

Unusual Case of Secondary Pneumothorax in a Patient of Allergic Bronchopulmonary Aspergillosis (ABPA)

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ABSTRACT

Pulmonary aspergillosis is a well-recognized fungal lung disease caused by the *Aspergillus* species (especially *Aspergillus fumigatus*). Allergic bronchopulmonary aspergillosis (ABPA) is milder form of pulmonary aspergillosis compared to other more invasive forms. However, if left untreated, ABPA can cause significant lung damage. We present the case of a 33-year-old man who came with complaints of shortness of breath, chest discomfort, and productive cough. The patient underwent High Resolution Computed Tomography (HRCT) scan of the chest which, suggested the diagnosis of ABPA with secondary tension pneumothorax.

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INTRODUCTION

Spontaneous pneumothorax is characterized by the presence of air in the pleural space. It can be divided into two types—primary and secondary. Primary spontaneous pneumothorax occurs in the absence of known lung disease, while secondary spontaneous pneumothorax occurs due to underlying lung diseases such as acute severe asthma, cystic fibrosis, tuberculosis (TB), necrotizing pneumonia, malignancy, interstitial lung diseases, etc.¹

Allergic bronchopulmonary aspergillosis (ABPA) is a lung disease that occurs due to an overreaction of the immune system to *Aspergillus* antigen, which triggers an inflammatory response that can cause damage to the airways. ABPA is often seen in patients with asthma and cystic fibrosis. An inflammatory response can cause symptoms such as shortness of breath, coughing with expectoration, and wheezing.^{2,3}

Pleural involvement in ABPA is less common. Pleural involvement may occur in the form of pleural thickening, ipsilateral pleural effusion, and lung collapse.⁴ ABPA complicating as secondary spontaneous pneumothorax which is unusual and a few cases have been reported in the literature to date.⁵

CASE DESCRIPTION

A 33-year-old male patient who worked as a farmer visited the radiology department for a high-resolution computed tomography (HRCT) scan of the thorax. He reported a history of difficulty in breathing for the last 2 days and a cough with expectoration for 7 days. Over the past 2 years, he has been experiencing symptoms of atopy and recurrent upper and lower respiratory tract

infections. He did not smoke or drink alcohol. There was no record of previous TB in his medical history.

The patient's vital parameters were assessed during the physical examination, revealing an oral temperature of 99.3°F, blood pressure of 122/78 mm Hg, pulse rate of 104 beats/minute, and respiratory rate of 24 breaths/minute. During chest auscultation, the left hemithorax exhibited diffuse wheezing, while the right hemithorax had absent breath sounds. Upon percussion, a hyper-resonant note was observed in the right hemithorax.

The HRCT scan of the chest showed central bronchiectasis in both lungs, with bronchoceles formation due to impacted mucoid secretions. A large pneumothorax was observed on the right side, with a visible connection between dilated bronchioles and the pleura (known as broncho-pleural communication or fistula), causing the right lung to collapse. Additionally, a small amount of pleural fluid was visible (Figs 1 and 2).

Follow-up laboratory investigations showed a hemoglobin level of 12 gm/dL, a total leukocyte count of 7100/mm³ with 25% eosinophils on the differential count, an erythrocyte sedimentation rate of 40 mm in 1 hour, and a fasting blood sugar level of 78 mg/dL. The coagulation profile was normal. Renal function tests, liver function tests, and electrolytes were within normal limits. The follow-up immunological survey showed a total serum immunoglobulin E (IgE) level of 2750 IU/mL, with increased levels of serum IgE and IgG against *A. fumigatus*. Sputum microscopy and Ziehl–Neelsen staining for acid-fast bacilli were negative.

Based on the patient's clinical, pathological, and radiological characteristics, the diagnosis

of secondary spontaneous pneumothorax complicated by ABPA was suggested. The patient underwent intercostal drainage under a water seal to treat the pneumothorax. Additionally, oxygen inhalation, nebulization, antibiotics, antifungal medication, and oral steroids were administered. The patient improved on follow-up.

DISCUSSION

In 1952, Hinson et al. were the first to describe ABPA among individuals suffering from asthma.⁶ Since then, further research has been conducted on ABPA to better

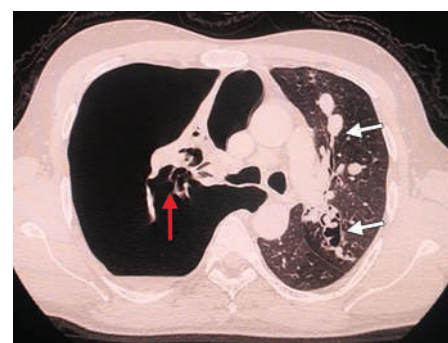


Fig. 1: Shows an axial image obtained from the HRCT chest, revealing bilateral central bronchiectasis with bronchoceles formation (indicated by white arrows). The image also demonstrates a large pneumothorax on the right side, with dilated peripheral bronchiole showing direct communication with the pleural air, suggestive of bronchopulmonary communication (indicated by a red arrow). Furthermore, a small amount of dependent pleural fluid is visible on the right side

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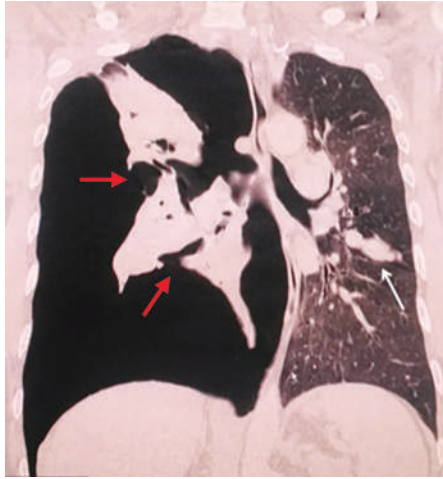


Fig. 2: Shows a coronal reformatted image obtained from the HRCT chest, revealing bilateral central bronchiectasis with bronchoceles formation (indicated by white arrows). The image also demonstrates a large pneumothorax on the right side, with dilated peripheral bronchioles showing direct communication with the pleural air, suggestive of bronchopulmonary communication (indicated by a red arrow). Furthermore, the image also reveals a complete collapse of the right lung

understand its pathophysiology, diagnosis, and treatment.

The clinical presentation of ABPA typically involves nonspecific respiratory and systemic symptoms such as shortness of breath, pleuritic chest pain, cough with expectoration, wheezing, fatigue, weight loss, low-grade fever, etc. Elevated total serum IgE levels, typically exceeding 1000 ng/mL, are commonly observed through laboratory testing in individuals with ABPA. Additionally, laboratory examination may also reveal peripheral blood eosinophilia in these patients.⁷

Patterson et al. in 1982 described five clinical stages of ABPA. The stages are acute, remission, exacerbation, corticosteroid-dependent asthma and fibrotic. Pleural complications that can arise from ABPA include pleural effusion, pleural thickening, and pneumothorax. These complications are

more commonly observed during the fibrotic stage of the disease.⁸

The International Society for Human and Animal Mycology (ISHAM) working group has classified ABPA into four major categories based on radiological findings, which indicate disease progression from mild to severe. These categories are as follows—(1) serologic ABPA (ABPA-S), (2) ABPA with bronchiectasis (ABPA-B), (3) ABPA with high-attenuation mucus (ABPA-HAM), and (4) ABPA with chronic pleuropulmonary fibrosis (ABPA-CPF).⁹

Radiologically, ABPA can be classified into different categories based on disease severity. HRCT can reveal several characteristic features of ABPA, including central bronchiectasis, mucus plugging, centrilobular nodular infiltrates, and in advanced stages, extensive fibrosis, and cavitation.¹⁰

Modified ISHAM working group 2013 criteria¹¹ for diagnosis of ABPA are as follows:¹¹

- Predisposing asthma or cystic fibrosis.
- Obligatory criteria—(1) IgE > 1000 IU/mL and (2) positive immediate skin test or increased IgE antibody to *Aspergillus*.
- Supportive criteria (two or more should be present)—(1) eosinophilia > 500 cells/μL, (2) precipitins or increased IgG antibody to *Aspergillus*, and (3) fixed or fleeting radiographic opacities.¹¹

Pleural involvement in ABPA is uncommon and usually presents as pleural effusions, pleural thickening and pleural calcifications. Secondary spontaneous pneumothorax is a rare complication or clinical presentation of ABPA. Secondary spontaneous pneumothorax with ABPA is a relatively uncommon condition.

In the literature, spontaneous pneumothorax cases in patients with ABPA have been reported by several authors (Ricketti et al., 1984, Judson et al., 1993, Das et al., 2014, Vishnukanth et al., 2017). These cases were successfully treated with primary intercostal chest tube drainage to evacuate the pneumothorax.

There is a high prevalence of ABPA as well as pulmonary TB (PTB) in India. In areas with a high prevalence of PTB, patients with ABPA

having atypical radiological presentations are often misdiagnosed with PTB. In some Indian studies, ABPA was misdiagnosed as TB in as high as 17–50% of cases.¹²

CONCLUSION

In the evaluation of chronic lung diseases that present with pneumothorax, it is essential to include ABPA in the differential diagnosis. However, the incidence of ABPA is often underestimated in India because of the high prevalence of PTB. To diagnose ABPA accurately and identify its complications, HRCT of the thorax is a reliable and precise imaging modality that provides both high sensitivity and specificity.

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