently underwent right craniotomy which exposed a small dural opening in the temporal region, as well as a small opening in the previously repaired frontal sinus. The frontal sinus defect was repaired by removing the remaining mucosa and muscle in the sinus, and cauterizing the walls. Temporalis muscle

was packed into the sinus and Mimix® bone cement was used to close off the frontal sinus. Duragen was subsequently laid over the hardened cement as well as the temporal dural defect and sealed with DuraSeal. A larger piece of Duragen was used to cover the remainder of the surgical exposure. The bone flap was secured to the skull with titanium plates and screws. The temporalis muscle was approximated and the galea and skin were closed. CT imaging preoperatively demonstrated large frontal pneumocephalus in the right cerebral hemisphere [Figure 2a] with significant decrease in size postcraniotomy and frontal sinus packing [Figure 2b].

On postoperative day (POD) 2, the patient developed a large and minimally reactive right pupil consistent with an oculomotor nerve III palsy. Repeat imaging showed persistent right pneumocephalus [Figure 3], which was treated with high-flow oxygen.

On POD 3, the patient was found to have a right CN III and VI palsy. The patient then underwent placement of a right-sided epidural drain and external ventricular drain (EVD) with subsequent improvement in pupil size and reactivity over the next couple of days [Figure 4].

Subsequent imaging demonstrated improved pneumocephalus, and the epidural and ventricular drains were removed on days 6 and 9, respectively. The patient was discharged POD 10 with no recurrence of cranial nerve palsy. A follow-up CT scan was done on POD 15 revealing decreased amounts of pneumocephalus [Figure 5]. The patient continues to remain neurologically stable with reactive pupils bilaterally.

DISCUSSION

Pneumocephalus is almost inevitable after cranial surgery, most specifically after supratentorial craniotomies as was the case with our patient. Reasoner et al. examined 240 scans of patients undergoing supratentorial craniotomies between 1986 and 1990 and found air in 100% of the scans within the first 2 PODs.[11] Markham described two theories that explain how air enters the cranial cavity. The first is best described as a "ball-valve mechanism." If the dural meninges are violated during surgery, air can enter during the PODs and become trapped through coughing, straining, or sneezing when the dura is closed off due to elevated intracranial pressure. The other theory is when negative intracranial pressure occurs from loss of CSF and brain volume, it allows air to enter through any pathologic defect, such as an opening in a sinus as seen in our patient.[5]

Review of the literature demonstrated reports ranging from no neurological symptoms to severe neurological deficits caused by distortion and displacement of the normal brain which required emergent surgery to evacuate the air. However, there are very few case reports of pneumocephalus postcraniotomy causing isolated cranial nerve palsies.

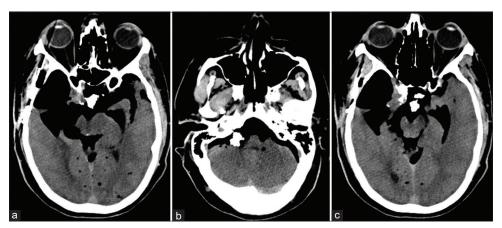


Figure 1: (a-c) Computed tomography head without contrast when the patient was admitted for nausea, vomiting, and cerebrospinal fluid rhinorrhea showing pneumocephalus in the right and left cerebral hemispheres, basal cisterns, and lateral ventricles.

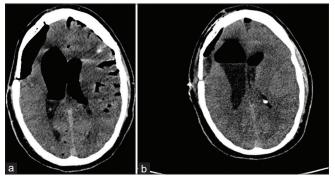


Figure 2: (a) Computed tomography (CT) head preoperatively. (b) CT head postoperatively.

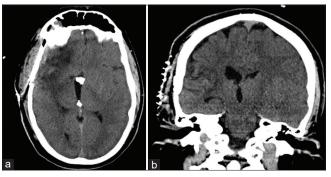


Figure 4: (a and b) Computed tomography head after improvement of pneumocephalus and subsequent improvement of palsy.

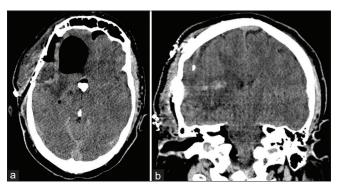


Figure 3: (a and b) Computed tomography head with pneumocephalus causing oculomotor nerve palsy.

Marupudi et al. described a case of a patient who developed a bilateral pupil-sparing oculomotor nerve palsy from pneumocephalus after a right occipital craniotomy with complete resection of the cystic pineal mass through a transtentorial interhemispheric approach. [6] It can be argued that pneumocephalus did not cause this nerve palsy, as pupil-sparing palsies are most commonly due to ischemic events and involve the nerve in its extra axial course. [9] Conversely, a nonpupil sparing palsy is due to compression

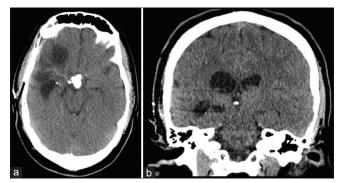


Figure 5: (a and b) Computed tomography head postoperative day 15 showing remaining pneumocephalus and resolving edema.

of the nerve's outer parasympathetic fibers that supply the sphincter pupillae.^[8] Two case studies were found involving a pneumocephalus-induced sixth nerve palsy and subsequent diplopia. Han et al. reported pneumocephalus after a craniotomy; whereas Li et al. encountered it due to an epidural injection.[4,13]

To the best of our knowledge, there are only two cases reported depicting a unilateral nonpupil sparing oculomotor

nerve palsy caused by pneumocephalus. This seems remarkably low considering the long course these nerves traverse, starting in the midbrain, and travelling along the lateral walls of the cavernous sinus to innervate the extraocular muscles and pupillary sphincter, which gives a significant amount of places for compression to occur.[14] These two prior cases occurred after an intradural Chiari I decompression and a microvascular decompression (MVD) for trigeminal neuralgia.^[2,13] In the patient who underwent the Chiari decompression, he woke up in the recovery room with a 5 mm dilated left pupil with a weak light reflex. He was immediately taken for cranial imaging which demonstrated pneumocephalus in interpeduncular and suprasellar cisterns. Interestingly, this was a transient event as his third nerve palsy resolved almost immediately after completion of imaging.^[2] In the MVD case, the patient developed rightsided ptosis with a fixed and dilated 7 mm pupil. She was also started on high-flow oxygen, with marginal improvement on discharge on POD 3. At her 3-month follow-up, her pupil was substantially improved and was completely resolved at 6 months.[12]

Our patient developed a nonpupil sparing oculomotor palsy on POD 2 with substantial improvement in pupil size after high-flow oxygen and placement of both epidural and EVD. Comparing our patient's rapid improvement to the previously mentioned patients' resolution over months, there may be an indication for an epidural and/or EVD in addition to high-flow oxygen. High-flow oxygen is thought to resolve pneumocephalus by reducing the partial pressure of nitrogen in the blood and brain parenchyma. Nitrogen gas makes up 78% of atmospheric air and is fairly insoluble in the blood. Its absorption depends on its partial pressure which is inversely proportional to the fraction of inspired oxygen. By giving 100% oxygen to treat pneumocephalus, the concentration of nitrogen gas is decreased, thus increasing its absorption concentration gradient.[12] However, in cases where pneumocephalus is causing neurological deficits, giving 100% oxygen may not work fast enough on its own, in which case an epidural drain and/or EVD would be beneficial to help facilitate the removal of pneumocephalus through negative pressure as seen in the case of our patient.

CONCLUSION

Pneumocephalus is an extremely common finding following craniotomy. This pathology is often caused by the trapping of air on closure, leakage through breaks in dural integrity, or traction secondary to negative intracranial pressure as a result of drainage. Our patient developed such severe pneumocephalus that it resulted in a unilateral oculomotor nerve palsy. Other causes of mass effect, such as tumor or hematoma, are more often associated with oculomotor nerve palsy, however, it is uncommon for pneumocephalus to cause similar symptoms. Although rare, the direct relationship between this cranial nerve deficit and pneumocephalus requires further research, as the oculomotor nerve pathway is relatively lengthy in comparison to the other cranial nerve tracts. In addition, presentation of a third nerve palsy is unique in regards to pupil and nonpupil-sparing symptomatology, which depends on central versus peripheral nerve fibers, respectively. Acute cranial nerve palsy secondary to pneumocephalus will often resolve without intervention as the air is resorbed, however, direct decompression may expedite resolution of deficits as seen in our case.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Goodrich ME, Wolberg AM, Kashyap S, Podkovik S, Bernstein J, Wiginton J, et al. Pneumocephalus causing oculomotor nerve palsy: A case report. Surg Neurol Int 2020;11:302.