Cerebral Schistosomiasis: MR and CT Appearance

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Summary: After returning from Africa, a 54-year-old man began to have episodes of headache and nausea, then a cerebral convulsion. Clinical and laboratory findings and response to chemotherapy indicated the diagnosis of cerebral schistosomiasis. Three lesions were seen on CT and MR studies: two appeared to be subacute intracerebral hematomas, one in the right parietal lobe and one in the frontal lobe; the third lesion, in the cortex of the left occipital lobe, appeared to be a cyst. These lesions could represent small granulomatous tissue reactions with secondary hemorrhages.

Index terms: Brain, infection; Brain, inflammation

Schistosomiasis, caused by trematode blood flukes of the species *Schistosoma,* is characterized by chronic inflammation of the intestines and urinary bladder. Cerebral involvement is a rare ectopic manifestation of schistosomiasis. Although ova can be found in the brains in a majority of autopsied cases with proved schistosomiasis, only 2% of persons with acute schistosomiasis incur complications of the central nervous system (1, 2). Clinical symptoms such as acute encephalopathy, seizure disorders, mass lesions, and paresis, as well as radiologic findings, can simulate a brain tumor, but in most cases thorough knowledge of the patient's history provides clues to the correct diagnosis.

We present a case of cerebral schistosomiasis and describe the computed tomographic (CT) and magnetic resonance (MR) imaging findings.

Case Report

A 54-year-old man, who until 3 months prior to admission had been working as an engineer in different parts of Africa for some years, was brought to the emergency department after an acute generalized cerebral convulsion at home. During the previous 5 weeks he had experienced intermittent episodes of headache and nausea. On admission he was found to have slow mental functions and showed signs of recent bite injuries of the tongue, but no major neurologic dysfunction was detected.

Laboratory findings revealed a leukocyte count of 11 000/μL, a normal red blood count and no eosinophilia, cerebrospinal fluid (CSF) with 7/3 cells, 0.81 g/dL proteins, and 3.7 mmol/L glucose. With the CSF ELISA, antibodies against *Schistosoma* organisms were detected, which had not been present at previous routine examinations. No antibodies against *Borrelia* organisms were found in the CSF. The serum contained no antibodies against neurophile virus; antibodies against Epstein-Barr virus–viral capsid antigen were 1:256. Stool contained vermicular eggs of *Schistosoma mansoni*. The patient had normal electrocardiographic and electroencephalographic findings, and results of chest radiography and abdominal CT were also normal. During colonoscopy no macroscopic signs of inflammation were found, but histologic evidence of chronic ileitis and colitis was evident. Cystoscopic examination showed no indications of schistosomiasis of the genitourinary system.

CT of the cerebrum was performed on the day of admission and showed an oval hyperdense area, 1.2 cm in diameter, located in the paraventricular zone dorsal to the right posterior horn (Fig 1A). The measured density was 56 Hounsfield units (HU). After injection of contrast medium, discrete central and marginal enhancement was noted. In the precentral gyrus of the left frontal lobe, a second, less clearly defined lesion of about the same size was observed, which was of high density (55 HU) on the unenhanced scan, with marginal enhancement of contrast medium. Both lesions caused only minimal displacement of surrounding tissue. The CT characteristics of the lesions indicated subacute hemorrhages. A third lesion was detected in the cortex of the left occipital lobe with density equivalent to fluid and a discrete ring-shaped contrast enhancement, indicative of a cyst. This lesion was about 1.5 cm, with no evidence of displacement of the surrounding tissue.

Five days later, cerebral MR imaging was performed before and after administration of contrast medium. Corresponding to the area of the lesion dorsal to the right posterior horn on CT scans, MR imaging revealed an oval area of intermediate signal intensity on spin-echo T1-weighted images and of low signal intensity surrounded by

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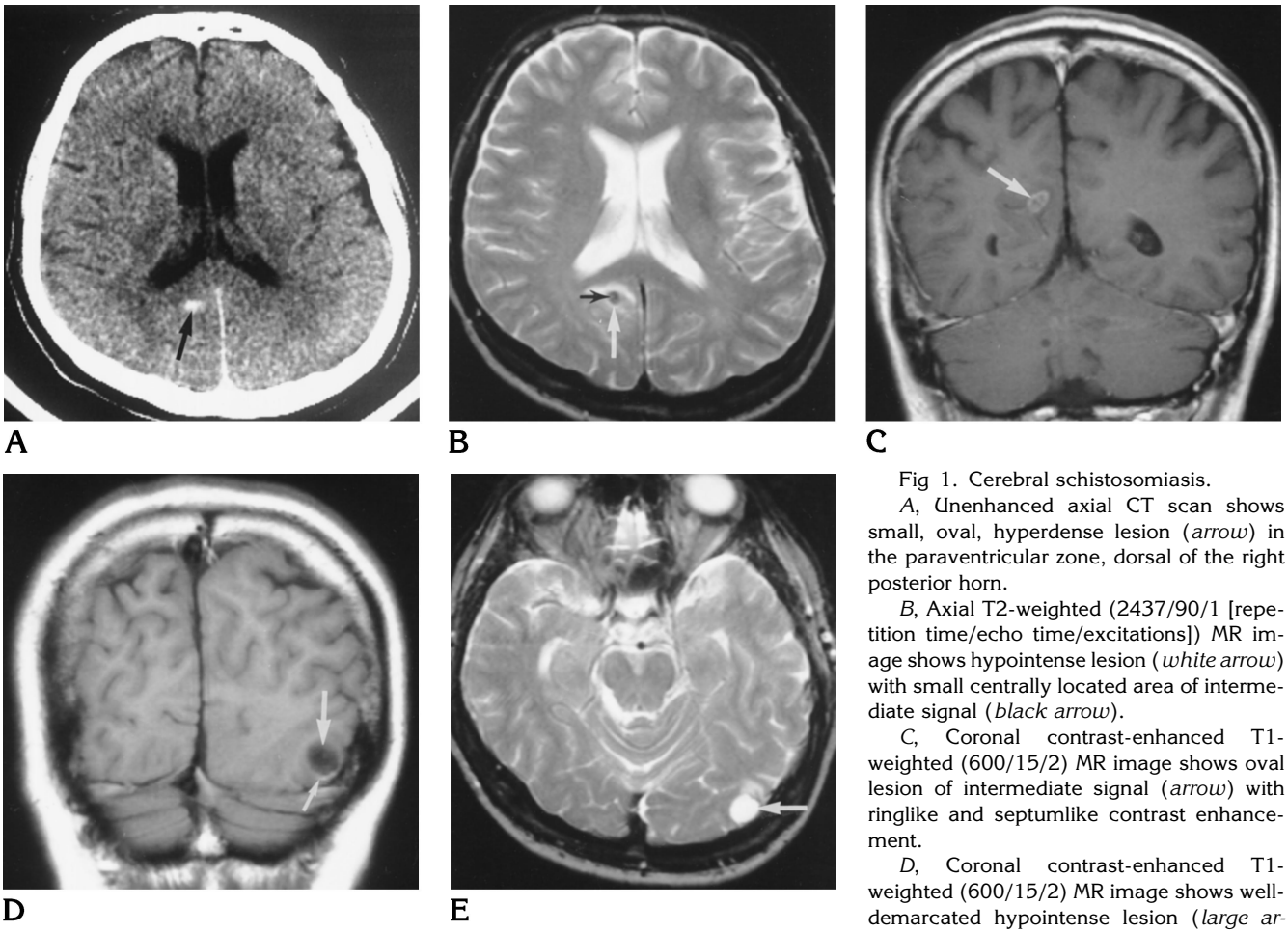

Fig 1. Cerebral schistosomiasis.

A, Unenhanced axial CT scan shows small, oval, hyperdense lesion (*arrow*) in the paraventricular zone, dorsal of the right posterior horn.

B, Axial T2-weighted (2437/90/1 [repetition time/echo time/excitations]) MR image shows hypointense lesion (*white arrow*) with small centrally located area of intermediate signal (*black arrow*).

C, Coronal contrast-enhanced T1-weighted (600/15/2) MR image shows oval lesion of intermediate signal (*arrow*) with ringlike and septumlike contrast enhancement.

D, Coronal contrast-enhanced T1-weighted (600/15/2) MR image shows well-demarcated hypointense lesion (*large arrow*) with discrete marginal contrast enhancement (*small arrow*) located in the cortex of the left occipital lobe.

E, Axial T2-weighted (2437/90/1) MR image shows fluid-equivalent hyperintense lesion (*arrow*) in corresponding location to that in *D*.

high signal on spin-echo T2-weighted sequences (Fig 1B). Centrally within the lesion a round area, only a few millimeters in diameter, with intermediate to slightly hyperintense signal could be observed. After administration of contrast medium the whole lesion showed marginal and septumlike enhancement on spin-echo T1-weighted images (Fig 1C). In the left precentral gyrus the second lesion showed low signal intensity on both spin-echo T1-weighted and T2-weighted sequences with a small focal area of intermediate signal centrally. The lesion was surrounded by high signal intensity on T2-weighted images and marginal contrast enhancement on T1-weighted images. The MR signal characteristics of both lesions were typical for subacute hemorrhages caused by underlying granulomatous lesions, as indicated by the focal areas in the center of both lesions and the septumlike contrast enhancement. MR characteristics of the third lesion in the cortex of the left occipital lobe included fluidlike hypointense signal on T1-weighted images and hyperintense signal on T2-weighted sequences with thin marginal enhancement clearly indicating a cyst (Fig 1D and E).

Diagnosis was confirmed by clinical and laboratory findings and was further supported by the recovery of the patient after chemotherapy with a single dose of praziquantel, 1 mg/kg body weight. The patient was lost to follow-up after a period of 8 months, during which time he refused further CT or MR examinations.

Discussion

Schistosomiasis is endemic throughout much of the tropics. Three different schistosomal species (*S haematobium, S japonicum, and S mansoni*) can cause infection that involves the brain and spinal cord (3). Brain involvement is found in about 4% of all patients infected by *S mansoni* (4). The life cycle starts with cercariae, which penetrate the human skin and transform into schistosomulae. From there they migrate to the lungs and the liver. The organisms then mature into mating pairs of male and female worms,

which settle in the mesenteric veins (5). The adult worm lay eggs that are excreted with stool or urine. Different mechanisms of invasion of the brain have been discussed: the eggs may reach the brain through the valveless venous plexus of Batson, which joins the deep iliac veins and inferior vena cava with veins of spinal cord and brain, or eggs may migrate to the brain via pulmonary arteriovenous shunts, or portal-pulmonary arteriovenous shunts (3, 6, 7). Finally, the worms themselves may enter the cerebral veins and place their eggs directly at the ectopic site, which could be the cerebrum, cerebellum, leptomeninges, brain stem, or choroid plexus (7, 8). The host then develops an immunologic reaction to the deposited ova; the response may cover a wide spectrum, from a very intense reaction, resulting in granulomas or space-occupying mass lesions, to a minimal reaction with no clinical manifestation at all.

Diagnosis can be difficult, as clinical findings are nonspecific and laboratory changes such as eosinophilia and evidence of schistosome ova in stool or urine may or may not be present. However, epidemiologic history in combination with positive results of CSF ELISA and the patient's recovery after chemotherapy can establish the diagnosis, although biopsy of the intracerebral lesions is considered the standard of reference (5, 7).

Cerebral schistosomiasis caused by *S haematobium* has been described as a nonspecific granulomatous lesion, hypodense on CT scans and with low signal intensity on T1-weighted MR images and isointense on T2-weighted MR images as a response of the host to the ova (9, 10). However, several studies have indicated that intracerebral deposition of worm eggs does not always cause granulomatous tissue reaction (11, 12).

Our patient had two lesions with the CT and MR imaging characteristics of subacute intracerebral hematoma and one cystic lesion, which might have been a focal area of an old cortical hemorrhage after resorption. The central areas of isointense to slightly hyperintense signal in the paraventricular lesion and the lesion in the precentral gyrus, including the septumlike contrast enhancement, might suggest that these lesions represent small granulomatous tissue reactions with secondary hemorrhages. Our observation goes along with the description of one Brazilian case in which *S mansoni* caused focal intrahepatic and intrapulmonary hemorrhages and intracerebral and intracerebellar hematomas of different stages of resolution (13). The latter signs were caused by an intense tissue reaction against the schistosomal eggs, leading to necrosis of the vascular walls with hemorrhagic transudation, which was responsible for the patient's symptoms (13).

Focal intracerebral hematomas can certainly also occur in other underlying lesions, such as angiomas or vascular malformations. Although our patient refused an angiographic study of his brain vessels, the radiologic appearance of the lesions as well as normal findings on contrast-enhanced CT studies obtained during routine examinations at 1 and 2 years before this infection nearly exclude these possibilities.

S mansoni is endemic in Africa, parts of the Near East, parts of South America, and in the eastern Caribbean islands (5). However, its increasing prevalence in the United States and Europe, caused by greater tourism to endemic regions and by emigration from these countries, make it important that physicians be aware of this entity, including its radiologic characteristics.

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