Case Report

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A 48-year-old male with allergic bronchopulmonary aspergillosis: a rare case report

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Introduction: Allergic bronchopulmonary mycoses, primarily from *Aspergillus fumigatus*, complicate asthma and cystic fibrosis, presenting diagnostic challenges due to overlapping respiratory symptoms.

Case presentation: A 48-year-old male with asthma and a history of Guillain-Barré syndrome presented with cough, chest pain, dyspnea, and weight loss. He was diagnosed with allergic bronchopulmonary aspergillosis after a series of investigations, including CT scans and bronchoscopy.

Clinical discussion: Allergic bronchopulmonary aspergillosis (ABPA) is a rare lung disease caused by an immune reaction to *Aspergillus fungi* in individuals with pre-existing respiratory conditions like asthma or cystic fibrosis. The primary treatment for ABPA involves systemic corticosteroids, often combined with antifungal agents, to reduce the need for long-term high-dose steroid therapy.

Conclusion: This case highlights the need for accurate and early diagnosis of ABPA, especially in patients with asthma or other respiratory diseases, which helps prevent potential complications. Additionally, the case provides valuable insights into how to manage patients with ABPA, contributing to the improvement of protocols and medical care in the future.

Keywords: allergic bronchopulmonary aspergillosis, antifungal agents, case report, systemic corticosteroids

Introduction

Allergic bronchopulmonary mycoses are multifaceted pulmonary disorders resulting from immune responses to fungal colonization, mainly by *Aspergillus fumigatus*, which invade the airways of patients suffering from chronic pulmonary diseases, most frequently asthma or cystic fibrosis (CF)[1]. ABPA develops in 1%–2% of patients with asthma and 1%–7.8% of patients with cystic fibrosis. ABPA typically refers to cases where the causative pathogen is *A. fumigatus*. In comparison, allergic bronchopulmonary mycosis (ABPM) describes a similar syndrome caused by fungi other than *A. fumigatus*[2]. Furthermore, there is ongoing debate regarding whether ABPA/ABPM is an eosinophilic or allergic disease. The International Society for Human and Animal Mycology's ABPA Working Group (ISHAM-AWG) recommendations state that the main criteria for diagnosing ABPA/ABPM are the existence of fungus-specific IgE and total IgE levels in serum over 500 IU/mL[3]. COPD, pulmonary tuberculosis, and bronchiectasis can all cause elevated levels of *A. fumigatus*-specific IgE and IgG; some of these individuals may develop ABPA. A significant portion of patients (about 30%) are initially misdiagnosed as pulmonary tuberculosis, and ABPA-complicating asthma is still poorly recognized. Treatments for ABPA/ABPM typically consist of three to five months of oral corticosteroids and/or triazole antifungals[4]. We report a case of ABPA for a patient with asthma, with indistinguishable symptoms compared to the more common symptoms of respiratory diseases; this is indeed what makes this condition difficult to diagnose.

HIGHLIGHTS

- The potential coexistence of ABPA with allergic fungal sinusitis and asthma warrants careful evaluation, as symptoms may overlap
- Elevated serum IgE levels greater than 1000 ng/m.
- Peripheral eosinophilia, indicating an allergic response
- The primary treatment for ABPA involves systemic corticosteroids and antifungal agents

Case presentation

A 48-year-old male patient was admitted to the pulmonology department with a cough producing yellow sputum without

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hemoptysis, generalized chest pain after coughing, and second-degree exertional dyspnea, according to the MMRC, without orthopnea or nocturnal paroxysmal spnea. The complaint was accompanied by an unmeasured fever, profuse night sweats, chills, and a weight loss of about 10 kg over a month and a half, along with a decreased appetite. He had a medical history of Guillain-Barré syndrome two years ago with complete recovery, asthma diagnosed since childhood, and poorly controlled medication with recurrent attacks and recurrent lower respiratory tract infections for the past five years. There are no surgical or allergic histories, and the family history includes asthma in the mother only. His medication history includes various antibiotics and salbutamol-formilar plus 125 inhalers regularly, along with leukast 10 mg and prednisolone repeatedly for each respiratory infection, the last time being five days before admission. Upon clinical examination, his blood pressure was 110/70, the respiratory rate was 25 breaths per minute, and his oxygen saturation was 95%. On auscultation of the chest, wheezing and prolonged expiration were noted bilaterally, with fine inspiratory crackles at the base of the right lung. We found, after performing a CT scan of the chest with contrast, the presence of lung densities of various shapes in the upper and lower lobes on both sides, some cavitary, and thickened bronchial walls. Granulomatous consolidations were also observed, indicating the presence of active pulmonary tuberculosis with mediastinal lymphadenopathy measuring between 1 and 2 cm. No pleural effusion, pneumothorax, or pericardial effusion was observed (Fig. 1). The search for Koch's bacillus through a sputum sample was also negative three times. A sample was sent for the tuberculosis culture and sensitivity, and a Gram stain showed *Streptococcus* bacteria, with the culture result being *Enterobacter*. An ECG was also performed, which showed a regular sinus rhythm with a right axis deviation and no electrocardiographic changes. Laboratory tests were conducted, revealing WBC 9.14, neutrophils 70%, lymphocytes 13%, eosinophils 11.5%, and CRP 3.7. Subsequently, a chest CT scan with contrast was performed, which showed a bronchiectasis syndrome in both lung bases, the left lung lobe, and some areas of the apices, along with apical pleural thickening and left apical peripheral density. Randomly distributed interstitial infiltrates were observed, giving the right middle lobe a ground-glass appearance, and no mediastinal lymphadenopathy was seen.

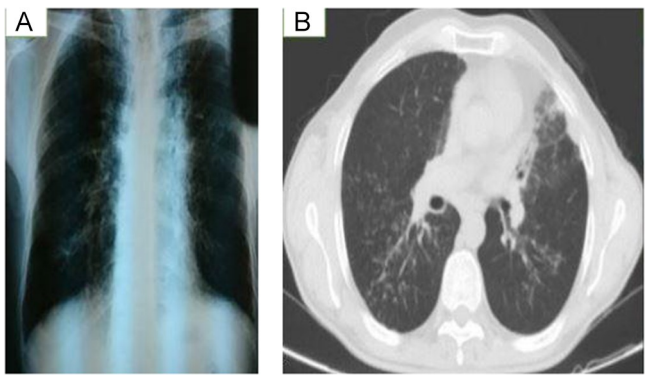

Figure 1. (A). X-ray demonstrated area of reticulation. (B). Ct scan shows bronchiectasis, high attenuation mucus plugging, tree-in-bud opacity and a little bit of ground glass opacity.

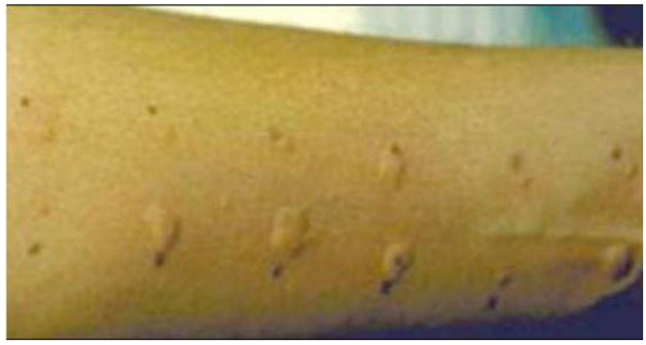

Figure 2. Positive skin prick test for aspergillus.

We then performed a bronchoscopy with bronchoalveolar lavage, which showed a differential count of 55% macrophages, 20% neutrophils, 15% lymphocytes, and 10% eosinophils. The IgE level was 515.3 IU/ml, indicating eosinophilic lung disease. We then conducted an ANCA test, which was negative, and a skin prick test for *Aspergillus*, which was positive (Fig. 2). The final diagnosis was allergic bronchopulmonary aspergillosis. After a follow-up of 6 months, the patient's condition was stable without any complications.

Discussion

The rare allergy lung disease known as allergic bronchopulmonary aspergillosis (ABPA) occurs due to an immune reaction to Aspergillus antigens that persist in the airways of patients with long-term respiratory tract diseases such as cystic fibrosis or asthma[1]. ABPA represents 1–7.8% of patients diagnosed with cystic fibrosis and 1–2% of individuals with asthma[2] and affects 2.5 million patients globally[3]. Assessing ABPA prevalence in asthmatics is problematic due to the absence of standardized diagnostic criteria[4]. ABPA symptoms include wheezing, dyspnea, and a heavy brown to black mucus producing cough sometimes with plugs. It may cause fever, malaise, anorexia, hemoptysis, and flu-like manifestations[5]. The immunological examination provides the basis for the diagnosis of ABPA. The most sensitive test for diagnosing ABPA at this time is a high level of *Aspergillus fumigatus*-specific IgE, which is also thought to be the best test for screening for ABPA in asthmatic patients, with a level over 0.35 kUA/l[4]. As of right now, the *Aspergillus* skin test is not recommended for ABPA screening in asthmatic patients. It may overlook 6% to 12% of individuals with ABPA due to its sensitivity of 88% to 94%[6]. Serum total IgE measurement is helpful for follow-up and can be a valuable diagnostic test for people with ABPA. Active ABPA is mainly ruled out as the underlying cause of a patient's symptoms if their blood total IgE level is normal. However, when it comes to detecting ABPA in asthmatic patients, serum total IgE (with a limit of 500 IU/mL) has a high sensitivity (96%) but low specificity (24%)[7] . Therefore, it is not a recommended screening test for ABPA . High-resolution chest CT is currently ABPA's preferred imaging modality for radiological studies[8]. Although central bronchiectasis is common and typical, in around 40% of patients, it can spread to the periphery. Since ABPA can occur without any radiological symptoms, it can only be detected immunologically. Since the value of MRI in diagnosing ABPA is still being determined, standard practice does not advise its use[9].

In our case, auscultation of the chest revealed bilateral wheeze and delayed expiration, along with little inspiratory crackles at the right lung's base. Following a contrast-enhanced CT scan of the chest, we discovered thicker bronchial walls, some cavitary, and lung densities of different shapes in the upper and lower lobes on both sides. Additionally, there were granulomatous consolidations, which showed mediastinal lymphadenopathy ranging from 1 to 2 cm and the presence of active pulmonary tuberculosis. There were no signs of pericardial effusion, pneumothorax, or pleural effusion. Additionally, a sputum sample used to search for Koch's bacillus came up negative three times. A Gram stain revealed *Streptococcus* bacteria in a sample sent for the tuberculosis culture and sensitivity, while the culture result revealed *Enterobacter*. Additionally, an ECG was taken, which revealed no electrocardiographic abnormalities and a typical sinus rhythm with a right axis deviation. WBC 9.14, neutrophils 70%, lymphocytes 13%, eosinophils 11.5%, and CRP 3.7 were found in laboratory testing. Following that, a contrast-enhanced CT scan of the chest revealed apical pleural thickening, left apical peripheral density, and bronchiectasis syndrome in both lung bases, the left lung lobe, and certain apices. There was no evidence of mediastinal lymphadenopathy, and the right middle lobe exhibited a ground-glass appearance due to randomly scattered interstitial infiltrates. A bronchoscopy with bronchoalveolar lavage was subsequently conducted, and the results revealed a differential count of 10% eosinophils, 20% neutrophils, 15% lymphocytes, and 55% macrophages . With an IgE level of 515.3 IU/ml, eosinophilic lung disease was diagnosed. After that, we performed a skin prick test for *Aspergillus*, which came back positive, and an ANCA test, which came back negative. The main treatments of ABPA are immunosuppression and complete eradication of the causative agent. However, eradicating the causative antigen is difficult due to its global nature[10]. The most essential treatment includes systemic corticosteroids, and therapeutic strategies also include using antifungal agents during the initial treatment, which can decrease the need for long-term high doses of systemic corticosteroids[11]. Using prednisolone and itraconazole together reduces the one-year exacerbation rate more effectively than using either drug alone[12].

Omalizumab emerges as novel therapeutic option for ABPA associated with asthma and cystic fibrosis. Meiling Jin et al. have demonstrated a significant decrease in the annualized exacerbations rate and the dosage of oral corticosteroids. Also, the study indicated marked improvements in lung function and overall asthma control.

Mepolizumab successfully decreases exacerbation frequency in patients with eosinophilic asthma. Previous work had successfully treated a 64-year-old women patient with ABPA by mepolizumab[13].

Methods

The work has been reported in line with the SCARE criteria[14].

Conclusion

In conclusion, this case highlights the significance of appropriate diagnosis and treatment of ABPA as a rare but impactful condition. Increasing medical understanding of this case can lead to improved healthcare for patients and better support for their needs.

Ethical approval

Ethics approval is not required for case reports deemed not to constitute research at our institution (Hama University).

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author's contribution

K.K. wrote a part of the manuscript. K.A. wrote a part of the manuscript. A.A.O. wrote a part of the manuscript. A.A. wrote a part of the manuscript. T.A. wrote a part of the manuscript. A.S. A.A.-K. wrote a part of the manuscript. S.F.A. wrote a part of the manuscript. M.Y.A. wrote a part of the manuscript. A.D. wrote a part of the manuscript. B.S. wrote a part of the manuscript. M. S. wrote a part of the manuscript. All authors approved the final manuscript.

Conflicts of interest disclosure

Not applicable.

Guarantor

Guarantor author is Bilal Sleiay.

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Provenance and peer review

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