Vertebral tuberculosis presenting as a posterior mediastinal mass

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Abstract
Vertebral tuberculosis (TB) or Pott’s disease has become uncommon since the advent of antituberculosis drugs and improved public health measures. However, it remains a cause of disease, especially in developing countries. It presents with nonspecific clinical features and atypical radiological findings which may cause delays in diagnosis and lead to mortality and serious morbidity including permanent neurological deficits. This report describes a case of vertebral TB that presented as a posterior mediastinal mass suggesting a tumorous lesion, leading to delay in diagnosis. TB may affect any organ in the body including bones and should always be kept in mind by physicians in the differential diagnosis of the posterior mediastinal lesions.

Keywords: Tuberculosis, vertebra, posterior mediastinal mass, COPD, malignancy

Introduction
The advent of antibiotics and adoption of directly observed therapy for the treatment of tuberculosis (TB) represented a major breakthrough in the fight against the disease. However, TB still remains as a leading cause of morbidity and mortality all over the world.

TB can infect any organ of the body, although it typically affects the lungs. In some cases of TB, the infection spreads outside of the lungs and causes “extrapulmonary TB” [1]. Vertebral TB accounts for 2% of all cases of TB and 15% of cases of extrapulmonary TB [2].

Although the characteristic clinical features of this rare type of TB were first described more than 200 years ago [3], this disease still presents with diagnostic difficulties and delays in clinical practice. Mimicking the radiological features of a vertebral or a lung tumor, nonspecific symptoms such as a backache on admission, and difficulties in the confirmation of diagnosis are the main causes of delay in diagnosis [4].

We describe a case of vertebral TB which presented with a posterior mediastinal mass suggesting a malignancy resulting in a delay in diagnosis.

Case presentation
A 69-year-old male presented to the emergency department of our hospital with shortness of breath, cough with sputum, one year history of a back pain, night sweats and weight loss of 10 kg. He was an illiterate and had been a farmer for approximately 45 years. He carried a diagnosis of Chronic Obstructive Pulmonary Disease (COPD) with an active smoking history of more than 80 pack-years. He was also suffering from type 2 diabetes mellitus since the age of 15 years, and from Alzheimer’s disease for the past 3 years. He had never consumed illicit drugs, had no allergies, and had no history of alcohol addiction.

On examination, blood pressure was 100/70 mmHg and electrocardiography showed sinus tachycardia with a rate of about 130 beats/minute. Examination of the chest revealed diminished breath sounds in the lower zone of the left lung. Examination of blood samples revealed leukocytosis (13800/mm^3), lymphocytosis (9600/mm^3), thrombocytosis (540000/mm^3) and anemia (haemoglobin: 9.5 g/dl). Results of blood chemistry were within normal limits. Erythrocyte sedimentation rate was 120 mm/h and serum level of C-reactive protein was 8.5 mg/dl. Tuberculin skin test was measured as 13 mm at the end of 72 hours and anti-HIV antibody tests were negative. Measurement of arterial blood gas analysis on room air revealed pH: 7.44, PaCO₂: 34 mmHg, PaO₂: 53 mmHg, HCO₃⁻: 24 mmol/L and SaO₂: 86%, compatible with hypoxemic respiratory failure. Chest radiograph findings included a paracardiac dense shadow blunting the left costophrenic angle (Figure 1a). Spine graph of the patient which he brought from another hospital included the area between lower thoracic and lumbar vertebrae, demonstrating osteophytes in the lumbar region (Figure 2).
He was hospitalized with a diagnosis of COPD exacerbation and respiratory failure. Most of his symptoms disappeared following the initiation of the bronchodilators, oxygen supply and nonspecific antibiotics (ceftriaxone sodium+clarithromycin). However, he had persistent night sweating and severe back pain refractory to conventional analgesics. He gave a history of multiple doctor visits for progressive backache. Analgesics and muscle relaxant drugs were prescribed to him for presumed myalgia. He underwent Computerized Tomography (CT) of the thorax which demonstrated radiological signs of centrilobular-type emphysema especially in the upper zones of the both lungs and a mass lesion located in the posterior mediastinum. This lesion had the dimensions of 4x7 cm and was invading the body of the 7th thoracic vertebra, prevertebral fascia and retroaortic area, suggesting a tumor (Figure 3).

Although CT-guided transthoracic needle biopsy was repeated twice to sample the lesion, histopathological studies revealed no evidence of a malignancy. No growth of a specific infectious agent could be demonstrated by microbiological studies, including M. tuberculosis, fungi or bacteria. The decision was made to continue administration of nonspecific antibiotics and observe the results of the treatment. However, one month later the radiological findings revealed enlargement of the mass lesion and the patient underwent Positron Emission Tomography-Computerized Tomography (PET-CT). PET-CT showed pathological 18-Fluorodeoxyglucose (18-FDG) uptake by the mass lesion (SUV$_{max}$=10.6) which was invading bodies of the 7th, 8th, 9th vertebrae, and paravertebral muscles. In addition, a subcarinal lymph node with a diameter of 1.2 cm (SUV$_{max}$=4.7) and pleural thickening adjacent to body of the 10th vertebra (SUV$_{max}$=9.9) demonstrated pathological 18-FDG uptake (Figure 4).
The patient was referred to the chest surgery department for further evaluation of the suspected malignancy. He underwent video-assisted thoracoscopic surgery and histologic examination of specimens taken at operation revealed necrotic tissue with a chronic inflammatory-cell infiltration and formations of caseating granulomas. Acid-fast bacilli were not seen on Erlich-Ziehl-Nielsen staining of the surgical material, but M. tuberculosis was grown after 6 weeks of incubation, confirming the diagnosis of vertebral TB.

He was treated with Isoniazid (300 mg/day), Rifampicin (600 mg/day), Pyrazinamide (1500 mg/day) and Ethambutol (1200 mg/day) for the first 2 months, followed by treatment with Isoniazid and Rifampicin, for a total of 9 months. On examination at 2 years since treatment he had no radiological sign of a TB or relapse (Figure 1b). He continues to cope with exacerbations of COPD.

Discussion
Vertebral TB is one of the oldest diseases of mankind and a British surgeon, Sir Percivall Pott, was the first physician who described vertebral TB in 1779 [3]. Hence, it is also called Pott’s disease.

Vertebral involvement is a result of hematogenous spread of M. tuberculosis into the vertebral bodies. The primary infection site is either a pulmonary or an extrapulmonary focus which may be active or dormant [5-6]. Predisposing factors for vertebral TB are similar to lung TB and include poverty, malnutrition, over crowding, alcoholism, drug abuse, immunosuppressive treatment, diabetes mellitus, chronic peritoneal dialysis, previous tuberculosis infection and HIV infection [4]. Predisposing factors in this case were probably combination of poverty, malnutrition and diabetes mellitus.

Infection usually begins in the anterior part of the vertebral body adjacent to the end plate and rarely affects the posterior vertebral elements including the pedicles. Then comes spread of infection to adjacent intervertebral disk and subsequent spread into the paraspinal tissues, with formation of a paravertebral abscess (Pott’s abscess or cold abscess) [7]. Lower thoracic and lumbar portion of the vertebral column is the most common site for vertebral TB and followed by middle thoracic and cervical vertebrae [8]. In this case, beginning site of the infection was also anterolateral part of the vertebral body and mainly lower thoracic vertebrae were involved. However, there was no formation of a Pott’s abscess.

Although plain radiographs are usually the initial investigation method for the diagnosis of vertebral TB, CT scanning provides much better detail of sclerosis, irregular lytic lesions, disc collapse and disruption of the bone circumference than plain radiograph. The presence of calcification within the abscess is virtually diagnostic of vertebral TB [2]. In addition, fat-saturated T1-weighted Magnetic Resonance Imaging (MRI) is more valuable to reveal presence of paravertebral abscesses, involvement of contiguous vertebrae and intervening intervertebral discs [9]. MRI has been suggested as a reliable imaging modality for diagnosing vertebral TB [10]. MRI not performed because thoracic CT images of the patient demonstrated disruption of the vertebral bodies suggestive of a tumor lesion.

Diagnosis of vertebral TB is generally made on the combination of clinical features, radiological findings, microbiological research and histopathological study. If possible, bone tissue or paravertebral abscess samples should be obtained to stain for acid-fast bacilli and isolate organisms for culture. Besides, histopathological study should be performed on the samples to demonstrate caseating granuloma formations. The methods widely used for this aim are CT or ultrasonography guided transthoracic needle aspiration biopsy and if needed surgical biopsy [11-12]. Enzyme-linked immunosorbent assay and polymerase chain reaction for detecting M. tuberculosis are other diagnostic helpers of vertebral TB [13]. In this case, diagnosis was obtained with the help of both microbiological and histopathological studies.

Diagnostic difficulties and delays arise in some cases because of similar presentation of the tumours and other infectious diseases [14]. The most common cause of delay in the diagnosis of this disease is failure to consider the diagnosis and long list of diseases in differential diagnosis of vertebral TB. Common differential diagnosis list of vertebral TB includes especially brucellar spondylitis, pyogenic spondylitis, multiple myeloma, lymphoma, metastatic tumours of the vertebrae, and primary tumours of the vertebral column.

Multidrug therapy remains the cornerstone of all types of TB disease, as well as vertebral TB [15]. Patients with early disease, without neurologic deficits, and little or no kyphosis can be treated with a multidrug combination alone. Continuation of the treatment for 9 months is recommended [15]. However, surgical intervention is necessary in advanced cases with extensive bony destruction, abscess formation, or neurological compromise [16].

This case highlights the importance of being aware that TB may affect any organ in the body including bones. Also physicians should keep in mind that vertebral TB may present with atypical radiological features and mimic a malignancy, in order to avoid delayed diagnosis and treatment.

Competing interests
The authors declare that they have no competing interests.

Authors’ contributions

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