

SILENT SINUS SYNDROME: RARE CAUSE OF ENOPHTHALMOS

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ABSTRACT

Silent sinus syndrome is a rare acquired cause of progressive enophthalmos and hypoglobus due to chronic maxillary sinus atelectasis that typically occurs in the absence of significant sinonasal symptoms.^{1,2} The condition results from persistent obstruction of the ostiomeatal complex with negative intrasinus pressure and gradual inward bowing of the maxillary walls and orbital floor.^{2,3} Because patients often present to ophthalmology or plastic surgery rather than otorhinolaryngology, radiologists play a key role in recognising this entity on cross sectional imaging and differentiating it from more common causes of unilateral enophthalmos.

We report a 61 year old pre diabetic man who presented with nasal blockage and subtle facial asymmetry and was found on CT to have classic features of silent sinus syndrome of the right maxillary sinus, including complete sinus opacification, marked volume loss, inward bowing of all walls, obstruction of the ostiomeatal complex, and inferior displacement of the orbital floor with enophthalmos. We compare his imaging and clinical features with the published series on silent sinus syndrome and related chronic maxillary atelectasis, highlighting key differentiating points from chronic sinusitis, maxillary sinus hypoplasia, mucocele, fibrous dysplasia, and syndromic midface hypoplasia.^{2,4,9} Awareness of this entity in unilateral sinus opacification with orbital changes can facilitate timely referral for functional endoscopic sinus surgery and prevent unnecessary work up for orbital or neoplastic disease.^{2,5,10}

Keywords: Silent sinus syndrome; chronic maxillary atelectasis; enophthalmos; hypoglobus; maxillary sinus; computed tomography.

Introduction

Silent sinus syndrome (SSS) is an uncommon clinical radiologic entity characterised by spontaneous enophthalmos and hypoglobus associated with ipsilateral maxillary sinus atelectasis and absence of overt sinonasal inflammatory symptoms.^{1,3,6} It was first named by Soparkar et al in 1994 in a series of patients with painless enophthalmos and unilateral maxillary sinus collapse on CT.¹ Subsequent work by Kass et al and Brandt and Wright integrated SSS into the broader spectrum of chronic maxillary atelectasis, with SSS now considered the symptomatic, stage III end of this continuum where orbital deformity becomes clinically apparent.^{2,6}

Most patients present in the third to fifth decade of life, although cases have been reported from adolescence to late adulthood.^{4,7,8} The rarity of the syndrome, subtle onset of facial asymmetry, and often minimal sinonasal complaints contribute to delayed diagnosis. CT is the imaging modality of choice and shows a fully pneumatized but volume reduced maxillary sinus with inward bowing of its bony walls, obstruction of the ostiomeatal complex, and descent of the orbital floor.^{3,5,9}

We present a case of SSS in an older adult and discuss how his demographic, clinical, and imaging features compare to the existing literature, with emphasis on differentiating radiologic features and implications for management.

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Clinical History

A 61 year old man with pre diabetes presented to the otorhinolaryngology clinic with several months of persistent right sided nasal blockage and gradually noticeable facial asymmetry. He denied a history of facial trauma, prior sinus surgery, chronic rhinosinusitis, or allergy. There were no visual complaints, including diplopia, blurring, or visual field defects, and he reported no facial pain, headache, or epiphora.

On examination there was subtle right sided facial asymmetry with mild enophthalmos and deepening of the superior sulcus. Extraocular movements were full and painless. Visual acuity and pupillary responses were normal. Anterior rhinoscopy showed no obvious mass or polyp. Routine blood tests were unremarkable apart from impaired fasting glucose. CT of the paranasal sinuses was requested to evaluate underlying sinonasal pathology and exclude neoplastic disease.

Image Findings

Noncontrast CT of the paranasal sinuses in axial, coronal, and sagittal planes demonstrated a fully formed right maxillary sinus that was completely opacified and markedly reduced in volume compared with the contralateral side. All bony walls of the right maxillary sinus showed inward bowing, including posterior displacement of the anterior wall and lateral bowing of the medial wall. The most striking feature was inferior bowing of the orbital floor with resultant increase in right orbital cavity volume, explaining the clinical enophthalmos.

The right ostiomeatal complex was completely obstructed, with lateralised uncinete process and narrowing of the infundibulum, consistent with chronic impairment of sinus aeration and drainage. No osseous destruction, expansile lesion, calcification, or focal mass was identified. There were no features of fungal sinusitis, mucocoele, or fibrous dysplasia. The contralateral maxillary sinus and the remaining paranasal sinuses were normally pneumatized and clear.

These findings of unilateral maxillary sinus contraction with total opacification, inward retraction of its walls, descent of the orbital floor, and associated enophthalmos in the absence of sinonasal symptoms were diagnostic of silent sinus syndrome.

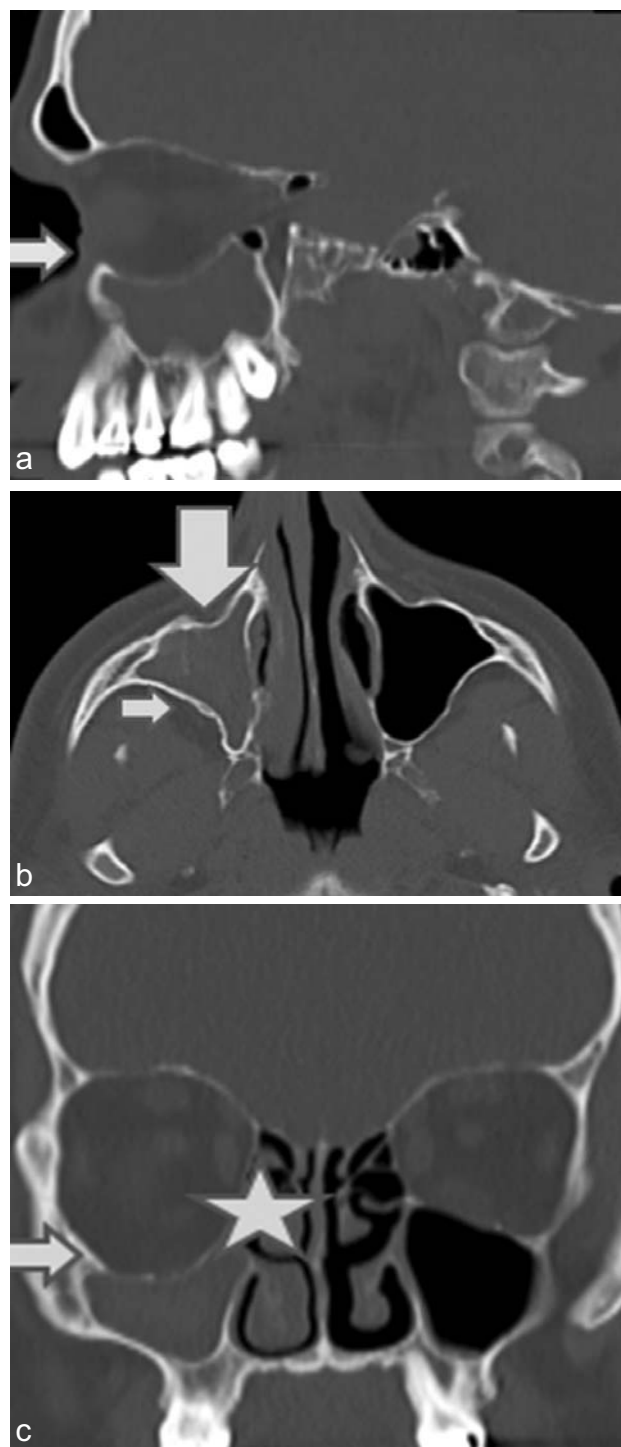


Figure 1 (a,b,c): CT paranasal sinuses. Sagittal (a), axial (b), and reconstructed coronal (c) images demonstrate a fully formed but contracted right maxillary sinus that is completely opacified with inward bowing of all four walls (arrows). There is inferior bowing of the roof (orbital floor) with increase in right orbital volume and enophthalmos. Posterior displacement of the anterior wall is marked by the thick downward arrow. The right ostiomeatal complex is occluded (star). The left maxillary sinus and remaining paranasal sinuses are normal.

Discussion

Large case series and systematic reviews report that SSS most often affects patients in middle age, with mean ages around 39 to 43 years and reported ranges from late adolescence to the eighth decade.^{4,7,8,9} Rosso et al, in a PRISMA compliant systematic review of 276 patients, reported a mean age of 40.4 years, with most patients in the third to fifth decade.⁹ Al badr et al described an age range from 19 to 82 years in their imaging focused series, again with a predominance of middle aged adults.⁸ Our patient was 61 years old, placing him toward the upper end of the reported age spectrum but within the documented range, which underlines that SSS cannot be excluded based on older age alone. Sex distribution across series is relatively balanced without a strong gender predilection.^{4,6,9} Brandt and Wright found no significant difference between males and females in their systematic review, 6 and similar findings have been reported in later cohorts.^{7,8,9} Consistent with this, our case in a male patient fits within the expected demographic pattern.

Classical descriptions highlight painless unilateral enophthalmos, hypoglobus, and deepening of the superior sulcus as the main clinical features, often without nasal complaints.^{1,3,5} In the original series by Soparkar et al most patients presented to ophthalmology for cosmetic asymmetry or diplopia, and sinonasal symptoms were minimal.¹ Illner et al and Rose et al similarly reported that ocular or cosmetic concerns, rather than nasal symptoms, drove presentation in the majority of patients.^{3,5}

More recent series and reviews recognise that a subset of patients report mild nasal obstruction, congestion, or intermittent sinus pressure, which likely reflect underlying chronic ostiomeatal obstruction yet remain clinically subtle.^{4,7,9} Numa et al, in their review of 84 cases, and Babar Craig et al, in a series of 16 patients, both described patients with minor nasal symptoms despite the label "silent".^{4,7} Our patient primarily complained of nasal blockage and only subtle facial asymmetry and did not have diplopia or significant pain, which aligns with this broader clinical spectrum. His presentation reinforces that SSS may present through ENT clinics rather than ophthalmology and that the absence of dramatic ocular symptoms does not exclude the diagnosis.

Chronic maxillary atelectasis (CMA) refers to progressive loss of maxillary sinus volume with inward retraction of sinus walls due to chronic negative intrasinus pressure.^{2,6} Kass et al coined the term and described three stages based on increasing wall deformity and clinical involvement.² Brandt and Wright systematically reviewed all reported cases of CMA and SSS and concluded that these entities represent the same disease process, with SSS corresponding to stage III CMA where enophthalmos and hypoglobus become clinically evident.⁶ Later radiologic prevalence studies and classification proposals have supported this unified concept and suggested that SSS should be considered a subtype of advanced CMA defined by the presence of orbital manifestations.^{6,9}

Our patient exhibited classic radiologic criteria for stage III CMA, including a contracted antrum, inward bowing of all maxillary walls, and inferior displacement of the orbital floor, together with clinical enophthalmos. This aligns closely with the definitions proposed by Kass et al, Brandt and Wright, and Rosso et al and supports the interpretation of SSS as part of the CMA spectrum rather than a completely distinct entity.^{2,6,9}

Several studies have explored the pathogenesis of SSS and CMA. The prevailing theory suggests that obstruction of the maxillary ostium leads to hypoventilation, with resorption of trapped gas and secretion into the mucosa and bloodstream, which creates a persistent negative pressure in the sinus.^{2,6,9} Manometric studies by Kass et al documented negative intrasinus pressures in completely occluded maxillary sinuses, supporting this mechanism.² Over time, this pressure gradient results in bone remodeling and inward bowing of the sinus walls and orbital floor, with gradual increase in orbital volume and onset of enophthalmos and hypoglobus.^{3,5,7}

In our case, CT showed complete obstruction of the right ostiomeatal complex with lateralised uncinate process and narrowing of the infundibulum, exactly mirroring the ostial obstruction described in the manometric and imaging studies of CMA and SSS.^{2,3,6,8} This supports the concept that even relatively mild or subclinical sinonasal disease that narrows the maxillary ostium can, over years, produce the characteristic antral implosion.

Illner et al first provided a detailed radiographic description of SSS, emphasising unilateral maxillary sinus opacification, marked reduction in sinus volume, inward

bowing of at least one sinus wall, lateral displacement of the infundibular wall, and inferior displacement of the orbital floor.³ Rose et al coined the term “imploding antrum” for the same constellation of findings in ophthalmic patients, highlighting the paradox of sinus contraction rather than expansion in a disease causing orbital deformity.⁵

Our patient demonstrated all of these key features: total opacification and volume loss of a previously fully developed maxillary sinus, inward bowing of the anterior, posterior, medial, and lateral walls, lateralisation and collapse of the infundibular wall, and inferior bowing of the orbital floor with increased orbital volume and enophthalmos. These findings are identical to those illustrated in the series by Illner et al, Rose et al, and Babar Craig et al and in imaging focused case reports by Albadr et al.^{3,5,7,8}

Albadr et al and other radiology based reports also emphasise the value of CT in excluding alternative causes of enophthalmos, such as post traumatic orbital floor defects, fat atrophy, metastatic disease, or prior orbital decompression.⁸ In our case there was no history of trauma or surgery and no focal mass, osseous destruction, or fat atrophy, which makes SSS the most plausible diagnosis when considered alongside the characteristic maxillary changes.

Several entities can mimic aspects of SSS on imaging, and accurate differentiation is essential. Chronic maxillary sinusitis typically demonstrates mucosal thickening and retained secretions without maxillary volume loss or inward wall bowing, and the orbital floor retains its normal position.^{3,8} Congenital maxillary sinus hypoplasia presents with a small sinus from birth, often bilaterally, with a shallow antrum rather than a contracted one and without the progressive orbital changes of SSS.^{3,6} Fibrous dysplasia shows a ground glass bony matrix with osseous expansion rather than collapse and usually involves multiple craniofacial bones.³

Maxillary mucoceles are expansile cystic lesions that cause outward bowing of sinus walls and often lead to proptosis rather than enophthalmos.^{3,8} Syndromic midface hypoplasia, such as in Crouzon or Treacher Collins syndrome, produces diffuse craniofacial abnormalities evident from childhood, with bilateral and symmetric involvement rather than the isolated unilateral sinus collapse seen in SSS.^{3,5}

Our case lacked any features of bone expansion, focal destructive mass, diffuse craniofacial dysplasia, or

bilateral sinus anomaly. The combination of sinus contraction, inward bowing of all walls, ostiomeatal obstruction, and inferior orbital floor displacement matches the diagnostic criteria for SSS and stage III CMA described by Illner et al, Kass et al, and Rosso et al.^{2,3,6,9}

Although our case report focuses on imaging diagnosis, the literature on SSS consistently recommends functional endoscopic sinus surgery as the primary treatment to re establish sinus ventilation and stop progression.^{5,7,9,10} Uncinectomy and middle meatal antrostomy create a permanent drainage pathway, normalise sinus aeration, and can lead to gradual improvement in orbital floor position over months.^{7,9,10}

There is ongoing debate regarding the need and timing of orbital floor reconstruction. Some authors advocate a staged approach, with initial endoscopic sinus surgery followed by orbital reconstruction only if enophthalmos or hypoglobus persist.^{5,7} Babar Craig et al and Sciarretta et al reported that many patients achieved satisfactory cosmetic and functional outcomes with endoscopic surgery alone or with delayed orbital reconstruction in more severe cases.^{7,10}

Rosso et al found in their systematic review that the majority of patients underwent endoscopic sinus surgery, with a smaller proportion receiving simultaneous or staged orbital reconstruction, and overall outcomes were favourable when diagnosis was made before marked orbital deformity became fixed.⁹

Early recognition on CT is therefore essential, particularly for radiologists, who may be the first to identify the entity when evaluating unilateral sinus opacification or unexplained enophthalmos. Prompt communication with ENT and ophthalmology can facilitate appropriate counselling and surgical planning tailored to the degree of orbital involvement.

Conclusion

Silent sinus syndrome is a rare but important cause of unilateral enophthalmos and hypoglobus that arises from chronic maxillary sinus atelectasis secondary to ostiomeatal obstruction. Our case of a 61 year old man illustrates that SSS can present in older adults with relatively modest sinonasal complaints and subtle facial asymmetry. CT findings of unilateral maxillary sinus

contraction with complete opacification, inward bowing of all bony walls, ostiomeatal complex obstruction, and inferior displacement of the orbital floor are highly characteristic and allow confident diagnosis when correlated with clinical features.

Comparison with published series shows that our patient fits the recognised demographic and radiologic profile of SSS, even though his dominant symptom was nasal obstruction rather than ocular complaint. Radiologists should consider SSS in any patient with unilateral maxillary sinus opacification, sinus volume loss, and orbital floor depression, and should explicitly raise this possibility in reports to prompt early referral for functional endoscopic sinus surgery and assessment of the need for orbital reconstruction.

All patient data in this manuscript and related files have been fully anonymised.

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