Case Report

Unusual cause of bilateral vocal cord paralysis

ABSTRACT

The most common cause of vocal cord paralysis (VCP) as a result of recurrent laryngeal nerve compression is malignant tumors. A benign and inflammatory causes of VCP is rarely reported in the literature, and in almost all reported cases it was a unilateral paralysis. We report a rare case of tuberculous mediastinal lymphadenopathy causing bilateral VCP in a young female patient.

Key words: Hoarseness of voice; tuberculous mediastinal lymphadenopathy; vocal cord paralysis

Introduction

The most common cause of vocal cord paralysis (VCP) as a result of recurrent laryngeal nerve (RLN) compression is malignant tumors, and of these tumors, bronchogenic carcinoma is the most frequent cause.[1] A benign and inflammatory cause of VCP is rarely reported in the literature, and in almost all reported cases were unilateral paralysis.[2-4] Here, we report a rare case of tuberculous mediastinal lymphadenopathy causing bilateral VCP and presenting with hoarseness of voice and stridor, which was treated by anti-tuberculous medications with subsequent full recovery.

Case Report

A 23-year-old female, presented with 6 months history of chest pain, dry cough, hoarseness of voice, and recently she was complaining of stridor. Chest pain was retrosternal, severe and continuous, radiated to the back, and associated with shortness of breath on mild exertion.

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Cough was dry, intermittent; progressive with time, and it was associated with hoarseness of voice and recently stridor, and dysphagia mainly for fluids. There was no history of fever, hemoptysis, night sweating, or weight loss. No history of smoking, recent travel, or contact with tuberculosis (TB) patient. Physical examination was unremarkable. All blood investigations were normal, sputum for Gram-stain, and culture showed no growth of organisms. Chest X-ray showed left mediastinal mass [Figure 1].

Enhanced chest computed tomography (CT) scan showed left upper mediastinal mass, cystic in nature, along the arch of the aorta, with thick, enhanced and irregular walls, measuring 5 cm in diameter [Figure 2]. In addition, there were multiple enlarged bilateral superior mediastinal lymph nodes. Fiber-optic bronchoscopy confirmed bilateral VCP; but the cords were not in the median position as the laryngeal entrance was opened, broncho-alveolar lavage for cytology was negative for malignant cells. CT-guided transthoracic tru-cut biopsy revealed changes characteristics of TB,

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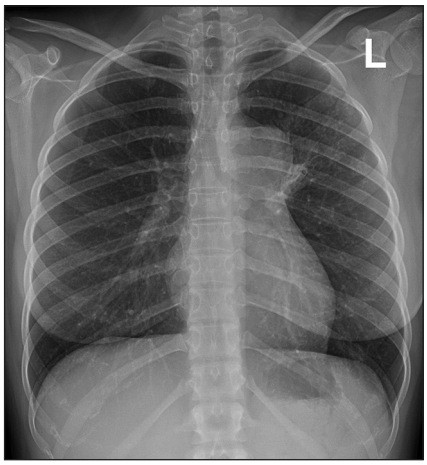

Figure 1: Chest X-ray showing left-side mediastinal mass

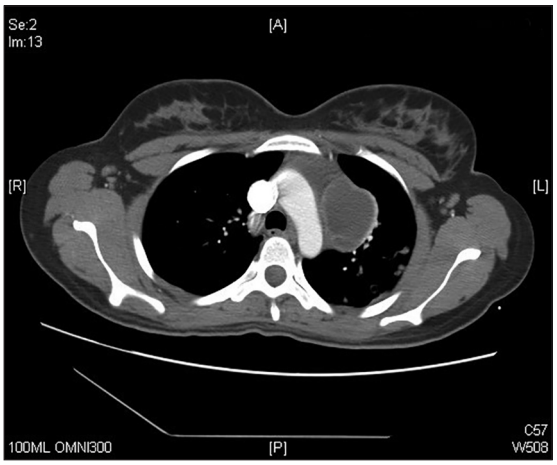

Figure 2: Enhanced chest computed tomography scan showing left upper mediastinal mass, cystic in nature, along the arch of aorta

includes granulomatous inflammation and caseous necrosis with chronic inflammatory cells, epithelioid cells infiltration, and multinucleated giant cells. A diagnosis of tuberculous mediastinal lymphadenopathy was made. The patient received anti-tuberculous medications. On 6 weeks follow-up, all symptoms were improved dramatically. The patient completed the course of anti-tuberculous medications successfully.

On outpatient clinic follow-up after 6 months, chest CT showed a marked reduction in the size of the mediastinal mass, and eventually full recovery clinically and radiologically was achieved.

Discussion

In our case report, bilateral VCP was due to both large left-sided mediastinal mass and the bilateral superior mediastinal lymphadenopathy which were affecting and compressing both RLNs, and this process of involvement was chronic not acute as explained by the pathology of TB, and the findings in the bronchoscopy as the vocal cords were not in the midline and the laryngeal entrance was opened, however, full clinical and radiological recovery was achieved with complete clearance of all symptoms following the early and full course of anti-tuberculous medications.

Hoarseness of voice as a result of RLN compression is frequently reported with primary or secondary malignant invasions, such as thyroid neoplasms, lung carcinoma, esophageal carcinoma, and malignant metastases to the mediastinum.[1] In these reports, unilateral left-sided VCP was more common, due to the long course of left RLN around the arch of the aorta, which usually gets compressed or invaded by malignancies located in the tracheobronchial or aorto-pulmonary window lymphadenopathy.[1,2]

While the right RLN arises at the level of the right subclavian artery, ascends to the neck and becomes in close contact with cervical pleura, so its involvement is usually associated with diseases involving the apex of the lung or right-sided mediastinal lymphadenopathy.[1-3]

There are several suggested mechanisms in the literature to explain VCP in TB patients. Some suggested that it could be caused by entrapment of the RLN by a scar but others suggested traction neuropathy and compression by TB lymphadenopathy as a cause, and some authors mentioned direct spread of infection to RLN as a cause of palsy,[3,4] but Rafay disagreed with that theory because of the complete recovery of RLN palsy after the administration of anti-tuberculous therapy.[2]

Moreover, Fowler and Hetzel stated that TB could lead to right VCP through chronic pulmonary TB predominantly affecting the right upper lobe of the lung.[3] However, VCP as a complication of primary tuberculous mediastinal lymphadenopathy is rare, bilateral VCP is extremely rare, and hoarseness of voice and stridor due to tuberculous lymphadenitis is a very unusual presentation.[2-4]

Bloomberg and Dow mentioned that pericardial effusion, broncho-esophageal fistula, bronchial erosion, superior vena cava obstruction and VCP are very uncommon complications of mediastinal TB.[5]

Although the prevalence of pulmonary TB has been noticed to fall recently in many developed countries, the notification of extrapulmonary TB has been increased. Among the different types of extrapulmonary TB, tuberculous lymphadenopathy is the most common form, and the first lymphoid tissue to be affected is often hilar and mediastinal lymph nodes.[6] However, the diagnosis of primary mediastinal tuberculous

lymphadenopathy is considered very difficult, because it often mimics mediastinal tumors and malignant lymphomas,[7] and also detection of acid-fast bacilli in microscopy and culture of materials other than sputum is relatively low.[4] Chest radiograph remains a useful screening study, but it is not sufficient for the diagnosis. CT scan chest is more accurate in detecting the extent and location of the responsible pathology than a chest radiograph.[1]

Recently, ultrasound-guided trans-bronchial needle aspiration has emerged as an important tool for diagnosis of mediastinal and hilar tuberculous lymph-adenopathy.[6] Nevertheless, in some circumstances, empirical anti-tuberculous therapy becomes necessary when the clinical and radiological features are highly suggestive of TB, even without microbiological evidence.[8] Early initiation of anti-tuberculous medication is the key in the successful management of tuberculous mediastinal lymphadenopathy.[9]

Conclusion

A benign and inflammatory causes of VCP is rarely reported in the literature, and in almost all reported cases, it was a unilateral paralysis. However, bilateral VCP caused by tuberculous mediastinal lymphadenopathy in otherwise healthy patient with full recovery is an extremely rare entity.

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Conflicts of interest

Nil.

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