CASE REPORT

Case report of Neuroschistosomiasis in a Child

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ABSTRACT *:* Neuroschistosomiasis is a serious complication of schistosomiasis, where Schistosoma parasites migrate to the central nervous system. It is often overlooked but can cause significant neurological symptoms. We present a 10-year-old male with headache and papilledema, emphasizing the importance of considering neuroschistosomiasis in patients with neurological symptoms and a history of schistosomiasis exposure. Early diagnosis and timely treatment with antischistosomal drugs and corticosteroids are crucial for positive outcomes. Raising awareness and implementing appropriate management approaches can improve the prognosis of neuroschistosomiasis.

CASE REPORT

This case involves a 10 y/o male from Tacloban, Leyte who presented with headache. He was apparently well until 11 months prior to admission, when the patient experienced intermittent rashes characterized by erythematous maculopapular lesions with raised borders, pruritus, and burning sensation on the extremities. He consulted an allergologist and was diagnosed with Acute Urticaria. After 1 month of antihistamine treatment without resolution of symptoms, the possibility of Cercarial Dermatitis was considered due to the presence of recurrent rashes and the high prevalence of schistosomiasis in the area. However, the family did not proceed with the recommended stool examination.

Nine months prior to admission, due to the persistence of symptoms a stool examination was finally conducted, leading to a diagnosis of Intestinal Schistosomiasis in the patient. The patient was given two courses of praziquantel computed at 42mg/kg/day, divided into two doses administered four hours apart. The first course was given immediately upon the initial diagnosis, and the treatment was repeated one month later.

The patient remained asymptomatic until 25 days prior to admission, when he complained of non-radiating dull periumbilical abdominal pain with a severity of 5/10, along with two episodes of non-projectile vomiting of previously ingested food.

No other associated symptoms were reported. A teleconsultation was conducted with a private pediatrician who managed the patient's condition as a case of Acute Gastroenteritis and prescribed probiotics. However, two days later, the abdominal pain and vomiting persisted, and the patient developed an associated bilateral frontotemporal headache with a severity of 5/10. The headache was described as non-radiating and squeezing in character, and there were no associated symptoms of dizziness or blurring of vision. Another teleconsultation took place, during which a complete blood count and urinalysis were requested and yielded normal results. He was then prescribed Omeprazole to be taken for two weeks, which provided temporary relief of the abdominal pain.

Twenty-one days prior to admission, the patient experienced a severe frontotemporal headache upon waking up, with a pain scale of 10/10. The headache was accompanied by transient slurring of speech lasting for about 5 minutes and one episode of vomiting. There were no associated seizures or focal weakness. The patient was taken to a nearby hospital, where the symptoms resolved during the stay in the emergency room. He was then advised to consult an ophthalmologist. Upon examination bilateral papilledema was observed during fundoscopy. As a result, patient was admitted to a hospital in Leyte for four days. The impression was Neuroschistosomiasis, and he received three doses of praziquantel, given four hours apart, at a calculated dosage of 45mg/kg/day. A Circumoval Precipitin Test (COPT) was performed during this admission, but the results were expected in two weeks. The patient was discharged in a well condition and advised to have child neurology consult in our institution which was scheduled approximately three weeks later.

Eight days prior to the current admission, the patient had no subjective complaints, but the mother decided to seek a second opinion from another Ophthalmologist regarding the previously noted papilledema. There was a concern about the possibility of leukemic infiltrates, and a complete work-up was recommended. After this consultation, the COPT result came in and was positive.

Three days prior to the current admission, the patient complained once again of a frontotemporal headache with the same character as before, but with a pain scale of 3/10. The patient also experienced two episodes of non-projectile vomiting. As a result, the patient was advised to be transferred to our institution for further evaluation and management.

The patient has a history of living in a flood-prone area during his early childhood. They lived in a rented bungalow house near a river that frequently floods during heavy rains and typhoons. The patient's mother mentioned that they occasionally had to wade through the floodwaters.

To avoid the frequent flooding, they eventually moved to a different house situated in a higher location. Additionally, the patient's school is also frequently affected by floods, indicating that the local area has a recurring issue with flooding. However, the last exposure to floodwater occurred before the COVID-19 pandemic.

The patient's vital signs are stable, and the physical examination does not reveal any significant abnormalities. Neurologically, the patient has intact mental status and higher cortical functions. The cranial nerve examination reveals bilateral papilledema, but the rest of the cranial nerve examination was normal. The motor examination does not show any notable abnormalities. There are no sensory deficits observed across all modalities, indicating that the patient's ability to perceive touch, pain, temperature, and other sensory stimuli is intact. The patient exhibits hyperreflexia in the bilateral lower extremities. The examination of cerebellar function and meningeal signs, as well as the autonomics, does not reveal any noteworthy abnormalities.

Based on the clinical presentation, the initial impression was Increased Intracranial Pressure secondary to Cerebral Neuroschisto-somiasis. Laboratory tests revealed an elevated erythrocyte sedimentation rate, which can be indicative of inflammation. Blood culture did not yield any growth, ruling out an acute bacterial infection. Other laboratory tests, including complete blood count, inflammatory markers, serum electrolytes, kidney and liver function tests, urinalysis, and stool examination, did not reveal any significant abnormalities. A chest X-ray was normal, suggesting no apparent lung involvement.

Ophthalmology consultation confirmed the presence of chronic papilledema in both eyes, further supporting the diagnosis. Cranial MRI was performed and revealed subcortical and sulcal lesions in the parietal lobes of both cerebral hemispheres, as well as vasogenic edema in the right frontal and bilateral parietal lobes (Figure). These findings were interpreted as cerebral tuberculomas. He was then initiated on anti-TB medication. TB work-up, including a purified protein derivative (PPD) test and sputum gene x-pert, yielded normal results.

A neuroradiologist reviewed the images and confirmed compatibility with neuroschistosomiasis. To further evaluate the possibility of seizures associated with neuroschistosomiasis, a 6-hour video EEG was performed, which was normal. A craniospinal MRI with contrast was also conducted, showing no remarkable findings in the spinal region.

Methylprednisolone was started at 15 mg/kg/day to manage the inflammation and reduce intracranial pressure, along with praziquantel at 50 mg/kg/day to target the schistosomiasis infection.

Subsequently, methylprednisolone was shifted to oral prednisone at 1 mg/kg/day after seven doses and tapered over the course of a month. Praziquantel was completed for five days, while anti-TB medication (HRZE) was continued and planned for a year.

The patient was also referred to the Hematology service to rule out the possibility of leukemic infiltrates. Lactate dehydrogenase and reticulocyte count were normal, and bone marrow aspiration revealed a normocellular marrow with complete trilineage hematopoiesis, effectively ruling out leukemia.

With no subjective complaints, stable vital signs, and no new onset neurological deficits, he was discharged on the 14th hospital day.

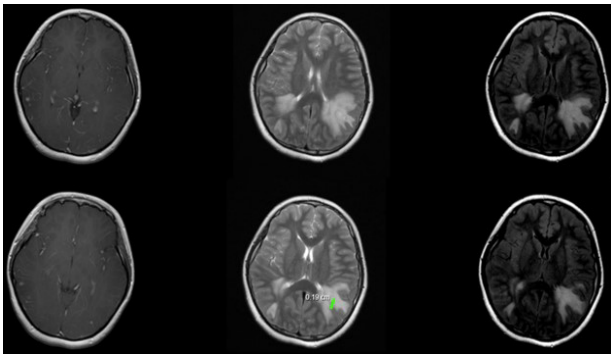

Figure: *T1-weighted hypointense and T2-weighted hyperintense subcortical and sulcal lesions on the parietal lobe of both cerebral hemispheres. There was also significant vasogenic edema in the right fontal and bilateral parietal lobes.*

Discussion

Schistosomiasis is a parasitic disease caused by blood flukes of the genus Schistosoma. The granulomatous reaction is the main mechanism of disease in schistosomiasis. The eggs of the Schistosoma parasite that are not excreted can become trapped in the intestinal or bladder wall or can be carried by the bloodstream to the liver and other sites, including the central nervous system (CNS).

Clinical forms of schistosomiasis include acute and chronic infections. Acute infections can manifest as cercarial dermatitis (swimmer's itch) or as Katayama syndrome (acute schistosomiasis syndrome). Chronic infections can result in intestinal schistosomiasis, hepatosplenic schistosomiasis, genitourinary schistosomiasis, or neuroschistosomiasis (schistosomal involvement of the CNS).[1,2]

Neuroschistosomiasis is a severe complication of schistosomiasis, characterized by the involvement of the central nervous system (CNS). It is an underrecognized complication and affects a small percentage of individuals with systemic schistosomal infections. There are two main types of neuroschistosomiasis depending on the species of Schistosoma involved. Spinal cord schistosomiasis is predominantly associated with Schistosoma mansoni and Schistosoma haematobium infections, while brain involvement, such as acute schistosomal encephalopathy and

and pseudotumoral encephalic schistosomiasis, is typically associated with Schistosoma japonicum infections.

Spinal cord schistosomiasis commonly presents with lower back pain that radiates to the lower limbs, lower limb weakness, bladder dysfunction, lower limb paraesthesia, hypoaesthesia or anaesthesia, deep tendon reflex abnormalities, constipation, and sexual impotence. The conus medullaris and cauda equina are often affected. The severity and distribution of symptoms can vary, and the medullary level identified by clinical examination is typically equal to or below T6, particularly at T11–L1. It is classified into three clinical forms: medullary, conus-cauda equina syndrome, and myeloradicular.

In cases of brain involvement, acute schistosomal encephalopathy (ASE) may occur. ASE presents with symptoms such as headache, altered mental status, seizures, sensory disturbances, weakness of extremities, ataxia, and transient visual and speech disturbances. Meningeal signs are less common in ASE.

Pseudotumoral encephalic schistoso-miasis, which predominantly occurs in individuals without other symptoms of schistosomiasis, is characterized by slow-expanding lesion-like masses in the brain. These masses cause increased intracranial pressure and can lead to severe and persistent headaches, focal neurological signs depending on the lesion site, and various types of seizures. The cerebellum is the most commonly affected site, followed by the occipital and frontal lobes.[3]

For the diagnosis of active schistosomiasis, the gold standard is the identification of schistosome eggs in stool or urine samples using microscopy. However, microscopy has limitations, particularly in cases of light infections or acute infections. The Kato-Katz method is commonly used for stool examination in endemic settings, but it may lack sensitivity in light infections. Serologic tests, such as the Circumoval Precipitin Test, can be useful in the absence of egg detection, especially for travelers with low parasite burden. Biopsy, particularly superficial rectal biopsies or "rectal snips," can be more sensitive than stool microscopy in certain cases.[4]

Regarding neuroschistosomiasis, MRI is a sensitive imaging modality for detecting abnormalities. In spinal cord schistosomiasis (SCS), the most common findings include signal hyperintensity on T2-weighted images, spinal cord enlargement (particularly lower cord and conus medullaris), thickening of spinal roots (especially cauda equina roots), and heterogeneous contrast enhancement on T1-weighted images. In acute schistosomal encephalopathy (ASE), edema and multifocal small contrast-enhanced lesions can be observed in the frontal, parietal, occipital lobes, and brainstem on MRI. Pseudotumoral encephalic schistosomiasis (PES) typically presents as a non-specific tumor-like lesion

surrounded by edema, with mass effect and heterogeneous contrast enhancement on MRI.

CSF examination in neuroschistosomiasis can show slight-to-moderate increases in total protein concentration and lymphocyte count. Eosinophils may be present in the CSF in approximately 50% of patients with spinal cord schistosomiasis. In ASE and PES, CSF findings can be normal or non-specific.

Treatment approaches for neuroschistosomiasis are not yet standardized. However, antischistosomal drugs, corticosteroids, and surgery are the available modalities. Prompt corticosteroid treatment, such as prednisone at a dose of 1 to 2 mg/kg per day, is essential to limit tissue damage caused by the intense inflammatory response to embolized eggs. Even if the diagnosis of neuroschistosomiasis is suspected but not confirmed, corticosteroid therapy should be initiated. The duration of corticosteroid therapy is uncertain, but it is generally continued for several months and then gradually tapered based on individual circumstances. Premature discontinuation or rapid tapering of steroids may result in clinical relapse.[3]

Control strategies for schistosomiasis in endemic areas include several approaches. Periodic mass treatment with antischistosomal drugs, particularly praziquantel, is a key intervention to reduce the parasite burden and prevent the development of severe disease. Water sanitation programs aim to minimize exposure to contaminated freshwater sources, which are the habitat of the intermediate snail hosts. Vaccine development is also an important area of research to provide long-term protection against schistosomiasis.

Interventions such as community education play a crucial role in raising awareness about preventive measures, including the use of protective clothing and footwear to reduce contact with contaminated water. Eradication of snail species, which act as intermediate hosts for the parasites, can also be a part of control efforts to disrupt the life cycle of the parasites.[4]

The prognosis of neuroschistosomiasis largely depends on early diagnosis and treatment initiation. With the advent of praziquantel and the addition of corticosteroids, the rates of recovery have significantly improved. In cases treated early with praziquantel and steroids, neurological symptoms have shown complete resolution in most patients. Before praziquantel became widely available, the rates of recovery were lower, with only 13% achieving full recovery, 74% experiencing partial recovery, and 13% having a poor recovery.[3]

Summary

Neuroschistosomiasis is a severe and underrecognized complication of schistosomiasis, a parasitic disease caused by blood flukes of the genus Schistosoma. It is characterized by the involvement of the central nervous system, including the spinal cord and brain.

This abstract summarizes the case of a patient with neuroschistosomiasis presenting with increased intracranial pressure and exhibiting symptoms such as bilateral papilledema, hyperreflexia, and subcortical lesions on imaging.

The patient, who had a history of living in a flood-prone area, underwent various diagnostic tests, including laboratory investigations, imaging studies, and cerebrospinal fluid examination. The diagnosis of neuroschistosomiasis was confirmed based on the presence of schistosome eggs in the stool and the characteristic findings on imaging, which showed T1-weighted hypointense and T2-weighted hyperintense lesions in the parietal lobes and significant vasogenic edema. Treatment was initiated with anti-kochs medication, including methylprednisolone and praziquantel. The patient showed improvement in symptoms and stable vital signs during the hospital stay. Follow-up evaluations, including a bone marrow aspiration, were performed to rule out other potential conditions.

Our report highlights the importance of early diagnosis and treatment in neuroschistosomiasis, as prompt administration of corticosteroids and antischistosomal drugs can lead to favorable outcomes and complete resolution of neurological symptoms. Control strategies for schistosomiasis in endemic areas, such as mass treatment, water sanitation programs, and community education, are crucial in preventing and managing this debilitating condition.

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