Exertional Dyspnea and Excessive Dynamic Airway Collapse

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KEYWORDS

• Dyspnea • Exercise • Excessive dynamic airway collapse • Tracheobronchomalacia
• Asthma • Chronic obstructive pulmonary disease

KEY POINTS

- Excessive dynamic airway collapse (EDAC), which is functional large airway collapse, can occur during exercise.
- EDAC symptoms include exertional dyspnea and centralized expiratory wheezing.
- EDAC may be identified in patients with chronic obstructive pulmonary disease or asthma.

INTRODUCTION

Excessive dynamic airway collapse (EDAC) is a recent term applied to individuals identified with functional collapse of the large airways. It is generally defined as excessive bulging of the posterior tracheal membrane into the airway lumen (usually >75%) during expiration without cartilage collapse to distinguish this entity from tracheobronchomalacia (TBM).[1] It has been primarily associated with underlying airway disorders, such as chronic obstructive pulmonary disease (COPD), asthma, and bronchiectasis.[2] Those patients with moderate to severe symptoms can present with cough, wheezing, dyspnea, or recurrent respiratory infections related to exacerbations of their lung disease. Likewise, it may become symptomatic after mild to moderate exertion. Current guidelines suggest that functional status (exertion, daily activities, or rest), length of

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the tracheobronchial wall affected, and severity of collapse are also important to define in patients diagnosed with EDAC.[2]

Much of the description of EDAC has focused on exacerbations of underlying disease with little discussion on its relationship to exercise. Weinstein and colleagues[3] recently published a case series of 6 individuals in the military with symptomatic EDAC only during exercise. All had exertional symptoms, expiratory wheezing, and confirmed visual evidence of large airway collapse (>75%) with fiberoptic bronchoscopy (FOB). The findings have expanded the differential in the challenging evaluation of exertional dyspnea patients with otherwise normal pulmonary function testing (PFT) and chest imaging.[4] In this review, the authors aim to discuss the relationship between exercise and EDAC and further elucidate its potential impact on exercise limitation.

EXCESSIVE DYNAMIC AIRWAY COLLAPSE DEFINITION

EDAC is generally defined as excessive bulging of the posterior tracheal membrane during expiration with greater than 75% collapse of the airway lumen. It is distinctly different from TBM because of the absence of cartilaginous involvement. These distinct clinical entities may be very similar in presentation and have been referred to collectively as expiratory central airway collapse.[2] However, there is no universally accepted cutoff value to separate normal from abnormal expiratory airway narrowing, but generally greater than 75% closure of the airway lumen has been suggested to be clinically important.

Reflecting tidal cycling of intrathoracic pressure, there is inward bulging of the posterior membrane of the trachea and main bronchi that occurs normally during exhalation. This airway compression, known as dynamic airway collapse, can ordinarily narrow the lumen of normal central airways by up to 50%. Posterior membrane collapse has also been described to be partly physiologic and associated with forced expiration or cough. Defining the clinical significance of EDAC can be problematic as functional airway collapse in healthy individuals has been documented. There is a wide variation in bronchial collapse with forced expiration on multidetector computed tomographic (CT) imaging in healthy volunteers with normal PFT and no significant smoke exposure.[5,6]

PATHOPHYSIOLOGY

It is currently theorized that large airway collapse related to EDAC is due to increased airway resistance distal to the equal pressure point, decreased lung elastic recoil, and increased pleural pressures. During exercise itself, there is increased luminal airflow velocity leading to a decrease in luminal pressures, especially in tapering airways, and creating a stress on luminal integrity with subsequent smooth muscle fatigue or strain. This muscle lassitude in the setting of continued pressure differential is the likely culprit for why this change in airway diameter is not identified on nonexertional pulmonary testing.[7]

The pathogenesis of EDAC has been described as being a result of 2 primary circumstances. One factor is weakening of the smooth muscle tone of the posterior membrane. The other factor is a decrease in luminal pressures in regions of tapering airways (Bernoulli's principle) in the setting of reduced elastic recoil, thus creating even greater narrowing leading to greater transmural pressure gradient.[8] The diagnosis of EDAC has primarily been described in patients with underlying lung disorders such as COPD or asthma, especially in those patients with identifiable TBM. The underlying lung pathology likely contributes to the chronic atrophy and strain of