Case Report

A 28-year-old male was admitted to the Government Medical College, Thiruvananthapuram, India in 2013 with hoarseness persisting for three months. He also complained of a history of dyspnoea both upon moderate exertion during the last year and at rest over the past two days. He did not report any symptoms of coughing, haemoptysis, difficulty in swallowing, paroxysmal nocturnal dyspnoea, rheumatic fever or previous surgical interventions or trauma to the head and neck region. Two years previously, he had been diagnosed with open pulmonary TB and was prescribed a regimen of antitubercular therapy including isoniazid, rifampicin, pyrazinamide and isoniazid, rifampicin, pyrazinamide and thiacetazone. Although he had undergone irregular treatment for pulmonary tuberculosis (TB) two years previously, he was diagnosed with left recurrent laryngeal palsy secondary to cardiovocal syndrome. Although reports exist of recurrent laryngeal palsy in patients with pulmonary TB, this case appears to be the first to report cardiovocal syndrome in a patient treated for pulmonary TB.

Keywords: Hoarseness; Pulmonary Tuberculosis; Vocal Cord Paralysis; Cor Pulmonale; Pulmonary Hypertension; Cardiovocal Syndrome; India.
Cardiovocal Syndrome

A rare cause of hoarseness in a patient with a history of pulmonary tuberculosis

The mitral valve apparatus was normal and there was no sign of aortic dilatation. A CT pulmonary angiogram ruled out a pulmonary embolism; however, arterial blood gas analysis showed hypoxaemia with hypercapnia.

A diagnosis of pulmonary hypertension group III was made according to the classifications of the World Health Organization. As such, the patient was treated with oxygen administration, bronchodilators, a low-salt diet, diuretics (furosemide) and calcium channel blockers (diltiazem). While the symptoms of breathlessness gradually improved over the course of the following week, there was no improvement in the hoarseness. Supplemental oxygen therapy was considered; however, this was not pursued due to financial constraints. The patient was discharged after two weeks of admission and was unfortunately lost to further follow-up.

Discussion

Also known as Ortner’s syndrome, cardiovocal syndrome was first described in 1897 among three patients with severe mitral stenosis, wherein it was postulated that left atrial dilatation can cause left recurrent laryngeal nerve palsy due to the compression of the nerve against the aorta. During autopsies, Fetterolf et al. found that the normal distance between the aorta and pulmonary artery in the aortic window was 4 mm; as such, compression of the recurrent laryngeal nerve between these structures can cause palsy. Other causes of hoarseness in cardiac disorders include lymphadenitis in the aortic window, pressure from the left bronchus, right ventricular hypertrophy, pulmonary artery atherosclerosis, the anatomical position of the ligamentum arteriosum, a dilated pulmonary artery, mitral regurgitation, atrial myxoma or a left ventricular aneurysm.
The left vagus nerve gives rise to the left recurrent laryngeal nerve at the level of the aortic arch. The recurrent laryngeal nerve then curves around the aorta on the outer side of the ligamentum arteriosum and ascends in the groove between the trachea and the oesophagus. This thoracic course makes the recurrent laryngeal nerve vulnerable to injury and it has been suggested that compression of the nerve between the dilated pulmonary artery and the aorta occurs among almost all patients with cardiovascular disease. In contrast, it is not unusual to find patients in India who have received pulmonary TB treatment for pulmonary TB to present with features of right heart failure; according to a recent study, the prevalence of cor pulmonale among Indian patients with pulmonary TB was 11%. Pulmonary hypertension in TB can be caused by the destruction of the vascular bed due to parenchymal abnormalities, endarteritis or vasculitis leading to the reduced cross-sectional area of the pulmonary vasculature. It has been suggested that recurrent secondary respiratory tract infections also play an important role in the pathogenesis of pulmonary hypertension in previously treated pulmonary TB patients. Commonly, such patients present with dyspnoea to a degree disproportionate to that indicated radiologically or with desaturation present even upon mild exertion. In rare instances, the presentation can also include overt heart failure with pedal oedema, raised jugular venous pressure and tender hepatomegaly. This is consistent with that seen in the present case, whereby the patient presented with features of right heart failure.

Among former TB patients presenting with worsening dyspnoea, the management and diagnosis can be challenging as such cases can be misdiagnosed as a TB relapse. The correct diagnosis and timely management of pulmonary TB can prevent future complications, such as those observed in the patient described in the present case report. Although the patient had previously received antitubercular therapy, his initial noncompliance with the treatment may have led to further lung damage and resulted in the subsequent development of pulmonary hypertension two years later.

Conclusion

Clinicians should be aware of cardiovocal syndrome as a cause of left recurrent laryngeal nerve palsy, particularly among patients with a history of TB who may also be at risk of developing pulmonary hypertension. Although reports exist of recurrent laryngeal palsy in TB cases, cardiovocal syndrome has never before been reported among patients with pulmonary TB.

References