B-ENT, 2005, **1**, 25-29

Chronic maxillary atelectasis

G. Hens, R. Hermans** and M. Jorissen**

*Department of Otorhinolaryngology, University Hospitals Leuven, Kapucijnenvoer 33, 3000 Leuven, Belgium; **Department of Radiology, University Hospitals Leuven, Herestraat 49, 3000 Leuven, Belgium

Key-words. Maxillary sinus; atelectasis; sinus surgery

Abstract. *Chronic maxillary atelectasis.* Chronic maxillary atelectasis (CMA) is characterized by a reduced maxillary sinus volume due to an inward bowing of one or more of the sinus walls. The disorder is probably caused by an obstruction of the maxillary ostium, leading to a persistent negative pressure within the sinus lumen.
To provide insight into the epidemiology, pathogenesis and treatment of this disorder, a retrospective study of twelve cases that met radiographic criteria of CMA was carried out. The patients were equally divided between both sexes and were on average 25 years old. Five of the twelve patients were under eighteen years of age. The patients had chronic sinonasal complaints except two, who had a "silent sinus syndrome", characterized by enophthalmos associated with a marked sinus deformation. This is the first report of CMA associated with a benign nasal tumour and also of CMA following cicatrisation due to nasal packing for bleeding after endoscopic sinus surgery. All patients were treated surgically by creating a middle meatal antrostomy, thus restoring sinus ventilation.
To conclude, CMA is rare and probably underestimated, especially in the paediatric population. Different entities causing a complete ostial occlusion can lead to CMA. Endoscopically restoring maxillary sinus ventilation is the recommended treatment.

Introduction

Unilateral enophthalmos due to a decrease in volume of the maxillary sinus was first reported by Montgomery in 1964.[1] In 1996, the term "chronic maxillary atelectasis (CMA)" was coined by Kass and colleagues who defined it as an inward bowing of one or more of the walls of the maxillary sinus, with at least partial opacification of the antrum due to mucous secretions.[2] The favoured hypothesis on the pathogenesis of CMA is that an obstruction to the maxillary sinus ostium causes persistent negative pressure in the antrum. Rabbits in which the maxillary ostium was occluded, developed a subatmospheric antral pressure probably as a result of absorption of sinus gases into the sinus mucosa.[3] In patients with CMA, the presence of negative pressures in the affected maxillary sinuses was manometrically confirmed, while atmospheric pressures were found both in the normal contralateral maxillary sinuses as well as in maxillary sinuses of patients with chronic sinusitis.[2,4] Similar to the development of serous otitis media following Eustachian tube obstruction, this negative pressure causes secretion of an acellular transudate, which is replaced by thick mucus in time. The inward directed pressure leads to bone remodelling and an inward displacement of the sinus walls, a process that may be enhanced by chronic inflammation of the sinus mucosa.[2]

A subgroup of patients without any preceding sinonasal complaints, presents with enophthalmos and hypoglobus due to an advanced stage of CMA. These patients usually consult an ophthalmologist before being referred to an ENT-specialist. Soparkar *et al.* named this disease presentation the "Silent Sinus Syndrome".[5]

In this article, we report our experience with twelve cases of CMA and discuss aspects of epidemiology, pathogenesis and treatment.

Materials and Methods

A retrospective study of twelve cases of CMA diagnosed at the ENT department of the University Hospitals Leuven from April 1996 until June 2003 was carried out. All cases met radiographic criteria of CMA, namely an inward bowing of at least one of the sinus walls and an opacification of the sinus antrum (Figure 1).

Presented at the Meeting of the Royal Belgian Society of E.N.T., February 22, 2003, Brussels, Belgium.

Table 1

Age, sex, side of involvement and presenting symptoms of twelve cases of CMA, diagnosed at the University Hospitals Leuven between April 1996 and June 2003

	Age (Years)	Sex	Side	Sinonasal symptoms	Enophthalmos
1	9	F	R	+	–
2	10	F	R+L	+	–
3	12	M	R	+	–
4	12	F	R	+	–
5	14	M	L	+	–
6	18	F	R	+	–
7	20	F	R	+	–
8	25	M	R	-	+
9	32	M	L	+	–
10	33	M	L	-	+
11	56	M	L	+	–
12	58	F	R	+	–

Results

The mean age of our patients was 25 years, with a range from 9 to 58 years (Table 1). Five of the twelve patients were under eighteen years of age. All but one had unilateral disease (right/left: 7/4). There was no gender predominance (male/female: 6/6).

Sinonasal symptoms (postnasal drip, runny nose, nasal obstruction and/or headache) were present in ten patients. The other two patients presented with enophthalmos (table 1, patient 8 and 10).

A middle meatal antrostomy was created in all patients during endoscopic sinus surgery. Peroperative biopsies of the mucosa of the affected maxillary sinus showed oedema and a mononuclear inflammatory infiltrate in all the cases. In two patients, eosinophils were also present. One of them had an allergic rhinitis due to house dust mite (table 1, patient 5), the other NARES (non-allergic rhinitis with eosinophilia syndrome) (table 1, patient 7).

In all patients the antrostomy was endoscopically patent during follow-up consultations at one, two, three and seven weeks. At the last visit the antral mucosa was endoscopically normal and the patients were free of complaints, except for two with persistent headache.

The CMA was idiopathic except in two patients. Patient 8 (table 1) had developed a rapidly progressive, right-sided enophthalmos six months after an endoscopic sinus procedure on the right anterior ethmoid and frontal sinus for chronic sinusitis. The procedure was complicated by severe epistaxis and nasal packing was performed immediately postoperatively. This lateralized the middle concha and caused cicatricial obstruction of the right maxillary ostium. A CT-scan showed maxillary atelectasis on the right side (Figure 2). In a subsequent endoscopic procedure, the middle concha was detached and an antrostomy was created.

Patient 11 (table 1) initially presented with unilateral nasal obstruction. A CT-scan showed an obstruction of the right maxillary ostium due to a solitary nasal polyp and ipsilateral maxillary

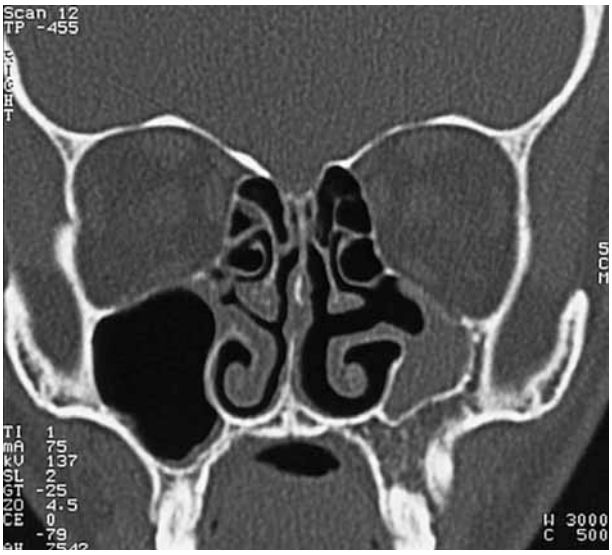

Figure 1
Patient 9 (table 1)

The left maxillary sinus displays typical features of CMA: inward bowing of the sinus walls and opacification of the sinus lumen.

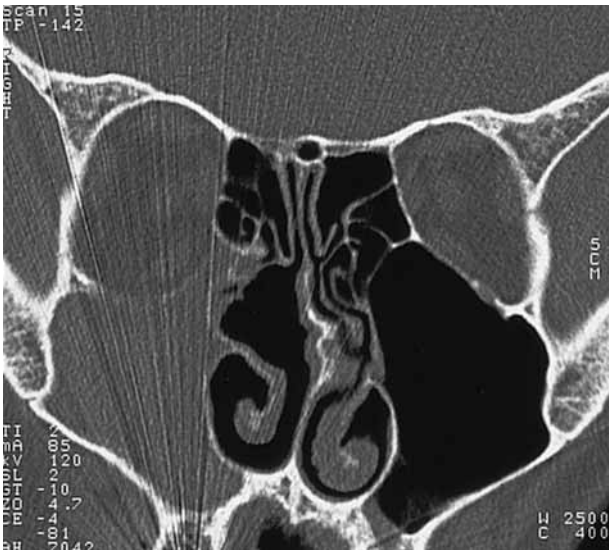

Figure 2
Patient 8 (table 1)

Right-sided silent sinus syndrome due to lateralization of the middle turbinate and scar tissue formation, six months after endoscopic sinus surgery complicated by severe epistaxis requiring nasal packing.

atelectasis (Figure 3 and 4). The polyp was resected and an antrostomy was created. Anatomopathologic examination revealed an hemangioma.

Discussion

Chronic maxillary atelectasis is a rare disorder. In our department, twelve patients were diagnosed with CMA over a period of seven years. This accounts for 0.5% of endoscopic sinus procedures performed in our center.

The primary cause of CMA is an obstruction of the sinus ostium due to, for instance, nasal oedema which leads to a negative antral pressure and initially a lateralization of the medial infundibular wall. Kass *et al.* described this as a “flap-valve mechanism”: the lateralized medial infundibular wall sustains the obstruction of the ostium, and thus the negative antral pressure, even if the primary cause (oedema) resolves.[6] Although in most cases of maxillary sinusitis an obstruction of the maxillary ostium is also present, only few patients will develop CMA. The air-tightness of the obstruction is probably a critical factor. Kass *et al.* suggested that certain anatomic features in patients (a high and mobile infundibular wall) make a total occlusion easier and thus these patients are more prone to develop

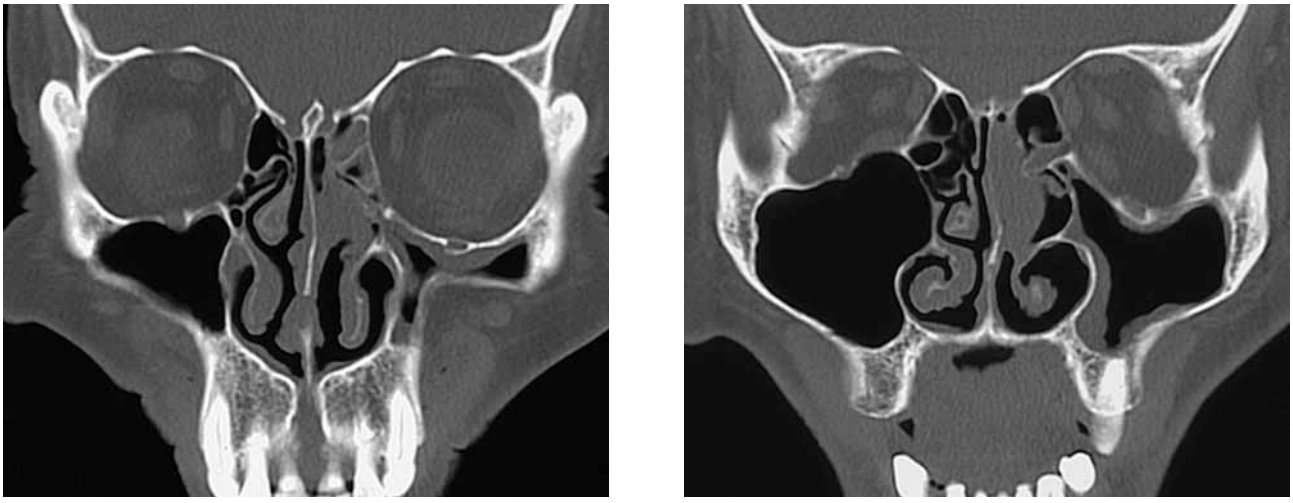

Figure 3 and 4
Patient 11 (table 1)

Solitary nasal polyp obstructing the left maxillary sinus ostium, and ipsilateral maxillary atelectasis.

CMA2. Secondly, a time factor could play a role, although in rabbits a negative antral pressure had developed 15 minutes after the occlusion.[3]

Besides oedema in anatomically prone patients, which probably accounts for the "idiopathic" cases, specific disease processes such as nasal polyps have been reported to cause ostial occlusion leading to CMA.[7] In our series, we describe for the first time CMA associated with, and most likely caused by, a (benign) nasal tumor, namely a hemangioma presenting as a solitary nasal polyp. We also report the first case with a silent sinus syndrome as a complication of nasal packing for severe epistaxis following on endoscopic sinus surgery. The tamponade lateralized the middle turbinate and scar tissue was formed which occluded the maxillary ostium.

In this series there is a high percentage of children. Although only three cases of CMA have previously been reported in the paediatric population,[8,9,10] five of the twelve patients in this series were under eighteen years of age. The prevalence of CMA in children may be underestimated, and this due to the difficulty in differentiating between CMA and maxillary sinus hypoplasia. The CT-graphic image of CMA resembles that of maxillary sinus hypoplasia (especially type II hypoplasia[11] which is associated with opacification of the affected sinus, a hypoplastic or absent uncinate process and an absent or pathologic infundibulum). During the period of sinus growth (until 15 years of age[12]), the differential diagnosis is even more problematic since maxillary hypoplasia can still develop at this age and even a previous normal CT-scan does not exclude maxillary hypoplasia. The degree of pneumatization of the maxilla is crucial in the differentiation, since hypoplasia is caused by growth arrest and thus marked by pneumatization that is underdeveloped in relation to the age of the patient, while CMA is marked by a sinus volume that is reduced by inward bowing of the sinus walls in a normally pneumatized maxilla (Table 2). Both disorders can occur simultaneously and CMA, with prolonged hypoventilation of the sinus, may even be the cause of an arrest in pneumatization of the maxilla.[13] In 1998, CMA in a 9-year-old boy was manometrically confirmed by Kass *et al.*[9]

Table 2
Differences between CMA and maxillary sinus hypoplasia

	CMA	**Maxillary sinus hypoplasia**
	Reduction of maxillary sinus volume	
Pathogenesis	Atelectasis of sinus walls due to negative antral pressure	Growth arrest of the maxillary sinus
Age of development	Any age	During period of sinus growth (< 15 years)
Radiology	Inward bowing of sinus walls Normal degree of pneumatisation in relation to age	Underdeveloped maxillary pneumatization in relation to age

Apart from maxillary sinus hypoplasia in the differential diagnosis of CMA, previous Caldwell-Luc surgery has to be considered since this may result in a decrease in antral volume[14] and is often associated with thickening of the sinus walls due to fibro-osseous proliferation.

In our series, ten patients had sinonasal complaints and two had the silent sinus syndrome with enophthalmos. If the diagnosis is not made incidentally in patients without nasal symptoms, this will only occur in more advanced stages of CMA when the deformity becomes clinically apparent. Indeed, there is an inverse correlation between the severity of the sinonasal symptoms and the degree of bony deformity in CMA.[15] The general prevalence of CMA in the population is thus likely to be underestimated. Analysis of large numbers of cranial CT scans for non-sinus related problems is warranted to understand the epidemiological importance of asymptomatic CMA.

Since it is not known how frequent and in which time period CMA progresses to a clinically apparent deformity, all cases are treated surgically with endoscopic techniques. Blackwell *et al.*[16] were the first to replace the Caldwell-Luc procedure by the endoscopic sinus surgery in the treatment of CMA. The goal is to restore sinus ventilation and drainage by creating a wide middle meatal antrostomy. It is crucial to give attention to the changed anatomy in CMA, since the lateral displacement of the uncinate process and the narrowed infundibulum increase the risk of an accidental penetration of the orbit. In patients with clinically apparent facial asymmetry, simultaneous orbital floor reconstruction can be performed. However, spontaneous improvement in

enopthalmos has been reported to occur within the first six months after maxillary antrostomy, and this is an argument for a two-staged approach.[17] In our series, the two patients with enophthalmos chose not to undergo reconstructive surgery as their facial asymmetry was mild.

In our series, no systematic long term follow-up consultations or control CT-scans were carried out. However, in the 11 to 96 months following endoscopic sinus surgery, none of the twelve patients complained of recurrence of sinonasal symptoms or progression of enophthalmos.

Conclusion

Although CMA is a rare disorder, its prevalence is probably underestimated because of the existence of a group of asymptomatic and undiagnosed patients. There was a high percentage of children in our case series. The underestimation of the prevalence of CMA in this group may be due to the difficulty in differentiating CT-graphically between CMA and maxillary sinus hypoplasia.

Treatment of CMA is based on restoring sinus ventilation. This is achieved endoscopically by creating a middle meatal antrostomy, while giving attention to the changed anatomy of the uncinate process and the infundibulum.

References

1. Montgomery WW. Mucocele of the maxillary sinus causing enophthalmos. *Eye Ear Nose Throat Mon.* 1964;43:41-44.
2. Kass ES, Salman S, Montgomery WW. Manometric study of complete ostial occlusion in chronic maxillary atelectasis. *Laryngoscope.* 1996;106:1255-1258.
3. Scharf KE, Lawson W, Shapiro JM, Gannon PJ. Pressure measurements in the normal and occluded rabbit maxillary sinus. *Laryngoscope.* 1995;105: 570-574.
4. Davidson JK, Soparkar CN, Williams JB, Patrinely JR. Negative sinus pressure and normal predisease imaging in silent sinus syndrome. *Arch Ophthalmol*. 1999;117:1653-1654.
5. Soparkar CN, Patrinely JR, Cuaycong MJ, *et al.* The silent sinus syndrome. A cause of spontaneous enophthalmos. *Ophthalmol*. 1994; 101:772-778.
6. Kass ES, Salman S, Rubin PA, Weber AL, Montgomey WW. Chronic maxillary atelectasis. *Ann Otol Rhinol Laryngol.* 1997;106:109-116.
7. Hayes EJ, Weber AL. Chronic sinus disease:a rare cause of enophtalmos. *Ann Otol Rhinol Laryngol.* 1987;96: 351-353.
8. Kass ES, Salman S. Developmental changes in the antrum of a child with chronic maxillary atelectasis. *Ann Otol Rhinol Laryngol.* 1999;108: 1091-1094.
9. Kass ES, Salman S, Montgomery WW. Chronic maxillary atelectasis in a child. *Ann Otol Rhinol Laryngol*. 1998;107:623-625.
10. Proctor T, Naclerio R. Development of a hypoplastic maxillary sinus. *Ann Otol Rhinol Laryngol.* 1996;105:327-328.
11. Bolger WE, Woodruff WW, Morehead J, Parsons DS. Maxillary sinus hypoplasia: classification and description of associated uncinate process hypoplasia. *Otolaryngol Head Neck Surg.* 1990;103:759-765.
12. Jovanovic S, Jelicic N, Kargovska-Klisarova A. Le développement postnatal et les rapports du sinus maxillaire. *Acta Anat*. 1984;118:122-128.
13. Milczuk HA, Dalley RW, Wessbacher FW, Richardson MA. Nasal and paranasal sinus anomalies in children with chronic sinusitis. *Laryngoscope.* 1993;103:247-252.
14. Ünlü HH, Caylan R, Nalça Y, Akyar S. An endoscopic and tomographic evaluation of patients with sinusitis after endoscopic sinus surgery and Caldwell-Luc operation: a comparative study. *J Otolaryngol.* 1994;23:197-203.
15. Hunt SM, Tami TA. Sinusitis-induced enophthalmos: the silent sinus syndrome. *Ear Nose Throat J.* 2000;79: 576,579-581,584.
16. Blackwell KE, Goldberg RA, Calcaterra TC. Atelectasis of the maxillary sinus with enophthalmos and midface depression. *Ann Otol Rhinol Laryngol*. 1993;102:429-432.
17. Thomas RD, Graham SM, Carter KD, Nerad JA. Management of the orbital floor in silent sinus syndrome. *Am J Rhinol.* 2003;17:97-100.

Mark Jorissen
Dienst Neus-, Keel- en Oorziekten
Kapucijnenvoer 33
B-3000 Leuven, Belgium
E-mail :
Mark.Jorissen@uz.kuleuven.ac.be