

# Pulmonary Arterial Hypertension (PAH) Secondary to Post-Tuberculosis Lung Fibrosis as a Rare Cause of Ortner's Syndrome

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## ABSTRACT

Hoarseness in tuberculosis (TB) patients can arise from vocal cord inflammation or recurrent laryngeal nerve palsy. Post-TB sequelae can be categorised into parenchymal, airway, pleural / chest wall, vascular, and mediastinal lesions based on radiological findings. However, pulmonary arterial hypertension (PAH) remains relatively underexplored as a complication of TB. PAH can lead to the dilatation of pulmonary arteries, resulting in the compression of the recurrent laryngeal nerve and leading to Ortner's syndrome. We present a case of a 35-year woman with a history of treated pulmonary TB who developed hoarseness due to left recurrent laryngeal nerve paralysis secondary to PAH-induced pulmonary artery enlargement, a rare cause of Ortner's syndrome. Imaging studies confirmed bronchiectasis and severe PAH. In a TB endemic area, clinicians must consider Ortner's syndrome as a rare but possible presentation of post-TB complication.

**Key Words:** Pulmonary arterial hypertension, Recurrent laryngeal Nerve palsy, Tuberculosis, Pulmonary TB.

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## INTRODUCTION

Hoarseness can occur among tuberculosis (TB) patients due to either inflammation of the vocal cords resulting from TB or recurrent laryngeal nerve palsy caused by lymph node enlargement.<sup>1,2</sup> Idiopathic pulmonary hypertension can lead to compression of the recurrent laryngeal nerve due to pulmonary arterial dilatation, resulting in Ortner's syndrome.<sup>3</sup> Post-TB sequelae can be categorised into parenchymal, airway, pleural / chest wall, vascular, and mediastinal lesions based on radiological findings.<sup>4</sup> However, pulmonary arterial hypertension (PAH) remains relatively underexplored as a complication of pulmonary TB.<sup>5,6</sup> This case report describes a case of cardiovocal syndrome (Ortner's syndrome) in a patient with pulmonary TB and PAH.

## CASE REPORT

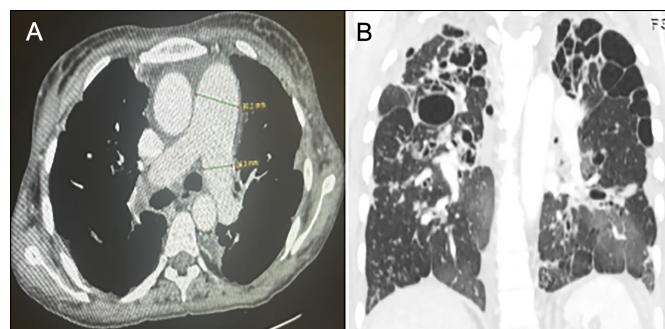
A 35-year woman presented with a history of occasional non-productive cough over six months, and worsening shortness of breath, abdominal distension, leg swelling, and hoarseness of voice for two months. She had no history of chest pain, fever, or haemoptysis. She had a history of pulmonary TB treated 25 years ago.

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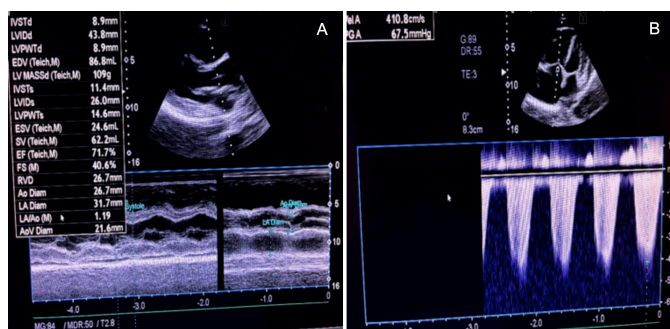
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**Figure 1: (A) CT scan chest axial view (mediastinal window) showing enlarged main pulmonary artery (diameter 39.1 mm) and left branch of pulmonary artery (diameter 26.3 mm). (B) Plain CT scan chest axial and coronal view showing cystic traction bronchiectasis involving bilateral upper lobe, with associated mosaic attenuation and air trapping in bilateral lung fields.**

On examination, she had bilateral end-inspiratory coarse crepitations in the chest, and central trachea, reduced chest expansion, and signs of the right heart strain including raised jugular venous pulse, right ventricular heave, loud P2, pedal oedema, and ascites. Arterial blood gas analysis revealed chronic respiratory acidosis. Sputum tests for active TB were negative. Imaging showed bronchiectasis in bilateral lung fields with associated mosaic attenuation, air trapping, and enlarged pulmonary artery consistent with the findings of long-standing pulmonary TB sequelae with PAH (Figure 1 A and B). Transthoracic echocardiography showed severe PAH with dilated right atrium, right ventricle, and pulmonary artery, severe tricuspid regurgitation and mean pulmonary artery pressure of 55 mmHg suggestive of grade III-PAH and cor-pulmonale (Figure 2 A and B). She

was severely dyspnoeic and unable to perform lung function tests such as spirometry. Fibre-optic laryngoscopy indicated left vocal cord paralysis due to recurrent laryngeal nerve involvement. She was managed with inhaled bronchodilators, oxygen therapy, and triple therapy (digoxin, spironolactone, and sildenafil), leading to improvement in symptoms over two months of follow-up.



**Figure 2: (A) Echocardiography showed a dilated right ventricle with size of 26.7 mm. (B) On colour Doppler imaging, there is a peak systolic tricuspid pressure gradient of 67.5 mm Hg suggestive of pulmonary arterial hypertension (PAH).**

## DISCUSSION

More than a century ago, Ortner presented a case in which he explained that compression of the recurrent laryngeal nerve by a dilated left atrium can cause hoarseness of voice. Since then, any bronchoalveolar, cardiac, or intrathoracic process that causes compression or stretching of the laryngeal nerve can cause vocal fold paralysis, now known as Ortner's syndrome.<sup>7</sup> The left recurrent laryngeal nerve, a branch of the vagus nerve, is found inferior to the aortic arch and posterior to the ligamentum arteriosum. It ascends between the trachea and oesophagus; thus, the anatomical location of this nerve makes it more prone to entrapment or injury due to any lesion of the nearby structures. Unilateral vocal cord paralysis can cause aspiration in about 40% of the patients. Therefore, early diagnosis and management of the cause of hoarseness should be pursued.<sup>8</sup>

TB sequelae include many conditions such as obstructive airway disease, amyloidosis, bronchiectasis, pulmonary hypertension, and heart failure. Interestingly, it is not yet recognised as a cause of pulmonary hypertension in the Western world. In patients with a history of TB, pulmonary hypertension can be caused by parenchymal damage, vasculitis, or any damage affecting the pulmonary vasculature.<sup>6</sup> There is a significant burden of patients with TB-induced destroyed lungs, presenting as obstructive and restrictive lung diseases, and efforts should be made to control their symptoms and prevent disease progression, thereby reducing socio-economic burden.<sup>9</sup> However, the burden of pulmonary vascular disease is still not well-defined in the literature, but it is not uncommon in TB-affected lungs. More data are needed regarding the pathophysiology, therapeutic options, and prognoses of patients with pulmonary hypertension due to the TB-destroyed lungs.<sup>10</sup> PAH can lead to the dilatation of pulmonary arteries, resulting

in the compression of the recurrent laryngeal nerve and leading to Ortner's syndrome.<sup>3</sup>

The scarcity of data on post-TB lung fibrosis and pulmonary hypertension, leading to hoarseness due to the compression of the recurrent laryngeal nerve in a TB-endemic area, prompted us to present this case.

In a TB-endemic region, clinicians must consider pulmonary hypertension as a complication of post-TB lung fibrosis and its consequences, including the destruction of bronchoalveolar structures. This can affect the laryngeal nerve, causing hoarseness of voice, a very rare but possible presentation.

## PATIENT'S CONSENT:

Written informed consent was obtained from the patient.

## COMPETING INTEREST:

The authors declared no conflict of interest.

## AUTHORS' CONTRIBUTION:

DF, RK, JL: All the authors contributed to the design, drafting, and critical revision of the manuscript.

All authors approved the final version of the manuscript to be published.

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