



Letter

Dynamic airway collapse: a frequently misdiagnosed asthma mimicker

Dynamic airway collapse (DAC) is a relatively common disease often misdiagnosed as asthma. It occurs in 5% to 10% of patients referred to pulmonologists for chronic cough and in 5% to 23% of patients undergoing bronchoscopic evaluation for respiratory symptoms.¹ The symptoms of DAC may closely mimic asthma, which often results in misdiagnosis, unnecessary procedures, and escalation of treatments. Patients with DAC may undergo multiple diagnostic procedures, have minimal improvement with standard asthma therapy, and have the potential for iatrogenic adverse effects. DAC must be distinguished from other proximal airway diseases, including vocal cord dysfunction and tracheo-bronchomalacia. It can occur concurrently with asthma, and because the management differs from that of asthma, it is important to increase awareness and consideration of this disease.

In this report, we describe a 58-year-old, nonsmoking, white woman from North Dakota with a history of allergic rhinitis who presented with shortness of breath and wheezing at the age of 30 years. She had an extensive workup and was diagnosed as having asthma. She was prescribed an albuterol inhaler to use as needed, with occasional use of prednisone during acute exacerbations. Triggers included upper respiratory tract infections, exercise, and dust exposure. Her collateral allergic history and family history were negative for other atopic diseases.

During the next 15 years, her symptoms became more frequent and problematic, requiring multiple emergency department visits annually. In 2012, after moving to California, she was evaluated by an allergist. Skin prick test results were positive to bluegrass, olive tree, and *Aspergillus* mold. She was prescribed an intranasal corticosteroid spray, montelukast, and inhaled mometasone-formoterol. Her symptoms continued to deteriorate, with daily audible wheezing, dyspnea, cough, and nocturnal awakenings. Levalbuterol was only partially effective. Inhaled ciclesonide was added, without improvement. In 2013, she became dependent on oral corticosteroids, using 60 mg/d of prednisone and higher interval bursts, with only partial relief.

The patient was evaluated at a tertiary care hospital by specialists in pulmonology, allergy/immunology, and gastroenterology. Pulmonary function tests revealed forced expiratory volume in 1 second (FEV₁) of 2.06 L (78% predicted) and ratio of FEV₁ to forced vital capacity of 71%. Exhaled nitric oxide level was 26 ppb. Bronchoscopy with bronchoalveolar lavage and endobronchial biopsy revealed chronic inflammation, lymphocytic

predominance, and negative culture results for bacteria, fungi, and atypical mycobacteria. A methacholine challenge result was positive with a provocation concentration that caused a decrease in FEV₁ of 20% of 0.31 mg/mL. Echocardiography results were normal. An esophageal pH study was diagnostic for gastroesophageal reflux disease, and she was prescribed a proton pump inhibitor, without significant improvement. Computed tomography (CT) of the paranasal sinuses was clear, and α_1 -antitrypsin levels were sufficient.

The patient was subsequently referred to our complex asthma program for further evaluation by pulmonology and allergy/immunology specialists. On physical examination, vital signs were normal, body mass index was 29.6, and lung examination revealed faint expiratory wheezes bilaterally. Serum specific IgE test results to aeroallergens were negative. Evaluation findings for allergic bronchopulmonary aspergillosis, collagen vascular disease, and primary immunodeficiency were normal. Pulmonary function testing revealed normal spirometry with absent bronchodilator response and normal lung volumes and diffusing capacity of lung for carbon monoxide.

A high-resolution CT of her chest with inspiratory and end-expiratory radiographs was performed (Fig 1), which revealed evidence of significant airway collapse on expiratory radiographs involving the trachea and mainstem bronchi. We subsequently diagnosed her as having DAC as the predominant cause of her recalcitrant symptoms, with a smaller component of asthma given her mild obstruction on spirometry and positive methacholine challenge results. She was administered continuous positive airway pressure (CPAP) of 10 cm H₂O, despite her sleep study revealing mild obstructive sleep apnea with an apnea-hypopnea index of 15. Subsequently, her inhalers and prednisone were weaned and then discontinued. Her symptoms significantly improved, and she has remained without any exacerbations.

As this case reveals, DAC is often misdiagnosed as asthma because of symptom overlap. Diagnosis of DAC requires a high index of suspicion and careful history and physical examination. Correct diagnosis is imperative because of morbidities associated with misdiagnosis. DAC is caused by pathologic collapse of the posterior tracheal membrane into the airway lumen during exhalation, causing narrowing of the lumen by at least 50%. In some cases, the laxity of the posterior membrane is due to atrophic elastic fibers.² Unlike in tracheo-bronchomalacia, the tracheal and bronchial cartilage remains intact.

DAC is seen in all age groups. The acquired form has increased incidence with age and a male predominance. Up to 22% of patients with chronic obstructive pulmonary disease (COPD) and asthma

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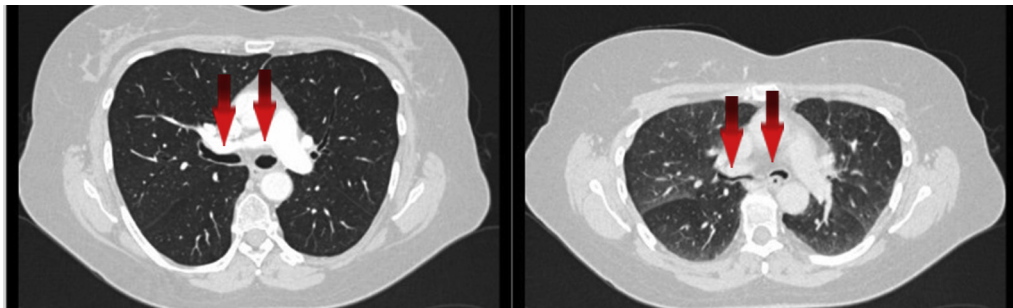


Figure 1. Comparison of inspiratory (left) and expiratory (right) computed tomographic images of the thorax, showing collapse of trachea and mainstem bronchi, loss of more than 50% luminal diameter, and frown sign on expiration.

who undergo bronchoscopy are found to have DAC.³ Secondary causes of DAC include COPD, asthma, bronchiectasis, bronchiolitis, obesity, and congenital atrophy of the elastic fibers known as Mounier-Kuhn syndrome.⁴ Differential diagnosis should include COPD, asthma, tracheal stenosis, relapsing polychondritis, long-standing extrinsic compression, and radiation.⁵

Symptoms of DAC include cough, dyspnea, orthopnea, wheezing, difficulty clearing the airway, stridor, and respiratory failure (including failure of extubation).³ Physical examination may reveal wheezing, rhonchi, decreased breath sounds, and poor air movement. Clinical suspicion for DAC should increase when these symptoms fail to improve with bronchodilators.

Diagnosis of DAC is confirmed during coughing or forced expiratory maneuvers, when a decrease in the transmural pressure fosters increased compliance and excessive collapsibility of the weak posterior membrane by the Bernoulli effect,⁶ resulting in reduction of airway caliber by at least 50%. Bronchoscopy is the criterion standard for diagnosis, and measurement of airway pressure via a double lumen catheter can be obtained to identify the most obstructed site.⁷ In our case, the patient refused dynamic diagnostic flexible bronchoscopy with CPAP titration for confirmation. However, dynamic and kinetic CT is comparable to bronchoscopy for evaluation.⁸ Comparison is made between inspiratory and end-expiratory images. The frown sign is a classic feature in which posterior tracheal membrane collapse results in a crescentic bowing of the membrane.⁹ Endobronchial ultrasonography can be used to distinguish between tracheobronchomalacia and DAC.²

In symptomatic DAC, first-line treatments include vocal exercises and pneumatic stenting with CPAP. If a flow-limiting segment is identified in the trachea or mainstem bronchi, placement of a silicone stent may be beneficial.⁴ Surgical tracheoplasty may be considered for severe or refractory cases.¹⁰

This case highlights the importance of considering dynamic airway disease when asthma symptoms fail to improve with traditional therapy. A detailed history, high index of suspicion, and appropriate diagnostic modalities are critical in making the diagnosis.

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