CASE REPORTS

Allergic bronchopulmonary aspergillosis mimicking pulmonary Tuberculosis

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Allergic bronchopulmonary aspergillosis (ABPA) is an uncommon disease with variable prevalence of 5 to 15%.1-7 It is found mainly in patients with asthma and cystic fibrosis.1-7 It results from exposure of the asthmatic bronchial tree to Aspergillus Fumigatus (AF). This may lead to hypersensitivity reaction involving eosinophilic infiltration of the bronchial wall and mucoid impaction or granulomatous inflammation.8.9 Repeated episodes of bronchial obstruction, inflammation and mucoid impaction can lead to bronchiectasis, fibrosis and respiratory impairment.10 Patients with ABPA usually have long standing asthma and present with recurrent attacks of fever, malaise, hemoptysis, bronchial obstruction, expectoration of brownish mucous plugs and peripheral eosinophilia.11,12 Therefore, with such a clinical presentation, and in the presence of radiological cavitating lesions due to bronchiectasis and as pulmonary tuberculosis is much more common than ABPA, some patients with ABPA have been misdiagnosed and treated as pulmonary tuberculosis. In one series from India almost one 3rd of the 35 patients with ABPA were misdiagnosed as pulmonary tuberculosis and treated with antituberculous therapy for varying periods of time.13 The aim of this study is to report 2 cases of ABPA misdiagnosed and treated as pulmonary tuberculosis and to define the major differences between these 2 conditions.

Case Report. ***Patient 1.*** A 42-year-old housewife was diagnosed to have pulmonary tuberculosis in April 1997. Since then she was placed on antituberculous therapy until her presentation at our institution. At that time, Ziehl-Neelsen staining for acid fast bacilli (AFB) in sputum and bronchial lavage were negative as well as her tuberculin test. The diagnosis of pulmonary tuberculosis was based mainly on radiological findings. She was a known asthmatic for 20 years. Her asthma was fairly controlled by beclomethasone and salbutamol inhalation, except in the past 2 years where her asthma was worse and she became more symptomatic. She was referred to our institution in July 1997 due to poor control of asthma and failure of antituberculous therapy. She presented with cough, shortness of breath, greenish sputum, wheezes and fever. On examination, she looked pale and underweight. Her height was 156 cm, weight was 38 kg, pulse rate was 85 beat/min. Blood pressure was 110/80 mmHg. Temperature was 38.5OC. Peak expiratory flow rate (PEFR) was 180l/min. She had no cyanosis or clubbing of the fingers. She had no palpable lymph nodes. Examinations of the head and neck were normal. Examination of cardiovascular system (CVS) was normal. Examination of the chest showed harsh vesicular breathing with prolonged expiration, occasional scattered crackles and rhonchi. Examinations of the abdomen and central nervous system (CNS) were unremarkable. Chest radiograph on presentation (Figure 1) showed chronic lung changes of both upper lobes with linear strands and loss of volume. The left upper lobe also showed ill defined opacity with irregular thickened wall cavity. There was also multiple different sized and shape cavities scattered on both lung fields. The radiological diagnosis (reported by 2 senior

radiologists) was consistent with an active tuberculosis. She was hospitalized and underwent the following investigations: WBC 11 x103 per mm3, Hb 12.2 g/dl, Eosinophils count 1.2x103cells/mm3 (n=00-0.73x103 cell/mm3), ESR 92. The high peripheral eosinophilic count in this lady has raised the possibility of pulmonary eosinophilia in which ABPA was among the differential diagnosis. So immunoglobulin E (IgE) level was measured and surprisingly it was very high 17802 IU/ml (n=0-195 IU/ml). Specific IgE was highly positive for AF as well as

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