Case Report

Allergic bronchopulmonary aspergillosis presenting with cough variant asthma with spontaneous remission

Hirofumi Matsuoka*, Towa Uzu, Midori Koyama, Yasuko Koma, Kensuke Fukumitsu, Yoshitaka Kasai, Daiki Masuya, Harukazu Yoshimatsu, Yujiro Suzuki

Department of Respiratory Center, Shinko Hospital, 1-4-47, Wakihama, Chuo-ku, Kobe 651-0072, Japan

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ABSTRACT

A 60-year-old woman presented with a dry cough without dyspnea or wheezing. Chest CT showed an image of mucoid impactions, which were identified as mucoid impactions by bronchofiberscopy. Aspergillus niger was cultured from her mucus. Her serum total IgE was 5150 IU/ml. Precipitins and IgE specific for Aspergillus were positive. She had no history of asthma and no evidence of bronchoconstriction by pulmonary function tests. Thus, a diagnosis was made of allergic bronchopulmonary aspergillosis without asthma. She refused to take oral corticosteroids, although she improved spontaneously. However, her dry cough persisted. Her cough was relieved by administering an inhaled β2 agonist; therefore, cough variant asthma was diagnosed. She was treated with an inhaled corticosteroid and her cough resolved completely.

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1. Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is an allergic pulmonary disorder caused by a hypersensitivity reaction to antigens of Aspergillus species. In general, an oral steroid is necessary to treat ABPA. Bronchial asthma has been considered an essential diagnostic criterion for ABPA. However, several cases without bronchial asthma have been reported. We report a case of ABPA presenting with cough variant asthma with spontaneous remission.

2. Case report

A 60-year-old woman presented with a 2-week history of dry cough without dyspnea or wheezing. She was a housewife, a non-smoker, and had no history of childhood asthma. She was afebrile and her chest was clear on auscultation. A chest radiograph showed bilateral infiltrates (Fig. 1). Laboratory findings showed a total leukocyte count of 8200 cells/mm³ with 65.4% neutrophils and 3.0% eosinophils, and a C-reactive protein concentration of 2.51 mg/dl. She was diagnosed with pneumonia and administered azithromycin.

One week later, she was afebrile and her chest radiograph showed improved infiltrates on chest radiography. A chest CT showed bilateral infiltrates (Fig. 2a). Laboratory findings showed a total leukocyte count of 8200 cells/mm³ with 65.4% neutrophils and 3.0% eosinophils, and a C-reactive protein concentration of 2.51 mg/dl. She was diagnosed with pneumonia and administered azithromycin.

Aspergillus niger was cultured from mucus aspirated by bronchofiberscopy. Her serum total IgE was 5150 IU/ml. IgE specific for Aspergillus was positive by ELISA. Precipitins to Aspergillus were also positive. Spirometry was normal with an FVC of 2.22 L (94% of predicted), FEV₁ of 2.03 L (103% of predicted), FEV₁/FVC ratio of 91%, and no changes after administering a bronchodilator. Peak expiratory flow variability was 6%. A provisional diagnosis of ABPA without asthma was made.

She refused recommendations to take oral corticosteroids; however, the infiltrates seen on chest radiograph gradually improved without the use of oral corticosteroids. Two months later, her serum IgE decreased to 2670 IU/ml, which was considered as a state of remission. Chest CT showed bronchiectasis in the lingula of the left lung, and the images of mucoid impaction had disappeared (Fig. 2b). However, her dry cough persisted. Her exhaled nitric oxide (ENO) was 77 ppb, and her cough was relieved by administering an inhaled β2 agonist; therefore, cough variant asthma was diagnosed. She was treated with an inhaled corticosteroid and her cough resolved completely.

3. Discussion

ABPA is an allergic pulmonary disorder caused by a hypersensitivity reaction to antigens of Aspergillus species. In general, an oral steroid is necessary to treat ABPA. Bronchial asthma has been considered an essential diagnostic criterion for ABPA. However,
several cases without bronchial asthma have been reported. To our knowledge, this is the first case of ABPA presenting with cough variant asthma that showed spontaneous remission without the use of oral corticosteroids.

Cough variant asthma is an asthma phenotype that presents solely with coughing. It involves airway eosinophilic inflammation, as does classic asthma with wheezing. About 30% of these patients may progress to classic asthma, which can be prevented by treatment with an inhaled corticosteroid. A diagnosis of cough variant asthma can be made when all of the following are present: (i) cough without wheezing lasting 8 weeks (or 3 weeks or more) and no wheezing heard on auscultation; and (ii) relief of cough with bronchodilator therapy. All of these criteria were satisfied in the present case. In addition, in the present case, ENO was elevated (77 ppb). ENO reflects airway eosinophilic inflammation and is useful for differentiating cough variant asthma from other causes of chronic cough.

Several cases of ABPA without bronchial asthma have been reported. However, in those reports, some patients developed bronchial asthma after the initial diagnosis. In those cases, elevated sputum eosinophils or increased airway responsiveness were found in some patients, which suggested that cough variant asthma patients might have been included. Oral steroid use can mask the cough due to cough variant asthma. In ABPA patients with cough variant asthma, cough variant asthma may progress to classic asthma after stopping or during the tapering of oral corticosteroids. Thus, in the case of ABPA without asthma, further examination should be performed to rule out cough variant asthma. And, if cough variant asthma is present, treatment with an inhaled corticosteroid should be considered.

In general, an oral steroid is necessary to treat ABPA; however, the present case improved spontaneously without using an oral steroid. She is being treated now only with an inhaled corticosteroid and has had no relapse. In this case, the peripheral eosinophil count did not increase, which might indicate a mild form of ABPA. Some reports have suggested that an inhaled corticosteroid could achieve adequate control of ABPA. However, relapse can occur. Thus, these cases should be closely followed and treated with oral steroids, as required.
In conclusion, in the case of ABPA without asthma, further examination should be performed to rule out cough variant asthma. Mild form of ABPA may improve without oral steroid therapy.

**Conflict of interest**

The authors have no conflict of interest.

**References**