

Case Report

Cerebral schistosomiasis in a Nigerian patient with end-stage kidney disease.

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Abstract

Cerebral schistosomiasis is an uncommon, probably less recognised, but serious neurological complication of *Schistosoma* infection. Its diagnosis is often delayed due to nonspecific clinical presentations and imaging findings.

We report the case of a 29-year-old Nigerian male with end-stage kidney disease on dialysis who presented with headaches, fever, generalised seizures, and left-side emiplegia. His brain MRI revealed a contrast-enhancing right frontotemporal mass lesion with surrounding oedema and midline shift, initially suggestive of a cerebral abscess.

Empirical treatment with antibiotics, steroids, and anticonvulsants provided partial relief. Surgical intervention via craniotomy and partial resection revealed a vascular, greyish-yellow mass. Histopathology confirmed *Schistosoma* eggs surrounded by granulomatous inflammation, establishing a diagnosis of cerebral schistosomiasis.

The patient was treated with praziquantel and dexamethasone, and levetiracetam, leading to complete resolution of neurological symptoms and seizure control. Follow-up imaging showed significant lesion regression.

This case underscores the diagnostic challenge of cerebral schistosomiasis, particularly in immunocompromised patients. Despite living in an endemic area, the absence of recent freshwater exposure and the absence of suspicious clinical history delayed diagnosis. Radiological features mimicked neoplastic or infectious lesions, emphasizing the need for histological confirmation.

Cerebral schistosomiasis should be considered in patients with space-occupying brain lesions in endemic regions. Early recognition and targeted therapy can yield favourable outcomes.

Keywords: Cerebral schistosomiasis, End-stage kidney disease,

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Introduction

Schistosomiasis is one of the most common parasitic diseases worldwide, estimated to affect more than 200 million people globally.[1] It is one of the neglected tropical diseases (NTD). The infection is prevalent in tropical and subtropical communities with inadequate potable water and sanitation.

Schistosomiasis is caused by a blood fluke of the genus *Schistosoma*. The three main species affecting human are the *S. Haematobium* causing urogenital schistosomiasis and *S. Mansoni* and *S. Japonicum* both causing intestinal diseases. [1,2,3]. *S. haematobium* and *S. Mansoni* are the main organisms causing diseases in Sub-Saharan Africa [2]. *Schistosoma* life cycle is complex involving an intermediate snail host and a mammalian definitive host. Individuals get infected by getting in contact with freshwater infested with the larvae (cercariae) of the parasite.

The adult organism reproduces sexually in the bladder (*haematobium*) or mesenteric vessels (*Mansoni*) and releases eggs which are carried via the blood stream to different tissues of the body where they cause inflammatory reaction. The disease can manifest as an acute or a chronic disease both of which are a result of inflammatory response to the *Schistosoma* eggs. [1,2]

Schistosoma infection affects mainly the urinary systems and gastrointestinal system. Involvement of the nervous system (neuroschistosomiasis) is the most serious manifestation of this disease, but probably under reported. Neuroschistosomiasis can affect any part of the CNS. [1,4,5] Cerebral schistosomiasis can manifest clinically as an Acute *Schistosoma* Encephalopathy (ASE) or as Pseudotumoral Encephalic Schistosomiasis (PES). It is believed that encephalopathy caused by *Schistosoma* is under-recognized in the tropics as most cases are misdiagnosed.[4] *Schistosoma Japonicum* is responsible for most reported cases of cerebral schistosomiasis with PES reported in 3.5% of individual infected with *S. japonicum*. [4,6] cases of cerebral schistosomiasis caused by *S. haematobium* and *S. mansoni* are also reported [7,8,9,10,11]. An autopsy study from Africa shows that half of the patients with urinary schistosomiasis had brain lesions.[12]

Nigeria has the highest prevalence of schistosomiasis in Africa with about 20 million people infected, yet only one case of cerebral schistosomiasis which was caused by *S. haematobium* was found in English literature.[7] This indicates possible under diagnosis of the condition in our environment.

We report a case of a 29-year-old man on treatment for chronic kidney disease with cerebral schistosomiasis who was managed with craniotomy and partial resection of lesion and medications with good outcome.

Case report

The patient was a 29-year-old man with five-day history of recurrent generalised tonic clonic seizures with progressive left sided hemiparesis. There was associated history of headache and intermittent high-grade fever. Four months prior to onset of seizures, he was diagnosed of chronic kidney disease (CKD) secondary to chronic glomerulonephritis based on markedly elevated serum creatinine of 1090 $\mu\text{mol/l}$ and eGFR of 5 mL/min/1.73m^2 (Table 1) and was on twice-weekly haemodialysis. He was being prepared for kidney transplantation. There was no history of recent contact with freshwater, nor previous history of terminal haematuria. Clinical examination revealed a young man who was febrile (T- 37.8) and confused with Glasgow coma scale score of 14. He has left hemiplegia with muscle power of 0/5 on Medical Council Scale (MCS). Other systemic examination was essentially normal. Laboratory tests revealed lymphocytic leucocytosis. (Table 2)

Table 1: Results of kidney function test

Test	Result Pre-dialysis	Result Post-dialysis	Reference	Unit
Sodium	139	136	135-145	mmol/L
Chloride	98	93	95-110	mmol/L
Potassium	4.5	3.8	3.5-5.5	mmol/L
Bicarbonate	27	25	21-30	mmol/L
Urea	26	14	2.5-6.5	mmol/L
Creatinine	1090	560	53-115	umol/L
eGFR	5	11	>90	mL/min/1.73m ²

Table 2: Full blood count result

Test	Result	Reference	Unit
PCV	33	38-52	%
Haemoglobin	10.9	13.5-17.5	g/dl
WBC count	13.1	4-10	10 ⁹ /L
Neutrophil	30	40-75	%
Lymphocytes	65	20-45	%
Mid	5	3-15	%
Neutrophil count	6.5	2-7	10 ⁹ /L
Lymphocyte count	6.1	0.8-4	10 ⁹ /L
RBC	3.8	4.5-6.5	10 ¹² /L
MCV	80	80-100	fL
MCH	30	27-34	Pg
MCHC	32	32-36	g/dL
Platelets	330	150-400	10 ⁹ /L

Brain MRI showed an irregular ring heterogeneously enhancing right frontotemporal mass with surrounding peri-lesional edema and midline shift (Figure 1).

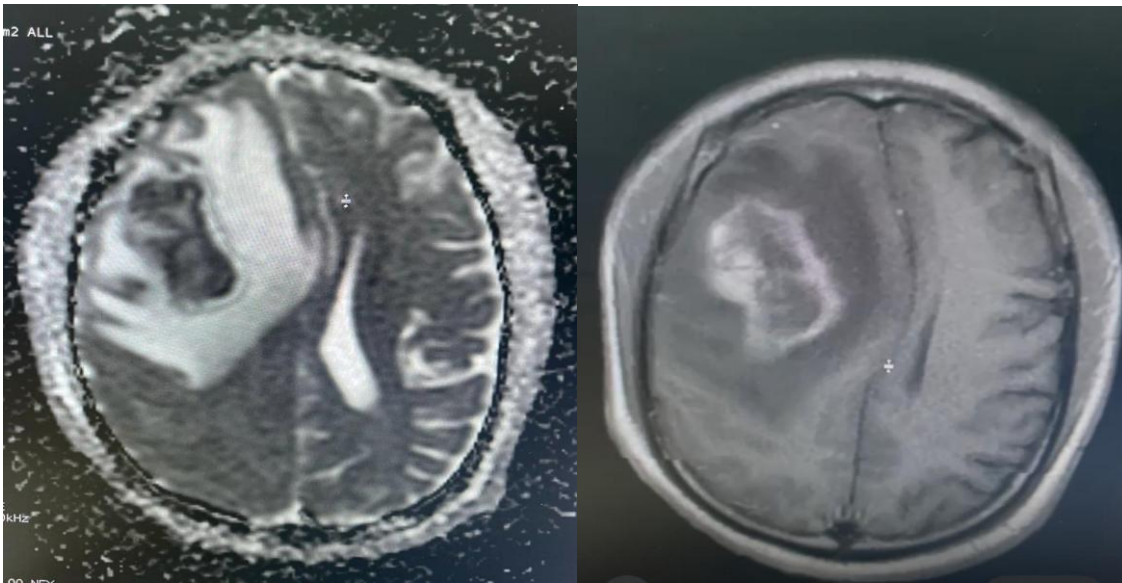


Figure 1: Brain MRI showing T2 and T1 contrast.

A diagnosis of right fronto-temporal cerebral abscess was made, and an empirical broad-spectrum parenteral antibiotic (Ceftriaxone and vancomycin), steroid (dexamethasone) and anticonvulsant (levetiracetam) were started. The Patient made some clinical improvement in the hemiplegia and seizure control. However, because the lesion was large with significant mass effect and the need for tissue diagnosis, we opted for surgical intervention. We did a craniotomy and partial resection of the lesion. Intraoperatively we found a highly vascular, grayish yellow lesion with surrounding gliotic brain tissue.

Histopathology revealed presence of Schistosomaeggs with surrounding granuloma (figure2).

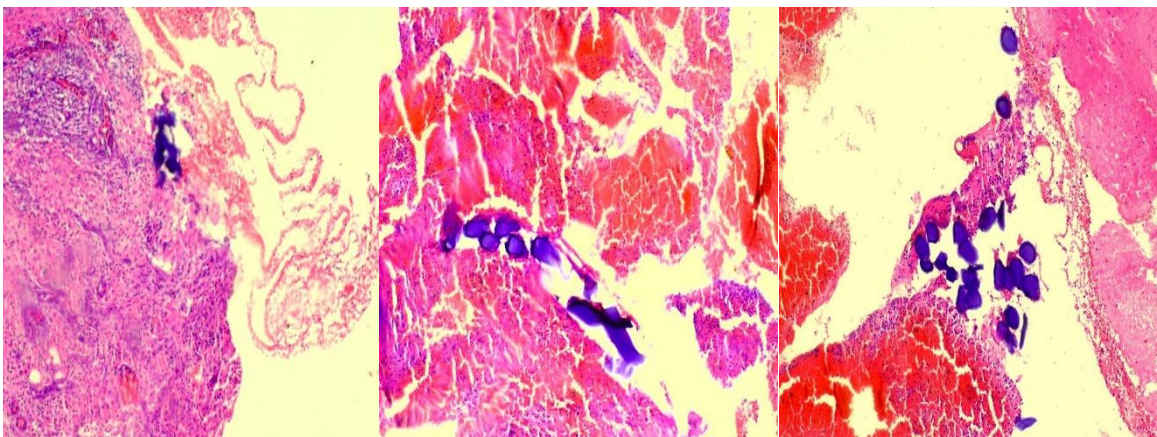


Figure 2: Pathological examination

He was treated with Praziquantel 40mg daily for 3 days and dexamethasone. There was complete resolution of the headache and limbs weakness. The patient has been seizure free for more than a year. A repeat brain MRI showed significant reduction in the size of the brain lesion.(figure 3)

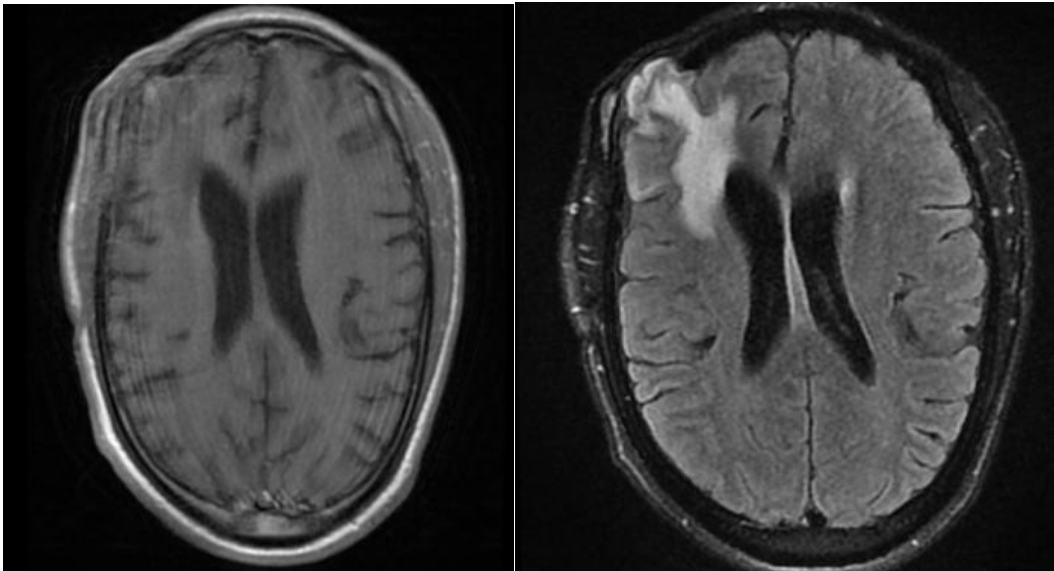


Figure 3: Post operative MRI T2 and T1 contrast.

Discussion

Our initial diagnosis in this patient was that of a possible pyogenic abscess based on the presentation with headache, seizures and hemiparesis and MRI finding of a tumor like lesion in the brain. We commenced treatment with use of broad-spectrum antibiotics and steroids while getting the patient ready for surgery. Even though our patient resides in an endemic area, we did not consider cerebral schistosomiasis as a differential diagnosis. The laboratory finding of lymphocytic leucocytosis was indicative of a possible parasitic infection. Many of the reported cases of cerebral schistosomiasis were in young healthy adults who resides or recently travelled to an endemic area, in many, a history of contact with freshwater as possible source of infection could be established. [8,10,11,13] This was not the case in our patient. Apart from been a resident of an endemic country we could not establish any history of possible recent contact with schistosomiasis infection. Most cases like our patient present with symptoms and sign of raised intracranial pressure and focal deficits depending on the location of the lesion in the brain. Headache as in our patient is the most frequent symptoms. Fever is usually not a prominent feature at presentation. [4,5] Our patient presentation with chronic kidney disease from glomerulonephritis and cerebral schistosomiasis represents an unusual presentation. Schistosomiasis related to kidney disease is not commonly reported in the literature. The incidence of glomerular involvement is estimated as 5% to 6% in clinical settings while glomerulonephritis is described in 10 -12 % of autopsy studies. [14]

Brain MRI in our patients was similar to previous reports of nonspecific mass lesion with heterogeneous enhancement with surrounding oedema. [4,5] Our patients had a single lesion in the brain; single and multiple Schistosoma lesions have been previously reported. [4,5,15,16] Other imaging modalities like CT scan and PET scan have also been used in investigating patients with suspected neuroschistosomiasis. [1,5,15,16] While presence of Schistosoma ova in the stool or urine through microscopic examination aid in establishing diagnosis in suspected cases of neuroschistosomiasis, these tests are positive in only 40 – 50 of cases. Serological tests have high sensitivity in making diagnosis but its fault with high false positive rates. A positive blood and/or cerebrospinal fluid serological result in patient with suspicious history and brain imaging lesions could prompt a therapeutic trial with medications. [13] Where these tests are negative or not done because managing physician did not consider cerebral schistosomiasis in the differential diagnosis. Surgery is also beneficial in patients with clinical manifestations of raised intracranial pressure and focal deficits because of mass effect of the tumor like lesion as in our index patient. Total or partial resection though invasive is safe and results in immediate clinical improvement as

demonstrated in our patients and other reported cases. [8,9,10]. In situation where diagnosis of schistosomiasis infection is established through microscopic and serological tests on stools, urine or CSF, medical treatment can proceed without a need for tissue biopsy and this has been reported to result in good clinical recovery. [5,13]

Following histological diagnosis, we treated our patient with Praziquantel 1.2g three times daily for 2 weeks and continued dexamethasone and levetiracetam. Praziquantel remains the most effective medication for all *Schistosoma* species. Steroid helps to reduce perilesional vasogenic cerebral edema thereby contributing to clinical improvement. There is no guideline as to the duration of steroid and anticonvulsant therapy in neuroschistosomiasis. Our patient received tapering doses of steroids for two weeks and anticonvulsants (Keppra) for six months.

While the species of *Schistosoma* organism responsible for our patient infection was not determined, we inferred that it is likely to be caused by *S. haematobium* because of the endemicity of this species in Nigeria and the fact that the only reported case from Nigeria was by *S. haematobium*. [7] Although *S. japonicum* is responsible for most cases of cerebral Schistosomiasis, brain infestation by other species has also been reported in literature. [5,8,9,11]

The clinical course of our patient was like previous case reports. There was complete resolution of headache and focal deficits and the patient remained seizures free as at last follow up.

Conclusion

Cerebral schistosomiasis, though rare, should be considered in patients presenting with space-occupying brain lesions, particularly in endemic regions. In this case, the diagnosis was obscured by the patient's underlying immunocompromised state due to CKD. Imaging findings mimicked an abscess or neoplasm, necessitating histologic confirmation. Prompt antiparasitic therapy led to radiological and clinical improvement.

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