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Case Report

The hidden airway: central airway collapse mimicking obstructive lung disease

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ABSTRACT

Central airway collapse is an underrecognized cause of respiratory failure and is often misdiagnosed as Chronic Obstructive Pulmonary Disease or Bronchial asthma. Traditionally central airway collapse was attributed to tracheomalacia (TM), it is now increasingly differentiated from excessive dynamic airway collapse (EDAC). Excessive dynamic airway collapse (EDAC) is an underrecognized cause of chronic respiratory symptoms and is frequently presents with clinical manifestations such as cough, wheezing and exertional dyspnoea. EDAC is characterized by excessive inward bulging of the posterior membranous wall of the trachea during expiration, resulting in marked airway narrowing despite preserved tracheal cartilaginous support. Recent advances in diagnostic modalities, particularly dynamic computed tomography and fiberoptic bronchoscopy, have improved the recognition of this condition and demonstrated its occurrence in both symptomatic and asymptomatic individuals. We report the case of an 81-year-old woman who had a significant history of occupational exposure to stone dust and was an ex-smoker presented with a 10-year history of chronic cough and progressive exertional dyspnoea. Further evaluation established the diagnosis of EDAC. The patient demonstrated marked symptomatic improvement following initiation of bilevel positive airway pressure therapy along with bronchodilator treatment. This case highlights the importance of considering EDAC in patients with persistent respiratory symptoms despite standard treatment.

Keywords: Central airway collapse, Bronchial asthma, EDAC

INTRODUCTION

Central airway collapse is an underrecognized yet clinically significant cause of respiratory morbidity, often misdiagnosed as Chronic Obstructive Pulmonary Disease or Bronchial asthma due to overlapping clinical features such as exertional dyspnoea, cough and wheezing.[1,2] Traditionally, this entity has been attributed to tracheomalacia (TM), characterized by structural weakness of the tracheal cartilaginous rings leading to airway collapse.[3] However, increasing evidence has identified excessive dynamic airway collapse (EDAC) as a distinct pathological condition with different underlying mechanisms. Unlike TM, EDAC is defined by excessive inward displacement of the posterior membranous wall of the trachea during expiration, resulting in significant luminal narrowing despite preserved cartilaginous support.[3]

This dynamic process is accentuated by increased intrathoracic pressure during exhalation and may contribute to airflow limitation, impaired mucociliary clearance and recurrent respiratory infections. Although varying degrees of expiratory airway narrowing may be

observed in healthy individuals, clinically significant EDAC can lead to persistent respiratory symptoms and in severe cases, respiratory failure.[4] The true prevalence of EDAC remains uncertain, partly due to under recognition and frequent coexistence with other chronic airway diseases. With advances in diagnostic techniques, particularly dynamic computed tomography and flexible bronchoscopy, EDAC is increasingly being identified in patients with unexplained or refractory respiratory symptoms.

Recognition of this condition is crucial, as it has important implications for management, including the use of noninvasive positive pressure ventilation and other targeted interventions. In this report, we describe a case of EDAC in an elderly patient with long-standing respiratory symptoms, highlighting the diagnostic challenges, pathophysiological considerations and therapeutic implications of this often-overlooked condition.

CASE REPORT

An 81-year-old woman presented with acute worsening of cough and breathlessness for one week, accompanied by intermittent high-grade fever with chills for three days. She had a 10-year history of progressive exertional dyspnoea.

The patient also reported a chronic cough of 10 years duration, initially associated with scanty sputum and predominantly nocturnal, which later became mainly dry. She had experienced five previous hospital admissions over the past decade due to exacerbations of cough and breathlessness.

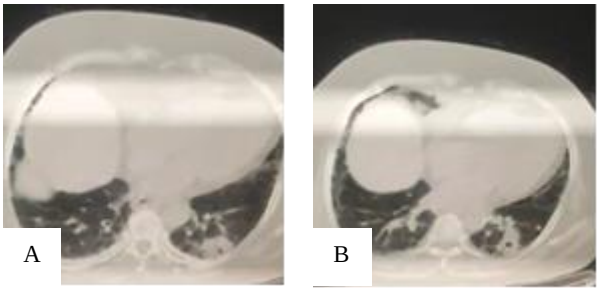

Figure 1(A and B): Peripheral subpleural fibrotic changes with basal predominance in both lungs. Mosaic attenuation with air trapping also seen bilaterally- interstitial lung disease possibilities include fibrotic HP/early UIP (usual interstitial pneumonia).

Her symptoms during the time of admission included scanty sputum production and worsening breathlessness, particularly in the evening and night, aggravated by the supine position and partially relieved by sitting upright and inhaled bronchodilator therapy (budesonide-formoterol). She denied hemoptysis, chest pain, palpitations, syncope, pedal edema, night sweats or significant weight loss.

Her medical history was significant for type 2 diabetes mellitus (3 years) and hypertension (6 years) on regular treatment. She had a smoking history of 7.5 pack-years (5 beedis/day for 30 years) but had quit 20 years ago, and a history of tobacco chewing from the age of 20 years, discontinued 5 years ago. Notably, she had worked for 50 years in a stone-cutting factory without proper respiratory protection, suggesting significant occupational dust exposure.

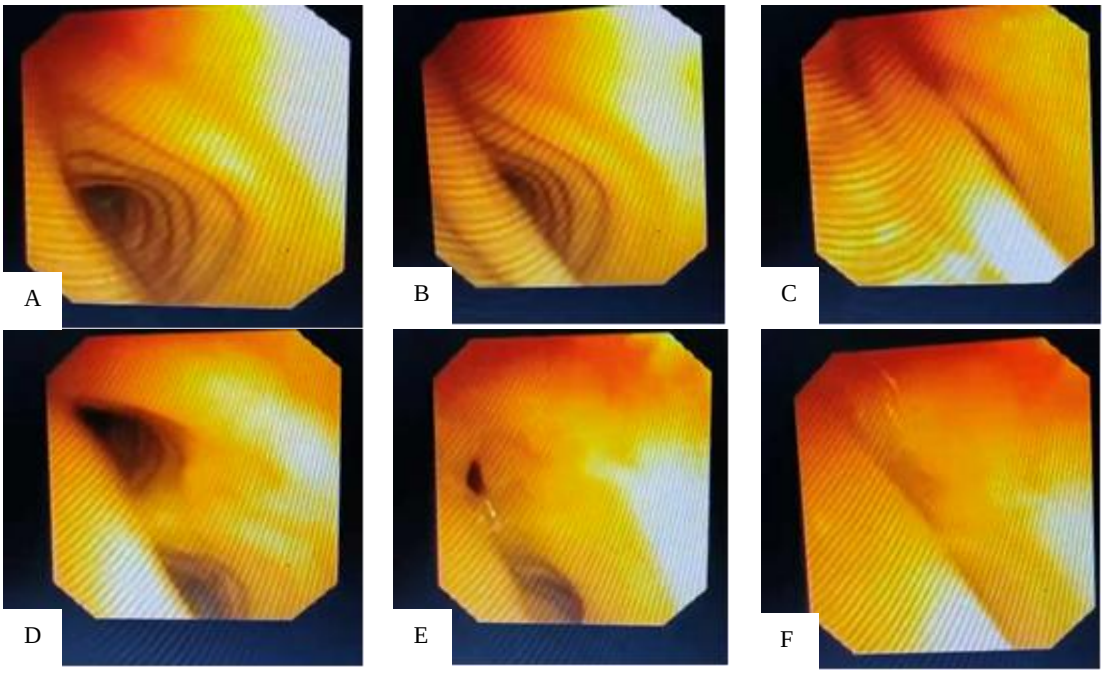

Figure 2 (A-F): Bronchoscopy: excessive narrowing of tracheobronchial tree. Suggestive of excessive dynamic airway collapse.

On examination, the patient was conscious and oriented, with a body mass index of 27 kg/m². Bilateral digital clubbing was noted. Respiratory examination revealed tachypnea with the use of accessory muscles of respiration. Percussion notes were dull over the bilateral infrascapular regions. Auscultation demonstrated reduced intensity of breath sounds in the inframammary, intraaxillary and intrascapular areas. Fine end-inspiratory "velcro" crackles were heard bilaterally over the infraaxillary and infrascapular regions. Vocal resonance was equal and normally heard in all lung fields.

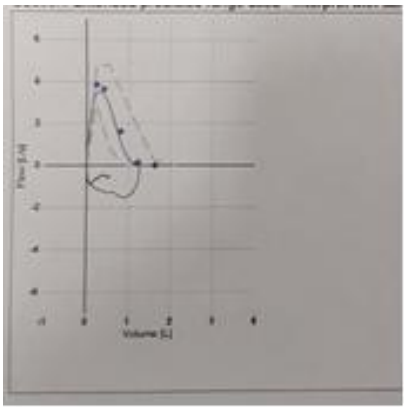

Figure 3: PFT: normal pattern.

Baseline laboratory investigations revealed mild anemia with normal leukocyte and platelet counts. Renal function tests were within normal limits. Liver function tests showed mild transaminase elevation. Serum electrolytes demonstrated mild hyponatremia and inflammatory markers were mildly elevated. Sputum microscopy was unremarkable; however, culture grew only normal upper respiratory tract flora. Evaluation for pulmonary tuberculosis including acid-fast bacilli smear and CBNAAT was negative. Bronchial washing microscopy was unremarkable, while aerobic culture yielded growth of Elizabethkingia meningoseptica. Rheumatoid factor was within normal limits (4.6 IU/ml), and D-dimer levels were normal. Two-dimensional echocardiography demonstrated no evidence of pulmonary artery hypertension. Inspiratory and expiratory chest CT imaging demonstrated excessive bowing of the posterior tracheal wall during expiration, resulting in greater than 50% reduction in luminal caliber, in the background of interstitial lung disease. Flexible bronchoscopy revealed marked narrowing of the tracheobronchial tree during expiration, consistent with excessive dynamic airway collapse (EDAC), correlating well with the radiological findings. The patient was initiated on bilevel positive airway pressure (BiPAP) along with bronchodilator therapy, following which she showed significant clinical improvement on follow-up.

DISCUSSION

EDAC is increasingly recognized causes of airway obstruction. The reported prevalence ranges from 4%-23% among patients undergoing bronchoscopy, while EDAC has been described in nearly 22% of patients with chronic obstructive pulmonary disease (COPD) and/or asthma.[5,6] Despite this, EDAC remains underdiagnosed, as its symptoms frequently overlap with those of coexisting airway diseases such as COPD or asthma.[7,8] Consequently, the diagnosis is often made incidentally during bronchoscopy or computed tomography (CT) performed for other clinical indications.[5]

The underlying mechanism of EDAC involves laxity and inward bulging of the posterior tracheal membrane during expiration.[6-9] Loring et al suggested that repeated mechanical stress from chronic coughing or increased expiratory pleural pressure in patients with airway obstruction may lead to progressive stretching and degeneration of the posterior membranous wall.[4] In the present case, the patient's long-standing history of chronic cough may have contributed to the development of posterior membrane laxity and dynamic airway collapse.

Although EDAC may remain asymptomatic, severe cases can present with persistent dry cough, dyspnea, recurrent respiratory infections due to impaired mucus clearance, and respiratory failure. Patients often exhibit wheezing that is poorly responsive to bronchodilators and corticosteroids. Impaired secretion clearance may also result in mucus plugging and bronchiectasis.

Dynamic bronchoscopy performed during spontaneous breathing remains the gold standard for diagnosis, allowing direct visualization of airway collapse during forced expiration or coughing. Dynamic CT provides a useful noninvasive alternative, whereas pulmonary function tests have limited diagnostic value.

Management depends on symptom severity and the degree of airway collapse. Treatment options include conservative therapy with bronchodilators and noninvasive positive pressure ventilation (NIPPV) with newer strategies like Manometry Optimized Positive Expiratory Pressure (MOPEP), Thermoablative Techniques, minimally invasive interventions such as airway stenting, and surgical procedures in selected cases.[10-12] NIPPV functions as a pneumatic stent, reducing airway resistance and improving airflow. Recognition of EDAC is important in patients with persistent respiratory symptoms despite optimal treatment for asthma or COPD, and such patients should undergo dynamic airway evaluation after exclusion of other common causes of chronic cough.

CONCLUSION

This case underscores EDAC as a critical yet underrecognized contributor to respiratory failure, often masquerading as refractory COPD or asthma. In this 81-year-old with occupational dust exposure and chronic cough, dynamic CT and bronchoscopy confirmed severe EDAC amid ILD, with BiPAP yielding rapid

improvement. Clinicians should prioritize dynamic airway evaluation in persistent dyspnea cases, leveraging NIPPV as a cornerstone therapy to avert exacerbations and enhance outcomes.

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