Three major species of *Schistosoma* are known to infect humans: *S. mansoni*, *S. hematobium*, and *S. japonicum*, each having a specific geographical endemic area. *S. mansoni* is seen in the Middle East, Africa, South America, and the Caribbean; *S. hematobium* is found in Africa and the Middle East; and *S. japonicum* is found in Japan, China, and the Philippines. All three are known to involve the central nervous system. Because it is rarely seen in North America, a diagnosis of schistosomiasis may be difficult to make, despite its characteristic clinical profile. This report describes a case of *S. mansoni* affecting the spinal cord and presents a short review of the literature.

**Case Report**

This 34-year-old man from Yemen was referred for evaluation because of progressive spastic paraparesis. In January 1992, the patient received 220 V electric current while working with electrical equipment in Yemen and fell 12 feet landing on his back. After approximately 10 minutes of unconsciousness, the patient fully recovered without any neurological deficit. The next morning he developed urinary retention, which has persisted since then. Three weeks later, he noted low-back pain, followed by a progressive paraparesis over the next few weeks. The patient was admitted to a hospital in Egypt for a 2-month period during which he underwent plasmapheresis for a suspected “degenerative neuron disorder.” He was transferred to a medical center in the United States and then referred to the Mayo Clinic for further evaluation in April 1992, 9 weeks after the last onset of the symptoms.

**Examination.** On admission, the patient had a moderate spastic paraparesis that was worse in the distal muscles, and he was unable to walk independently. There was moderately decreased sensation for light touch and pain below the T-10 dermatome. Proprioception and vibration sensation were absent below T-10 on both sides. His bladder and bowel dysfunction required catheterization and intermittent digital removal of stool. The upper extremities were intact both for sensory and motor testing. Hematocrit was 37.6%; white blood cell count was 5000 (34% neutrophils, 2% band forms, 47% lymphocytes, 6% monocytes, and 11% eosinophils). Blood chemistries with liver function tests were normal. The patient’s cerebrospinal fluid examination showed total protein at 102 mg/dl, nucleated cells at 108 cells/ml with 100% lymphocytes, and glucose at 55 mg/dl. A myelogram showed mild enlargement of the spinal cord below the T-5 level without any significant block; magnetic resonance (MR) imaging showed an enlarged spinal cord with spotty enhancement with gadolinium injection below T-9 (Fig. 1), and increased T₂-weighted signal intensity below T-5.

**Operation.** With the patient under general anesthesia, a T8–12 laminectomy was accomplished. The dura appeared normal. The arachnoid was opaque over these segments. The spinal cord was gray-yellow and irregularly enlarged showing a lumpy surface. Many sensory rootlets were enlarged forming small 1- to 2-mm nodules. Biopsy specimens were obtained from one of the abnormal roots and also from the left posterior column, both revealing ova of *S. mansoni* associated with granulomatous inflammation (Fig. 2). The dura was closed with a fascia lata graft.
**Postoperative Course.** The patient remained unchanged neurologically following the procedure. Stool examination was done the same day and revealed ova of *S. mansoni*. To detailed inquiry, the patient stated that he had been swimming in a river in March 1991 and had “swimmer’s itch” immediately after that. The patient was diagnosed with transverse myelitis caused by *S. mansoni* and was given praziquantel 40 mg/kg in a single dose orally, as well as dexamethasone 4 mg every 6 hours. He showed significant improvement in strength in his lower extremities shortly after the treatment was initiated, and in the stool collected 10 days after surgery, no ova of *S. mansoni* were identified. On examination 4 months later, the patient was able to walk with forearm crutches and could void spontaneously. An MR image showed a mildly atrophic spinal cord without any abnormal intensity or gadolinium enhancement.

**Discussion**

Schistosomiasis infects humans percutaneously as cercariae, which are liberated by a freshwater host snail. The characteristic dermatitis known as “swimmer’s itch” occurs occasionally at the sites of the penetration within 24 hours of infection, with an intensely pruritic reddened lesion measuring several millimeters in diameter, often with a necrotic center. The cercariae migrate into the lungs and liver and then descend to the venous system contiguous with the colon. *S. mansoni* and *S. japonicum* inhabit portal veins; *S. hematobium* inhabits veins of the bladder. They mature to adult worms in 4 to 8 weeks, then begin to lay eggs. An acute reaction known as “Katayama fever” is sometimes seen at this period, which consists of an acute onset of fever, chills, sweating, and cough.

Spinal cord schistosomiasis is a relatively rare manifestation of chronic schistosomiasis with less than 100 cases reported in the literature. The time between exposure to infection and onset of the spinal cord dysfunction varies from 38 days to years. The ova migrate into the spinal cord via the valveless vertebral venous plexus of Batson and cause marked inflammation at encysted sites. The most frequent neurological consequence is an acute or subacute transverse myelitis. The thoracolumbar portion is most commonly affected. Urinary retention and motor and sensory disturbance of lower extremities associated with back pain are the early symptoms. Granuloma formation is the other, but less common, type of neurological consequence, being usually seen at conus level. Conus medullaris and cauda equina syndrome are the manifestations.

Schistosomiasis is a treatable disease. Praziquantel, the most favored drug for the treatment, is given in a single oral dose of 40 mg/kg to kill adult worms and prevent further oviposition. High-dose steroid therapy is reported to improve the clinical symptoms by reducing the inflammatory process caused by ova in the spinal cord. The prognosis is more favorable with early institution of therapy, making early diagnosis mandatory for optimum results.

Diagnosis is made by demonstration of evidence of schistosomiasis infection. In *S. mansoni*, this is made by recovering ova from the stool or by retrieval of adult worms in a rectal biopsy specimen. In *S. hematobium*, ova are not seen in stool but in urine. Eosinophilia in periph-

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**Fig. 1.** A T₁-weighted gadolinium-enhanced magnetic resonance image revealing a slightly enlarged spinal cord below T-9 with spotty enhancement.

**Fig. 2.** Photomicrograph from a biopsy specimen of the spinal cord showing the characteristic appearance of *Schistosoma mansoni* egg with surrounding granulomatous inflammation. H & E, original magnification × 300.
Schistosomiasis involving the spinal cord

eral blood may be seen, but occasionally are not obvious, especially in the chronic phase. Cerebrospinal fluid examination typically shows mild pleocytosis and an elevated protein content. An enzyme-linked immunosorbent assay test of cerebrospinal fluid is reported to be positive in most cases.4

Myelography with thin-slice computerized tomography shows expansion of the conus medullaris in cases of granuloma formation, but normal or equivocal findings in transverse myelitis;3,5,6 the infected nerve roots may show an irregularly matted appearance.6 Magnetic resonance findings of spinal cord schistosomiasis have not been reported. In our case, MR images showed a mildly enlarged spinal cord below the low thoracic level with increased T2-weighted intensity and patchy enhancement by gadolinium injection on T1-weighted images. These findings correlated well with the pathological process and hence would be characteristic for this rare disease.

The only direct diagnostic procedure is surgery. Even if the schistosomiasis infection is demonstrated by the non-invasive tests mentioned above, it can be a coincidental finding especially if the patient is from an endemic area. Therefore, it may be necessary to obtain tissue diagnosis in questionable cases, so that treatment is not delayed. In retrospect, a preoperative stool examination might have been appropriate in our case, but would probably not have changed our management, including our decision to perform an open biopsy. Decompressive laminectomy also is a reasonable option for the patient with a conus granuloma.3,6

The relation between the injury and the development of symptoms observed in our case is unclear; however, there might have been an increase of abdominal pressure at the injury that subsequently caused the oviposition at the spinal cord.

One must have an index of suspicion to recognize the characteristic clinical profile of this rare disease, which will lead to prompt and appropriate treatment.

References


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