



Allergic Bronchopulmonary Aspergillosis

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Abstract

Allergic bronchopulmonary aspergillosis (ABPA) is type I and II hypersensitivity reaction to *Aspergillus fumigatus* (a species of fungus which is found in household dust, soil and plants) following colonization in patients' airway with background of asthma or cystic fibrosis [1-4]. This disease also affects patients with chronic granulomatous disease, bronchiectasis, lung transplant recipient and hyper immunoglobulinemia IgE. The main symptoms including exacerbation of asthma and productive cough usually respond well to prednisolone and this is necessary to consider early diagnosis of ABPA in any patient with bronchial asthma at early stages to prevent bronchiectasis development and permanent damage. Following CD (4) +Th2 cells activation in response to antigens of *Aspergillus fumigatus*, immunoglobulins are produced (IgE, IgG and IgA). Diagnosis of ABPA is considered based on clinical findings, radiological abnormalities and biological criteria [3]. The mainstay of treatment is corticosteroids which is considered for flare up of disease or acute phase and to be continued for 6-8 weeks. In addition, antifungal medication, itraconazole, is recommended nowadays with daily dose of 200 mg, for period of 16 weeks [4].

Keywords: Allergic bronchopulmonary aspergillosis; Complex hypersensitivity reaction; Asthma; Bronchiectasis; Corticosteroids

Introduction

The first 3 cases of ABPA reported in United Kingdom [5]. *Aspergillus fumigatus*, an opportunistic fungal infection (this is one type of environmental mold), affects predisposed patients with asthma or cystic fibrosis and can represent in five types with different clinical manifestation and severity, resulting from excessive or impaired immune response [6].

Case Report

A 17-year-old female referred to respiratory clinic with 12-month history of chronic cough with intermittent mucoid expectoration along with coughing up some brownish plaques. In addition, she suffers from dyspnoea on exertion only. She experienced some wheezing in the past. The symptoms commenced when she was back from travel to Japan. During the past 12 months, she had been treated by courses of antibiotics and prednisolone with no complete resolution of symptoms. Patient has background history of asthma. She is not diabetic and does not report cystic fibrosis, rheumatic disease or AIDS. She is neither active nor passive smoker and lives with her family. Moreover, patient is not on any

regular medication. On examination, she was afebrile with Bp of 121/76 mm/Hg, pulse rate of 81 and respiratory rate of 12 per minute. On chest auscultation there were bilateral expiratory rhonchi. There was no finger clubbing or peripheral cyanosis. Heart sounds were dual with no murmur and jugular venous pressure was not elevated. Initial investigations including chest X-ray (Figure 1) showed lingular consolidation, therefore she treated with a course of antibiotic and steroid. Unfortunately, soon after treatment completed, the previous sign and symptoms recurred and repeat chest X-ray showed some fleeting shadows and patchy pneumonitis (Figure 2). Patient underwent chest CT scan for more accurate findings (Figures 3,4).

Ct chest revealed central bronchiectasis with high mucoid impaction with involvement of the anterior basal segment of the lower lobes bilaterally and the left upper lobe. Moreover, the lung function showed significant reversibility. FEV1 was 1.51L which is 45.3% of predicted value. After post bronchodilator FEV1 was 1.71 which is 51.1% of predicted value with significant reversibility in the form of 12.9%. The remainder of investigations were the *Aspergillus Fumigatus* IgE level of 4.13

Received date: 29 November 2020; **Accepted date:** 06 December 2020; **Published date:** 10 December 2020

Citation: Korkchi N, Kumar P (2020). Allergic Bronchopulmonary Aspergillosis. SunText Rev Case Rep Image 1(2): 112.

DOI: <https://doi.org/10.51737/2766-4589.2020.012>

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SUNTEXT REVIEWS

kU/L, Total IgE level of 833 KIU/L (< 100), Aspergillus Fumigatus IgG and IgA level of 7 mg/L and 33 mg/L respectively. Blood eosinophil was $2.79 \times 10^9 /L$. Allergy serology including dust mite, grass mix and animal mix were negative. Patient commenced on Prednisolone 25 mg daily based on 0.5/kg per day for four weeks. On the 4-week review, her symptoms had drastically improved, her coughs were nearly gone, and her shortness of breath was mainly under control. The follow-up chest X-ray detected the changes in the lingula has improved. Although there were still some patchy changes in the right middle lobe. Her blood eosinophil had come down to $0.70 \times 10^9/L$ from initial level of $2.79 \times 10^9/L$. As patient had improved clinically and slightly radiologically, the prednisolone dose reduced to 25 mg daily for another 4 weeks with tapering plan of reducing the dose by 5 mg every four weeks. The next review organized for her in 8-week time with repeat chest X-ray and lung function test. This involves all segments but particularly the anterior basal segments of both lower lobes and left upper lobe.

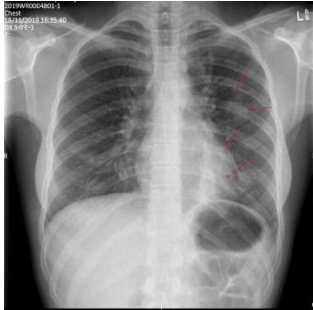


Figure 1: Sub segmental consolidation and atelectasis seen across the base of the left lung projected over the left side of the heart Background of mild bilateral bronchial wall thickening suggesting bronchitis.

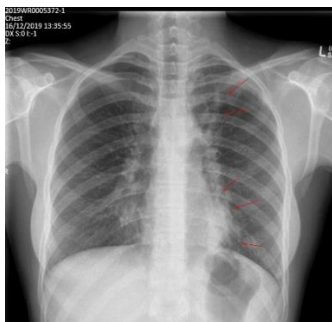


Figure 2: The changes behind the heart shadow are in keeping with a persistent sub segmental left lower lobe and singular pneumonia. The left upper lobe changes have almost completely resolved. This is slowly resolving bronchopneumonia.

Case Discussion

Aspergillus fumigatus is one of the most common fungal infections that affects lungs. Aspergillus fumigatus is a type of mould which is found in moist decaying organic matter, potting soil and compost piles [7]. Pulmonary aspergillosis mainly develops in patients with intrinsic lung disease or immunodeficiency [8]. It can affect both upper and lower respiratory tract and present in different ways [9] (Table 1).

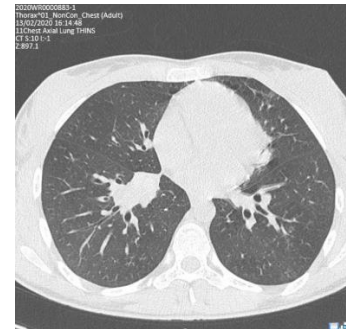


Figure 3: A pattern of central bronchiectasis with high attenuation mucoid impaction (HAM) which is said to be pathognomonic of allergic bronchopulmonary aspergillosis (ABPA).



Figure 4: This is consistent with central bronchiectasis with mucoid impaction which has a branching pattern in the lower lobes known as a finger in glove appearance.

Table 1: Shows various clinical manifestation of infection with aspergillus fumigatus.

Affecting Lower respiratory tract	Affecting Upper respiratory tract
Allergic bronchopulmonary aspergillosis (ABPA)	Allergic aspergillosis (Allergic Aspergillosis sinusitis-AAS)
Immunoglobulin E- mediated aspergillosis-induced asthma (AIA)	Invasive disease including: Acute fulminant invasive sinusitis Chronic invasive sinusitis Granulomatous invasive sinusitis

Hypersensitivity pneumonitis	
Saprophytic colonization-Simple and complex aspergilloma	Saprophytic colonization (Sinus fungal balls)
Invasive disease- Acute and subacute invasive aspergillosis	

Table 2: The diagnostic criteria for ABPA is outlined.

Predisposing conditions (one must be present)
1.Cystic Fibrosis
2.Asthma
Obligatory criteria (both must be present):
Serum IgE levels against <i>Aspergillus fumigatus</i> (>0.35 kU/L) or <i>Aspergillus</i> skin test positivity
Elevated total IgE concentration (typically >1000 IU/mL, but if the patient meets all other criteria, an IgE value <1000 IU/mL may be acceptable, especially if <i>A. fumigatus</i> -specific IgG levels are >27 mg/L)
Other criteria (at least two must be present):
Precipitating serum antibodies to <i>A. fumigatus</i> or elevated serum <i>Aspergillus</i> IgG by immunoassay (>27 mg/L)
Total eosinophil count >500 cells/microL in glucocorticoid-naïve patients (may be historical)
Radiographic pulmonary opacities consistent with ABPA

Table 3: Five stage of ABPA which is outline.

Conventional staging of Allergic bronchopulmonary aspergillosis	
I-	Acute: Patient presents with typical features meeting the diagnosis criteria for ABPA for the first time
II-	Remission: Patient does not have new pulmonary infiltrates and no rise in total IgE for minimum of 6 months
III-	Exacerbation: New pulmonary infiltrates on chest Xray, peripheral eosinophilia and >50% increase in remission total IgE level
IV-	Corticosteroid dependant asthma: patient is dependant to oral corticosteroid and cannot be tapered off completely
V-	Fibrosis lung disease: evidence of irreversible fibrosis and cavitation on chest Radiography. Negative serological parameters usually

Allergic Broncho pulmonary aspergillosis (ABPA) presents with wheeze, cough, dyspnoea, mucus plugs and recurrent pneumonia in individuals with atopy or other hypersensitivity states. Invasive aspergillosis affects immunosuppressed and immune deficient patients (HIV, Leukemia, burns etc.) Aspergilloma (Mycetoma) is found as a fungal ball in pre-existing cavity (mainly resulting from sarcoidosis or TB) [10]. Patients are usually asymptomatic but in some others haemoptysis (secondary to cavity wall erosion) could be fatal when airborne *Aspergillus* spores are inhaled in normal individuals without atopy, body can eliminate the fungal spores resulting in IgG and IgA production. In contrast, exposure of atopic patients to *Aspergillus* spores results in formation of IgE and IgG antibodies [11,12]. The diagnostic criteria for ABPA is outlined in Table 2, as per the latest international Society for Human & Animal Mycology (ISHAM) criteria [13,14].

Investigations

Chest radiograph: In allergic Broncho pulmonary aspergillosis, parenchymal opacities found in upper lobes usually. In addition, we can see bronchiectasis changes and gloved finger shadows resulting from bronchial wall thickening [15].

High resolution CT scan: Shows cylindrical bronchiectasis with upper lobes predominance, three in bud pattern, mucous plugging, high attenuation mucous (HAM), ground glass opacities and atelectasis [16-18].

Pulmonary function tests

PFT is mainly a kind of the disease monitoring measure rather diagnostic test. Normalization of obstructive or restrictive patterns may indicate treatment or disease remission.



Treatment and follow up

The mainstay of treatment is systemic corticosteroid. There are different suggestions in term of dosage and duration of prednisolone. However, his most common regimen is prednisolone 0.5 mg/kg per day for a period of 1 to 2 weeks followed by decreasing dose to 0.5 mg/kg every other day for 6-8 weeks and tapering gradually by 5-10 mg every two weeks. (Tapering should be considered when radiologic infiltrates are resolved and total serum IgE reduced by $\geq 35\%$). Although, based on patient's response to treatment (serum IgE level, chest imaging findings and etc. the prednisolone dosage might be increased. The suggested follow up plan is reviewing patient every 6 to 12 weeks during the first year to check the total serum immunoglobulin E, aiming for reduction by 25%-50% in association with clinical and radiological findings improvement.

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