SHORT REPORT

Transverse myelitis due to trypanosomiasis in a middle aged Tanzanian man

G S Kibiki, D K Murphy

J Neurol Neurosurg Psychiatry 2006;**77**:684–685. doi: 10.1136/jnnp.2005.079533

We report the case of a middle aged Tanzanian man who developed a spinal cord syndrome over 6 weeks, along with a mild encephalopathy. Investigations ruled out the usual major causes of such a syndrome in our setting in northern Tanzania. Examination of his cerebrospinal fluid revealed trypanosomes, and he made a slow but dramatic improvement after a full course of suramine and melarsoprol. We postulate that he had a transverse myelitis due to African trypanosomiasis, a rare and barely recognised cause.

Acute, non-traumatic myelopathy is a common clinical problem in medical practice in developing countries,¹ including sub-Saharan Africa.² For resource limitation and other reasons, a definite cause for such a myelopathy is often not found. Common causes include tuberculosis (TB) of the spine (Pott's disease), which usually affects the thoracic spine.^{3–4} HIV/AIDS is now an important cause of a myelopathy,⁵ including a vacuolar type, lymphoma, and opportunistic infections. Acute transverse myelitis, implying an inflammatory or demyelinating disorder of the spinal cord, occurs as a disordered response to, rather than the direct effect of, an infectious agent.⁶ It is seen in a variety of viral, bacterial, fungal, parasitic, and granulomatous diseases. In some tropical settings, schistosomiasis (bilharziasis) is a recognised cause of myelitis, with the spinal cord being a target for all three common forms of *Schistosoma*: *S. haematobium*, *S. japonicum*, and *S. mansoni*.⁷

Human African trypanosomiasis,⁸ or sleeping sickness, is caused by a flagellate protozoan, the trypanosome, which is transmitted by the bite of the tsetse fly. Only two species, belonging to the subgroup *Trypanosoma brucei*, are implicated in human disease: *T. brucei gambiense* in west Africa, and *T. brucei rhodesiense* in east Africa. South American trypanosomiasis (or Chagas' disease) is caused by *T. cruzi*, and transmitted to humans by the bite of infected reduviid bugs. A diffuse meningoencephalitis occurs with both African trypanosomiasis and Chagas' disease. Experimental work in rats, using *T. cruzi*, demonstrated spinal cord destruction.⁹ Myelitis has been described in Chagas' disease.¹⁰

In the UK, a case of African trypanosomiasis complicated by transverse myelitis was reported in a 67 year old woman returning from Zimbabwe.¹¹ She presented initially with a mainly encephalopathic picture, and only developed a transverse myelitis after 4 weeks of treatment.

We report a patient with African trypanosomiasis, where the main clinical finding was an acute spinal cord syndrome from the beginning, with recovery following treatment with anti-trypanosome drugs.

CASE REPORT

A previously well, middle aged native Tanzanian man presented with a 6 week history of progressive tingling,

numbness, and a prickly burning sensation that started in both feet and progressed upwards, with bilateral leg pain and weakness. This progressed to the point of being unable to walk. There was no history of back pain or trauma. He had no symptoms in his upper limbs. Bladder function was intact, but he had incontinence of bowel function. For 2 months, he had an intermittent fever, which was variously treated as malaria and typhoid. Review of systems revealed him to have anorexia and to have had some weight loss over the previous few months, with loose stools occurring 2–3 times per day. He worked in a rural location in northern Tanzania. He drank alcohol occasionally, and did not smoke cigarettes. He had healthy children. There was no history of blood transfusion or drug abuse.

On examination, he was febrile at 38°C, blood pressure was 90/50 mmHg, and pulse 120 beats/min (regular). He was wasted and pale, and had bilateral pitting oedema of his legs to upper thighs. There was no rash. He had ascites and reduced anal tone. He was minimally lethargic, but oriented in time, place, and person. There was no papilloedema, and cranial nerves I to XII were normal. He had wasting in all four limbs, reflecting generalised wasting. There was normal tone, strength, and coordination in the upper limbs, with normal reflexes and sensation. In his lower limbs, tone was flaccid, with 2/5 strength, areflexia, and a mute response to plantar stimulation bilaterally. Sensory examination showed reduced light touch and pinprick bilaterally to the ankle, with reduced proprioception in both sets of toes and normal ankle proprioception.

The initial impression was of an acute flaccid paraparesis and a mild encephalopathy in a patient with a systemic illness. The considerations were acute transverse myelitis, perhaps related to schistosomiasis, TB or other infectious or parasitic organisms; acute radiculomyelopathy; spinal cord compression (despite the absence of a clear sensory level) including Pott's disease; neurological manifestations of HIV/AIDS; other spinal cord lesions such as subacute combined degeneration of the cord (B12 deficiency), other toxic or metabolic causes of myelopathy; or spinal cord tumour.

Investigations

Repeated measurements of the patient's erythrocyte sedimentation rate were elevated at 145–155 mm/hour. His white cell count was increased to $10.2 \times 10^9/l$ (normal range (NR) $3.5–9.0 \times 10^9/l$), with neutrophilia $9.2 \times 10^9/l$ (NR $1.2–5.5 \times 10^9/l$), lymphocytes $1.0 \times 10^9/l$ (NR $0.6–2.9 \times 10^9/l$), haemoglobin 125 g/l (NR 100–140 g/l), and platelets $181 \times 10^9/l$ (NR $150–450 \times 10^9/l$).

Radiography of the thoracic and lumbar spine revealed only a mild scoliosis, convex to the left. Pelvic and chest radiographs were normal. Serology for VDRL (Venereal Disease Research Laboratories test), schistosomiasis, and HIV were all negative, as was Widal test. Stool analysis was

Abbreviations: CSF, cerebrospinal fluid; TB, tuberculosis

repeatedly negative for ova, cysts, and parasites. Stool culture was negative. Urinalysis for protein was repeatedly negative. Serum creatinine was repeatedly in the normal range. Total protein was 59 g/l (normal range 60–80 g/l). A lumbar puncture showed a colourless CSF, with 35 white cells, mostly polymorphonuclear cells, glucose 2.4 mmol/l (normal range 2.2–4.4 mmol/l), protein 0.42 g/l (normal range <0.5 g/l), no organisms or acid fast bacilli seen, and no growth on culture. On CSF microscopy, a moderate number of trypanosomes were seen. Ascitic fluid was slightly turbid, with protein of 18 g/l, and 49 white cells, mainly lymphocytes, seen. Gram stain was positive for pus cells, and no organisms were detected.

Clinical course

The patient was treated with metronidazole, ciprofloxacin, ceftriaxone, and multivitamins while investigations were undertaken. Initially, the weakness in his lower limbs got worse over the next 3 weeks, and he became paraplegic, with flaccidity and areflexia. When the CSF revealed trypanosomiasis, he was treated with suramine and melarsoprol. Two weeks later, he was documented as having “mild improvement”, with a flicker of movement in his toes; he was not encephalopathic. Four months later, he was documented as having “paraparesis” rather than a paraplegia. Nine months after the start of his illness, he could walk without support. About 1 year after hospitalisation, his lower limb muscle bulk and tone were normal, while muscle power was 4/5 in the right leg and 5/5 in the left. Leg reflexes were now exaggerated bilaterally, with toes mute to plantar stimulation, no clonus, and normal sensation. Upper limbs were normal.

DISCUSSION

The onset, progression, and clinical outcome in this case support a spinal cord syndrome, best fitting with a transverse myelitis. The absence of a clear sensory level and areflexia (at least initially) do not militate against this. An acute inflammatory demyelinating polyneuropathy (Guillain-Barré syndrome) is argued against by the absence of upper limb and facial involvement, the CSF findings, and the ultimate development of exaggerated leg reflexes. Pott's disease is very unlikely because of the radiological findings and the good clinical outcome without anti-TB treatment. Similarly, other causes of spinal cord compression or a spinal cord tumour would not have had such a good outcome. The negative HIV test and clinical course rule out a myelopathy associated with HIV/AIDS. The absence of specific anti-schistosomal treatment, negative serology, and a good clinical outcome all argue against schistosomiasis as an aetiology. A tropical spastic paraparesis would have presented more slowly, and would have continued to progress. The full blood picture and subsequent clinical course do not support metabolic disorders, such as B12 deficiency. The negative VDRL and clinical course make a syphilitic myelopathy unlikely.

Because of resource limitations, not all possible investigations could be carried out. However, the investigations that were performed help to rule out common causes of a spinal cord syndrome in our setting. These results, along with the findings of trypanosomes in the CSF and the response to anti-trypanosomal treatment, lead us to believe that this patient had a transverse myelitis related to *T. brucei*. His mild

encephalopathy would also be consistent with this. In addition, his work exposed him to the tsetse fly, and bites are difficult to detect in people with dark skin colour. We believe that this was a parainfectious immunological phenomenon rather than a direct invasion of the spinal cord and roots, although this remains speculative. Pathophysiologically, *T. brucei* invades and has a marked tropism for the CNS, giving rise to perivascular inflammatory lesions, immune complex formation, and a generalised vasculitis. A demyelinating encephalitis is the terminal stage of the disease.¹² A well described diffuse meningoencephalitis is the usual dominant clinical picture. Perhaps the usually dominant cerebral symptoms may mask a myelopathy in patients with African trypanosomiasis. There are, however, no theoretical reasons why the pathological changes described above could not occur in the spinal cord. Clinically, the time course in our patient fits with an inflammatory or demyelinating myelopathy secondary to *T. brucei*, with a good recovery. We contend that such a potentially reversible spinal cord syndrome related to *T. brucei* is rarely recognised. We suggest that trypanosomiasis should be considered as a possible cause of an acute and treatable myelopathy in a tropical setting.

Authors' affiliations

G S Kibiki, D K Murphy, Department of Internal Medicine, Kilimanjaro Christian Medical Centre, Tumaini University, Moshi, Tanzania

Competing interests: none declared

Correspondence to: Dr D K Murphy, Department of Clinical Neurological Sciences, Beaumont Hospital, Dublin 9, Ireland; kevdamurphy@oceanfree.net

Received 1 September 2005

In revised form 23 October 2005

Accepted 2 November 2005

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