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RHINOLOGY

Chronic maxillary atelectasis and silent sinus syndrome: two faces of the same clinical entity

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Abstract Chronic maxillary atelectasis (CMA) and silent sinus syndrome (SSS) are rare clinical entities characterized by an implosion of the maxillary sinus that may or may not be associated with sinonasal symptoms, and are complicated by ipsilateral enophthalmos. The objective of this article is to discuss the definitions, physiopathology, clinical and radiographic characteristics, and surgical management of these entities. We retrospectively reviewed 18 patients (7 women, 11 men, aged 12–70 years) diagnosed and treated in the ear, nose, and throat departments of four Belgian teaching hospitals between 2000 and 2015. Nine patients had a history of sinus disease. In all cases, a computed tomography scan showed downward displacement of the orbital floor, increased orbital volume, and maxillary sinus contraction. Five patients met the criteria for grade II CMA and 13 for grade III CMA. Four patients met the criteria for SSS. All patients underwent wide endoscopic middle maxillary antrostomy. There were no orbital complications and all patients experienced resolution or a dramatic reduction of their symptomatology. Only one patient asked for an orbital floor reconstruction to correct a persisting cosmetic deformity. Although CMA and SSS are usually regarded as different entities in the literature, we believe that they lie on the same clinical spectrum. Treatment for both conditions is similar, i.e., middle meatal antrostomy to halt or even reverse the pathological evolution and reconstruction of the orbital floor in the event of persistent cosmetic deformity.

Keywords Chronic maxillary atelectasis · Silent sinus syndrome · Imploding antrum syndrome · Enophthalmos · Endoscopic surgery · Middle meatal antrostomy

Introduction

A paranasal sinus is an air-filled space surrounding the nasal cavity. From Jankowski's theory concerning evo-devo evolution, a sinus cavity results from resorption of the bone marrow with production of air bubbles. The ostium establishes a communication with the nasal cavity and takes over the ventilation and drainage of the sinus [1].

Constitutionally, the sinuses can be normal, small (hypoplastic), or large (megasinus or pneumocoele) as shown in Table 1. Occasionally, the size may change over time. An enlargement is called a pneumosinus dilatans and a reduction can be secondary to chronic atelectasis, e.g., chronic maxillary atelectasis (CMA) or silent sinus syndrome (SSS) or to thickening of the sinus walls during a chronic inflammatory process, e.g., in association with a fungus ball.

A reduction in maxillary sinus volume, through downward displacement of the orbital floor, can lead to unilateral enophthalmos. In 1964, Montgomery described two cases of enophthalmos associated with maxillary "mucocoeles" [2]. In 1994, Soparkar et al. coined the term "silent sinus syndrome" to describe 19 patients with spontaneous

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Table 1 Anatomical variations of the maxillary sinus

	Small sinus	Large sinus
Constitutional	Incomplete development	Overpneumatisation
	Hypoplastic maxillary sinus	Megasinus
Acquired	Imploding sinus	Exploding sinus
	Silent sinus syndrome or chronic maxillary atelectasis	Pneumosinus dilatans
	Volume reduction by thickening of the sinus wall	
	Chronic inflammatory process	

enophthalmos and unilateral collapse of the maxillary sinus in the absence of sinonasal symptoms [3]. In 1997, Kass et al. used the term "chronic maxillary atelectasis" to describe the process of maxillary sinus implosion, which is associated in many cases with sinus-related symptoms [4]. Kass et al. divided the disease spectrum of CMA into grades I, II, and III (Table 2) [5].

In this article, we report on a cohort of patients presenting with either one of these two conditions. We describe their clinical and radiographic characteristics as well as their surgical management, and discuss the definitions, pathogenesis, and treatment of these conditions.

Patients and methods

After receiving approval from our ethical committee, (Comité d'Ethique Médicale, Centre Hospitalier Universitaire UCL Namur, No. 20/2017), we retrospectively reviewed the charts of patients with a reduced maxillary volume and deformation of the orbital floor in the presence of a completely developed maxillary sinus. The patients were diagnosed and underwent surgery in the ear, nose, and throat departments of four Belgian teaching hospitals between 2000 and 2015. Exclusion criteria were previous facial or orbital trauma (including sinus surgery), congenital facial deformity, and other causes of acquired enophthalmos.

For the diagnosis of SSS, we used the diagnostic criteria devised by Soparkar et al., i.e., modification of facial appearance, enophthalmos and/or hypoglobus, a reduction of maxillary sinus volume on computed tomography (CT) scan, and the absence of sinonasal symptoms [3]. For CMA, we used the criteria developed by Kass et al. and described in Table 2 [4].

Results

Demographic and clinical characteristics

Eighteen patients [7 women, 11 men, mean age 44.0 ± 16.9 (range 12–70) years] met the inclusion criteria. Table 3 reports the characteristics, symptoms, and history of sinus disease in each patient. Thirteen patients were found to have enophthalmos on clinical examination, of which 7 had spontaneously noticed facial asymmetry (Figs. 1, 2). The five remaining patients were diagnosed by CT. Nine patients had a history of sinus disease. Endoscopic findings showed an enlargement of the middle meatus on the affected side in all cases (Fig. 3). The right side was affected in 13 cases (72%). In 14 of the 18 cases (77%), there was an ipsilateral nasal septal deformity, and in five (27%), the middle turbinate was lateralised. Five patients met the criteria for grade II CMA and 13 for grade III CMA. Four patients met the criteria for SSS.

Radiographic characteristics (Table 4)

Opacity of the pathological maxillary sinus was seen on coronal CT scans in all patients. This opacity was complete in ten cases and partial in the remaining eight cases. All patients had maxillary sinus contraction, with a decreased sinus volume and an increased orbital volume because of downward displacement of the orbital floor (maxillary roof). The orbital floor was thinned and depressed, with abnormal concavity toward the sinus lumen (Fig. 4), and was dehiscent in 12 cases. We also observed a loss of bone density in the anterior, posterolateral, and medial walls in six, four, and three cases, respectively. In the 17 cases where the sinonasal wall

Table 2 Classification of chronic maxillary atelectasis according to Kass et al. [5]

Grade I	Membranous deformity	Lateralised maxillary fontanelle
Grade II	Bony deformity	Inward bowing of one or more osseous walls of maxillary antrum
Grade III	Clinical deformity	Marked deformation of the antral walls, enophthalmos, hypoglobus, midfacial deformity

Table 3 Clinical characteristics of 18 reported patients

Characteristic	Patient number																	
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Age at presentation, years	66	12	41	22	48	45	71	23	36	59	41	40	52	26	51	67	30	59
Sex	F	F	F	M	F	M	M	M	M	M	F	F	F	M	M	M	M	M
Side affected	L	R	R	L	R	R	R	R	R	R	R	L	R	R	L	L	R	R
Altered facial appearance	N	N	N	N	N	Y	N	N	N	N	Y	N	N	Y	Y	Y	Y	Y
Enophthalmos	N	N	N	N	N	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Diplopia	N	N	N	N	N	N	N	N	N	Y	U	N	N	N	N	Y	Y	N
General symptoms of sinusitis	Y	N	Y	N	N	N	Y	Y	Y	N	Y	Y	Y	Y	N	N	N	N
Rhinorrhoea	Y	Y	Y	N	N	N	Y	Y	Y	N	N	Y	Y	N	N	N	N	N
Nasal congestion	Y	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	N	N	Y	N	N	N
Postnasal drip	Y	Y	Y	N	N	N	Y	Y	Y	N	N	Y	N	N	N	N	N	Y
Facial pressure/pain	Y	Y	U	N	N	N	Y	Y	Y	Y	Y	Y	Y	N	N	N	N	N
History of sinus disease	Y	N	Y	N	N	Y	Y	Y	Y	N	U	Y	Y	N	N	N	N	N

M male, *F* female, *R* right, *L* left, *Y* present, *N* absent, *U* unknown

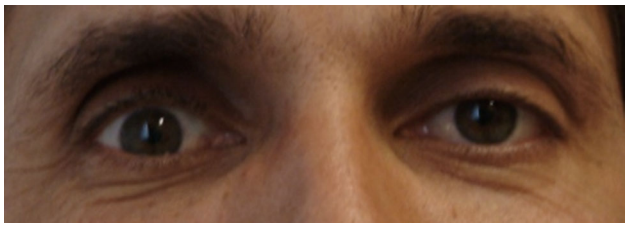

Fig. 1 Frontal view of a patient with facial asymmetry and right hypoglobus

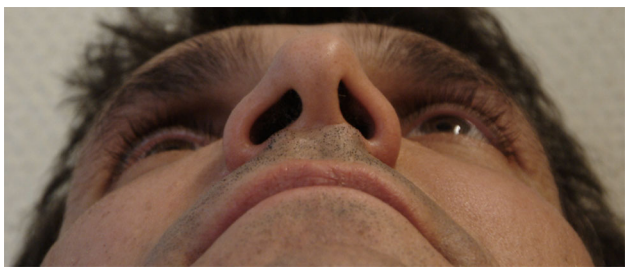

Fig. 2 Posterior displacement of the eye globe in the orbital cavity, known as "enophthalmos"

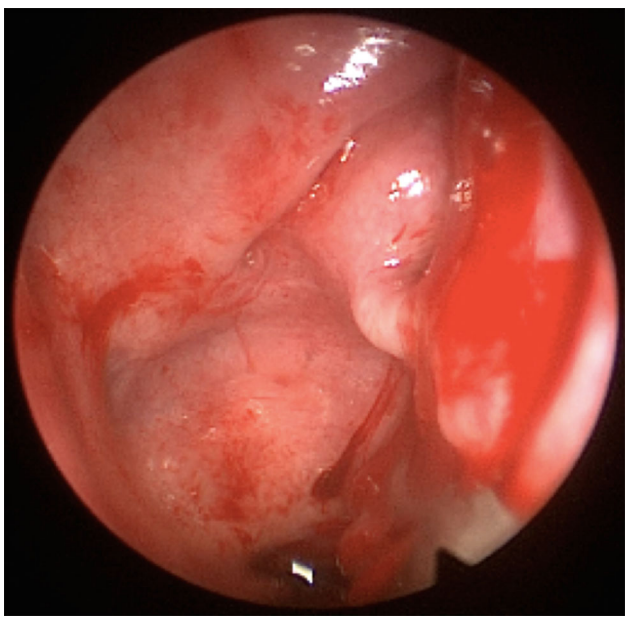

Fig. 3 Endoscopic view of the right nasal fossa showing lateral displacement of the medial maxillary wall and enlargement of the middle meatus

could be observed in the axial plane, 16 showed deformation towards the antrum; the same deformation was seen in the other walls (Fig. 5). Seven of the 18 patients had other sinus disease or opacification (mainly of the ethmoidal and frontal sinuses). Fourteen patients had ipsilateral septal deviation to the affected sinus; in five cases, this was associated with lateralisation of the ipsilateral middle turbinate.

Surgical management

All patients underwent endoscopic middle meatal antrostomy under general anaesthesia. The antrostomy was performed from backward to forward with medialisation of the uncinate process because it is considered to be safer [6]. The uncinate was then cut in two parts with backbiting forceps and the horizontal portion was resec-

Table 4 Radiographic characteristics of 18 reported patients

Characteristic	Patient number																	
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Maxillary opacification	C	C	C	I	I	C	I	C	I	I	C	C	I	C	I	I	C	C
Maxillary sinus contraction	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Downward displacement of the orbital floor	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Inward bowing of the maxillary walls	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	U	Y	Y	Y	N	Y	Y	Y
Nasal septal deviation	Ips	Con	Ips	Ips	Ips	Ips	Ips	Ips	Con	Ips	Ips	Ips	Nl	Con	Ips	Ips	Ips	Ips
Ipsilateral middle turbinate lateralisation	N	Y	Y	N	Y	Y	N	N	N	N	U	Y	N	N	N	N	N	N
Other sinus disease	N	N	N	N	N	Y	Y	Y	N	N	N	Y	Y	N	Y	Y	N	N
Site of loss of bone density in the maxillary wall																		
Roof	N	N	N	Y	Y	Y	Y	Y	N	N	U	Y	Y	Y	Y	Y	Y	Y
Anterior wall	N	N	N	Y	Y	N	N	N	N	N	U	N	N	Y	Y	Y	Y	N
Medial wall	N	N	N	N	N	N	N	N	N	N	U	N	Y	Y	N	N	Y	N
Posterolateral wall	N	N	N	N	N	N	N	N	N	N	U	N	N	Y	Y	Y	Y	N

C complete, *I* incomplete, *Y* present, *N* absent, *Ips* side affected, *Con* opposite to side affected, *Nl* normal, *U* unknown

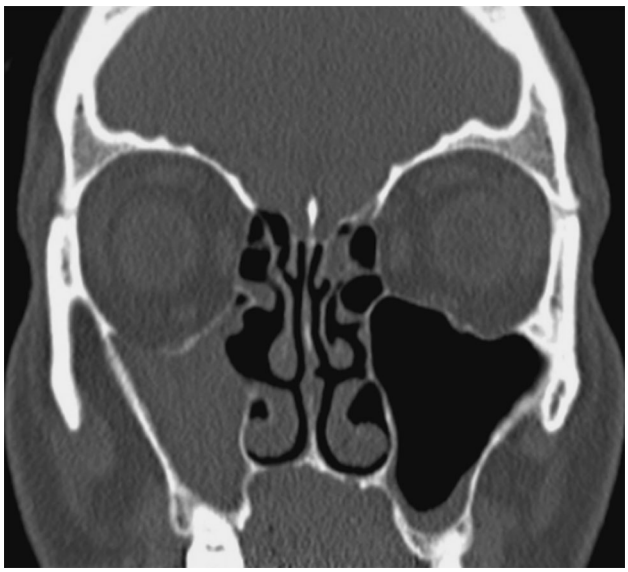

Fig. 4 Computed tomographic image in coronal plane with the downward displacement of the orbital floor and right maxillary sinus opacity

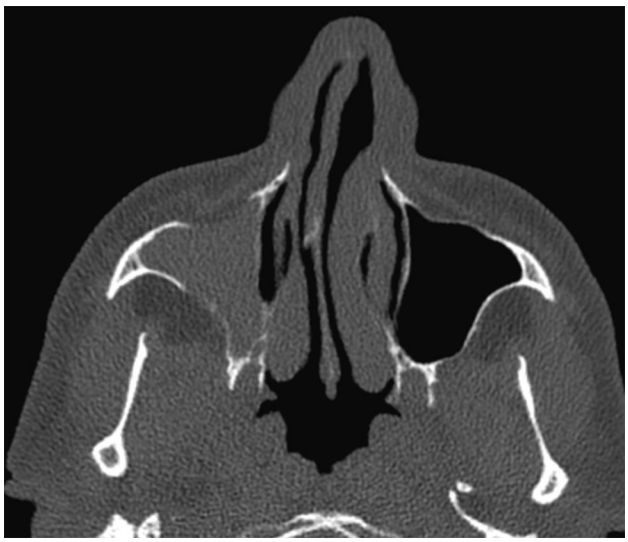

Fig. 5 Computed tomographic image in the axial plane showing inward bowing of the anterior and posterior maxillary walls and septal deviation toward the affected sinus

ted. The opening was then enlarged posteriorly and inferiorly using a microdebrider or cutting (Blakesley) forceps (Fig. 6). An ethmoidectomy was performed if other sinuses were found to have chronic inflammatory disease. In one case, the procedure was completed with bilateral frontal sinus drainage (modified endoscopic Lothrop or Draf type III).

When performed, bacteriological examination showed the drained mucus to be sterile and mucosal biopsies showed chronic non-specific inflammation signs on histopathology.

All surgeries were free of complications. Figure 6 shows a postoperative CT scan in the coronal view. Disease evolution was halted by six months in all but one patient. Slight enophthalmos with or without facial asymmetry persisted in some patients, but only one patient asked for a second surgical procedure (for reconstruction of the orbital floor).

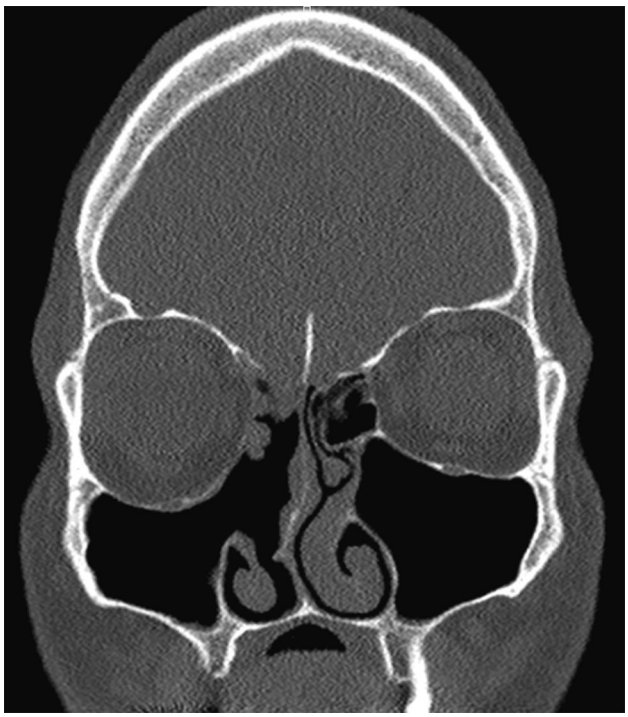

Fig. 6 Computed tomographic image in the coronal view after middle meatal antrostomy

Discussion

Definitions

Historically, the term "silent sinus syndrome" has been defined as a unilateral spontaneous, progressive, and painless enophthalmos associated with atelectasis of a fully developed maxillary sinus [3]. As described by Montgomery and Soparkar, the typical patient presenting with this unusual syndrome is a middle-aged man or woman who consults for spontaneous, progressive, and painless enophthalmos but without typical symptoms of chronic rhinosinusitis [2, 3]. The definition of SSS highlights the absence of sinus symptoms and complaints. However, in the literature, many cases of a spontaneous decrease in volume of the maxillary sinus secondary to antral collapse are associated with symptoms of sinusitis. The term "chronic maxillary atelectasis", defined by Kass et al. [4] as a "persistent decrease in the sinus volume of the maxilla from inward bowing of the antral walls", can be used to describe those patients.

The question that remains unresolved in the literature is whether SSS and CMA are really different entities. According to the definition by Kass et al. [4], and as discussed in a recent literature review [7], we believe that SSS might be considered grade III CMA. Even though SSS and CMA are still often described and discussed separately in the literature, the only difference between the criteria defined for grade III CMA and those for SSS is the presence or absence of sinus-related symptoms. We have attempted to summarise these criteria in Table 5. Indeed, in our series, all patients whose symptoms corresponding to the diagnostic criteria for SSS corresponded to the CMA III definition but only four (31%) patients with CMA III met the criteria for SSS.

Pathophysiology and natural history

Atelectasis of the maxillary sinus is defined as a persistent reduction of the volume of the maxillary sinus, secondary to a centripetal contraction of its walls [4]. While its clinical presentation is well known, its pathogenesis remains uncertain, and different theories have been put forward in the literature.

Blackwell et al. proposed a mechanism whereby a mechanical obstacle such as an anatomical variation (e.g., concha bullosa, paradoxical middle turbinate, septal deviation) or chronic rhinosinusitis causes chronic hypoventilation of the maxillary sinus [8]. Other authors describe the possibility of atelectasis secondary to occlusion of the ostium by fat after orbital decompression [9], trauma [10], a benign nasal tumour [11], or even a foreign body [12].

More recently, it has been postulated that lateralisation of the uncinate process could cause chronic hypoventilation of the cavity via a valve effect. This infrequent anatomical variation could explain why only a few patients develop this type of primary atelectasis [13].

Regardless of the initiating event, it is likely that as suggested by Illner et al., the final common pathway leading to sinus atelectasis is an obstruction to mucus drainage that leads to chronic hypoventilation of the sinus [14]. Over time, the negative pressure gradient and low-grade inflammation induce progressive osteolysis of the sinus walls (osteopenia secondary to diminution of osteoblast activity). These walls, which are thinned and weakened by this inflammatory reaction and attracted by the negative pressure gradient in the cavity, deform progressively with the diminution of sinus volume and augmentation of orbital volume [15–18].

This negative pressure theory is supported by the animal studies performed by Sharf et al., who measured negative pressures in rabbits with occluded maxillary sinus ostia [15]. In humans, Kass et al. reported negative antral pressures during corrective endoscopic surgery in patients with CMA. Similar to control patients, the contralateral antral pressure was equal to atmospheric pressure [16]. Gillman et al. likened these phenomena to chronic Eustachian tube dysfunction, hypoventilation of the middle ear, and tympanic membrane retraction [19].

Table 5 Diagnostic criteria for chronic maxillary atelectasis and silent sinus syndrome

	CMA grade I	CMA grade II	CMA grade III	SSS
Enophthalmos	Absent	Absent	Present	Present
Maxillary sinus volume reduction	Radiological membranous deformity	Radiological bony deformity	Radiological volume reduction with clinical repercussions	
Symptoms of sinusitis or facial pain	Absent or present	Absent or present	Absent or present	Absent

CMA chronic maxillary atelectasis, *SSS* silent sinus syndrome

Clinical and radiographic characteristics

The demographic and clinical characteristics of our cohort of patients are consistent with those previously described by Sopakar et al. [3], Babar et al. [20], and Rose et al. [21]. Most of the patients are in their third to fifth decade of life, and no frank gender preponderance has been reported in the literature. The right side was affected more frequently in our series (72%) than in the reports published by Soparkar et al. and Rose et al. The difference is probably explained by the small sample sizes.

Endoscopically, we frequently observed septal deviation toward the affected sinus and enlargement of the middle meatus. Lateralisation of the middle turbinate was frequently noticed. This association was also observed by Rose et al., who suggested that this nasal conformation might be a predisposing factor [9]. They also suggest that deformation of the medial maxillary wall might act to relieve this longstanding narrowing of the nasal space.

Radiographic evaluation of the paranasal sinuses and orbital characteristics is essential to confirm the diagnosis. While T_2-weighted magnetic resonance imaging can be helpful for study of the soft tissues, CT remains the most useful [6]. The classical radiographic findings observed are, a fully developed maxillary sinus with partial or complete opacification, an ostiomeatal occlusion with lateralisation of the uncinate process, enlargement of the ipsilateral middle meatus with or without lateralisation of the middle turbinate, and a loss of bone density in the sinus wall that ranges from thinning to dehiscence. Consequently, the pathognomonic scanographic features are retraction of the sinus walls with downward displacement of the orbital floor, diminution of maxillary volume, and augmentation of orbital volume [14]. CT also allows examination of other sinus cavities; the differential diagnosis includes sinus hypoplasia (where the cavity is incompletely developed) and other causes of enophthalmos (e.g., tumour) [6].

Surgical management

When SSS was described in the 1990s, the Caldwell–Luc procedure was commonly used. However, the current trend in the treatment of CMA is endoscopic middle meatal antrostomy.

The main controversy in the literature is the necessity and timing of orbital floor reconstruction via a subconjunctival or transconjunctival approach [22]. Some authors prefer a one-stage approach, with endoscopic antrostomy and reconstruction of the orbital floor performed at the same time [6, 9]. Others opt for a two-stage approach [20, 23] with a currently recommended delay between the two steps of 6 months [7]. Arguments against one-step surgery are the risk of orbital infection and also the fact that evolution of atelectasis is stopped after widening of the ostium. A certain degree of spontaneous re-expansion and sometimes even resolution of the atelectasis can be observed [20, 24]. Thus, for some patients, the two-stage approach allows avoidance of an orbital floor implant. All 18 patients in our series underwent the first stage of this two-staged approach and only one asked for an orbital floor reconstruction six months later. This supports the notion that the enophthalmos will improve spontaneously following endoscopic antrostomy alone in certain patients.

However, it is obvious that the same surgical treatment is needed in both CMA and SSS. Consideration of these conditions as the same clinical entity may influence preventive therapeutic intervention. As previously described, we encountered five patients who did not present with enophthalmos but whose CT scans showed typical features of maxillary sinus atelectasis. Those patients were classified as having grade II CMA and underwent antrostomy, which prevented pathological evolution to grade III CMA and avoided ocular and aesthetic symptoms. Even if it remains uncertain regarding whether all of these patients would have developed ocular and aesthetic complications, clinical experience has shown that early and appropriate surgical treatment could prevent this evolution [25].

Conclusion

Implosion of the maxillary sinus is a rare evolving process that can lead to enophthalmos. Nasal endoscopy, CT scan, and clinical evaluation are essential for the diagnosis.

Middle maxillary antrostomy is the treatment of choice to stop the natural evolution of the disease and leads to resolution of the symptomatology. In a minority of patients, an orbital implant is necessary to correct persistent enophthalmos. SSS and CMA are still often described and discussed separately in the literature, but the only difference between the definition of grade III CMA and SSS is the presence or absence of sinus-related symptoms. It seems clear that grade III CMA and SSS meeting the criteria for grade III CMA are part of the same disease spectrum. We believe that the staging classification for CMA is better adapted in describing the natural evolution of the disease and should be adopted.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflicts of interest regarding this article.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed consent As this is a retrospective chart review, the ethical committee at our institution granted a waiver of the need for informed patient consent. The patient appearing in Figs. 1 and 2 gave his explicit permission and informed consent to publication of these images.

References

1. Jankowski R (2013) The evo-devo origin of the nose, anterior skull base and midface. Springer, Berlin
2. Montgomery W (1964) Mucocele of the maxillary sinus causing enophthalmos. Eye Ear Nose Throat Mon 43:41–44
3. Soparkar CN, Patrinely JR, Cuaycong MJ et al (1994) The silent sinus syndrome. A cause of spontaneous enophthalmos. Ophthalmology 101:772–778
4. Kass ES, Salman S, Rubin PA et al (1997) Chronic maxillary atelectasis. Ann Otol Rhinol Laryngol 106:109–116
5. Ende K, Mah L, Kass ES (2002) Progression of late-stage chronic maxillary atelectasis. Ann Otol Rhinol Laryngol 111:759–762
6. Bossolesi P, Autelitano L, Brusati R, Castelnuovo P (2008) The silent sinus syndrome: diagnosis and surgical treatment. Rhinology 46:308–316
7. Brandt MG, Wright ED (2008) The silent sinus syndrome is a form of chronic maxillary atelectasis: a systematic review of all reported cases. Am J Rhinol 22:68–73
8. Blackwell KE, Goldberg RA, Calcaterra TC (1993) Atelectasis of the maxillary sinus with enophthalmos and midface depression. Ann Otol Rhinol Laryngol 102:429–432
9. Rose GE, Lund VJ (2003) Clinical features and treatment of late enophthalmos after orbital decompression: a condition suggesting cause for idiopathic “imploding antrum” (silent sinus) syndrome. Ophthalmology 110:819–826
10. Pawar SS, Hong S, Poetker DM (2010) Delayed presentation of silent sinus syndrome after orbital trauma. Am J Otolaryngol 31:61–63. doi:10.1016/j.amjoto.2008.09.003
11. Hens G, Hermans R, Jorissen M (2005) Chronic maxillary atelectasis. B-ENT 1:25–29
12. Fidan V (2008) Silent sinus syndrome associated with nasal foreign body. Int J Pediatr Otorhinolaryngol Extra 3:75–77. doi:10.1016/j.pedex.2007.10.007
13. Hobbs CGL, Saunders MW, Potts MJ (2004) Spontaneous enophthalmos: silent sinus syndrome. J Laryngol Otol 118:310–312. doi:10.1258/002221504323012102
14. Illner A, Davidson HC, Harnsberger HR, Hoffman J (2002) The silent sinus syndrome: clinical and radiographic findings. AJR Am J Roentgenol 178:503–506
15. Scharf KE, Lawson W, Shapiro JM, Gannon PJ (1995) Pressure measurements in the normal and occluded rabbit maxillary sinus. Laryngoscope 105:570–574
16. Kass ES, Salman S, Montgomery W (1996) Manometric study of complete ostial occlusion in chronic maxillary atelectasis. Laryngoscope 106:1255–1258
17. Boyd JH, Yaffee K, Holds J (1998) Maxillary sinus atelectasis with enophthalmos. Ann Otol Rhinol Laryngol 107:34–39
18. Wan MK, Francis IC, Carter PR et al (2000) The spectrum of presentation of silent sinus syndrome. J Neuroophthalmol 20:207–212
19. Gillman GS, Schaitkin BM, May M (1999) Asymptomatic enophthalmos: the silent sinus syndrome. Am J Rhinol 13:459–462
20. Babar-Craig H, Kayhanian H, De Silva DJ et al (2011) Spontaneous silent sinus syndrome (imploding antrum syndrome): case series of 16 patients. Rhinology 49:315–317. doi:10.4193/Rhino10.103
21. Rose GE, Sandy C, Hallberg L, Moseley I (2003) Clinical and radiologic characteristics of the imploding antrum, or “silent sinus”, syndrome. Ophthalmology 110:811–818. doi:10.1016/S0161-6420(02)01993-0
22. Cardesín A, Escamilla Y, Romera M, Molina JA (2013) Single surgical step for endoscopic surgery and orbital reconstruction of a silent sinus syndrome. Acta Otorrinolaringol Esp 64:297–299
23. Vander Meer JB, Harris G, Toohill RJ, Smith TL (2001) The silent sinus syndrome: a case series and literature review. Laryngoscope 111:975–978
24. Thomas RD, Graham SM, Carter KD, Nerad JA (2003) Management of the orbital floor in silent sinus syndrome. Am J Rhinol 17:97–100
25. Sciarretta V, Pasquini E, Tesei F et al (2006) Endoscopic sinus surgery for the treatment of maxillary sinus atelectasis and silent sinus syndrome. J Otolaryngol 35:60–64