Silent Sinus Syndrome: An Acquired Condition

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Summary: We present the case of a 30-year-old man with silent sinus syndrome. A CT scan obtained 10 years earlier showed completely normal maxillary sinuses. This case illustrates the acquired nature of this disorder.

The silent sinus syndrome, also known as imploding antrum and chronic maxillary sinus atelectasis, consists of findings of painless enophthalmos and inward retraction of the ipsilateral maxillary sinus walls on imaging studies (1, 2). The resultant volumetric loss in the maxillary sinus accounts for orbital enlargement and enophthalmos. The distinction between the imaging appearance of hypoplastic maxillary sinus and silent sinus syndrome is not well understood. Some authors believe that a congenital underdevelopment of the maxillary sinus is responsible for the development of silent sinus syndrome, but the acquired nature of this condition is now more readily apparent (3). Obstruction of the maxillary ostium appears to play a critical role in the development of silent sinus syndrome. Events leading to retraction of the walls of the obstructed sinus are not clearly understood.

Case Report

A 30-year-old man was found to have asymmetrically enlarged palatine tonsils, right greater than left, during an upper respiratory infection. The tonsillar asymmetry did not resolve with proper antibiotic treatment. A CT scan of the neck performed for evaluation of a potential underlying mass revealed a completely opacified maxillary sinus on the right. There was significant volume loss of the right maxillary sinus with inward retraction of the slightly thickened walls and consequent enlargement of the right orbit and middle meatus (Fig. 1). The uncinate process was thinned and retracted to the orbital wall, obstructing the maxillary sinus infundibulum. Of note, there was no underlying mass to account for tonsillar asymmetry. The patient did not have any symptoms that might be related to sinusitis. He did not notice any enophthalmos but retrospectively recalled a girlfriend several years before noting his right “eye line” being lower than that on the contralateral side. Right enophthalmos and 1-2 mm of hypoglobus were observed on repeat examination (Fig 2).

The patient had undergone a CT scan of the paranasal sinuses 10 years previously for presurgical evaluation of a posttraumatic nasal septum deformity that occurred during childhood. Findings showed that the right maxillary sinus size and wall thickness were normal (Fig 3). Soon after that initial CT, he underwent nasal septal reconstruction and revision rhinoplasty, including lateral nasal osteotomies. The patient elected not to pursue surgical treatment of the maxillary sinus disease. His tonsillar asymmetry remained stable during a 14-month follow-up.

Discussion

The silent sinus syndrome is a spontaneous unilateral maxillary atelectasis with complete or partial opacification of the sinus. Silent sinus syndrome is a rare disorder, but it is probably underdiagnosed because of a lack of recognition (1). The typical patient with silent sinus syndrome is an adult in the third through fifth decades of life who presents with spontaneous, painless, and occasionally progressive enophthalmos and hypoglobus (1, 2). No symptoms attributable to chronic sinonasal disease are present. Physical examination findings may include upper lid retraction, deepened upper lid sulcus, malar depression, facial asymmetry, and diplopia. The diagnosis of silent sinus syndrome can be made clinically, but it should be differentiated from other causes of spontaneous enophthalmos such as Parry-Romberg syndrome and linear scleroderma.

The imaging findings are characteristic (4). The primary finding is maxillary sinus volume loss due to inward retraction of the sinus walls, which accounts for the increased orbital volume and enlargement of the middle meatus. Typically, all 4 walls of the sinus are retracted, though one of the medial, anterior, or posterolateral walls may be spared. The orbital floor (maxillary roof) is always retracted and commonly thinned. The other walls may be thinned, normal, or slightly thickened. The maxillary infundibulum is always occluded and the sinus is opacified. The uncinate process is retracted against the inferomedial aspect of the orbital wall.

Our patient exhibited all the characteristic findings of silent sinus syndrome and slight thickening of the walls of the sinus including the orbital floor. The orbital floor thickening is uncommon and was reported to be present in 3 of 12 patients of Rose et al (2) and none of 68 patients of Soparkar et al (1). We speculate that the slight thickening of all walls of the sinus and ipsilateral tonsillar hypertrophy are secondary to chronic inflammation. Soparkar et al argued that the wall thickening may indicate a coexistent developmental hypoplasia, which was clearly not the case in our patient because imaging before disease onset showed completely normal sinus walls (1).

The pathophysiology of silent sinus syndrome remains unclear. Initially, it was suggested that a devel-
FIG 1.  (A) Coronal, (B) transverse, and (C) sagittal CT images of the sinuses show inward retraction of all walls of the right maxillary sinus with enlargement of the orbit and the middle meatus. The uncinate process is not clearly visualized because it is markedly thinned and retracted to the inferomedial orbital wall (confirmed with nasal endoscopy). (D) Thick-slab volume reconstruction in the coronal plane and (E) curved reconstruction along the optic nerves better demonstrates the maxillary sinus volume loss and enlargement of the orbit and middle meatus.
opmentally small sinus with chronic obstructive sinus-
itis was the cause (3). The acquired nature of this con-
dition, however, is now well recognized. Negative in-
trasinus pressure has been demonstrated in patients
with silent sinus syndrome (5). Obstruction of the
sinus ostium is always present, but it is not clear
whether this is the cause or the result of sinus wall
retraction. Complete obstruction of the mucous mem-
brane–lined sinus resulting in gas resorption and neg-
ative pressure formation, in a similar manner to
middle ear atelectasis due to Eustachian tube dys-
function, is the most plausible explanation. Given the
rarity of silent sinus syndrome and the very high
prevalence of maxillary sinus obstruction, however,
one has to question this explanation. This theory also
fails to offer an explanation for the exclusive in-
volvement of the maxillary sinus. Perhaps, other com-
pounding factors such as trauma or anatomic predis-
position play a role (6).

In our patient, childhood nasal trauma and subse-
quent surgical trauma were likely factors in the later
insidious development of silent sinus syndrome. An
osteotomy performed during septal reconstruction
and rhinotomy typically passes through the nasal pro-
cess of the maxilla; however, an osteotomy extending
more posteriorly could enter and destabilize the max-
illary sinus. Despite the absence of chronic sinusitis
symptoms in our patient’s case, subsequent scar con-
tracture or maxillary sinus hypoventilation might then
have resulted in the sinus atelectasis, hypoglobus, and
enophthalmos typical of silent sinus syndrome.

The initial management in this syndrome should be
conservative. If this is inadequate, then normal sinus
drainage is restored by enlarging the maxillary ostium
with functional endoscopic sinus surgery. Surgical in-
tervention to improve sinus aeration typically halts
the progress of maxillary sinus contraction but does
not restore sinus volume. In patients with diplopia or
severe cosmetic deformity, repair of the orbital floor
with placement of a subperiosteal implant can be
performed at the same time or after functional endo-
scopic sinus surgery.

In summary, this case supports the notion that
silent sinus syndrome is an acquired condition. Thick-
ening of the sinus walls is probably related to chronic
inflammation and not to underlying developmental
hypoplasia. Surgical or other trauma to the maxilla
and sinus ostium may play a role in the pathogenesis
of this condition.

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