Systemic and Odontogenic Etiologies in Chronic Rhinosinusitis

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KEYWORDS

- Chronic rhinosinusitis • Cystic fibrosis
- Eosinophilic granulomatosis with polyangiitis • Granulomatosis with polyangiitis
- Sarcoidosis • Primary ciliary dyskinesia • Odontogenic sinusitis

KEY POINTS

- Cystic fibrosis and primary ciliary dyskinesia result in pansinusitis and inspissated, thick mucopurulent secretions. Surgical therapy requires extended procedures to achieve antrostomies or ventilation and to treat obstructive polyposis.
- Patients with sinonasal sarcoidosis tend to present with nasal crusting, anosmia, and epistaxis. The nasal mucosa of the septum and turbinates may demonstrate nodules.
- Vasculitides present with lower respiratory tract disease. Medical therapy is the mainstay of therapy, with surgery indicated for severe symptoms, recurrent infections, and anatomic obstruction.
- Odontogenic sinusitis should be suspected in any patient with unilateral maxillary sinusitis with a longstanding history of maxillary dental problems or a recent history of a maxillary dental procedure.
- Otolaryngologists should personally review imaging studies to look for any bony dehiscence or anomalous connections between the maxillary sinus and the oral cavity.

INTRODUCTION

Although the majority of cases are largely attributed to local factors (ie, anatomic, allergic, immunologic, infectious), chronic rhinosinusitis (CRS) may also be caused by systemic or odontogenic disease. It is thus important for otolaryngologists to be aware of systemic and odontogenic etiologies of CRS, and to approach the management of these patients from a multidisciplinary standpoint. A thorough history, with

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special attention to comorbidities and medical conditions, should be elicited from each patient. Because all of these conditions have manifestations in multiple organ systems, consultations with other specialists should be sought in each case. In this review, we discuss the most common systemic diseases with distinguishing sinonasal manifestations, including cystic fibrosis (CF), sarcoidosis, vasculitides, primary ciliary dyskinesia (PCD), and odontogenic sinusitis.

SYSTEMIC ETIOLOGIES OF CHRONIC RHINOSINUSITIS

Cystic Fibrosis

CF is a relatively common autosomal-recessive genetic disorder involving a derangement in the gene encoding the CF transmembrane conductance regulator (*CFTR*). The most common mutation is ΔF508, or deletion of the phenylalanine codon at position 508, of which 1 out of 25 to 30 Caucasians are carriers.[1] This mutation results in restricted efflux of anions, such as chloride and bicarbonate, thus leading to thick, obstructive, inspissated sinobronchopulmonary secretions, further promoting mucosal inflammation and superinfections.[2] The pancreas and reproductive systems are also affected, frequently leading to exocrine insufficiency and male infertility, respectively. More recent studies have classified CF patients as either high or low risk based on *CFTR* genotype, with an apparent impact on prognosis.[3–7] Specifically, high-risk CF patients have an earlier age of diagnosis, worse pulmonary status (ie, lower forced expiratory volume at 1 second), increased colonization by *Pseudomonas aeruginosa*, increased sweat chloride, and worse overall survival.[8,9]

Patients tend to present with clinical manifestations of CF at a young age. A history of recurrent sinonasal and bronchopulmonary infections in a child should prompt consideration of a workup for CF. Similarly, the finding of nasal polyposis in a child is suggestive of unrecognized CF, occurring in one-third to one-half of patients.[10–12] On computed tomography (CT) imaging of the sinuses, CF patients tend to have hypoplasia of all sinuses (**Fig. 1**).[13,14] In a study by Ferril and colleagues,[9] comparison of high- and low-risk CF patients additionally found that high risk-patients are even more likely to have sinus hypoplasia. CF patients also tend to develop mucoceles.[15] Throughout their lives, CF patients require close care by pulmonologists, gastroenterologists, geneticists, otolaryngologists, respiratory therapists, and nutritionists to optimize their medical condition and preserve functional status.

Newborn screening for CF using serum immunoreactive trypsinogen and genetic testing for *CFTR* mutations is now nearly universal in the United States.[16,17] A diagnosis of CF can be made by (1) clinical signs and symptoms indicative of the diagnosis and (2) one of the following: (a) abnormal sweat chloride test on 2 separate occasions,

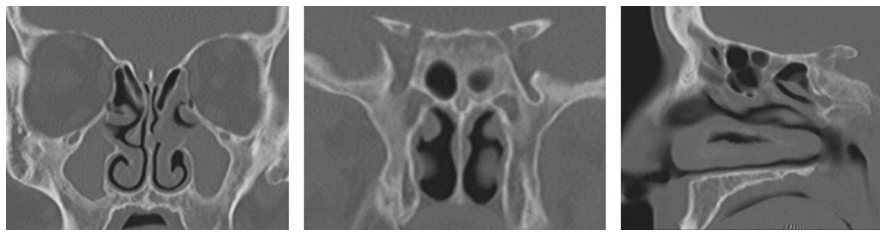

Fig. 1. Computed tomography image of the paranasal sinuses in a patient with cystic fibrosis showing maxillary (*left*), sphenoid (*center*), and frontal (*right*) sinus hypoplasia. Also note the significant bony thickening and osteitic changes surrounding the borders of each sinus, suggesting chronic inflammation.