Common Symptom: Uncommon cause

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**Introduction:** Hoarseness of voice is a very common condition and is caused by recurrent laryngeal nerve palsy. Many possible causes of unilateral vocal cord paralysis are known, including malignant or less often benign tumors, iatrogenic injury, inflammation, or post radiation fibrosis causing nerve palsy due to encasement. Cardiovocal syndrome or Ortner’s syndrome is hoarseness due to left recurrent laryngeal nerve palsy caused mainly by mechanical affection of the nerve from enlarged cardiovascular structures. This interesting case report discusses a patient who developed hoarseness of voice due to venous thromboembolism of pulmonary venous system. Cardiovocal syndrome subsequent to pulmonary hypertension secondary to venous thromboembolism is exceptionally rare.

**Case report:** A 53 year old male presented to his primary for breathlessness experienced during his morning walks. He had history of eosinophilia with seasonal cough and cold. Patient had been never smoker, tee-totalar with atheletic built. Hemogram, Chest xray, spirometry, Echocardiogram and TMT were done.

Chest skiagram was normal (Fig 1).Spirometry revealed mild airway obstruction (Fig2).2D echo was reported to have normal LV function with mild RA/RV dilatation. TMT was negative for reversible ischemia with tachycardiac response. Hemogram revealed mild eosinophilia which was usual as per history. He was put on treatment for asthma with inhaled bronchodilator plus steroid, antihistaminics and diethylcarbazine(DEC).
The patient experienced worsening of his breathlessness. Repeat assessment with chest x-ray (Fig3) and CT scan (Fig4) showed Left parahilar consolidation with breakdown. He was prescribed anti tubercular treatment (ATT) empirically.

However even after 2 months of treatment his symptoms persisted and hoarseness appeared at this point of time. He was referred for ENT evaluation. Videolaryngoscopy revealed fixed LVC (Fig 5). Cause of hoarseness was not clear as there was no mediastinal mass or lymphadenopathy in the CT scan. Repeat chest skiagram (Fig6) after completion of intensive phase of ATT showed progression of the lesion with appearance of large LUL cavity. This was confirmed by CT thorax (Fig7).

He was further investigated and CT pulmonary angiogram (Fig8a,b) was done revealing extensive pulmonary thromboembolism (PTE) and venous Doppler lower limbs showing evidence of DVT. 2D echo measures PASP>60mmHg with gross dilatation of RA and RV. He was treated with low molecular weight heparin to which his symptoms responded dramatically. Repeat echo cardiogram showed decrease in pulmonary artery systolic pressure.

Left Upper Lobe cavity remained unexplained primarily to PTE and it was investigated further. Bronchoscopy showed normal airways and bronchial wash from left upper lobe was negative for AFB and malignancy. LUL wedge resection was done. Histopathology of lung specimen was reported to have inflammatory tissue with thrombosis in supplying vasculature.

Patient was kept on oral anticoagulant monitored with PT/INR. His hoarseness disappeared in next follow up after two weeks. He could resume his active life and job of bank manager.

Discussion:

Cardiovocal syndrome or Ortner’s syndrome is hoarseness due to left recurrent laryngeal nerve palsy caused mainly by mechanical affection of the nerve from enlarged cardiovascular structures [1]. Cardiovocal syndrome was originally described in 1897 by Nobert Ortner in three patients with severe mitral stenosis [2]. Later it has been encountered with other mediastinal structures causing mass effect [3,4] and in many cardiac conditions for example, congenital heart diseases, mitral valve disorders, ventricular and aortic aneurysms, atrial enlargement and in iatrogenic conditions [5]. Cardiovocal syndrome caused by idiopathic pulmonary artery hypertension and dilated pulmonary trunk has also been described in the literature [6–8]. Pathophysiological mechanism of this syndrome is thought to be compression of the left recurrent laryngeal nerve between the aorta and dilated pulmonary artery [7] our patient developed hoarseness preceded by cough and breathlessness. Hoarseness lead to think both the family as well as the treating physician of more sinister etiology. The presence of thick walled left upper lobe cavity prima facie directed thoughts towards lung malignancy. However, investigations lead to final diagnosis of pulmonary hypertension secondary to venous thromboembolism. To the best of our knowledge cardiovocal syndrome associated with
pulmonary embolism is a very rare condition and it has been described with extreme rarity [9, 10].

**Conclusion:**

Cardiovocal syndrome is a rare cause for left recurrent laryngeal nerve palsy. Hoarseness subsequent to cardiovascular cause is of much higher consequence for the individual than hoarseness itself as in the presented case. Radiological imaging, computerised tomography of chest in particular, is needed to differentiate the syndrome from other causes such as mediastinal masses or lung cancer.
Figures:

Fig 1. Chest X-ray: Unremarkable

Fig 2. PFT: Mild obstruction

Fig 3. Chest X-ray: LPH opacity

Fig 4. CT scan: LUL cavity

Fig 5. Videolaryngoscopy: LVC palsy
Fig. 6. Chest X-ray: Progressive lesion

Fig. 7. CT scan: Thick walled cavity (LUL)

Fig. 8(a, b). CT Pulmonary Angiogram: Thrombi in pulmonary artery
References:


