



ORIGINAL RESEARCH PAPER

Otorhinolaryngology

ORTNER'S SYNDROME: A CARDIOVOCAL CONUNDRUM: CASE REPORT

KEY WORDS: Ortner's Syndrome, Cardiovascular Syndrome, Hoarseness, Rheumatic Heart Disease

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ABSTRACT

Background: Ortner syndrome (OS), or cardiovocal syndrome, is a rare clinical entity where hoarseness arises from the mechanical compression of the left recurrent laryngeal nerve (LRLN) by distorted or enlarged intrathoracic cardiovascular structures. While classically tied to mitral stenosis stemming from chronic rheumatic carditis, its current incidence remains low, representing under 0.6% of modern cardiovascular cases. **Case Description:** We present the case of a 42-year-old female with a childhood history of medically managed rheumatic heart disease who presented with a 6-month history of progressive, insidious dyspnea and worsening hoarseness. Video laryngoscopic evaluation confirmed left vocal fold immobility in the paramedian position. A high-resolution CT (HRCT) scan of the neck and thorax revealed severe cardiomegaly, marked left atrial dilatation (6.2 cm), and a prominent left atrial appendage compressing the nerve path. The patient was referred to cardiology for optimized management, resulting in notable improvement of her dysphonia at subsequent follow-up. **Conclusion:** Although historically classic, OS is easily overlooked in contemporary primary care. Clinicians must maintain a high index of suspicion for cardiovascular etiologies when evaluating unexplained vocal fold paralysis, ensuring comprehensive imaging across the entire anatomical course of the LRLN to avoid unnecessary laryngeal interventions.

INTRODUCTION

Vocal fold paralysis secondary to mechanical compression of the left recurrent laryngeal nerve (LRLN) was first documented in 1897 by Austrian physician Norbert Ortner in a patient suffering from mitral valve disease [1]. Over time, the scope of Ortner syndrome (OS)—often called cardiovocal syndrome—has expanded to include any benign thoracic or cardiovascular anomaly that stretches, compresses, or exerts traction on the recurrent laryngeal nerve path, leading to dysphonia [1].

Because of its specific loop under the aortic arch, the left recurrent nerve is roughly 1.75 times more susceptible to compression than its right-sided counterpart [2]. While left atrial enlargement secondary to rheumatic mitral lesions remains the textbook etiology, the modern syndromic definition encompasses conditions like pulmonary arterial hypertension, aortic aneurysms, aberrant right subclavian anomalies, and ductus arteriosus ectasia [3].

True cardiovocal hoarseness is rare; the vast majority of recurrent laryngeal nerve palsies stem from surgical trauma or malignant mediastinal invasion. Mitral stenosis develops as a delayed sequela of childhood rheumatic fever. If left unmanaged, the progressive valvular restriction can lead to severe hemodynamical changes [4]. While typical presentations involve dyspnea, hemoptysis, or chest pain, massive left atrial dilation can rarely manifest through mechanical compression of adjacent structures. This results in insidious, sometimes intermittent hoarseness that can advance to absolute aphonia if the nerve insult persists [5,6].

Furthermore, patients may occasionally exhibit dysphagia (dysphagia aortica) if the tense left atrium encroaches upon the neighboring esophagus or impacts the regional autonomic nerve plexuses, disrupting normal peristaltic patterns [7,8]. Literature indicates that OS accounts for up to 11% of all

non-traumatic, benign RLN palsies [9], though its overall prevalence among cardiac patients sits at a mere 0.5% to 0.6% (10). Mitral valve disease specifically is noted to be the primary driver in roughly 0.25% to 0.5% of comprehensive nerve palsy series [4].

Anatomically, the RLN originates from the vagus nerve (CN X) and follows a markedly asymmetric course. The right RLN splits at the root of the neck near the T1–T2 level, looping beneath the right subclavian artery before ascending within the tracheoesophageal groove. Conversely, the left RLN descends deep into the mediastinum, looping beneath the aortic arch adjacent to the ligamentum arteriosum before tracking superiorly back toward the larynx [11,12]. This anatomical vulnerability forms the structural basis for Ortner syndrome.

We report a classic yet increasingly rare presentation of isolated left vocal cord immobility secondary to severe left atrial dilatation driven by chronic rheumatic mitral stenosis.

OBJECTIVE

To identify and discuss a rare case of recurrent nerve palsy due to cardiac pathology.

Case Report

A 42-year-old female who is a known case of Rheumatic Heart Disease since childhood on regular medication, presented to the outpatient department with complaints of progressive dyspnea and a change in voice for the past 6 months.

The dyspnea was insidious in onset, progressive in nature, initially present on exertion, and later progressed to dyspnea at rest (NYHA Class I to Class IV).

The patient also reported hoarseness of voice and mild throat discomfort.

There was no history of fever, cough, hemoptysis, trauma, recent infection, or foreign body aspiration.

No history of smoking, alcohol intake, or previous similar episodes was noted.

Examination Findings:

- On general examination, the patient was conscious and oriented.
- Water Hammer pulse was observed, and auscultation revealed a low-pitched diastolic murmur at the apex, suggestive of mitral stenosis.
- Bilateral jugulodigastric lymphadenopathy was noted.
- Indirect laryngoscopy showed left vocal cord immobility in the paramedian position, with normal right vocal cord mobility.
- There was no visible laryngeal mass or mucosal abnormality.

Investigations

An HRCT scan of the neck and thorax was performed to trace the course of the nerve. The scan demonstrated severe cardiomegaly characterized by profound multi-chamber dilation. The anteroposterior (AP) diameters of the chambers were severely increased: the left atrium measured 6.2 cm, the right atrium measured 5.8 cm, and the right ventricle reached 8.1 cm. Notable prominence of the left atrial appendage was observed, encroaching directly onto the aortopulmonary window. Video laryngoscopy confirmed the examination findings.

Mild medialization of the left true vocal cord was documented, without any intrinsic laryngeal or pulmonary apical lesions.

DISCUSSION

Ortner syndrome represents a fascinating intersection between cardiovascular pathology and laryngology. While historically tied almost exclusively to rheumatic mitral stenosis, epidemiological shifts over the last few decades show that thoracic aortic aneurysms and primary pulmonary hypertension have overtaken valvular disease as the leading causes of cardiovocal syndrome. Modern registry data indicates that aortic aneurysms account for nearly 41% of OS cases, followed by pulmonary hypertension (35%), while mitral stenosis now represents roughly 17% [13]. Interestingly, among patients who undergo successful surgical or transcatheter correction of the underlying vascular anomaly, approximately 85.4% achieve partial or total resolution of their hoarseness [13].

The asymmetric course of the RLN explains the clear predisposition for left-sided pathology in these cases. Because the left RLN must clear the aortic arch and loop near the ligamentum arteriosum before ascending between the trachea and the esophagus, any expansion of the aortopulmonary space directly pinches the nerve fibers against the aortic wall.

Consequently, encountering isolated left RLN palsy without an obvious primary head, neck, or lung mass should immediately prompt a comprehensive cardiovascular workup. A routine chest X-ray is often the initial screening tool to rule out apical lung tumors or gross mediastinal widening; however, standard radiographs can appear completely normal in the aortopulmonary window in up to 72% of patients who are later found on CT to have definitive mechanical compression structures [14].

Therefore, cross-sectional imaging (CT or MRI) spanning from the brainstem down to the lower border of the aortic arch is essential for any idiopathic left vocal cord paralysis.

Clinical Significance

Because patients presenting with voice changes naturally seek care from otolaryngologists or primary care clinics, OS is prone to misdiagnosis or delayed recognition. Although the global burden of Rheumatic Heart Disease is decreasing due

to better antibiotic prophylaxis, it remains a major health concern in developing regions and can manifest with atypical, extracardiac symptoms.

This case highlights the varied presentations of chronic valvular disease and underlines why ENT clinicians must maintain diagnostic suspicion for cardiac abnormalities. Recognizing cardiovocal syndrome early prevents unnecessary, invasive laryngeal biopsies or interventions and redirects the patient immediately to life-saving cardiac care.

CONCLUSION

Ortner syndrome remains a clinically significant, diagnostic consideration for unexplained hoarseness secondary to recurrent laryngeal nerve compression. This case highlights the value of linking localized ENT symptoms to systemic cardiovascular diseases, specifically rheumatic mitral stenosis causing severe left atrial expansion. Utilizing high-resolution cross-sectional imaging of the neck and chest is crucial to confirm the diagnosis and rule out malignancy. Ultimately, managing the primary cardiovascular condition remains the main treatment path, often leading to excellent recovery of both cardiac function and vocal cord mobility. This case reinforces the need for close multidisciplinary collaboration between otolaryngologists and cardiologists.

Images

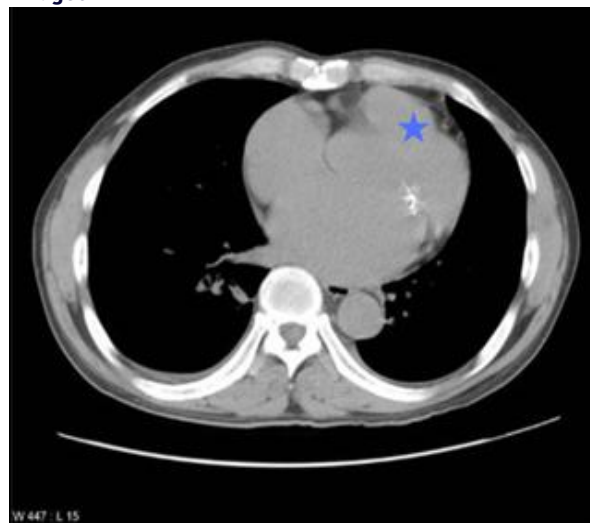


Fig 1. Cardiomegaly With Marked Left Atrial Enlargement, Producing A Prominent Left Cardiac Border/left Atrial Appendage.

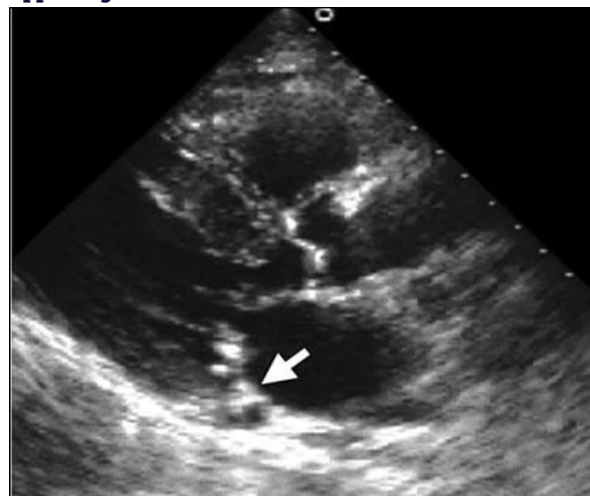


Fig 2. Two-dimensional Echocardiography Demonstrated Severe Mitral Stenosis with Left Atrial Enlargement and Pulmonary Hypertension.



Fig 3. Mild Medialization of Left True Vocal Cord.

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