Pseudotumoral form of cerebral Schistosomiasis Mansoni

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ABSTRACT

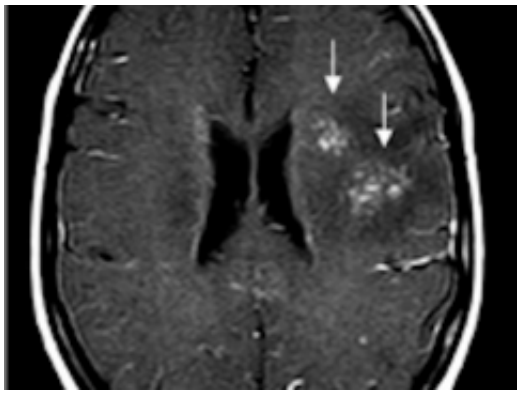
The authors report a case of 36-year-old woman presented with epileptic seizures and headaches. Magnetic resonance imaging (MRI) revealed an enhancing lesion with surrounding edema and mild mass effect in the left frontal lobe. Stereotactic brain biopsy demonstrated intraparenchymal granulomas surrounding *S. mansoni* eggs. Praziquantel was started (60mg/kg of body weight, in a single dose), followed by Prednisone (80mg/day) for seven days to treat the cerebral edema. The patient's symptoms resolved following medical treatment and the follow-up MRI yielded normal findings.

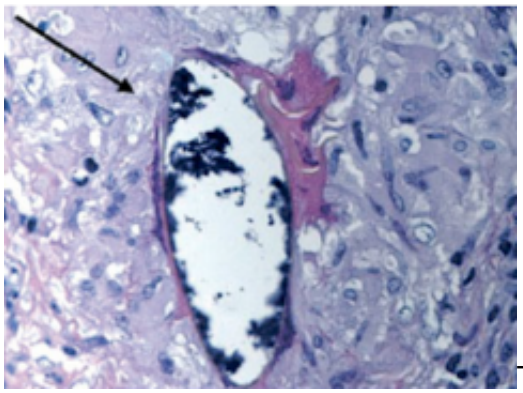
INTRODUCTION

Schistosomiasis is a common parasitic infection in some regions, transmitted to human by skin contact (1,2). Three major *Schistosoma* species can infect humans: *Schistosoma haematobium* (endemic in Africa and the eastern Mediterranean), *S. mansoni* (endemic in Africa, the Middle East, the Caribbean, and South America), and *S. japonicum* (endemic mainly in China, Japan, and the Philippines). More than 200 million people are infected by schistosomiasis worldwide (1-3). Humans are the definitive host, although certain aquatic snails act as the intermediate host. Adult parasite is a flat, elongated fluke that lives in the mesenteric (*S. mansoni* and *S. japonicum*) or pelvic *(S. haematobium)* veins (4). Their females lay hundreds to thousands of eggs per day, which are excreted in human urine or feces. Intestinal or urinary symptoms are predominant, when eggs in bowel or bladder walls produce a localized inflammatory response (2,4). Although gastrointestinal system infection is common, cerebral schistosomiasis is very rare. Cerebral lesions most frequently occur with *S. japonicum*, whereas *S. mansoni* and urinary or bladder schistosomiasis may involve the spinal cord (5). The authors describe an unusual case of cerebral pseudotumoral form of *S. Mansoni.*

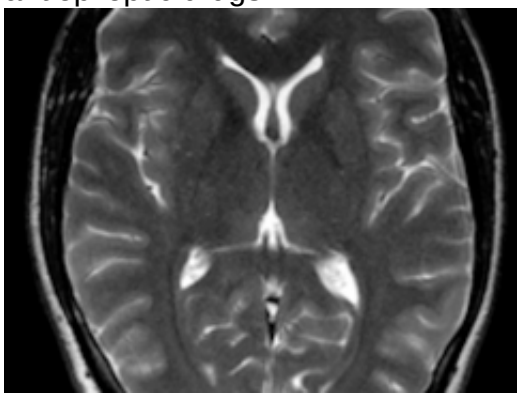
CASE REPORT

A 36-year-old female presented with severe headache followed by seizures five days before her admission in the hospital. Neurological examination showed mild right hemiparesia. A computed tomography scan (CT) showed a contrast-enhanced tumor mass in the left parietal and frontal lobes, surrounded by edema. Axial contrast-enhanced T1-weighted MR imaging showed a focal multinodular pattern (Fig. 1).





The histological exam (H&E 400x) revealed presence of *Schistosoma mansoni* eggs, surrounded by proliferative granulomata (Fig. 2). Praziquantel was started (60mg/kg of body weight, in a single dose), followed by Prednisone (80mg/day) for seven days. She was discharged from hospital 10 days later in a good general condition. Two months later, she was asymptomatic. No motor or sensory deficits were associated with the biopsy or the presence brain lesion. Seizures were controlled with antiepileptic drugs.



DISCUSSION

Schistosmoiasis is transmitted through skin contact with infected water. *S. mansoni* larvae enter to human circulation penetrating the skin. After several days, pairs of worms migrate to the inferior mesenteric veins. Egg production begins 4 to 6 weeks after initial infection passing through the blood vessel and the intestinal mucosa to finally shed in the feces. The life cycle is complete when the eggs hatch, releasing miracidia that infect specific freshwater snails (*Biomphalaria* species). Miracidia will then develop into sporocysts and produce cercariae (6). Central nervous

system (CNS) involvement is a rare ectopic manifestation of schistosomiasis, more commonly seen in *S. japonicum* infections and only few documented cases were reported in the literature (7,8). Pathogenesis of cerebral schistosomiasis is not completely understood. Clinical findings are associated with host inflammatory response against the eggs siting the brain by embolization through venous shunts, secondary to hepatic and pulmonary hypertension. Some authors believe that the cerebral form is caused by aberrant worms migration to the vertebral venous plexus (Batson plexus), producing eggs directly in the CNS (8). Diagnosis is difficult because clinical findings are nonspecific and laboratory changes such as eosinophilia and evidence of *Schistosoma* ova in stool or urine may or may not be present (9). Neuroimaging usually shows a tumor-like lesion with mass effect and heterogeneous contrast enhancement mainly at the temporoparietal, occipital, and frontal regions. Antibody detection in blood samples or cerebrospinal fluid is useful in a few specific circumstances, and eosinophilia is not a constant finding in cerebrospinal fluid analysis (1). The most practical laboratory examination for the investigation of cerebral schistosomiasis is wet smear stool examination, which can determine the presence of eggs in feces, but positive results are only supportive for the diagnosis (10). Patients treated with complete surgical resection or biopsy and antihelmintic medication (Praziquantel or Oxamniquine) normally have a good outcome (2). Oxamniquine or praziquantel cause death of the adult worm decreasing the inflammatory response being used in non CNS *S. mansoni* infection (2). Corticosteroids are expected to diminish granulomatous inflammation and edema, thereby preventing further tissue damage (9,10). Our patient was treated with Prednisone and Praziquantel therapy improving all symptoms. S. mansoni is an unusual cause of cerebral mass lesions although schistosomal eggs can be frequently found in the brains of individuals in countries where S. mansoni is endemic (1). We described a patient with a space-occupying cerebral lesion and schistosomal granulomas on pathological examination and S. mansoni identified in brain biopsy. The cerebral lesion responded to therapy with Praziquantel and corticosteroids. The authors report a case of 36-year-old woman presented with epileptic seizures and headaches