REVIEW

Rhinology, 43, 178-182, 2004

The antrochoanal polyp*

Miguel Maldonado[1,2], Asunción Martínez[2], Isam Alobid[1], Joaquim Mullol[1,2]

[1] Rinology Unit, Otolaryngology Department, Hospital Clinic, Barcelona, Spain
[2] Clinical and Experimental Respiratory Immunoallergy, IDIBAPS, Barcelona, Spain

SUMMARY

Antrochoanal polyps (ACP) are benign lesions that arise from the mucosa of the maxillary sinus, grow into the maxillary sinus and reach the choana, and nasal obstruction being their main symptom. It is an interesting model to compare the pathophysiological mechanisms with those of bilateral nasal polyposis (NP). There is a lack of research in some areas of ACP, which have to become the main aims for future investigations of this disease. In comparison to NP, ACPs are usually unilateral and appear in younger patients. Macroscopically, they have a cystic intramaxillary portion and a solid intranasal portion. Microscopically, they are similar to a maxillary cyst of the mucosa. Nasal endoscopy, computed tomography and magnetic resonance are the main diagnostic techniques. Surgery is the indicated treatment for ACP, with endoscopic resection the most recommended. Simple avulsion of the polyp has a high rate of recurrence, whereas the Caldwell-Luc procedure is associated with damage of the maxillary and dental growth centres. More research is needed to establish the relationship between chronic sinusitis and ACP. Further work is also needed to investigate the role of inflammatory mediators (histamine, IgE, adhesion molecules, PAF), as well as metalloproteases and nasal remodelling, and tumour marker expression in ACP.

Key words: antrochoanal polyps, inflammation, epidemiology, diagnosis, treatment

INTRODUCTION

Although great effort has been made during the last two decades in research into the molecular and cellular basis of bilateral polyposis, little is known about the pathophysiological mechanisms of antrochoanal polyps (ACP). ACP does not represent a severe condition, but it may be considered as a good model to compare with bilateral polyposis. Some evident differences between both diseases are known, but many questions have still to be answered. Why do two diseases involving nasal polyps manifest in different ways? Is antrochoanal polyposis an inflammatory process? Can ACP be considered a benign tumour? What is its intimate pathophysiological origin? The present review will try to present the similarities and differences of both diseases, as well as to point out the steps to follow in future research.

Palfyn reported the first case of an ACP in 1753 (Palfyn, 1753), although Killian was the first to describe this disease in detail in 1906 (Killian, 1906). In contrast to inflammatory nasal polyposis, which grows from the ethmoid cells (Larsen and Tos, 1991), an antrochoanal polyp is a benign lesion originating from the oedematous mucosa of the maxillary sinus, growing through the main or accessory ostium which is usually enlarged (Hong et al., 2001) into the middle meatus and protruding posteriorly to the choana and nasopharynx (Figure 1). Stammberger found that antrochoanal polyps left the sinus through an accessory ostium in 70% of the cases (Stammberger and Hawke, 1993). Clinical manifestations usually start with unilateral nasal obstruction, although there are reports of cases starting with epistaxis (Robson et al., 1990), purulent rhinorrea (Basak et al., 1998), polyp strangulation (Ole-Lengine and Manni, 1993), spontaneous amputation (Rashid et al., 1994), dyspnoea and dysphagia (Grewal and Sharma, 1984), falling into the hypopharynx (Lavetskii and Protasevich, 1973), occupying the mouth to produce dysphagia and speech disturbances (Sharma and Daud, 1997), and obstructive sleep apnoea and cachexia (Salib et al., 2000).

EPIDEMIOLOGY

Antrochoanal polyps are usually unilateral with only two bilateral cases reported in the literature (Myatt and Cabrera, 1996; Basu et al., 2001), both in paediatric patients. Different authors have reported a high incidence of ACP in young patients. In a recent prospective study involving 252 patients, Larsen and Tos (2002) found that at the time of diagnosis the mean age of nasal polyposis and antrochoanal polyposis was 50 and 27 years respectively. Chen et al. (1989) found 28% of antrochoanal polyps occurred in children.

Conversely inflammatory nasal polyposis presents much later in life, being very rare in children. Settipane and Chaffee (1977) studied 4986 adult patients from the Rhode Island

*Received for publication: October 9, 2003; accepted: July 7, 2004

Hospital Adult Allergy Clinic and found a rate of 4.2% nasal polyposis, observing a higher incidence in non-atopic asthmatic patients (12.5%) in comparison to atopic asthmatics (5%). In patients suffering from rhinitis, the incidence of bilateral nasal polyposis was 2.2%. The rate of nasal polyps was also higher in non-atopic rhinitic patients (4.7%) than in atopic rhinitic patients (1.5%). This rate increases with age in all groups (1.8% in asthmatic aged 10 to 19 in comparison with 14.6% in patients older than 50), probably due to a longer time of evolution of the inflammatory process. In the same study, 1051 paediatric patients with asthma and rhinitis with a mean age of 6 years were analysed, finding only one case of inflammatory nasosinusal polyposis (0.1%). This study did not analyse antrochoanal polyposis.

While no preponderance of gender has been found in ACP patients (Chen et al., 1989; Orvidas et al., 2001), bilateral nasal polyposis is more frequent in males (Settipane and Chaffee, 1977). However, nasal polyposis associated with aspirin induced asthma is more frequent in women (Szczeklik et al., 2000). In a series of 16 patients from the army medical academy in Gülhane, Turkey, a higher rate of ACP was found in males (87.5%) (Aktas et al., 1998), but the type and low number of patients in this study probably represents a significant bias in relation to sex incidence.

HISTOPATHOLOGY

Macroscopically, the antrochoanal polyp is composed of a cystic part filling the maxillary sinus and a solid part coming out through the maxillary ostium into the middle meatus and thence to the choana (Berg et al., 1988). Microscopically, the polyp shows a central cavity surrounded by a homogeneous oedematous stroma bearing few cells. The polyp surface is covered with respiratory epithelium.

According to Hellquist and from a histological point of view, nasal polyps can be classified in four groups: (1) oedematous, eosinophilic or "allergic" is the most frequent type, formed by oedematous connective tissue and isolated glands, without development of cysts with a rich eosinophilic infiltrate and goblet cell hyperplasia; (2) ductal, formed by glands and cysts; (3) fibrous or fibroinflammatory, with a great proliferation of fibroblasts and collagen, and a lymphocyte inflammatory infiltrate; and (4) polyps with stromal atypia, which are very scarce and distinguished from a genuine neoplasm by the absence of mitosis (Hellquist, 1997).

Inflammatory cells

The inflammatory infiltrate in bilateral nasal polyps consists of leucocytes, mainly eosinophils, mast cells and lymphocytes (T suppressors being more frequent than T helper cells) (Lui et al., 1994). There is a predominance of EG2+ eosinophils, showing the activation of these cells (Picado and Mullol, 1998). Polyps from cystic fibrosis patients are rich in mast cells whereas the presence of eosinophils is rare (Henderson and Chi, 1992).

On the other hand, antrochoanal polyps show clear histologic differences when compared to bilateral polyps, consisting of a lower inflammatory infiltrate and a significantly lower eosinophil infiltrate (Min et al., 1995), whilst oedema is essentially the same. The fibrous type is significantly more frequent, possibly due to its long evolution that leads to a scarring stage. Submucous glands are less frequent than in non-antrochoanal polyps.

There are some similarities between antrochoanal polyps and maxillary mural cysts which have led to the hypothesis that the former are the intranasal form of the latter. Berg et al. (1988) found that both tissues had a fluid content that turned to gel at room temperature. The ratio between proteins in the aspirated fluid and serum proteins (IgA, IgG, IgM, albumin, α1-antitrypsin, C3, C3d and of origin C4) did not differ in both types, although in each case the implantation area was located in the inferolateral wall of the maxillary sinus. No histological differences were found between intramural cysts and antrochoanal polyps.

Since a high rate of ACP has been found in children with chronic rhinosinusitis (Wolf et al., 1994) it has also been suggested that antrochoanal polyps can be caused by acinar mucous gland obstruction in the recovery period of a chronic infection (Chen et al., 1989). However, other studies have not corroborated these findings (Basak et al., 1988).

Inflammatory mediators

A variety of chemical mediators with different proinflammatory activities have been described in nasal polyposis. These mediators can be classified into four groups (Hoffman and Wasserman, 1997):

1. Substances with a vasoactive function: histamine, platelet activating factor (PAF), prostaglandins, leukotrienes, norepinephrine, neuropeptid Y (NPY), and vasoactive intestinal peptide (VIP).
2. Substances influencing cell migration: high molecular weight neutrophil chemotactic factor (HMW-NCF), eicosanoids (prostaglandins and leucotrienes), interleukins, chemokines, and adhesion molecules (selectins, integrins, cadherins, mucins and immunoglobulins).
3. Substances acting on the extracellular matrix: transforming growth factor (TGF), tumor necrosis factor (TNF) and vasoactive intestinal peptide (VIP).
4. Mediators affecting cell growth, differentiation and proliferation: transforming growth factor (TGF), colony stimulating factors (CSF), insulin-like growth factor (IGF), tumor necrosis factor (TNF), and interleukins (IL).

ACP

Few inflammatory markers have been studied in ACP.

a) Cytokines play an important role in chemotaxis, differentiation, growth and cell proliferation. Proinflammatory cytokines such as IL-1ß, IL-6, and IL-8, have a neutrophil chemotactic effects and are implicated in acute rhinosinusi-

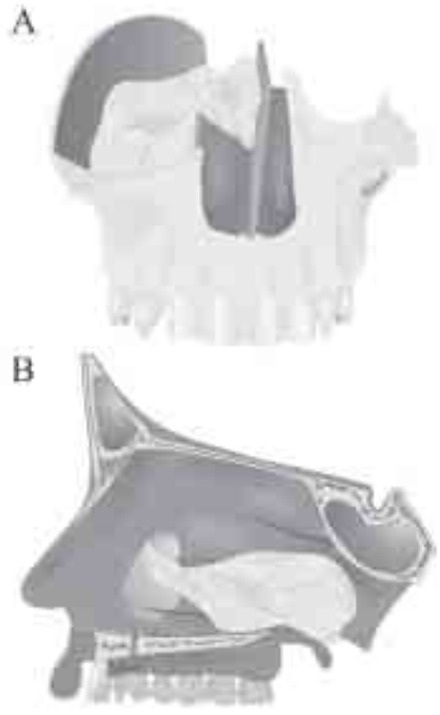

Figure 1. Diagram of an antrochoanal polyp.
A. Frontal view: the polyp grows from the mucosa that lines the maxillary sinus and exits into the middle meatus.
B. Saggital view: from the middle meatus, the polyp reaches the posterior choana and the cavum.

tis, whereas IL-3 is more related to chronic rhinosinusitis (Rudack et al., 1998) IL-5 has a chemotactic effect for eosinophils, and its synthesis is increased in nasal polyposis when compared to healthy mucosa or antrochoanal polyp (Rudack et al., 1998; Bachert and Van Cawenberger, 1997). ACPs show an enhanced expression of IL-6 in comparison to controls, and therefore more clearly resemble an acute infection than bilateral polyposis.

b) Plasminogen activator inhibitor type 1 (PAI-1) is the most efficient t-PA and u-PA known inhibitor, and plays an important role in the regulation of plasminogen activating system. u-PA has been related to progression in nasopharyngeal tumours and a suppressor activity for PAI-1 (Sunagawa et al., 1997) has been suggested. Some studies have demonstrated the existence of tissue plasminogen activator (t-PA) in mucosa from patients affected with chronic rhinosinusitis. In antrochoanal polyps, urokinase type plasminogen activator (u-PA) (Yamashiro et al., 1992) has also been found. Urokinase does not have a specific affinity for fibrin (Majerus et al., 1996) and it has been suggested that u-PA is not engaged in vascular fibrinolysis, but it may play a role in tissue remodelling and cell migration (Sunagawa et al., 1999).

Tumour markers

When considering antrochoanal polyps as benign tumour-like tissues, tumour markers need to be evaluated with a potential involvement in their development. Tumours arise from cells that have undergone specific genetic alterations in proto-oncogenes or tumour suppressor genes (Forastiere et al., 2001). These cells accumulate DNA staggered changes that favour cell survival. Several markers have been related to carcinogenesis. However, no studies have been carried out in ACP.

Diagnosis

Nasal endoscopy, computed tomography (CT) and magnetic resonance imaging (MRI) are the core diagnostic techniques used in bilateral and unilateral nasal polyposis. By using CT scanning, the diagnosis of ACP is made when a mass fills the maxillary sinus growing through the accessory or natural ostium into the middle meatus and the posterior choana. MRI (Vuysere et al., 2001) shows T1 hypointense and T2 enhanced signals in sinochoanal polyps (antrochoanal as well as those originating in other sinuses). When intravenous gadolinium is administered during MRI, the intrasinusal cystic part of the polyp is only peripherally enhanced, whereas the nasal and choanal regions show hyperintense images.

In childhood, the differential diagnosis with other paediatric masses should be assessed (Pruna et al., 2000). Among them, the most frequent maxillary masses are mucoceles and mucopyoceles. With the use of intravenous contrast these masses usually show a characteristic enhanced ring. A retention cyst derived from a salivary or mucous gland can also occur. However, this normally bears a crescent moon air density that helps to differentiate it from an antrochoanal polyp.

The sphenochoanal polyp is usually diagnosed by demonstrating its stalk growing from the sphenoid sinus wall and passing through the ostium into the posterior choana (Lessa et al., 2002; Weissman et al., 1991). Sphenochoanal polyps are less frequent than antrochoanal polyps, although they may have been underdiagnosed in the past, due to the use of plain X-ray, which cannot determine from which sinus the choanal polyp originates (Tosun et al., 2001).

Other less frequent differential diagnoses are adenoid hypertrophy, an ethmoidochoanal polyp, turbinate hypertrophy, Tornwaldt´s cyst, as well as tumours such as angiofibroma, olfactory neuroblastoma, or heamangioma. Other bone destroying diseases such as lymphoma, Wegener granulomatosis, or rhabdomyosarcoma (Grainger and Zammit-Maempel, 2001) should also be considered. Inverted papilloma is a typically unilateral lesion which must be differentiated from ACP. Lopatin et al., studied 20 cases of choanal polyps (11 antrochoanal, 3 sphenochoanal, and 5 ethmoidochoanal) finding 2 cases of inverted papilloma among them (Lopatin et al., 1997).

Treatment

The accepted treatment of antrochoanal polyps is removal by surgery. Several techniques have been described, including

simple polyp avulsion, an antral approach using a Caldwell-Luc technique, and endoscopic sinus surgery (El-Guindy and Mansour, 1994; Basak et al., 1998; Özdek et al., 2002). No studies regarding the efficacy of drug therapy efficacy in antrochoanal polyps have been reported. Simple polypectomy and the Caldwell-Luc approach, used for many years, have been relegated in favour of endoscopic sinus surgery (Basak et al., 1998). Simple polypectomy has a high rate of recurrence due to insufficient resection of the intramaxillary portion of the polyp (El-Guindy and Mansour, 1994). The Caldwell-Luc procedure can damage the growth centres of the maxillary bone and dental germs, cause cheek or tooth inflammation and/or anaesthesia. The endoscopic technique consists of an uncinectomy and resection of the polyp and its attachment to the maxillary wall (Stammberger and Hawke, 1993). In recurrent cases antrostomy through the canine fossa to resect the antral part of the polyp can be added to endoscopy (El-Guindy and Mansour, 1994; Özdek et al., 2002). The use of a microdebrider through the canine fossa as a way to properly resect a broad place of attachment (Hong et al., 2001) may be indicated as complementary to endoscopic surgery. We propose a diagnostic and therapeutic flow chart for antrochoanal polyposis (Figure 2).

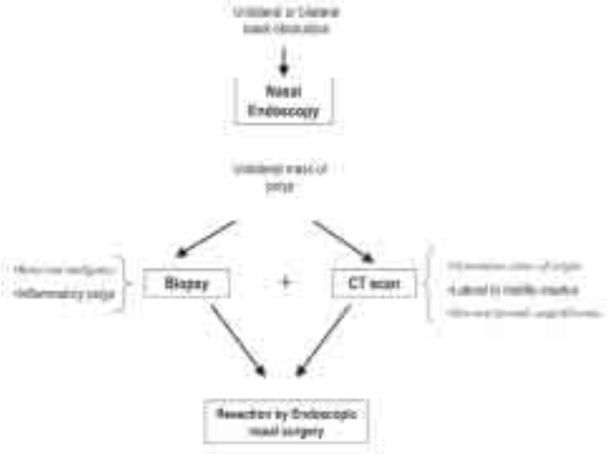

Figure 2. Flowchart showing the stepwise approach for the diagnosis and treatment of antrochoanal polyp.

CONCLUSIONS

The antrochoanal polyp is an interesting model to study and compare the pathophysiological mechanisms with those of bilateral polyposis. However, there is a lack of research in a number of areas, which should become the main focus of future investigation. More research is needed to establish the relationship between chronic rhinosinusitis and antrochoanal polyps. Further work is needed to investigate the role of inflammatory mediators (histamine, IgE, adhesion molecules, PAF), as well as metalloproteases and nasal remodelling, and tumour marker expression in this interesting condition.

REFERENCES

1. Aktas D, Yetiser S, Gerek M, Can C, Kahramanyol M (1998) Antrochoanal polyposis; analysis of 16 cases. Rhinology 36: 81-85.
2. Bachert C, Van Cawenberger PB (1997) Inflammatory mechanisms in chronic sinusitis. Acta Otorhinolaryngol Belg 51: 209-217.
3. Basak S, Karaman CZ, Akdilli A, Metin KK (1998) Surgical approaches to antrochoanal polyps in children. Int J Pediatr Otolaryngol 46: 197-205.
4. Basu SK, Bandyopadhyay SN, Bora H (2001) Bilateral antrochoanal polyps. J Laryngol Otol 115: 561-562.
5. Berg O, Carenfelt C, Silfverswärd C, Sobin A (1988) Origin of the choanal polyp. Arch Otolaryngol Head Neck Surg 114: 1270--1271.
6. Chen JM, Schloss MD, Azouz ME (1989) Antrochoanal polyps: a 10 year retrospective study in the pediatric population with a review of the literature. J Otolaryngol 18: 168-172.
7. El-Guindy A, Mansour MH (1994) The role of transcanine surgery in antrochoanal polyps. J Laryngol Otol 108: 1055-1057.
8. Forastiere A, Koch W, Trotti A, Sidransky D (2001) Head and neck cancer. N Eng J Med 345: 1890-1900.
9. Grainger AJ, Zammit-Maempel I (2001) Antrochoanal polyps in children. Eur Radiol 11: 347.
10. Grewal DS, Sharma BK (1984) Dyspnea and dysphagia in a child due to an antrochoanal polyp. Auris Nasus Larynx 11: 25-28.
11 Hellquist HB (1997) Histopathology. In: Settipane GA Ed. Nasal polyps: epidemiology, pathogenesis and treatment. Oceanside Publications. Providence, pp. 31-39.
12. Henderson WR Jr, Chi EY (1992) Degranulation of cystic fibrois nasal polyp mast cells. J Pathol 166: 395-404.
13. Hoffman HA, Wasserman SI (1997) Chemical mediators in polyps. In: Settipane GA Ed. Nasal polyps: epidemiology, pathogenesis and treatment. Oceanside Publications. Providence, pp. 41-47.
14. Hong SK, Min YG, Kim CN, Byun SW (2001) Endoscopic removal of the antral portion of antrochoanal polyp by powered instrumentation. Laryngoscope 111: 1774-1778.
15. Killian G (1906) The origin of choanal polipi. Lancet 2: 81-82.
16. Kowalski ML, Pawliczak R, Wozniak J, Siuda K, Poniatowska M, Iwaszkiewicz J, Kornatowski T, Kaliner MA (2000) Differential metabolism of arachidonic acid in nasal polyp epithelial cells cultured from aspirin-sensitive and aspirin-tolerant patients. Am J Respir Crit Care Med 161: 391-398.
17. Larsen K, Tos M (2002) The estimated incidence of symptomatic nasal polyposys. Acta Otolaryngol 122: 179-182.
18. Larsen PL, Tos M (1991) Origin of nasal polyposis. Laryngoscope 101: 305-312.
19. Lavetskii RT, Protasevich GS (1973) Choanal polyp descending into the hypopharynx. Zh Ushn Nos Gorl Bolezn 33: 75.
20. Lessa MM, Voegels RL, Pádua F, Wiikmann C, Romano FR, Butugan O (2002) Sphenochoanal polyp: diagnose and treatment. Rhinology 40: 215-216.
21. Liu CM, Shun CT, Hsu MM (1994) Lymphocyte subsets and antigen specific IgE antibody in nasal polyps. Ann Allergy 72: 19-24.
22. Lopatin A, Bykova V, Piskunov G (1997) Choanal Polyps: one entity, one surgical approach? Rhinology 35: 79-83.
23. Majerus PW, Broze GJ Jr., Miletich JP, Tollefsen DM (1996) Anticoagulant, thrombolytic and antiplatelet drugs. In: Hardman JG, Limbird LE Eds. Goodman & Gilman´s The pharmacological basis of therapeutics. 9th Ed. McGraw Hill. New York pp. 1341-1359.
24. Min YG, Chung JW, Shin JS, Chi JG (1995) Histologic structure of antrochoanal polyps. Acta Otolaryngol 115: 543-547.
25. Myatt HM, Cabrera M (1996) Bilateral antrochoanal polyps in a child: a case report. J Laryngol Otol 110: 272-274.
26. Nigam S, Kunkel G, Herold D, Baumer FE, Jusuf L (1986) Nasal polyps and their content of arachidonic acid metabalites. NER Allergy Proc 7: 109-112.
27 Ole-Lengine L, Manni JJ (1993) A strangulated antrochoanal polyp. A case report. J Laryngol Otol 107: 342-343.
28. Orvidas LJ, Beatty CW, Weaver AL (2001) Antrochoanal polyps in children. Am J Rhinol 15: 321-325.

29. Özdek A, Samim E, Bayiz Ü, Meral I, Safak MA, Oguz H (2002) Antrochoanal polyps in children. Int J Pediatr Otolaryngol 65: 213-218.
30. Palfyn J (1753) Anatomie Chirurgicale. Paris.
31. Picado C, Mullol J (1998) The nose in aspirin-sensitive asthma. In: Szczerklik A, Gryglewski RJ, Vane JR Eds. Eicosanoids, aspirin and asthma. Marcel Dekker Inc. New York pp. 493-505.
32. Pruna X, Ibañez JM, Serres X, Garriga V, Barber I, Vera J (2000) Antrochoanal polyps in children: CT findings and differential diagnosis. Eur Radiol 10: 849-851.
33. Rashid AM, Soosay G, Morgan D (1994) Unusual presentation of a nasal (antrochoanal) polyp. Br J Clin Practice 48: 108-109.
34. Robson AK, Barker CS, Whittet HB (1990) Epistaxis as an unusual presentation of an antrochoanal polyp. J Laryngol Otol 104: 643-644.
35. Rudack C, Stoll W, Bachert C (1998) Cytokines in nasal polyposis, acute and chronic sinusitis. Am J Rhinol 12: 383-388.
36. Salib RJ, Sadek SA, Dutt SN, Pearman K (2000) Antrochoanal polyp presenting with obstructive sleep apnoea and cachexia. Int J Pediatric Otorhinolaryngol 54: 163-166.
37. Settipane GA, Chafee FH (1977) Nasal polyps in asthma and rhinitis. A review of 6037 patients. J Allergy Clin Immunol 59: 17-21.
38. Sharma HS, Daud ARA (1997) Antrochoanal polyp - a rare paediatric emergency. Int J Pediatric Otorhinolaryngol 41: 65-70.
39. Stammberger H, Hawke M (1993) Essentials of functional endoscopic sinus surgery. Mosby, St Louis pp. 103-105.
40. Sunagawa M, Huang GW, Nakamura M, Kosugi T (1997) The concentration of u-PA and PAI-1 antigen in tissue extracts of nasopharyngeal carcinoma Eur Arch Otolaryngol 354: 277-280
41. Sunagawa M, Kinjoh K, Nakamura M, Kosugi T (1999) Urokinase-type plasminogen activator inhibitor antigen in tissue extracts of paranasal sinus mucous membranes affected by chronic sinusitis and antrochoanal polyps. Eur Arch Otolaryngol 256: 237-241.
42. Szczeklik A, Nizankowska E, Duplaga M and AIANE investigators (2000) Natural history of aspirin-induced asthma. Eur Respir J 16: 432-436.
43. Szczeklik A, Stevenson DD (2003) Aspirin-induced asthma: advances in pathogenesis, diagnosis and management. J Allergy Clin Immunol 111: 913-921.
44. Tosun F, Yetiser S, Akcam T, Özkaptan Y (2001) Sphenochoanal polyp: endoscopic surgery. Int J Pediatr Otorhinolaryngol 58: 87-90.
45. Vuysere S, Hermans R, Marchal G (2001) Sinochoanal polyp and its variant, the angiomatous polyp: MRI findings. Eur Radiol 11: 55-58.
46. Weissman JL, Tabor EK, Curtin HG (1991) Sphenochoanal polyps: evaluation with CT and MR Imaging. Radiology 178: 145-148.
47. Wolf G, Greistorfer K, Kainz J (1994) Unsere erfahrungen mit der endoskopisch-endonasalen operationstechnik bei der therapie der sinusitis im kindesalter. Laryngo-Rhino-Otol 73: 258-263.
48. Xaubet A, Mullol J, López E, Roca-Ferrer J, Rozman M, Carrión T, Fabra JM, Picado C (1994) Comparison of the role of nasal polyp and normal nasal mucosa epithelial cells on in vitro eosinophil survival. mediation by GM-CSF and inhibition by dexamethasone. Clin Exp Allergy 24: 307-317.
49. Yamashiro Y, Nakamura M, Huang GW, Kosugi T (1992) Presence of urokinase-type plasminogen activator (u-PA) in tissue extracts of antrochoanal polyp. Laryngoscope 102: 1049-1052.

Miguel Maldonado, MD
Otolaryngology Department
Hospital Clinic
C/ Villarroel 170
E-08036 Barcelona
Spain

E-mail: mmaldonadof@mixmail.com