

International Journal of Current Research in Medical Sciences

ISSN: 2454-5716 P-ISJN: A4372-3064, E -ISJN: A4372-3061 www.ijcrims.com



**Case Report** 

Volume 3, Issue 10 - 2017

DOI: http://dx.doi.org/10.22192/ijcrms.2017.03.10.003

# Allergic bronchopulmnary aspergillosis presenting with haemoptysis in a non asthmatic patient mimicking like tuberculosis.

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#### Abstract

Aspergillosis is the group of diseases caused by the Aspergillus species. Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitive disease showing various radiographic and clinical manifestations. Allergic bronchopulmonary aspergillosis (ABPA) is typically associated with asthma. Presence of asthma is one of the minimal essential diagnostic criteria for ABPA. Rarely, ABPA has been described in association with other diseases without asthma. We are presenting a case report of female patient who presented with haemoptysis due to allergic bronchopulmonary aspergillosis but without asthma.

Keywords: Asthma, Aspergillosis, Haemoptysis, Bronchoalveolar lavage, HRCT.

#### Introduction

Aspergillus species are ubiquitous spore-forming fungi present in the environment. There are about 180 species of Aspergillus but the common ones which affect the humans are *Aspergillus fumigates* (AF), *Aspergillus flavus*, *Aspergillus niger*. Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity reaction, which occurs predominantly in asthmatic patients and patients with cystic fibrosis. Its clinical and diagnostic manifestations originate from an allergic response to multiple antigens expressed by fungi, most notable among which is *Aspergillus fumigatus*, which can colonize the bronchial mucus .<sup>1</sup> The prevalence of ABPA in asthma clinics is reported to be around 1-2%.<sup>2</sup> and in up to 10% of cystic fibrosis patients,<sup>3</sup> 7-14% in

steroid-dependent asthmatics,<sup>4</sup>. ABPA is rarely observed in the absence of asthma.<sup>5</sup> Prevalence of ABPA presenting as haemoptysis is 31%.<sup>6</sup>

### **Case Report**

A 40 years old female presented with H/o blood in sputum and low grade fever. Fever was not associated with chills and rigors, evening rise of temperature, night sweats and cough with sputum. H/o weight loss was present. She had no h/o hypertension, diabetes mellitus, bronchial asthma and tuberculosis in past. On examination vitals were stable. Clinical examination was also normal. As this area is endemic to tuberculosis, first possibility OF Pulmonary tuberculosis was kept. But early morning sample (repeated 3 times) of sputum for acid fast bacilli (AFB) and culture was negative.ESR was 22. X-Ray chest was normal and Mantoux was negative. Coagulation profile done to rule out bleeding diathesis, was normal. Complete haemogram showed Hb 12gm%, TLC 7700/cmm, N49%, L36%, M04%, E11%. Peripheral blood film showed no toxic granulation, normocytic normochromic anemia with eosinophilia. Due to hypereosinophilia, differential of fungal, parasitic infection and hypersensitivity pneumonitis was kept. Total serum IgE was 5.942 IU/Ml and Aspergillusspecific IgE (52 kUA/L was elevated as well as positive skin reaction to Aspergillus fumigatus. HRCT chest was planned which showed predominantly central bronchiectasis with hyperdense contents in bronchial lumen seen in lingula giving finger in glove appearance along with mild volume loss of lingula. There were areas of consolidation, ground glass density, soft tissue nodules in right upper lobe as well as areas of central saccular bronchiectasis with bronchial wall thickening in right upper lobe. Few soft tissue nodules giving tree in bud appearance in inferior right middle lobe were also detected. No mediastinal lymphadenopathy was documented. Bronchoscopy was planned but patient was noncooperative. Bronchoalveolar lavage (BAL) was taken, which showed budding yeast cells with psuedohyphae. Spirometry was normal, with a forced vital capacity (FVC) of 2.78 L (82% of predicted), a forced expiratory volume in 1 sec (FEV<sub>1</sub>) of 2.28 L (85% of predicted), and an

 $FEV_1/FVC$  ratio of 82%. Hence, all the diagnostic criteria for ABPA were met except for the presence of asthma. The patient was advised oral prednisolone, 40 mg daily that was tapered over five to six months as the patient improved. Oral itraconazole was also prescribed, 200 mg twice daily for 12 weeks. Currently, the patient is under follow-up and no episode of haemoptysis occurred yet.

#### Discussion

The prevalence of ABPA in asthma clinics is reported to be around 1-2%.<sup>2</sup> and in up to 10% of cystic fibrosis patients,<sup>3</sup> 7-14% in steroid-dependent asthmatics.<sup>4</sup> Both environmental factors and a genetic predisposition may be present as far as the prevalence of the disease in asthmatics is concerned. The familial occurrence of ABPA supports the same.<sup>7</sup> The HLA-DR molecules, especially DR2, DR5, and possibly DR4 and DR-7, are associated with susceptibility to ABPA, while HLA-DRQ2 has shown to have resistance to ABPA.<sup>8</sup>

Diagnostic criteria for ABPA without cystic fibrosis:-

- 1. Asthma
- 2. Immediate cutaneous reaction to A. fumigatus
- 3. Total serum IgE concentration (>1000 ng/ml)

4. Elevated A. fumigatus-specific serum IgE levels

5. Precipitating antibodies to *A. fumigatus* in the serum

6. Peripheral blood eosinophilia (not essential for diagnosis)

7. Chest Roentgenographic infiltrates (not essential for diagnosis)

8. Central bronchiectasis

Pulmonary infiltrates, when coupled with eosinophilia in an asthmatic subject, have raised an initial suspicion of some kind of eosinophilic lung disease such as ABPA, chronic eosinophilic pneumonia, or Churg-Strauss syndrome.<sup>9</sup> However, ABPA is only very rarely observed in the absence of asthma. This trend is so pronounced that bronchial asthma has classically been considered an essential diagnostic criterion for ABPA, and has also been believed to play a crucial role in the development of the disease.<sup>10</sup> However, some cases of ABPA have been reported in non-asthmatic patients, albeit extremely infrequently, with less than 20 cases ever having been described in the literature.<sup>11</sup>

CT of the thorax can provide a sensitive method for the assessment of bronchial, parenchymal and pleural abnormalities in patients with ABPA and should constitute a part of the diagnostic work-up of the disease along with plain chest radiographs.<sup>12</sup> Bronchial abnormalities observed in CT were:- central bronchiectasis (100%), dilated and totally occluded bronchi (48%) as evidenced by beaded, tubular opacities and dense, circular opacities, air-fluid levels within dilated bronchi (22%), bronchial wall thickening (10%) and parallel-line opacities extending to periphery (30%). On high-resolution CT, high attenuation mucus plugs have also been reported in 28% of patients with ABPA. Atelectasis, due to proximal mucoid impaction can sometimes be a presenting feature. The atelectasis may be segmental, lobar or may involve the entire lung. Parenchymal abnormalities observed were in the form of consolidation (43%), non-homogenous patchy consolidation (67%), collapse (17%), parenchymal scarring of varying extent (83%), cavities (13%) and emphysematous bullae (4%). Involvement of pleura with thickening has also been observed. The pleura were involved in 43% of patients. In our patient HRCT findings were consistent with ABPA.

Bronchoscopy plays a crucial role in diagnosis and differentiating tuberculosis from ABPA, when there is overlap between two diseases. Patients with ABPA might have atypical manifestations of disease and may not meet standard diagnostic criteria. Bronchoscopy with bronchial biopsy and washing plays a role in detecting these patients. The presence of "allergic" mucin might be overlooked in the small biopsy fragments obtained by bronchoscopy.<sup>13</sup> In our patient diagnosis of ABPA was made on radiological and laboratory investigations .To rule out tuberculosis as a cause of haemoptysis which is endemic in our area, bronchoscopy with bronchoalveolar lavage was done which showed no evidence of mycobacterium tuberculosis.

In the present case, the diagnosis of ABPA was established on clinical. hematological, immunological and radiological grounds. ABPA frequently misdiagnosed as pulmonary is tuberculosis<sup>14</sup> thus delaying the initiation of proper therapy. Almost half are initially misdiagnosed pulmonary as tuberculosis. Frequently, symptoms like haemoptysis, cough, fever etc. caused by ABPA are attributed to active tuberculosis and managed incorrectly.

The case of ABPA reported here is unique. Firstly, this was a case of ABPA in a patient exhibiting no evidence of asthma and, secondly, presence of haemoptysis which is present in 31% patients of ABPA and common in tuberculosis.

# Conclusion

ABPA should be included in the differential diagnosis of patients presenting with haemoptysis even in endemic areas of tuberculosis and in non-asthmatic patients. HRCT has important role in diagnosis even if X-Ray is normal. Bronchoscopy with broncho- alveolar lavage should be done in overlap patients so that appropriate therapy can be started.

#### **Financial support and sponsorship:** Nil

**Conflicts of interest:** there are no conflicts of interest

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How to cite this article:

N.S Neki, Neeraj Joshi, Gaurav Joshi, Gagandeep Singh Shergill, Deepali Kaushal, Mandeep Singh, Aarti Vaid. (2017). Allergic bronchopulmnary aspergillosis presenting with haemoptysis in a non asthmatic patient mimicking like tuberculosis. Int. J. Curr. Res. Med. Sci. 3(10): 11-14. DOI: http://dx.doi.org/10.22192/ijcrms.2017.03.10.003