Commentary

Tracheobronchomalacia in obstructive airway diseases

Ravindran Chetambath

Department of Pulmonology, DM Wayanad Institute of Medical Sciences, Wayanad, Kerala, India.
E-mail: crcalicut@gmail.com

Tracheobronchomalacia (TBM) is characterized by generalized or focal airway wall weakness, resulting in excessive variation of the luminal diameter during the respiratory cycle. The weakness of the trachea is due to a reduction and/or atrophy of the longitudinal elastic fibers or impaired cartilage integrity so that the airway become softer and more susceptible to collapse.[1,2] This disease may arise congenitally, or it may be acquired due to trauma, infections, or chronic inflammation.[3] It is seen more commonly in the pediatric population, and the symptoms usually subside with age as the cartilaginous rings become more rigid, with reduction of tracheal wall compliance. Acquired TBM has been reported to be present in 12.7% of all patients undergoing bronchoscopy evaluation for respiratory diseases, and in 44% of patients undergoing bronchoscopy who have a history of chronic bronchitis.[3]

TBM has been associated with airway inflammatory diseases such as chronic obstructive pulmonary disease (COPD) and asthma. Chronic airway inflammation might play a role in exacerbations of these disorders. TBM acquired in the background of such inflammation results in persistence of symptoms.[4] TBM is associated with a substantial invagination of the posterior wall of the tracheobronchial tree which is called excessive dynamic airway collapse (EDAC).[5] The clinical impact of these abnormalities is quite high. The pathologic weakening of the airway can produce dynamic outflow obstruction and severe symptoms, in the form of cough, dyspnea, and wheezing. These symptoms are resistant to corticosteroids and inhaled bronchodilators. Clearing of secretions may be affected leading to recurrent pneumonia, atelectasis, or respiratory insufficiency. Air trapping was also observed with greater severity in these patients when compared to patients of similar ages without TBM.[6] Up to 7% of patients with severe TBM may require mechanical ventilation due to respiratory failure.[7]

Although bronchoscopy is widely used to diagnose TBM, recent advances in computed tomography (CT) imaging can be used as a noninvasive investigation to diagnose this entity with a high level of accuracy.[8] Dynamic CT of the chest exhibits tracheal collapse with crescentic bowing of the posterior membranous trachea during expiration.[9] Examining the airway with flexible bronchoscopy during passive exhalation can confirm collapse of the central airway typical of TBM.

The presence of TBM or EDAC should be considered whenever COPD or asthma persists even after appropriate pharmacological intervention. Current treatments include techniques that splint the central airways such as continuous positive pressure ventilation,[10] silicone airway stents,[11] and surgical tracheobronchoplasty.[12] Surgical central airway stabilization with posterior tracheobronchial splinting using a polypropylene mesh improves the symptoms.

In this issue of the journal, result of a pilot study on the influence of TBM and EDAC in patients of COPD is presented. This study is based on the hypothesis that TBM/EDAC may be a cause of persistent wheeze in patients with obstructive airway disorders even after optimal medical management. The authors conclude that 40% of their patients with persistent wheeze have features of TBM/EDAC. Even though they could not find any significant correlation with severity of disease and exacerbation, it is postulated that identification of TBM/EDAC in COPD may provide additional treatment options. It could have been better if COPD without persistent wheeze were also included in this study so that the true impact of TBM/EDAC in COPD could be ascertained.

REFERENCES

1. Johnson TH, Mikita JJ, Wilson RJ, Feist JH. Acquired tracheomalacia. Radiology 1973;109:576-80.
2. Jokinen K, Palva T, Sutinen S, Nuutinen J. Acquired tracheobronchomalacia. Ann Clin Res 1977;9:52-7.
3. Jokinen K, Palva T, Nuutinen J. Chronic bronchitis. Abronchologic evaluation. J Otorhinolaryngol Relat Spec 1976;38:178-86.
4. Palombini BC, Villanova CA, Araújo E, Gastal OL, Alt DC, Stolz DP, *et al*. A pathogenic triad in chronic cough: Asthma, postnasal drip syndrome, and gastroesophageal reflux disease. Chest 1999;116:279-84.
5. Murgu DS, Colt GH. Tracheobronchomalacia and excessive dynamic airway collapse: Novel diagnostic tools clarify the issues. Pulm Perspect 2005;22:7-10.
6. Zhang J, Hasegawa I, Hatabu H, Feller-Kopman D, Boiselle PM. Frequency and severity of air trapping at dynamic expiratory CT in patients with tracheobronchomalacia. AJR Am J Roentgenol 2004;182:81-5.
7. Ernst A, Majid A, Feller-Kopman D, Guerrero J, Boiselle P, Loring SH, *et al*. Airway stabilization with silicone stents for treating adult tracheobronchomalacia: A prospective observational study. Chest 2007;132:609-16.
8. Lee KS, Sun MR, Ernst A, Feller-Kopman D, Majid A, Boiselle PM. Comparison of dynamic expiratory CT With bronchoscopy for diagnosing airway malacia: A pilot evaluation. Chest 2007;131:758-64.

9. Gilkeson RC, Ciancibello LM, Hejal RB, Montenegro HD, Lange P. Tracheobronchomalacia: Dynamic airway evaluation with multidetector CT. AJR Am J Roentgenol 2001;176:205-10.
10. Ferguson GT, Benoist J. Nasal continuous positive airway pressure in the treatment of tracheobronchomalacia. Am Rev Respir Dis 1993;147:457-61.
11. Sommer D, Forte V. Advances in the management of major airway collapse: The use of airway stents. Otolaryngol Clin North Am 2000;33:163-77.
12. Wright CD. Tracheomalacia. Chest Surg Clin N Am 2003;13:349-57, viii.

Access this article online

Quick Response Code:

Website: www.lungindia.com

DOI: 10.4103/0970-2113.184929

How to cite this article: Chetambath R. Tracheobronchomalacia in obstructive airway diseases. Lung India 2016;33:451-2.