Case Report: Ortner’s Syndrome: A Rare Cause of Hoarseness.

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Abstract: We report the case of a 71 years old lady with a history of hoarseness of voice due to aortic arch aneurysm, one of the causes of the cardiovocal syndrome. 

Key Words: Cardiovocal hoarseness, Ortner’s syndrome, Aortic aneurysm, Mitral stenosis.

Introduction:
Cardiovocal syndrome or Ortner’s syndrome is a clinical condition with hoarseness attributable to left recurrent laryngeal nerve palsy in variety cardiovascular diseases like aortic aneurysm, patent ductus arteriosus, primary pulmonary hypertension, atrial and ventricular septal defect, Eisenmenger’s syndrome and recurrent pulmonary embolism and mitral stenosis.

Case Report
A 72 years old lady, a home maker, and a hypertensive on treatment, presented with change in voice (Hoarseness) for 3 months which was gradually progressive. There was no history of difficulty in breathing or difficulty in swallowing. There were no history of trauma and nor of any addiction. Cardiac examination revealed normal heart sounds S1 and S2 with ejection systolic murmur of grade IV/VI radiating to the carotids. There was no neurological deficit. All blood investigations were within normal limits. ECG showed frequent ectopies. 2D echo showed a normal mitral, aortic, pulmonary and tricuspid valve with trace mitral and tricuspid regurgitation, and mild concentric left ventricular hypertrophy.

Patient was subjected for flexible nasopharyngoscopy which showed paralysis of the left vocal cord paralysis with phonatory gap. X-Ray chest showed mild cardiomegaly with clear lung fields. CT scan of neck showed a normal appearance of the soft tissue structure of the neck. CT thorax showed pseudo aneurysm of the arch of aorta with surrounding thrombus and left recurrent laryngeal nerve compression. CT chest angiography showed a saccular aneurysm of size 4.0X3.5 with peripheral thrombus. Patent lumen showed a size of 2.7X2.0 cm. It was seen protruding inferiorly and to the left side. Aneurysm was seen arising 2.5 cm distal to the origin of left subclavian artery. The Descending aorta and right brachiocephalic artery was mildly sclerosed. Myocardial perfusion SPECT-Post DPM Stress and rest study stress protocol (post dipyridamole) showed normal myocardial perfusion scan and normal resting LVEF.

Figure 1: 4.0X3.5 cm saccular aneurysm with peripheral thrombus as marked by yellow arrow.
Discussion
Cardiovocal syndrome is a clinical entity manifested by hoarseness caused by an impaired ability of the left recurrent laryngeal nerve to transmit impulse to the laryngeal musculature because of stretching or impingement of the nerve from the disease induced changes in cardiac or greater vessel anatomy. Hoarseness of voice due to paralysis of the left recurrent laryngeal nerve caused by a dilated left atrium in mitral stenosis was first discussed by Nobert Ortner in 1897. A variety of cardiac problems can lead to paralysis of the left recurrent laryngeal nerve, these include thoracic aortic aneurysm, patent ductus arteriosus, primary pulmonary hypertension, trial and ventricular septal defect, Eisenmenger’s syndrome and recurrent pulmonary embolism. In mitral stenosis it ranges from .6 to 5%. Leonetti reported that 6 of 168 patients (4.8%) with thoracic aortic aneurysm presented with hoarseness and all of these had type I aneurysms (de baek classification) involving the ascending root and aortic arch.

The onset of hoarseness is usually insidious and, in the beginning it may be intermittent, but in due course, it progresses to complete aphonia. To understand how cardiovocal syndrome arises, one has to consider the anatomical position of the recurrent laryngeal nerve and the left atrium. The lengthy course of the recurrent laryngeal nerve in the thoracic cavity especially around the aortic arch makes it vulnerable to compression, traction and erosion by enlarged or displaced cardiac chambers, dilated pulmonary arteries and a dilated aorta. Contrary to its name, the left atrium does not lie on the left side but forms the most posterior chamber of the heart. It is closely related to the esophagus, spine, left recurrent laryngeal nerve, pulmonary vessels, lung parenchyma and bronchi. Therefore, when the left atrium enlarges, it causes complication by compressing the adjacent structures.

Treatment and prognosis for this syndrome depends on the possibilities of managing the underlying cause. It is not necessary to perform any additional surgical procedure on the vocal fold if the patient shows sufficient improvement during speech therapy. Definitive treatment should be considered if aspiration is severe or if no improvement is visible after alleviation of the cardiac problem.

Conclusion
Cardiovocal syndrome is rare in the general population but associated with causes such as aneurysms and mitral valve stenosis. A high index of suspicion is needed to make an early diagnosis which can lead to surgical correction of the potentially life threatening, underlying cardiovascular disease. Following such treatment, the cardiovocal syndrome usually resolves spontaneously as vocal cord function returns, often without a need for additional invasive treatment.

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References