Mitral Regurgitation: A Rare Cause of Ortner’s Syndrome

SIBES KUMAR DAS, INDRANATH GHOSH, ANIRBAN DAS, SUBIR KUMAR DEY

ABSTRACT

Ortner’s syndrome (Cardiovocal syndrome) is characterized by left recurrent laryngeal nerve palsy due to cardiovascular diseases. Very rarely, it can be caused by mitral regurgitation. We are reporting here, a case of a 32-years old male who presented with a history of hoarseness of voice for the past 3 months. His physical examination revealed a pansystolic murmur in the apical area. Laryngoscopy revealed left vocal cord palsy. Echocardiography confirmed mitral regurgitation, left atrial enlargement and pulmonary arterial hypertension. He was offered surgical correction of the mitral regurgitation, but he refused it and was lost from the follow up.

Key Words: Left recurrent laryngeal nerve palsy, Mitral regurgitation, Hoarseness of voice

INTRODUCTION

Hoarseness of voice which is caused by the paralysis of the left recurrent laryngeal nerve as a result of cardiovascular causes known as the Ortner’s syndrome or the Cardiovocal syndrome. This was first described by Ortner in 1897 in two patients of left atrial enlargement due to mitral stenosis [1]. This condition is very common previously, due to the high frequency of untreated, rheumatic, valvular heart diseases. Though this condition is very rare now-a-days, it is still a diagnostic and therapeutic challenge. We are reporting here, a case of Ortner’s syndrome which was secondary to mitral regurgitation, due to its rarity in our settings.

CASE REPORT

A 32-years old non-smoker, non-alcoholic and normotensive male was referred to the Chest Outpatients Department by an ENT surgeon, with a history of progressive hoarseness of the voice for the past three months. He also had a history of progressive shortness of breath (Modified Medical Research Council grade-II) [2] and easy fatigability for the past two years. He had no significant past illness. On examination, he was found to have a regular pulse rate of 80 beats/min., a respiratory rate 20 breaths / min., and a blood pressure of 118/72 mmHg. There was no peripheral oedema, cyanosis, jaundice or clubbing. The examination of his cardiovascular system revealed a displaced hyperdynamic apex beat, a soft first heart sound and a pansystolic murmur at the apical area, which was radiating into the axilla. The examination of other systems revealed that they were within normal limits. Direct laryngoscopy showed left vocal cord palsy with paramedian positioning, without any abnormality in the larynx. His chest skiagram showed cardiomegaly [Table/Fig-1a] and his barium swallow studies [Table/Fig-1b]- lateral view revealed an enlarged left atrium with displacement of the oesophagus . His complete haemogram and blood biochemistry studies revealed those values to be within normal limits. A contrast enhanced CT scan of the thorax revealed cardiomegaly [Table/ Fig-2]. Fibreoptic bronchoscopy confirmed left vocal cord palsy [Table/Fig-3] but was otherwise normal. Upper gastrointestinal endoscopy revealed no abnormality. Echocardiography revealed grade 2+ mitral regurgitation, pulmonary hypertension (systolic pulmonary arterial pressure of 40 mm Hg), the left atrium to be 45 mm and a left ventricular ejection fraction of 55% [Table/Fig-4].

He was put on digoxin, metoprolol, ramipril and torsemide. His voice improved to some extent. He was advised to attend the
echocardiography), as originally postulated by Ortner, though here, was the enlarged left atrium (as was evidenced by radiography and pulmonary artery and the aorta near the ligamentum arteriosum. In our case, the cause of the left recurrent laryngeal nerve palsy and degeneration of the nerve fibres [1]. However, subsequent compressing it against the aortic arch, leading to ischaemic injury and degeneration of the nerve fibres [1]. However, subsequent investigators [9,10] have contradicted this theory and have concluded that the primary mechanism was the compression of the left recurrent laryngeal nerve between the hypertensive enlarged left pulmonary artery and the aorta near the ligamentum arteriosum. In our case, the cause of the left recurrent laryngeal nerve palsy was the enlarged left atrium (as was evidenced by radiography and echocardiography), as originally postulated by Ortner, though here, the underlying cause was mitral regurgitation. The ancillary factors that can contribute to the primary mechanism of the injury include mediastinitis, pericarditis, lymphadenopathy and scarring within the aortic window, and anatomic variations of the left bronchus and the ligamentum arteriosum [2]. Once the more common non cardiac causes of the left recurrent laryngeal nerve palsy, e.g., laryngitis, malignant invasion of the superior mediastinum, iatrogenic trauma, metabolic or neurologic causes are excluded, as were excluded in our case either clinically or by investigations like CT scan thorax etc. Ortner’s syndrome should be considered as a possibility, especially in patients with cardiovascular disorders. The natural cure of the disorder can be achieved by resolution of the symptoms if there is early detection and correction or treatment of the underlying abnormality. Our patient was offered medical treatment and was he advised mitral valve repair. His hoarseness improved to some extent on medical treatment, but he refused surgery which was advised for a radical cure.

DISCUSSION

Ortner’s syndrome is manifested by hoarseness of the voice which is caused by left recurrent laryngeal nerve palsy due to cardiac or great vessel disease. The incidence of the Cardiovocal syndrome is infrequent. Patients with mitral stenosis are at an increased risk of developing this disease, having a frequency of 0.6–5% [3]. However, it has been reported in other cardiopulmonary disorders including left atrial myxoma, mitral valve prolapse, aortic aneurysm, patent ductus arteriosus, primary pulmonary hypertension, atrial and ventricular septal defects, Eisenmenger’s syndrome, and recurrent pulmonary embolism [4-8]. Our patient did not have mitral stenosis, but had significant mitral regurgitation and pulmonary hypertension.

Ortner postulated that the enlarged left atrium which was caused due to mitral stenosis, pushed the laryngeal nerve upwards, compressing it against the aortic arch, leading to ischaemic injury and degeneration of the nerve fibres [1]. However, subsequent investigators [9,10] have contradicted this theory and have concluded that the primary mechanism was the compression of the left recurrent laryngeal nerve between the hypertensive enlarged left pulmonary artery and the aorta near the ligamentum arteriosum. In our case, the cause of the left recurrent laryngeal nerve palsy was the enlarged left atrium (as was evidenced by radiography and echocardiography), as originally postulated by Ortner, though here, the underlying cause was mitral regurgitation. The ancillary factors that can contribute to the primary mechanism of the injury include mediastinitis, pericarditis, lymphadenopathy and scarring within the aortic window, and anatomic variations of the left bronchus and the ligamentum arteriosum [2]. Once the more common non cardiac causes of the left recurrent laryngeal nerve palsy, e.g., laryngitis, malignant invasion of the superior mediastinum, iatrogenic trauma, metabolic or neurologic causes are excluded, as were excluded in our case either clinically or by investigations like CT scan thorax etc. Ortner’s syndrome should be considered as a possibility, especially in patients with cardiovascular disorders. The natural cure of the disorder can be achieved by resolution of the symptoms if there is early detection and correction or treatment of the underlying abnormality. Our patient was offered medical treatment and was he advised mitral valve repair. His hoarseness improved to some extent on medical treatment, but he refused surgery which was advised for a radical cure.

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AUTHOR(S):
1. Dr. Sibes Kumar Das
2. Dr. Indranath Ghosh
3. Dr. Anirban Das
4. Dr. Subir Kumar Dey

PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Respiratory Medicine, Medical College & Hospitals, West Bengal, India.
2. Assistant Professor, Department of Respiratory Medicine, Calcutta National Medical College & Hospital, 24, Gorachand Road, Kolkata, West Bengal-700 014, India.
3. Assistant Professor, Department of Respiratory Medicine, Medical College & Hospitals, West Bengal, India.
4. Corresponding Author

NAME, ADDRESS, TELEPHONE, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Subir Kumar Dey
Professor and HOD, Department of Pulmonary Medicine, Calcutta National Medical College, 24-Gorachand Road P.S. Beniapukur, Kolkata -700014, West Bengal, India.
Phone: +91-9831169389
E-mail: deysk2000in@yahoo.com; subirdhey2011@gmail.com
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None.