

Pulmonary Vocal Syndrome

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Abstract

Vocal cord paralysis is a common entity with diverse causes clinically manifesting as dysphonia. Vocal cord paralysis due to respiratory cause is due to involvement of left recurrent laryngeal nerve usually secondary to bronchogenic carcinoma. However, it can also be seen in association with other less well recognised causes such as pulmonary tuberculosis. We present to you a patient with hoarseness of voice due to left recurrent laryngeal nerve paralysis secondary to endobronchial tuberculosis.

Introduction

Hoarseness of voice can occur due to anatomical or functional abnormality of larynx. Common cause includes laryngeal infections, blunt trauma, iatrogenic affection of recurrent laryngeal nerve, malignancies of thyroid, oesophagus and lung and cardiovascular conditions such as

mitral stenosis. Vocal cord paralysis due to respiratory cause is known as pulmonary vocal syndrome. We present a case of pulmonary vocal syndrome due to endobronchial tuberculosis.

Case Report

A 30-year-old lady presented with symptoms of three months duration of intermittent fever, cough and hoarseness of voice. There was no

history of preceding viral illness, vocal cord abuse, paroxysmal nocturnal dyspnoea or rheumatic heart disease. Her general physical and systemic examination was within normal limits.

Chest radiograph showed left upper lobe fibrosis. Sputum examination did not reveal acid fast bacilli. High resolution computerised tomography of thorax showed fibrosis in left upper lobe and lingula with absence of any other intrathoracic lesion or enlarged mediastinal lymph nodes (Figure 1). Fiberoptic bronchoscopy revealed left vocal cord palsy along with an additional finding of scarring of trachea at the distal end with the stenosis of left main stem bronchus (Figure 2). Bronchial washing gene Xpert detected *Mycobacterium Tuberculosis* thus confirming the diagnosis of endobronchial tuberculosis.

Discussion

Hoarseness of voice is a frequently encountered symptom seen due to structural or functional involvement of larynx or secondary to involvement of recurrent laryngeal nerve. Common causes include iatrogenic such as following thyroid surgeries (41%), idiopathic causes (33%) and well defined causes (25%)¹ such as lung malignancy, thyroid malignancy, oesophageal malignancy, cardiac causes such as Ortner's syndrome and chronic benign inflammatory conditions such as tuberculosis.

Earlier hoarseness of voice after ruling out malignancy of the lung was largely attributed to cardiovascular causes such as mitral stenosis, left atrial enlargement or pulmonary hypertension. This entity was known as Ortner's syndrome. Hoarseness of voice due to involvement of recurrent laryngeal nerve secondary to a respiratory cause is known as pulmonary vocal syndrome. The involvement of left recurrent laryngeal nerve is more common when compared to right as it arches around the arch of aorta and has a substantial intrathoracic course.² Such an entity is more often encountered in association with

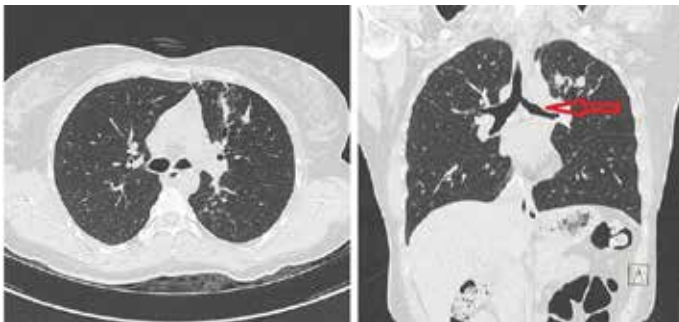


Fig. 1: HRCT axial and sagittal images showing upper lobe fibrosis and narrowing of left main bronchus (red arrow)

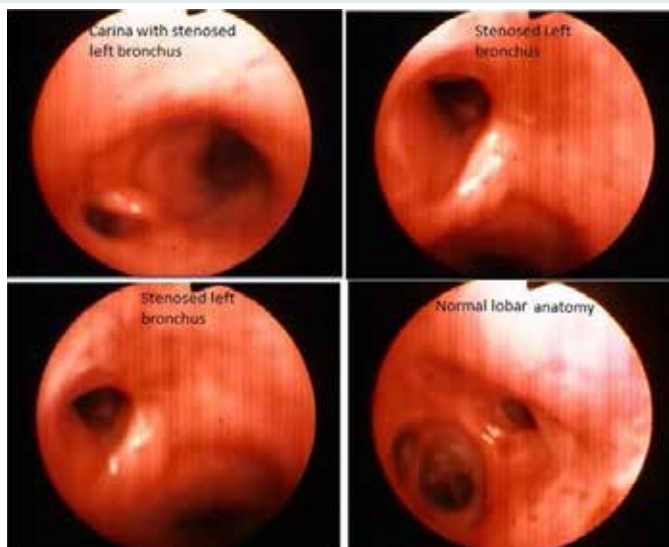


Fig. 2: Bronchoscopy images showing left main bronchus stenosis

bronchogenic carcinoma³. However, it can also be seen in association with chronic benign inflammatory conditions such as tuberculosis. Later Radner⁴ quoted in his article about the possibility of tuberculosis leading to recurrent laryngeal nerve palsy. The various mechanisms responsible in tuberculosis that might lead to pulmonary vocal syndrome are⁵⁻⁷

1. The nerve passing through caeateating lymph node.
2. The nerve might get stretched due to dense pleural thickening or fibrosing mediastinitis.
3. The nerve being stretched due to retraction of upper lobe bronchus towards apex as in endobronchial tuberculosis.
4. The nerve being compressed by enlarged pulmonary artery.

Cases of pulmonary tuberculosis associated with mediastinal lymphadenopathy⁸ or upper lobe fibrosis leading to pulmonary vocal syndrome have commonly been reported in literature. However, endobronchial tuberculosis leading to pulmonary vocal syndrome is rarely documented. Endobronchial tuberculosis is defined as tuberculous infection of the tracheobronchial tree. Clinically a

case of endobronchial tuberculosis may present with cough with sputum production, fever, haemoptysis and rarely with hoarseness of voice as in our case. Endobronchial tuberculosis has been classified into seven subtypes as non-specific, granular, stenotic type with fibrosis, stenotic type without fibrosis, actively caseating type, ulceroproliferative and tumorous type.⁹ Establishing the diagnosis of endobronchial tuberculosis is often challenging as the lesion is usually not evident radiologically. Sputum examination is often unyielding and bronchial washings need to be obtained. The mainstay of therapy is eradication of tubercle bacilli with appropriate antituberculous regime followed by prevention of stenosis or fibrosis of endobronchial tree.

Conclusion

Our patient was a case of pulmonary vocal syndrome secondary to endobronchial tuberculosis. To the best of our knowledge pulmonary vocal syndrome attributable to primary endobronchial tuberculosis has not been reported earlier. Hence endobronchial tuberculosis should be considered as a differential diagnosis in cases presenting with vocal cord palsy after

ruling out other etiologies in the correct clinical context.

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