Sir,

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.\[1\] Although a patient may be asymptomatic most of the time, the pathological airway weakening can lead to dynamic airflow obstruction leading to symptoms such as dyspnea, orthopnea, intractable cough, and sometimes respiratory failure. EDAC presenting as exertional dyspnea, chronic wheeze, and postoperative respiratory failure has been mentioned in the literature.\[2-4\] EDAC presenting as intractable cough is a rare phenomenon. Nevertheless, cough is one of the common symptoms requiring a pulmonologist expertise in day-to-day practice.

A 45-year-old woman presented to the emergency department (ED) with a complaint of intractable cough of 3-week duration to the extent of even causing disturbed sleep. She was evaluated for similar complaints 2 weeks back. Routine investigations done then, including pulmonary function test (PFT) [Figure 1a] was normal. Computed tomography (CT) thorax [Figure 1b] showed minimal central bronchiectasis with mucous plugging, especially involving lower lobes. Total IgE level was normal. The patient was treated with steroids and nebulized bronchodilators suspecting hyperactive airway/cough variant asthma. She had partial response to treatment and was discharged. The patient had a history of intermittent episodes of dry cough in the past which aggravated during cold seasons and on exposure to dust. She was also prescribed inhalers for the same to which she was noncompliant. One week following discharge, the patient got readmitted with severe intractable cough. This admission, suspecting EDAC, she was given a trial of noninvasive positive-pressure ventilation (NIPPV) to which she responded. Bronchoscopy was done under conscious sedation which showed excessive dynamic expiratory collapse (>70%) of the trachea and both main bronchi with almost complete collapse during cough [Figure 1c and d]. The patient was managed with NIPPV, chest physiotherapy, and airway clearance techniques to which she showed significant improvement. 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.

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.[6] 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.

Loring et al. speculated that 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.[6] Our patient had a history of chronic cough, probably being cough-variant asthma, 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. This could be the reason for the CT findings of central bronchiectasis and mucus plugging in our case.

Dynamic bronchoscopy done under conscious sedation remains the gold standard for diagnosis of EDAC. Bronchoscopy should be preferably performed with a flexible instrument during spontaneous breathing with the patient conscious and alert so that he/she can follow the instructions of deep breathing, forced expiration, and coughing that increase the dynamic collapse of the airways. Dynamic CT remains the noninvasive method of diagnosis. PFT is of least diagnostic value.

Once identified a clear differentiation between TBM and EDAC should be made and specific cause, if any, should be looked for and treated. Treatment for EDAC depends on the severity of symptoms and the extent of airway collapse. Asymptomatic patients require no treatment. Proposed management strategies include conservative methods (bronchodilators and NIPPV), minimally invasive therapy (endoluminal airway stents and laser therapy), and surgical methods (tracheostomy, airway splinting, and tracheal resection).[1] NIPPV acts as a pneumatic stent, thereby decreasing the airway resistance and improving airflow. The response to NIPPV therapy was what prompted us of the possibility of EDAC.

To conclude, the presence of EDAC should be considered in patients with asthma and COPD not responding to standardized treatment. 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 after ruling out other common possibilities such as sinusitis, gastroesophageal reflux disease, nonasthmatic eosinophilic bronchitis, and asthma.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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