

Pneumocephalus in the Absence of Craniofacial Skull Base Fracture

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We report a rare case of intracerebral pneumocephalus. It was not accompanied by the typical craniofacial skull base fracture. A 77-year-old woman presented with pneumocephalus following a pedestrian traffic accident. Neurologic and physical examination revealed multiple extensive emphysemas, multiple rib fractures, and lung contusions, but no facial or skull bone fractures. Computed tomography (CT) and simple X-ray did not reveal a craniofacial skull base fracture, although both imaging methods showed an air shadow in the internal carotid artery (ICA) pathway. Pneumocephalus and pneumoventricle are defined as an intracranial gas collection, with several reports in the literature describing various portals of entry and clinical situations that favor the introduction of air to the skull. Our report presents the possibility that pneumoventricle and pneumocephalus can occur even in the absence of a bony skull fracture.

Key Words: Pneumocephalus · Craniofacial skull base bone · Internal carotid artery canal



INTRODUCTION

Pneumocephalus, or intracranial air or gas, is associated with several neurological procedures, cranial trauma, and, rarely, open neural tube defects^{3,11,12}. The condition has been reported in both traumatic and non-traumatic instances, with the most common traumatic causes being craniofacial bone fractures with communication through an injury to the dura, accompanied by CSF leakage. Naturally, traumatic CSF leakage is a complication in 2% of all head-injured patients and in 12 to 30% of all cases of basilar skull fractures^{4,5,14}. Traumatic pneumocephalus is also possible in the absence of a craniofacial skull fracture, but this is rare. Two mechanisms have been proposed to explain pneumocephalus without craniofacial skull fracture, both involving low intracranial pressures that result in the "sucking" of air through a dural defect^{8,15,18}. The first mechanism involves vertical pressure that creates a pressure gradient within the CSF system, while the second involves a ball valve effect that allows air to enter the

skull base through multiple foramen or the craniocervical junction pathway. Here, we report a case of massive intracerebral pneumocephalus and pneumoventricle, in the absence of any skull fracture, that may have occurred by the entrance of air into the brain parenchyma, subarachnoid space, and ventricle system via the ICA canal pathway during blunt cranial trauma.



CASE REPORT

A 77-year-old woman was the victim of a pedestrian traffic accident wherein she sustained multiple contusions to the chest, head, and extremities. Upon arriving to the hospital by ambulance, the patient complained of a headache, dyspnea, and chest discomfort, but was alert and oriented to her surroundings. No traumatic brain injuries were revealed in the patient's medical history, although five years previously she had received medical treatment for hypertensive intracerebral hemorrhage in the frontal lobe.

Examination revealed multiple extensive air emphysemas in the masticator space, parapharyngeal space, retropharyngeal space, neck, face, and chest wall. There was no facial bone tenderness. No Battle's sign or Raccoon eyes⁶ were noted, nor were rhinorrhea or hemotympanum. A CT scan of the head revealed extensive pneumocephalus and pneumoventricle, primarily in the anterior horn of the lateral ventricle and in the CSF pathway. Images of

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the facial bones and skull base did not reveal a fracture. While coronal and axial images of the orbits, facial bones, and skull base did not reveal a fracture, an initial chest X-ray showed an air shadow in the ICA canal at the neck (Fig. 2). Subcutaneous emphysema was also seen at the neck in anteroposterior and lateral view X-rays (Fig. 1). A CT scan of the chest and abdomen revealed multiple left rib fractures; hemopneumothorax, pneumomediastinum and a contusional hemorrhage in the left upper lobe; atelectasis of the right upper lobe; and liver contusions and perihepatic fluid collection. The patient was admitted for observation.

Because of the hemopneumothorax in the left lung field, a chest tube was inserted in the left lung cavity. Prophylactic antibiotic was administered for the pneumocephalus and thoracotomy. The patient reported that her headache gradually resolved, and a repeat brain CT scan on hospital day 10 revealed almost complete resolution of pneumocephalus and pneumoventricle. The

patient was discharged at follow-up.



DISCUSSION

Pneumocephalus and pneumoventricle imply communication between the intracranial vault and the air-containing cavity and are most commonly caused by a traumatic event such as a craniofacial bone fracture that allows communication through an injury to the dura. In our patient, however, images of the facial bones and skull base revealed no fractures (Fig. 2). Our patient also showed no evidence of CSF leakage. We propose that pneumocephalus and pneumoventricle arose in the present case from an air pathway through the intracranium.

Common pathways of air trapping are known to include a weak entry point (basal cistern, carotid canal, cavernous sinus), the foramen ovale, foramen lacerum (ICA canal), choroidal fissure,



Fig. 1. Patient's Simple X-rays. **A.** Chest anteroposterior view showing the internal carotid artery canal (ICA) pathway (black arrow). **B.** Skull anteroposterior view shows a pneumoventricle in the frontal horn of the lateral ventricle. **C. D.** Neck anteroposterior and lateral views. An air-like density is observed in the retropharyngeal space (white arrow) and internal carotid artery pathway (black arrow).

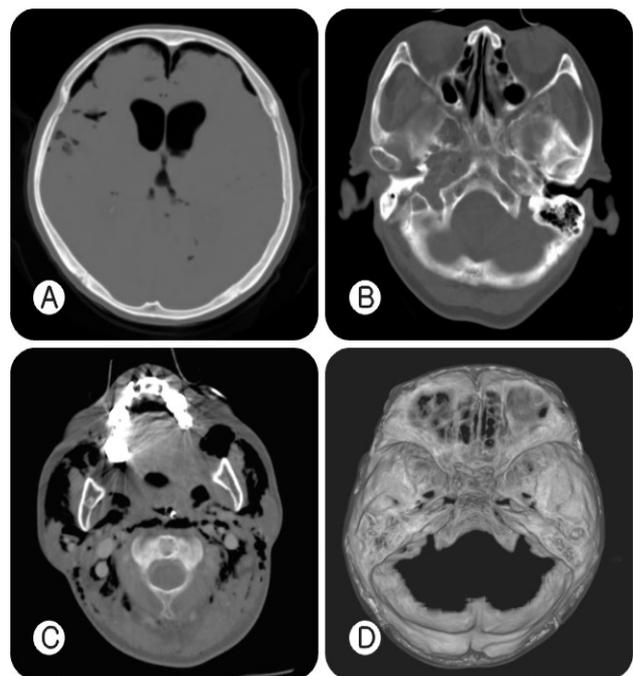


Fig. 2. Patient's Computed Tomography (CT) scan & Three-Dimensional CT. **A.** Axial CT shows massive pneumocephalus and pneumoventricle. **B.** Axial CT shows an air bubble in the foramen lacerum (internal carotid artery canal; black arrow). **C.** Air densities around the internal carotid artery canal and jugular vein area are indicated (black arrow) and show widening of the retropharyngeal space (white arrow). **D.** No skull base fracture is observed in 3-dimensional CT.

jugular foramen, and thecal sac of the thorax^{1,17,19}). However, the pathway leading to pneumocephalus in patients without craniofacial skull fractures remains unknown. It is clear that both intracranial and extracranial pneumocephalus can be induced, depending on patient factors and the anatomy of a few skull base foramina. The relevant anatomy, the carotid canal air bronchogram, is revealed by chest anteroposterior, neck anteroposterior, and lateral X-rays.

Based on the anatomy of the neck and skull base, we hypothesize that hemopneumothorax can lead to pneumocephalus. The brief pressure gradient between the intracranial and extracranial spaces associated with hemopneumothorax could, in our opinion, allow air to be canalized through a subcutaneous tract in the patient's body. Further, this air would be canalized through an internal carotid artery pathway consisting of various soft tissue spaces contained within the carotid artery, jugular vein, pharynx, and other skull base structures. The air would then enter through a foramen of the skull base, next to an air-containing space such as the foramen lacerum (ICA canal), foramen ovale, or jugular foramen. After the air entered the subarachnoid space, it would move to the ventricle through the choroidal fissure or lamina terminalis of the third ventricle. Consequently, pneumocephalus would occur through the weak entry point.

Clinically, our patient exhibited no evidence of a skull fracture on 3-mm CT cuts of the craniofacial bones. The most significant finding was extensive pneumocephalus and pneumoventricle in the absence of an obvious fracture, and we were faced with perplexing questions regarding the origin of intraventricular pneumocephalus. Two hypotheses have been reported to describe the pathophysiologic basis of pneumocephalus; they are known as the "ball valve" and "inverted bottle" mechanisms^{2,7,16}.

In the ball valve mechanism, it is postulated that air enters through a fracture or foramina of the skull base bone adjacent to an air-containing space. The only requirement, then, is a force to push air into the intracranial space. Once this has occurred, the air remains trapped and a seal is created by the arachnoid membrane, cerebral cortex, or ventricle. The second theory, the inverted bottle mechanism, hypothesizes that as CSF flows out of the skull, negative pressure is created within the intracranial space. This negative pressure will not allow the efflux of more CSF until air enters to take its place and equilibrate the pressure

differential. In our patient it is reasonable to conclude that the "ball-valve" mechanism was responsible for pneumocephalus, as no loss of CSF was recorded. Based on the premises of the ball valve mechanism, occult dural damage must have occurred at the time of our patient's traffic accident.

Malca SA¹⁰, in a report of pneumocephalus following thoracotomy without skull fracture, found that pneumocephalus had resulted from a fistula between the thoracic cavity and subarachnoid space. Hwang SL⁹ reported massive cerebral pneumocephalus after cardiopulmonary resuscitation. Although the case involved no pneumothorax or extravascular pneumocephalus, air was found in the ICA. Thus, massive cerebral pneumocephalus is possible if air enters the circulatory system via ruptured pulmonary vessels during cardiopulmonary resuscitation. As in these reported cases, carotid canal air bronchogram was revealed in our patient by chest anteroposterior, neck anteroposterior, and lateral X-ray views. Based on this similarity, we believe that the air pathway leading to pneumocephalus in our case may be similar to those in the previous cases.

McMurtrie R Jr¹³ reported a case of pneumocephalus following the placement of an epidural catheter using the loss-of-resistance to air technique, without apparent evidence of dural puncture. This case confirms the possibility of subarachnoid pneumocephalus in the absence of a skull fracture. The articles briefly reviewed here make clear that, though rare, pneumocephalus in the absence of craniofacial skull fracture is possible in the cranium.

In cases of pneumocephalus due to craniofacial fractures, air-like densities are typically seen in the subarachnoid space because of dural tearing and the proximity of the skull base to the basal cistern. In our case, air was primarily located in the temporal horn and frontal horn of the lateral ventricle. We thus proposed that the route of air entry was the choroidal fissure. According to our explanation, air was sucked into the ventricle through the ICA and anterior choroidal artery.

A review of the literature identified several cases of pneumocephalus in patients with ventriculoperitoneal shunts and thoracic bursting fractures with dural tearing. Our case seems to be the first, however, of pneumocephalus without any pre-existing condition to explain its origin. We have re-evaluated our radiologic findings with double-blind methods to confirm that they are indeed negative for craniofacial bone fractures. Further investiga-

tions, including a mimic of fracture and possible entry point, are necessary to fully explain the phenomenon of pneumocephalus.



CONCLUSION

Intraventricular pneumocephalus in the absence of craniofacial fracture is rare. The case presented here has led us to conclude that this condition can be caused by minor fractures of the skull base bone not revealed by traditional imaging methodologies, as well as by other causes in the complete absence of bony skull fracture.



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