ABSTRACT

Objective:
1. Describe a rare case of sphenoid sinus barotrauma after air travel resulting in pneumocephalus and meningitis.
2. Review presentation, pathology, and management of sinus barotrauma.

Method:
This is a case report of a woman with a prior history of head trauma who presented with pneumocephalus after recent air travel due to sphenoid sinus barotrauma. A pertinent review of the literature is presented.

Conclusion:
The paranasal sinuses communicate with ambient atmosphere. The free air flow through the ostia compensates for barometric pressure changes. Blocked sinuses of patients with sinusitis lead to a buildup of positive pressure during airplane ascent. As the barometric pressure decreases, air cannot escape from the sinus ostia resulting in a relative positive pressure. Air can then flow along the path of least resistance to the cranial cavity leading to injuries such as pneumocephalus, meningitis, and orbital emphysema. Bone defects in the sinus wall resulting from trauma or surgical injury enable unidirectional egress of air into the cranial compartment. Hyperpneumatization of sphenoid sinuses in some individuals may predispose them to spontaneous pneumocephalus.

INTRODUCTION

The middle ear spaces and paranasal sinuses, two body cavities that communicate with ambient air, must compensate for rapid equilibration of air pressure during aviation and underwater activities such as self-contained underwater breathing apparatus diving. The pressure-volume relationship of air in enclosed body cavities is described by the Boyle’s law, which states that the volume of a gas is inversely proportional to the pressure on it, when temperature is constant. Inability to equilibrate from blocking of sinus ostia from preexisting inflammation or polyposis may result in rapid changes in pressure inside sinuses relative to the surrounding spaces such as cranial cavity. Sinus barotrauma can occur during either ascent or descent. Although descent barotrauma or “squeeze” is more common by a ratio of at least 2:1, ascent barotrauma or “reverse squeeze” has more severe sequelae.

Rapid ascent in diving or aviation leads to a buildup of positive pressures in the obstructed sinuses. Inability to rapidly equilibrate air pressures may lead to traumatic movement of air to adjacent cavities which offer the least resistance leading to injuries such as pneumocephalus, meningitis, and orbital emphysema. The major identifiable preflight risk factors for civilian aerosinusitis include active upper respiratory infection and allergic rhinitis. In addition, a history of recurrent sinus barotrauma which is generally restricted to military aircrew, but conceivably exists in civilian patients with nasal polyps predisposes to aerosinusitis.

CASE REPORT

A 61 year old female was traveling by airplane when she began to develop severe headache, nausea and vomiting along with confusion. Symptoms started after the plane took off. She was suffering from an active upper respiratory tract infection. Her symptoms continued to worsen throughout the duration of flight. She was transferred to the emergency room after the plane landed. She reported a history of fall resulting in head trauma about 2 years back.

Her physical exam was significant for nuchal rigidity. Initially, she followed commands with upper and lower extremity movement and no vision changes were noted. Lumbar puncture was performed and she was intubated for deteriorating mental status in the emergency room.

Initial non-contrast CT scan of the brain showed pneumocephalus involving the sella. A sinus CT scan revealed extensive paranasal sinus disease involving the frontal, sphenoid, and ethmoid sinuses bilaterally with air fluid levels in the maxillary sinus and complete opacification of sphenoid and ethmoid sinuses. An irregularity of the posterior sphenoid sinus wall with posterior displacement of a tiny piece of bone into the pontine cistern was noted. CT cisternogram revealed opacification of subarachnoid spaces with contrast pooling in the posterior aspect of the right sphenoid sinus. The contrast in the pontine cistern outlined the amorphous debris regional to the deficit thus suggesting free communication through the defect.

CSF cultures were negative despite a gram stain revealing gram positive cocci in pairs and rods. She was treated empirically with Meropenem and Vancomycin. The navigation endoscopic sinus surgery was performed to repair the defect with Durepair and Tisseel. Patient has been on the plane after this repair and has not experienced headache or CFF leak.

DISCUSSION

Trauma secondary to barometric pressure variation occurs more frequently in the frontal sinuses secondary to the small outlet of the nasofrontal duct creating resistance to flow. The frontal sinus is most commonly affected (70 – 80%), followed by the maxillary sinus (19 – 29%). The ethmoids and sphenoid sinuses have rarely been implicated in this condition.

The sphenoid sinus varies between individuals in the degree of pneumatization. The extensively pneumatized sphenoid sinuses can cause dehiscence of bone over vital structures and may precipitate individuals to spontaneous and barotraumatic pneumocephalus.

Pneumocephalus is most frequently associated with trauma or iatrogenic injury from surgical repairs. Pneumocephalus generally develops because there is a discontinuity of the skull base through which air enters the cranial cavity. In the absence of gas-forming organisms, two mechanisms have been proposed to explain this. The first requires a cerebral spinal fluid leak. The resultant development of a relatively negative intracranial pressure can create a sufficient vacuum to introduce air into the cranium. This mechanism will cause air to be distributed along the cisterna and leptomeningeal space. The second mechanism, consists of a valve mechanism. In the presence of positive sinus pressure (example nasal blowing, coughing, sneezing, or a Valsalva maneuver), air can be forced from pneumatic cavities to the endocranium. When the endocranial pressure exceeds the pressure in the pneumatic cavity, the valve closes, preventing egress of the entrained air. This air will normally be distributed in the extradural space. Our patient may have developed pneumocephalus from the valve mechanism precipitated by the pressure changes during airplane ascent.

Prompt decompression of intracranial air is the initial treatment of symptomatic pneumocephalus. The principles of subsequent treatment parallel those for a cerebrospinal fluid leak with meningitis. It is of fundamental importance to identify the communication to the pneumatic cavity, if present, and seal it, thereby decreasing the possibility of recurrence.

CONCLUSIONS

Our patient developed pneumocephalus secondary to inability of her sinuses to equalize pressure changes from obstructed ostia and pre-existing dehiscence in the posterior wall of the sphenoid sinus during the aircraft ascent. It is not clear if history of fall with a mild head injury facilitated the dehiscence of the posterior sphenoid sinus wall. Posterior displacement of the sphenoid sinus wall likely created a valve mechanism to enable unidirectional egress of air into the cranial cavity. To our knowledge, there is no previous report of a case of sphenoid sinus barotrauma resulting from civilian aviation causing clinically significant pneumocephalus.

REFERENCES


6. Contact

Mona Shete, M.D.
UTHSC Department of OTO-HNS
Email: mshete@uthsc.edu
Phone: (901) 907-0703
Website: www.uthsc.edu

Figure 1. CT head with pneumocephalus around sella.

Figure 2. Coronal reconstruction of computerized scan of sinus.

Figure 3. CT Cisternogram
Opacification of subarachnoid spaces with contrast. Pooling of contrast in the posterior aspect of the right sphenoid sinus.