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Case Report Open Access

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Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is a most frequently recognised manifestation of pulmonary aspergillosis. Being an immunologically mediated disease, it usually occurs in atopic individuals and is caused by hypersensitivity reaction to aspergillus fumigatus. Patients with chronic obstructive pulmonary disease (COPD) have mucus hypersecretion and may favour occurrence of ABPA. However, ABPA is rarely reported in other lung disorders including COPD. Here we are reporting a case of COPD complicated by ABPA.

Case Report

A 52-year old ex-smoker (pack year 15) was diagnosed to have chronic obstructive pulmonary disease (COPD) 4 years back. Diagnosis of COPD was considered based on symptoms of cough and breathlessness with a history of exposure to risk factor and con rmed on spirometry (post-bronchodilator FEV1/FVC <0.7 and post-bronchodilator FEV1 59% without reversibility). His symptoms were well controlled on inhaled bronchodilators since the time of diagnosis. He came to the outpatient of chest department with the complaint of worsening cough and breathlessness of 2 weeks duration. ere was no history of fever, anorexia and weight loss. He was having history of repeated exacerbation in the past 3 months with response to steroids. Examination revealed respiratory rate of 24 breaths per minute. Chest auscultation revealed generalised decrease in breath sounds, prolonged expiratory phase and ronchi. Rest of the examination was unremarkable. Saturation on room air was 94%. He was initially managed with antibiotics, theophylline, inhaled steroids and bronchodilators. But he had one episode of haemoptysis of moderate amount. He was evaluated further for the aetiology of haemoptysis.

Chest radiograph was normal (Figure 1). Sputum for acid fast bacilli was also negative for three consecutive days. Computed tomography (CT) thorax was done as a work-up of haemoptysis. CT thorax showed bilateral central bronchiectasis. Haematological and biochemistry investigation were normal except absolute eosinophil counts of 2850 cells/µl. Skin testing was done to exclude the diagnosis of allergic bronchopulmonary aspergillosis (ABPA). Both skin prick tests (type 1 and type 3) were positive. Subsequently total serum IgE was also found to be elevated at 1636 IU/ml (normal 1-87 IU/ml). Aspergillus speci c IgE and IgG were 19 kU/l (normal 0-0.35 kU/l) and 28.2 (normal

cause marked airway in ammation. e epithelial damage results in di usion of soluble antigen and mycelial fragments into interstitium with further release of in ammatory mediators, and in ux of in ammatory cells [5]. e antigens are also presented to 2 cells which lead to total and A. fumigatus speci c IgE synthesis, mast cell degranulation and promotion of a strong eosinophilic response. e in ammatory cells lead to tissue injury and characteristic pathology of ABPA [5].

Patients with COPD have mucus hypersecretion and impaired mucociliary clearance which may predispose these patients to the colonisation of aspergillus fumigatus resulting in development of ABPA. Till date only one such case was reported from Chandigarh [6]. A case control study from the same place also showed high probability of aspergillus hypersensitivity/ABPA in patients of COPD [7].

Conclusion

ABPA can also complicate the patients with COPD and besides bronchial asthma or cystic brosis; COPD can also be taken as major criteria for the diagnosis of ABPA.