MR Imaging of Schistosomal Myelitis

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Myelitis is a rare complication of Schistosoma mansoni, and is characterized by a dramatic involvement of the lower part of the spinal cord [1, 2]. Early antischistosomal therapy is able to improve markedly clinical signs [3, 4]. Myelography and CT [3, 5–10] are normal in most cases of Schistosoma mansoni transverse myelitis [2, 10]. As far as we know, no MR studies have been reported. We present a patient in whom MR was of great value.

Case Report

A 29-year-old man had lived in Zaire since 1983 and had traveled to Zambia in 1987. He swam in stagnant waters in both countries. In January 1989, he suffered pain in the left calf that, after a few days, extended to both legs and the lumbar spine, with nocturnal exacerbations that required pentazocine administration. Seven days later, leg weakness and bladder and bowel disturbances were noted. The patient was admitted on the 15th day and clinical examination revealed a paraparesis, mainly distally; loss of patellar reflexes; indifferent cutaneous plantar reflexes; and a tactile hypesthesia in both legs. Usual biological tests were normal, including ESR, lymphocyte subsets typing, serology for HIV1, HIV2, HTLV1, and Treponema pallidum. Schistosoma mansoni ova were found in stools. Hepatic sonography showed a few hyperechogenic foci in the left hepatic lobe, suggesting punctate calcifications. Dorsal and lumbar myelography were performed on the 18th day. Frontal, oblique, and lateral views were obtained in the prone and supine position, and no spinal cord enlargement was observed at any level. CSF studies demonstrated 194 white cells/mm³, mainly lymphocytes with 5% eosinophils, and a protein content of 88 mg/dL. Antibodies against Schistosoma mansoni were positive by ELISA technique in serum (optic lecture: 0.360; cut-off <0.100) and strongly positive in CSF (optic lecture: 0.766; cut-off <0.050). MR of the conus medullaris performed 3 days after myelography demonstrated a mild enlargement of the spinal cord with centromedullary hyperintense signal on the mid-sagittal T2-weighted image (Fig. 1A). A mild enlargement was also noted on T1-weighted images (Fig. 1B). After IV injection of gadopentetate dimeglumine (0.1 mmol/kg body weight), small and heterogeneous areas of intramedullary contrast uptake were noted (Fig. 1C).

Praziquantel (30 mg/kg, twice at 4-hr intervals) was administered on the 19th day concurrently with steroids. Pain quickly diminished and 4 months later the patient was able to walk much better; however, mild dysuria and painful dysesthesias remained. Clinical examination showed bilateral Babinski signs, asymmetric patellar reflexes, and disturbed tactile and vibratory sensations in both legs. Neurophysiological studies demonstrated modest peripheral nerve involvement and much less pronounced disturbances of somatosensory evoked potentials than in February. CSF analysis had returned to normal, and antibodies against Schistosoma mansoni were lowered in serum (0.272) and CSF (0.360). Control MR studies of the spinal cord were normal at that time (Fig. 2). Pain was markedly reduced with carbamazepine, and the patient was able to resume his work.

Discussion

Our patient presented with a subacute paraparesis preceded by pain and associated with loss of reflexes and bladder and bowel disturbances. These clinical signs were caused by a transverse myelitis at the thoracic and lumbar levels. Schistosoma mansoni rarely provokes neurologic symptoms; instead, it is usually characterized by low spinal cord syndromes attributed to ova deposition from inferior mesenteric venules [1, 2, 4, 9]. Host granulomatous reaction to the ova is the major factor in the pathogenesis of schistosomiasis [1, 2, 9]. Typically, an early symptom is low back pain, which sometimes radiates to the perineum and legs, followed within a few days by paraplegia with sensory disturbances and incontinence [2]. However, the severity and the speed of evolution vary considerably. Four separate but associated syndromes have been distinguished: medullary compression, acute transverse myelitis, granulomatous root involvement, and anterior spinal artery occlusion [2, 4, 9]. Fifty-three histologically proved cases of spinal cord involvement by Schistosoma mansoni have been reported [1, 10], as well as 15 similar cases due to Schistosoma haematobium. According to the Center for Disease Control a presumptive diagnosis can be based, in absence of histologic proof, on low thoracic and lumbar spinal cord symptoms, serologic or parasitologic demonstration of exposure to Schistosoma, and exclusion of other known causes of transverse myelitis [4]. Our patient meets these diagnostic criteria. Demonstration with the ELISA technique of antibody titers against Schistosoma mansoni higher in the CSF than in the serum is further evidence. Indeed, similar comparative data have been reported previously in spinal cord schistosomiasis with immu—
Fig. 1.—A–C, Mid-saggital MR scans of mildly enlarged conus medullaris at 1.5 T with section thickness of 6 mm. TR/TE = 1800/100 (A) and 415/20 (B and C). Arrow in C indicates uptake of contrast material after IV injection of gadopentetate dimeglumine.

Fig. 2.—Mid-saggital MR image (1800/200) shows normal appearance of conus.

myelitic type, and only MR was able to demonstrate spinal cord lesions. However, these MR findings are nonspecific, since the same characteristics may be encountered in intramedullary stroke and other types of inflammatory disease [13].

REFERENCES