

Brief communications

Fusarium allergic fungal sinusitis

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Allergic fungal sinusitis (AFS) was first reported in 1981 when Millar et al.¹ described five patients with chronic sinusitis caused by *Aspergillus fumigatus*. Since that original description, multiple small series and case reports have identified *Bipolaris*, *Curvularia*, *Alternaria*, *Rhizomucor*, *Exserohilum*, and *Chrysosporium* species as culpable pathogens in AFS.²⁻⁷ Patients with AFS are immunocompetent with positive epicutaneous skin test results and serum precipitins to the relevant mold. They have high serum IgE levels, both total and mold-specific, and usually have nasal polypoidosis and varying degrees of asthma. In more advanced cases, they can have signs and symptoms caused by sinus cavity enlargement and erosions including orbital proptosis, diplopia, and epiphora. Surgical specimens show a characteristic thick mucin that contains large numbers of eosinophils and Charcot-Leyden crystals. Fungal hyphae are sometimes found in the mucin, and growth of a mold species to which the patient has positive immediate skin test sensitivity helps confirm the diagnosis. A new AFS pathogen is identified in the case presented below.

CASE REPORT

A 12-year-old boy presented to our allergy clinic with a 2- to 3-year history of perennial nasal congestion while living in southern Alabama. Sinusitis and nasal polyps had been recently diagnosed by referring physicians. Systemic and nasal topical steroids decreased the size of his polyps, but standard antibiotic therapy did not clear his sinus disease. The patient had no previously recognized history of sinusitis, pneumonia, or other major infections. No atopic disorders had previously been identified, and growth and development

Abbreviation used

AFS: Allergic fungal sinusitis

were normal. He had experienced "bronchitis" with β -agonist-responsive cough on three prior occasions. Family medical history was notable for a paternal aunt and cousin with asthma. Results of physical examination were normal except for a small left-sided nasal polyp. Results of spirometry, sweat chloride test, quantitative immunoglobulins, IgG subclasses, and complete blood count were all normal. Total IgE was markedly elevated at 4606.5 IU/ml. Total eosinophil count was 600 per cubic milliliter. Epicutaneous skin tests were 4+ to histamine control and to several molds: *Fusarium*, *Bipolaris*, *Curvularia*, *Helminthosporium*, *Alternaria*, and *Cladosporium* species. He had no reaction to all other aeroallergens tested. Serum precipitins to *Fusarium* were present with heavy bands on the Ouchterlony plates. Sinus computed tomographic scan demonstrated pansinusitis with "speckled" hyperattenuation in the soft tissue density-filled sphenoid sinuses (Fig. 1). Surgically obtained sinus specimens showed allergic mucin with eosinophils and Charcot-Leyden crystals. Potassium hydroxide preparation showed broad, septated hyphae. A *Fusarium* species grew in a heavy pure growth.

Approximately 7% of patients with chronic sinusitis requiring surgery have AFS. *Aspergillus* and *Bipolaris* species have been identified in the majority of AFS reports. Although *Aspergillus* is not, many of the identified AFS pathogens are dematiaceous (black) molds commonly found in the outdoor environment. This case represents the first known report of AFS caused by *Fusarium*, a nondematiaceous white mold species commonly found in the southern United States. It is a soil organism and plant pathogen. Allergic hypersensitivity to *Fusarium* species is well recognized.⁸ It has also caused nonatopic human disease, most commonly mycotic keratitis and onychomycosis.

The identification of yet another nondematiaceous mold suggests that many more mold species could potentially be found to be AFS pathogens if we search appropriately. The patient in this report presented with the typical clinical, radiographic, and laboratory characteristics of the patient with AFS, and surgical specimens confirmed the diagnosis. He has done well after surgical debridement and systemic steroid therapy.

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FIG. 1. Soft tissue density-filled sphenoid sinus with centrally located area of hyperattenuation.

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