INTERNATIONAL JOURNAL OF CURRENT RESEARCH IN BIOLOGY AND MEDICINE
ISSN: 2455-944X
www.darshanpublishers.com
DOI:10.22192/ijcrbm Volume 3, Issue 3 - 2018

Original Research Article

DOI: http://dx.doi.org/10.22192/ijcrbm.2018.03.03.004

Allergic bronchopulmonary Aspergillosis mimicking as bilateral cavitatory pulmonary tuberculosis.

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Abstract

Allergic Bronchopulmonary Aspergillosis (ABPA) is an idiopathic inflammatory disease of the lung, characterized by an allergic inflammatory response to colonization of the airways by *Aspergillus fumigatus* or other fungi. It is a hypersensitivity reaction rather than a true infection. ABPA is oftenly wrongly diagnosed as pulmonary tuberculosis. Therefore we present a case report of a young male with complaints of cough with expectoration, fever and dyspnea. He was on anti tuberculosis regimen for the last three months and there was no improvement seen. He was later diagnosed as ABPA.

Keywords: Allergic Bronchopulmonary Aspergillosis (ABPA), Pulmonary tuberculosis

Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is a lung disease resulting from hypersensitivity to *Aspergillus fumigatus*, clinically characterized by impaired mucociliary clearance, mucoid impactions, episodic bronchial obstruction, and pulmonary infiltrates.[1]Early recognition allows treatment with corticosteroids, which are effective but may be required indefinitely. There is some evidence to support the use of newer antifungal azoles as corticosteroid-sparing agents. Patients must be followed closely for recurrent disease. ABPA should be considered in all patients with asthma or cystic fibrosis, but especially in those with difficult to control disease. The relationship between asthma and ABPA is incompletely understood. It is unclear whether having asthma directly increases the risk for ABPA or whether asthma and ABPA share a common predisposition. As many as 25% of subjects with

asthma are sensitized to aspergillus, yet only a small fraction develop ABPA.[2]The differential diagnosis for ABPA includes refractory asthma, newly diagnosed CF, tuberculosis, sarcoidosis, infectious pneumonia, eosinophilic pneumonia, aspergillus sensitive asthma, Churg-Strauss syndrome, and bronchocentric granulomatosis.[3]Almost half are initially misdiagnosed aspulmonary tuberculosis.[4] Diagnostic criteria for ABPA include the presence of bronchial asthma, immediate skin test reactivity to *A fumigatus*, elevated serum IgE levels- total and *A fumigatus*-specific, pulmonary infiltrates (transient or fixed), central bronchiectasis, peripheral blood eosinophilia, and presence of serum precipitins against aspergillus antigen.[5]

Occasionally, patients can develop a syndrome similar to ABPA, but it is caused by fungi other than *A. fumigatus* and is called *allergic bronchopulmonary mycosis*.[6]

Case Report

A 15 year old male patient was admitted in emergency department with the chief complaints of cough with mucopurulent copious sputum production for last 10 years. He also gave history of fever on and off and breathlessness with wheeze for last 10 years. There was no history of hemoptysis. Patient was on antituberculosis treatment (Rifampicin, Isoniazid, Ethambutol, Pyrazinamide) for the last three months based on clinico radiological basis with no significant improvement. On physical examination, the patient was afebrile with a pulse rate of 86/min, respiratory rate of 22/min and a right arm sitting blood pressure of 116/70 mmHg. There was no pallor, cyanosis or clubbing. Chest examination was unremarkable on inspection, palpation and percussion. On auscultation bilateral wheeze were audible. Examination of other systems was unremarkable.

Labortary investigations revealed Hb-12gm , total leukocyte count- 11,000 /mm^3, differential leukocyte count- 78/18/4/0 and ESR-70mm at the end of first hour .Chest radiograph revealed non homogenous opacities in left middle and lower zones (see fig 1). After one month, chest radiograph revealed a new homogenous opacity with well defined margins in right middle and lower zone suggesting of transient fleeting opacities see fig 2). Again after 15 days, chest radiograph was repeated that revealed air fluid levels in both lower zones with slight breaking down see fig 3). Sputum for acid-fast bacilli was negative. Tuberculin skin test was negative at 72 hours. As the patient also gave history of episodic breathlessness with wheezing for the last 10 years, he was investigated further and HRCT was done. High-resolution computed tomography showed evidence of central bronchiectasis and mucus filled dilated bronchi involving both the lower lung fields. In addition, centrilobular nodules were also seen. Location of bronchiectasis was noted as central when it extended within medial one-half of the lung fields and peripheral when it extended outside the medial half. Thus a possibility of allergic bronchopulmonary aspergillosis was suspected. Total IgE was 11924kUA/L (reference range <64kUA/L). Specific IgE against *A. fumigatus* by ELISA were 33kUA/L (normal is<0.35) and the total eosinophil count came out to be 400/mm3 (reference range 40-400/mm3).

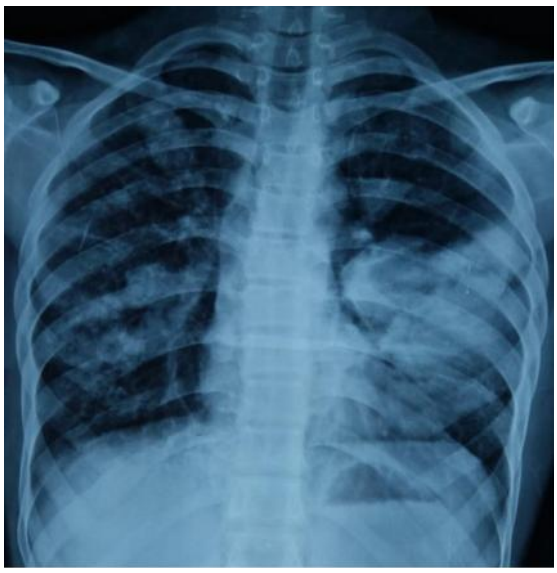

Fig 1- Chest Radiograph revealed non homogenous opacities in left middle and lower zones

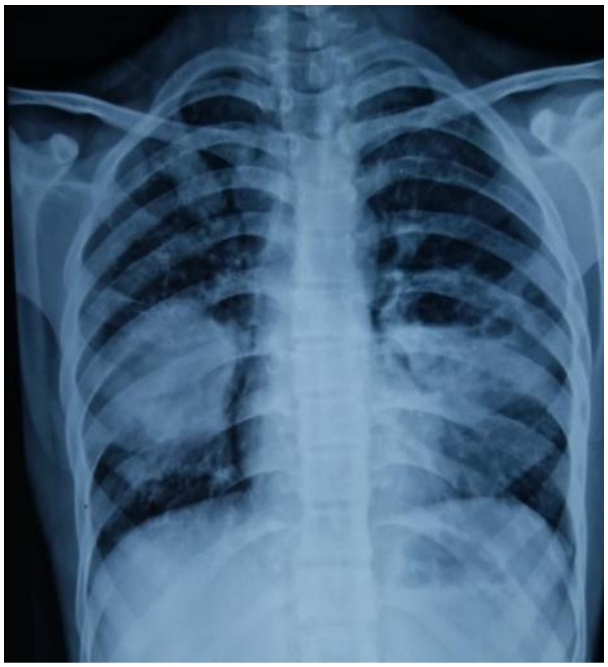

Fig 2- Chest Radiograph revealed a new homogenous opacity in right middle and lower zone.

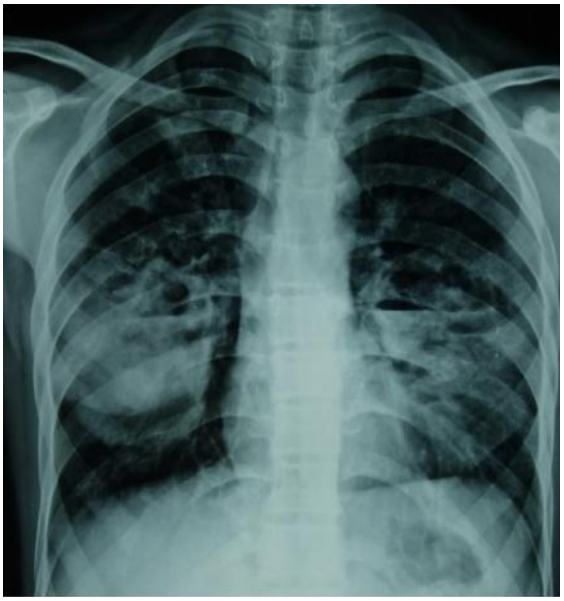

Fig 3 - Chest Radiograph revealed air fluid levels in both lower zones with breaking

Spirometry showed features of mild obstruction (FEV1 57%, FEV1 /FVC ratio 76) with significant bronchodilator reversibility (post bronchodilator FEV1 67% [% change 16]). Thus a diagnosis of ABPA was established. As the patient was young, the patient was investigated for cystic fibrosis.

It was decided to keep the patient under observation while treatment for ABPA with oral prednisolone, starting with a daily single dose of 15mg (weight of patient 30 kg)(0.5mg/kg) along with inhaled budesonide 200micrograms with formetrol 6micrograms in two divided doses and inhaled salbutamol as and when required. The patient was also prescribed 400mg/day itraconazole.

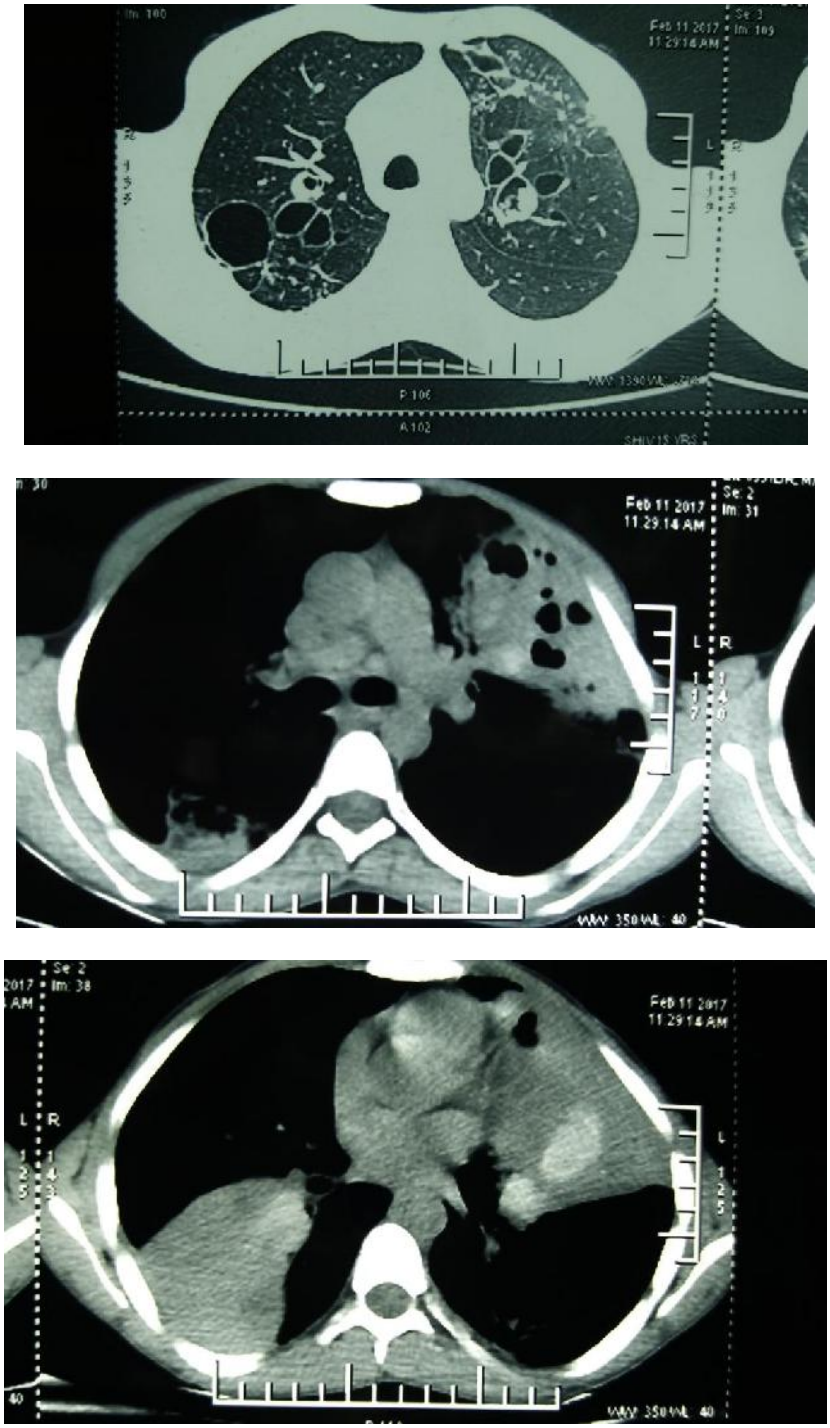

Fig 4-6: Lung and Mediastinal windows of the HRCT scans showing evidence of central bronchiectasis and mucus filled bronchi which involved both the lungs.

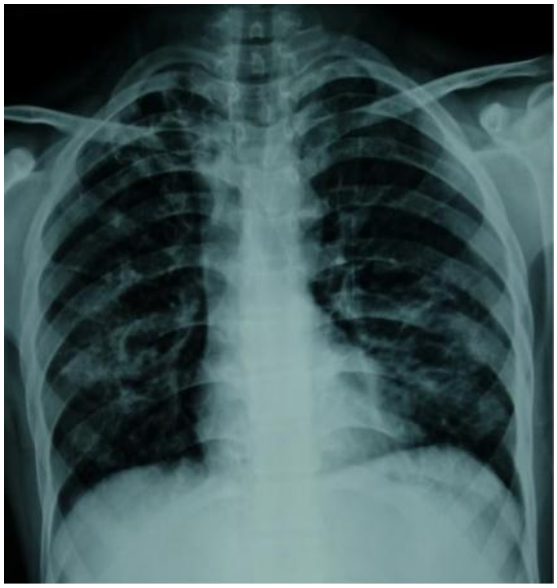

Fig 7: Chest X –ray PA view showing resolution of persisting opacity after a course of steroid.

The patient was also started on oral N acetyl cysteine, multivitamins, calcium, and protein supplements. He was vaccinated with influenza and pneumococcal vaccinations. He was advised follow-up visits at monthly intervals to record for symptoms and was noted for compliance of therapy, any exacerbations, adverse events due to medications, and any non-institutional consultations for exacerbations or any adverse events. Serum IgE was recorded at monthly intervals for 2 months and thereafter bimonthly till the end of 6 months.

Discussion

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity lung disease due to bronchial colonization by *Aspergillus fumigatus* that occurs in susceptible patients with asthma and cystic fibrosis (CF). It is a ubiquitous, saprophytic mold found in both outdoor and indoor air, in potting soil, crawl spaces, compost piles, mulches, freshly cut grass, decaying vegetation, and sewage treatment facilities. [7,8]

The chest radiographic findings are generally nonspecific, although the manifestations of mucoid impaction of the bronchi suggest a diagnosis of ABPA. HRCT of the chest can be normal in almost one-third of the patients, and at this stage it is referred to as serologic ABPA (ABPA-S). The importance of central bronchiectasis (CB) as a specific finding in ABPA is debatable, as almost 40% of the lobes are involved by peripheral bronchiectasis. High-attenuation mucus (HAM), encountered in 20% of patients with ABPA, is pathognomonic of ABPA.

ABPA most commonly complicates the course of bronchial asthma and cystic fibrosis (CF).[9] The clinical presentation of ABPA is usually with poorly controlled asthma, hemoptysis, expectoration of mucus plugs, malaise, and fever. The diagnosis can be made on the basis of a combination of clinical, immunological, and radiological findings.

Radiological findings are nonspecific or subtle in the early stages of the disease, and the diagnosis is often difficult.[10-12]There is preferential involvement of the upper lobes.Plain chest radiography is not sufficiently sensitive to assess the extent of bronchiectasis.[13] Bronchography has been traditionally considered the investigation of choice for the diagnosis of bronchiectasis. On the bronchogram, a characteristic pattern of proximal bronchial dilatation with normal peripheral filling is observed in ABPA.[14,15] However, bronchography is invasive, relatively contraindicated in asthma, and may be associated with adverse effects.[16] HRCT of the chest is safe, allows better assessment of the pattern and distribution of bronchiectasis, and also detects abnormalities that are

not apparent on chest radiography. *Central bronchiectasis* (CB) is believed to be a characteristic finding in ABPA although there are no uniform criteria for the diagnosis of CB. Depending on the proximity of the dilated bronchi from the hilum at a point midway between the hilum and the chest wall, bronchiectasis is arbitrarily defined as central if confined to the medial two-thirds or the medial half of the lung.[17] Bronchiectasis can however extend to the periphery as well, and peripheral bronchiectasis has been described in 26%–39% of the lobes involved by bronchiectasis.[18] The bronchiectasis in ABPA usually involves the upper lobes although, rarely, there may be involvement of the lower zones without involvement of the upper lobes.

Other radiologic findings (ORF) described in ABPA include pulmonary fibrosis, blebs, bullae, parenchymal scarring, emphysematous change, multiple cysts, fibrocavitary lesions, and pleural thickening.[19] The presence of aspergilloma in dilated bronchiectatic cavities has also been documented.

The prognosis of ABPA is good if the disease is detected early and treatment started promptly. It is important that the diagnosis is made and treatment commenced before there is permanent lung damage from bronchiectasis. In such patients, there should be no progression of the disease, although relapses can occur many years later, and long-term followup is recommended.

Source of funding: Nil

Conflict of interest: None declared

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How to cite this article:
Gurleen Kaur, Gurpreet Singh, Rajwinder Kaur, Mandeep Singh, Lakhvir Kaur, Ashi Singh, NS Neki. (2018). Allergic bronchopulmonary Aspergillosis mimicking as bilateral cavitatory pulmonary tuberculosis.. Int. J. Curr. Res. Biol. Med. 3(3): 23-29.
DOI: http://dx.doi.org/10.22192/ijcrbm.2018.03.03.004