3-12-2021

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Recommended Citation
Turfe Z, Saleh N, George C, Rock J, and Craig JR. Cavernous Sinus Syndrome After Barotraumatic Sneeze. J Neuroophthalmol 2021.

Section Editors: Robert Avery, DO
Karl C. Golnik, MD

Cavernous Sinus Syndrome After Barotraumatic Sneeze

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Cavernous sinus syndrome (CSS) is a potentially life-threatening condition characterized by unilateral or bilateral proptosis, diplopia, and facial hypesthesia. CSS may be caused by a number of inflammatory, infectious, neoplastic, or neurovascular pathologies.

Sinus barotrauma results from an inability to equalize pressure changes between the sinus cavity and surrounding atmosphere (1). Sinus barotrauma can result in various complications including orbital complications, meningitis, and pneumocephalus (2–5). Sphenoid sinus barotrauma can cause pneumocephalus after sneezing (2), scuba diving (3), and nose blowing (4), but has not been reported to cause CSS. The purpose of this case report was to describe a rare case of unilateral CSS occurring due to sphenoid sinus barotrauma from sneezing.

A 75-year-old woman presented with a 2-day history of left-sided retrobulbar pain, cheek and forehead hypesthesia, and blurred vision after a sneezing episode. The patient denied any recent trauma, sinus surgery, or recent air travel or scuba diving. She was started on intravenous (IV) antibiotics and steroids for presumed CSS. She was afebrile with no leukocytosis, and examination revealed hypesthesia in left cranial nerve (CN) V1 and V2 distributions, CN VI paralysis, and CN III and IV paresis. There was no evidence of lid edema, orbital emphysema, chemosis, or proptosis. Nasal endoscopy revealed no evidence of purulence. Ophthalmology evaluation demonstrated no evidence of visual acuity loss. Sinus and head computed tomography (CT) scan demonstrated no stroke or neoplasm and no sphenoid sinus mucosal thickening or opacification. The left sphenoid sinus was hypoplastic with dehiscence of the lateral wall, including absent bone over the cavernous portion of the internal carotid artery. Notably, 2 foci of air were identified in the left cavernous sinus (Fig. 1). MRI demonstrated decreased enhancement of the left cavernous sinus and enhancement of the left intraconal orbital apex (Fig. 2). A cerebral angiogram showed normal filling of the cavernous sinus without thrombosis. A lumbar puncture was negative for meningitis, and there was no laboratory evidence of vasculitis.

Because of a lack of improvement over 48 hours from admission, left-sided endoscopic sinus surgery was performed. All sinonasal mucosa was well-vascularized, with no evidence of infection in any sinus. The left sphenoid sinus was hypoplastic, and there was bony dehiscence over the cavernous portion of the internal carotid artery, but the overlying mucosa was completely intact.

Given the low suspicion for an infectious etiology, intravenous antibiotics were discontinued and the patient numbness and extraocular motion gradually improved. She was eventually discharged on a short steroid taper. By 2 months postoperatively, the patient had complete resolution of all CSS signs based on examinations by both an otolaryngologist and ophthalmologist and a follow-up orbital MRI demonstrating no cavernous sinus disease.

Sinus barotrauma is the second most common type of barotraumatic injury after otologic barotrauma. Sinus barotrauma is characterized by an inability to equalize pressure changes between the sinus cavity and the surrounding atmospheric pressure, which can lead to transient sinus mucosal or bony injury due to either a negative pressure "squeeze" effect or a positive pressure "reverse squeeze"

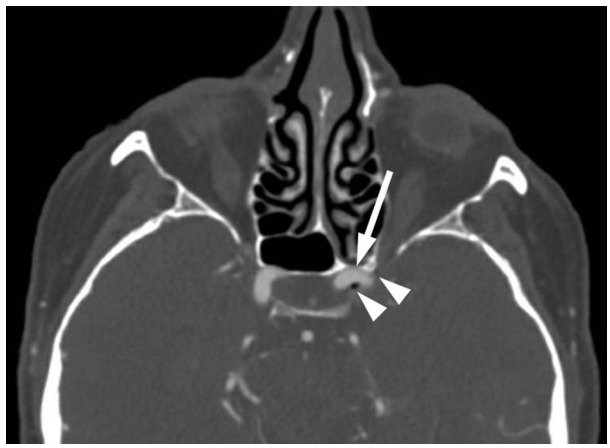

FIG. 1. Computed tomography scan demonstrating air bubble around the ICA (between *arrowheads*), with dehiscence of the lateral wall of the sphenoid sinus (*arrow*).

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Poster presentation at 2020 Triological society Combined Sections Meeting, January 23, 2020, Coronado, CA.

The authors report no conflicts of interest.

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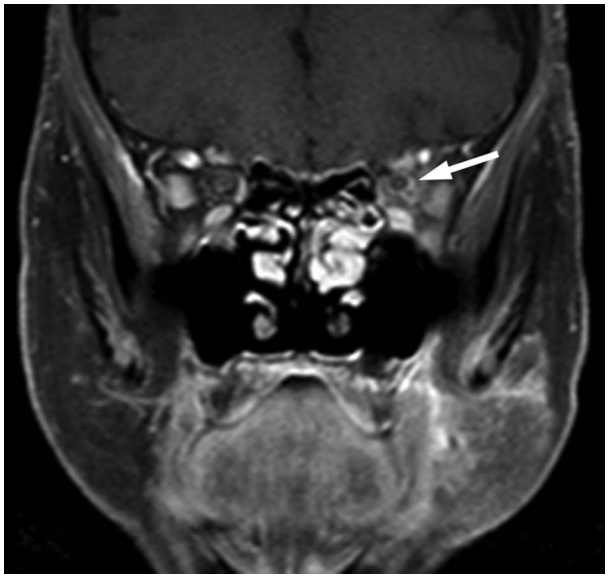

FIG. 2. MRI on presentation demonstrated increased enhancement of the left cavernous sinus region, with subsequent 6-month follow-up demonstrating residual mucosal thickening of the left sphenoid (*arrow*).

effect (1). Barotraumatic sinus injuries can lead to intrasinus or extrasinus complications.

Extrasinus complications of sphenoid sinus barotrauma can occur because of the close proximity of the sinus to the intraorbital and intracranial spaces. Sphenoid sinus barotrauma can cause pneumocephalus and possibly intraorbital or intracranial infections (2–5). Bony dehiscence of sinus walls presumably increases the risk of extrasinus spread of air or infection in the setting of sinus barotrauma. For example, Babl et al reported a case of a child who developed sellar and suprasellar pneumocephalus after a bout of forceful sneezing, and CT demonstrated dehiscence of the posterior wall of the sphenoid sinus (2).

Depending on the extrasinus spread of air, inflammation, or infection after sphenoid sinus barotrauma, a CSS can also develop. Tryggvason G et al reported a case of pneumocephalus from sphenoid sinus barotrauma, which occurred on ascent during scuba diving. Sinus CT demonstrated left sphenoid sinus partial opacification, and air in the sella turcica, and left cavernous and petrosal sinuses. The patient reported severe headaches and clinically improved over the next week. A follow-up head CT a month later showed resolved pneumocephalus (3). Orrell et al reported a case of septic cavernous sinus thrombosis and meningitis after repeated forceful nose blowing. CT demonstrated a small focus of air within the cavernous sinus and opacification of the adjacent sphenoid sinus, but the patient did not have symptoms of CSS. The patient had complete disease resolution with IV antibiotics (4). Canavan and Osborn (5) also reported a case of pneumocephalus within the cavernous sinus after barotrauma secondary to airflight. The patient reported a severe headache but which resolved with analgesics and conservative management.

Although sneezing has been reported as a cause of sinus barotrauma leading to pneumocephalus (2), it has not been reported as a cause of CSS. The patient in the current report developed air in her cavernous sinus after an isolated episode of sneezing, after which she immediately noticed severe retrobulbar pain followed by diplopia and facial numbness. Similar to previous studies, this patient had bony dehiscence of the lateral wall of the sphenoid sinus which presumably allowed for the extrasinus spread of air (2–4). The current report helps support the theory that sinus wall bony dehiscence places one at risk of pneumocephalus or orbital emphysema in the setting of sinus barotrauma. Whether the barotraumatic insult caused transient mucosal injury, or intrasinus air under high pressure directly traversed intact sinus mucosa, the end result was air entering the cavernous sinus which led to a unilateral CSS.

Barotrauma is a rare but important etiology to consider as a cause of CSS, once other more life- or vision-threatening conditions have been ruled out. If barotraumatic CSS is not considered, patients could undergo unnecessary diagnostic and therapeutic interventions. Fortunately, if CSS is due to barotrauma, patients should resolve with limited to no neurologic morbidity.

STATEMENT OF AUTHORSHIP
Category 1: a. Conception and design: Z. Turfe, C. George, N. Saleh, J. Rock, and J. Craig; b. Acquisition of data: Z. Turfe, C. George, N. Saleh, J. Rock, and J. Craig; c. Analysis and interpretation of data: Z. Turfe, C. George, N. Saleh, J. Rock, and J. Craig. Category 2: a. Drafting the manuscript: Z. Turfe, C. George, N. Saleh, J. Rock, and J. Craig; b. Revising it for intellectual content: Z. Turfe, C. George, N. Saleh, J. Rock, and J. Craig. Category 3: a. Final approval of the completed manuscript: Z. Turfe, C. George, N. Saleh, J. Rock, and J. Craig.

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