The Role of Allergy in Chronic Rhinosinusitis

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KEYWORDS

• Chronic rhinosinusitis • Chronic rhinosinusitis with nasal polyps
• Chronic rhinosinusitis without nasal polyps • Allergic fungal rhinosinusitis • Allergies
• Allergic rhinitis

KEY POINTS

- Allergy and CRS often co-occur but a direct link establishing causality has not been identified.
- There is far more overlap in the pathophysiology of allergy and chronic rhinosinusitis with nasal polyps and allergic fungal rhinosinusitis than there is between allergy and chronic rhinosinusitis without nasal polyps.
- Seasonal allergies do not appear to contribute to rhinosinusitis in any form; however, perennial allergies may.
- Weak evidence exists for the use of immunotherapy in CRS with significant limitations of current available studies.

INTRODUCTION

Chronic rhinosinusitis (CRS), both with and without polyps, has an estimated prevalence of 4.9% ± 0.2% (or 490 in 10,000 people) in the United States.[1,2] The prevalence of allergic rhinitis (AR) in the general population is between 10% and 30%.[3,4] Some studies have shown that patients with sinusitis have a higher incidence of positive allergy skin prick tests (SPTs) than the general population,[5–7] other studies do not support this.[8,9] Likely, the 2 disease processes often coexist, but the data supporting the role or association of allergy and CRS are mixed.[10] This is not surprising because the definitions of both allergy and CRS can vary widely between studies, introducing an inherently heterogeneous study population over the whole of the literature. Furthermore, there is some evidence that the type of allergy (seasonal vs perennial) also needs to be taken into consideration because studies have shown that seasonal allergies do not seem to predispose a person to sinusitis but perennial allergies might.[7,11,12]

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CRS is likely a multifactorial disease with environmental, immunologic, microbacterial, genetic, and, possibly, allergic factors at play. Over time, a complex picture of the molecular and cellular mechanisms underlying CRS has begun to emerge and has led to the conceptualization of CRS endotypes. Endotypes represent subtypes of CRS defined by unique pathophysiologic mechanisms and biomarkers.[13] Until these endotypes have been better characterized and understood, the recognition of specific clinical CRS phenotypes and consideration of them as having different underlying pathophysiologies are of the utmost importance. As such, this review considers the role of allergy in the following disease states: CRS without nasal polyps (CRSsNP), CRS with nasal polyps (CRSwNP), and allergic fungal rhinosinusitis (AFRS).

CHRONIC RHINOSINUSITIS WITHOUT NASAL POLYPS

Diagnostic criteria[14] for CRSsNP include the presence of 2 or more of the following symptoms for at least 12 weeks:

- Nasal drainage (anterior rhinorrhea or postnasal drip)
- Nasal obstruction/congestion
- Hyposmia or anosmia
- Facial pain/pressure

And in addition

- Lack of nasal polyps

And either

- Evidence of paranasal sinus inflammation on CT
- Evidence of purulence coming from the sinuses or ostiomeatal complex on endoscopy

T cells are thought to be a crucial driver for the inflammatory cascade observed in most CRSsNP cases, which is generally marked by elevated levels of tumor necrosis factor (TNF)-α, interleukin (IL)-1β, IL-5, and IL-8.[15] As discussed previously, the exact underlying cause of CRSsNP is not completely understood and is likely multifactorial. Unfortunately, no controlled studies on the role of allergy in the pathophysiology of CRSsNP have been performed; therefore, only associations, such as the following, can be made.

A proposed mechanism by which allergy leads to the development of sinusitis is allergy-induced mucosal inflammation leading to ostial obstruction. Ostial obstruction in turn may promote an environment for bacterial overgrowth and/or perpetuation of inflammation. Certainly, in the nasal passages, IgE-mediated degranulation of mast cells leads to mucosal edema. Whether this same process occurs in the paranasal sinuses is less clear. In 1 study, Baroody and colleagues[16] performed a nasal allergen challenge in patients confirmed allergic on SPT followed by nasal and maxillary sinus lavage. After nasal challenge, they found a significant increase in the levels of histamine, albumin, and number of eosinophils in maxillary sinus lavage. These levels were smaller in magnitude than nasal passage levels, but this study did demonstrate a parallel inflammatory response within the maxillary sinuses.[16] Another study biopsied the inferior turbinate of patients confirmed to be allergic on SPT who were prone to sinusitis (defined as at least 2 sinus infections per year confirmed on maxillary puncture) to controls. Biopsies were taken in each patient during an acute viral upper respiratory infection and later during convalescence. The allergic patients prone to sinusitis were found to have significantly elevated T cells and lower levels of mast cells

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