SINONASAL POLYPOSIS: SIMPLE POLYPECTOMY VERSUS FUNCTIONAL ENDOSCOPIC SINUS SURGERY

(THESIS)
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Abstract

We recommend functional endoscopic sinus surgery for patients with polyposis when surgery is indicated as a primary surgery or the ***first attack surgery***, to obtain effective, anatomical surgery, also better postoperative improvement and longer disease free interval. Simple polypectomy is recommended for revision surgery to obtain its advantages of rapid, safe, low cost, minimal postoperative pain, short postoperative period of care and trouble, no complications, capability on outpatient bases and easy idea of revision.

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Introduction:

Nasal polyposis is a common problem facing the otolaryngologist. It's incidence is estimated to be in the range of ٠.٢ to ٤.٣ per cent **(Larsen K and Tos M, ٢٠٠٢).** It may be associated with other conditions such as bronchial asthma (about ٢٥%-٣٠% of polyposis patients) and Aspirin sensitivity (١٢ %) **(Vancil ME, ١٩٦٩).**

The main problem in management of nasal polyposis is its known high incidence of recurrence. Medical treatment is usually the first line of treatment, however, a considerable number of patients may not respond. Patients with associated asthma or ASA sensitivity are known to be more prone to lack of response. Non-responsers genrerally will be candidates for surgical intervention **(Vento SI. Etal ٢٠٠٠).**

Surgical treatment of nasal polyposis is even associated with remarkable rate of recurrence. The extent of surgery ranges from simple polypectomy to radical removal of all polypoid mucosa using the classic endoscopic sinus surgery.

Aim of work:

The aim of this study is to evaluate and compare the treatment outcome of simple polypectomy versus functional endoscopic sinus

surgery in the management of patients with allergic sinonasal polyposis, as regards symptom improvement, recurrence rate and complications.

Basic considerations

A- Etiology:

The etiology remains unknown or unclear and it is possible that it is not the same in all patients or may be a multifactorial one. Nasal polyps may represent a relatively uniform response to different etiologic agents. Also others consider nasal polyps are a physical finding or local manifestation of a systemic disease rather than a disease **(Caplin I etal, ١٩٧١)**.

Chronic Infection of the nose and paranasal sinuses is frequently present in patients with nasal polyps. As regards **Bacterial infection,** The most common pathogens are . -hemolytic streptococci, *Staphylococcus aureu*s, *Streptococcus pneumoniae*, and *Haemophilus influenza*e. Bacterial specific IgE has been detected in patients with nasal polyposis but not in patients with allergic rhinitis. Neutrophils and not eosinophils infiltrate the polyps in this setting. Recent investigations of experimental maxillary sinusitis in rabbits demonstrated both granulation-like polyps and edematous-like polyps. In these studies, the sinuses were inoculated with *S. pneumoniae* type ٣, *Bacteroides fragilli*s, or *S. aureu*s, after simple blocking of the sinus ostium. Granulation-like polyps were noted in regions of deep inflammatory trauma and edematous polyps were found in regions of superficial inflammatory trauma. Polyps were found in all sinusitis groups irrespective of an inducing agent and were not directly related to the presence of a certain microorganism (**Calenoff E. etal, ١٩٩٣ and Stierna PLE, ١٩٩٦)**.

Recently, it was shown that the presence of IgE antibodies to Staphylococcus aureus enterotoxins (SAEs) was related to the severity of eosinophilic inflammation in nasal polyp tissue. In a study, to determine, whether aspirin sensitivity was related to an immune response to SAEs, and how both criteria would be related to eosinophilic inflammation, however, a direct impact of S. aureus could not be established. It seems that aspirin sensitivity and immune reactions to SAEs are independently related to eosinophilic inflammation. **(Perez-Novo etal, ٢٠٠٤).**

Viral infections also have been mentioned as a possible cause of nasal polyps. However, a viral etiology has not been shown, despite investigation for adenovirus, Epstein-Barr virus, herpes simplex virus, and human papilloma virus. In a search for human papilloma virus, using polymerase chain reaction, the virus also did not detected in nasal polyps **(Weille FL. and Gohd RS. ١٩٥٧).**

Fungal infection in allergic fungal sinusitis, the syndrome of nasal polyposis combined with positive Aspergillus cultures from the paranasal sinuses was recognized by Safirstein in ١٩٧٦. Further clinical reports supported this finding and noted its similarities with allergic bronchopulrnonary aspergillosis **(Ponikau JU etal, ١٩٩٩).**

After the recognition that species other than Aspergillus were associated with the disease the term 'allergic fungal sinusitis; was introduced. Diagnostic criteria for allergic fungal sinusitis have been proposed. The diagnosis relies on the confirmation of polypoid sinus disease, the identification of 'allergic mucin' with fungal elements and the absence of invasive fungal disease. Patients are typically

immunocompetent young adults with a mean age of presentation in the third decade (**Marple BF. ٢٠٠١**).

The initiating event is thought to be an immunomodulated reaction to fungal antigens, although this is not a universal finding. Evidence of atopy and/or hypersensitivity to fungi is common, with elevated serum total IgE levels present in between ٦٩ and ٧٥ per cent, serum fungal specific IgE present in between ٦٦ and ١٠٠ per cent and positive skin prick tests to fungal specific IgE present in between ٥٨ and ١٠٠ per cent (**Cody DT. Etal ١٩٩٤).**

The exact nature of the immune reaction is unclear but it is thought that it extends beyond a simple type I hypersensitivity reaction. The presence of fungal specific IgG would indicate that a type III reaction could be involved. This may result in a cycle of mucosal oedema, and further presentation of the antigen in sensitized individuals leading to chronic rhinosinusitis and nasal polyposis (**Feger TA, etal ١٩٩٧**).

Recently the role for fungi in the pathogenesis of chronic rhinosinusitis has been questioned. However the ever-present nature of fungal spores in the paranasal sinuses of subjects with, and without, sinus disease makes proving causation difficult **(Mabry RL and Manning S, ١٩٩٥)**.

Asero and Bottazzi also found ٤٠ per cent of a series of polyp sufferers showed skin reaction to *Candida albicans,* a common commensal of the upper respiratory tract **(Park et al. ١٩٩٧)**.

As regards **allergy and atopy**, traditionally, allergy has been assumed to be the underlying cause of nasal polyps. Indeed polyps removed at surgery continue to be labelled as allergic type polyps' by histopathologists. The presence of history of allergy, eosinophilia, mast cell degranulation, and high levels of IgE suggest an allergic basis for nasal polyps. This has been challenged by a number of clinically based studies which found objective measures of atopy (i.e. skin prick testing) to be no more common in polyp patients than the general population. Nasal polyps are also no more common in atopic individuals. However polyps are observed to occur in patients with adult-onset asthma, where allergy does not feature, rather than childhood asthma where concurrent allergic conditions such as rhinitis and eczema are common (**Drake-Lee A. ١٩٨٤).**

Recently a possible role for allergy in the pathogenesis of nasal polyps has again been suggested. Park *et al.* found allergen-induced *in vitro* release of granulocyte-macrophage - colony stimulating factor (GM-CSF) and IL-٨ from polyp tissue in atopic individuals and this was associated with increased eosinophil survival. These authors suggested that therefore an allergic response may contribute to polyp eosinophilia. Although they did not include a control group, Asero and Bottazzi also found ٤٠ per cent of a series of polyp sufferers showed skin reaction to *Candida albicans,* a common commensal of the upper respiratory tract. Also, bacterial specific IgE has been detected in patients with nasal polyposis but not in patients with allergic rhinitis (**Bachert C. etal ١٩٩٧).**

In a study, nasal polyps were reported in ٢٥.٦% of patients with allergy compared to ٣.٩% of a control group without allergy. The

prevalence of allergy in patients with nasal polyps varies from ١٠% to ٥٤% and ٦٤%. There is a coincidence of asthma and nasal polyps, but the association between polyps and atopic allergy is not clear. Patients with nasal polyps have the same prevalence of positive skin prick tests to a series of allergens as the normal population. It was believed that most clinical evidence is against allergy as a causal factor. The mechanisms involved in eosinophilic infiltration of the nasal mucosa and the nasal polyp are not fully elucidated. Studies on tissue cultures of epithelial cells from nasal polyps and normal nasal mucosa, generating human epithelial cell-conditioned media, suggest that eosinophil infiltration into the nasal mucosa during allergic reaction and nasal polyposis may be modulated, at least in part, by epithelial cells. Epithelial cells from nasal polyps might have a more potent effect on inducing eosinophilic infiltration of the respiratory mucosa than epithelial cells from healthy nasal mucosa. Other studies showed that eosinophil migration into tissues is controlled, in part, by interactions between eosinophil adhesion receptors and counterstructures on the vascular epithelium. This p-selectin–dependent adhesion is important (**Jamal A and Maran AGD, ١٩٨٧)**.

A possible role for **Food allergy Food allergy** in the pathogenesis of nasal polyps has also been suggested although the evidence for this is limited. Pang *et* al. reported positive intradermal tests relating to food allergy in ٨١ per cent of polyp patients versus ١١ per cent in controls and in a separate study ٣١ per cent patients with polyps gave a history of food or drug allergy. In this study patients with aspirin sensitivity were included, and the diagnosis of food sensitivity was made solely on the basis of the clinical history **(Pang YT, et al. ٢٠٠٠)**.

About **local allergy**, The high level of IgE in polyp tissue, even in the absence of other markers of atopy (such as skin prick testing), may indicate local production. Recent work in idiopathic rhinitis has implicated local IgE production as a possible aetiologic factor **(Donovan R, et al. ١٩٧٠)**.

In vitro production of IgE from nasal polyp tissue in both atopic and non-atopic subjects, has been identified. Bachert et al. demonstrated that total and specific IgE in polyp tissue was unrelated to positive skin-prick tests but correlated with the degree of eosinophilia. It remains possible therefore, that local allergic mechanisms could play a role in the pathogenesis of polyps. This would be compatible with both the characteristic histological appearances and the previous clinical studies showing no link between polyps and generalized atopy **(Bachert C, etal ٢٠٠١)**.

Allergy versus infection

Prospective descriptive study to evaluate the relationship of nasal polyps to allergy, sinonasal infection and histopathological type. There was no statistically significant difference in prevalence of polyposis with eosinophil- and neutrophil-dominated inflammations. Or there was no statistical significant difference between allergy and infection. This may reflect some sort of interplay or interaction between them, in the form of causal relationship to each other. Allergy may be to bacterial allegins, or allegy may encourage local infection (**Kirtsreesakul V, ٢٠٠٤).**

Although there is some evidence for a **Genetic predisposition** to nasal polyps, the current state of knowledge in this area is limited. In

١٩٩٢ Drake-Lee reported nasal polyposis in monozygotic twins and high rates of nasal polyposis (٥٢.٦ per cent) and asthma (٤٣.٦ per cent) have been reported in the family history of patients presenting with nasal polyps **(Drake-Lee A. ١٩٩٢ and Rugina M etal, ٢٠٠٢)**.

An association between HLA-A١/B٨ has been described with severe polyposis and asthma and between HLA-A٧٤ and nasal polyposis. In a case-controlled study Molnar-Gabor *et al*. showed an increased susceptibility for developing nasal polyposis in association with the HLA-DR٧-DQAI*٠٢٠١ and -DQBI*٠٢٠٢ haplotype **(Luxenburger W. etal. ٢٠٠٠)**.

The intriguing possibility that mutations of the cystic fibrosis transmembrane regulator (CFTR) gene in phenotypically non- cystic fibrosis individuals (individuals with no other evidence of the disease) are responsible for nasal polyps has been examined but no association has been found (**Irving RM. etal ١٩٩٧)**.

Other causes, like autonomic dysfunction, ciliary abnormalities, as found in primary ciliary dyskinesia (Kartagener's syndrome) can be associated with polyps as can the hyperviscous mucus in Young's syndrome (**Settipane GA etal, ١٩٩٧)**.

B- Structure:

Anatomically, the majority of nasal polyps enter the nasal cavity from the region of the middle meatus, although in conditions such as aspirin-sensitive asthma they may be widespread throughout the nasal cavity. It has been observed that polyps form at points of mucosa to

mucosa contact and one proposed explanation for this is that mucosal contact stimulation leads to the release of pro-inflammatory cytokines from the epithelium. Other authors have observed that anatomical abnormalities causing mucosal contact occur as commonly in patients without rhinosinusitis as those with the disease. In addition, the increased use of the nasendoscope, which frequently reveals contact areas within the nasal cavity in the absence of polyps, would suggest that mucosal contact is not the key to polyp formation. The reason why polyps are most commonly localized to such a small area of the upper airway remains a mystery **(Larsen PL and Tos M. ١٩٩٦)**.

Autopsy Evaluation of the origin of nasal polyps was done in several cadaveric studies. In one study, nasal polyps were noted in ٢% of specimens depending on conventional anterior rhinoscopy alone. The nasoethmoidal complexes were then removed transcranially in these six specimens, and polyps were documented photographically. In other study, the nasoethmoidal complex was removed transcranially first, and examined directly; anterior rhinoscopy was not performed. Polyps were found in ٢٦%. In more recent study, specimens were evaluated with endoscopic dissection. Polyps were noted in ٤٢% specimens. So with more tedious search for polyps their insidence increases. Important characteristics of nasal polyps were noted in these studies, Polyps were found mainly in the transition spaces between the nose and sinuses. ٧٥% of polyps were related to ethmoidal recesses and clefts. Most of the polyps were unilateral (٦٣%), and bilateral nasal polyps were found in ٣٧% of the cadavers. The high frequency of nasal polyps seems to show that nasal polyps often are small and do not always reach a size that would produce symptoms. **(Larsen PL, and Tos M, ١٩٩٤,)**

It is also possible that most small polyps may remain small or regress spontaneously as has been shown in the modified respiratory epithelium of the middle ear. Although too small for statistical calculations, the results suggest that nasal polyps start small and solitary in the narrow channels of the osteomeatal complex. It was postulated that nasal polyposis can be the result of a vicious cycle starting with infection or another etiologic factor, which leads to the formation of a single polyp. This polyp can cause reduced ventilation in the meatus and of the ethmoidal cells, thereby creating a milieu in which other polyps can form, causing further blockage of the sinus ostia, recesses, and clefts. This may lead to sinus infection and subsequent polyp formation within the ethmoidal cells **(Larsen PL, and Tos M, ١٩٩٥)**

Stammberger evaluated nasal polyps in ٢٠٠ consecutive patients undergoing functional endoscopic sinus surgery. He noted that ٨٠% of polyps originated from the middle meatal mucosa, uncinate process, and infundibulum. In ٦٥%, polyps originated from the ethmoidal bulla and hiatus semilunaris and from the frontal recess in ٤٨%. Polyps were found inside the ethmoidal bulla in ٣٠% (**Stammberger H. ١٩٩١)**.

Nasal polyps vary in size, depending on growth rate, polyp age, degree of inflammation and edema, and several other factors. The polyp body or apex is connected by a stalk of various thickness and length to the base of the polyp, its site of origin on the nasal mucosa. Polyp shape varies and depends mainly on the anatomy of the meatus, the space available for its extension, and the number and size of neighboring polyps. A study was undertaken for further view about the natural history, their anatomical site of origin, etiology, and pathogenesis of nasal polyps.