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CODIAGNOSIS OF ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS AND SARCOID IN A SEVERE ASTHMATIC PATIENT

Allergic bronchopulmonary aspergillosis (ABPA) is a respiratory disease that can occur in patients with cystic fibrosis and asthma, resulting in increased morbidity.¹ ABPA is characterized by mucoid impaction and inflammation, which can lead to bronchiectasis, fibrosis, and progressive lung function decline.¹ The classic computed tomography findings of late-stage ABPA are central bronchiectasis with upper lobe predominance. Peripheral nodules and ground glass opacities can also be found earlier, but mediastinal lymphadenopathy is rare.

Sarcoid is a disease often described as the “great imitator.” Patients with sarcoidosis present with vague symptoms of fatigue, shortness of breath, cough, night sweats, and occasionally rashes.² On computed tomograms, sarcoid classically shows bilateral hilar adenopathy and reticular opacities, and diagnosis is made by the finding of noncaseating granulomas on biopsy.² Other common causes of mediastinal and hilar lymphadenopathy include infection, neoplasm, granulomatous disease, and reactive hyperplasia. We describe a case study of severe persistent asthma presenting with bronchiectasis and mediastinal and hilar lymphadenopathy with a diagnosis consistent with ABPA and sarcoidosis.

We present a case of a 38-year-old man with a 3-year history of allergic rhinitis, sinusitis, and severe persistent asthma. In the previous 18 months, he had received 2 courses of systemic corticosteroids and had been treated for pneumonia in the past. His medications included fluticasone propionate and salmeterol (250/50 μ g) and montelukast. He noted nocturnal symptoms occurring 2 to 3 times per week and use of albuterol inhaler 2 to 3 times per week. Family history was significant for a father and 2 brothers with severe sinus disease. He owned a landscaping business. The patient had a 5-pack-year smoking history, although he had quit 4 years ago. The results of the physical examination were unremarkable.

Pulmonary function testing showed obstruction with a forced expiratory volume in 1 second (FEV₁) of 2.54 L or 63% of predicted and a FEV₁/forced vital capacity (FVC) ratio of 57% of predicted. Diffusion capacity was normal. Allergy skin prick testing revealed positive reactions to cottonwood tree, ragweed, cat dander, and *Aspergillus fumigatus*. Laboratory testing demonstrated an elevated absolute peripheral blood eosinophil count (0.9 g/ μ L) and elevated serum IgE level (451 kU/L), which was drawn off systemic corticosteroids. A serum angiotensin-converting enzyme level was not obtained. The results of precipitating serum antibody tests were negative. High-resolution computed tomography demonstrated central bronchiectasis with mucoid impaction suggestive of ABPA and mediastinal lymphadenopathy with the largest lymph node measuring 1.3 $\times$ 2.1 cm (Figure 1A). The patient was diagnosed as having asthma complicated by ABPA. Treatment included an extended course of systemic corticosteroids (slowly tapered during 3 months) and increased inhaled corticosteroid dosing. Treatment with omalizumab was also initiated.³ However, because of the finding of mediastinal adenopathy, which is not common in ABPA, the patient was referred for a video-assisted thoracoscopy. The biopsy results showed noncaseating granulomas, consistent with the diagnosis of sarcoid (Figure 1B). Culture of lung tissue was negative for infection. The results of ophthalmologic examination and 24-hour urinalysis for calcium were normal. At 1-year follow-up, he reported improvement in symptoms with objective improvement in pulmonary function testing with an FVC of 5.31 L (108% predicted), an FEV₁ of 4.31 L (108% predicted), an FEV₁/FVC ratio of 0.75 with omalizumab, fluticasone propionate–salmeterol (500/50 μ g), montelukast, nasal irrigations, mometasone nasal spray, and cetirizine.

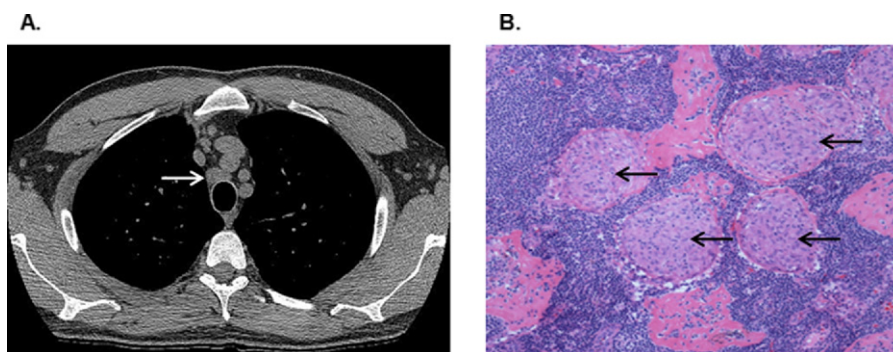


Figure 1. Mediastinal lymph node. A, High-resolution computed tomogram of mediastinal lymph node enlargement measuring 1.3 $\times$ 2.1 cm. B, Histologic analysis (hematoxylin-eosin) of mediastinal lymph node (fourth lymph node, right) shows nonnecrotizing (noncaseating) granulomatous inflammation composed of small, tight, “naked” granulomas (arrows), consistent with sarcoid.

The incidence of sarcoid is approximately 10 to 20 per 100,000 persons, and the incidence of ABPA is approximately 1% to 2% of asthma patients.² Thus, the overall incidence of these diseases is thought to be rare, and concomitant occurrence has not been previously reported, to the best of our knowledge. Although the cause of sarcoidosis remains to be established, it has been linked to infectious agents, such as *Mycobacterium tuberculosis*, propionibacterium, and human herpesvirus 8.^{4–6} Sarcoidosis has not been previously associated with fungal infections or colonization. There is a case study describing the onset of ABPA after treatment with infliximab in a patient with sarcoidosis.⁷ Our case is unique in that the patient presented with severe persistent asthma complicated by ABPA, but due to the unusual finding of mediastinal adenopathy, further evaluation revealed sarcoidosis. Of note, features of restriction or diminished diffusing capacity were not observed in the patient, which suggests either an early diagnosis of sarcoid or the predominance of his severe asthma. The finding of sarcoid is important because disease progression of eye and kidney involvement occurs in up to 20% to 80% of patients.^{4–8} Sarcoid affects many other organs compared with ABPA, which primarily affects the lungs. In conclusion, the finding of lymphadenopathy on chest imaging of patients with ABPA should prompt the physician to conduct further evaluation because mediastinal lymphadenopathy is not a classic finding of ABPA.

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