Case Report

Coexistent ABPA and Tuberculosis Presenting as Lung Collapse with Respiratory Failure

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Abstract

A 30-year-old female presented with respiratory failure and left lung collapse. Possibility of tumour related endobronchial obstruction was considered. Bronchoscopy revealed thick mucus in the left main bronchus, acid-fast bacilli were present on examination of bronchial washings and Gene-Xpert test was positive for *Mycobacterium tuberculosis*. In view of significant mucoid impaction, possibility of allergic bronchopulmonary aspergillosis was considered. Serum *Aspergillus* specific immunoglobulin E levels, *Aspergillus* precipitins and total serum immunoglobulin E levels were elevated. A diagnosis of allergic bronchopulmonary aspergillosis co-existent with pulmonary tuberculosis was made. Patient was treated with anti-tuberculosis therapy along with oral corticosteroids. **[Indian J Chest Dis Allied Sci 2019;61:147-149]**

Key words: Allergic bronchopulmonary aspergillosis, Tuberculosis, Lung collapse.

Introduction

Acute respiratory failure is frequently encountered in clinical practice and prompt recognition and treatment are key to optimising outcomes. Causes of acute respiratory failure can be variable depending upon patient's age, co-morbidities and precipitating factors. Allergic bronchopulmonary aspergillosis (ABPA) is usually diagnosed in patients with a previous history of asthma and respiratory failure at the time of diagnosis is rare. Pulmonary tuberculosis can also present with acute respiratory failure though a combination of these two diseases presenting as respiratory failure is a rare occurrence. We herein describe a case of a young female who presented with acute respiratory failure due to lung collapse and was eventually diagnosed as a case of ABPA with coexistent pulmonary tuberculosis.

Case Report

A 30-year-old female presented to emergency department with history of breathlessness and dry cough for one month, that worsened acutely for the two days. There was history of low-grade fever, weight loss and loss of appetite for one month. She had a history of episodic shortness of breath with wheezing for the last three years that used to improve with inhalers. There was no history of biomass smoke exposure. There was no history of haemoptysis, chest pain or any abdominal, urinary or neurological symptoms.

On physical examination, oxygen saturation was 82% on room air, pulse rate of 112 per minute and respiratory rate 32 breaths per minute. There was no pallor, icterus, lymphadenopathy, cyanosis, clubbing or pedal oedema. Respiratory system examination showed use of accessory muscles, decreased chest movements and absent breath sounds on the left side. Polyphonic wheeze was audible on the right side. Chest radiograph (postero-anterior view) showed left lung collapse with compensatory hyperinflation and herniation of the right lung across the midline (Figure 1). A working diagnosis of acute exacerbation of bronchial asthma with left lung collapse was made. Possibility of foreign body inhalation/ tumourous endobronchial obstruction was also considered. Flexible fiberoptic bronchoscopy demonstrated thick

Figure 1. Chest radiograph (postero-anterior view) demonstrating left lung collapse with compensatory hyperinflation of the right lung.

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mucus plug causing complete luminal occlusion of the left main bronchus (Figure 2). Few mucus plugs were also seen in the right bronchial segments. Multiple mucous plugs were suctioned out. Cytological examination of the bronchial washings revealed acid-fast bacilli and *Mycobacterium tuberculosis* was detected on Gene-Xpert test (Rifampicin resistance not detected). In view of history of asthma and mucoid impaction, investigations for allergic bronchopulmonary aspergillosis were performed. Absolute eosinophil count (1360 cells/μL), total serum IgE levels (>5000 kUA/L) and *Aspergillus fumigatus* specific immunoglobulin E (IgE) levels [4.08 kUA/L (Normal <0.35 kUA/L)] were elevated. Serum *Aspergillus* precipitin test was also positive. *Aspergillus* skin test and *Aspergillus* specific IgG levels could not be performed. A diagnosis of ABPA with coexistent pulmonary tuberculosis was made.

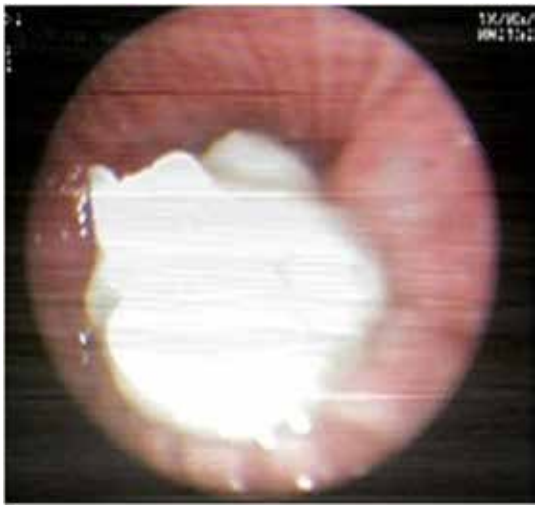

Figure 2. Bronchoscopic image showing large mucous plug occluding the left main bronchus.

Treatment with a four-drug anti-tuberculosis regimen (isoniazid, rifampicin, pyrazinamide and ethambutol) was initiated concurrently with a tapering regimen of oral prednisolone (initiated at dose of 0.5 mg/kg). Patient had marked improvement with therapy. Chest radiograph performed six months later showed complete resolution of radiological abnormalities (Figure 3). Eosinophil count normalised to 180 cells/ μL and serum IgE levels reduced to 680 kUA/L.

Discussion

Allergic bronchopulmonary aspergillosis is a hypersensitivity reaction to *Aspergillus fumigatus* in patients with underlying bronchial asthma or cystic fibrosis.[1] ABPA as a cause of acute respiratory failure has been reported rarely.[2] ABPA has also been reported following pulmonary tuberculosis in patients without history of bronchial asthma.[3,4] Co-existence of ABPA and tuberculosis has been occasionally described. In a report by Kim *et al*[5], diagnosis of tuberculosis was achieved after lobectomy in a patient recently diagnosed as ABPA.

Figure 3. Follow-up chest radiograph (postero-anterior view) demonstrating complete expansion of the collapsed lung.

Acute respiratory failure in our patient was likely precipitated by left lung collapse due to mucous plugging. Acute lung collapse can present as respiratory failure and may be caused by foreign body aspiration, tumours or blood clots.[6] Foreign body aspiration and blood clot obstruction due to haemoptysis are usually medical emergencies. Airway tumours leading to lung collapse is usually a subacute process. In our patient, right lung was hyper-inflated at presentation, which suggests long standing obstruction that allowed the right lung to expand. It is likely that minimal symptoms were initially ignored by the patient while bronchial obstruction was evolving.

Allergic bronchopulmonary aspergillosis usually presents as a complication in a patient with pre-existing asthma or cystic fibrosis. The prevalence of ABPA in bronchial asthma in western countries has been reported to be 6% and in referral centres, 12.9% in a systematic review.[7,8] Diagnosis is usually suspected when patients do not respond to treatment. *Aspergillus fumigatus* specific IgE has been recommended as a screening test for ABPA in patients at risk. *Aspergillus skin* test may be performed if IgE is negative and clinical suspicion is high. Skin test can also be used if specific IgE facility is unavailable, though the sensitivity of skin test is lower.[9] Further evaluation includes blood eosinophil count and total serum IgE levels. Migratory pulmonary infiltrates, central bronchiectasis, sub-segmental or segmental collapse and centrilobular nodules are common radiological findings.[10] Presence of high-attenuation mucus is considered pathognomic of ABPA. In tuberculosis endemic settings, nearly one-third of ABPA patients may be mis-diagnosed as tuberculosis and receive anti-tuberculosis treatment before a diagnosis of ABPA is achieved. Similarly, tuberculosis masquerading as ABPA has also been reported.[11] Other unusual presentations of ABPA included lung mass, pleural

effusion and pneumothorax. Although there are specific radiological features described for tuberculosis as well as ABPA, it may not always be possible to achieve a diagnosis on radiological basis. Coexistent tubercular pleural effusion in a patient with ABPA has also been described.[12]

The probable hypothesis for association of tuberculosis and ABPA includes imbalance between Th1 and Th2 lymphocytes driven inflammation. In bronchial asthma, the predominant inflammation is Th2 CD4 lymphocyte driven which remains true for the patients with ABPA.[13] To the contrary, the protective immunity for tuberculosis is driven by Th1 CD4 lymphocytes.[14] It includes the development of cell-mediated immunity and interferon gamma production. Toll-like receptors play an important role in recognition of mycobacterial antigen and development of protective immunity. This swaying of immune system towards Th2 inflammation may probably increase the susceptibility towards mycobacterial infection. This case highlights that physicians may consider ABPA in the list of differential diagnosis in patients with lung collapse. Pulmonary tuberculosis can have varied clinical presentations and sometimes patients may have acute respiratory failure at presentation.[15] *To the best of our knowledge,* this is the first case in the literature describing coexistent ABPA with tuberculosis presenting with acute respiratory failure.

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