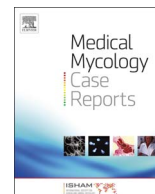




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## Allergic Bronchopulmonary Aspergillosis masquerading as recurrent bacterial pneumonia



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### ABSTRACT

Allergic Bronchopulmonary Aspergillosis (ABPA) can be diagnosed in an asthmatic with suitable radiologic and immunological features. However ABPA is likely to be misdiagnosed with bacterial pneumonia. Here we report a case of ABPA masquerading as recurrent bacterial pneumonia. Treatment with high-dose inhaled corticosteroids was effective. To our best knowledge, this is the first reported case of ABPA in Vietnam.

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### 1. Introduction

Allergic Bronchopulmonary Aspergillosis (ABPA) is the hypersensitive status of airway to *Aspergillus* which colonizes the bronchial mucosa, occurring mainly in patients with asthma or cystic fibrosis. The diagnostic criteria for ABPA articulated by Rosenberg, and later revised by Greenberger, has been widely used [1]. However, clinical presentation of ABPA is indistinguishable with pneumonia or pulmonary tuberculosis, especially at developing countries where the prevalence of pulmonary tuberculosis is still high [2–4]. This is really a diagnostic challenge to most clinicians.

The mainstay treatment of ABPA includes the systemic corticosteroid and the adjunctive antifungal agent [1]. Several published case reports mentioned inhaled corticosteroids (ICS) in management of ABPA, specially in serologic ABPA (ABPA-S) [7–9]. Here we report the first ABPA case at Vietnam misdiagnosed as recurrent bacterial pneumonia and treated with high-dose inhaled corticosteroid effectively.

### 2. Case

A 74 year-old male patient whose past medical history included allergic rhinitis, hypertension, and type 2 diabetes mellitus was a non-smoker and did not take alcohol. Four months ago (on day

– 120), he complained of cough and his chest X ray (CXR) showed right perihilar airspace opacities (Fig. 1A). He was diagnosed and treated as a bacterial pneumonia. His cough improved and his CXR (on day –30) came back almost normal three months later (Fig. 1B).

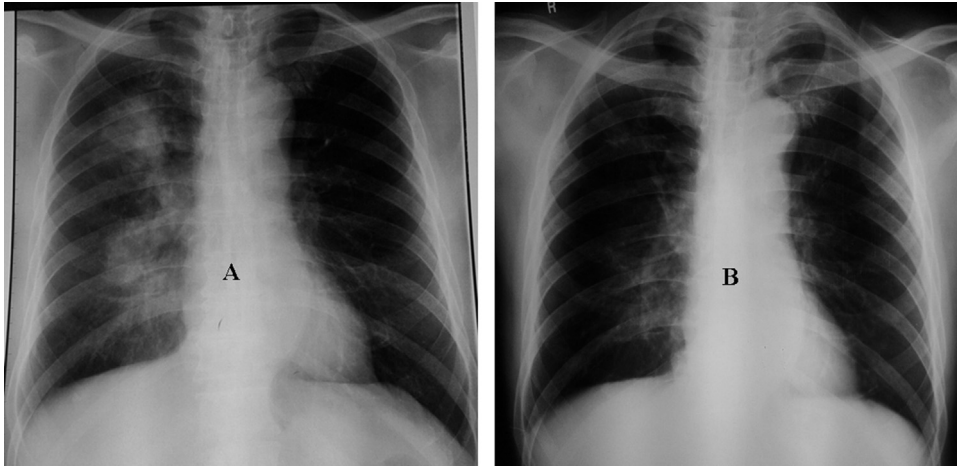
Subsequently, he had a 10- day history of coughing up white-cloudy and viscous sputum before admission. On day 0 he presented at the emergency department with fatigue and shortness of breath. On examination, his pulse was 100/min, blood pressure 120/80 mmHg, temperature 37 °C, respiratory rate 28/min, peripheral capillary oxygen saturation (SpO<sub>2</sub>) 88% while breathing room air. Use of accessory muscles for respiration and scattered rhonchi were also recorded.

The cell blood count revealed eosinophilia which was 3400/mm<sup>3</sup>. The blood glucose level was 336 mg/ml. CXR on admission showed bilateral perihilar airspace opacities (Fig. 2A). Both serologic tests for parasitic infections (*Strongyloides stercoralis*, *Toxocara canis*, *Clonorchis sinensis*, *Paragonimus westermani*, *Entamoeba histolytica*, *Ancylostoma*) and the direct faecal smear were negative on hospital day +1. Three consecutive sputum samples for acid-fast bacilli (AFB) testing were negative. However the sputum culture was positive for *Klebsiella pneumoniae* (2.10<sup>6</sup> cfu/ml) on hospital day +3. Therefore, diagnosis of bacterial pneumonia was confirmed and he continued antibiotic therapy.

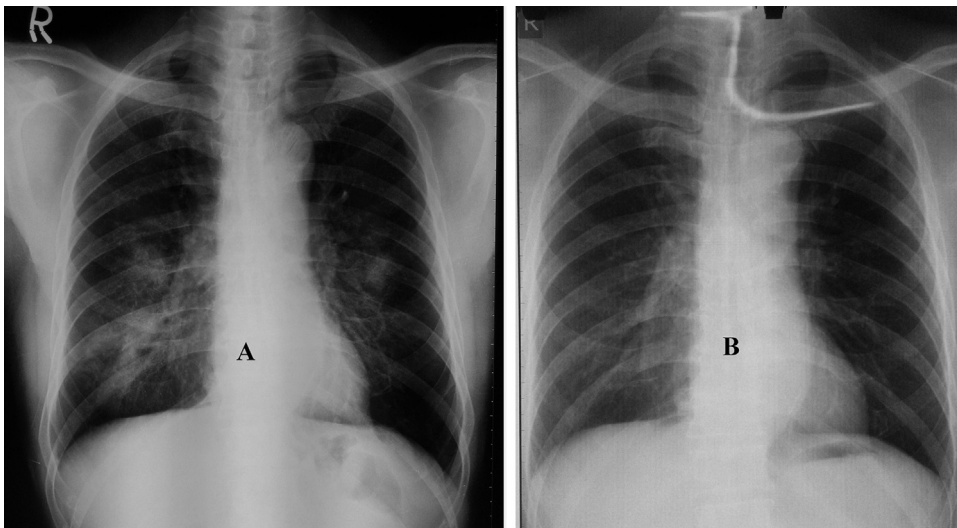
The total serum IgE level and serum level of specific IgG to aspergillus was 5110 IU/ml and 1000 U/ml, respectively. Although the prick skin test with *Aspergillus* mix (*A. fumigatus*, *A. nidulans*, and *A. niger*) from Stallergenes France was negative, the serum IgE specific to *Aspergillus* spp was elevated (38 LU). The contrast-enhanced high-resolution chest computed tomography on hospital

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**Fig. 1.** Chest X rays (CXR) in the first course of presumed bacterial pneumonia. A) CXR at four months before admission showed right perihilar airspace opacities. B) CXR at one month before admission.



**Fig. 2.** Chest X rays (CXR) related to the second course of presumed bacterial pneumonia. A) CXR on admission showed bilateral perihilar airspace opacities. B) CXR at one month after treatment with high-dose inhaled corticosteroids.

day +6 showed scattered nodules with halo signs and no sign of bronchiectasis (Fig. 3). Bronchoscopy showed mucous plugs occluding the central bronchi, which contained infiltrated eosinophils (Fig. 4). Bronchoalveolar lavage (BAL) showed that 30% of cell count was eosinophil. The BAL fluid for AFB testing was negative but its culture was positive for *Aspergillus spp.* Thus, the diagnosis of ABPA-S was established based on the aforementioned clues and a spirometry result suitable to diagnosis of asthma. On management of ABPA-S, we used ICS (salmeterol/fluticasone 25/250 with dose of fluticasone 1000 microgram per day). After one month, the blood eosinophil cell count was  $206/\text{mm}^3$  and his CXR showed disappearance of airspace opacities (Fig. 2B). He achieved remission at one-year follow-up.

### 3. Discussion

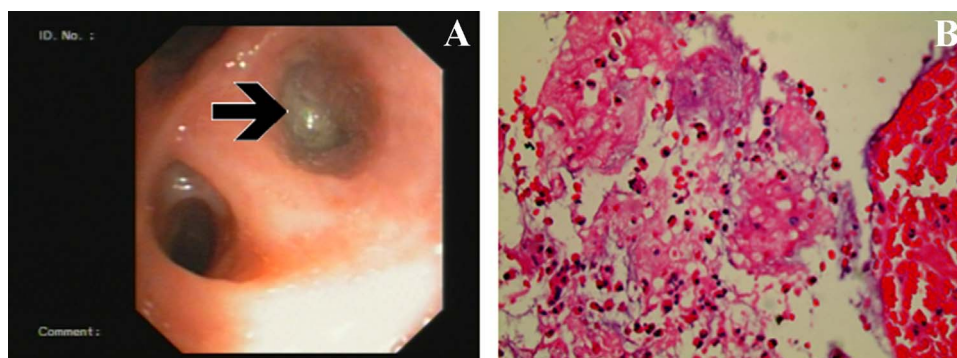
Two differential diagnoses of ABPA include bacterial pneumonia [2] and pulmonary tuberculosis [3,4] which should be given a great caution because of the high prevalence in Vietnam. In this case, diagnosis of bacterial pneumonia four months ago and on admission was presumed because the features were as follows: (1) clinical and radiographic response with antibiotic therapy,

(2) the sputum isolate with *Klebsiella pneumoniae* at the concentration  $2.10^6$  cfu/ml, and (3) new infiltrates on CXR. Recurrent pneumonia with fleeting pulmonary opacities on CXR and blood eosinophilia were the main clues that urged us to investigate further. With exception of the negative skin prick test, diagnostic criteria of ABPA-S were met in this case. Several factors that include the technical expertise in performing the test, the component of allergen extracts, and the patient's age can affect the outcome of the skin prick test [5]. The common incriminating species of *Aspergillus* are *A. fumigatus*, *A. flavus*, *A. niger*, *A. terreus*, and *A. nidulans* [6] while the *Aspergillus* mix applied to this patient only composed of three species. These can lead to the discordant result between the skin prick test and the serum test.

In 2011 Agarwal et al. concluded that high-dose ICS showed no role in management of ABPA-S [7]. However, several published case reports showed the usefulness of high-dose ICS [8,9]. We decided on treating ABPA-S with high-dose ICS (fluticasone propionate) because the patient's past medical history was hypertension and type 2 diabetes mellitus, which in turn could be influenced seriously when using systemic corticosteroid. During follow-up, improvement of clinical symptoms, disappeared lesions on CXR and normal eosinophil count proved patient's response to ICS treatment, although serum level of total IgE was not re-



**Fig. 3.** High-resolution computed tomography of the chest revealed scattered nodules with halo sign (yellow arrow) and finger in glove sign (red arrow) (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).



**Fig. 4.** A) Bronchoscopy showed mucus plug (black arrow). B) Hematoxylin and Eosin staining revealed the infiltration of eosinophils in mucus plug. Image was captured at 100 × magnification. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

measured. This patient was stable during follow up until 12 months later. This emphasises the possible therapy choice of high-dose ICS in ABPA-S patients with high risk of systemic corticosteroid and the closely follow-up of these patients should be considered.

#### Conflict of interest

There are none.

#### Acknowledgements

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