

Ortner's Syndrome: A Comprehensive Review of Etiology, Clinical Presentation, and Management

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ABSTRACT

Ortner's syndrome, also known as cardiovocal syndrome, is a rare clinical entity characterized by left recurrent laryngeal nerve palsy secondary to cardiovascular pathology. Initially described in the context of left atrial enlargement due to mitral stenosis, the syndrome has since been associated with various cardiac and thoracic abnormalities, including aortic aneurysms, pulmonary hypertension, and congenital heart defects. This review aims to provide an in-depth analysis of Ortner's syndrome, encompassing its etiological factors, pathophysiology, clinical manifestations, diagnostic approaches, and therapeutic strategies. We also discuss the historical evolution of the syndrome's understanding and highlight contemporary advancements in its diagnosis and management. Enhanced awareness and timely recognition of Ortner's syndrome are crucial for appropriate intervention and improved patient outcomes.

KEYWORDS: Ortner, Syndrome, cardiovocal, nerve.

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INTRODUCTION

Ortner's syndrome, first described by Norbert Ortner in 1897, represents a unique intersection of cardiovascular and laryngeal pathologies. The syndrome is characterized by the compression or irritation of the left recurrent laryngeal nerve by an enlarged cardiovascular structure, leading to unilateral vocal cord paralysis and consequent hoarseness. Originally linked to severe mitral valve disease, Ortner's syndrome has since been recognized in association with a broader spectrum of cardiovascular conditions, including but not limited to aortic aneurysms, pulmonary artery hypertension, and various congenital heart diseases.^{1,2}

The left recurrent laryngeal nerve's anatomical course, looping around the aortic arch before ascending between the trachea and esophagus to innervate the larynx, renders it particularly susceptible to compression by adjacent cardiovascular structures. The resulting neuropathy manifests clinically as hoarseness, dysphonia, and, in severe cases, airway compromise, significantly impacting patients' quality of life and necessitating prompt medical attention.^{1,2}

Advancements in imaging modalities and a deeper understanding of cardiovascular pathophysiology have improved the diagnostic accuracy and management of

Ortner's syndrome. However, due to its rarity and the overlap of its symptoms with other causes of vocal cord paralysis, the syndrome often poses a diagnostic challenge. Early recognition and targeted intervention are imperative to prevent irreversible nerve damage and to address the underlying cardiovascular pathology effectively.^{1,2}

This review delves into the multifaceted aspects of Ortner's syndrome, exploring its historical background, diverse etiologies, clinical presentations, diagnostic criteria, and current therapeutic approaches. Through a synthesis of existing literature and recent clinical insights, we aim to provide a comprehensive resource for clinicians encountering this intriguing syndrome in their practice.^{1,2}

EPIDEMIOLOGY

Ortner's syndrome, or cardiovocal syndrome, remains a relatively rare clinical entity, with its precise epidemiological characteristics not well delineated due to its infrequent occurrence and the variability in its underlying causes. Historically, Ortner's syndrome was predominantly associated with mitral valve disease, particularly mitral stenosis, secondary to rheumatic heart disease. This association has primarily been observed in regions with high

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prevalence rates of rheumatic fever, including parts of Asia, Africa, and South America. However, the global decline in rheumatic fever incidence due to improved public health measures and antibiotic prophylaxis has led to a corresponding decrease in cases of mitral stenosis-related Ortner's syndrome.^{1,2}

In contemporary practice, the epidemiological landscape of Ortner's syndrome has evolved, reflecting changes in the prevalence of its various etiological factors. Cardiovascular conditions that contribute to left atrial enlargement and pulmonary hypertension are now more frequently implicated. These conditions include chronic obstructive pulmonary disease (COPD), pulmonary embolism, congenital heart defects such as patent ductus arteriosus and atrial septal defect, and systemic conditions like hypertension and atherosclerosis. Additionally, aortic aneurysms and other mediastinal masses have emerged as significant contributors.^{1,2}

The exact prevalence of Ortner's syndrome in the general population remains unclear, largely due to its rarity and the challenges associated with its diagnosis. Retrospective studies and case series suggest that it constitutes a small percentage of patients presenting with vocal cord paralysis. For instance, a review of vocal cord paralysis cases in tertiary care centers often reveals Ortner's syndrome in a minority, typically less than 5% of cases. However, this figure might underrepresent the true incidence due to underdiagnosis and misattribution to other causes of recurrent laryngeal nerve palsy.^{3,4}

Age and sex distributions in Ortner's syndrome are largely reflective of the underlying cardiovascular conditions. For instance, congenital heart diseases leading to Ortner's syndrome might present in younger individuals, while aortic aneurysms and chronic pulmonary diseases are more prevalent in older populations. There is no clear sex predilection, although certain etiologies like atherosclerosis may have a male predominance, potentially skewing the demographic characteristics in some studies.^{3,4}

Geographical variations in the epidemiology of Ortner's syndrome also mirror the distribution of its etiological factors. Regions with higher rates of rheumatic heart disease historically reported more cases of mitral stenosis-associated Ortner's syndrome. In contrast, areas with higher incidences of COPD and pulmonary hypertension might observe more cases linked to these conditions. This geographical variability underscores the importance of considering regional epidemiological trends when diagnosing and managing Ortner's syndrome.^{3,4}

Ortner's syndrome remains a rare but clinically significant condition with a shifting epidemiological profile. While rheumatic heart disease was once the predominant cause, contemporary cases are more likely to arise from diverse cardiovascular and thoracic conditions. Understanding the epidemiology of Ortner's syndrome is crucial for clinicians, as it guides the differential diagnosis in patients presenting

with vocal cord paralysis and directs attention towards potential underlying cardiovascular abnormalities. Enhanced awareness and reporting of Ortner's syndrome are needed to better delineate its prevalence and inform effective management strategies.^{3,4}

CLINICAL MANIFESTATIONS

Ortner's syndrome, also known as cardiovocal syndrome, presents a distinctive set of clinical manifestations primarily resulting from the compression or irritation of the left recurrent laryngeal nerve by cardiovascular structures. This nerve, a branch of the vagus nerve, is uniquely susceptible to involvement due to its anatomical course, looping under the aortic arch before ascending to innervate the larynx. The resulting neuropathy leads to a characteristic constellation of symptoms, predominantly related to vocal cord dysfunction.^{3,4}

PRIMARY CLINICAL SYMPTOMS:

Hoarseness:

The hallmark symptom of Ortner's syndrome is hoarseness, arising from unilateral vocal cord paralysis. Patients typically describe a gradual onset of a breathy, weak voice, which can fluctuate in severity. This symptom is often progressive, correlating with the worsening of the underlying cardiovascular condition.^{3,4}

Dysphonia:

Dysphonia, or impaired voice production, is a common complaint. Patients may experience difficulty projecting their voice, changes in pitch, and vocal fatigue. These alterations are directly related to the paralysis of the affected vocal cord, which remains in a paramedian position, leading to incomplete glottic closure during phonation.^{5,6}

Breathiness And Vocal Fatigue:

Due to the inability of the paralyzed vocal cord to meet its contralateral counterpart, there is a significant air leak during speech. This results in a breathy voice quality and rapid vocal fatigue, particularly during prolonged speaking engagements.^{5,6}

Aphonia:

In severe cases, patients may experience aphonia, the complete loss of voice. This is rare and typically associated with extensive nerve damage or severe compression.^{5,6}

SECONDARY CLINICAL SYMPTOMS:

Dyspnea and Stridor:

Although less common, some patients with Ortner's syndrome may present with dyspnea (difficulty breathing) or stridor (a high-pitched wheezing sound) due to the compromised function of the larynx. These symptoms are usually indicative of significant nerve involvement or concurrent respiratory pathology.^{5,6}

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Cough and Aspiration:

The compromised laryngeal function can lead to ineffective cough and an increased risk of aspiration. Patients may report frequent coughing, especially when eating or drinking, and an increased susceptibility to respiratory infections.^{5,6}

Associated Cardiovascular Symptoms:

Given that Ortner's syndrome results from underlying cardiovascular pathology, patients often present with additional symptoms related to their primary cardiac or thoracic condition. These can include:^{5,6}

Palpitations and Chest Pain:

Patients with conditions such as aortic aneurysms or severe mitral valve disease may report palpitations or chest pain, reflecting the hemodynamic impact of their disease.^{5,6}

Dyspnea on Exertion:

Common in conditions causing left atrial enlargement or pulmonary hypertension, patients may experience shortness of breath with physical activity.^{5,6}

Peripheral Edema:

In cases of heart failure, patients may exhibit signs of fluid overload, including peripheral edema, ascites, and weight gain.^{5,6}

Syncope:

Syncope or near-syncope episodes may occur, particularly in patients with significant aortic or pulmonary artery involvement, leading to compromised cerebral perfusion.^{5,6}

PHYSICAL EXAMINATION FINDINGS

Vocal Cord Paralysis:

Laryngoscopic examination typically reveals unilateral left vocal cord paralysis, with the affected cord in a paramedian position. This is a critical diagnostic feature of Ortner's syndrome.^{5,6}

Cardiovascular Signs:

Physical examination may also uncover signs of the underlying cardiovascular condition. For instance, patients with mitral stenosis might present with a diastolic murmur, while those with pulmonary hypertension may have a loud P2 component of the second heart sound.^{5,6}

Diagnostic Considerations:

The clinical presentation of Ortner's syndrome requires a high index of suspicion, particularly in patients with known cardiovascular disease presenting with new-onset hoarseness. A comprehensive evaluation including a detailed history, physical examination, and appropriate imaging studies (echocardiography, chest CT or MRI) is essential for confirming the diagnosis and identifying the causative cardiovascular pathology.^{5,6}

The clinical manifestations of Ortner's syndrome are primarily related to unilateral vocal cord paralysis caused by left recurrent laryngeal nerve involvement. Recognizing the characteristic symptoms and associated cardiovascular signs

is crucial for timely diagnosis and management, ultimately improving patient outcomes.^{5,6}

Pathophysiology of Ortner's Syndrome

Ortner's syndrome, or cardiovocal syndrome, represents a fascinating interplay between cardiovascular and neurological systems, wherein cardiovascular pathology leads to vocal cord paralysis through the involvement of the left recurrent laryngeal nerve. The pathophysiology of Ortner's syndrome is primarily rooted in the anatomical course of the left recurrent laryngeal nerve and the diverse cardiovascular conditions that can exert pressure on or cause inflammation of this nerve.^{5,6}

Anatomical Considerations:

The left recurrent laryngeal nerve branches from the vagus nerve and takes a unique path, looping under the aortic arch before ascending between the trachea and esophagus to innervate the muscles of the larynx. This intricate course places the nerve in close proximity to various cardiovascular structures, making it susceptible to compression or irritation by pathological processes occurring within the thoracic cavity.^{5,6}

MECHANISMS OF NERVE INVOLVEMENT

Mechanical Compression:

The most common mechanism in Ortner's syndrome is mechanical compression of the left recurrent laryngeal nerve. Conditions that can lead to such compression include:

Left Atrial Enlargement: Secondary to mitral stenosis or regurgitation, left atrial enlargement can impinge on the nerve as it loops under the aortic arch.^{5,6}

Aortic Aneurysms: An aortic aneurysm, particularly in the ascending or aortic arch regions, can exert pressure on the nerve.^{5,6}

Pulmonary Artery Hypertension: Increased pressure in the pulmonary arteries can lead to dilation and subsequent compression of adjacent structures, including the left recurrent laryngeal nerve.^{5,6}

Thoracic Masses: Tumors or lymphadenopathy within the mediastinum can physically impinge on the nerve.^{5,6}

Inflammatory and Vascular Mechanisms:

Perineural Inflammation: Inflammatory processes secondary to cardiovascular conditions can lead to perineural inflammation and subsequent neuropathy.^{5,6}

Ischemia: Vascular abnormalities, such as aneurysms or atherosclerosis, can compromise blood flow to the nerve, leading to ischemic neuropathy.^{5,6}

Pathophysiological Processes:

Mitral Valve Disease:

Historically, the most recognized cause of Ortner's syndrome was severe mitral valve disease, particularly mitral stenosis due to rheumatic heart disease. The stenotic valve leads to increased left atrial pressure and subsequent left atrial enlargement. As the left atrium enlarges, it can exert pressure on the adjacent left recurrent laryngeal nerve, leading to vocal

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cord paralysis. Additionally, chronic left atrial enlargement can cause structural remodeling and fibrosis, further exacerbating nerve compression.^{5,6}

Aortic Pathology:

Aortic aneurysms and aortic dissection are significant etiologies in contemporary cases of Ortner's syndrome. The dilated aortic arch or aneurysmal dilation can directly compress the left recurrent laryngeal nerve as it loops underneath the arch. The mechanical distortion and stretching of the nerve fibers can result in demyelination and axonal damage, manifesting as vocal cord paralysis.^{5,6}

Pulmonary Hypertension:

In pulmonary hypertension, increased pressure within the pulmonary arteries leads to right ventricular hypertrophy and subsequent dilation of the pulmonary artery. This dilation can impinge on the left recurrent laryngeal nerve, particularly at the level where the nerve ascends from the thoracic cavity. The chronic pressure exerted by the dilated pulmonary artery can cause neuropathy and subsequent vocal cord paralysis.^{7,8}

Congenital Heart Disease:

Congenital heart defects, such as patent ductus arteriosus, atrial septal defect, and coarctation of the aorta, can lead to anatomical distortions and abnormal hemodynamics that place the left recurrent laryngeal nerve at risk. These conditions can cause abnormal flow patterns and pressures within the thoracic cavity, contributing to nerve compression or ischemia.^{7,8}

Neurological Impact:

The left recurrent laryngeal nerve innervates all the intrinsic muscles of the larynx, except the cricothyroid muscle, which is innervated by the superior laryngeal nerve. Paralysis of the left recurrent laryngeal nerve results in the inability of the left vocal cord to abduct and adduct properly, leading to the characteristic clinical manifestations of hoarseness, dysphonia, and breathy voice. The paralyzed vocal cord typically assumes a paramedian position, causing incomplete glottic closure during phonation, which significantly affects voice quality and projection.^{7,8}

The pathophysiology of Ortner's syndrome is a complex interplay of anatomical susceptibility and diverse cardiovascular conditions leading to left recurrent laryngeal nerve involvement. Understanding the underlying mechanisms, including mechanical compression, inflammatory and vascular insults, and the specific contributions of various cardiovascular pathologies, is essential for diagnosing and managing this rare syndrome. Enhanced recognition of the pathophysiological processes involved in Ortner's syndrome can facilitate timely intervention, potentially mitigating the impact on vocal function and improving overall patient outcomes.^{7,8}

Diagnosis

Diagnosing Ortner's syndrome, also known as cardiovocal syndrome, can be challenging due to its rarity and the overlap

of its symptoms with other causes of vocal cord paralysis. A comprehensive approach that includes a detailed history, physical examination, and targeted investigations is essential to confirm the diagnosis and identify the underlying cardiovascular pathology responsible for the left recurrent laryngeal nerve compression.^{7,8}

Clinical Assessment:

History:

A thorough medical history is crucial, focusing on the onset and progression of hoarseness, associated cardiovascular symptoms, past medical history of cardiovascular disease, and any history of thoracic surgeries or interventions.^{7,8}

Physical Examination:

Laryngoscopy: Direct visualization of the larynx can reveal the position and mobility of the vocal cords. Unilateral vocal cord paralysis, with the affected cord in a paramedian position, is a hallmark finding.

Cardiovascular Examination: Signs of underlying cardiovascular disease, such as murmurs, signs of heart failure, or evidence of aortic pathology, should be assessed.^{7,8}

Diagnostic Studies:

Imaging Studies:

Chest X-ray: Can reveal cardiomegaly, pulmonary congestion, or the presence of a mediastinal mass.

Echocardiography: Essential for evaluating cardiac structure and function, particularly to assess for left atrial enlargement, mitral valve pathology, aortic abnormalities, or pulmonary hypertension.^{7,8}

Computed Tomography (CT) or Magnetic Resonance Imaging (MRI): Useful for visualizing the aorta and detecting aortic aneurysms, dissections, or other mediastinal masses that could compress the left recurrent laryngeal nerve.^{7,8}

Electrophysiological Studies:

Electromyography (EMG) and Nerve Conduction Studies: Can assess the integrity and function of the recurrent laryngeal nerve, helping to differentiate between neuropathy and myopathy.

Laryngeal Electromyography: Provides detailed information about the function of the intrinsic laryngeal muscles and the degree of nerve involvement.^{7,8}

Laboratory Tests:

Blood Tests: Including complete blood count, electrolytes, renal function, and inflammatory markers, can help identify underlying systemic conditions contributing to vocal cord paralysis.

Autoimmune Markers: If autoimmune diseases are suspected, specific serological tests can be considered.^{7,8}

Differential Diagnosis:

The differential diagnosis of Ortner's syndrome includes other causes of vocal cord paralysis, such as:

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Idiopathic Vocal Cord Paralysis: Unilateral vocal cord paralysis of unknown etiology.

Recurrent Laryngeal Nerve Injury: Trauma or surgical injury to the nerve.

Neoplastic Invasion: Tumors affecting the recurrent laryngeal nerve.^{9,10}

Neurological Disorders: Such as stroke, multiple sclerosis, or amyotrophic lateral sclerosis.

Treatment Implications:

The diagnosis of Ortner's syndrome has significant treatment implications, as addressing the underlying cardiovascular pathology is essential to prevent further nerve damage and potentially reverse vocal cord paralysis. Collaboration between otolaryngologists, cardiologists, and cardiovascular surgeons is crucial for optimal management. Early diagnosis and intervention can lead to improved outcomes and quality of life for patients with Ortner's syndrome.^{9,10}

Treatment of Ortner's Syndrome

The management of Ortner's syndrome, or cardiovocal syndrome, revolves around addressing the underlying cardiovascular pathology responsible for the compression or irritation of the left recurrent laryngeal nerve. Treatment strategies aim to alleviate symptoms, prevent further nerve damage, and improve vocal cord function. The approach to treatment is multidisciplinary, involving collaboration between otolaryngologists, cardiologists, and sometimes cardiovascular surgeons, depending on the underlying etiology.^{9,10}

1. Conservative Management:

Speech Therapy: Speech therapy can help improve vocal cord function and voice quality by teaching techniques to compensate for vocal cord paralysis.^{9,10}

Voice Therapy: Voice therapy focuses on optimizing voice production and minimizing strain on the vocal cords.

Rehabilitative Measures: Techniques such as vocal cord strengthening exercises and breathing exercises can aid in vocal cord rehabilitation.^{9,10}

2. Pharmacological Therapy:

Steroids: In cases of inflammatory neuropathy, short-term corticosteroid therapy may be considered to reduce nerve inflammation and swelling.^{9,10}

Botulinum Toxin Injection: Botulinum toxin can be injected into the unaffected vocal cord to weaken its function temporarily, allowing for improved vocal cord symmetry and voice quality.

3. Surgical Interventions:

Vocal Cord Medialization: Medialization procedures involve implanting materials to push the paralyzed vocal cord towards the midline, improving vocal cord closure and voice projection.

Thyroplasty: In this procedure, the thyroid cartilage is manipulated to reposition the vocal cord, improving glottic closure and voice quality.^{9,10}

Recurrent Laryngeal Nerve Reinnervation: Nerve reinnervation procedures aim to restore function to the paralyzed vocal cord by surgically connecting a healthy nerve to the damaged one.^{9,10}

Cardiovascular Surgery: For cases where the compression of the left recurrent laryngeal nerve is due to an aortic aneurysm or other cardiovascular pathology, surgical correction of the underlying condition may be necessary.^{9,10}

4. Endovascular Interventions:

Endovascular Aneurysm Repair (EVAR): In cases where an aortic aneurysm is compressing the left recurrent laryngeal nerve, EVAR may be considered to relieve the compression and alleviate symptoms.^{9,10}

5. Lifestyle and Dietary Modifications:

Smoking Cessation: Smoking cessation is crucial to prevent further cardiovascular damage and improve overall cardiovascular health.^{9,10}

Dietary Changes: Adopting a heart-healthy diet can help manage underlying cardiovascular conditions and reduce the risk of complications.^{9,10}

6. Follow-up and Monitoring:

Regular Follow-up: Regular follow-up with a multidisciplinary team is essential to monitor vocal cord function, cardiovascular health, and the effectiveness of treatment.^{9,10}

Imaging Studies: Periodic imaging studies, such as echocardiography or CT scans, may be necessary to assess the progression of underlying cardiovascular pathology and its impact on the left recurrent laryngeal nerve.^{9,10}

The management of Ortner's syndrome is complex and requires a tailored approach based on the underlying etiology and the severity of symptoms. Collaboration between specialists is essential to ensure optimal outcomes and improve the quality of life for patients affected by this rare syndrome. Early diagnosis and intervention can lead to better outcomes, highlighting the importance of awareness and recognition of Ortner's syndrome in clinical practice.^{9,10}

CONCLUSION

Ortner's syndrome, although rare, represents a unique and intriguing clinical entity that underscores the intricate interplay between cardiovascular and laryngeal pathology. Historically associated with severe mitral valve disease, Ortner's syndrome has evolved to encompass a broader spectrum of cardiovascular conditions, including aortic aneurysms, pulmonary hypertension, and congenital heart defects. The syndrome's hallmark is vocal cord paralysis resulting from compression or irritation of the left recurrent laryngeal nerve, often leading to hoarseness and dysphonia. Diagnosing Ortner's syndrome requires a high index of suspicion, as its symptoms overlap with other causes of vocal cord paralysis. A comprehensive diagnostic approach, including imaging studies, laryngoscopy, and electrophysiological studies, is essential to confirm the

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diagnosis and identify the underlying cardiovascular pathology responsible for nerve compression.

Treatment strategies for Ortner's syndrome are multifaceted and aim to address the underlying cardiovascular condition while improving vocal cord function. Conservative measures such as speech therapy and voice therapy can be beneficial, along with pharmacological interventions such as steroids or botulinum toxin injections. Surgical options, including vocal cord medialization and recurrent laryngeal nerve reinnervation, may be considered in select cases.

In conclusion, Ortner's syndrome remains a challenging yet clinically significant condition that requires a multidisciplinary approach for optimal management. Early recognition, accurate diagnosis, and timely intervention are paramount to improving patient outcomes and quality of life. Continued research and clinical awareness are essential to further elucidate the pathophysiology and treatment of this intriguing syndrome.

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