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REVIEW ARTICLE

Remodeling in Chronic Sinusitis and Nasal Polyps

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ABSTRACT

Remodeling in chronic sinusitis and nasal polyps is discussed. In chronic sinusitis, epithelial shedding, which is characteristic of asthma, is not observed in the maxillary sinus. An increase of mirovillous cells, squamous metaplasia, and goblet cells is observed in many patients with chronic sinusitis. The decreased ciliary area increases postoperatively in the maxillary ostium and in the maxillary sinus. There is no significant difference in the number of goblet cells between normal controls and chronic sinusitis. On the other hand, the number of submucosal acinar cells in chronic sinusitis is significantly higher than that in normal controls. Nasal polyps show a diversity of histogical findings. Although squamous metaplasia and goblet cells hypertrophy is observed in many patients, epithelial shedding, which is characteristic of asthma, is not observed in nasal polyps. The most striking finding of glands in nasal polyps is long shape. Histochemical analysis reveals deposition of types I, III, and V collagens in nasal polyps. Myofibroblasts, which are abundant in nasal polyps but rare in nasal mucosa, could be involved in the growth process of nasal polyps by inducing extracellular matrix accumulation. Although accumulation of extracellular matrix is a main feature of nasal polyps, its pathogenesis is not clearly known.

KEY WORDS

airway remodeling, asthma, chronic sinusitis, extracellular matrix, nasal polyps

INTRODUCTION

Not much attention has been paid to the remodeling process of chronic sinusitis and nasal polyps. Here we will discuss remodeling of chronic sinusitis and nasal polyps. Historically, sinusitis was divided into three categories based on disease duration, with chronic sinusitis referring to patients whose symptoms were of greater than 6 weeks' duration.[1] At the time this terminology was developed, all sinusitis was thought to be infectious. It is now clear, however, that the majority of patients with chronic sinusitis do not have an infectious disorder, and this has led to the need to develop more appropriate terminology to describe the myriad of conditions that make up chronic sinusitis. There are 4 major pathophysiologic processes responsible for chronic sinusitis.[1] Only a small subset of patients in fact have *chronic infectious sinusitis*. These patients typically have underlying humoral immune deficiencies, HIV, Kartagener syndrome, and cystic fibrosis. In contrast, most patients with chronic sinusitis have an inflammatory disorder with prominent hyperplasia of immune cells. *Chronic inflammatory sinusitis* is thought to result from chronic or recurrent occlusion of the sinus ostia caused by viral rhinitis, allergic rhinitis, anatomic predisposition, or other causes. These processes lead to recurrent acute bacterial infections, possibly in association with barotrauma of the sinus cavities and damage to the respiratory epithelium, ciliary destruction, mucous gland and goblet cell hyperplasia, bacterial colonization, and, ultimately, chronic inflammatory changes. Eosinophils are not a feature of chronic inflammatory sinusitis, although this disorder might produce nasal polyps. Chronic inflammatory sinusitis is generally responsive to surgical interventions. In contrast to chronic inflammatory sinusitis, the other immune inflammatory disease is characterized by prominent expression of eosinophils and perhaps should be referred to as *chronic hyperplastic eosinophilic sinusitis*. This disease is frequently associated not only with nasal polyps but also with asthma, atopy, aspirin sensi-

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tivity, and, as described in our article, the overproduction of cysteinyl leukotrienes. Chronic hyperplastic eosinophilic sinusitis represents the majority of patients seen with chronic sinusitis, and in contrast to chronic inflammatory sinusitis, it does not respond well to surgery. The final condition associated with chronic sinusitis is *allergic fungal sinusitis.* This presumably represents a severe variant of chronic hyperplastic eosinophilic sinusitis associated with the colonization of fungi within the sinus cavities and the presence of an IgE- and Th2-like lymphocyte-mediated allergic inflammatory response.[1]

CHRONIC SINUSITIS

EPITHELIAL ABNORMALITIES

The normal nasal and paranasal epithelium is a stratified structure consisting of a columnar layer comprising ciliated and secretory cells supported by basal cells. In the normal maxillary sinus, more than 90% of the mucosal surface area is covered with cilia. In order to qantitate epithelial abnormalities in chronic sinusitis, the maxillary mucosa of both the superolaterla wall and the ostium were sampled during endonasal sinus surgery. Ciliary surface was determined using scanning electron microscopy and was expressed in terms of ciliary area, which is the percentage of mucosal surface occupied by cilia. The mean ciliary area at the time of surgery was 61% and 40% in the superolateral wall of the maxillary sinus and the ostium of the maxillary sinus, respectively.[2] Although the increase of mirovillous cells, squamous metaplasia, and goblet cells was observed in many patients, epithelial shedding, which is characteristic of asthma,[3] was not observed in the maxillary sinus. In the inferior turbinate mucosa, epithelial shedding was not observed in chronic sinusitis. After 7.6 months postoperatively, samples were taken from the same sites and examined using scanning electron microscopy. The mean postoperative ciliary area value was 74% in the superolateral wall and 51% in the ostium.[2] These postoperative values were significantly higher than the preoperative values. These result suggests that loss of cilia in chronic sinusitis is a reversible phenomenon. However, those with low ciliary area at the time of surgery did not recover to the same extent as those with higher ciliary area.

Unlike allergic rhinitis, nasal mucosal sensitivity to histamine is not enhanced in chronic sinusitis. We instilled a histamine solution with serial dilution on the inferior nasal mucosa until sneezing occurred. There was no significant difference in the histamine threshold between chronic sinusitis patients and normal control subjects. Thus epithelial abnormalities in chronic sinusitis are not severe enough to cause enhanced mucosal reactions to environmental stimuli as in the case of asthma.

Recently, Ponikau *et al.*[4] reported that epithelial damage (shedding) was observed in all specimens from twenty-two randomly selected patients with refractory chronic rhinosinusitis undergoing endoscopic sinus surgery. The discrepancy between the results in this study and our observations may come from differences in subtype classification of sinusitis. Since 68% of their subjects had been previously diagnosed with asthma, most of their patients had diagnoses of chronic hyperplastic eosinophilic sinusitis according to the classification explained in the introduction. On the other hand, the majority of our subjects had chronic inflammatory sinusitis.

MUCOSAL THICKENING AND SUBEPITHELIAL FIBROSIS

Electron microscopy of asthmatic basement membrane demonstrated that the true basement membrane, the lamina rara and densa, is normal in these tissues. The "thickening" is the result of a dense fibrotic response that occurs primarily in the lamina reticularis.[5,6] This response is characterized by the enhanced accumulation of fibronectin and types I, III, and V collagens. In chronic sinusitis, mucosa in the maxillary sinus is thickened.[7] Sobol *et al.* [8] examined submucosal collagen deposition using van Gieson stain. The mean grade of subepithelial collagen deposition was significantly higher in adult patients with chronic sinusitis and pediatric patients compared with control subjects. Although the presence of subepithelial fibrosis has been associated with disease severity and correlated with a decline in FEV_1 in asthma,[9] significance of subepithelial collagen deposition in chronic sinusitis is not known.

GLAND CELL METAPLASIA

Mucus hypersecretion is a well-documented feature of chronic sinusitis. In humans, subepithelial glands are the major source of airway mucus. In order to determine whether goblet cells or submucosal gland cells are important in chronic sinusitis, Majima *et al.*[10] histochemicaly quantitated the number of goblet cells and submucosal acinar cells in the nasal mucosa from 65 patients with chronic sinusitis and 18 normal control subjects. Results showed no significant difference in the number of goblet cells between normal controls and chronic sinusitis. On the other hand, the number of submucosal acinar cells in chronic sinusitis was significantly higher than that in normal controls ($p<0.01$). The area occupied by the acini in lamina propria was also increased in chronic sinusitis ($p<0.001$). This was also true in the case of maxillary sinus. Thus, hyperplasia and hypertrophy of nasal acinar cells may have an important role in mucus hypersecretion in chronic sinusitis.

In order to more clearly determine the nature of the mechanism of submucosal gland hyperplasia and hypertrophy, Guo *et al.*[11] established a serum-free three dimensional culture system for human nasal gland cells and Kimura *et al.*[12] examined the effects

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