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# Excessive dynamic airway collapse presenting as intractable cough and wheezing : A case report

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## ABSTRACT

Excessive dynamic airway collapse (EDAC) is a condition characterized by significant airway narrowing, often mistaken for asthma or COPD. This case study highlights a 35-year-old woman with chronic cough and wheezing, resistant to standard treatments. Through bronchoscopy, EDAC (>90%) was diagnosed, exacerbated by a history of pulmonary tuberculosis. Treatment involved serial bronchial dilatation and NIPPV, resulting in symptom relief. EDAC, frequently underdiagnosed, warrants consideration in patients with refractory respiratory symptoms, especially those with past tuberculosis. Dynamic bronchoscopy remains the gold standard for diagnosis, guiding tailored management strategies ranging from conservative to surgical interventions.

**Key Words**: *Excessive dynamic airway collapse (EDAC); Bronchoscopy; Pulmonary tuberculosis; NIPPV (Non-Invasive Positive Pressure Ventilation); Respiratory symptoms.*

## INTRODUCTION

Excessive dynamic airway collapse (EDAC) is defined as the pathological collapse and narrowing of the airway lumen by 50% or more of the sagittal diameter which occurs as a result of laxity of the posterior wall membrane with intact cartilage.[1] Patient is mostly asymptomatic , except at times when dynamic airflow obstruction resulting from the pathological weakening of the airway membrane leading to symptoms akin to dyspnea, orthopnea , cough, wheezing , and sometimes respiratory failure. Symptomatic presentation of EDAC as exertional dyspnea, and postoperative respiratory failure has been mentioned in the literature.[2,3] EDAC presenting as intractable cough or wheezing is not a common phenomenon. Nevertheless, can be one of the common symptoms requiring a pulmonologist expertise in day-to-day practice. There are two classic patterns of collapse: crescentic and circumferential [6]. The crescentic pattern shows marked anterior bulging of the posterior membrane and is also known as the “frown” sign [5]. In the circumferential pattern, the collapse is more evenly distributed [6]. Airway collapse is frequently accompanied by air trapping [5,6].

A 35-year-old woman presented to the OPD with a complaint of intractable cough and chronic wheezing for a duration of around 10 years to the extent of even causing disturbed sleep. Symptoms get aggravated on exposure to dust and with changes in weather conditions. She was evaluated at multiple occasions for similar complaints, mostly diagnosed as Bronchial Asthma, with no relief even from maximum ICS/LABA combinations and on multiple occasions OCS lasting for 7-10 days .Past history of adequately treated pulmonary tuberculosis was also present around 11 years back. Routine investigations done , including pulmonary function test (PFT) [Figure 1a] was suggestive of airway obstruction. Computed tomography (CT) thorax [Figure 1b] showed minimal upper and lower lobe bronchiectasis, especially involving lower lobes. Total IgE level was normal. The patient was treated with steroids and nebulized bronchodilators suspecting hyperactive airway/Bronchial asthma. She had no response to treatment. Bronchoscopy was

done under conscious sedation which showed excessive dynamic expiratory collapse (>90%) of the trachea with stenosis of right main bronchus with almost complete collapse during cough. Serial dilatation of right main bronchus using CRE pulmonary ballon catheter was done and patient was given trial of NIPPV, with chest physiotherapy, and airway clearance techniques which she showed significant improvement in symptoms. She was discharged with advice to continue NIPPV at home. On follow-up, after 1 month, the patient was reported to have significant relief of her symptoms.

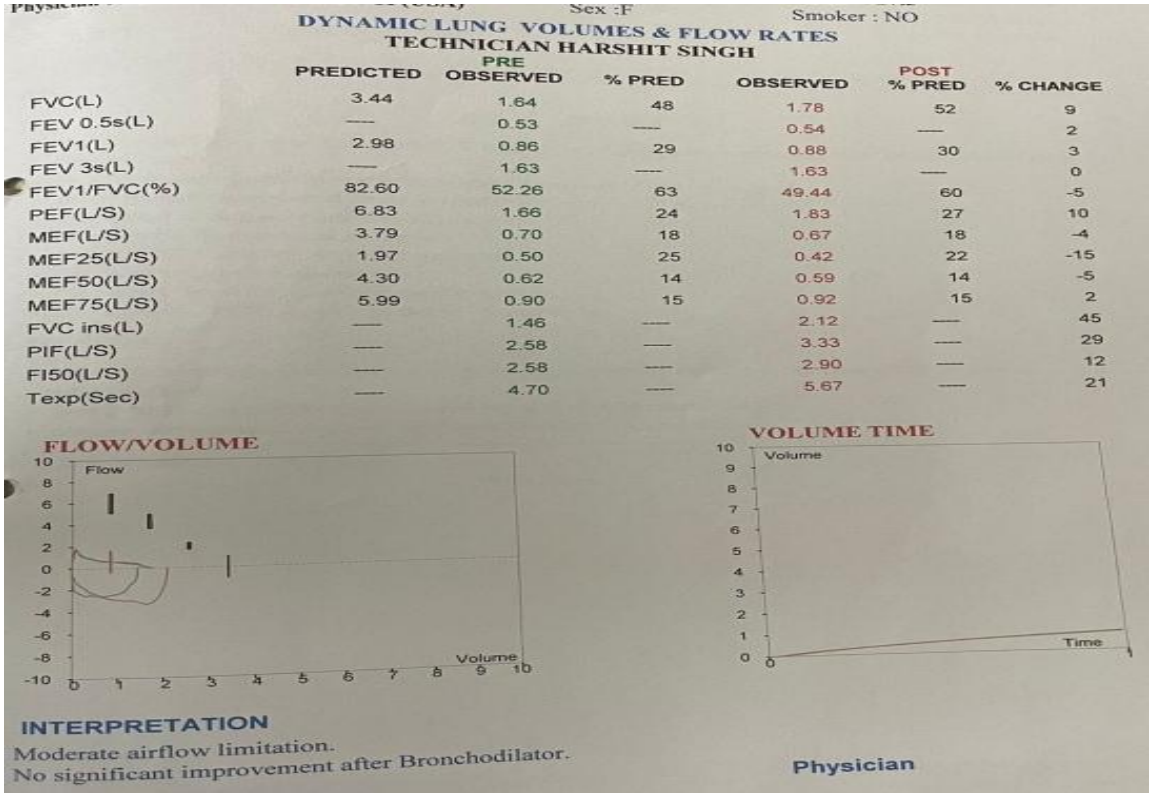
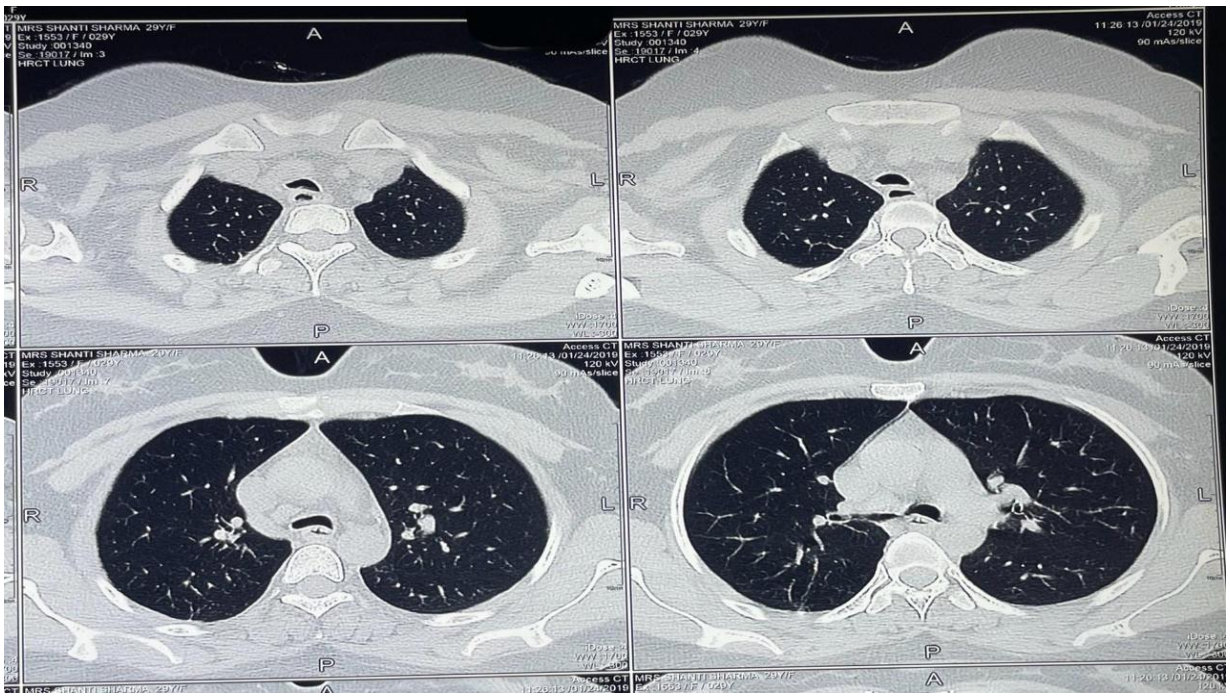
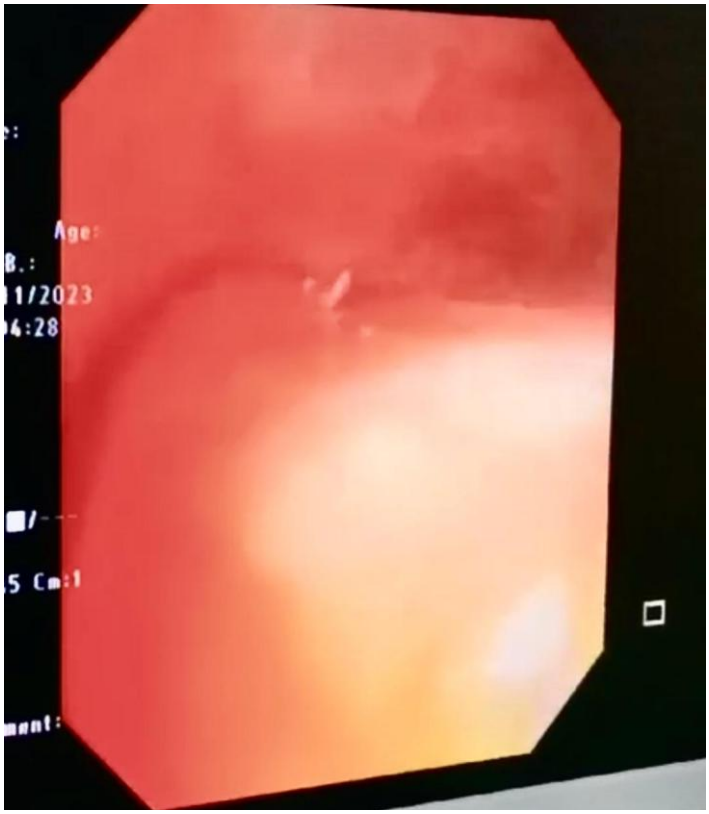
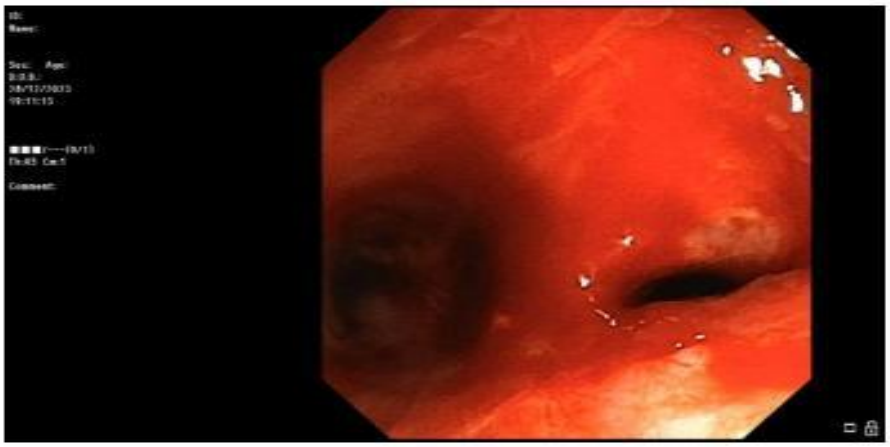


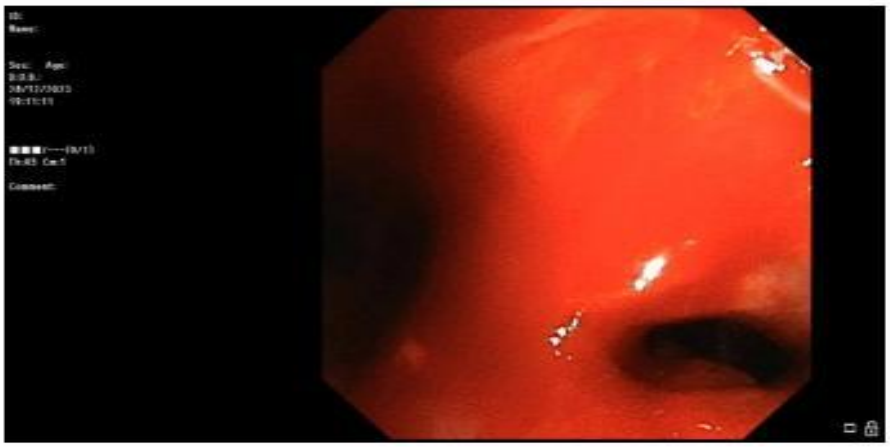
Figure 1 (a) Spirometry showing very severe obstruction with flattening of inspiratory curve on P-V loop


(b) Computed tomography at the level of the upper and lower lobes showing narrowed trachea and both right and left (right>left) main bronchus with some bronchiectasis




(c) Fiber-optic bronchoscopy picture of the trachea showing severe dynamic collapse with the bulging posterior membrane.





(d) Fiber-optic bronchoscopy picture showing stenosed right main bronchus (pre and post CRE dilatation)

The prevalence of EDAC and tracheobronchomalacia (TBM) varies with reported incidence varying from 4% to 23% in patients undergoing bronchoscopy for various reasons.[1] EDAC is supposed to be present in 22% of people with chronic obstructive pulmonary disease (COPD) and/or asthma. It is also found in patients with past history of pulmonary tuberculosis.[7] The disease is mostly underdiagnosed as the symptoms are ascribed to its accompanying pathologies (COPD and asthma), while the actual pathology is accidentally individuated through a bronchoscopy or CT scan performed for other reasons.

Repeated mechanical stretch from coughing or high expiratory pleural pressure during exercise in patients with airway obstruction might cause stretching and degeneration of the posterior membrane over a period of time.[8] Our patient had a history of chronic cough and wheezing , probably secondary to tuberculosis, which would have resulted in the laxity of membrane. EDAC is asymptomatic most of the time. In severe cases, the symptomatology is characterized by dry cough, dyspnea, recurrent airway infections due to difficult expectoration, and respiratory failure. A typical symptom is a wheezing that resists corticosteroid and bronchodilating therapy. EDAC may also be considered in a patient with difficulty to wean from a ventilator. Moreover, patients with EDAC have problem in mobilizing secretions which leads to mucus plugging and eventually may result in bronchiectasis.

Dynamic bronchoscopy done under conscious sedation remains the gold standard for diagnosis of EDAC. Bronchoscopy be preferably performed with a flexible instrument while the patient is spontaneously breathing with the patient being conscious and alert so that he/she can follow the instructions of deep breathing, forced expiration, and coughing that increase the likelihood of dynamic collapse of the airways. Alternatively dynamic CT can be used as the non invasive method of diagnosis. PFT is of less diagnostic value.

Treatment for EDAC depends on the severity of symptoms and the extent of airway collapse. Patients who are asymptomatic do not require treatment. Proposed management strategies include conservative methods such as bronchodilators and NIPPV, minimally invasive therapy like endoluminal airway stents and laser therapy, which have been subsequently planned in the described case, and surgical methods such as tracheostomy, airway splinting, and tracheal resection.[8] NIPPV acts as a pneumatic stent, thereby decreasing airway resistance and improving airflow. Many times, the response to NIPPV therapy prompts the possibility of EDAC.

To conclude, the presence of EDAC should be considered in patients with asthma and COPD not responding to standardized treatment and in patients with known history of tuberculosis in past. Such patient should be considered for a dynamic CT/fiber-optic bronchoscopy under conscious sedation. A peep into the airway is necessary when evaluating a patient with cough but only after ruling out other common possibilities such as sinusitis, gastroesophageal reflux disease, nonasthmatic eosinophilic bronchitis, and asthma.

**Discussion:**

Excessive dynamic airway collapse (EDAC) presents a diagnostic challenge due to its varied symptomatic manifestations and frequent association with other respiratory pathologies like asthma, COPD, and tuberculosis. This case underscores the importance of considering EDAC in patients with refractory respiratory symptoms, particularly in those with a history of pulmonary tuberculosis. The classic crescentic and circumferential collapse patterns aid in the recognition of EDAC, often confirmed through dynamic bronchoscopy, the gold standard for diagnosis. However, dynamic CT serves as a non-invasive alternative for diagnosis.

Treatment strategies for EDAC depend on symptom severity and airway collapse extent. Conservative measures like bronchodilators and NIPPV are beneficial, with potential consideration for minimally invasive or surgical interventions in severe cases. NIPPV emerges as a promising therapy, acting as a pneumatic stent to alleviate airway resistance and improve airflow. The response to NIPPV can serve as an indicator of EDAC, prompting further diagnostic and therapeutic interventions.

**Conclusion:**

In conclusion, EDAC should be considered in patients with refractory respiratory symptoms, especially in those with a history of tuberculosis. Early recognition and appropriate management are essential to alleviate symptoms and improve patient outcomes. Dynamic bronchoscopy remains pivotal for diagnosis, while NIPPV offers a promising therapeutic approach. Further research and awareness are warranted to enhance the diagnosis and management of this underdiagnosed condition, ultimately improving the quality of life for affected individuals.

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