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Case Report

Neuroschistosomiasis presenting as recurrent seizures: A case report

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ABSTRACT

Background: Cerebral pseudotumoral schistosomiasis is an uncommon and underreported condition, posing significant diagnostic challenges due to its ability to mimic other neurological conditions, especially in patients presenting with persistent seizures and imaging findings indicative of an infectious etiology.

Case Description: We report the case of a 16-year-old male who presented with persistent headaches and recurrent seizures despite adherence to antiseizure medications. Neuroimaging findings suggested an infectious process but were inconclusive in differentiating between a tuberculoma and cerebral schistosomiasis. Given the differing therapeutic approaches required for these conditions, a definitive diagnosis was pursued through a brain tissue biopsy, which confirmed cerebral schistosomiasis. This diagnosis guided appropriate treatment, leading to clinical improvement.

Conclusion: This case highlights the critical role of biopsy in establishing a definitive diagnosis when imaging results are inconclusive and suggests the importance of exploring the use of adjunct diagnostic methods like magnetic resonance spectroscopy, hence decreasing or potentially eliminating the need for an open biopsy.

Keywords: Biopsy, Cerebral schistosomiasis, Pseudotumoral, Seizure

INTRODUCTION

Human schistosomiasis is a parasitic infection caused by hematogenous invasion of trematode flukes of the genus *Schistosoma*, found in contaminated freshwater bodies harboring infected intermediate-host snails.[24] Several species of *Schistosoma* infect humans, such as *Schistosoma mansoni*, *Schistosoma japonicum*, *Schistosoma mekongi*, and *Schistosoma intercalatum,* which cause intestinal disease and *Schistosoma haematobium* causing urogenital disease.[24] More than 230 million individuals are afflicted with schistosomiasis worldwide, and almost 800 million are at risk of developing the illness in the endemic regions.[26] Due to its high morbidity, untreated illness has considerable long-term economic consequences, resulting in an estimated 3.3 million disability-adjusted life years as of 2019, as mentioned in a study done by Murray *et al.*, which comprised data from over 21 regions, including Southeast Asia.[25] Neuroschistosomiasis, referring to schistosome involvement of the central nervous system (CNS), occurs less frequently compared to its gastrointestinal and genitourinary manifestations and accounts for <2.3% of reported cases.[4] The CNS pathology early during infection is thought to occur through the *in situ* egg deposition following aberrant migration of adult worms to the brain or spinal cord.[32]

The presence of the ova in the CNS induces a periovular granulomatous reaction causing mass effect attributed to the large granulomas concentrated within the brain or spinal cord, thus explaining the signs and symptoms of increased intracranial pressure, myelopathy, radiculopathy, and subsequent clinical sequelae. Cerebral complications include encephalopathy with headache, visual impairment, delirium, seizures, motor deficits, and ataxia.[32]

We report a case of cerebral schistosomiasis presenting as a new-onset seizure in an adolescent with a multifocal brain lesion. His clinical and radiological findings resembled that of a brain neoplasm. On further workup, a magnetic resonance spectroscopy (MRS) was done to assess the biochemical composition of the brain lesion, which revealed an infectious etiology suggestive of either tuberculosis (TB) or schistosomiasis. The biopsy demonstrated the presence of *Schistosoma* ova with chronic granulomatous inflammation, thus establishing the diagnosis of schistosomiasis and appropriate treatment with praziquantel single dose was administered.

PATIENT INFORMATION

This is a case of a 16-year-old male from Davao del Sur, Philippines, who presented at the clinic with a 6-month history of recurrent seizures. The initial symptoms included severe generalized headaches, occasional dizziness, and nausea. The patient's condition was later complicated with generalized onset tonic-clonic seizure, accompanied by postictal loss of consciousness and vomiting. Despite repeated seizures, financial constraints hindered timely access to medical care. The patient eventually consulted an adult neurologist who managed the seizures with levetiracetam 500 mg/tablet, one tablet 3 times a day. After a brief period of relief, the seizures recurred, prompting them to see a pediatric neurologist, wherein cranial magnetic resonance imaging (MRI) and electroencephalogram (EEG) were done. The EEG was unremarkable, but the cranial MRI scan revealed a contrast-enhancing irregularly shaped foci aggregately measuring 2.92 × 2.32 × 1.32 cm clustered in the right insula and temporal operculum [Figures 1a and b].

Since the cranial MRI revealed a markedly enhancing lesion in the right cerebrum causing surrounding vasogenic edema and mass effect, a cranial MRS was done to assess the biochemical composition of the lesion further. The lesion was noted to have increased choline: creatine ratio and decreased n-acetylaspartate: choline and n-acetylaspartate: creatine ratios. The results were suggestive of an infectious etiology, with primary considerations being TB or schistosomiasis, both of which were considered due to endemicity in the area.

To investigate for schistosomal infection, a Kato-Katz thick stool smear test to check for *Schistosoma* ova and a whole abdomen ultrasound for hepatic involvement was done. Kato-Katz

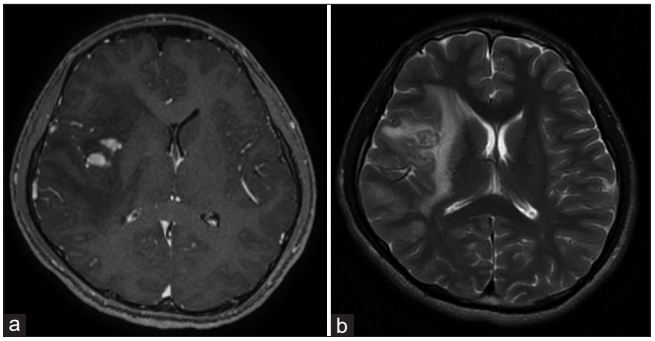

Figure 1: (a) A 2.92 × 2.32 × 1.32 cm aggregate of contrast-enhancing irregularly shaped foci are seen in the T1-weighted contrast magnetic resonance imaging (MRI). The lesion is primarily focused on the right temporal operculum and right insula. No restricted diffusion was noted (not shown). (b) Significant hyperintense vasogenic edema can be observed on T2-weighted MRI surrounding the previously mentioned foci.

revealed no *Schistosoma* ova which would usually be apparent in active infection.[42] Aside from an incidental finding of a 0.4 cm gallbladder polyp, the whole abdomen ultrasound showed no signs of periportal fibrosis, which would have indicated hepatic involvement of the fluke.[2,5] A chest radiograph was done to investigate signs of TB, such as hilar lymphadenopathy or pleural effusion; [6] however such findings of TB were absent in our case. The patient's growth, development, and medical history were reviewed, revealing normal development, complete immunization, and a history of pneumonia requiring hospital admission in 2009, wherein he was discharged well. He has no known allergies to food or medications. There is no significant family history of seizure disorders. His environmental history included frequent visits to a farm in Bukidnon, raising concerns about environmental exposures. A comprehensive psychosocial assessment revealed that the patient is part of a blended family and is in a same-sex relationship, with no history of drug use, smoking, or suicidal ideation.

The patient did not report any fever, weight loss, night sweats, dizziness, visual disturbances, respiratory and gastrointestinal issues, or neurological changes.

After consideration, a thorough anamnesis was done, which revealed that the patient's latest exposure to rice fields was 18 months before the presentation of seizures.

To resolve the issue of diagnosis, an open biopsy was scheduled.

Clinical presentation

On examination, the patient was alert, cooperative, and not in respiratory distress, with normal vital signs. The patient had a normal body mass index for age, warm skin with good turgor, and no skin abnormalities. The head was normocephalic, with anicteric sclerae, pink palpebral conjunctivae, and pupils equally reactive to light and accommodation. The

chest, cardiovascular, and abdominal examinations were unremarkable. Neurological examination showed no changes in gait, normal mental status, cranial nerve function, no sensory or motor deficits, and intact reflexes, with no signs of meningism or cerebellar dysfunction. No papilledema was seen on the ocular fundoscopy. Laboratory results for complete blood count and liver enzymes were unremarkable [Table 1].

Therapeutic intervention

The patient underwent a right frontotemporal craniotomy and debulking of the mass lesion with a frozen section biopsy. The surgery was uneventful. The histopathologic findings reported scattered perivascular aggregates of ova and surrounding gliosis, morphologically consistent with *Schistosoma*. The staining pattern and morphology were characteristic of *S. japonicum* species because of the noted lateral knob [Figures 2a and b]. The patient was started on Praziquantel 600 mg/tablet, and 6 tablets were given as a single dose with a 56 ml dose. The patient had no adverse effects or allergic reactions to the medication.

Postoperatively, the patient was noted with no recurrence of seizures and with good wound healing. The patient was maintained on levetiracetam 500 mg 1 tablet twice a day for seizure prophylaxis. On the 3rd postoperative day, the patient was discharged well.

Postoperative course

Upon discharge, dexamethasone was prescribed for a total of 4 days with a tapering dose before discontinuation. This was given to prevent tissue destruction by diminishing granulomatous inflammation. There is evidence suggesting that they also decrease ova deposition.[16] Levetiracetam was continued for 3 weeks for seizure prophylaxis.

Follow-up and outcomes

After 3 weeks, the patient has his first follow-up at the clinic. There was noted good healing at the postoperative site and no recurrence of seizures; hence, levetiracetam was discontinued. At follow-up 6 months later, he was seizure-free and was experiencing no sequelae of his previous infection or its treatment. The patient has returned to school and is performing all pre-disease activities.

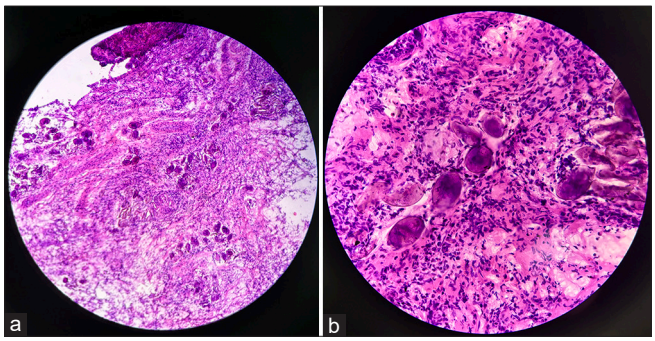

Figure 2: Biopsy demonstrating scattered perivascular aggregates of schistosoma eggs with surrounding gliosis. (a) Low-power field. (b) High-power field. Stain used: Hematoxylin and eosin.

DISCUSSION

Yamagiwa, a student of Rudolf Virchow in 1889, published the first case of cerebral schistosomiasis in literature.[43] *S. japonicum*, the endemic species of *Schistosoma* in the Philippines, initially gained international attention during World War II, when American forces congregated on Leyte Island in the Philippines as part of the Pacific war.[8] Troops wading through freshwater wetlands and streams, where Oncomelania snails, the intermediate host, were abundant, developed the disease through cercarial penetration released by the snail in the water. The cercariae penetrate human skin, entering the bloodstream, where they mature into adult larvae.[12] These adult *Schistosoma* larvae, especially *S. japonicum and S. mansoni*, latch onto the inner walls of the hepatic portal and mesenteric veins using suction cups located in their mouth and abdomen, allowing them to resist being swept away by the rapid blood flow.[37] Schistosomes are dioecious, with the sexual maturation and egg-laying of females entirely regulated by males. The pairing with males stimulates ova production by the females, which is the infective stage.[10] Many eggs are trapped in tissues, causing

Table 1: Laboratory test results

Variables	Result	Reference Range
Aspartate transaminase (AST) (U/L)	54.0	<37.00
Alanine transaminase (ALT) (U/L)	46.0	<45.00
Total bilirubin (µmol/L)	17.50	3.0-22
Direct bilirubin (µmol/L)	4.0	<5.0
Indirect bilirubin (µmol/L)	13.5	<19.0
Creatinine (µmol/L)	77.8	71-115
Sodium (mmol/L)	140.0	135.0-145.0
Potassium (mmol/L)	3.75	3.50-5.10
Hemoglobin (g/dL)	16	13.5-17.5
Hematocrit (%)	48	33-44
Leukocytes (x10^9/L)	8.8	3.8-9.8
Neutrophil (%)	48	50-70
Lymphocytes (%)	35	20-60
Eosinophils (%)	10	1-6
Platelet (x10^9/L)	310	150-400
Prothrombin time (seconds)	10.4	10.7-13.7
Partial Thromboplastin time (seconds)	29.4	24.1-30.7
International normalized ratio (INR)	0.88	2.0-3.0

an immune host response that leads to granuloma formation and tissue damage. The early inflammatory Th1-type immune response results in cytokine release, which can escalate into tissue fibrosis and organ damage, particularly in the liver and intestines.[22] Acute schistosomiasis manifests with symptoms such as fever, cough, abdominal pain, and hepatosplenomegaly.[18]

Out of the 1200 soldiers with acute *Schistosoma* infection during World War II, 2.3% presented with cerebral complications such as seizures and headaches.[31] Such cerebral manifestations may indicate neuroschistosomiasis. It can cause infection in any region of the CNS and can be classified into three different subtypes: acute schistosomal encephalopathy, spinal schistosomiasis, and pseudotumoral cerebral schistosomiasis.[15] The latter form is caused most often by *S. japonicum* infection in East Asia, as expected in our case. The pathogenesis is not fully understood, but evidence suggests that in most symptomatic cases, the damage is caused by the host's immune response to Schistosoma ova in nervous tissue. Ova can reach the CNS through two routes: first, the arterial system, through pulmonary shunts, or portopulmonary anastomoses due to portal hypertension. Second, through the retrograde venous flow through Batson's venous plexus, which links the portal venous system to the spinal cord and cerebral veins.[30] Multiple ova may also be laid near the CNS following aberrant migration of adult worms to the brain or spinal cord. These eggs, surrounded by granulomas, damage nearby nervous tissue and create a mass effect[15,30], as seen in our case.

In a study done by Ferrari and Moreira (2011), they hypothesized that predilection of *S. japonicum* for the CNS is a direct consequence of their small-sized ova and the absence of a well-developed spine facilitates their migration to the brain[15] as revealed in the histopathology of our patient. This hypothesis is strengthened by Scrimgeour and Gajdusek (1985), wherein they discussed that the larger, spine-protruding eggs of *S. mansoni* and *S. haematobium* typically remain in the lower spinal cord, although, in rare cases, eggs from *S. mansoni* and *S. haematobium* might reach the brain.[33]

A prolonged asymptomatic period of 18 months in our patient was a pertinent finding as a similar case report done by Suthiphosuwan *et al*. in 2018 on pediatric cerebral schistosomiasis, the patient had a prolonged asymptomatic period of 4 years compared to our patient, but such delays have been reported in literature, and it might have reflected several factors including egg load, intensity of inflammatory reaction surrounding the eggs and/or worms, and the location of the lesion.[35] The asymptomatic form of this disease is associated more frequently with hepatosplenic and cardiopulmonary disease[15], both of which were screened for and found to be absent in our patient.

The diagnosis of cerebral schistosomiasis was challenging in our case because the patient lived in an urban area and was a student. Schistosomiasis predominantly affects rural populations engaged in agricultural activities such as farming and fishing, where they are exposed to contaminated freshwater sources. Studies consistently demonstrate that middle-aged individuals and farmers in endemic areas are more susceptible to the disease due to their reliance on natural water bodies for their livelihoods. A study done by Klohe *et al*., 2021 highlighted that rural populations are at higher risk because of their close interactions with contaminated freshwater bodies.[21] In contrast, schistosomiasis remains uncommon in school-going populations and urban dwellers, who have limited exposure to such water sources. However, peri-urban areas with inadequate sanitation and access to clean water are becoming new zones of vulnerability.[41] Consequently, the patient's occupational status and living environment further complicated the diagnosis. The key factor in establishing the diagnosis was the recent travel history in which there was exposure to rice fields. This diagnostic dilemma led to the consideration of other conditions causing cerebral manifestations.

In urban pediatric populations, neuroschistosomiasis is rare. In endemic regions like the Philippines, where TB remains highly prevalent, tuberculomas must be included in differential diagnoses. Studies show that the Philippines continues to be a high-burden country for TB, with both latent and active cases widespread across various regions.[7] Tuberculomas are a significant manifestation of CNS TB. These lesions may occasionally present without ring enhancement on imaging, causing a diagnostic conundrum.[28] Some documented cases highlight instances where tuberculomas remained systemically asymptomatic and present with nonspecific clinical presentations such as headaches, seizures, or focal neurological deficits.[1,29] These cases necessitated biopsy due to inconclusive imaging results. Abbasi *et al*. (2021) reported a case where the diagnosis of tuberculoma was confirmed only after an open brain biopsy revealed noncaseating granulomas, underscoring the difficulties in distinguishing tuberculomas from other intracranial pathologies causing granulomatous lesions as seen in our case.[1] Additional studies have documented solitary, asymptomatic brain tuberculomas, further illustrating the complexity of diagnosis.[29]

Pediatric cases of brain neoplasms are also more relevant, complicating diagnoses in school-aged individuals presenting with neurological symptoms but lacking systemic signs of schistosomiasis or TB.[7,27]

MRS was used as a diagnostic modality for our case to investigate the biochemical composition of the contrast-enhanced lesion revealed on MRI. While nonspecific, MRS revealed a choline peak indicative of cell membrane turnover,

which can occur in various conditions such as neoplasms, demyelination, inflammation, and gliosis.[3] MRS done in our patient revealed increased levels of choline (Cho)/N-acetyl-aspartate (NAA) ratio, elevated choline at the lesion site, elevated lipid lactate peaks in the same region, and decreased levels of NAA. Another case report by Llenas-García *et al*. (2009)[23] demonstrated increased lipid lactate peak and decreased NAA peak in the cerebral *Schistosoma* lesion. Considering the lipid lactate peak, a tuberculoma could be considered; however, this condition would also present with increased NAA levels[34], which were decreased in our patient. Similarly, Wilbers *et al*. (2010)[39] also reported increased Cho/NAA ratio and increased lactate lipids with additional findings of aspartate and glutathione. In 2020, Ghimire and Wu[17] attempted to describe the MRI features at different stages of cerebral schistosomiasis in 25 cases, showing increased levels of Cho/NAA ratio in all. The above studies give insight into the use of MRS as an adjunct tool to diagnose neuroschistosomiasis; however, there is limited literature to infer this adequately.

Due to the limited literature available, the sensitivity and specificity of MRS in the diagnosis of schistosomiasis remain inadequately defined. A *Schistosoma* serology of blood using Falcon assay screening test – enzyme-linked immunosorbent assay[19] has a specificity of 99% and sensitivity of 99% for *S. mansoni*, 95% for *S. haematobium*, and approximately 50% for *S. japonicum* infection.[40] However, cross-reactivity with other parasitic infections due to shared glycan epitopes and proteins[11,14] can lead to false positives.[13] Furthermore, blood serology alone is not sufficient for diagnosing neuroschistosomiasis because the CNS involvement may not always elicit a strong peripheral immune response.[15] Cerebrospinal fluid (CSF) serological testing can be helpful if the results are positive (sensitivity, 83–88%), but the test has relatively low specificity (range, 38–67%).[20] In particular, patients with neuroschistosomiasis typically present with elevated CSF eosinophils and *Schistosoma*-specific antibodies, making it a more targeted test than blood serology in cases with neurological involvement.[9] In our case, no CSF immunoanalysis was done due to unavailability.

In this case, a tissue biopsy of the lesion was necessary. The patient presented with persistent headaches and recurrent seizures despite adherence to antiseizure medications. Neuroimaging suggested an infectious origin, but the findings were inconclusive in differentiating between a tuberculoma and neuroschistosomiasis. Although MRS provided valuable biochemical insights, it did not confirm the diagnosis. Given the increasing frequency of seizures and the need for timely initiation of the correct treatment – since TB and schistosomiasis require entirely different regimens – a tissue biopsy became essential. This case underscores the importance of biopsy when imaging and laboratory results are inconclusive and when a trial of medication would delay effective treatment. It also highlights the role of MRS as an adjunct diagnostic tool that could potentially reduce the need for unnecessary biopsies and enable earlier treatment initiation.

The treatment of cerebral schistosomiasis consists of anti-schistosomal (namely, praziquantel) therapy, corticosteroids to control the inflammation, anticonvulsants when indicated, and/or surgery. The cornerstone of treatment is praziquantel, an anti-schistosomal agent highly effective in eliminating the parasite. Praziquantel's mechanism of action involves increasing the permeability of the parasite's cell membrane to calcium ions. This results in muscle contraction followed by paralysis and eventual death of the parasite. Studies indicate that high doses (40–60 mg/kg) of praziquantel, administered over 1–2 days, effectively eradicate the parasite load in patients with neuroschistosomiasis. In our case, a single dose of 60 mg/kg was administered over 1 day. However, despite its efficacy in eliminating adult worms, the inflammatory response caused by the dead parasites often necessitates adjunct therapies.[38] Interestingly, praziquantel reduces posttreatment seizures by eliminating parasites and diminishing CNS inflammation. Patients often show improvement in neurological symptoms, including reduced seizure frequency, once the infection and inflammation are controlled.[36]

In managing the inflammatory sequelae of neuroschistosomiasis, corticosteroids, particularly dexamethasone, are used to reduce perilesional edema and mitigate the immune response before the administration of praziquantel. By inhibiting pro-inflammatory cytokine production, dexamethasone minimizes the risk of neurological damage due to the host's inflammatory response to dying schistosomes.[16] Following medical treatment, regular follow-up is essential to monitor neurological recovery and seizure control, especially in patients who undergo biopsy or surgery, as seen in our case. Imaging and clinical assessments are critical in tracking the resolution of granulomatous lesions, and long-term seizure management may be required depending on the severity of the pretreatment neurological involvement.[36]

CONCLUSION

In cases where neuroschistosomiasis is a potential diagnosis, several important lessons emerge. First, although neurological manifestations of schistosomiasis are uncommon, clinicians should maintain a high index of suspicion in patients from endemic regions, even when the patient's occupational background and locality do not align with typical risk factors. For example, in our case, the patient was a student residing in an urban setting rather than a farmer who was exposed to contaminated freshwater areas such as rivers or farmlands, leading to the initial oversight of the diagnosis. A thorough history and physical examination

remain indispensable, particularly in identifying potential environmental exposures, such as this patient's contact with rice fields. In addition, while neuroimaging techniques like MRI are crucial, they may be inconclusive in distinguishing neuroschistosomiasis from other infectious etiologies such as TB. However, MRI, particularly when supplemented by modalities like MRS, can play an important role in supporting early diagnostic efforts, potentially reducing the need for invasive procedures like brain biopsy. Physicians are advised to incorporate both clinical and imaging findings into their diagnostic approach to ensure prompt and accurate treatment, avoiding unnecessary delays of interventions.

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REFERENCES

1. Abbasi F, Ozer M, Juneja K, Goksu SY, Mobarekah BJ, Whitman MS. Intracranial tuberculoma mimicking neurosarcoidosis: A clinical challenge. Infect Dis Rep 2021;13:181-6.
2. Abdel-Wahab MF, Esmat G, Milad M, Abdel-Razek S, Strickland GT. Characteristic sonographic pattern of schistosomal hepatic fibrosis. Am J Trop Med Hyg 1989;40:72-6.
3. Ahmed MM, Osman HH, Mohamed AH, Ginawi A. Surgical management outcome of cerebral schistosomiasis: A case report and review of the literature. J Med Case Rep 2021;15:268.
4. Alanazi RF, Al Karawi M, Almalki A, Sufiani F, Al Karawi S. Schistosomiasis involving the central nervous system: Case report of a rare complication. Case Rep Surg 2023;2023:9968155.
5. Albano JE, Ma-Alat RB. Misdiagnosis of hepatosplenic schistosomiasis as hepatic tuberculosis: A case report. Cureus 2023;15:e35169.
6. Alzayer Z, Al Nasser Y. Primary lung tuberculosis. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2023.
7. Bai W, Ameyaw EK. Global, regional and national trends in tuberculosis incidence and main risk factors: A study using data from 2000 to 2021. BMC Public Health 2024;24:12.
8. Blas BL. Handbook for the control of *Schistosomiasis* japonica. Manila, Philippines: Department of Health; 1988.
9. Carod-Artal FJ. Neuroschistosomiasis. Expert Rev Anti Infect Ther 2010;8:1307-18.
10. Chen R, Wang J, Gradinaru I, Vu HS, Geboers S, Naidoo J, *et al*. A male-derived nonribosomal peptide pheromone controls female schistosome development. Cell 2022;185:1506-20.e17.
11. Cognato BB, Handali S, de Mattos Pereira L, Barradas JR, da Silva AJ, Graeff-Teixeira C, *et al*. Identification of cross-reactive markers to strengthen the development of immunodiagnostic methods for angiostrongyliasis and other parasitic infections. Exp Parasitol 2020;218:107999.
12. Colley DG, Bustinduy AL, Secor WE, King CH. Human schistosomiasis. Lancet 2014;383:2253-64.
13. Elbaz T, Esmat G. Hepatic and intestinal schistosomiasis: Review. J Adv Res 2013;4:445-52.
14. El-Faham MH, Gai F, Igetei JE, Richter S, Falcone FH, Schramm G, *et al*. Antigenic cross-reactivity between *Schistosoma mansoni* and allergenic invertebrates putatively due to shared glycanic epitopes. Sci Rep 2020;10:3350.
15. Ferrari TC, Moreira PR. Neuroschistosomiasis: Clinical symptoms and pathogenesis. Lancet Neurol 2011;10:853-64.
16. Fowler R, Lee C, Keystone JS. The role of corticosteroids in the treatment of cerebral schistosomiasis caused by *Schistosoma mansoni*: Case report and discussion. Am J Trop Med Hyg 1999;61:47-50.
17. Ghimire P, Wu G. Spectrum of imaging findings in oriental cerebral schistosomiasis and their categorization by MRI. Nepalese J Radiol 2020;10:16-26.
18. Gryseels B, Polman K, Clerinx J, Kestens L. Human schistosomiasis. Lancet 2006;368:1106-18.
19. Hancock K, Tsang VC. Development and optimization of the FAST-ELISA for detecting antibodies to *Schistosoma mansoni*. J Immunol Methods 1986;92:167-76.
20. Imai K, Koibuchi T, Kumagai T, Maeda T, Osada Y, Ohta N, *et al*. Cerebral schistosomiasis due to *Schistosoma haematobium* confirmed by PCR analysis of brain specimen. J Clin Microbiol 2011;49:3703-6.
21. Klohe K, Koudou BG, Fenwick A, Fleming F, Garba A, Gouvras A, *et al*. A systematic literature review of schistosomiasis in urban and peri-urban settings. PLoS Negl Trop Dis 2021;15:e0008995.
22. Liu Z, Zhang L, Liang Y, Lu L. Pathology and molecular mechanisms of *Schistosoma japonicum*-associated liver fibrosis. Front Cell Infect Microbiol 2022;12:1035765.
23. Llenas-García J, Guerra-Vales JM, Alcalá-Galiano A, Domínguez C, Pérez-Nuñez A, Lizasoaín M, *et al*. Rare disease: Cerebral neuroschistosomiasis: A rare clinical presentation and review of the literature. BMJ Case Rep 2009;2009:bcr04.2009.1787.
24. Loscalzo J, Fauci A, Kasper D, Hauser S, Longo D, Jameson JL. Harrison's principles of internal medicine. 21st ed., Vol. 2., Ch. 234. United States: McGraw Hill LLC; 2022. p. 1784-8.
25. Murray CJ, Vos T, Lozano R, Naghavi M, Flaxman AD, Michaud C, *et al*. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: A systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380:2197-23.
26. Nelwan ML. Schistosomiasis: Life cycle, diagnosis, and control. Curr Ther Res 2019;91:5-9.

27. Ostrom QT, Cioffi G, Waite K, Kruchko C, Barnholtz-Sloan JS. CBTRUS statistical report: Primary brain and other central nervous system tumors diagnosed in the United States in 2014-2018. Neuro Oncol 2021;23:III1-105.
28. Pagnoux C, Généreau T, Lafitte F, Congy F, Chiras J, Herson S. Brain tuberculomas. Ann Med Interne (Paris) 2017;151:231-48.
29. Perez-Malagon CD, Barrera-Rodriguez R, Lopez-Gonzalez MA, Alva-Lopez LF. Diagnostic and neurological overview of brain tuberculomas: A review of literature. Cureus 2021;13:e20133.
30. Pittella JE. Neuroschistosomiasis. Brain Pathol 1997;7:649-62.
31. Rollinson D. Human schistosomiasis. Trans R Soc Trop Med Hyg 1994;88:716-7.
32. Ross AG, McManus DP, Farrar J, Hunstman RJ, Gray DJ, Li YS. Neuroschistosomiasis. J Neurol 2012;259:22-32.
33. Scrimgeour EM, Gajdusek DC. Involvement of the central nervous system in *Schistosoma mansoni* and *S. haematobium* infection. A review. Brain 1985;108:1023-38.
34. Shingade RG, Prakashchandra SP. Role of advanced diagnostic imaging in intracranial tuberculoma: MR spectroscopy. J Clin Diagn Res 2015;9:TJ03.
35. Suthiphosuwan S, Lin A, Gao AF, Munoz DG, Spears J, Bharatha A. Delayed presentation of cerebral schistosomiasis presenting as a tumor-like brain lesion. Neuroradiol J 2018;31:395-8.
36. Vezzani A, Fujinami RS, White HS, Preux PM, Blümcke I, Sander JW, *et al*. Infections, inflammation and epilepsy. Acta Neuropathol 2016;131:211.
37. Wang J, Paz C, Padalino G, Coghlan A, Lu Z, Gradinaru I, *et al*. Large-scale RNAi screening uncovers therapeutic targets in the parasite *Schistosoma mansoni*. Science 2020;369:1649-53.
38. Watt G, Long GW, Ranoa CP, Adapon B, Fernando MT, Cross JH. Praziquantel in treatment of cerebral schistosomiasis. Lancet 1986;2:529-32.
39. Wilbers J, Idema A, Gijtenbeek A. A bilateral foot drop due to neuroschistosomiasis. J Neurol 2010;257:853-5.
40. Wilkins PP, Keystone JS. Schistosomiasis serology is valuable and reliable. Clin Infect Dis 2013;56:312.
41. World Health Organization. Schistosomiasis. Geneva: World Health Organization; 2023.
42. World Health Organization. The control of schistosomiasis. Report of a WHO Expert Committee. World Health Organ Tech Rep Ser. 1985;728:1-113.
43. Yamagiwa K. Beitrag contribution to the etiology of Jacksonian Epilepsy. Arch für Pathol Anat Physiol für Klin Med 1890; 119:447-60.

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