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Relationship between mucoceles, nasal polyposis and nasalisation*

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SUMMARY

The etiology of sinus mucoceles remains somewhat obscure, but favorizing factors can be broadly divided in two: inflammation and trauma. Patients suffering from nasal polyposis offer a unique group for the study of mucoceles as they present the factor of inflammation related to their polyposis, and the factor of trauma when being treated surgically. In order to establish the relationship between nasal polyposis, mucoceles and nasalisation, we performed a retrospective study on the files of 501 patients operated according to the nasalisation technique. We then selected all the patients who also presented with a mucocele that could be diagnosed before, during or after the surgery. We also noted the presence of associated pathologies like bronchial asthma or the Fernand Widal's syndrome (Samter's triad), in order to see if they played a role in mucocele formation.

Our study group finally included 36 patients that presented one or several mucoceles in association with nasal polyposis. Only three of them did not have any antecedent of surgery or known trauma, giving an incidence of mucoceles associated to a non-surgically treated polyposis of 0.6%. The mean incidence rate of mucocele formation after nasalisation for nasal polyposis was estimated to be of 2.5/100 patients per year. The real incidence may be greater as some patients may have been lost in follow up. Sixteen patients presented multiple mucoceles. The most frequent location was the fronto-ethmoidal region. The formation of mucoceles, or their multiplicity, did not appear to be influenced by the presence of an associated pathology, as up to one half of the patients presented an isolated nasal polyposis. Nevertheless, when associated pathologies were present, mucoceles were more frequently observed in the Widal's triad. Most of the mucoceles were diagnosed during the first 6 years after nasalisation, with a peak incidence around year 2 and 3.

We conclude that nasalisation, like other endonasal endoscopic techniques, can be related to a greater incidence of mucoceles than non-surgically treated nasal polyposis. Associated pathologies do not seem to influence mucocele formation. It is important for the surgeon to follow-up patients operated of a nasal polyposis as mucoceles can develop very lately.

Key words: mucocele, nasal polyposis, nasalisation, Fernand Widal.

INTRODUCTION

After almost two centuries since the first description of sinus mucoceles by Langenbeck in 1818, their pathogenesis remains somewhat obscure. Mucoceles have been defined as mucus-filled pseudo-cystic formations (Raynal et al., 1999) that are initially limited by bony walls which are expanded progressively during their development. They behave like real space-occupying lesions that cause bone erosion and surounding structures displacement.

In thirty percent of the cases, a cause can not be elucidated, and this fact adds to the confusion of describing a clear etiopathology. Mucoceles also represent 18% of all secondary orbital tumors, and over 80% originate from the frontal or ethmoid sinus (Henderson, 1994). They can be present at almost any age group with a median age of 51 years, and their gender distribution is equal (Kennedy et al., 1989; Henderson, 1994; Raynal et al., 1999; Har-El, 2001). When there are identifiable causes, these can be broadly divided into two main mechanisms: obstruction and inflammation. Obstruction can be related to craniofacial trauma, surgical trauma, tumors and nasal polyps, and inflammation is present with infection, allergy, cystic fibrosis and increased mucus secretion (Lund, 1987).

There is no doubt that inflammation is by far the mechanism that explains better the clinical behavior of mucoceles. Lund already demonstrated the presence of inflammation mediators

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in the epithelial lining of mucoceles (Lund, 1987). In a study on dogs, Schenck et al. failed to produce a mucocele by blocking the nasofrontal duct (Schenck et al., 1974).
Trauma is by itself another important favorizing factor, and even if an obstructive mechanism does not explain by itself the appearance of mucoceles, several cases are preceeded by craniofacial trauma, or surgical trauma, sometimes several years before (Lund, 1987; McFadden et al., 1996). Surgical trauma can be accounted for another somewhat considerable amount of mucoceles, and several reports in the literature relate sinus surgery, should it be by external or by endonasal approach, to the development of these space-occupying lesions. The expansion of endonasal endoscopic surgical techniques has then logically been followed by a dramatic rise in the number of cases of mucoceles (Raynal, 1999).

In our otolaryngology and Head & Neck department the main surgical technique that has been used for several years for the treatment of nasal polyposis is nasalisation. This technique is nothing else than a complete ethmoido-sphenoidectomy with wide middle meatus antrostomy and resection of the middle turbinate. This is performed systematically with the complete removal of all the ethmoidal mucosa as described by Jankowski (1995). The technique has already been described in other reports and is not the object of the present discussion, but since all endoscopic endonasal surgery can lead to the apparition of mucoceles, it is logical to think that nasalisation is not free of this complication.
Indeed we have observed that some post-operated patients have presented mucoceles, but there are also some patients that present mucoceles related to a nasal polyposis without any prior surgical procedure. Patients operated of a nasalisation for nasal polyposis offer a unique group for studying the mucoceles, as they present the two main factors of inflammation (by their chronic polyposis) and trauma (because of the surgery itself).
It seemed important to us to consider the fact that some pathologies associated with nasal polyposis, like bronchial asthma or Fernand Widal syndrome (Samter's triad), could play a role in mucocele formation, as they are tightly bound to inflammation mechanisms. Reports in the literature have effectively shown that patients with Fernand Widal presented more agressive nasosinusal involvement with higher incidence of orbital complications, including mucoceles (Mc Fadden, 1996).

In the present paper, we first tried to determine the frequency of spontaneous or non surgical-related mucoceles in patients with nasal polyposis, as polyps are associated with chronic inflammation, and cause obstruction by themselves.
Second, we tried to determine the frequency of mucoceles after nasalisation, and third, to determine if associated pathologies like asthma or Fernand Widal syndrome may play a role in the apparition of mucoceles.

PATIENTS AND METHODS

All the files from patients that presented mucoceles or were operated of a nasalisation between January 1997 and January 2003 were reviewed. Only patients who presented mucoceles in association with nasal polyposis were retained, whether they had been submited to a prior nasalisation or not.
Mucoceles were diagnosed on CT-Scan by the presence of a well-defined, homogeneous, smooth and rounded enlargement of a sinusal cavity or air cell, which presented a low density on CT-Scan (Weber, 1987). When MRI was available, signal intensity could vary depending on the relative protein content of the mucocele and its degree of hydration.
In all cases, the diagnosis was confirmed surgically by the presence of mucoid material within an expanded cavity limited by thin bone walls.

All patients with nasal polyposis that had been treated surgically but with other surgical techniques instead of nasalisation (e.g. polypectomy or anterior ethmoidectomy) were excluded, and so were the patients that presented mucoceles related to other causes (e.g. craniofacial trauma).
All the incomplete files were eliminated.
The age and gender of the patient, the site of the mucocele, the number of mucoceles, the antecedent of prior nasalisation and the presence of associated pathologies like asthma or Fernand Widal syndrome were documented in each case.

RESULTS

Between January 1997 and January 2003, a total of 501 nasalisations were performed in our institution by the senior author, and a total of 62 patients were operated for mucoceles of different etiologies. Only 36 patients presented mucoceles in association with nasal polyposis, and these constituted our main study group. In this late group, age distribution was between 17 and 77 years, with a median age of 47.4 years. There were 13 females and 23 males.
From all 36 patients, 33 had been operated of a nasalisation before, and 3 presented a mucocele in association with a non-operated nasal polyposis. In these three patients, the mucoceles were discovered during the nasalisation. A total of 69 mucoceles were operated on these 36 patients (16 patients presented multiple mucoceles).
The sites of the mucoceles were quite variable (Figure 1). Thirty-six were fronto-ethmoidal, 18 were in the ethmoidal sinus, and 7 were purely frontal. There were also 5 sphenoidal mucoceles and one located in the maxillo-ethmoidal junction.

Of all 36 patients, 18 (50%) presented an isolated nasal polyposis, while the other half presented pathologies associated to the nasal polyposis (Figure 2). Of this last group, 13 presented the Fernand Widal triad (nasal polyposis, asthma and aspirin intolerance), 4 patients were asthmatic and 1 presented a Churg-Strauss disease (Figure 3).

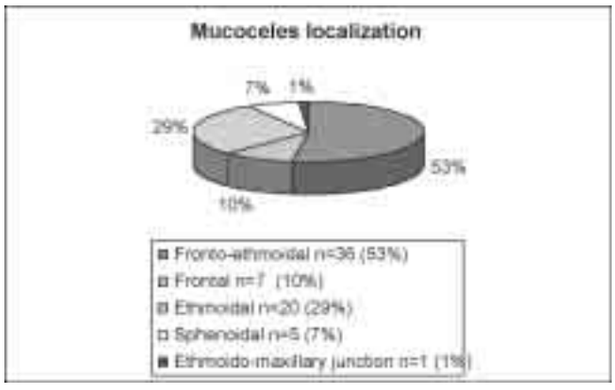

Figure 1. Localization of the 69 mucoceles in 36 patients with nasal polyposis.

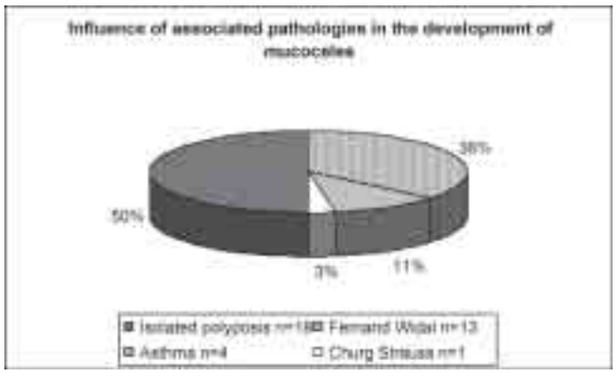

Figure 2. Incidence of mucoceles in nasal polyposis patients, with and without associated pathologies. Note that half of the patients present an isolated polyposis.

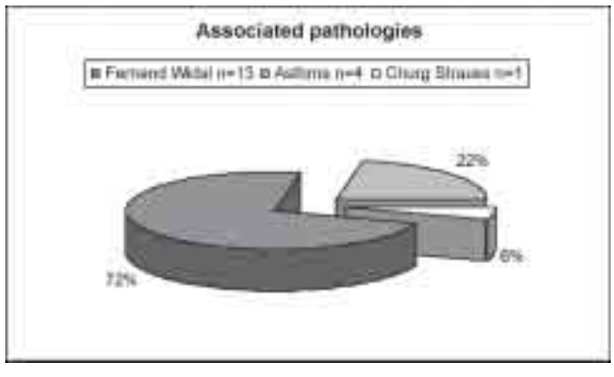

Figure 3. Associated pathologies to nasal polyposis. When these are present, they are dominated by the Fernand Widal's syndrome.

From the 16 patients that presented multiple mucoceles (Table 1), half had an isolated nasal polyposis, while the other half (8 patients) had an associated condition (Figure 4). Of these, 5 had a Fernand Widal syndrome, 2 were asthmatics, and 1 was the patient with the Churg Strauss disease.

Table 1. Presence of multiple mucoceles in patients with isolated nasal polyposis, and in patients with an associated pathology. Note that half of the patients present an isolated nasal polyposis.

	Single mucocele	Multiple mucoceles	Total
Isolated Polyposis	10	8	18
Polyposis with Fernand Widal	8	5	13
Polyposis with asthma	2	2	4
Polyposis with Churg Strauss	0	1	1
Total	20	16	36

Only 3 patients (0.6%) had not been operated before and their mucocele was discovered during the first sinus surgery. Two of these patients presented an isolated nasal polyposis, and 1 presented a complete Widal's triad.

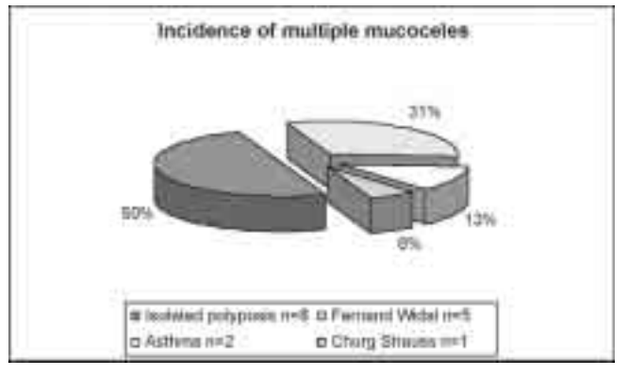

Figure 4. Incidence of multiple mucoceles in nasal polyposis patients with and without associated pathologies. Note that half of the patients present an isolated nasal polyposis.

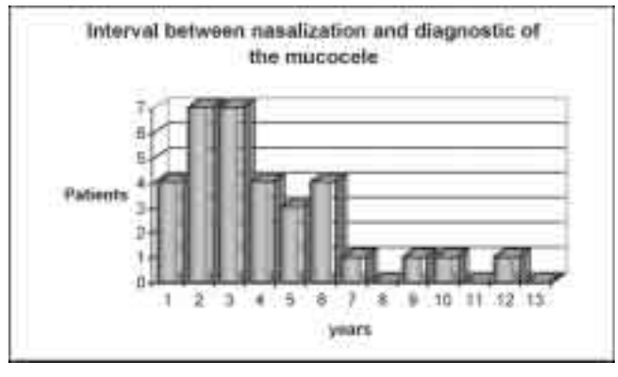

Figure 5. Time interval between the nasalisation and the diagnostic of the mucocele. Note that most of the mucoceles are diagnosticated during the first 6 years, with a peak incidence between year 2 and 3.

The time between the nasalisation and the diagnostic of mucocele was determined in each case (Figure 5). Most of the postoperative mucoceles were diagnosed during the first 6 years after the nasalisation, with a peak incidence between 2 and 3 years. However some patients developed a mucocele more than

Table 2. Year by year incidence of mucocèles expressed in percentage. The averaged percentage for the 6 years is 2.6%. This is a minimal incidence as some patients may have been lost on follow up.

Year	1997	1998	1999	2000	2001	2002
Number of nasalisations	86	82	95	85	81	72
Total of mucoceles in January 2003	3	3	5	0	2	0
Percentage	3.5%	3.7%	5.3%	0	2.5%	0
Years since nasalisation	6	5	4	3	2	1

10 years later. Table 2 shows the number of nasalisations that were performed each year from January 1997 to January 2003 (mean= 83.5/year; range: 72-95). On a total of 501 nasalisations over these 6 years, only 13 patients out of our series of 36 (2.5%) developed a mucocele at endpoint January 2003. The table also shows that 3 out of the 86 patients that were operated in 1997 (3.5%) developed a mucocele after 6 years of follow up. Similar data are obtained for each year (Table 2). These results show that the risk of developing a mucocele after a nasalisation lies between 3.5% and 5.3% after a follow-up of 4 to 6 years, and between 0 and 2.5% after only 1 to 3 years. The mean incidence rate of mucocele formation after nasalisation for nasal polyposis can be estimated at 2.5 patients/ 100 nasalisations per year.

DISCUSSION

Nasal polyposis has indeed been considered as a risk factor for developing mucoceles (Lund, 1987). Nevertheless, we can observe from our results that only 3 out of 501 patients presented a mucocele associated with a non-surgically treated nasal polyposis. The risk of developing spontaneously a mucocele in association with a nasal polyposis can therefore be estimated at around 0.6%. When compared to the percentage of patients presenting a mucocele after a nasalisation (mean percentage of 2.5%) the difference is quite significant. This last averaged percentage has to be considered cautiously, as it only considers the number of patients that presented symptomatic mucoceles that led them to seek for medical atention in our department. Some patients can be presenting asymptomatic mucoceles at this very moment, and some other might have been treated for a symptomatic mucocele in other institutions. Thus this percentage represents just a minimal average of patients that presented a mucocele after a nasalisation.

There is no doubt that endonasal endoscopic surgery is a major risk factor for the developement of mucoceles. Raynal et al. (1999) report in a study with 42 patients presenting a mucocele, that 22 of them (52.38%) had the antecedent of prior sinus surgery. Fourteen of these patients had been operated endoscopically, and the 8 others through an external approach.

Previous studies report that 60 to 80 percent of the mucoceles arise from the frontoethmoidal region (Lund, 1987; Kennedy et al., 1989; Raynal et al., 1999; Har-El, 2001). In our series, from 69 mucoceles, 36 where fronto-ethmoidal and 7 where purely frontal. This gives a percentage of 62.31% of frontal and fronto-ethmoidal mucoceles, similar to what has been described in the literature. It is interesting to note that 20 mucoceles out of 69 (28.98%) where limited exclusively to the ethmoidal sinus, without affecting the frontal sinus.

Our results confirm that surgery may be a more important factor in the development of mucoceles than nasal polyposis itself. Nevertheless, the role of inflammation must not be underestimated. Nasal polyposis is an inflammatory condition, and whether treated surgically or not, its role in mucocele formation has to be considered. Polyps can cause obstruction, but most of all, the diseased mucosa is the siege of inflammatory conditions that could favorize the formation of the mucocele. Jankowski states that polyposis is just the last stage of the development of a chronic non-allergic rhinitis with eosinophilia, in which the ethmoidal mucosa is the siege of major inflammatory changes (Jankowski, 1991). The studies by Lund et al. have demonstrated that inflammation mediators are present in mucoceles epithelial lining. High levels of PGE2, which appears to play a major role in the osteolytic process, were noted in the mucocele tissue and these could help to explain the locally agressive behavior of these expanding masses (Lund, 1987). Lund also demonstrated that the mucous lining of mucoceles contains a high amount of inflammation mediators (IL-1, TNF) and expresses the presence of cytokine-inducible vascular endothelial adhesion receptors like inter-cellular adhesion molecule (ICAM)-1 and E-Selectin (Lund et al., 1993). Then, polyposis is an ideal inflammatory condition that could favorize the development of mucoceles.

Reports have shown that nasal polyposis tends to be more agressive when associated to asthma and aspirin intolerance. McFadden et al. (1996) compared the incidence of orbital complications between a group of 81 patients presenting a Widal's triad and a group of 51 patients presenting chronic sinusitis, with or without polyposis. They report that 7 patients from the first group presented orbital involvement, 3 of these were cases of mucoceles. There were no orbital complications in the second group. This study suggested that nasal polyposis could increase the risk of mucocele development when accompanied by aspirin intolerance and asthma (Mc Fadden et al., 1996). In normal subjects 60% of the arachidonic acid is metabolized through the cyclooxigenase pathway, and 40% through the

lipoxygenase pathway, but in Fernand Widal patients 90% of the metabolism of the arachidonic acid is deviated towards the lipoxygenase pathway. This leads to an increase in leucotrienes and eicosateranoic acids that have major bronchoconstrictor and vasoconstrictor effects, and increase vascular permeability (Sherman, 1983). Theoretically, this alteration of the arachidonic acid metabolism could be responsible of a more agressive behaviour of nasal polyposis in patients with Fernand Widal syndrome, and facilitate more the development of mucoceles. This does not seem to be the case in our series, as we can observe that the incidence of mucoceles is the same in patients presenting associated pathologies as in patients with an isolated polyposis. Thus, mucocele formation does not seem to be influenced by the presence or absence of a bronchial asthma or of a Widal syndrome. Nevertheless, it is important to note from our series that when present, associated pathologies are strongly dominated by the Widal's syndrome.

Moreover, we can also observe that the development of multiple mucoceles does not seem to be influenced either by the presence or abscence of asthma and/or aspirin intolerance, and we observed that the number of patients presenting multiple mucoceles (n=18) is the same in patients with and without associated pathologies to nasal polyposis.

It would be interesting to know if a Fernand Widal syndrome or a bronchial asthma could play a role in the development of mucoceles in patients with a non-surgically treated nasal polyposis. In these cases, the inflammatory stimuli from the nasal polyposis would not be disturbed by surgical trauma, and eventually the role of these associated pathologies on mucocele formation could be defined more precisely. Unfortunately in our series, only 3 patients had a nasal polyposis without any prior surgical treatment (2 with an isolated nasal polyposis and 1 with a Widal's syndrome). The number of patients in this case is too small to draw any conclusion.

Another interesting point to discuss is the delay that usually exists between sinus surgery and the apparition of mucoceles. Classically, authors have described that a long period of time, sometimes as long as 23 years, elapses between surgery and the diagnosis of mucoceles (Lund, 1987; Moriyama et al., 1992; McFadden et al., 1996). However, our results show that in our patients, mucoceles tend to develop more frequently during the first 6 years after surgery, with a peak incidence between the second and third year. These results are comparable to the ones obtained by Raynal et al. (1999). These authors described from a study in 42 patients, that when sinus surgery was performed through an endonasal endoscopic approach, the interval between surgery and the apparition of the mucocele averaged 22 months. Moreover, when the sinus surgery was performed through an external approach, the same interval averaged 13 years (Raynal et al., 1999). Endoscopic endonasal surgery is then associated with an earlier development of mucoceles. This is important as we observed that our minimal estimated percentage of patients presenting a risk of mucocele is of 2.5%, and we know that non-treated mucoceles can undertake dangerous complications like orbital or intracranial complications, and even optic nerve compression (Lund and Rolfe, 1989; Moriyama et al., 1992; McFadden et al., 1996). A possible way to avoid these complications could be an early detection of the mucoceles by including in the post-operative follow-up one annual visit with endoscopic control during the first 6 years after surgery, and the systematic realization of a control CT-scan or a MRI of the sinuses at post-operative year 1, 3, and 6. This is just a proposed guideline for the follow up of patients after nasalisation or endoscopic sinus surgery. However, further studies are needed in order to establish if this follow up is adequate and if it will permit to detect mucoceles earlier in order to avoid severe complications. This also has to be the object of further discussion, as mucoceles are characterized by having a variably long inital asymptomatic phase (Raynal et al., 1998), and they also can recur several years later after their detection and marsupialization (Kennedy, 1989).

CONCLUSION

Nasal polyposis is by itself a predisposing factor for the develoment of mucoceles, as this disease is characterized by a disregulation in inflammatory processes, and polyps could cause drainage obstruction by themselves. Nevertheless, the risk of mucoceles in non-surgically treated nasal polyposis is quite low (0.6%) when compared to the risk of developing mucoceles after nasalisation (minimal estimated percentage of 2.5%). Surgery then remains as the most important favorizing factor for the development of mucoceles. The presence of aspirin intolerance and bronchial asthma does not seem to play a major role in the formation of mucoceles, nor in their multiplicity, as up to one half of the patients do not present this associated pathologies. It is important to establish new guidelines for the follow-up of patients operated on nasal polyposis, as this could enable the surgeon to detect asymptomatic mucoceles and avoid severe complications.

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