Obstructive lung disease secondary to compression of the bronchus by an enlarged pulmonary artery

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ABSTRACT

In clinical practice, the presence of wheezing generally indicates an airway disease. In rare circumstances, adjacent mediastinal structures may compress the tracheobronchial tree leading to obstructive physiology. Compression of the tracheobronchial region by an enlarged pulmonary artery (PA) is exceedingly rare. We present here a case of pulmonary hypertension, where the enlarged PA resulted in obstructive lung physiology with a relevant review of the literature.

KEY WORDS: Bronchial compression, intrathoracic obstruction, obstructive lung disease, pulmonary artery dilatation, pulmonary hypertension

INTRODUCTION

Diseases of the airways present with airflow limitation leading to obstructive lung physiology. Rarely, problems outside of the conducting airways can lead to such a phenomenon. Airway compression by enlarged blood vessels is one such cause.⁹ We present here a case of dynamic right mainstem bronchus compression caused by the enlargement of the pulmonary artery (PA) in a patient with pulmonary hypertension (PHTN).

CASE REPORT

A nonsmoking 74-year-old male with a history of pulmonary embolism was evaluated for dyspnea on exertion and cough. On examination, he was hemodynamically stable, with a room air saturation of 88%. On examination, he had wheezing in both lower lung posterior hemithoraces but was more pronounced on the right side. Echocardiogram showed pulmonary arterial systolic pressure of 65 mmHg. His 6-min walk test was 317 m. Right heart catheterization showed right atrial pressure of 13 mmHg; mean pulmonary arterial pressure of 44 mmHg; mean pulmonary arterial wedge pressure of 14 mmHg; cardiac output of 2.65 L/min; and pulmonary vascular resistance of 11.32 wood units.

Spirometry showed an obstructive pattern [Table 1]. The flow-volume loop also showed flattening in the expiratory phase [Figure 1]. Computed tomography (CT) angiography showed an enlarged PA measuring 7 cm with compression of the right mainstem bronchus by the right PA [Figure 2]. Therapeutic stenting of the airway was entertained; however, he was deemed a high-risk surgical candidate. Instead, we continued medical management and follow-up.

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DISCUSSION

This case outlines a rare cause of dyspnea in a patient with pulmonary arterial hypertension with enlarged PA or its branches causing airflow limitation due to extrinsic compression of the airways.

Enlargement in thoracic vascular structures is a known cause of airway compression, which can be classified as congenital or acquired. Congenital vascular malformations causing extrinsic airway obstruction are common in children. In this population, airway cartilage is more elastic and hence susceptible to compression. Acquired vascular malformations leading to tracheobronchial compression are more common in adults. Severe aortic aneurysm secondary to atherosclerosis is the most common cause of bronchial compression in this clinical setting. Vascular bronchial compression may be misdiagnosed with asthma because of shared presenting symptoms of dyspnea and cough. Chest X-ray is an initial test in patients with respiratory complaints. However, its usefulness is limited in cases of vascular airway compression as the only abnormality seen on a chest X-ray may be mediastinal widening. Pulmonary function tests are a more accurate diagnostic test. The flow-volume loop in bronchial compression may show a characteristic expiratory flattening, as seen in this case. During expiration, the pleural pressure becomes positive relative to intra-airway pressure. In the presence of external compression, this difference is amplified and is reflected by end-expiratory loop flattening representing variable intrathoracic obstruction. CT angiography is considered the gold standard for defining the vasculature and the spatial relationship between the vessels and the airways. This modality can be combined with bronchoscopy. After identifying the cause, treatment of the lesion is typically attempted by surgical decompression of the vascular abnormality or airway stenting. Although airway stenting in such cases appears to be an attractive alternative as it is less invasive, there is always a risk of wall erosion by the stent hence it has not been much reported in these cases. Surgical interventions to relieve the extrinsic airway compression are of high risk in patients with PHTN.

CONCLUSION

Wheezeing in patients can occur from many causes. In patients with PHTN, the cause of wheezeing may be the...
first indication of compression of the tracheobronchial tree from an enlarged PA.

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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**Table 2: List of cases of symptomatic bronchial vascular compression by enlarged pulmonary artery**

| Author          | Journal                      | Patient age and sex | WHO PHTN Group | PA dilatation | Intervention       |
|-----------------|------------------------------|---------------------|----------------|---------------|--------------------|
| Achouh et al.  | European Respiratory Journal  | 34 male             | Group I        | Pulmonary trunk to aortic diameter ratio >2 | Medical management Bosentan |
| Achouh et al.  | European Respiratory Journal  | 56 male             | Group I        | Pulmonary trunk to aortic diameter ratio >1.5 | Medical management Bosentan |
| Morjaria et al. | Pulm Circ 2012               | 55 male             | Group IV       | Not reported  | Medical management Bosentan |
| Jaijee et al.  | Pulm Circ 2015               | 73 female           | Group I        | 91 mm         | Medical management Bosentan |
| Jaijee et al.  | Pulm Circ 2015               | 63 male             | Group I        | 51 mm         | Medical management Sildenafil |
| Arimura et al. | Am J Respir Crit Care Med 2015| 42 female           | Group I        | 79 mm         | Medical management Bosentan |
| Nokes et al.   | Am J Respir Crit Care Med 2019| 63 female           | Group I        | Dilation see in APCs | Not reported          |

APCs: Aortopulmonary collaterals, PHTN: Pulmonary hypertension, PA: Pulmonary artery