

The Role of Atopy in Nasal Polyposis

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Patients with sino-nasal complaints presenting to an allergist's or otolaryngologist's practice frequently are diagnosed with conditions including allergic rhinitis (AR) and chronic rhinosinusitis (CRS). Furthermore, patients with CRS are phenotypically classified as CRS with and without nasal polyposis (CRSwNP and CRSsNP, respectively). There is an intuitive sense that atopy is an underlying etiologic factor in the development of CRS, particularly CRSwNP. Certainly, many patients with CRS will have atopy. AR is observed in approximately 50% of CRS patients [1] versus 15-20% in the population at large [2]. However, this clinical association does not prove causality and may reflect a selection bias by which patients with symptoms of CRS, presenting to an allergist's or otolaryngologist's practice are more likely to undergo allergy testing. Additionally, histopathological specimens of surgically resected sino-nasal polyps often yield a diagnosis of "allergic polyp" secondary to the observation of eosinophils in the tissue. The preponderance of recent evidence, however, suggests that despite the clinical comorbidity of these conditions, AR and CRSwNP are distinct entities with overlapping presentation, rather than a continuum of disease.

There are published reports illustrating that patients with AR do have increase burden of sino-nasal inflammation by computed tomographic criteria [3,4]. However, in these studies, patients were not carefully stratified by polyp status. Others have observed an increased prevalence of dust mite allergy in CRSwNP patients [5], but an expanding body of more recent evidence calls a potential etiologic association into question. Data from the Northwestern Allergy & Sinus Center [1] revealed a prevalence of atopy, as defined by positive skin prick to any of 27 tested antigens, of 49% in a sample of 106 CRS patients. When examining patients by NP status, CRSwNP was similar between atopic (38%) and non-atopic (37%) patients. In that study, the most significant predictor of the presence of polyps was comorbid asthma, but this observation was independent of atopic status. In fact, polyps were significantly ($p < 0.01$) more common in non-allergic asthmatics (13%) versus allergic (5%) asthmatics. These findings are supported by another study of 193 CRS patients [6], where the rate of atopy was 32% and 28% for CRSsNP and CRSwNP, respectively. Recent work has focused upon local up-regulation of IgE to specific allergens including *Staphylococcus aureus* enterotoxins, as well as deficiencies in the protective epithelial barrier [7]. Defects in genes associated with integrity of the mucosal barrier may be associated with microbial colonization and a dysfunctional or deregulated host response to microorganisms as well as other immunologic stimuli.

The observation of AR in a sizable subset of CRS patients compared

to the general population may reflect a selection bias of those subjected to allergy testing, but may also signal an etiologic contribution. However, the presently available body of data does not establish a causal relationship between IgE-mediated disease and the development of CRS. Furthermore, despite the common observation of tissue eosinophilia in nasal polyp tissue, atopy may be even less frequent in CRSwNP than is observed in CRSsNP. Recent investigations have suggested multiple other immunologic mechanisms by which eosinophils can be recruited and activated. These observations indicate, that although AR may contribute to the clinical symptoms suffered by CRS patients, its mechanistic role remains unclear and is far from definite. Thus, terms like "allergic polyp" should be avoided and regarded as misnomer.

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