

Spontaneous enophthalmos: silent sinus syndrome

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Abstract

Spontaneous enophthalmos unrelated to trauma or surgery is rare. The term 'silent sinus syndrome' has been used to describe this process where, in particular, there is an absence of any sino-nasal symptoms. The enophthalmos and hypoglobus that occurs in these subjects is caused by atelectasis of the maxillary antrum, which itself appears to be due to chronic maxillary hypoventilation. We report a case of silent sinus syndrome that arose following insertion of a nasogastric tube. Whilst acute paranasal sinusitis is a well-described sequela of nasal intubation, this association with a rare, and as yet unexplained, phenomenon may go some way to explain its aetiology.

Key words: Enophthalmos; Maxillary Sinusitis

Introduction

The orbital complications of acute sinusitis are familiar to both otolaryngologists and ophthalmologists. In addition, diplopia and proptosis resulting from a more chronic sinus disease such as frontal or ethmoidal mucoceles is also well described. Enophthalmos is rare however, particularly when it occurs on its own without any preceding symptoms or history of trauma.

The term 'silent sinus syndrome' was first described by Soparkar *et al.* who presented 14 patients with unilateral enophthalmos and hypoglobus associated with asymptomatic maxillary sinus disease.¹ All these patients demonstrated dramatic thinning or complete resorption of the orbital floor with consequential inferior displacement of the orbital contents.

Case report

A 41-year-old lady presented to the ophthalmology clinic with diplopia and enophthalmos of the right eye. On further questioning, she had experienced some facial pressure in the past but no orbital or facial pain. She was found to have vertical diplopia on downward gaze, but had normal visual acuity. There was 2 mm of enophthalmos.

On subsequent review in ENT out-patients, she denied any nasal congestion, rhinorrhoea or post-nasal discharge. There was no history of previous trauma or surgery to the face and no significant dental disease. She did, however, have a history of Crohn's disease and during her first pregnancy a year previously, she had developed small bowel obstruction, which required an emergency right hemicolectomy. During this time she had had a nasogastric tube inserted in her right nostril for three days. Examination in the clinic revealed a narrowed middle meatus on the right side but no mucopus or polyps. The left nasal cavity was normal.

CT scanning showed opacification of the right maxillary antrum together with significant mucosal thickening in the right frontal and anterior ethmoidal sinuses (Figure 1). There was an increase in the orbital volume due to thinning of the orbital floor and a decreased maxillary sinus volume. The middle turbinate was lateralized with an inwards 'C-shaped' bowing of the uncinate.

During functional endoscopic sinus surgery, the right middle turbinate was found to be lateralized and required trimming inferiorly to allow access. A middle meatal antrostomy was performed together with an anterior ethmoidectomy and opening of the nasofrontal recess. The right antrum contained mucoid material, but bacterial and fungal cultures were negative. The posterior ethmoid system was normal.

She made a good recovery from the surgery and at follow up, was found to have a widely patent middle meatal antrostomy with a relatively healthy looking antrum. The patient declined orbital floor reconstruction.

Discussion

The process of spontaneous asymptomatic enophthalmos related to chronic maxillary sinus disease has not been satisfactorily explained. It was first reported by Montgomery who described it in two patients with maxillary sinus mucoceles, but who had no symptoms of past sinus disease.²

In the later description by Soparkar *et al.*, the majority of patients were in their fourth or fifth decade, the average amount of enophthalmos was 3 mm and only a third of the patients had any history of sinus disease in childhood. Significantly, all but one of the cases showed ipsilateral maxillary sinus hypoplasia. They went on to speculate that this hypoplasia could result from temporary obstruction of the maxillary sinus during the second decade of life (during which remodelling occurs) resulting in growth arrest and bone resorption. However, the same group more recently

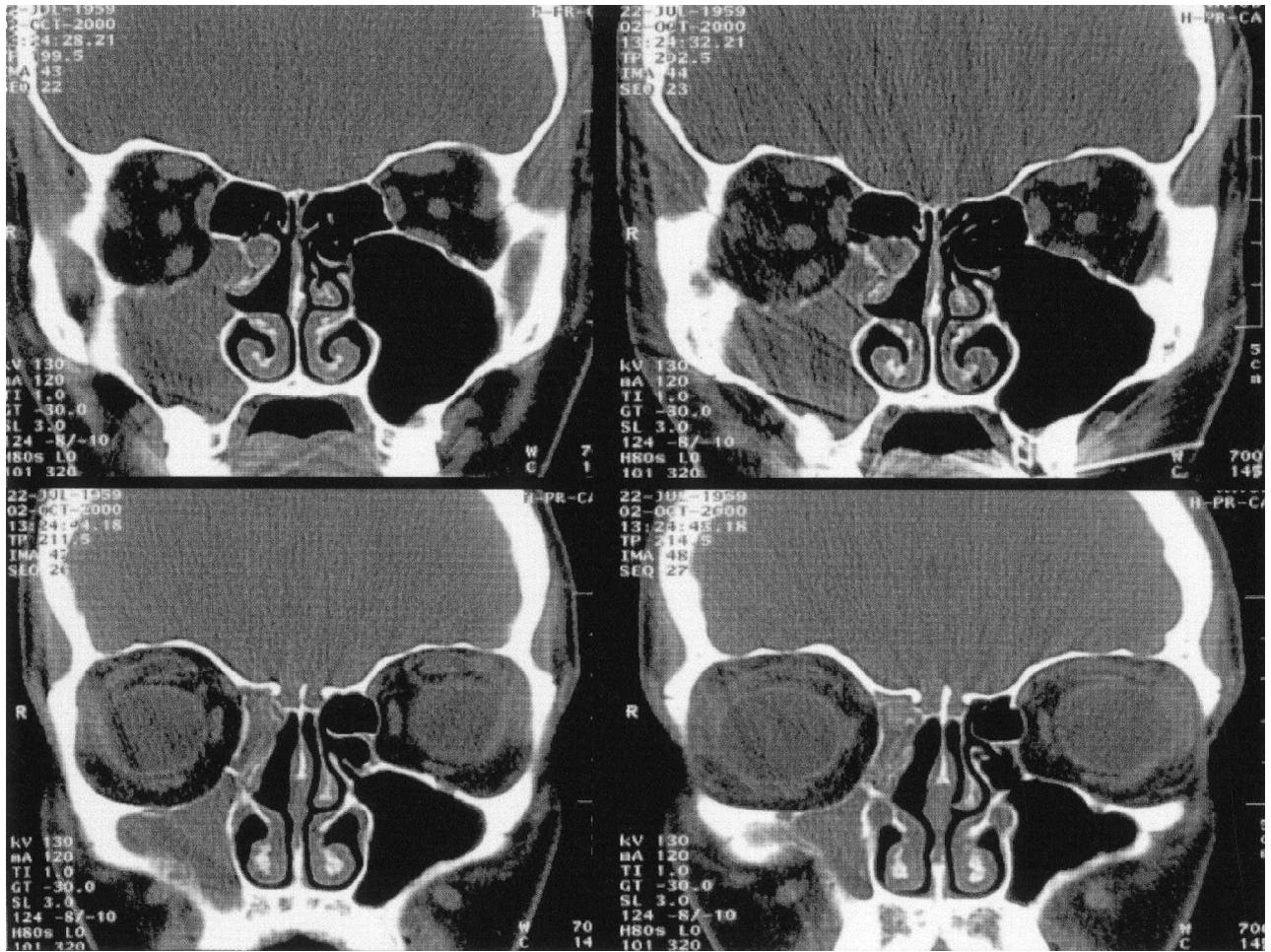


FIG. 1

Coronal CT scans showing complete opacification of the right maxillary sinus and inferior displacement of the orbital contents.

described a case where normal pre-morbid imaging was available suggesting a more rapid acquisition of the pathological changes than previously thought.³ Interestingly, they also demonstrated a negative sinus pressure of -23 mmHg at the time of surgery.

Since then, there have been scattered case reports with varying theories as to how the syndrome arises.⁴⁻¹¹ A similar condition of chronic maxillary sinus atelectasis has also been reported.¹²⁻¹⁴ Most notably, Gillman *et al.* suggests that the probable cause is hypoventilation of the maxillary sinus secondary to obstruction of the ostiomeatal complex. This creates a negative pressure, which leads to thinning of the sinus walls in a similar manner to eustachian tube dysfunction causing atelectasis and retraction in the middle ear.^{5,6} This theory is supported by experimental evidence both in the animal model¹⁵ and in patients with reduced maxillary volume on computed tomography (CT) scans.¹⁶ Secretions within the sinus may then act as support for the orbital floor until decompression at a later date.^{3,5} However, despite several authors agreeing with this possible aetiology, no one has yet provided any evidence to suggest how the ostial occlusion occurs.

Acute sinusitis is an important complication of nasotracheal or nasogastric intubation particularly in the intensive care setting. In a case series of nosocomial sinusitis in a trauma unit over two years, all were found to have had an indwelling nasal tube on the side of the

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infected sinus at the time of diagnosis.¹⁷ Subsequent randomized controlled trials have shown that the use of long-term nasotracheal intubation significantly increases the likelihood of sinusitis developing in patients who are being mechanically ventilated.¹⁸⁻²⁰ Although one can only speculate that mechanical outflow obstruction of the maxillary sinus was the cause of the infection at least in a proportion of these cases, a further study of iatrogenic sinusitis found that those who underwent emergency blind nasotracheal intubation had a more rapid and severe sinus infection.²¹ This indicates that trauma to the nasal architecture may be a factor in the development of ostiomeatal obstruction. Unfortunately, there are no reports of long-term follow up in patients who have had nosocomial sinusitis secondary to nasopharyngeal instrumentation.

We suggest that in our case, the nasal intubation that occurred in the preceding year may have resulted in the maxillary sinus outflow obstruction and subsequent hypoventilation. At the time of surgery the patient had a severely lateralized middle turbinate, which would support this aetiology. Indeed, surgery for these cases is directed at removing the obstruction at the ostiomeatal complex, thereby allowing normal pressure to occur in the antrum. The extent of altered uncinata anatomy and lateralization often requires very careful dissection so that penetration of the lamina papyracea is avoided.⁶

A further report in which 5–6 mm of orbital displacement recovered completely following spontaneous resolution of the maxillary sinus disease, does suggest that the initial management in this syndrome should be conservative.²² If this is inadequate, then a simple middle meatal antrostomy (without orbital floor reconstruction) should be sufficient—as was the case in our example and in fact, in many of the descriptions in the literature.

In summary, 'silent sinus syndrome' is a rare phenomenon that presents with spontaneous enophthalmos and hypoglobus rather than any sino-nasal symptomatology. It appears to arise from chronic maxillary atelectasis, which we suggest occurred in the presented case following the insertion of a nasogastric tube. We believe this is the first report of this process occurring after nasal intubation and suggest that ostial occlusion resulting in maxillary hypoventilation is the cause, at least in a proportion of cases.

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Mr C. Hobbs takes responsibility for the integrity of the content of the paper.

Competing interests: None declared
