

Case Report

Co-existence of Bronchiectasis and Chronic Obstructive Pulmonary Disease

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Abstract

We present the case of a 56-year-old male who presented with cough and breathlessness. Chest radiograph (postero-anterior view) showed bulla and signs of hyperinflation in the right upper zone with cystic shadows in the left lower zone. Spirometry showed severe irreversible airflow obstruction with restriction and decreased diffusion capacity for carbon monoxide. On high resolution computed tomography (HRCT), right upper lobe bulla with emphysematous changes and left lower lobe cystic bronchiectasis were seen. Considering its rarity of occurrence and paucity of data in literature regarding co-existence of bronchiectasis with emphysema, this case is being reported. [Indian J Chest Dis Allied Sci 2015;57:125-127]

Key words: COPD, Bronchiectasis, Exacerbation.

Introduction

Chronic obstructive pulmonary disease (COPD) and bronchiectasis are two different but related diseases that occur separately, but can co-exist, although this association is even more uncommon. We report a case of middle-aged male who presented with a clinical picture suggestive of bronchiectasis in whom radiographic evaluation revealed right upper lobe bulla with emphysematous changes with left lower lobe cystic bronchiectasis. Considering its rarity of occurrence and paucity of data in literature regarding co-existence of bronchiectasis with emphysema, this case is being reported.

Case Report

A 56-year-old male shopkeeper, presented with productive cough for the last 20 years. He also reported exertional dyspnoea for last two years and fever on and off for the last two years. He denied any history of chest pain, loss of weight and appetite. He gave a history of 25 pack years of smoking *bidi*. He quit smoking two years back. He also gave a history of episodes of acute worsening of symptoms 2-3 times per year for the last two years and had received treatment with inhaled corticosteroids and bronchodilators for the same. General physical examination revealed clubbing of fingers. Respiratory system examination revealed vesicular breath sounds with prolonged expiration and coarse crepitations in the left infra-scapular region. Laboratory testing including blood counts, serum biochemistry and urine analysis were within normal limits. Serological testing for human immunodeficiency virus (HIV) was negative. Alpha-1 antitrypsin level

was measured to be 160 mg/dL that was within normal range (100-300 mg/dL). The chest radiograph (postero-anterior view; Figure 1) and high resolution computed tomography (HRCT) of the chest in the axial plane (Figures 2A and 2B) showed a bulla in the right upper zone, signs of hyperinflation and cystic shadows in the left lower zone. Sputum was negative for acid-fast bacilli (AFB) and fungi.

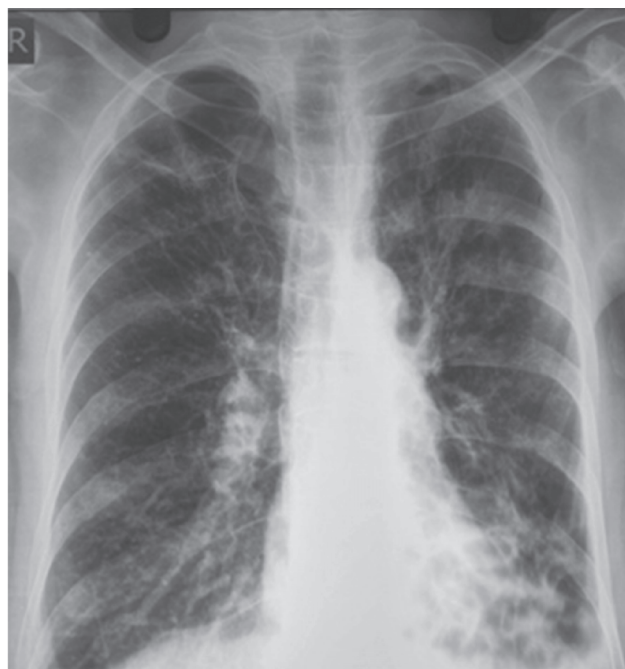


Figure 1. Chest radiograph (postero-anterior view) showing a bulla, signs of hyperinflation in the right upper zone and cystic shadows in the left lower zone.

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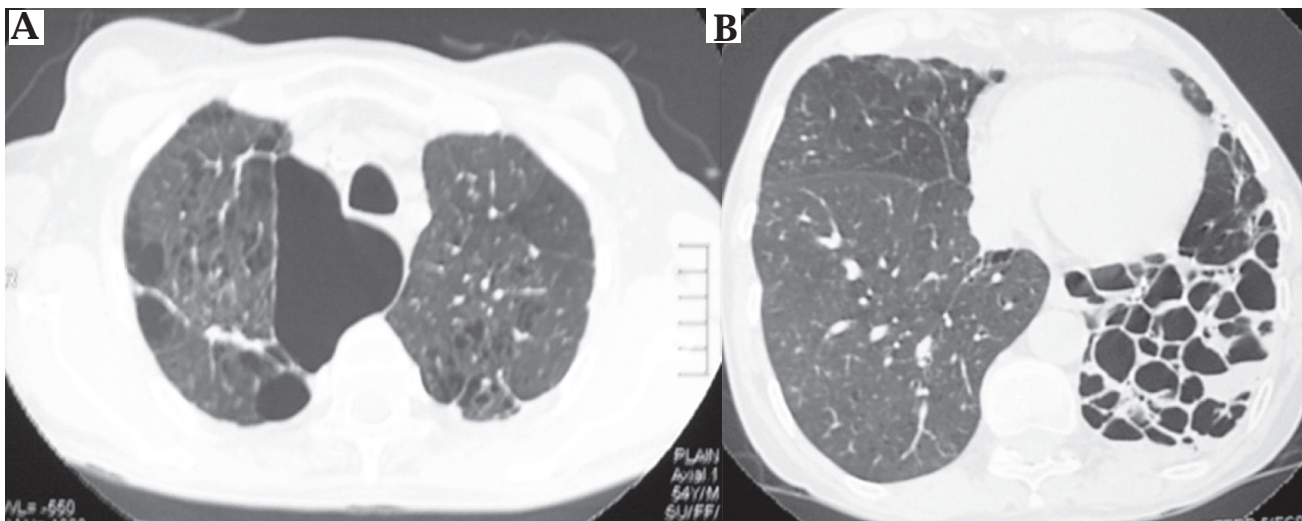


Figure 2. HRCT chest of the same patient showing (A) right upper lobe bulla with emphysematous changes and (B) left lower lobe cystic bronchiectasis.

Sputum bacterial culture grew *Pseudomonas aeruginosa* sensitive to amoxicillin-clavulanic acid, cefoperazone-sulbactam, piperacillin-tazobactam, ciprofloxacin, levofloxacin, amikacin and gentamicin. Spirometry showed severe irreversible airflow obstruction with restriction and decreased diffusion capacity for carbon monoxide as per international guidelines.¹ A diagnosis of left lower lobe bronchiectasis with COPD was made on the basis of clinical, radiologic and spirometric findings. The patient was treated with a combination of intravenous antibiotics (piperacillin-tazobactam and amikacin) for two weeks based on culture sensitivity report and inhaled bronchodilators and corticosteroids were continued. The patient was advised long-term antibiotics (oral azithromycin 500 mg for 7 days, 250 mg for next 7 days followed by 250 mg thrice weekly for next 3 months), postural drainage and physical rehabilitation on discharge.^{2,3}

Discussion

Bronchiectasis is an abnormal permanent dilatation of bronchi and bronchioles due to repeated cycles of airway infection and necrotising inflammation.⁴ Considered to be an orphan disease in the developed world in the late twentieth century, bronchiectasis is now being diagnosed with increasing frequency around the globe.⁵ Regardless of the underlying cause, bronchiectasis results when inflammatory and infectious damage to the bronchial and bronchiolar walls leads to a vicious cycle of airway injury.⁶ Establishing the cause of bronchiectasis may be difficult. Almost 50% to 80% of cases of bronchiectasis remain to be classified as of unknown aetiology/ idiopathic, even after clinical, laboratory and pathologic testing.⁷ The HRCT is now accepted as the imaging modality of choice for the evaluation of both bronchiectasis⁸ and emphysema.⁹ Due to increasing use of HRCT scanning in patients with pulmonary symptoms, bronchiectasis is more frequently

being recognised in patients with chronic cough and dyspnoea. Radiographic features may include cylindrical, varicose and cystic/saccular type of bronchiectasis. Overt bullous emphysema is seldom reported in bronchiectasis, except in patients with alpha-1-antitrypsin deficiency (AAT).¹⁰ The computed tomography (CT) finding of widespread areas of decreased attenuation in bronchiectasis as considered to be suggestive of emphysema.¹¹ In the present case, CT findings of bullous emphysema could not be explained solely by bronchiectasis. The prevalence of bronchiectasis in patients with moderate to severe COPD has been reported to be 47.8% and it was primarily of cylindrical type and mainly localised in the lower lobes.¹² In another study¹³, a higher prevalence of bronchiectasis was described in the lobes most affected by emphysema. In contrast to this, bulla with emphysematous changes in right upper lobe and bronchiectasis in the left lower lobe were observed in the present case. Hence, the two diseases, i.e. COPD and bronchiectasis, can be considered to be of separate origin co-existing together in the present case. Also, in the present report bronchiectasis was primarily of cystic type and not cylindrical as reported in previous studies of COPD leading to bronchiectasis.¹² The AAT deficiency can be associated with both emphysema and bronchiectasis but AAT levels were normal in our patient. Bronchiectasis can cause airflow obstruction that produces clinical symptoms similar to COPD. Patients with COPD and bronchiectasis have greater bronchial inflammation, chronic colonisation of bronchial mucosa by a pathogenic micro-organism and longer duration of acute infectious exacerbations.¹⁴ *Pseudomonas aeruginosa* is the most frequent pathogen in COPD patients associated with bronchiectasis that was also the scenario in present report.¹⁵ The goals of bronchiectasis treatment are to reduce the number of exacerbations and to improve quality of life. Regular follow-up, postural drainage and

physical rehabilitation are key components of management.

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