Case In Point: Recognizing allergic bronchopulmonary aspergillosis

April 01, 2006
By Truptesh H. Kothari, MD [1], Michael Boyars, MD [2], Dipti B. Kothari, MD [3], and Bernard Karnath, MD [4]

A 28-year-old man presented with chest pain, hemoptysis, and wheezing. He had a history of intermittent shortness of breath that occurred at least 3 times a year in the past 3 years; fever; and loss of appetite associated with headache, vomiting, and weakness. His medical history also included asthma, chronic gastritis, and more than 5 episodes of pneumonia since 1996. A test for hepatitis C virus (HCV) had yielded positive results.

THE CASE
A 28-year-old man presented with chest pain, hemoptysis, and wheezing. He had a history of intermittent shortness of breath that occurred at least 3 times a year in the past 3 years; fever; and loss of appetite associated with headache, vomiting, and weakness. His medical history also included asthma, chronic gastritis, and more than 5 episodes of pneumonia since 1996. A test for hepatitis C virus (HCV) had yielded positive results.

On physical examination, the patient was in acute distress because of shortness of breath. He was febrile, with a temperature of 38.5°C (101.3°F); pulse rate, 120 beats per minute; blood pressure, 180/110 mm Hg; and respiration rate, 30 breaths per minute. Decreased breath sounds in the right lower lobe were noted on auscultation, and a dull sound was heard on percussion. The initial workup included a complete blood cell count, basic metabolic panel, hepatitis panel, HIV testing, antineutrophil cytoplasmic antibody testing, and measurement of serum IgG and IgE antibody levels. The white blood cell count was 14,000/µL; erythrocyte sedimentation rate, 57 mm/h; serum IgE level, 17,259/µL; and HCV antibody titer, positive. Cultures of blood, urine, and bronchoalveolar lavage (BAL) fluid for fungi and bacteria were unremarkable, except for a few Gram-positive cocci on BAL fluid culture. The results of a tuberculin purified protein derivative (PPD) test were negative. A chest radiograph showed an irregular opacity in the right lower lobe (Figure 1), consistent with pneumonia, and circular opacities in both lungs, which were confirmed by a CT scan of the chest (Figure 2). A transbronchial biopsy specimen from the right lower lobe demonstrated eosinophilia and hemosiderin-laden macrophages (Figure 3).

DISCUSSION
ABPA was first described by Hinson and associates [1] in the United Kingdom in 1952. It is an allergic reaction to the fungus Aspergillus, characterized by inflammation of the bronchi or alveoli. Patients with ABPA commonly present with worsening symptoms of asthma, wheezing, cough that may produce brownish plugs or bloody sputum, shortness of breath, and fever. [2,3] The prevalence of ABPA is relatively high in patients with asthma and cystic fibrosis. [3,4] Data from the Epidemiologic Registry of Cystic Fibrosis (ERCF) indicate that the overall prevalence of ABPA in the ERCF population was 7.8% (range, 2.1% in Sweden to 13.6% in Belgium). The prevalence was low in persons younger than 6 years, but was almost constant—about 10%—thereafter. No differences based on sex were observed. ABPA affected 8% of the patients who had a DF508/DF508 genotype and 5% to 6% of those with DF508/G551D, DF508/G542X, and DF508/N1303K genotypes. [4] The presence of asthma and cystic fibrosis is a predisposing factor for ABPA. In this setting, Aspergillus resides in the patient's airways, where it appears to be an important antigen. Both type I and type III immune reactions to the organism develop in affected persons. [5] The pathogenesis of ABPA depends on impaired respiratory clearance and dense respiratory epithelial exposure to Aspergillus fumigatus spores, with subsequent chemotactic recruitment to lung tissue of CD4+ T helper 2 lymphocytes specific for A fumigatus. Susceptibility to ABPA appears to involve certain risk factors such as the presence of atopy and defined major histocompatibility complex-restricted alleles. Distinct cytoplasmic A fumigatus molecules (2, 4, and 6), available as recombinant allergen reagents, appear to be associated with ABPA. [6] The diagnosis of ABPA is based on clinical evaluation and invasive diagnostic tests. Diagnostic
criteria include asthma, a history of pulmonary infiltrates, peripheral blood eosinophilia, immediate-type skin reactivity, serum precipitating antibodies to Aspergillus-specific IgE and IgG, and central (proximal) bronchiectasis.

The differential diagnosis includes chronic eosinophilic pneumonia, bronchiolitis obliterans with organizing pneumonia (BOOP), Wegener granulomatosis, and eosinophilic granuloma. In patients with chronic eosinophilic pneumonia, the chest radiograph shows persistent patches mainly in the outer zones of the lungs. Microscopic examination of sputum or BAL fluid typically shows clumps of eosinophils. Patients with BOOP have CT or radiographic evidence of bilateral areas of consolidation and ground-glass opacities, usually with a peripheral location.6

Transbronchial biopsy can help differentiate these diseases. In the case described here, there was no evidence of granulomas, vasculitis, or viral inclusions. The presence of eosinophilia and hemosiderin-laden macrophages on a transbronchial biopsy specimen is consistent with a diagnosis of ABPA. The negative PPD test result ruled out tuberculosis.

CT can play an important role in the workup of ABPA.7-9 High-resolution CT has a sensitivity and specificity for bronchiectasis similar to that of bronchography; yet unlike bronchography, CT can be performed safely and quickly on most patients.8

The key points in the evaluation of the patient described here include the following:

- A history of asthma.
- Recurrent pneumonia since 1996.
- Pulmonary nodules on chest radiograph and CT scan.
- Peripheral eosinophilia and elevated IgE levels.

**Treatment**

Systemic corticosteroids remain the mainstay of treatment for ABPA. Itraconazole has an established role as a corticosteroid-sparing agent if the patient has a slow or poor response to corticosteroids, relapses, or is at risk for or has corticosteroid toxicity. Monitoring the patient's clinical, radiographic, and laboratory responses (especially total serum IgE level) is essential.3

The response to therapy for ABPA is usually good, although relapses requiring treatment are common. The best preventive measures include the avoidance of exposure to Aspergillus by staying away from areas with decaying vegetation or standing water; living in a dust-free house; and remaining in an air-filtered, air-conditioned environment whenever possible.

**References: REFERENCES**


**Source URL:**
http://www.psychiatrictimes.com/case-point-recognizing-allergic-bronchopulmonary-aspergillosis

**Links:**