CLINICAL IMAGES

Spinal tuberculosis: Diagnostic biopsy is mandatory

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'Assumption makes an ass out of you and me' was unfortunately the case in a presumptive diagnosis of tuberculous spinal infection. With the escalating incidence of HIV and tuberculosis (TB), there is an increasing incidence of spinal disease. Frequently, patients with pre-existing pulmonary TB develop back pain. In the South African environment of reduced access to tertiary care, decisions are often made on the basis of probability and not confirmed diagnostically. This can be dangerous, as highlighted by the following cases where patients were incorrectly assessed and treated for spinal tuberculosis, to their detriment.

Case 1

A 35-year-old man presented with a 2-month history of ascending sensory loss and paraparesis of both legs. A thoracic gibbus and collapse of the third thoracic vertebral body was noted. A presumptive diagnosis of spinal TB was made and Rifafour anti-TB medication initiated. The patient continued to deteriorate neurologically over the next 2 weeks to a paraplegic state with reduced but sensory preservation. He was then referred to a tertiary spinal surgery unit.

His X-rays were re-assessed as a T3 vertebro plana. Magnetic resonance imaging (MRI) revealed a complete T3 vertebral body collapse with pre- and post-vertebral soft-tissue masses. A narrowed spinal canal with cord compression and abnormal cord signal changes was found (Fig. 1). He had mild leucocytosis and an erythrocyte sedimentation rate (ESR) of 64 mm/h.

A provisional diagnosis of spinal TB was made and, with the history of progressive neurological deficit, the patient was taken to theatre for an anterior transthoracic decompression and fusion. Surprisingly, despite the MRI evidence of epidural mass, no free pus was found. A small amount of yellow-gray granulomatous tissue was curetted out. Microbiology revealed no organisms, but histological investigation suggested an eosinophilic granuloma with dense eosinophilic infiltrates, histiocytes and scanty multinucleated giant cells. S-100, CD 68 and CD-1a stains were positive and Birbeck granules were seen on electron microscopy (EM), confirming the diagnosis.

Following surgical decompression, there was slow neurological recovery and at 6 months the patient was walking with assistance.

Case 2

A 32-year-old woman, with a proven history of disseminated TB and HIV, on anti-TB treatment for 18 months and antiretroviral agents for a year, presented with increasing lumbar pain over a period of 6 months. This was assessed as spinal TB and not investigated further as she was already on TB medication. She had been placed on an alternative TB regimen on a presumptive diagnosis of multidrug-resistant TB (MDR-TB).

After 6 months of back pain, she developed a 1-week history of right leg pain and decreased sensation around the right foot and ankle. She was referred to the tertiary spinal unit where she was found to be pyrexial, confused and tachypnoeic. There was a tender right paraspinal soft-tissue mass without any noticeable spinal deformity, decreased sensation and loss of power in both lower limbs ranging from 1 to 4/5 (MRC grading), with reflexes and anal tone well maintained. She had leucocytosis, a C-reactive protein (CRP) level of 246 mg/l and an erythrocyte sedimentation rate (ESR) of 150 mm/h. Radiographs demonstrated destruction of the L4 vertebral body with maintenance of the adjacent disc spaces and sagittal alignment (Fig. 2). MRI confirmed destruction of the L4 body with large soft-tissue masses present in the psoas muscles and posterior muscles of the back (Fig. 3).

Broad-spectrum antibiotics were initiated for what was believed to be pneumonia and, after a positive response, she was taken to theatre for drainage of the pus with biopsy of the lesion and fixation from L3 to L5. Culture confirmed *Salmonella* group B (non-typhoid) infection and an anaplastic (T-cell) large-cell lymphoma on histology. She died during her hospital admission.

Case 3

A 68-year-old woman, with a history of 2 episodes of successfully treated pulmonary TB, presented with mid-thoracic back pain, progressive leg weakness, loss of perineal sensation, and urinary incontinence for 2 weeks. There was an associated productive cough and significant weight loss for the preceding 2 months. She was started on TB medication but

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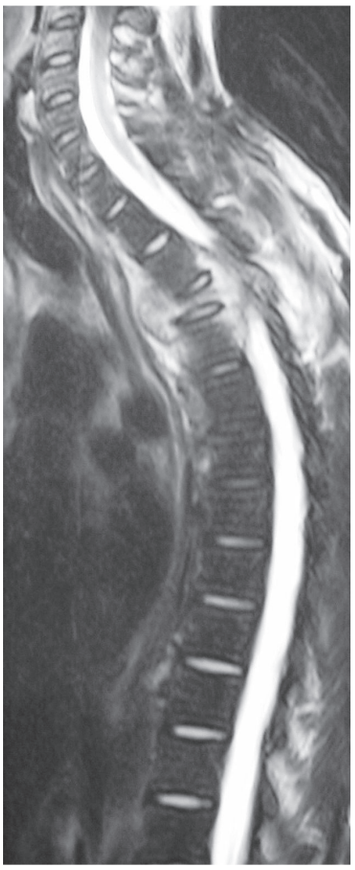

Fig. 1. MRI: Single-body involvement with epidural and paraspinal soft-tissue involvement.

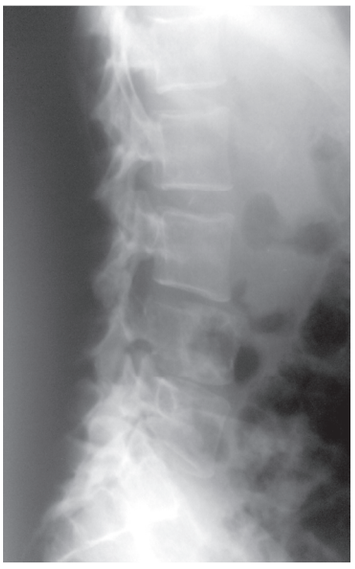

Fig. 2. X-ray: Loss of L4 cortical definition with reduced disc space.

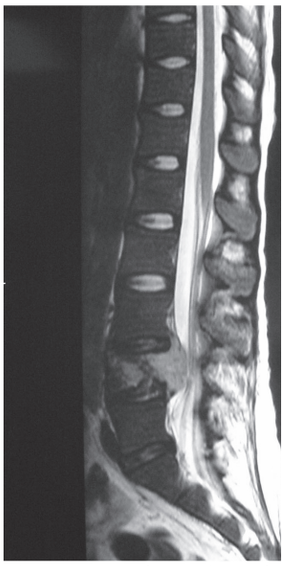

Fig. 3. MRI: L4 destruction and epidural soft-tissue extension.

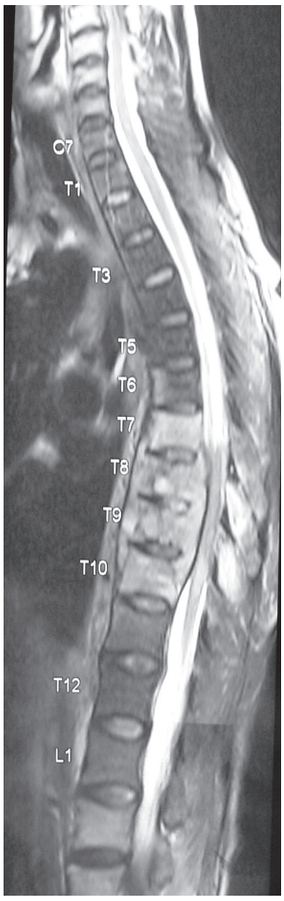

Fig. 4. MRI: Multilevel vertebral body infiltration indicative of metastatic tumour.

neurological deterioration continued and she was referred to the tertiary spinal unit after 2 weeks of medication.

She appeared chronically ill and wasted with anaemia and clubbing. There was mid-thoracic tenderness with no spinal deformity. Neurological examination revealed a sensory level at T10 with no sacral sparing, complete loss of motor power in her left leg with 2 - 4/5 power in her right leg, and loss of anal tone. Her ESR was 117 mm/h and CRP 105 mg/l. Radiographs demonstrated destructive bony lesions in T8 - T10 with maintenance of the disc spaces and alignment. MRI confirmed multiple levels of vertebral body involvement from T7 to T10 with soft-tissue masses extending under the anterior and posterior longitudinal ligaments (Fig. 4).

On anti-TB treatment and strict bed rest, there was mild improvement of her neurological function in the first week after admission. Concern then arose about the possibility of MDR-TB, and an alternative regimen was started. However, she worsened to complete paralysis with the development of renal failure and sepsis. Surgery for a decompression and fusion revealed metastatic adenocarcinoma. She died in hospital.

Discussion

Bone and joint TB continues to be a major problem in most developing countries, with evidence of resurgence in the developed world. Several Medical Research Council (MRC) trials established that drug therapy is the mainstay of treatment for TB of the spine, and that surgery is only indicated when there is gross destruction, deformity or associated neurological deficit not responding to chemotherapy. A cause of such destruction and deformity is delay in diagnosis. More than 50% of the vertebra has to be destroyed before a lesion can be seen on a plain radiograph, a process which takes 6 months.[1,2]

MRI is the modality of choice for imaging in suspected spinal TB because it demonstrates bony and soft-tissue involvement. TB spondylitis typically manifests with juxta-discal body involvement and collapse with paraspinal and epidural pus. The intervertebral disc may have normal height and normal signal on MRI, reflecting the resistance of the disc to TB infection. Gadolinium-contrasted scans may

help distinguish abscesses from granulation tissue, with the abscess showing only rim enhancement, whereas the whole mass enhances with granulation tissue.

In endemic areas, there may be atypical cases of up to 30% with a variety of other MRI manifestations. This leads to most lesions with epidural collections being considered as tuberculotic if the patient has a raised ESR and is clinically not septicaemic. Reliance on new imaging techniques alone is insufficient.

Our cases highlight the need for laboratory confirmation of TB before accepting it as the diagnosis. Many patients have pulmonary TB, and concomitant spinal complaints cannot simply be presumed to be related, especially in a country with a TB incidence approaching 1%.

TB is the great mimicker. Biopsy is mandatory, and all efforts should be made to confirm the diagnosis despite constraints in the state health sector. This is even more the case if features are atypical on X-ray or unresponsive to medical intervention.

1. Desai SS. Early diagnosis of spinal TB by MRI. *J Bone Joint Surg Br* 1994; 76-B: 863-869.
2. Lifeso RM, Weaver P, Harder EH. Tuberculous spondylitis in adults. *J Bone Joint Surg Am* 1985; 67-A: 1405-1413.