World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(REVIEW ARTICLE)

Neuroschistosomiasis: Exploring the Clinical and Epidemiological Dimensions of a Neglected Tropical Disease

Abdul Muhymin Alam Khattak [1], Sayyed Muhammad Taha Hussain [2], Muhammad Mustafa [3], Naveed Ahmed [3], Muhammad Usman Sharif [3], Sadia Shafique [4], Abdul Mohaimin Muhammad [5], Azlan Shah [6], Zahir Rehman [7], Asad Riaz [8,*] and Imtiaz Khan [9]

[1] *Department of Medical Oncology, St Vincent's Private Hospital, Dublin, Ireland.*
[2] *Department of Pulmonology, Lady Reading Hospital, Peshawar, Pakistan.*
[3] *Department of Internal Medicine, Ayub Teaching Hospital, Abbottabad, Pakistan.*
[4] *Department of Neurosurgery, Lady Reading Hospital, Peshawar, Pakistan.*
[5] *Medical Officer at Shaukat Khanum Memorial Cancer Hospital and Research Centre, Peshawar, Pakistan.*
[6] *Demonstrator at Abbottabad International Medical College, Abbottabad, Pakistan*
[7] *Department of Neurosurgery, Shifa International Hospitals Limited, Islamabad, Pakistan.*
[8] *Department of Urology, Institute of Kidney Diseases, Peshawar, Pakistan.*
[9] *Department of Internal Medicine, Ayub Teaching Hospital, Abbottabad, Pakistan.*

World Journal of Advanced Research and Reviews, 2025, 27(02), 933-940

Publication history: Received on 11 June 2025; revised on 19 July 2025; accepted on 21 July 2025

Article DOI: https://doi.org/10.30574/wjarr.2025.27.2.0499

Abstract

Neuroschistosomiasis is a rare but severe neurological complication of schistosomiasis, a parasitic disease caused by *Schistosoma* species. Although primarily affecting the gastrointestinal and urogenital systems, aberrant migration of eggs or adult worms to the central nervous system (CNS) can result in significant morbidity and disability.

This narrative review provides a comprehensive overview of neuroschistosomiasis, with a focus on its epidemiology, pathogenesis, clinical manifestations, diagnostic challenges, treatment strategies, and public health implications. A literature review was conducted using databases such as PubMed, Scopus, and Google Scholar to identify peer-reviewed articles, case reports, clinical guidelines, and review articles on neuroschistosomiasis. Emphasis was placed on studies describing CNS pathology, diagnostic approaches, therapeutic outcomes, and disease burden.

Neuroschistosomiasis occurs in less than 5% of infected individuals but may be underreported. *S. mansoni*, *S. haematobium*, and *S. japonicum* are the primary species implicated. Spinal forms, presenting as transverse myelitis or conus medullaris syndrome, are most commonly seen, while cerebral forms may present with seizures, focal deficits, or signs of raised intracranial pressure. Diagnosis relies on MRI, CSF analysis, serology, and occasionally CNS biopsy.

Management includes praziquantel and corticosteroids, with supportive rehabilitation. Prognosis depends on timely intervention, and long-term sequelae are common.

Neuroschistosomiasis remains a neglected yet important cause of neurological disease in endemic areas and among travelers. Enhanced clinical awareness, improved diagnostics, and coordinated public health interventions are crucial to reduce morbidity and improve outcomes. Further research into pathogenesis and vaccine development is urgently needed.

* Corresponding author: Asad Riaz

Keywords: Neuroschistosomiasis; Schistosomiasis; Bilharzia; Katayama Fever; Flatworm; Platyhelminths; Imported Diseases; Acute Schistosomiasis; Cercarial Dermatitis; Chronic Schistosomiasis

1. Introduction

1.1. Overview of Schistosomiasis

Schistosomiasis, also referred to as bilharzia, is a neglected tropical disease caused by parasitic blood flukes belonging to the genus Schistosoma. It impacts over 240 million individuals worldwide and is second only to malaria in terms of its socioeconomic and public health effects among parasitic diseases. The disease burden is concentrated in more than 70 endemic nations, mainly in sub-Saharan Africa, South America, the Middle East, and parts of Asia, where limited access to clean water and sanitation facilitates its transmission. Human infection occurs when skin comes into contact with freshwater contaminated by cercariae, the larval stage of the parasite released from infected snails. Clinically, schistosomiasis usually manifests as intestinal or urogenital disease, depending on the infecting species—S. mansoni and S. japonicum primarily cause hepatointestinal disease, while S. haematobium is linked to urinary tract involvement. However, in a small number of cases, schistosome eggs or adult worms migrate abnormally to the central nervous system (CNS), leading to neuroschistosomiasis. This rare but severe complication is marked by granulomatous inflammation, immune-mediated tissue damage, and neurological dysfunction, which can be disabling or even fatal if not treated. Neuroschistosomiasis may affect the spinal cord or brain, with the clinical presentation influenced by the infecting species, immune response, and timing of intervention. Spinal forms often result in acute or subacute transverse myelitis, while cerebral involvement may present as seizures, focal neurological deficits, or intracranial hypertension. Due to its ability to mimic other neurological conditions such as tumors, autoimmune disorders, or infections, diagnosis is often delayed—especially in non-endemic areas where clinical suspicion is low. Despite its serious consequences, neuroschistosomiasis remains under-recognized and underreported, representing a critical gap in global health awareness. This narrative review aims to synthesize current knowledge on neuroschistosomiasis, focusing on its clinical spectrum, diagnostic challenges, and evidence-based management strategies. Through a comprehensive examination of the literature, this review also highlights public health priorities and future directions for research in addressing this neglected manifestation of a globally significant parasitic infection. `

Epidemiology and Global Burden Schistosomiasis remain one of the most prevalent and impactful neglected tropical diseases, affecting an estimated 220 million people worldwide, with over 90% of cases occurring in sub-Saharan Africa [1]. In addition to Africa, endemic regions include parts of South America (particularly Brazil), the Middle East, Southeast Asia, and certain provinces in China. The disease is primarily caused by five medically significant species of Schistosoma: S. mansoni, S. haematobium, S. japonicum, S. intercalate, and S. Mekong. Each species has a distinct geographic distribution and is associated with characteristic clinical syndromes, most commonly hepatointestinal or urogenital forms of schistosomiasis.

In contrast, neuroschistosomiasis is a relatively rare yet severe complication, representing a small fraction—estimated at less than 5%—of overall schistosomiasis cases [2]. However, this figure may be an underestimation because of the disease's non-specific neurological presentation and underreporting, especially within low-resource settings, as well as lack of access to diagnostic tools. Most documented neuroschistosomiasis cases are in Brazil, Egypt, China, and various sub-Saharan African countries [3]. However, in recent years, global travel, population migration, together with refugee movements occurred, leading to diagnoses that were sporadic in non-endemic regions like Europe and North America. These foreign examples stress a global need for more healthcare provider clinical awareness. Schistosomiasis is typically not encountered in some settings, particularly so, and awareness is necessary [4].

2. Life Cycle and Pathogenesis

Schistosoma species have a complicated life cycle that includes an intermediate host of freshwater snails as well as a definitive host of humans. Cercariae, the parasite's free-swimming larval stage, are discharged from infected snails and enter humans through unbroken skin when they come into touch with tainted freshwater. Once inside the host, cercariae travel hematogenous to the lungs and subsequently to the liver, where they mature into adult male and female worms. These adult worms pair and move to their preferred venous sites: S. mansoni and S. japonicum to the mesenteric venous plexus, and S. haematobium to the *pelvic and vesical venous plexus. From there, they lay eggs that traverse tissue walls and are expelled in feces or urine, depending on the species [5].

Both a human definitive host and a freshwater snail intermediate host are involved in the complicated life cycle of Schistosoma species. When contaminated freshwater comes into touch with undamaged human skin, the parasite's free-

swimming larval stage, cercariae, which are released from infected snails, causes human infection. Following their hematogenous migration to the lungs and liver within the host, cercariae develop into adult male and female worms. S. mansoni and S. japonicum migrate to the mesenteric venous plexus, while S. haematobium migrates to the *pelvic and vesical venous plexus. These mature worms couple and migrate to their chosen venous locations. Depending on the species, they then lay eggs that pass-through tissue walls and are expelled as either urine or feces [5].

A freshwater snail serves as the intermediate host in the complicated life cycle of Schistosoma species, which also has a definitive host in humans. The parasite's free-swimming larval stage, cercariae, are discharged from infected snails and enter the human body through undamaged skin when contaminated freshwater comes into contact with it. Once within the host, cercariae undergo hematogenous migration to the liver and lungs, where they develop into adult male and female worms. The preferred venous locations of these adult worms are the mesenteric venous plexus for S. mansoni and S. japonicum, and the *pelvic and vesical venous plexus for S. haematobium. They pair up and migrate, respectively. Depending on the species, their eggs are then expelled in either urine or feces after passing through tissue walls [5].

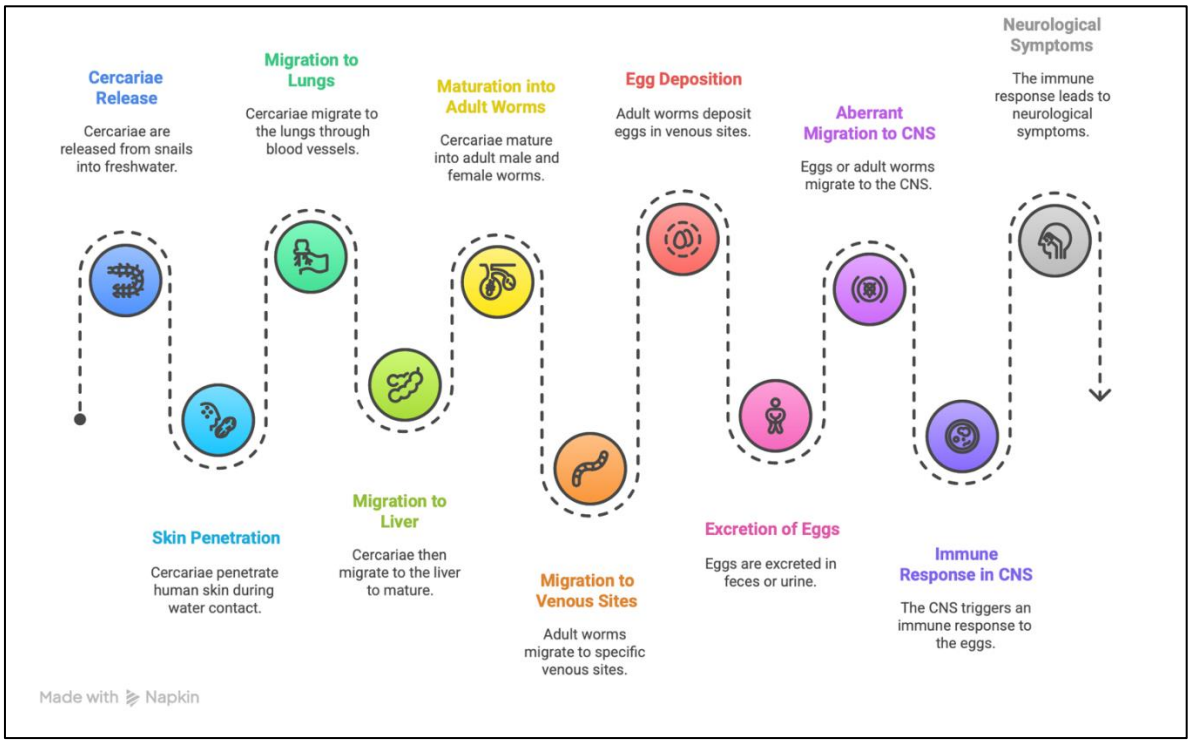

Figure 1 Schistosoma Life Cycle and Neurochistosomiasis

3. Clinical Manifestations

Eosinophils, macrophages, lymphocytes, and fibroblasts mediate the strong granulomatous immunological response that schistosome eggs induce once they are deposited in the central nervous system. In the end, this immune activation causes neurological symptoms that differ depending on the region of involvement through tissue swelling, demyelination, necrosis, and mass impact. The pathogenic process can resemble viral, autoimmune, or neoplastic illnesses, making prompt diagnosis and identification much more difficult [8].

The most prevalent clinical type of spinal neuroschistosomiasis is caused by Schistosoma mansoni or Schistosoma haematobium. Usually, it manifests as conus medullaris syndrome, cauda equina syndrome, or acute or subacute transverse myelitis. Depending on the degree of involvement, patients frequently describe bilateral lower limb weakness, which can be either flaccid or spastic. Common sensory abnormalities typically have a distinct sensory level upon assessment. Early-stage areflexia or later-stage hyperreflexia are examples of reflex alterations. With Schistosoma mansoni or Schistosoma haematobium as the most common cause, spinal neuroschistosomiasis is the most common clinical type. Conus medullaris syndrome, cauda equina syndrome, or acute or subacute transverse myelitis are the usual ways it manifests. Bilateral lower limb weakness, which can be either flaccid or spastic depending on the degree of involvement, is frequently reported by patients. Sensory disturbances are frequent and typically exhibit a distinct sensory level upon evaluation. Areflexia in the early stages of the disease or hyperreflexia as it worsens are examples of

reflex alterations. It is not unusual to experience altered mental status, disorientation, and behavioral or cognitive abnormalities, particularly when there is diffuse cortical involvement. Because granulomatous lesions in the cortex or white matter can closely resemble tumors or abscesses on imaging, these symptoms could be confused with encephalitis, stroke, or neoplastic processes. Vasculitis alterations in the cerebral vasculature may cause ischemic stroke-like episodes in certain people [10].

There is growing recognition of neuroschistosomiasis's unusual and chronic manifestations. These include increasing myelopathy, cognitive deterioration, and even mental manifestations like mood disorders or psychosis, and they might appear gradually over weeks to months. In rare cases, granulomatous blockage of the ventricular system or poor CSF absorption at the arachnoid granulations can cause hydrocephalus. These atypical cases often elude early diagnosis, particularly when presenting in non-endemic regions where schistosomiasis is not routinely considered [11].

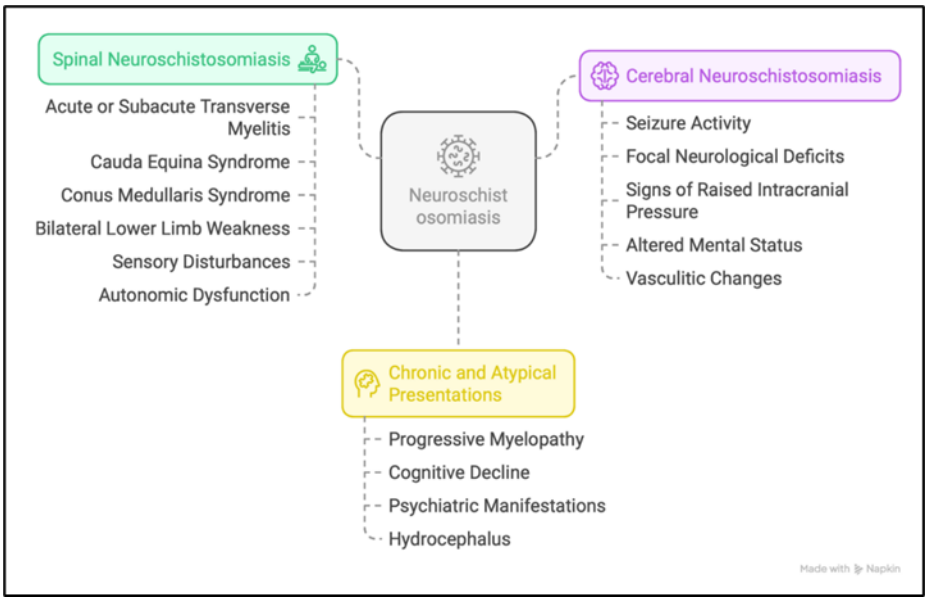

Figure 2 Neuroschistosomiasis clinical manifestations and diagnostic challenges

4. Diagnostic Challenges

Because neuroschistosomiasis has nonspecific and variable clinical symptoms, diagnosis is frequently delayed. Clinicians may initially mistake symptoms for more prevalent neurological conditions such multiple sclerosis, autoimmune or viral transverse myelitis, malignancies, or vascular insults in both endemic and non-endemic situations. Frequent misdiagnosis is a result of the condition's rarity as well as the way it mimics other diseases on imaging and clinical evaluation.

Patients with epidemiological risk factors, such as living in or visiting endemic areas, having a history of freshwater exposure, or having systemic schistosomiasis concurrently, require a high index of suspicion. Irreversible neurological damage may arise from delayed diagnosis, highlighting the significance of healthcare personnel' understanding. Combining laboratory tests, targeted imaging, and clinical history

Patients with epidemiological risk factors, such as living in or visiting endemic areas, having a history of freshwater exposure, or having systemic schistosomiasis concurrently, require a high index of suspicion. Irreversible neurological damage may arise from delayed diagnosis, highlighting the significance of healthcare personnel' understanding. Timely detection and better results can be achieved by combining clinical history with targeted imaging, laboratory tests, and, if necessary, serologic or genetic testing.

5. Diagnosis

Particularly in individuals with epidemiological risk factors including living in or visiting endemic areas, having a history of exposure to freshwater, or having systemic schistosomiasis concurrently, a high index of suspicion is crucial. Irreversible neurological damage might arise from delayed diagnosis, which emphasizes how crucial it is for healthcare

professionals to be informed. Clinical history combined with targeted imaging, lab work, and, if necessary, molecular or serologic testing can help identify problems early and enhance results.

Analysis of cerebrospinal fluid (CSF) provides additional support for the diagnosis, but it is not very specific. Elevated protein levels and lymphocytic pleocytosis are common observations that indicate a continuing inflammatory response. Even though it strongly suggests a parasite etiology, CSF eosinophilia is not always present and may not be present in a sizable portion of cases. Although they are more frequently linked to autoimmune diseases and can complicate the diagnostic process, oligoclonal bands can also occasionally be identified [13].

The diagnosis is further supported by cerebrospinal fluid (CSF) analysis; however, this method is not very specific. Common signs of an ongoing inflammatory reaction include elevated protein levels and lymphocytic pleocytosis. CSF eosinophilia is not usually present and may not be present in a significant number of cases, despite the fact that it strongly supports a parasite origin. On occasion, oligoclonal bands can also be detected, but they are more commonly associated with autoimmune disorders and can make diagnosis more difficult [13].

Serology and parasite tests provide valuable additional information. connected to enzymes.

Tests for parasites and serology offer useful further data. Schistosoma-specific antibodies in serum can be found with high sensitivity and specificity using immunoblot and enzyme-linked immunosorbent assays (ELISA), especially in patients from endemic areas [14]. However, because egg excretion does not usually occur at the same time as neurological involvement, regular stool and urine microscopy for ova may produce negative results in patients with CNS-limited disease. In situations when conventional techniques are equivocal, molecular diagnostics, such as polymerase chain reaction (PCR)-based assays on CSF or serum, have demonstrated promise in improving diagnostic sensitivity and can assist in confirming infection.

6. Treatment and Management

The management of neuroschistosomiasis focuses on two primary goals: eradication of the parasitic infection and modulation of the host's inflammatory response to prevent permanent neurological damage.

Testing for parasites and serous diseases yields useful supplementary data. Immunoblot and enzyme-linked immunosorbent assay (ELISA) tests have good sensitivity and specificity for detecting Schistosoma-specific antibodies in serum, especially in individuals from endemic areas [14]. However, because neurological involvement is not frequently accompanied by egg expulsion, conventional stool and urine microscopy for ova may produce negative results in patients with CNS-limited disease. When conventional approaches are unable to confirm an infection, molecular diagnostics, such as polymerase chain reaction (PCR)-based assays on CSF or serum, have demonstrated promise in improving diagnostic sensitivity.

Praziquantel is frequently used in conjunction with corticosteroids to treat the inflammatory component. These are essential for lessening the immune-mediated tissue damage and edema brought on by egg deposition in the central nervous system. One gram of methylprednisolone is usually administered intravenously for three to five days, after which oral prednisone is gradually tapered down. Individualized corticosteroid therapy duration is determined by patient tolerance, radiologic improvement, and clinical response [17]. In many cases, extended corticosteroid therapy may be required to achieve full neurologic recovery, particularly in spinal neuroschistosomiasis.

Together, timely diagnosis and a combined therapeutic strategy targeting both the pathogen and the host response offer the best chance for neurological improvement and prevention of long-term sequelae in patients with neuroschistosomiasis.

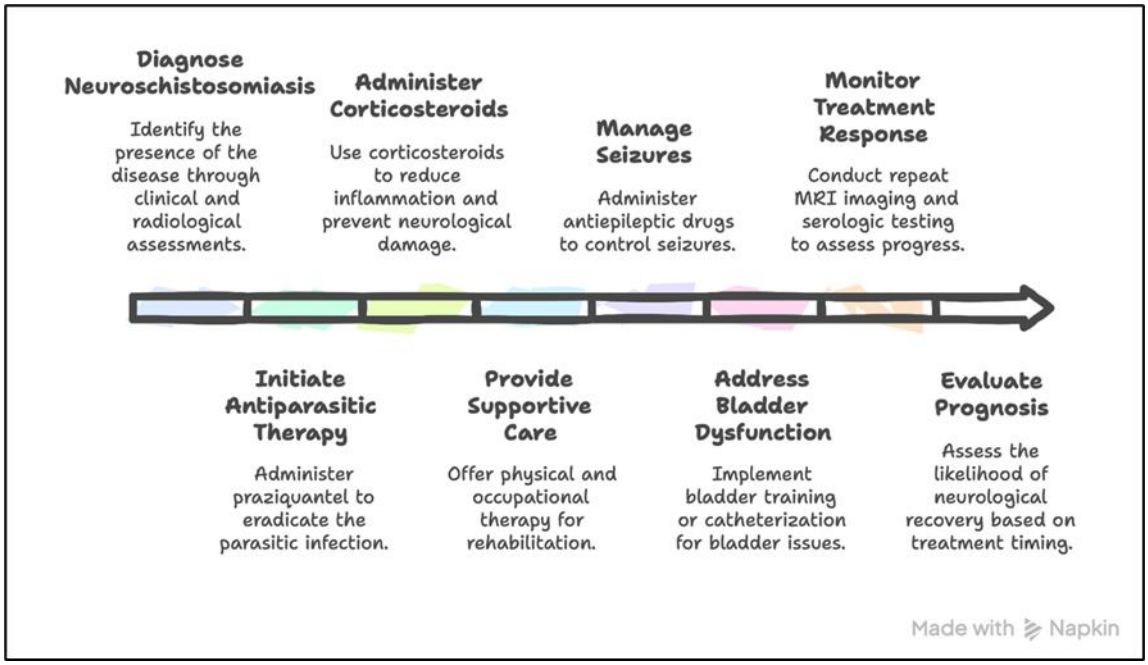

Figure 3 Neuroschistosomiasis management process

7. Supportive Care and Follow-Up

Supportive care is essential to the overall management of neuroschistosomiasis, especially in patients with moderate to severe neurological impairments, in addition to antiparasitic and anti-inflammatory treatment. Rehabilitation requires physical and occupational treatment, particularly for those with functional impairments brought on by spinal cord involvement, motor weakness, or abnormalities in gait. The likelihood of a functional recovery is increased and quality of life is improved with early rehabilitation commencement. Antiepileptic medications should be used when seizures are a presenting symptom of cerebral neuroschistosomiasis in order to guarantee seizure control and lower the chance of recurrence or neurological decline. In order to prevent urinary tract infections and maintain renal function, bladder dysfunction—which is commonly seen in cases with spinal involvement—may also call for bladder training, intermittent catheterization, or even long-term urological follow-up [18].

Supportive care, especially for patients with moderate to severe neurological impairments, is essential to the overall management of neuroschistosomiasis in addition to antiparasitic and anti-inflammatory treatment. Rehabilitation requires physical and occupational therapy, particularly for people with spinal cord involvement-related functional deficits, motor weakness, or abnormalities in gait. Early rehabilitation improves quality of life and increases the likelihood of a functional recovery. When seizures are a presenting symptom of cerebral neuroschistosomiasis, antiepileptic medications should be used to manage seizures and lower the chance of neurological decline or recurrence. Furthermore, bladder dysfunction, which is commonly seen in cases of spinal involvement, may require intermittent catheterization, bladder training, or even long-term urological follow-up in order to maintain renal function and avoid UTIs [18].

7.1. Prognosis and Outcomes

In the entire management of neuroschistosomiasis, supportive care is essential, especially for patients with moderate to severe neurological impairments, in addition to antiparasitic and anti-inflammatory treatment. Particularly for those with motor weakness, abnormalities in gait, or functional impairments brought on by spinal cord involvement, physical and occupational therapy are crucial for rehabilitation. Rehabilitation that is started early increases quality of life and increases the likelihood of a functional recovery. Antiepileptic medications should be used when seizures are a presenting symptom of cerebral neuroschistosomiasis in order to guarantee seizure control and lower the chance of recurrence or neurological decline. Furthermore, periodic catheterization, bladder training, or even long-term urological follow-up may be necessary to avoid UTIs and maintain renal function in cases of bladder dysfunction, which is commonly seen in spinal involvement [18].

The timely diagnosis and timely initiation of adequate medication are critical factors in the prognosis of neuroschistosomiasis. Early treatment often has positive results for patients, with many obtaining partial or full neurological recovery, particularly during the acute period of symptom development. On the other hand, chronic pain syndromes, paraplegia, sensory loss, and sphincter dysfunction are among the irreversible neurological impairments that are more likely to occur in cases of delayed diagnosis. Mortality is uncommon, although it can happen in extreme circumstances because to complications like sepsis, hydrocephalus, or aspiration pneumonia, especially when brain involvement raises intracranial pressure.

7.2. Public Health, Prevention, and Research Gaps

Neuroschistosomiasis has both opportunities for preventive intervention and problems for public health because it is a neglected tropical illness. Mass drug administration (MDA) of praziquantel to at-risk groups, especially school-aged children, who have the largest infection burden, is the cornerstone of prevention in endemic areas. Furthermore, limiting exposure to contaminated freshwater through health education programs, sanitary infrastructure, and enhanced access to clean water are essential elements in breaking transmission cycles [20].

However, several challenges continue to hinder effective control. These include limited access to diagnostic imaging and serologic testing in resource-poor settings, low clinical awareness of neuroschistosomiasis among healthcare providers in non-endemic regions, and the absence of robust surveillance systems for neurological complications of schistosomiasis. These factors contribute to underdiagnosis and undertreatment, exacerbating patient morbidity.

Nonetheless, a number of obstacles still stand in the way of efficient control. These include the lack of effective monitoring systems for schistosomiasis neurological sequelae, low clinical knowledge of neuroschistosomiasis among healthcare personnel in non-endemic locations, and restricted access to diagnostic imaging and serologic testing in settings with minimal resources. These elements exacerbate patient morbidity by causing underdiagnosis and undertreatment.

The development of more precise and sensitive diagnostic instruments, especially point-of-care assays appropriate for use in endemic regions, is urgently needed from a scientific standpoint. To improve treatment plans, including the best dosage, length of time, and combinations of antiparasitic and anti-inflammatory medications, randomized clinical trials are also required. Additionally, a deeper comprehension of the host-parasite interactions and immunopathogenesis in CNS disease may provide new targets for treatment. Lastly, research into vaccines against Schistosoma species is still ongoing.

8. Conclusion

A dangerous and usually underdiagnosed side effect of Schistosoma infection, neuroschistosomiasis presents formidable diagnostic and treatment obstacles. From acute spinal cord syndromes to long-term cognitive and behavioral abnormalities, its clinical presentation is quite diverse and frequently resembles that of more prevalent neurological conditions like tumors, multiple sclerosis, or stroke. In non-endemic areas or among returning tourists, where schistosomiasis is not frequently taken into account in the differential diagnosis, this clinical variability leads to a high rate of misdiagnosis or delayed detection.

Significant diagnostic and treatment issues arise from neuroschistosomiasis, a dangerous and commonly underdiagnosed consequence of Schistosoma infection. Acute spinal cord syndromes and persistent cognitive and behavioral abnormalities are just two examples of its extremely varied clinical presentation, which frequently mimics more prevalent neurological conditions like tumors, multiple sclerosis, or stroke. Because schistosomiasis is not frequently taken into account in the differential diagnosis, this clinical variability frequently results in misdiagnosis or delayed detection, especially in non-endemic areas or among returning tourists.

Improvements in diagnostic techniques, such as the creation of molecular and sensitive serologic assays, as well as increased accessibility to imaging in endemic areas, are also necessary to address the burden of neuroschistosomiasis. From a public health standpoint, preventing CNS involvement and reducing transmission require integrated measures that include mass drug administration (MDA), enhanced sanitation, health education, and surveillance systems. In order to address this overlooked but significant manifestation of a parasite disease that is widespread around the world, more study into the immunopathogenesis, therapy optimization, and vaccine development will be essential.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] World. Schistosomiasis (Bilharzia). Who.int. Published February 28, 2020. Accessed July 2, 2025. https://www.who.int/health-topics/schistosomiasis#tab=tab_1

[2] Ferrari TC. Involvement of central nervous system in the schistosomiasis. Mem Inst Oswaldo Cruz. 2004;99(5 Suppl 1):59-62. doi:10.1590/s0074-02762004000900010Ross AG, Bartley PB, Sleigh AC, et al. Schistosomiasis. N Engl J Med. 2002;346(16):1212–1220.

[3] Gryseels B, Polman K, Clerinx J, Kestens L. Human schistosomiasis. Lancet. 2006;368(9541):1106-1118. doi:10.1016/S0140-6736(06)69440-3

[4] Carod-Artal FJ. Neurological complications of Schistosoma infection. Trans R Soc Trop Med Hyg. 2008;102(2):107-116. doi:10.1016/j.trstmh.2007.08.004

[5] Scrimgeour EM, Gajdusek DC. Involvement of the central nervous system in Schistosoma mansoni and S. haematobium infection. A review. Brain. 1985;108 (Pt 4):1023-1038. doi:10.1093/brain/108.4.1023

[6] Suchet I, Klein C, Horwitz T, Lalla S, Doodha M. Spinal cord schistosomiasis: a case report and review of the literature. Paraplegia. 1987;25(6):491-496. doi:10.1038/sc.1987.82.

[7] Pittella JE. Neuroschistosomiasis. Brain Pathol. 1997;7(1):649-662. doi:10.1111/j.1750-3639.1997.tb01080.x

[8] Zaqout A, Abid FB, Murshed K, et al. Cerebral schistosomiasis: Case series from Qatar. Int J Infect Dis. 2019;86:167-170. doi:10.1016/j.ijid.2019.07.002

[9] Llenas-García J, Guerra-Vales JM, Alcalá-Galiano A, et al. Cerebral neuroschistosomiasis: a rare clinical presentation and review of the literature. BMJ Case Rep. 2009;2009:bcr04.2009.1787. doi:10.1136/bcr.04.2009.1787

[10] Saleem S, Belal AI, El-Ghandour NM. Spinal cord schistosomiasis: MR imaging appearance with surgical and pathologic correlation. AJNR Am J Neuroradiol. 2005;26(7):1646-1654.

[11] Liu H, Lim CC, Feng X, et al. MRI in cerebral schistosomiasis: characteristic nodular enhancement in 33 patients. AJR Am J Roentgenol. 2008;191(2):582-588. doi:10.2214/AJR.07.3139

[12] Chala B. Advances in Diagnosis of Schistosomiasis: Focus on Challenges and Future Approaches. Int J Gen Med. 2023;16:983-995. Published 2023 Mar 18. doi:10.2147/IJGM.S391017.

[13] Jauréguiberry S, Paris L, Caumes E. Acute schistosomiasis, a diagnostic and therapeutic challenge. Clin Microbiol Infect. 2010;16(3):225-231. doi:10.1111/j.1469-0691.2009.03131.x

[14] Inobaya MT, Olveda RM, Chau TN, Olveda DU, Ross AG. Prevention and control of schistosomiasis: a current perspective. Res Rep Trop Med. 2014;2014(5):65-75. doi:10.2147/RRTM.S44274

[15] Ferrari TC, Moreira PR. Neuroschistosomiasis: clinical symptoms and pathogenesis. Lancet Neurol. 2011;10(9):853-864. doi:10.1016/S1474-4422(11)70170-3

[16] Lu CY, Zhao S, Wei Y. Cerebral schistosomiasis: MRI features with pathological correlation. Acta Radiol. 2021;62(5):646-652. doi:10.1177/0284185120934475

[17] Ferreira MS, Costa-Cruz JM, Gomes MA. Esquistossomose do sistema nervoso central: relato de um caso [Schistosomiasis of the central nervous system: report of a case]. ArqNeuropsiquiatr. 1990;48(3):371-375. doi:10.1590/s0004-282x1990000300018

[18] Preventive chemotherapy. Who.int. Published 2015. Accessed July 2, 2025. https://www.who.int/teams/control-of-neglected-tropical-diseases/interventions/strategies/preventive-chemotherapy