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A case of allergic bronchopulmonary aspergillosis in a patient with a history of pulmonary tuberculosis and chronic obstructive pulmonary disease (COPD)

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Abstract:

Allergic bronchopulmonary aspergillosis (ABPA) is an inflammatory disease that is usually seen in patients with uncontrolled asthma, who have allergic sensitization to *Aspergillus* antigen. However, patients without asthma may rarely develop ABPA. We present a case of a patient who was hospitalized due to chronic obstructive pulmonary disease (COPD) exacerbation and was referred to the allergy clinic due to elevated total Immunoglobulin E (IgE), leading to a diagnosis of ABPA. The patient, previously treated for pulmonary tuberculosis, had COPD and a total IgE value over 2,500 IU/mL. *Aspergillus*-specific IgE was positive. While receiving systemic steroid therapy, the patient's blood eosinophil count was 190 cells/ μ L, and the pulmonary function test showed an obstructive pattern. Radiological imaging revealed emphysema, parenchymal distortion, bronchial enlargement, and cavitation. These findings led to a diagnosis of ABPA. ABPA may develop in patients with post-tuberculosis structural lung disease or COPD who have *Aspergillus* sensitization. Therefore, if these patients exhibit clinical and radiological findings compatible with ABPA, an investigation for ABPA should be conducted.

Keywords:

Allergic Bronchopulmonary Aspergillosis, asthma, chronic airway diseases, chronic obstructive pulmonary disease (COPD), tuberculosis

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Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is an inflammatory disease resulting from the immune

response initiated against *Aspergillus fumigatus* colonizing the airways of patients with asthma and cystic fibrosis.^[1] The incidence of ABPA is 1–2.5% in asthmatics and 10% in adults with cystic fibrosis.^[2] ABPA

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is more common in patients with allergic sensitization to *Aspergillus* antigen and in those with severe asthma.^[3,4] Most patients present with uncontrolled asthma symptoms, but ABPA can rarely develop in patients without asthma. In this report, we present a case of ABPA in a patient with post-tuberculosis (TB) structural lung disease and chronic obstructive pulmonary disease (COPD).

Case Report

A 57-year-old male patient was admitted to the ward due to COPD exacerbation and was referred to the immunology and allergy clinics due to elevated total Immunoglobulin E (IgE). The patient had been diagnosed with COPD for ten years and experienced increased dyspnea and yellow-green sputum over the past ten days. He had no accompanying symptoms of rhinitis.

The patient had been treated for pulmonary tuberculosis in his youth. His complaints of shortness of breath and cough had persisted throughout the day for ten years and worsened in the winter months. He also had recurring coughs and sputum, especially in winter. He was using supplemental oxygen and inhaler treatment for COPD. His smoking history was significant, at 100 pack-years. He worked as a scrap worker and had biomass exposure. He had no history of allergies or indoor mold exposure, and there was no evidence of asthma in his family history.

On lung auscultation, respiratory sounds were severely reduced. Pulmonary radiology revealed bullous emphysema in both lungs, occasional parenchymal distortion, and of consolidation in the right upper lobe's posterior segment with irregular contours of cavitation leaning against the pleura. Soft tissue densities with calcifications in the cavitation suggested a fungus ball [Fig. 1]. The patient's total IgE level was above 2,500 kU/L. There was no histamine response in the patient's skin prick test. *Aspergillus*-specific IgE was 27.8 kU/L, which was above the normal range (0–0.35 kU/L) and positive. The blood eosinophil count during treatment after hospitalization was determined to be 190 cells/ μ L. The pulmonary functions were measured while the patient was using systemic steroid treatment, with results as follows: forced expiratory volume in 1 second (FEV₁) at 660 mL (19%) and FEV₁/forced vital capacity (FVC) at 44%, and maximal expiratory flow rate 25–75 percent at 10%. With all these findings, the patient was considered to be compatible with ABPA (Table 1).

Discussion

Although ABPA primarily affects patients with asthma, it can rarely develop in those without asthma. The vast majority of ABPA patients without asthma have underlying bronchiectasis. It has also been reported that ABPA develops in lung diseases such as bronchiectasis, post-tuberculosis lung disease, Kartagener's Syndrome, Macleod's Syndrome, COPD, chronic granulomatous disease, and Hyper IgE Syndrome.^[1] ABPA sans asthma has been identified as a distinct subset of ABPA, with these patients showing better lung function and fewer ABPA exacerbations.^[5]

The case presented involved a patient with post-TB structural lung disease and COPD. He had high levels of total IgE and *Aspergillus*-specific IgE. His blood eosinophil level was not above 500 cells/ μ L, but it is known that blood eosinophil values may be low or normal in ABPA patients receiving oral steroid therapy.^[6] The patient was using systemic steroid treatment for COPD exacerbation when the examinations were performed.

It has also been reported that ABPA radiology may present with atypical radiological findings in patients without asthma. Cases of ABPA have been reported with mass lesions and miliary nodules in the lung.^[7–9]

Aspergillus species may colonize in patients with post-TB structural lung disease, leading to pulmonary aspergillo-

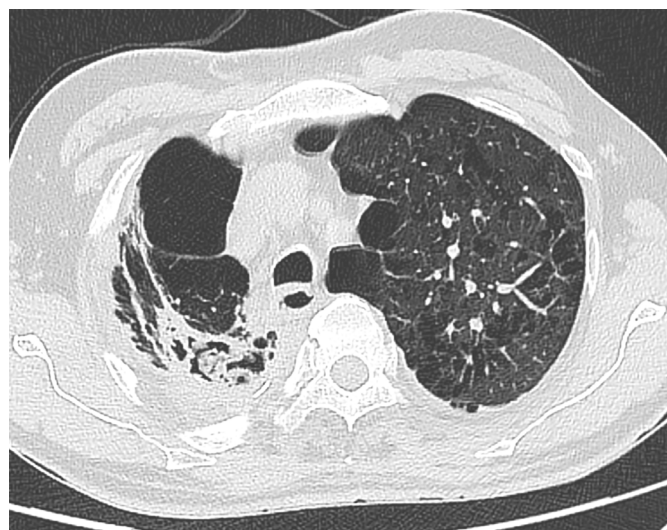


Figure 1: Computed tomography section revealing bullous emphysema, parenchymal distortion, consolidation areas in the right upper lobe with cavitation, and soft tissue densities with calcifications in the cavitation

Table 1. Diagnostic criteria for allergic bronchopulmonary aspergillosis (ABPA)^[6]**Predisposing conditions**

Bronchial asthma, cystic fibrosis

Obligatory criteria (both should be present)Type I *Aspergillus* skin test positive (immediate cutaneous hypersensitivity to *Aspergillus* antigen) or elevated IgE levels against *Aspergillus fumigatus*
Elevated total IgE levels (> 1,000 IU/mL)***Other criteria (at least two out of three)**Presence of precipitating or IgG antibodies against *A. fumigatus* in serum

Radiographic pulmonary opacities consistent with ABPA**

Total eosinophil count > 500 cells/ μ L in steroid-naive patients (may be historical)

*: If the patient meets all other criteria, an IgE value < 1,000 IU/mL may be acceptable, **: The chest radiographic features consistent with ABPA may be transient (e.g., consolidation, nodules, tram-track opacities, toothpaste/finger-in-glove opacities, fleeting opacities) or permanent (e.g., parallel line and ring shadows, bronchiectasis, and pleuropulmonary fibrosis)

sis and *Aspergillus* sensitization. In a study, *Aspergillus* sensitization was found positive in 32% of patients with post-TB fibrocavitary lung disease.^[10] Therefore, clinicians should be aware that ABPA may develop in patients with post-tubercular lung lesions, as reported in the literature.^[11,12] Moreover, due to the similar clinical and radiological findings of ABPA and pulmonary TB, patients with ABPA may be misdiagnosed as having pulmonary tuberculosis and may receive anti-TB treatment before an ABPA diagnosis is made.^[12]

ABPA complicating the clinical presentation in COPD patients has been previously reported.^[13–15] According to the report by Agarwal et al.,^[16] in COPD patients, *Aspergillus* sensitization was found in 8.5% and ABPA in 1% of COPD patients. They reported that, theoretically, ABPA may develop in COPD patients during the disease process due to increased mucus secretion and decreased mucociliary clearance. In addition, ABPA was found to increase mortality in COPD patients.^[17]

In conclusion, in patients with post-TB structural lung disease or COPD, *Aspergillus* sensitization is possible. Therefore, ABPA should be investigated in such patients in case of clinical and radiological findings compatible with ABPA.

Informed Consent

Written informed consent was obtained from the patient for the publication of the case report and the accompanying image.

Conflicts of interest

There are no conflicts of interest.

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Peer-review

Externally peer-reviewed.

Authorship Contributions

Concept – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Design – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Supervision – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Funding – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Materials – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Data collection &/or processing – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Analysis and/or interpretation – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Literature search – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Writing – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.; Critical review – O.T., H.Ç.T., M.Y., Ö.A., F.D.Ç., K.A.

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