

# 11

## A 45-Year-Old Male Security Guard from Malawi With Difficulties in Walking and Back Pain

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### Clinical Presentation

#### History

A 45-year-old security guard from Malawi is admitted to a local tertiary hospital because of back pain and progressive difficulty in walking.

His troubles started 1 year earlier with back pain and he presented to a local health centre. He was given paracetamol and sent home. The pain did not improve. Over the following weeks he also developed difficulty in walking and 'pins and needles' sensation in his legs.

Three months after the first visit he presented again to the same health centre. His temperature was slightly elevated (37.5°C, 99.5°F). He was given antimalarials, a single dose of praziquantel and paracetamol. He consulted a local traditional healer who applied tattoos to his chest and his back (Fig. 11.1A). Over the following 6 months his condition further deteriorated and he finally became bedridden.

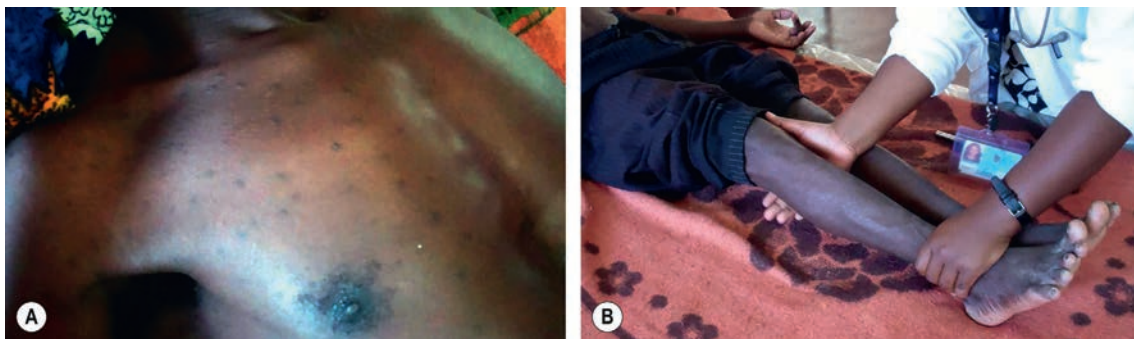
The patient denies fever, night sweats and weight loss, and there is no chronic cough. There is neither haematuria nor

diarrhoea and he is continent for stool and urine. There is no history of trauma or past tuberculosis (TB). He has never been tested for HIV.

He is a non-smoker, but drinks two paper cartons (about one litre) of Chibuku, a locally brewed beer, per day. He is married with three children, who are all well. He resides in an urban area and used to work as a security guard but has been unemployed for the last 6 months because of his illness.

#### Clinical Findings

He looks well and is afebrile with normal vital signs. There is tenderness over the lower thoracic spine and severe spasticity of both legs (Fig. 11.1B). The power in his legs is 1/5 (visible muscle flicker). Deep tendon reflexes of the lower limbs are exaggerated. The plantar reflexes are upgoing. There is a sensory level for pain and temperature sensation between T9 and T11, with diminished joint sense in his big toes bilaterally. The examination of his cranial nerves and the upper limbs is normal.



• **Fig. 11.1** Physical examination of a Malawian patient with back pain and difficulties walking. (A) Tattoos from a traditional healer on the chest of the patient. (B) Severe spasticity and contractures of both lower limbs.

## Laboratory Results

His Full blood count results are normal.

## Questions

1. What is the neuroanatomical syndrome and what is your differential diagnosis?
2. What further management should be carried out?

## Discussion

A 45-year-old Malawian man presents with chronic back pain and slowly progressive spastic paraparesis. On examination there is tenderness over the lower thoracic spine and a thoracic sensory level. He denies any constitutional symptoms. His past medical history is unremarkable. His HIV status is unknown.

### Answer to Question 1

#### *What is the Neuroanatomical Syndrome and What is Your Differential Diagnosis?*

The clinical signs – spastic paraparesis with hyperreflexia, upgoing plantar reflexes and thoracic sensory level – localize the lesion to the spinal cord. The bladder is usually involved in spinal cord disease, but the absence of bladder symptoms does not rule out spinal cord involvement, particularly in slowly progressive lesions, as in our case.

Spinal cord disease can be traumatic or non-traumatic. Nothing in the patient's history suggests trauma. Non-traumatic spinal cord disease can be compressive or non-compressive. Compressive disease is sometimes amenable to spinal surgery.

Common causes of adult non-traumatic compressive spinal cord disease in sub-Saharan Africa are spinal TB ('Pott's disease'), spinal metastases and degenerative spinal disease including slipped disc. Common causes of non-compressive spinal cord disease are schistosomiasis, autoimmune transverse myelitis and HIV-associated vacuolar myelopathy.

### Answer to Question 2

#### *What is the Further Management?*

The diagnosis of spinal cord disease in resource-limited settings is often clinical (Table 11.1). Management should focus on diagnosis and treatment of the underlying aetiology and on prevention and treatment of the complications of spinal cord disease. Diagnostic clues and possible treatment regimens are summarized in Table 11.2.

All patients should be tested for HIV, ova of *Schistosoma* spp. in urine and stool and evidence of TB or neoplasia on chest- and spinal radiography. If available, ultrasound examination of the abdomen is very valuable in tumour-screening and TB work-up. CSF should be examined, which is of particular importance in immunosuppressed patients.

Realistically, most patients admitted to hospital with paraplegia because of spinal cord disease will leave the

**TABLE 11.1**

### Important Causes of Spinal Cord Disease in the Tropics and Their Typical Clinical Features

	Typical Onset and Course	Clinical Features
Spinal tuberculosis	Insidious onset, chronically progressive over weeks, with months of back pain	Spasticity common, bladder may be spared, spinal deformity on examination
Spinal metastases	Subacute onset, chronically progressive over weeks	Spasticity or flaccidity, bladder may be spared
Transverse myelitis (incl. autoimmune)	Acute onset, often non-progressive	Bladder involvement common
Schistosomiasis	Acute (days) or subacute (a couple of weeks)	Often flaccid paresis, bladder involvement common

**TABLE 11.2**

### Diagnostic Clues and Possible Treatment Regimens for Important Causes of Spinal Cord Disease in Resource-Limited Settings

	Diagnostic Clues	Treatment
Spinal tuberculosis	Typical spinal radiograph (see Box) Epidemiological evidence	Antituberculous treatment Spinal surgery if available and applicable (see Box)
Spinal metastases	Clinical evidence of the primary tumour (e.g. prostate, breast)	Very limited options: radiotherapy rarely available; corticosteroids to decrease oedema
Transverse myelitis (incl. autoimmune)	Young adults Inflammatory CSF	Corticosteroids
Schistosomiasis	Exposure to freshwater in endemic regions, young adults in endemic countries or non-immune travellers; CSF eosinophilia; other manifestations of schistosomiasis may or may not be present.	Praziquantel, corticosteroids

**TABLE 11.3****Common Complications of Spinal Cord Disease and Their Prevention and Management**

Complication	Prevention/Management
Pressure sores	Nursing, training and counselling of guardians (two hourly turning)
Urinary retention	Catheterization
Contractures	Physiotherapy, training of guardians for home-based physiotherapy
Pain	Pain relief by NSAID/opiates, involvement of local palliative care team
Immobilization	If available, prescription of walking aids/wheelchairs
Depression	Spiritual and mental support, occupational therapy/community projects, pharmacotherapy, involvement of local palliative care team

hospital paraplegic. The prognosis is overall poor, and often the secondary complications rather than the primary pathology dictate the further course of the disease. The prevention of complications of spinal cord disease is therefore of paramount importance. Health workers should work hand in hand with guardians, physiotherapists and the local palliative care team (Table 11.3).

### The Case Continued...

The patient was found to be HIV-positive. His urine dipstick was normal and there were no ova of *Schistosoma* spp. detected in his urine and stool. The spinal radiograph showed a collapse of T11 vertebral body as well as soft tissue swelling around the spine (Fig. 11.2). Chest radiography and abdominal ultrasound examination were normal.

A presumed diagnosis of spinal TB was made and the patient was started on standard first-line antituberculous treatment. Vitamin B<sub>6</sub> was prescribed to prevent peripheral neuropathy. Physiotherapy and intensive guardian counselling were initiated at the hospital. The patient was reviewed by a spinal surgeon who did not recommend surgical intervention at that time, but suggested a review 3 months later. The patient was discharged home. The hospital palliative care team was involved and put him on their list for monthly home visits.

At 4 weeks he was followed up in the HIV outpatient clinic. His neurological deficits were unchanged. His CD4 count was 331 cells/ $\mu$ L and he was started on antiretroviral therapy.

At the 3-month follow-up by the palliative care team, the patient reported some subjective improvement in his gait. Clinically though, his deficits remained unchanged. He was taking his antituberculous and antiretroviral medication



• **Fig. 11.2** AP radiograph demonstrating a paravertebral soft tissue mass in the lower dorsal region with collapse of the T11 vertebral body. (Waldman, S.D., Campbell, R.S.D., 2011. *Imaging of Pain*, 1<sup>st</sup> ed. Saunders, Amsterdam. pp. 147–8)

regularly and was still waiting for a wheelchair. He did not attend the neurosurgical outpatient clinic because of problems with transportation.

### SUMMARY BOX

#### Spinal Tuberculosis (Pott's Disease)

Spinal TB is a collective term for spinal involvement in *Mycobacterium tuberculosis* infection. It comprises tuberculous spondylitis, tuberculous spondylodiscitis and tuberculous epidural and paraspinal abscess. Often, all manifestations occur together.

Spinal TB is the most common cause of tuberculous paraplegia and represents two-thirds of cases. Other causes are tuberculous radiculomyelitis (= arachnoiditis), tuberculous myelitis and intramedullary tuberculoma.

Constitutional symptoms are absent in more than half of patients, and simultaneous active pulmonary involvement is rather the exception than the rule. Inflammatory markers (ESR and CRP) are elevated in most cases and may be used as a screening method for patients from endemic regions presenting with back pain. Spinal radiographs may typically show focal areas of erosions and osseous destruction in the anterior corners of the vertebral bodies, involvement of the adjacent disc or vertebral body, 'wedging,' gibbus deformity or paraspinal abscess formation.

Compressive spinal cord involvement is a common and most feared complication caused by accumulation of epidural caseous debris or by vertebral collapse or dislocation.

CT-guided percutaneous vertebral or paravertebral biopsy and aspiration is the diagnostic gold standard. If biopsy is available, polymerase chain reaction (PCR)-based technology such as GeneXpert might expedite the diagnostic process and allow the early recognition of antibiotic resistance.

Antituberculous therapy is the same as for pulmonary TB; however, most national guidelines and centres recommend longer treatment for nine instead of 6 months.

Progression of bone destruction may continue for up to 1 year after effective antituberculous treatment has been initiated and should not be taken as a sign of treatment failure. Adjunctive corticosteroid treatment is not recommended for spinal TB. However, some centres use steroids in severe spinal cord compression to reduce oedematous swelling, which is a non-evidence-based approach. Surgery has been recommended for patients with (1) extensive extradural compression with features of spinal cord involvement, (2) no improvement or worsening of deficits after conservative treatment and (3) potentially unstable spine or kyphosis of more than 60 degrees. Surgical treatment relieves compression of the spinal cord, corrects kyphosis, may facilitate fusion and lead to faster pain relief.

## Further Reading

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