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# A 38-Year-Old European Expatriate Living in Malawi With Difficulty Passing Urine

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## Clinical Presentation

### History

A 38-year-old European expatriate presents at the medical outpatient clinic in a tertiary hospital in Malawi with progressive constipation and difficulty passing urine for the past 3 weeks. He noticed that he had to use increasing abdominal pressure to pass urine and eventually became only able to pass small amounts at a time. This is associated with increasing lower abdominal discomfort, abnormal sensations in the groins and around the genitals and some erectile dysfunction. He has no weakness and there is no dysaesthesia and no loss of sensation in his legs. However, he mentions that walking did not feel normal, although he cannot fully explain what the abnormality is. He has no backache.

Over the past 2 months he has experienced unintentional weight loss of 8 kg and fatigue without fever or night sweats. He blames this on stressful circumstances at work. He has sexual contact with his wife only, to whom he has been married for several years.

One week prior to presentation he had noticed reddish discoloration of his urine. He had no painful micturition or fever at that time. He visited a local clinic and was prescribed four different types of medication, which he completed. There is no history of trauma and the rest of his previous medical history is unremarkable.

### Physical Examination

He looks healthy and has normal vital signs. There are no abnormalities on general examination, except for a palpable bladder. The rectal examination reveals a low anal sphincter tone and the genital examination is normal. He has lively, symmetrical tendon reflexes in the legs without clonus or pathological plantar reflexes, and no abnormalities in the rest of the neurological examination.

## Questions

1. Which important pieces of information are missing?
2. What are possible diagnoses and which investigations would you order?

## Discussion

A 38-year-old European expatriate living in Malawi presents with constipation, bladder retention, an episode of probable haematuria, erectile dysfunction, genital paraesthesia, significant weight loss and fatigue. There are no abnormal neurological findings on examination, however most of the complaints are compatible with a conus syndrome because of a lesion within the conus medullaris or because of compression. The constitutional symptoms suggest an underlying chronic infection or malignancy.

### Answer to Question 1

#### *Which Important Pieces of Information Are Missing?*

The important missing pieces of information are details of the recent visit to the local clinic and exposure to fresh water in Malawi. On physical examination, the anal reflex should have been tested.

### Answer to Question 2

#### *What Are Possible Diagnoses and Which Investigations Would You Order?*

The most important differential diagnosis for a conus medullaris syndrome in a young, otherwise healthy man living in tropical Africa is spinal cord schistosomiasis. Other important infections to consider are spinal tuberculosis, a spinal abscess and neurosyphilis. A tumour or metastases impinging on the conus medullaris should be ruled out.

Urine and stool microscopy for ova of *Schistosoma haematobium* and *S. mansoni,* respectively, should be ordered. A full blood count should be done, including a white cell differential count to look for eosinophilia. Creatinine and C-reactive protein should be tested.

Schistosomiasis serology would be useful in this expatriate.

A VDRL and an HIV test should be done. An ultrasound of the abdomen assessing bladder volume before and after micturition and imaging of the spinal cord should be requested.

## The Case Continued…

The patient was able to retrieve details of his urine microscopy from the local clinic. *Schistosoma haematobium* eggs had been identified in his urine and he had been treated with a full dose of praziquantel, a course of ciprofloxacin, buscopan and bisacodyl.

He had swum regularly in Lake Malawi.

An abdominal ultrasound scan after attempted micturition showed a large bladder residue without further abnormalities.

Full blood count showed a normal absolute WBC of $9 \times 10^9$/L with a relative eosinophilia of 21.8% (reference range: 1–6%) and an absolute eosinophil count of $1.15 \times 10^9$/L (reference: $<0.45 \times 10^9$/L). ALT was slightly elevated. Creatinine and CRP were normal.

An HIV test and the VDRL were negative and microscopy of a stool and urine sample were normal. Serological tests for schistosomiasis were not available.

An MRI scan of the spinal cord was performed (Fig. 63.1). An enhancing 1 cm large lesion was seen at the anterior aspect of the conus, with associated high T2 signal representing oedema extending from the lower end of the conus to the level of T9. In addition, there was nodular thickening of the urine bladder wall at the base and the posterior aspect.


• **Fig. 63.1** T2-weighted MRI scan of the spinal cord.

The diagnosis of an infection with *Schistosoma haematobium* was made as evidenced by the urine findings and supported by the eosinophilia and the abnormalities seen in the bladder wall on MRI scanning. This was complicated by spinal cord schistosomiasis (see Summary Box) with lesions in the conus medullaris, causing a conus syndrome. A dual infection with *S. haematobium* and *S. mansoni* is possible, because *S. mansoni* is the more common pathogen causing spinal cord schistosomiasis, and both pathogens are endemic in Malawi. Theoretically, the spinal cord abnormalities could have been caused by other conditions, such as tuberculosis and metastatic cancer, for instance from a bladder carcinoma. Although it has been argued that *S. haematobium* infection is a risk factor for bladder carcinoma, this is mainly observed in long-term heavy infections, which are uncommon in expatriates. Further tests to rule out such distinct possibilities are not readily available in Malawi and were not deemed necessary.

The patient required urinary catheterization for 2 weeks. He made a full recovery after a second course of praziquantel and a course of high-dose prednisolone, tapered off over 2 months. He was well after more than 2 years of follow-up.

### SUMMARY BOX

#### Neuroschistosomiasis

Neuroschistosomiasis can occur in the brain and more frequently in the spinal cord. Cerebral schistosomiasis can be asymptomatic or present with seizures, lateralizing signs and meningo-encephalitis. Spinal cord schistosomiasis (SCS) is more frequent in *S. mansoni* than in *S. haematobium* infections and often causes severe disability because of paraparesis and bladder dysfunction. It is also the most important severe complication of schistosomiasis in travellers.

Our understanding of the pathophysiology of neuroschistosomiasis is limited. For unknown reasons, ectopic worms lodge in the venous plexus around the CNS instead of their normal habitat. Laminectomy with biopsy of the nervous tissue is the only method that gives a definite diagnosis of SCS. However, this procedure should be avoided because of its risks. Diagnosis is based upon spinal cord imaging and proof of exposure to the parasite. Urine should be examined for *S. haematobium* eggs; for *S. mansoni,* stool microscopy may be attempted but lacks sensitivity, which is much higher for rectal biopsies. The circulating cathodic antigen (CCA) test is a urine antigen test, which may prove *S. mansoni* infection. It detects an antigen regurgitated by adult worms of *S. mansoni.* It is less sensitive for *S. haematobium* and generally works better if the parasite burden is high. A novel test for the circulating anodic antigen (CAA) is under development and seems to be more promising for proof of both parasite species in urine and blood. Serological tests are useful in patients from non-endemic countries. They lack sensitivity and specificity in endemic populations. Detection of schistosomal antibodies in the CSF is specific but not widely validated. Eosinophils are present in the CSF in about 50% of patients.

Other causes of myelitis should be ruled out, which is often impossible in low-resource settings where pragmatic treatment for suspected neuroschistosomiasis may be justified.

Treatment of neuroschistosomiasis is with antischistosomal drugs (praziquantel) plus corticosteroids and is based on case series and expert opinion. The pathology is caused by inflammation around *Schistosoma* eggs. Praziquantel kills adult flukes only and thereby stops additional eggs being shed into the spinal cord. Most early improvement of the neurological presentation is thought to result from the antiinflammatory effects of corticosteroids. The optimal dose and duration are not well known. Up to 6 months of high-dose corticosteroids is recommended; however, data from randomized controlled trials are lacking.

Laminectomy should be considered in patients with severe spinal cord compression.

About 65% of patients with spinal cord schistosomiasis who are treated early, recover completely or are left with negligible deficits that do not cause any functional limitations; the remaining patients are left with sequelae that vary from mild to severe.

## Further Reading

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