

Allergic Broncho-Pulmonary Aspergillosis Presenting as Flitting Opacities on Chest Radiograph

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ABSTRACT

Allergic broncho-pulmonary aspergillosis (ABPA) is hypersensitivity reaction to *Aspergillus fumigatus* in the bronchial tree of young asthmatic patients. A 28 years old female patient presented with one year history of fever with generalised body aches and pains and had already received treatment for pulmonary tuberculosis. Her chest radiograph showed flitting opacities in both lung fields with a TLC of $13.2 \times 10^9/L$ having 25% eosinophils and ESR of 87 mm at first hour. Her serum IgE were markedly raised and CT scan of the chest showed dilated large and medium sized bronchi forming mucocoeles, finger in glove appearance and nodular shadowing in the lung parenchyma. Sputum also showed fungal hyphae by direct microscopy. All the findings were consistent with the diagnosis of ABPA, which responded to oral Itraconazole and Prednisolone. ABPA is a potentially destructive lung disease requiring high index of suspicion for an early diagnosis to prevent irreversible lung damage.

Key words: Allergic bronchopulmonary aspergillosis. *Aspergillus*. Eosinophilia. Flitting opacity. Lung. Chest X-ray.

INTRODUCTION

Allergic broncho-pulmonary aspergillosis (ABPA) is an immunologic pulmonary disorder caused by hypersensitivity to *Aspergillus fumigatus* colonizing the bronchial tree.¹ It occurs in young asthmatic patients for which radiological techniques with ancillary supportive laboratory investigations are required for diagnosis.² Although the disease has been a subject of recent highlights in medical literature but neither frequently diagnosed nor an early stage diagnosis is made leading to inappropriate therapy and irreversible lung damage.^{3,4} One such case is thereby reported.

CASE REPORT

A 28 years old female patient presented with one year history of intermittent low grade fever having evening spikes associated with generalised body aches and pains. She had been treated for pulmonary tuberculosis about 5 years back. She was mother of 5 children and belonged to lower socioeconomic strata. Her preliminary laboratory investigations showed haemoglobin (Hb) of 12.8 g/dL, total leucocyte count (TLC) of $10.9 \times 10^9/L$ and an erythrocyte sedimentation rate (ESR) of 87 mm at the end of first hour by Westergren's method. Her initial chest radiograph showed reticulonodular shadowing in both lung fields with patchy inhomogenous shadowing

with confluence of opacities (Figure 1a). Considering the possibility of a relapse of pulmonary tuberculosis her sputum for acid fast bacilli on 3 consecutive days and Mantoux test were advised which were negative. However, she was started with anti-tuberculosis therapy empirically after which her fever and complaints subsided. She was advised monthly follow-up with liver function tests and chest radiograph in the out-patient department.

Three months later she again complained of persistent fever with productive cough, difficulty in breathing and weight loss. On physical examination, she had bilateral rhonchi with crepitations in both lung fields. A subsequent chest radiograph showed bilateral haziness in both lung fields with blunting of the left costo-phrenic angle and consolidation in the right middle zone. There was reticulo-nodular shadowing with confluence in the right peri-hilar region and left upper zone with a differential diagnosis of interstitial lung disease or tuberculosis (Figure 1b). Her repeat complete blood counts showed TLC of $13.2 \times 10^9/L$ with 25% eosinophils and ESR of 87 mm at first hour. Her blood total eosinophil count was $3.1 \times 10^9/L$ (normal $0.06-0.6 \times 10^9/L$) while her autoimmune profile for ANA, anti-Ds DNA and ANCA was negative. ABPA was added to the list of differential diagnosis and subsequent serum Ig E levels were found markedly raised with a value of $> 800 IU/mL$ (normal $< 120 IU/mL$) and sputum showing septate fungal hyphae by direct microscopy using KOH method. A high resolution CT scan of the chest was done which showed scattered bronchiectasis predominantly involving large and medium sized bronchi forming mucocoeles. Finger in glove appearance was seen with nodular shadowing in the lung parenchyma and consolidation in

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Received July 30, 2009; accepted January 15, 2010.

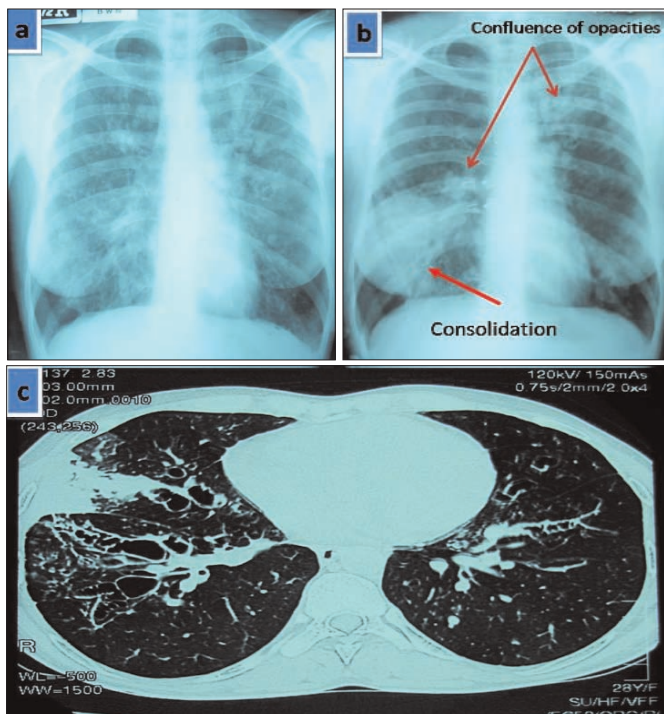


Figure 1: Photoradiographs showing reticulo-nodular shadowing in both lung fields with patchy inhomogeneous confluence of opacities (a) which after 3 months was followed by consolidation in the right middle zone with confluence of opacities in the right peri-hilar region and left upper zone (b). HRCT chest showing scattered bronchiectasis with mucocoeles, nodular shadowing in the lung parenchyma and consolidation in the right middle lobe (c).

the right middle lobe (Figure 1c). The findings in the light of clinic-pathological findings were consistent with the diagnosis of ABPA. She was started on tablet itraconazole 200 mg. once daily for 2 weeks along with bronchodilators and oral Prednisolone in a dose of 1 mg/kg body weight. She became asymptomatic and was subsequently continued on a maintenance dose of Prednisolone at 10 mg. per day.

DISCUSSION

Aspergillus is a genus of fungi with worldwide distribution found in decomposing organic matter, ceilings and walls with water salination.⁵ Pulmonary aspergillosis is caused by inhalation of 2-3 μ m spores which reach alveoli. However, certain pre-disposing factors and host characteristics are required for the spectrum of clinic-pathological syndromes caused by *Aspergillus* infection. Host immune status, hypersensitivity response or an underlying pre-existing chronic lung disease, i.e. chronic obstructive airway disease, tuberculosis or bronchiectasis are central to the pathogenesis of ABPA.⁶

The disease ABPA affects adults aged between 20-90 years with a male pre-disposition having male to female ratio of 3:1. It occurs in 1-2% of asthmatics and 10% of patients with cystic fibrosis accounting for 0.02% of hospital admissions.⁷ Epidemiological studies are difficult because the incubation period of the disease is long (4-14 days), making it difficult to distinguish between

nosocomial and community acquired infections. Also nasal cultures have low sensitivity and specificity. The clinical presentation is non-specific with low grade fever, wheezing productive cough, weight loss, malaise and chest pain with recurrent pneumonia as was also seen in this case.⁸

ABPA can not be diagnosed on a plain chest radiograph but fleeting alveolar subsegmental or lobar infiltrates, tram-line appearance of bronchial walls, V-or Y-shaped bronchial tubular opacities (finger-in-glove sign) can be seen. Features of lobar consolidation, atelectasis, cavitation, parenchymal scarring or fibrosis may also be present.⁹ On CT scan, bronchiectasis and peribronchial thickening remain the most common finding typically involving segmental and sub-segmental bronchi of predominantly upper lobes. High attenuating mucoid impaction is the most characteristic finding in 30% patients. Mucus plugging of the small airways with centrilobular nodularity and tree-in-bud sign is better appreciated on high resolution CT scans.¹⁰

The primary diagnostic criteria includes symptoms of asthma, blood eosinophilia, elevated serum IgE levels, positive skin tests and elevated serum IgE and IgG levels for *Aspergillus fumigatus* whereas the primary radiologic criteria include fixed or transitory pulmonary infiltrate and central bronchiectasis. Secondary criteria consists of fungal hyphae in sputum and expectoration of brown mucus plugs.⁷

Treatment is decided by the severity of symptoms and pulmonary function derangement with an aim to reduce inflammation and immunologic activity. Prednisolone remains the mainstay of therapy for control and stability of ABPA supported by anti-inflammatory and bronchodilator agents.³ Antifungal agents are used to eradicate and prevent the growth of fungal mycelia within the bronchial tree. The newer imidazoles such as itraconazole and fluconazole are preferred because of fewer side effects than ketoconazole and help in reduction in the dose of corticosteroids.⁷ Recently monoclonal anti-IgE (omalizumab) antibody has proved to be of greater benefit showing improvement in pulmonary symptoms and lung functions which were not achieved previously with antibiotics or prednisone alone.¹¹

The disease process can be staged using combined clinical and radiological data with stage I as acute presentation of 6-8 weeks while stage V is at the other end of the spectrum having irreversible lung damage resulting in fibrosis.⁷

To conclude, ABPA is a potentially destructive lung disease which requires high index of suspicion for an early diagnosis. The treatment is prolonged and protracted, but it is essential to prevent and ameliorate the advanced development of end stage lung fibrosis.

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