Review

Cardiovocal Syndrome: A Systematic Review

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Hoarseness associated with mitral stenosis was initially described by Ortner. Several cardiopulmonary conditions were associated with left recurrent laryngeal nerve palsy over the last 100 years; thus, the syndrome is termed as cardiovocal syndrome or Ortner's syndrome. This study aimed to classify the various predisposing conditions and to explain the pathophysiology and treatment opportunities available for these patients.

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Introduction

Nobert Ortner ascribed hoarseness of voice to the left recurrent laryngeal nerve palsy (LRLNP) in three patients with severe mitral stenosis.[1] He postulated that an enlarged left atrium was responsible for the recurrent laryngeal nerve palsy. Although it was initially associated with mitral stenosis, several other case reports suggested that hoarseness can be caused by a myriad of clinical situations (Table 1). The association of hoarseness with a cardiovascular pathology was termed as cardiovocal syndrome. The term cardiovocal syndrome was first comprehensively described in English journals in 1958 by Stocker and Enterline.[2]

Anatomy of Laryngeal Nerves

The nerves that supply the larynx are terminal branches of the vagus nerve. After entering the neck from the jugular foramen, the vagus nerve runs in the carotid sheath between the vein and the artery. It has two main branches that innervate the larynx.

The superior laryngeal nerve that runs behind the internal carotid artery divides into internal and external laryngeal nerves. The internal laryngeal nerve, along with the superior laryngeal artery, pierces the thyrohyoid membrane and serves as a sensory nerve supply to the pyriform fossa and mucous membranes above the vocal cords. The external laryngeal nerve runs along with the superior thyroid artery beneath the thyroid gland and supplies the cricothyroid muscle.

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Recurrent laryngeal nerve is also another terminal branch of the vagus nerve that innervates the larynx with a different course on each side. On the right side, it crosses the first part of the subclavian artery and hooks around to travel between the trachea and oesophagus. On the left, the recurrent laryngeal nerve arises from the left vagal trunk in the thorax when it crosses the arch of aorta and hooks around the ligamentum arteriosum and ascends in the groove between the trachea and the oesophagus. The recurrent laryngeal nerves supply all the muscles of the larynx except cricothyroid as well as sensory supply to the larynx below the vocal cords and the upper part of trachea.

Clinical Features of LRLN Palsy

The LRLN paralysis causes the left vocal cord to be in the paramedian position; on a laryngoscopy, the position can be variable. Symptoms include hoarseness, dysphagia, and shortness of breath during speech because of loss of air, which is secondary to glottic incompetence. Effective cough cannot be mounted. LRLN palsy can be a significant risk for aspiration because the paralysed vocal cord cannot protect from aspiration especially from liquids. The degree of symptoms depends on the extent of paresis and compensation by the other vocal cord. LRLN palsy during the immediate postoperative period can also lead to reduction in pulmonary function as a result of loss of natural positive end expiratory pressure (PEEP) that occurs with normal glottic closure.

Incidence

A prospective study from Scotland suggested that left sided recurrent laryngeal nerve palsy is more common than the right recurrent laryngeal palsy and it is more

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Table 1. *Clinical Conditions Associated with LRLN Palsy*

1. Congenital	Atrial septal defect Ventricular septal defect Double outlet right ventricle Eisenmenger's complex Patent ductus arteriosus Ebstein's anomaly Aortopulmonary window
2.Mitral valve disorders	Mitral stenosis Mitral valve prolapse Mitral regurgitation
3. Adult disorders	Left atrial enlargement Left ventricular aneurysm Pulmonary hypertension (primary as well as secondary) Ductus aneurysm Pulmonary embolism Thrombosed giant left atrium Tortuosity of great vessels Atrial myxoma
4. Aortic aneurysms	Saccular Atherosclerotic Pseudoaneurysms Dissections Traumatic Mycotic
5. Iatrogenic	Closure of patent ductus arteriosus Cardiac surgery Repair of aortic aneurysms Thoracic surgery Heart lung transplant Defibrillation Atrial fibrillation ablation procedure
6. Miscellaneous	Foreign body causing oesophago-broncho-aortic fistula

common in men and can occur in any age group. Lung cancer was the most common cause (42%) and surgical manipulation accounted for 24% of the cases. The idiopathic causes that included the cardiovocal syndrome accounted for less than 11% of the cases.[34] However, in the same study, LRLN palsy caused by lung cancer was noted to be high compared to the previous studies.

Conditions Associated with LRLN Palsy

Cardiovocal syndrome was described in various congenital abnormalities like atrial septal defect, ventricular septal defect, and type 2 aortopulmonary window.[3,4] It was also associated with double outlet right ventricle,[5] Ebstein's anomaly,[6] patent ductus arteriosus (PDA),[7] and Eisenmenger's complex.[8] Ductal ligation and transcatheter closure of PDA is associated with a risk of LRLN palsy.[9,10] Infants, who are less than 1500 g, premature, and younger, are have a higher risk of developing LRLN palsy.[9,11] The use of clips had a higher rate of LRLN palsy compared with the use of suture ligatures. Transcatheter approach for closure of PDA in a patient with long ductus and narrow diameter is associated with a higher risk for vocal cord palsy.[10]

Left atrial enlargement in association with giant thrombus,[12] mitral stenosis,[1] mitral valve prolapse,[13] mitral regurgitation,[14] and atrial myxoma[15] has been described to cause LRLN palsy. The incidence of cardiovocal syndrome in mitral stenosis ranges from 0.6% to 5%.[16] LRLN palsy has also been reported with cardiac defibrillation[17] and transcatheter ablation of atrial fibrillation.[18] Isolated ductus aneurysm can also be present in adults with hoarseness of voice.[19] Primary pulmonary hypertension[21] and several secondary causes of pulmonary hypertension including pulmonary embolism[20] can cause LRLN palsy. Left ventricular aneurysm[22] and tortuosity of the great vessels[23] in the mediastinum were also associated with left recurrent nerve palsy.

Aneurysms of aorta and the pulmonary artery[24] were reported to cause LRLN palsy. The aneurysms of the aorta can be traumatic,[25] saccular,[26] atherosclerotic, mycotic,[28] and pseudoaneurysm[27] and can have an associated dissection.[29] Repair of aneurysms have also been associated with this syndrome. The incidence of postsurgery hoarseness because of RLN palsy was 32%. The incidence, which was much higher when the surgery was done for type A aneurysms, was around 65%. Most patients still have hoarseness of voice six months after surgery.[30] Paediatric, adult cardiothoracic surgery,[32,33] and heart–lung transplant[31] has been associated with LRLN palsy. Foreign body, induced oesophago-bronchial-aortic fistula, has been associated with LRLN palsy.[35]

Pathophysiology

In 1990, Sunderland classified nerve injuries into five major types with prognostication with each class and modified the previous Sneddon's classification (Table 2). He suggested a classification depending on injury to different parts of the nerve. Class I injuries with virtually no damage to the nerve were associated with complete recovery, whereas class V injuries with disruption of perineurium had a dismal prognosis unless the offending agent is removed and surgical reconstruction of the nerve is done.[36]

Ortner initially postulated that enlarged left atrium pushing up the LRLN and compressing against the arch of aorta was responsible for the palsy. However, a series of careful autopsies and radiological studies in the early part of the 20th century disputed his hypothesis. On the basis of the autopsy studies, Fetterrolf and Norris showed that the distance between the aorta and pulmonary artery within the aortic window is only 4 mm and suggested that compression of the nerve between the two structures is responsible for palsy.[37] This hypothesis was further strengthened when compression of LRLN was observed by Ari et al. between aorta and pulmonary artery near the ligamentum arteriosum in patients with mitral stenosis undergoing mitral commissurotomy.[38] The authors of the present study believe that the causes of LRLN palsy can be broadly classified as mechanical, electrical or thermal energy leading to palsy and ischemic causes. Pressure on the LRLN by various structures in the mediastinum can cause palsy and chance of recovery depends on the degree and duration of injury. LRLN caused by defibrillation and

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