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Tracheobronchomalacia☆

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ABSTRACT

The term tracheobronchomalacia refers to excessively compliant and collapsible central airways leading to symptoms. Although seen as a coexisting condition with various other pulmonary condition, it may cause symptoms by itself. The condition is often misdiagnosed as asthma, bronchitis or just chronic cough due to a lack of specific pathognomonic history and clinical findings. The investigation revolves around different modes of imaging, lung function testing and usually confirmed by flexible bronchoscopy. The treatment widely varies based on the cause, with most cases treated conservatively with non-invasive ventilation. Some may require surgery or stent placement. In this article, we aim to discuss the pathophysiology behind this condition and recognize the common symptoms and causes of tracheobronchomalacia. The article will highlight the diagnostic steps as well as therapeutic interventions based on the specific cause.

Introduction

The term “malacia” refers to weakness of a structure inside the human body. Tracheobronchomalacia (TBM) refers to a weak or damaged trachea and bronchus which may or may not lead to dynamic airflow limitation and symptoms arising thereof. The condition is referred to as tracheomalacia (TM) when a segment or the entire trachea is involved. The term TBM signifies involvement of the trachea and mainstem bronchus. Bronchomalacia indicates isolated weakness and collapsibility of one or both mainstem bronchus.[1] What is important to recognize is that there is a structural and a functional component to this condition – structural weakness as witnessed by the collapse of the airways and then a functional airflow limitation, which may or may not be present. Any perturbation of the laminar airflow may lead to symptoms and may be detected on lung function tests. TBM affects a broad age group; paediatric and adult TBM are often disparate entities requiring distinct approaches towards diagnosis and treatment. We will keep our discussion limited mostly to TBM in the adult population with an occasional mention of the paediatric condition, only if there is a similarity with adult TBM.

The compliant nature of the intrathoracic tracheal walls leads to some degree of collapse with expiration and dilatation with inspiration due to relative pressure changes between the intrathoracic and intraluminal pressures.[2–4] Tracheobronchial distention results from the generation of negative intrathoracic forces leading to movement of air into the thoracic cage with each inspiratory effort. A positive intrathoracic pressure pushes air out of the chest cavity during expiration and hence, compresses the compliant intrathoracic trachea with variable degree collapse proportional to the degree of respiratory effort. Such physiological changes in airway diameter are normal and usually do not lead to symptoms. An abnormality is said to exists when such variation is beyond

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normal physiological limits and produce symptoms by virtue of the expiratory tracheal collapse.[5] The diagnosis of clinically relevant TBM requires thoughtful assessment and careful determination of the presence of a functional limitation resulting from weakness of the trachea and other central airways. We will analyse the diagnostic criteria for TBM, enumerate its etiology and discuss means to classify the severity of the disease through this review article. Finally, we will determine the best course of action for each individual condition and review the pros and cons of each such therapy.

Definitions

It is necessary to mention upfront that the definition of TBM is controversial and still debated upon. Pulmonologists have been able to determine that a structural abnormality and functional airflow limitation resulting from it do not have a simple proportional relationship. The determination of the extent of a "normal degree of tracheobronchial collapse" is extremely difficult. The exact percentage of airway collapse that qualifies for further work-up is dependent on associated comorbidities such as obstructive airway diseases.[6] This leads to a heterogeneity in the definition of "clinically relevant TBM" and thereupon introduces some difficulty with creating a uniform diagnostic criteria. Hence, we will attempt to define this condition based on the most accepted and updated views on this topic.

Expiratory collapse of the central airway can result from weakness of the cartilaginous tracheobronchial walls or from excessive invagination of the posterior membranous portion of the trachea without any obvious weakness of its structure or integrity or combination of weakened cartilaginous wall and excessive invagination of membranous airway wall. Together these entities are called expiratory central airway collapse (ECAC) and can be further classified into following subgroups.

Expiratory central airway collapse (ECAC)

A common term to define the expiratory narrowing of the central airway (trachea and bronchus) from a wide variety of causes leading from damage of the cartilaginous tracheal superstructure (TBM) or weakness of the posterior membranous part of the airway (EDAC).

Tracheobronchomalacia (TBM)

TBM refers to collapse of tracheobronchial due to loss of structural integrity of the trachea-bronchial cartilage as well as hypotonia of the myoelastic elements.[8,9] This may lead to reduction of the tracheobronchial lumen and building up of airway secretions. Patients may complain of dyspnoea, cough and wheezing and present with recurrent pneumonia.

Excessive dynamic airway collapse (EDAC)

A reduction of the airway lumen by 50% or more in sagittal diameter is abnormal and, if due to invagination of the posterior membrane with structurally intact cartilage, is referred to as EDAC.[10–12] The advancement in diagnostic flexible bronchoscopy, dynamic CT scans allows for better and more accurate identification of this entity.

Normal breathing leads to some degree of collapse of the pars membranosa (posterior membrane of the trachea) during expiration. Dynamic CT scans reveal normal variation in central airway cross sectional airway (11 to 61%) area during quite breathing.[5] Thus, a certain degree of expiratory dynamic collapse is expected and speculated to aid with expectoration and secretion clearance. In healthy individuals, the tracheobronchial lumen collapses by 18–39% of the maximal diameter while coughing.[11–13] The invagination of the pars membranosa is noted to be exaggerated in some patients with obstructive pulmonary disease such as chronic bronchitis, emphysema, asthma and TBM.[11–13] Excessive collapse may also be seen as an isolated finding in healthy patients during cough and forced expiration.

It is very important to make the distinction between these two entities since treatment for these differ considerably. Although initial reviews on the topic of TBM did not make a distinction between these two entities,[5,14,15] we will attempt to clarify the difference between the terms EDAC and TBM.

Diagnosis of TBM and EDAC

Patients with expiratory central airway collapse often remain undiagnosed or misdiagnosed because of the similarity of their symptoms with that of asthma or chronic bronchitis.

Traditionally, a decrease in lumen size by 50% or less during exhalation is considered normal. Collapse of central airways beyond is considered to be abnormal. Between 51 to 75% collapse indicates mild obstruction, 76 and 90% moderate obstruction, and 91 and 100% regarded as severe obstruction.[16] The diagnosis of TBM or EDAC thus requires obstruction greater than 50% during exhalation in the presence of respiratory symptoms attributable to such an obstruction.[17,18] Flexible bronchoscopy is considered to be the gold standard investigation although dynamic CT scan technology has shown promise and is thought to have an acceptable degree of correlation with bronchoscopic assessment.[19,20]

Having said that, it is essential to point out that the degree of airway collapse necessary to diagnose EDAC has not been universally agreed upon. In addition to the disagreement regarding the degree of collapse, disagreement also exists in identifying the best investigation modality. To add to the confusion, the critical degree of collapse necessary to be of clinical significance varies