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Case Report
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Schistosomiasis Mansoni Cerebral Pseudotumor

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Abstract

We describe a case report of a potentially treatable condition of Schistosomiasis mansoni cerebral pseudotumor in Uganda, an ectopic form of the disease that is mainly associated with Schistosoma japonicum. It is presumed to be extremely rare, neglected and underestimated despite the high prevalence of schistosomiasis mansoni in many developing countries. This is a 25 year-old man who presented with a history of recurrent seizures despite anticonvulsants and headaches unresponsive to non-steroidal anti-inflammatory drugs. Brain Magnetic resonance imaging revealed a T1 Gadolinium enhancing lesion with surrounding edema and mild mass effect in the right occipital lobe. He underwent brain surgery for possible meningioma but biopsy demonstrated intra-parenchymal granulomas surrounding Schistosoma mansoni eggs on histology. Praziquantel was started (60 mg/kg of body weight, in a single dose) and Prednisone (80 mg/day) for seven days to treat the cerebral edema. The patient's symptoms resolved following medical treatment and the follow-up brain MRI was normal.

Keywords: Schistosomiasis; Brain; Pseudo tumor

Introduction

Schistosomiasis is endemic in Africa, South America and Asia with over 200 million people affected worldwide [1]. It is estimated that approximately 110 million people are infected with Schistosoma mansoni worldwide [2]. The main human species that occur in Africa are Schistosoma mansoni and Schistosoma haematobium [3]. In Uganda it is estimated that approximately 4 million people have schistosomiasis [4].

Despite a high burden of schistosomiasis in Africa [2], few cases of cerebral schistosomiasis have been described. The Schistosoma mansoni cerebral pseudotumor formation is extremely rare [5-7]. We therefore describe a case in which Schistosoma mansoni infection simulated a cerebral tumor in Uganda, a developing country.

Case Presentation

A 25year old male was referred and admitted in January 2013 following a three week history of progressively worsening low grade fevers, associated with throbbing occipital headache, occasional blurring of vision, episodes of confusion and generalized tonic clonic convulsions that lasted 5 min. The convulsions were associated with postictal confusion lasting about two minutes. These were the index seizures with no history of febrile seizures during childhood, trauma or a familial history of epilepsy. He also denied history of alcohol consumption. He self-medicated with Non-Steroidal Anti Inflammatory drugs and anti malarial with no response. A presumed diagnosis of bacterial meningitis was made at the peripheral health center with lumber puncture performed to confirm the diagnosis. He was initiated on intravenous Ceftriaxone and phenytoin with some improvement in the seizure frequency and fever after 3 days but further deteriorated with the worsening headache and convulsions which prompted a referral to Mulago National Referral Hospital. On examination, axillary temperature was 37.6°C, had projectile vomiting, frequent convulsions, Eye opening scored 4, Verbal and Motor reponses were 4 and 6 respectively. Glasgow Coma Scale was 14/15. He had a left hemiparetic gait, left upper and lower limb muscle powers of 4, increased reflexes, tone and an up going plantar on the same side.

The differential diagnoses included a brain abscess and a space occupying lesion probably tumour, He was admitted on the neurosurgical ward at Mulago Hospital.

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Results and Discussion

A Brain Computerised Tomography (CT) scan (Figure 1) revealed an extensive homogenous hypodense non contrast enhancing lesion in the right parietal occipital lobe with surrounding cerebral edema and significant midline shift of 8 mm displacing the midline structures to the left. There were no features of intra-cerebral hemorrhage or fractures. A working diagnosis of cerebritis was made and the patient was started on intravenous Metronidazole, Ceftriaxone, Oral phenytion and Prednisolone with minimal improvement.

The repeat brain CT scan done after one week was unchanged and the brain magnetic resonance imaging scan (Figure 2) done at the time revealed a hyper-intense mass lesion in the right parietal occipital region on T2 imaging. A differential diagnosis of a meningioma was suspected and the patient underwent surgery. The right occipital craniotomy revealed a highly vascular, grayish-whitish mass of firm consistency, attached to the internal surface of the dura mater that was completely resected. Histopathologic specimens (Figure 3) showed a number of voluminous granulomas with Scistosoma mansoni eggs. A final diagnosis of cerebral schistosomiasis mansoni pseudo tumor was made.

The Complete blood cell count revealed a significantly raised eosinophils count of 10.8%. His liver function tests revealed raised alkaline phosphatase of 135.0 U/L and Gamma glutamate transferase was 393.5 U/L about 7 times above normal. The random blood glucose, the HIV antibody test, urinalysis, stool examination, repeat HIV test and an abdominal ultra sound scan were normal.

He was initiated on Praziquantel tablets 20 mg/kg four times a day for three days, intravenous Dexamethasone 4 mg six hourly for two days and switched to tablets Carbamazepine for his seizures. The headache and seizures stopped after surgery. He was discharged 5 days after surgery on a tapering dose of prednisolone over three months and tablets Carbamazepine. His anti-convulsant medications were stopped after three months; he remained seizure free with no attendant problems.

The mechanism of injury of cerebral schistosomiasis is thought to arise from the eggs. Our patient had granulomatous lesions surrounding the eggs within the brain. It is thought that the schistosomal eggs enter the brain by embolization through venous shunts as a result of hepatic and pulmonary hypertension. It's also thought that the cerebral form is caused by aberrant migration of the worms to the vertebral venous plexus (Batson plexus). In the absence of valves, the worms migrate and produce eggs directly in the brain [8-10].

Neuroschistosomiasis is one of the most severe clinical outcomes associated with schistosome infection [11]. Cerebral schistosomiasis may manifest as an acute or subacute encephalitic-like syndrome, as a slowly growing inflammatory pseudotumor with mass effect, or as a stroke syndrome [12]. Patients with the tumoral form of Schistosoma mansoni infection are usually male and young; headache, focal neurologic deficits, and seizures are the main manifestations just like in our patient [13,14]. These lesions lead to seizures and headache due to the rising intracranial hypertension caused by the mass lesions or hydrocephalus [8,15].

However, misdiagnosis is common and confirmation of the diagnosis is always difficult. Brain neuroimaging (computed tomography and magnetic resonance imaging) usually shows a tumor

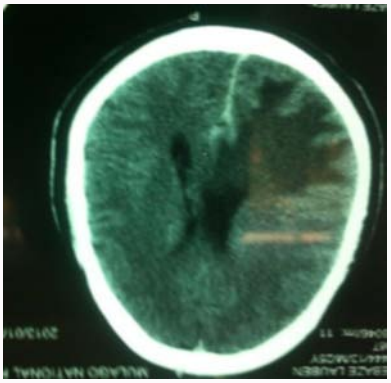

Figure 1: Non-contrasted and contrasted brain computed tomography scan showing a hypodense lesion in the white matter of the right parietal.

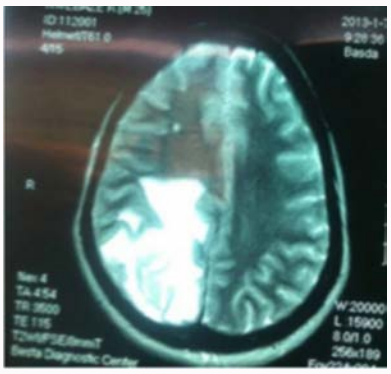

Figure 2: Is a brain magnetic resonance imaging scan showing an ill-defined moderately enhancing lesion in the right parietal and right occipital region associated with brain edema.

Figure 3: Is the histology specimen showing an intense granulomatous inflammation, numerous stromal eosinophils suggestive of a parasitic infection and scattered Schistosoma mansoni eggs.

allesion with mass effect and heterogeneous contrast enhancement mainly at the temporoparietal, occipital, and frontal lobes [2,13,16,17]. For our patient a diagnosis of meninigioma was initially made based on the radiological features [18]. However, definitive diagnosis was made by pathologic examination. Histologic analysis showed exudative necrotic granulomas surrounding the Schistosoma mansoni parasite's eggs [11,14].

Antibody detection in samples of blood or cerebrospinal fluid is useful in only a few specific circumstances [19]. Eosinophilia is not a constant finding in cerebrospinal fluid analysis. Kato-Katz thick-smear stool examination, which is the most practical laboratory examination for the investigation of cerebral schistosomiasis, can determine the presence of eggs in feces. Positive results constitute supportive evidence for the diagnosis. This diagnosis should be considered in patients that travelled to or originate from endemic schistosomiasis areas.

In patients with schistosomiasis mansoni cerebral pseudotumor, surgery is important in reducing mass effect and getting a biopsy specimen for confirmation of the diagnosis [6,7]. There are however reports where after biopsy confirmation, the cerebral Schistosoma mansoni pseudotumor has resolved with only medical treatment alone [20,21]. The medical treatment encompasses praziquantel and steroids. The steroids are administered in high doses and early in the course of treatment to prevent complications [22,23].

Conclusion

This case high lights the need for a high index of suspicion in patients that present with seizure disease and suspected cerebral tumor residing in schistosomiasis high endemic areas or travelling from endemic areas. Schistosomiasis cerebral pseudo tumor is a potentially reversible condition that responds to treatment if diagnosed early. The challenge in resource limited settings still remains availability of investigative ability.

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