Tuberculosis of the spine (Pott's disease) presenting as hemiparesis

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Abstract

A middle-aged man with spinal tuberculosis (TB) (Pott's disease) presented with severe back pain and right hemiparesis which was initially diagnosed as stroke. Physicians should include Pott's disease in the differential diagnosis when patients present with back pain, hemiparesis and evidence of vertebral collapse.

A review of the literature on the pathogenesis, pathophysiology, clinical presentation, diagnostic methods, treatment, and prognosis of spinal TB was carried out. After an initial delay a correct diagnosis of spinal TB was made. Microbiology diagnosis confirmed *Mycobacterium tuberculosis* and appropriate medical treatment was instituted.

Spinal TB is common in developing countries but initial presentation with hemiparesis is uncommon. Magnetic resonance imaging (MRI) is an excellent procedure for the diagnosis of spinal TB; however microbiological diagnosis is essential. Mycobacterium may be cultured from other sites, otherwise, biopsy of the spine lesion should be done for pathologic diagnosis followed by culture, and staining for *M tuberculosis*. Clinicians should consider Pott's disease in the differential diagnosis of patients with back pain, hemiparesis, and destructive vertebral lesions. Proper diagnosis and anti-TB treatment with or without surgery will result in cure.

Introduction

Tuberculosis (TB) is a common disease in Nigeria and other developing countries of the world. Extrapulmonary TB may involve any organ systems, and signs and symptoms are non specific. The presenting symptom of spinal TB (Pott's disease) is usually spinal pain. When a patient presents with hemiparesis it may be mistaken for a stroke. This report describes a 53-year-old man who had Pott's disease initially diagnosed as stroke.

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Case report

Mr U A, a 53-year-old cargo inspector presented to a general practitioner (GP) with a history of severe waist pain radiating to both thighs, and weakness of right upper and lower limbs of 1-month's duration. The GP made a diagnosis of stroke and referred Mr U A to a tertiary institution where he had received several sessions of physiotherapy without improvement. During the night about a month later, after a strenuous day, he noticed increasing waist pain which he thought was due to the previous day's activity. The following morning he was unable to stand or support his weight on his hands. A couple of hours later he developed urinary incontinence and was unable to open his bowel. He was initially rushed to a prayer house where he was given some native medications. Hot water bottles were used to stimulate his legs but this caused multiple injuries to both legs. He was moved to our hospital because he had developed a fever and was getting weaker.

At presentation to the hospital the patient had a persistent fever and also gave a history of occasional sputum expectoration but had no cough. There was associated history of weight loss and night sweats. He had no contributory medical history.

Mr U A was married with five children. He had never smoked but drank alcohol occasionally. On examination the patient was conscious but lethargic; he was febrile (temperature 38.2°C) and had no lymphadenopathy. Neurological examination revealed supple but moderate tenderness in the neck. Strength was two over five in the right shoulder and elbow in flexion and extension and the same in the right hip and knee joints. The left upper limb was three over five in flexion and extension in the shoulder and elbow and two over five in the left hip and knee. He had no clear sensory level but had isolated areas of loss of patchy sensations and no gibbus. Systemic examinations of the chest, cardiovascular signs, and abdomen were unremarkable. He had shallow ulcers on both legs measuring 3–4 cm across.

Initial diagnosis of cord compression syndrome was made with tuberculosis of spine and cervical spondylosis as differentials. In addition, the patient had sepsis and was started on antibiotics and fluid resuscitation.

A full blood count was done which showed haemoglobin of 11 g/dl, white blood cells (WBC) 13 000, erythorocyte sedimentation rate (ESR) 88 mm in the first hour, WG. A Mantoux test was significant at 12 mm; serum

electrolyte, urea, and creatinine were normal. Fasting blood glucose, lipid profile, and urinalysis were normal, and urine culture yielded no organisms. Prostate-specific antigen (PSA) and serum acid phophatase were normal. Sputum stain for acid fast bacilli yielded *M tuberculosis*. Radiologic findings revealed a normal chest X-ray; cervical X-ray showed degenerative changes with marginal osteophytes at C3, C4 and C5, C6.

A lumbo-sacral/pelvic X-ray showed marginal osteophytes and subchondrial sclerosis. A magnetic resonance imaging (MRI) test revealed hypointense lesions on T2 weighted images at C3/C4 and C5/C6 with subtle reduction in the disc height of the aforementioned disc spaces. Our conclusions were: multiple cervical cord compression/myelopathy and secondary disc prolapsed; multiple disc degeneration; and cervical spondylosis (see Figure 1).

The patient was placed on anti-TB drug combination therapy of INH (isonicotinylhydrazine), rifampicin, ethambutol, and pyrazinamide for 2 months; followed by INH and rifampicin for a further 10 months. By the second week of the continuation phase of treatment, the strength in the right upper limb was grossly 4, right lower limb 3+, left upper limb 4+, and left lower limb 3+. The sphincteric functions had fully returned and he was discharged. By the 6th month of treatment he was standing with support.

Discussion

Tuberculosis of the spine (Pott's disease, TB spondylitis) is one of the oldest neurological diseases for which clear evidence is found. In 1779, Sir Percival Pott described spinal TB and surgical treatment of paravertebral abscess;[1,2] hence TB of the spine was named after him. Typical features have been described in Egyptian mummies as far back as 4000BC. DNA typing from a vertebral lesion in a 12-year-old dating back to AD1000 identified *M tuberculosis*.[2,3]

Spinal TB is common in many developing countries where pulmonary TB is prelevant owing to poor nutrition and environmental sanitation. It is a common cause of morbidity in children and adults in Nigeria and other developing nations,[4] but more common in elderly people in the developed world.[4] Spinal TB accounts for about 50% of all skeletal TB, about 15% of all cases of extrapulmonary TB, and about 1% to 2% of all cases of TB.[5,6] While the HIV pandemic has led to the resurgence of TB, it has had little impact on the epidemiology of spinal TB. In a large French study, none of the 82 cases of spinal TB was HIV positive.[7,8] In other large longitudinal studies among HIV-infected patients few were reported with spinal TB even after long-term follow-up.[9,10]

(a) (b)

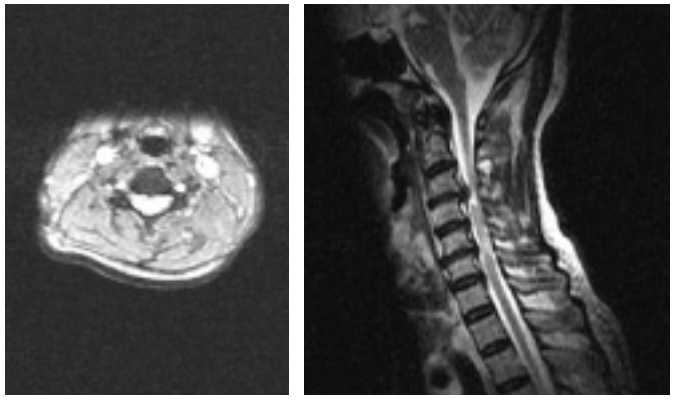

Figure 1 (a) side view and (b) cross-sectional view from MRI (T2 weighted) showing spinal cord compression at the C3/C4 and C5/C6 levels.

Most reported cases of spinal TB are in the lower thoracic and thoraco-lumbar areas, while spinal TB in a cervical region is less common.[11–13] The normal route of entry of *M tuberculosis* is the respiratory tract with haematological spread. Secondary haematological seeding can occur from a silent focus elsewhere in the body, e.g. genito-urinary tract, gut, or tonsils. Another mode of spread to the vertebral bodies is through the lymphatic system, usually from involved contiguous para-aortic lymph nodes.

Damage by the tubercle bacilli starts in the cancellous bone and extends to the cortex. The inflammation slowly spreads to the vertebra via the disc space. When the disease has advanced there is progressive vertebral collapse resulting in kyphosis and gibbus formation. Onset of symptoms is insidious in spinal TB and disease progression is slow. Most cases present with pain overlying the affected vertebrae. The patient may present with low-grade fever, chills, weight loss, and non-specific symptoms. Paraplegia or paraparesis can be the first sign of spinal disease, with varying degrees of weakness, nerve root compression, and sensory involvement. Duration of symptoms ranges from several weeks to several months and sometimes years. Neurologic deficits may occur early in the course of Pott's disease. Signs of such deficits depend on the level of spinal cord or nerve root compression. Pott's disease that involves the upper cervical spine can cause rapidly progressive symptoms as in our patient. Retropharyngeal abscesses occur in almost all cases. Neurologic manifestations also occur early and range from single nerve palsy to hemiparesis or quadriparesis as in our patient.

Diagnosis of spinal TB in a developing country like ours may be difficult, especially when there is an atypical presentation such as a single nerve palsy, hemiparesis, or quadriparesis as seen in upper cervical compression. These are also features of cerebrovascular disease. It may present in a similar fashion to malignant deposits in the spine from cancer of the prostate, cervix, or colon. The patient usually presents with pain and systemic symptoms like weight loss and elevated ESR. This presentation is similar to cases of spinal metastasis.[7] Other differential diagnoses of spinal TB include *Staphylococcus aureus* osteomylitis, brucellosis, actinomycosis, candida, histoplasmosis, blastomycosis, multiple myeloma, and

eosinophilic granuloma.

A positive PPD (purified protein derivative) skin test has been reported in 62–100% of TB spine cases. Our patient had a Mantoux test of 12 mm.[14,15] A positive tuberculin test supports the diagnosis but a negative test should not be considered as evidence for excluding TB.

Radiologic investigation is very important in spinal TB. A confident radiological diagnosis is made in less than 50% of cases, with the principal alternative being malignant disease.[7] Most studies on different imaging procedures such as plain radiography, CT, and MRI fail to describe how much information is provided by each test. Conventional radiographs give a good overview; CT visualises the disco-vertebral lesions and para vertebral abscesses, while MRI is useful in determining the spread of the disease to the soft tissues and spinal canal.[7,16] The plain radiograph describes changes consistent with TB spine in 91–99% of cases.[14,17] Radiographs may reveal advanced lesions with vertebral osteolysis and disc space narrowing; these findings are similar in patients with pyogenic TB. A CT scan is very important in the diagnosis of spinal TB as it shows abnormalities earlier than plain radiography. The pattern of bone destruction may be fragmentary in 47% of cases, osteolytic in 34%, localised and sclerotic in 10%, and subperiostal in 30%.[7,18] Other findings suggestive of spinal TB in CT include soft tissue involvement and paraspinal tissue abscess.

MRI is the best diagnostic modality of spinal TB.[7,16,19] It is more sensitive than radiography and more specific than CT. MRI can also provide the diagnosis 4–6 months earlier than conventional methods, offering the benefits of earlier case detection and treatment.[9] Because radiological films are commonly non diagnostic and imaging studies are not fully reliable for differentiating spinal TB from other infection or neoplasm, bacteriologic and/or histologic confirmation must be obtained. Few patients with spinal TB have concomitant renal or pulmonary disease, therefore microbiological diagnosis should be done with pus aspirate or tissue sampling. However, a collection of spinal or paraspinal specimens is not absolutely necessary if pulmonary or lymph node TB is present or if extraskeletal sites can be sampled.[7] Fine-needle aspiration biopsy as a diagnostic tool is accurate, safe, and cost effective because the procedure does not require hospitalisation.[7] CT-guided fine aspiration gives a better yield than blind aspiration.

The treatment of spinal TB has undergone some revolutionary changes in the last 60 years. Modern treatment will cure up to 90% of cases. Medical treatment may be combined with surgery.[7,20] Skeletal TB is treated for a duration of 12 months. Combination therapy uses the four drugs INH, rifampicin, pyrazinamide, and ethambutol for 2 months; followed by INH and rifampicin for 10 months. Directly observed therapy (DOT) is recommended as this enhances patient compliance. Surgery was once the mainstay of treatment of spinal TB but is now required less frequently, even in patients with cord compression. Chemotherapy alone has been reported as a successful and effective treatment modality.

Early diagnosis yields excellent results in the treatment of spinal TB. Increasing back pain requires investigation with plain radiograph and possibly CT or MRI where the facility exists. Our patient presented with back pain and later hemiparesis. He initially presented to a GP who diagnosed stroke and referred him to a larger centre. A good history and reassessment would have suggested the diagnosis especially when the risk factors for cerebrovascular disease were remote. His treatment began with physiotherapy, which may have worsened the problem. Timely intervention in treatment of spinal TB can avoid extensive investigations, treatment delays, and adverse long-term outcomes, including compression fractures with neurological deficits as seen in our patient. Patients with spinal TB experience severe pain, therefore measures to alleviate pain should include appropriate spinal bracing and a combination of analgesics, including narcotic analgesics.

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