Silent Sinus Syndrome and Scuba Diving

Case Report and Literature Review

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Abstract

A 42-year-old patient presents with *unilateral enophthalmos, hypoglobus,* and no recent orbital trauma to the left eye. This case report and literature review discusses *silent sinus syndrome* with a possible relationship to *scuba diving*.

Introduction

Silent sinus syndrome is an underdiagnosed condition with characteristic findings, specifically presenting with unilateral, spontaneous enophthalmos and hypoglobus. The presented patient, seen in our oculoplastic clinic, meets most of the requirements for the diagnosis, but also has an interesting history of recreational scuba diving. While reviewing available literature on this topic, we discover no previous references of barotrauma from diving in patients diagnosed with silent sinus syndrome.

Case Report

A 42-year-old man, referred by an otolaryngologist, presented to the oculoplastic surgeon's office complaining that his left eye gradually appeared to be "pulled back" into its socket for the past three months. The patient had no recent history of orbital trauma, fractures, diplopia, or vision changes in general, but complained of intermittent, dull aches around the left orbit. He was not using any form of ocular treatment and denied a history of similar episodes. His past ophthalmic history was significant for excision of a growth on the right eye as a child, removal of a lead BB pellet over the left orbit as a teenager, and uncomplicated LASIK surgery for both eyes four years ago. Pertinent ocular or medical history within his family was unremarkable.

Social information gathered from the patient included his vast history of scuba diving in Guam, specifically during 1986-1990, when he completed five to six dives per week, descending between 30-60 feet at time limits recommended by dive tables given to the patient, with a maximum of 140 feet on few occasions while wreck diving. The patient used compressed air and remembered having intense facial and sinus pain during descent on each dive with severe ear infections occurring after the final trip. The patient admitted to needing nasal decongestant sprays before his dives but never suffered from epistaxis while underwater or after completing trips. The patient's last dive was in 1990, and his subsequent occupation rendered no association with orbital signs or symptoms.

In general, our patient used atenolol for hypertension and levothyroxine for hypothyroidism, both of which were well controlled. He had a history of a broken nose and two concussions as a teenager. Five months ago, the patient experienced an episode of dizziness, confusion, headaches, and vomiting. At that time, a CT of the brain with and without contrast was ordered and showed only a benign appearing osteoma in the right frontal sinus, but no brain or paranasal sinus disease otherwise. After discovering the sinus osteoma, he was referred to an ENT specialist. Systemic symptoms subsided in the meantime, but the patient began noticing misalignment between both upper eyelids, which later necessitated a visit to the oculoplastic

surgeon. Four months after the CT scan, an MRI of the brain was taken and showed a largely opacified left maxillary sinus that appeared smaller than the right.

On initial ophthalmic examination, visual acuity measurements were J1+ in both eyes without correction using a Rosenbaum near card. No RAPD was appreciated in either eye and extraocular muscle testing showed no restrictions. Diplopia was not initiated during any ophthalmologic visit. Upon gross examination, the patient had asymmetric palpebral fissures along with left upper eyelid retraction. Marginal reflex distances were recorded as MRD 1: 5 mm in the right eye and 8 mm in the left eye. Initial Hertel measurements were 16 mm in the right eye and 13 mm in the left eye at a base of 97, and the patient had 2 mm of left hypo-ophthalmos.

At that time, axial and coronal CT scans of the orbits as well as an MRI of the orbits, face, and neck with and without contrast were ordered. MRI of the orbits, face, and neck showed similar results as before with a significantly smaller left maxillary sinus. Downsloping of the left orbital floor was noted with suggestion of enophthalmos by the radiologist. There was no pathologic enhancement or enlargement of extraocular muscles, no orbital mass lesion, and optic nerve sheath complexes appeared normal. CT, however, revealed complete opacification of the left maxillary sinus and osteomeatal complex with severe mucosal thickening and a hyperlucent mass in the face with thinning of the walls, indefinite whether the mass was located intranasal or interfacial.

Upon receiving MRI results, the patient had endoscopic sinus surgery with debridement, excision of a left facial mass, left total ethmoidectomy, and a left maxillary antrostomy with removal of mucosal membrane. During the procedure, the ENT specialist commented on complete opacification of the left maxillary sinus with severe mucosal thickening, erosion of the orbital floor and lamina paprycea with herniation of the globe into the left orbit, and an audible vacuum after opening the maxillary sinus. The surgeon also found dehiscence of bone throughout the lamina paprycea and orbital floor with only stabilization of the orbit from a thin bridge formed by the nasal maxillary bone and the ethmoid complex. The aforementioned nasal mass proved to be an inspissated mucous clot in the maxillary sinus, and from pathology reports after biopsy, showed only signs of chronic sinusitis. Following the above procedures, the enophthalmos and hypoglobus resolved to some extent, but the patient was not completely satisfied with the aesthetic outcome. One month later, he requested insertion of a left orbital implant by the oculoplastic surgeon to resolve the remaining hypoglobus and enophthalmos. A 1.5mm Porex orbital implant was inserted via a transconjunctival inferior fornix-based incision. During the procedure, the surgeon noticed some hypoplasia, but no bony fractures. Before completion of the surgery, the left eye was documented to have elevated 1.5mm and enophthalmos resolved.

Discussion

Scuba divers learn of the common medical side effects of barometric pressure throughout the body prior to plunging underwater. Some of these manifestations include decompression sickness, arterial gas embolism, ear, sinus, and pulmonary barotrauma, and even osteogenic necrosis of weight bearing bones in the body.[1,4] As Boyle's Law explains, while external pressure increases during descent, the volume in a gas-filled space decreases. In essence, a squeeze is created. That squeeze is equalized by air traveling through patent sinus pathways in cases of non-obstructed anatomy. However, with nasal pathology or congestion of mucosal tissues, a negative pressure is created without equalization and an inward collapse of mucosal

tissue can occur. Middle ear squeeze, or barotrauma, is the most common diving injury, found in up to thirty percent of novice divers, and sinus squeeze is another similar example.[4] One study quotes paranasal sinus barotrauma in sports scuba divers to occur at a rate of twenty-seven percent per diver and one-half percent per dive.[20] During a squeeze phenomenon, the diver may encounter ear, sinus, or facial pain, depending on the area of barotrauma, and hemorrhage into the small, congested compartments can also occur. Treatment, prior to diving, is use of decongestants and analgesics to try and minimize obstruction to these pressurized areas.

In the case presented, our patient has no prior history of chronic sinusitis, but could have suffered from obstructed nasal passageways due to his history of a broken nose, warranting treatment before dives with decongestants to prevent barotrauma. Pain upon descent classically suggests sinus barotrauma. One could suspect recurring negative pressure over a four year period to cause an inward suction of mucosal tissue resulting in a vacuum effect of the orbital floor and subsequent hypoglobus if the bony foundation is compromised. However, this would not explain why the patient presented twenty years later with symptoms.

A more reasonable scenario would include a discussion of maxillary atelectasis, also referred to as imploding antrum or silent sinus syndrome. Typically, this entity affects adults ages 39-65 with progressive onset of atraumatic eyelid and orbital misalignment. Patients experience unilateral eyelid retraction, enophthalmos, usually between two to five millimeters by Hertel measurement, and hypoglobus of two to three millimeters. Eye muscle restriction is not appreciated in these patients, therefore diplopia is absent. Negative sinus pressure, once explored via endoscopic sinus surgery, is common.

Several proposals exist to describe the etiology of this disease. Many believe the reason for spontaneous enophthalmos to be as discussed below by researchers at the University of Utah:

> The leading theory regarding the pathogenesis of silent sinus syndrome is based on chronic maxillary sinus obstruction with hypoventilation, a state that eventually causes negative pressure to develop within the sinus [1,2,3,4,5,6,7,8,9]. After occlusion of the maxillary infundibulum, mucous begins to accumulate and eventually fills the sinus. The stagnant mucous incites a low-grade inflammatory response within the sinus and causes osteolysis of the sinus walls. The sinus walls, thinned by inflammation, are pulled into the sinus by negative sinus pressure.[8]

Other explanations for its etiology include congenital hypoplasia of the orbital floor, which can lead to increased fragility of the maxillary bone. Also hypothesized is the thought that mild inflammation over time results in mucosal thickening and introducing negative pressure allows resorption and a subsequent inward collapse of non-thickened walls.[10]

Specific imaging findings are necessary for the diagnosis. Volume loss, which occurs within the maxillary sinus secondary to inward retraction of the walls, is the primary radiographic finding. The orbital floor retracts and is commonly thinned, the maxillary infundibulum is always occluded with sinus opacification, and the uncinate process retracts against the inferomedial aspect of the orbital wall.[7] Our patient presents with three of the four findings on CT and MRI scans. Position of the uncinate process is not mentioned in the final readings, perhaps secondary to complete opacification of the sinus or due to the large mucous plug.

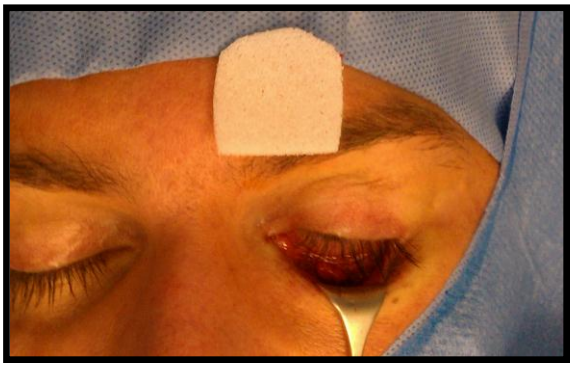
Until this point, neither specific occupations nor activities have been associated with silent sinus syndrome, whether not asked by the recorder or offered by the historian it is unknown. Our patient in particular likely has a component of sinus barotrauma and chronic

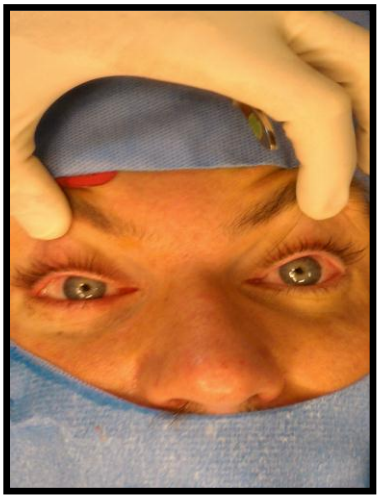
maxillary atelectasis with the enophthalmos and hypoglobus resulting from silent sinus syndrome. It is possible that his recurrent exposures to increasing pressures while scuba diving led to an underlying chronic sinus inflammatory process that with time and increasing negative pressure was followed by ongoing orbital wall thinning. Based on the hypothesized mechanisms of the disease, it would be interesting to study groups of patients exposed to long-term significant barometric pressure changes to see if they have an increased propensity to developing silent sinus syndrome.

Patient at presentation

1 week post-operative views

The figure below shows the inferior transconjunctival approach used during surgery with a 1.5mm Porex implant. The figure on the right was taken after insertion of orbital implant, left eye.





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