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Case Report

Losing the Voice to the Lungs: Ortner's Syndrome in Pulmonary Vascular Disease

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Abstract

Ortner's syndrome, also known as cardiovocal syndrome, is a rare clinical condition marked by left recurrent laryngeal nerve palsy. It is most commonly caused by mechanical compression or traction of the nerve between the aortic arch and the pulmonary artery, typically triggered by underlying cardiovascular diseases like aortic aneurysms or mitral stenosis. In 1897, Norbert Ortner elucidated the association between left recurrent laryngeal nerve palsy and severe mitral valve stenosis, which leads to hoarseness of voice. Although it was originally described in association with severe mitral stenosis, the syndrome may arise from several other causes. These include compression of the nerve by vascular conditions such as aortic aneurysm, aortic dissection, and pulmonary hypertension. We present a case of Ortner's syndrome in a 41-year-old man, characterized by dysphonia from left recurrent laryngeal nerve compression, secondary to severe pulmonary arterial hypertension and embolic pulmonary occlusion. This case emphasizes the need to carefully consider cardiac causes in patients with vocal cord dysfunction and supports the use of a comprehensive, team-based approach to diagnosis and treatment.

Keywords: Ortner's syndrome, Dysphonia, Pulmonary hypertension, Cardio-vocal syndrome.

Introduction

Norbert Ortner[1] first established the link between severe mitral valve stenosis and left recurrent laryngeal nerve (RLN) paralysis in 1897. Hoarseness arising from vocal cord paralysis in the setting of an underlying cardiovascular disorder constitutes a rare and noteworthy clinical manifestation.[2]

The RLN, a branch of the vagus nerve, provides sensory innervation inferior to the vocal folds and motor supply to all intrinsic laryngeal muscles, excluding the cricothyroid. Its course is asymmetric; the right RLN loops around the subclavian artery, while the left RLN loops around the ligamentum arteriosum near the aortic arch before ascending in the tracheoesophageal groove. The left RLN is more prone to injury due to its longer intrathoracic pathway and proximity to mediastinal structures.[3] Modern documented cases of Ortner's syndrome are most frequently linked to aortic aneurysms. Nevertheless, the syndrome has also been described in association with ductus arteriosus, left atrial enlargement, pulmonary hypertension and pulmonary thrombo-embolism.[4,5] This case report outlines the clinical progression of a patient who developed cardio-vocal syndrome secondary to pulmonary arterial hypertension and pulmonary thromboembolism, and further examines the underlying pathophysiology, diagnostic evaluation, and multidisciplinary management approach.

Case Report

A 41-year-old male presented with increasing breathlessness, which was progressive in nature for a duration of three months, followed by hoarseness of voice for two months. Patient was a former smoker with a pack-year of 12, and had a prior history of tuberculosis for which the patient had taken

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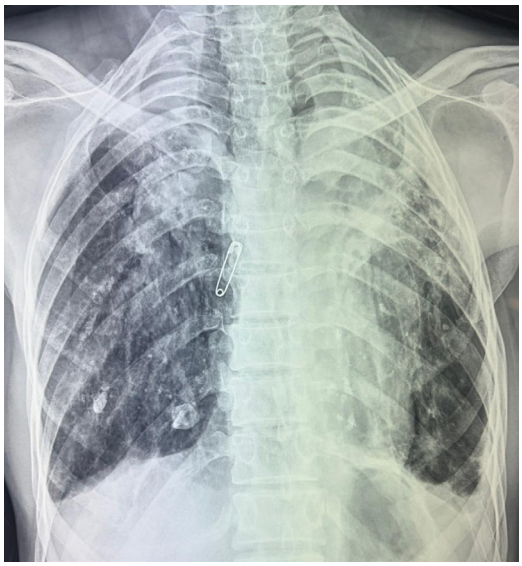

Figure 1: chest x-ray showing mediastinal widening, bilateral upper zones fibrosis along with pleural thickening and features of lower zone hyperinflation

anti-tuberculosis therapy for 6 months ten years back. There was no history of prior cardiac disease. On general physical examination, elevated jugular venous pressure and digital clubbing were noted. Clinical evaluation further revealed reduced oxygen saturation with associated tachycardia and tachypnoea. Cardiovascular assessment demonstrated an accentuated pulmonary component of the second heart sound, while neurological examination confirmed hoarseness of voice in the absence of other cranial nerve deficits.

A chest X-ray was done which showed mediastinal widening and bilateral upper zone fibrosis (Figure 1). Electrocardiography (ECG) was suggestive of sinus tachycardia, right axis deviation, right bundle branch block, P-Pulmonale, right atrial enlargement, and right ventricular hypertrophy; findings suggestive of right heart strain associated with pulmonary hypertension. Transthoracic echocardiography revealed an enlarged right atrium and ventricle with severe tricuspid regurgitation, with right ventricular systolic pressure of 95 mmHg. Fibreoptic bronchoscopy revealed an immobile left vocal cord, while both the right and left bronchial trees appeared normal. A contrast-enhanced CT scan of the chest demonstrated an eccentric filling defect within the left pulmonary artery, suggestive of pulmonary thromboembolism and a dilated pulmonary artery trunk with a diameter of 36 mm. A D-dimer was done, which was elevated. Based on the findings from contrast-enhanced CT of the chest, transthoracic echocardiography, and fibreoptic bronchoscopy in the clinical context, a diagnosis of Ortner's syndrome was established.

The patient was managed with oxygen therapy, diuretics to alleviate volume overload, bronchodilators for symptomatic relief, and sildenafil to reduce pulmonary arterial pressure, along with anticoagulation therapy for pulmonary thromboembolism. The patient was successfully discharged after two weeks in a stable condition with optimized medical therapy and a structured follow-up plan. Over time, there was a gradual and sustained improvement in symptoms. Dyspnoea progressively diminished, with a corresponding improvement in exercise tolerance and daily functional capacity. Additionally, the hoarseness of voice resolved, with restoration of near-normal vocal quality after three months, indicating recovery of left vocal cord function. Overall, the clinical course following discharge was favorable, reflecting an effective response to treatment for pulmonary hypertension and pulmonary thromboembolism.

Discussion

Voice changes can occur when there is a disruption of the normal mechanisms involved in phonation. Such disturbances may be due to diseases affecting the larynx directly or conditions arising elsewhere that influence vocal cord function. Persistent hoarseness for more than two weeks, particularly in the absence of a recent respiratory infection, should prompt detailed clinical assessment. Paralysis of the vocal cords is often linked to conditions such as thoracic, thyroid, or oesophageal malignancies. However, it may also develop following surgery, traumatic injury, infections, inflammatory disorders involving the recurrent laryngeal nerve, neurological disease, or even tuberculosis of the larynx.[6] In a proportion of patients, no specific cause can be established despite extensive evaluation.[7]

Ortner's syndrome is a rare clinical entity characterized by the development of hoarseness of voice due to underlying cardiopulmonary pathology.[1] In most instances, the condition arises from extrinsic compression of the nerve by structures bordering the aortopulmonary window. Pulmonary artery enlargement associated with Ortner's syndrome may be seen in primary pulmonary hypertension[8], recurrent embolic events[9], and congenital heart anomalies.[10] Beyond hoarseness, this syndrome may present with other compressive symptoms such as dysphagia, as described by Shah *et al.*[11], in a patient with primary pulmonary arterial hypertension associated with Ortner's syndrome manifesting with unstable angina, oesophageal compression, and left RLN palsy, culminating in voice changes and difficulty in swallowing. Right atrial and ventricular enlargement were prominent in our patient, reflecting chronic hemodynamic overload. This sustained pressure burden facilitates right heart failure progression and further expansion of the pulmonary vasculature. Mechanistically, these cardiopulmonary alterations cause Ortner's syndrome via mechanical compression of the left recurrent laryngeal nerve by the engorged cardiac structures.

This case demonstrates that Ortner's syndrome may be reversible if the underlying vascular pathologies are promptly and adequately managed. In the present patient, a combination of pulmonary vasodilator therapy, anticoagulation, and diuretic treatment resulted in substantial clinical improvement.

Evidence demonstrates that effectively managing pulmonary hypertension can relieve mechanical compression of the left recurrent laryngeal nerve, which may facilitate partial or full restoration of vocal cord function.

Conclusion

This case underscores the importance of recognizing Ortner's syndrome as an uncommon but clinically meaningful cause of persistent hoarseness in patients with underlying cardiopulmonary pathology. It highlights the need for a high index of suspicion and a thorough multidisciplinary evaluation when vocal cord paralysis is identified, particularly in the absence of primary laryngeal disease. Early diagnosis and timely, targeted management of contributing conditions such as pulmonary arterial hypertension and pulmonary thromboembolism are crucial, as they can reduce vascular compression of the left recurrent laryngeal nerve. As demonstrated in this patient, appropriate therapy, including pulmonary vasodilators, anticoagulation, and supportive care, can result in substantial clinical improvement, not only in respiratory symptoms but also in restoration of vocal cord function. This case therefore emphasizes that, despite its rarity, Ortner's syndrome may be potentially reversible when the underlying hemodynamic disturbances are effectively addressed.

References

1. Verma S, Talwar A, Talwar A, Khan S, Krishnasastry KV, Talwar A. Ortner's syndrome: a systematic review of presentation, diagnosis and management. Intractable Rare Dis Res. 2023;12(3):141-47.
2. Dutra BL, Campos LC, Marques HC, Vilela VM, Carvalho RE, Duque AG. Ortner's syndrome: a case report and literature review. Radiol Bras. 2015;48(4):260-62.
3. Loughran S, Alves C, MacGregor FB. Current aetiology of unilateral vocal fold paralysis in a teaching hospital in the West of Scotland. J Laryngol Otol. 2002;116: 907-10.
4. Zhang W, Zhang J, Zhao L. Cardiovocal syndrome (Ortner's syndrome) caused by pulmonary hypertension: a case report. BMC Pulm Med. 2021;21(1):50.
5. Alouazen O, Chetoui A, Meqor A, Amar AO, Rachidi SA. Ortner's syndrome: hoarseness of voice revealing severe secondary pulmonary hypertension. Radiol Case Rep. 2024;19(12):6618-20.
6. Dixit R, George Jacob. Dysphonia in an asthma patient taking inhaled fluticasone: not always a side effect. Int J Tuberc Lung Dis 2010; 14(12): 1653.
7. Shahul HA, Manu MK, Mohapatra AK, Magazine R. Ortner's syndrome. BMJ Case Rep. 2014;2014: bcr2013200950.
8. Kagal AE, Shenoy PN, Nair KG. Ortner's syndrome associated with primary pulmonary hypertension. J Postgrad Med. 1975;21(2):91-95.
9. Albertini RE. Vocal cord paralysis associated with pulmonary emboli. Chest. 1972;62(4):508-10.
10. Stocker HH, Enterline HT. Cardio-vocal syndrome: laryngeal paralysis in intrinsic heart disease. Am Heart J. 1958;56(1):51-59.
11. Shah H, Shah K, Dixit R, Shah N, Shah K. Ortner's Syndrome with unstable angina and dysphagia secondary to primary pulmonary hypertension. Current Med Trends. 2004; 8: 1639-34.