

Unilateral Vocal Cord Immobility in a Massive Pericardial Effusion Secondary to Lung Carcinoma

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ABSTRACT

Ortner syndrome or cardiovocal syndrome is characterized by vocal cord immobility due to recurrent laryngeal nerve palsy resulted from a cardiovascular pathology. It is due to the compression of the recurrent laryngeal nerve along its pathway, from the enlargement of the pulmonary artery, aorta and dilatation of left atrium. We report a middle-aged lady presented with hoarseness after four months of breathlessness. Laryngoscopy revealed that left vocal cord immobility and computed tomography showed a globular-shaped heart with a lung mass and multiple mediastinal lymphadenopathies. We highlight the multiple possible causes for the Ortner syndrome in this case.

KEYWORDS: Ortner syndrome, cardiovocal syndrome, hoarseness, lung carcinoma

INTRODUCTION

Ortner syndrome is not an uncommon pathology. Norbert Ortner in 1897 first described it after he found the association of hoarseness in 3 patients with mitral stenosis. The mitral stenosis caused the left atrium dilatation and subsequently compression to the left recurrent laryngeal nerve (RLN) [1]. However, this cardiovocal syndrome aetiology may arise from non-cardiogenic causes as well. Some pathological lesions of the mediastinal contents can impinge the RLN as well, especially on the left.

CASE PRESENTATION

A 58-year-old lady presented with progressive breathlessness for four months. It was associated with reduced effort tolerance, dry cough, and orthopnea and weight loss. There were also recurrent aspiration

episodes in one-month duration. She denied smoking history or passive smoking, no noisy breathing, no odynophagia and no family history of malignancy. There was no history of intubation. The breathlessness worsened despite treatment with oral antibiotics, which she took from the recent hospitalization for community-acquired pneumonia.

At the emergency department, the clinical examination, chest radiography (Figure 1) and Focused Assessment with Sonography for Trauma (FAST) scan revealed a massive pericardial effusion. She was admitted and had pericardial catheter insertion with 1065 ml of hemorrhagic fluid drained. An echocardiogram post pericardiocentesis revealed minimal pericardial effusion with diastolic dysfunction grade II.

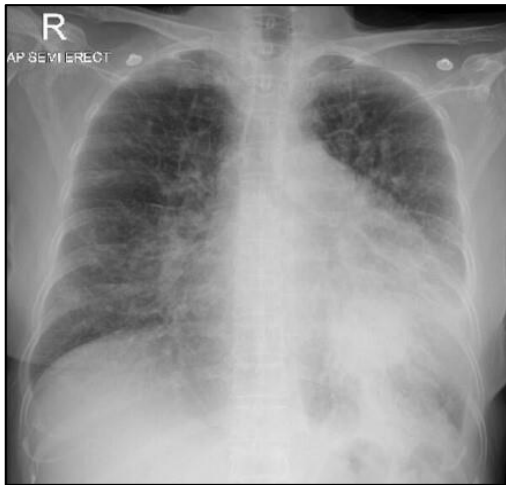


Figure 1 Chest radiograph showing the globular shape of the heart

After the procedure, she complained of voice change. She was referred to the otolaryngologist (ORL). Upon voice assessment, the hoarseness was persistent and associated with vocal fatigue and required multiple breaths to converse in a sentence. There was no audible stridor heard but noticed poor cough effort. Neck

examination revealed a right supraclavicular mass measuring 3x3cm, firm, not mobile with normal overlying skin. Voice assessment using GRBAS (Grade, Roughness, Breathiness, Asthenia, Strain scale) score was 3, with the main component of breathiness. She could only count 1 to 3 in a single breath and maximum phonation time was 3 seconds. Laryngoscopy revealed left vocal cord immobility with the presence of glottal gap during phonation.

Later, a contrasted computed tomography (CT) of neck and thorax revealed bilateral heterogeneous lung mass at the right middle lobe and left lower lobe with multiple enlarged mediastinal lymph nodes at level 2R, 4R, 5, 6 and 7 (Figure 2). The cytological study of the pericardial fluid revealed malignant cells with the possible primary site being the lung. She was planned for CT-guided lung biopsy to confirm the diagnosis. She was also offered injection laryngoplasty to medialize the paralyzed vocal cord to improve her voice. However, she refused both because of the invasiveness of the procedures in nature.

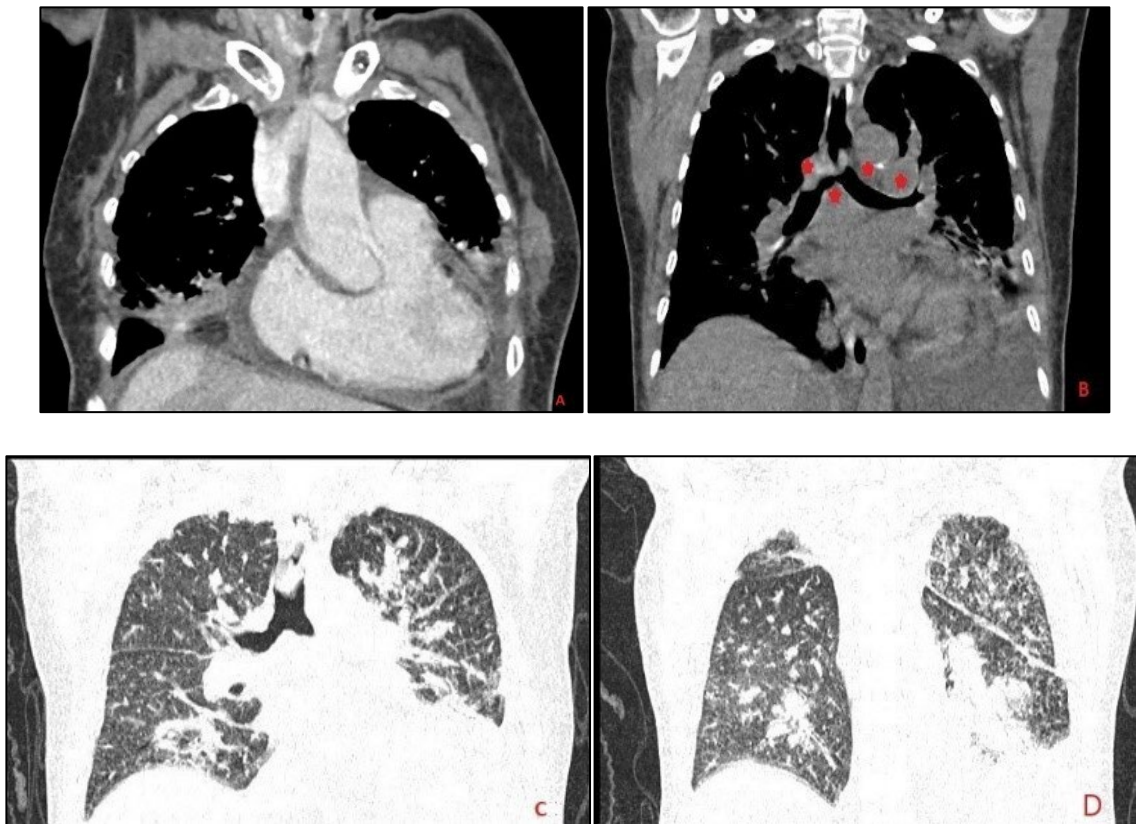


Figure 2 Contrast CT thorax showed the pericardial effusion with multiple lung lesion, coronal view (A), mediastinal lymph nodes enlargement at level 4R, 5, 6 and 7 (red asterisk), coronal view (B) and multiple lung masses seen bilaterally in high-resolution CT lung, coronal view (C, D)

DISCUSSION

Hoarseness is a common presentation in ORL clinic due to impairment of the vocal cord mobility. The vocal cord immobility mechanism either from innervation problem or mechanical fixation of the cord [2]. The underlying cause of this problem should be identified to decide the definitive treatment plan. Complete history and physical examination, including endoscopic examination, are essential to find the causes. Laryngoscopy is a procedure done in a clinic setting to assess the mobility of the vocal cord and to look for the presence of any supraglottic or glottic mass that might obstruct the vocal cord mobility. We have picked up all the crucial signs from our examination in this patient, which was later confirmed with the laryngoscopic evidence.

Other investigations such as an echocardiogram, angiogram, direct laryngoscopy, bronchoscopy and esophagoscopy are to be done in investigating the aetiology of unilateral vocal cord palsy depend on the clinical suspicion [2]. An echocardiogram is a non-radiating and fast tool to detect the possible cardiac cause in the Ortner syndrome by identifying cardiac abnormalities such as chamber dilatation, vascular dilatation, valvular problem and presence of pericardial effusion [3]. If the cause of hoarseness is identifiable in echocardiogram, CT or MRI can be deferred to avoid radiation exposure and minimize the cost.

A chest radiograph is a primary radiology assessment that could suggest few pathologies in the thoracic cavity such as the widening of the mediastinum or any lung lesion that possible affecting the recurrent laryngeal nerve. However, it has a limitation in assessing the complex structure such as inside the mediastinal structures. In our patient, chest radiography was done as one of the initial investigations for breathlessness. It revealed cardiomegaly; however, it did not give much information regarding the hoarseness's possible aetiology. In addition, the echocardiogram also showed no valvular lesion or any heart chamber dilatation. Thus, CT is a valuable modality in viewing the course of RLN and detect the pathological location and its extension, especially in the thoracic cavity [4].

RLN is a pair of nerves branching from the vagus nerve that innervates the larynx's ipsilateral intrinsic muscles. Left RLN is more vulnerable to injury due to the long course compared to the right side. The right RLN hooks at the right subclavian artery and left RLN at the arch of aorta, lateral to ligamentum arteriosum, before both ascend inside tracheoesophageal groove to the larynx.

Enlargement of the left atrium, aorta and pulmonary artery dilatation are sequelae to the mitral valve pathology and other pathologies such as aortic aneurysm and septal heart defect causing stretching and compression to the left RLN in the Ortner syndrome. Besides that, diverse causes that are non-cardiac condition were reported such as enlargement of mediastinal lymph nodes especially at superior mediastinal nodes (level 2 to 6), compression from the left bronchus, pulmonary artery atherosclerosis [1] and pericardial effusion [5]. Thus, contrasted CT from the base of the skull until thorax is a useful diagnostic tool beside the MRI.

Ortner syndrome in a case of pericardial effusion is rarely reported. The RLN might be injured due to the pressure over the pulmonary artery caused by the pericardial effusion [5]. In our case, we believed the pericardial effusion due to the metastasis of the lung carcinoma to the pericardium. The pericardium is a common site of metastasis in a lung, breast carcinoma, lymphoma, leukaemia and melanoma [5]. Pericardiocentesis is a minimally invasive procedure to remove the pericardial effusion to normalize the cardiac output. Subxiphoid, apical and parasternal approaches can be used for the aspiration depending on the collection site. It is unlikely to cause an injury to the RLN as the puncture sites are targeted away from the RLN courses. In our case, the pericardiocentesis was done at subxiphoid approached, which is away from RLN.

In addition, we believed the RLN injury was present earlier before the procedure, evidenced by bowing of the left vocal cord during laryngoscopy examination and muscle atrophy from the denervation that indicate it was a chronic process and the aspiration symptoms that already happened for the past one month also a clinical sign of uncompensated unilateral vocal cord immobility. It is also unlikely the lung lesion

causing the RLN palsy as from the CT study most of the lung lesions located at the lower lobe of both lungs, where the RLN more superiorly. Mediastinal lymph nodes enlargement can cause compression to the RLN, especially at the lymph nodes at level 5, 6 on the left side and 2R on the right side where it is closely related with RLN course. In our case, the impingement of the left RLN may also be contributed by the lymphadenopathy at level 5 and 6.

Despite the malignant cause of the unilateral vocal palsy, there is still a chance to reverse the voice problem. Surgical management of the unilateral vocal cord immobility is indicated by the history of aspiration pneumonia and the patient's desire to improve the voice quality [6]. Temporary management with injection laryngoplasty recommended in a high degree of vocal disability, high vocal demand, a small glottic gap which is 2-3mm with no posterior glottic gap and dysphagia. It is recommended to manage the straining during vocalization and swallowing problem prior to the definitive surgery. As for our patient, the injection laryngoplasty may help manage her aspiration symptoms and improve her voice quality.

CONCLUSION

There are many potential etiologies in this case. The massive pericardial effusion, the lung carcinoma, the mediastinal node or any cardiac intervention might be the possible cause. Ortner syndrome in a massive pericardial effusion is an uncommon pathology. The prognosis of unilateral vocal cord immobility depends on the aetiology and the duration of the illness. As in our case, despite its malignant aetiology, the voice dysfunction still can be reversible.

Conflict of Interest

Authors declare none.

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Authors' contribution

NSM and NAMU were involved in writing the case report while NFHNH, WYHWI and IM contributed the critical review for the case report. All authors read and approved the case report before the submission.

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