Silent sinus syndrome is a rare condition that can pose a diagnostic challenge. The patient may present with unilateral ptosis or retraction, a deep superior sulcus or orbital asymmetry. The medical history is often noncontributory. This condition is characterized by unilateral spontaneous enophthalmos and hypoglobus due to increased orbital volume and retraction of the orbital floor. This occurs because of atelectasis of the ipsilateral maxillary sinus and, when the condition is left untreated, may result in complete obliteration of the sinus with worsening enophthalmos and hypoglobus.

Patients typically are unaware of preexisting sinus disease and deny orbitofacial trauma. Rarely, they will complain of vertical diplopia, although extraocular motility and the rest of the eye examination are usually normal. There is no gender or racial predilection, and patients tend to present during the third to fifth decade of life.

The first two reported cases were reported in 1964, but the term “silent sinus syndrome” was coined 30 years later by Soparkar and colleagues.1 Since that time, several case series have been published in both the ophthalmology and otolaryngology literature.

Our patient was a 32-year-old Caucasian woman, referred for evaluation of an acquired right upper eyelid ptosis (Fig. 1). She was otherwise healthy with no past ocular history.

Not Just Another Droopy Eyelid
The evaluation of ptosis requires a complete ocular and periocular examination, including pupillary examination, cranial nerve evaluation and measurement of the margin-to-reflex distances 1 and 2, eyelid fissure height and levator function.

The differential diagnosis of ptosis is extensive, including neurogenic, neuromuscular junction, myogenic and aponeurotic causes. It is critical to rule out serious conditions such as myasthenia gravis, third nerve palsy and Horner syndrome.

Consideration must also be given to those conditions that present with an apparently smaller palpebral fissure but otherwise normal levator function. These cases of “pseudoptosis” include contralateral exophthalmos or contralateral eyelid retraction, and ipsilateral enophthalmos.

In our patient, we noted the deep ipsilateral superior sulcus compared with the contralateral side. Although this can suggest levator dehiscence, we confirmed ipsilateral enophthalmos with Hertel exophthalmometry readings of 15 mm in the right eye and 17...
mm in the left. Our patient also demonstrated 2 mm of right hypoglobus. The rest of her eye examination was completely normal. Our concern about the orbital asymmetry prompted us to review an old MRI, which revealed evidence of an enlarged right orbit consistent with silent sinus syndrome.

**Spontaneous Enophthalmos**
The most common cause of enophthalmos is traumatic expansion of the orbital cavity secondary to a blowout fracture of the orbital floor and/or medial wall. Other causes of enophthalmos include rare atrophy of orbital contents or a cicatricial process of the orbit. Conditions that exhibit such features include Parry-Romberg syndrome, linear scleroderma or metastatic scirrhous carcinoma of the breast. Orbital imaging is essential in evaluating these conditions.

**Orbitofacial Imaging**
The decision whether to order MRI as opposed to CT requires weighing the advantages and disadvantages of each modality. While an MRI scan spares the patient from exposure to ionizing radiation and provides excellent soft tissue detail, a CT scan is faster and cheaper and provides superior bony detail. Either modality may be used to help establish the diagnosis.

In general, the main radiological findings of silent sinus syndrome are thinning and retraction of the orbital floor (Fig. 2A), ipsilateral maxillary sinus hypoplasia and opacification, lateralization of the uncinate process resulting in blockage of the ostiomeatal complex (Fig. 2B) and retraction of the posterolateral and medial walls of the maxillary sinus (Fig. 2C).

**Pathophysiology**
Optimal management of silent sinus syndrome requires an understanding of the underlying pathophysiology. The most widely accepted theory is that an inciting event causes occlusion of the ostiomeatal complex through which the maxillary sinus drains into the middle meatus of the nasal antrum. This occlusion results in an accumulation of secretions that eventually are resorbed, causing a vacuum effect. The chronic subatmospheric pressure and hypoventilation of the sinus results in negative pressure, leading the sinus walls to migrate inward. In addition to the orbital floor being pulled downward, there may be bone remodeling and thinning due to increased osteoclast activity. Typically, the peristomeum is not affected.

Our patient developed enophthalmos after the natural delivery of her first child. Currently, there is only one reported case of silent sinus syndrome occurring in a pregnant patient. Although pregnancy-related rhinitis and maxillary sinusitis are known to occur, our patient had no signs or symptoms suggesting this etiology. We speculate that the birthing process might be implicated in precipitating the blockage of the ostiomeatal complex. In a contrary model, pregnancy may be associated with new-onset proptosis secondary to orbital vascular lesions that enlarge during pregnancy. These cause typical symptoms of an expanding orbital mass, such as proptosis and limited eye motility.

**Treatment**
The definitive treatment for silent sinus syndrome is surgical, and otolaryngological consultation may be necessary. The blockage of the ostiomeatal complex (Fig. 2B) must be relieved by functional endoscopic sinus surgery. Typically, endoscopic uncinectomy and opening of the maxillary sinus ostium are done. Once sinus drainage has been normalized, orbital floor augmentation surgery may be needed to restore orbital volume and decrease the enophthalmos. Medpor, titanium, autologous bone or another material may be fashioned into a subperiosteal implant, which is placed on the orbital floor. This aids in the repositioning of the globe with improvement of enophthalmos and eyelid position.

**Summary**
Silent sinus syndrome is an acquired condition that occurs in adults with unilateral, progressive, spontaneous enophthalmos and hypoglobus secondary to maxillary sinus hypoventilation caused by blockage of the ostiomeatal complex. The patient often presents with a droopy eyelid and a deep superior sulcus, and the history is usually not contributory. It is important to look for orbital asymmetry by performing Hertel exophthalmometry in such patients as well as to have orbitofacial imaging done when indicated.

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