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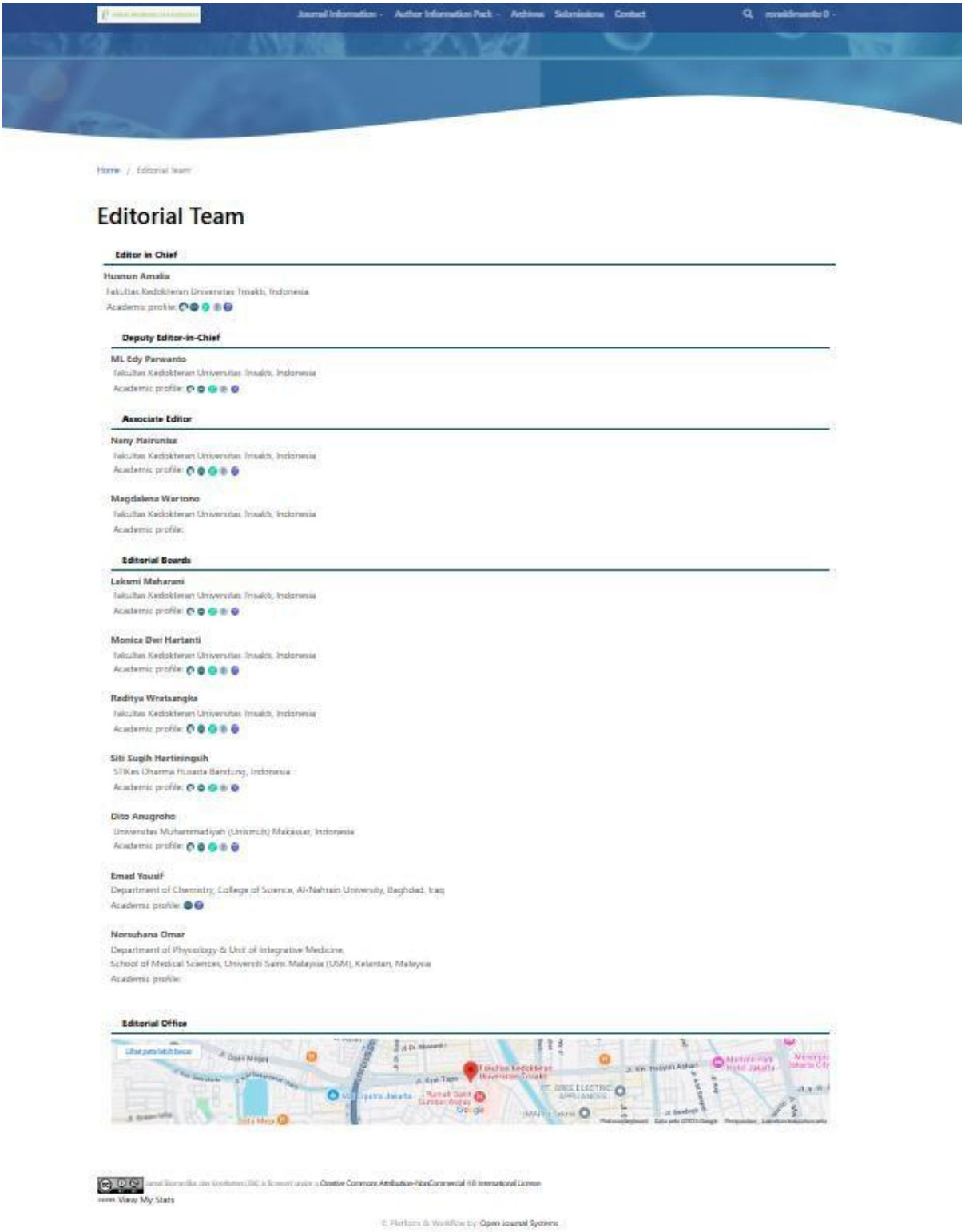

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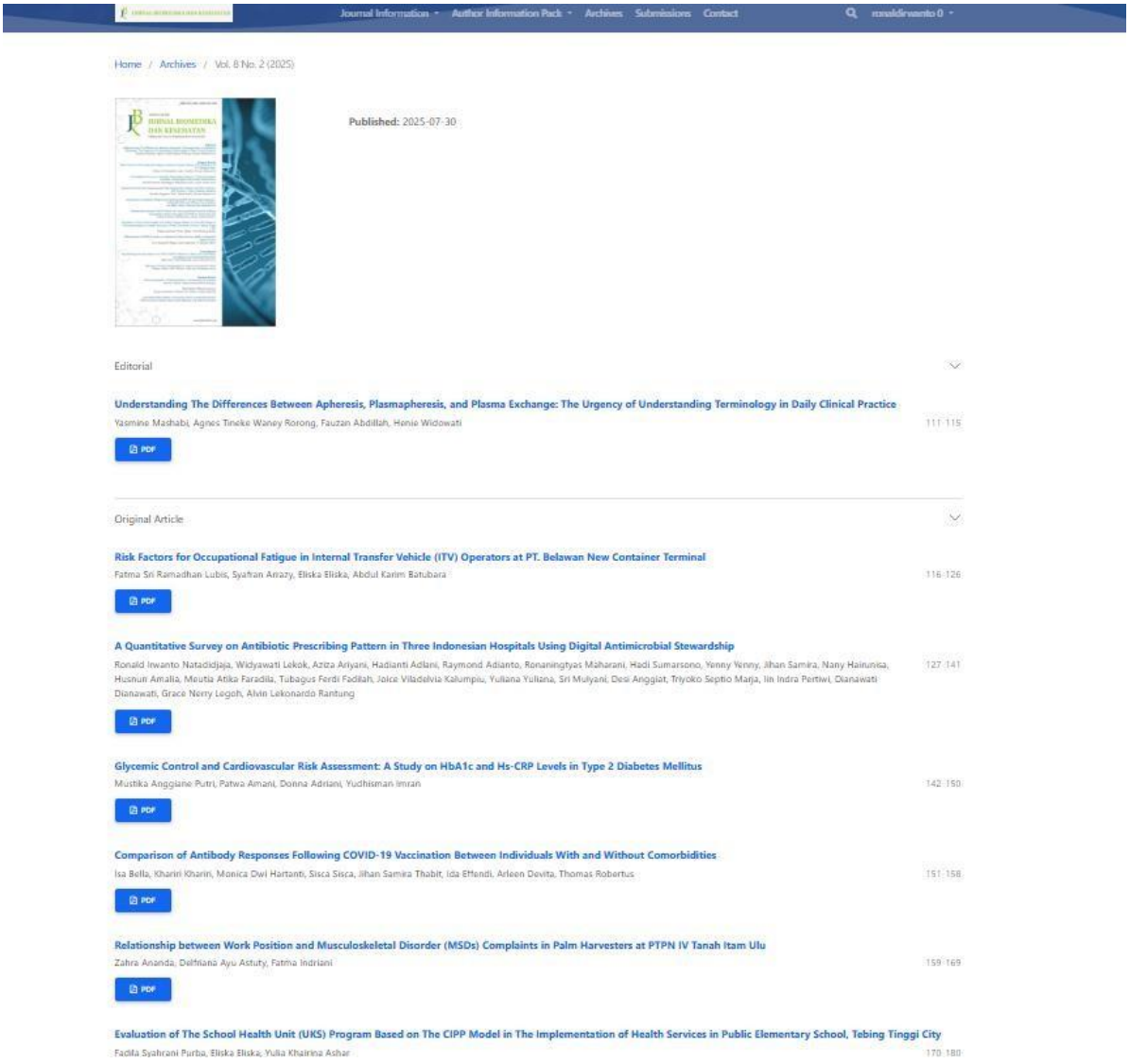

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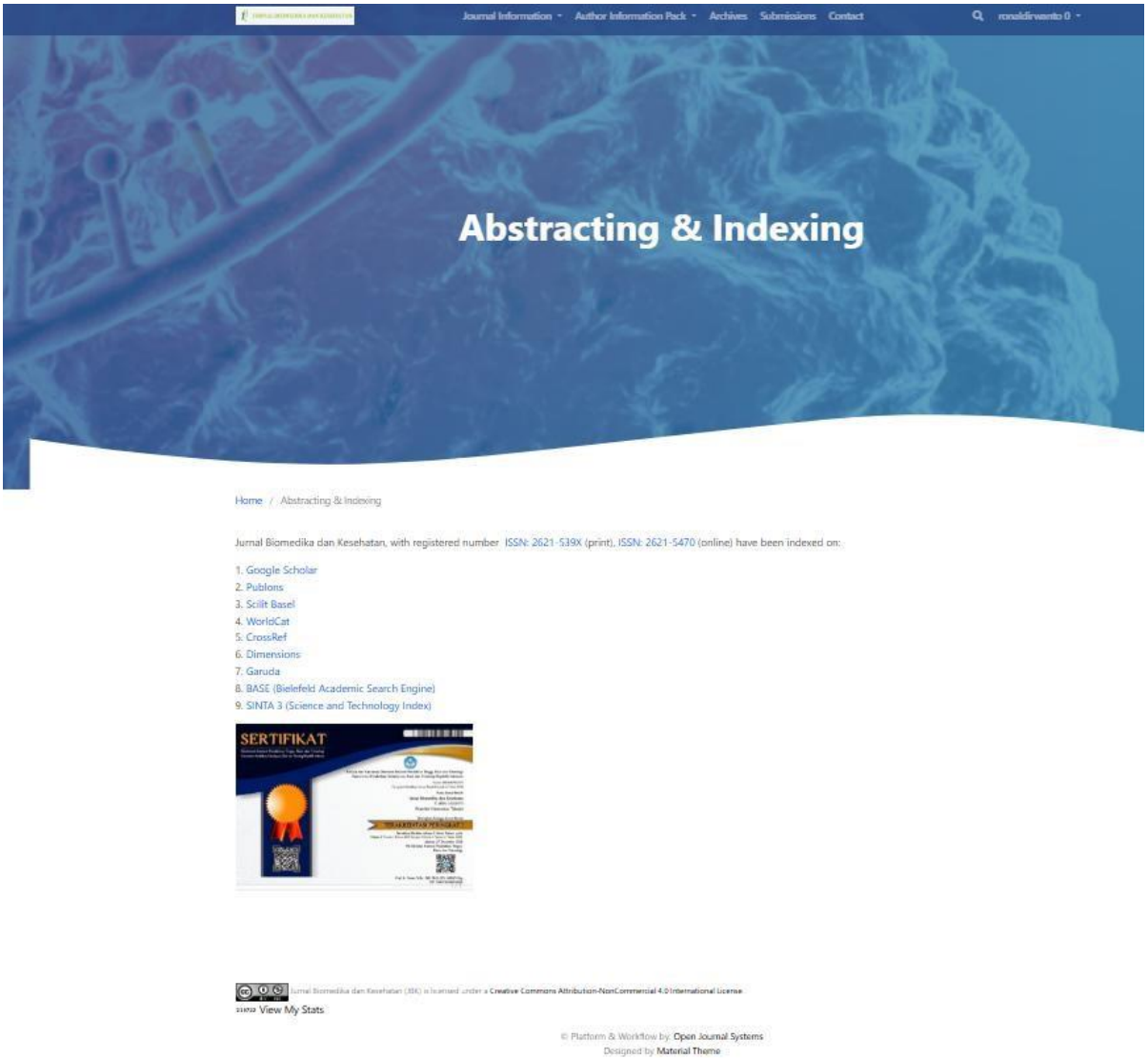

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- **Blood trematodes: Schistosomiasis in Central Nervous System**

Yuliana Yuliana, Machrumnizar Machrumnizar

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RESEARCH ARTICLE

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Blood trematodes: Schistosomiasis in Central Nervous System

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Keywords:

Brain, Schistosoma, Schistosomiasis, Spine, Tropical Disease

Abstract

Schistosomiasis (bilharzia) is a disease caused by blood trematode worms of the genus *Schistosoma, which can be acute or chronic*. There are five species of *Schistosoma* (*S. japonicum, S. mansoni, S. intercalatum, S. Mekongi, and S. haematobium*) known to infect humans, which are distributed across a wide geographic range. Severe clinical symptoms of schistosomiasis infection on the central nervous system (CNS) are caused by the deposition of eggs when adult worms migrate abnormally to the brain or spinal cord. As a result, radiculopathy, myelopathy, increased intracranial pressure, and clinical sequelae occur. Each species shows different tendencies for atopic infections and clinical symptoms. The most common neurological symptom associated with *Schistosoma mansoni* or *S. haematobium* infections is myelopathy, while severe encephalitis is more likely to be caused by *S. japonicum* infections. The symptoms of encephalopathy can manifest as headache, vision disturbances, delirium, convulsions, motor limitations, and ataxia. Spinal abnormalities may lead to backache, lower limb radiating discomfort, muscular weakness, loss of sensation, and urinary impairment. Histopathological analysis, which shows granulomas and *Schistosoma* eggs, is necessary for a definitive diagnosis. Current management involves surgical procedures, steroids, and schistosomicide drugs. Corticosteroids are administered in the acute stage, and praziquantel is given after the female worm begins to lay eggs. If medical intervention fails to relieve compression or medullary degeneration, surgery should be postponed in some instances. The earlier a diagnosis is identified and proper therapy begins, the better the patient's outcome.

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REVIEW ARTICLE

Blood trematodes: Schistosomiasis in Central Nervous System

Trematoda Darah: Schistosomiasis pada Sistem Saraf Pusat

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ABSTRACT

Schistosomiasis (bilharzia) is a disease caused by blood trematode worms of the genus *Schistosoma, which can be acute or chronic*. There are five species of *Schistosoma* (*S. japonicum, S. mansoni, S. intercalatum, S. Mekongi, and S. haematobium*) known to infect humans, which are distributed across a wide geographic range. Severe clinical symptoms of schistosomiasis infection on the central nervous system (CNS) are caused by the deposition of eggs when adult worms migrate abnormally to the brain or spinal cord. As a result, radiculopathy, myelopathy, increased intracranial pressure, and clinical sequelae occur. Each species shows different tendencies for atopic infections and clinical symptoms. The most common neurological symptom associated with *Schistosoma mansoni* or *S. haematobium* infections is myelopathy, while severe encephalitis is more likely to be caused by *S. japonicum* infections. The symptoms of encephalopathy can manifest as headache, vision disturbances, delirium, convulsions, motor limitations, and ataxia. Spinal abnormalities may lead to backache, lower limb radiating discomfort, muscular weakness, loss of sensation, and urinary impairment. Histopathological analysis, which shows granulomas and *Schistosoma* eggs, is necessary for a definitive diagnosis. Current management involves surgical procedures, steroids, and schistosomicide drugs. Corticosteroids are administered in the acute stage, and praziquantel is given after the female worm begins to lay eggs. If medical intervention fails to relieve compression or medullary degeneration, surgery should be postponed in some instances. The earlier a diagnosis is identified and proper therapy begins, the better the patient's outcome.

Keywords: Brain; *Schistosoma*; Schistosomiasis; Spine; Tropical Disease.

ABSTRAK

Schistosomiasis adalah suatu penyakit akut sekaligus kronis yang disebabkan oleh cacing trematoda darah dari genus *Schistosoma*. Terdapat lima spesies *Schistosoma* (*S. japonicum, S. mansoni, S. intercalatum, S. Mekongi,* dan *S. haematobium*) yang diketahui menginfeksi manusia dan tersebar di rentang geografis yang luas. Gejala klinis berat akibat infeksi schistosomiasis pada sistem saraf pusat (SSP) disebabkan oleh deposit telur ketika cacing dewasa bermigrasi secara abnormal ke otak atau sumsum tulang belakang. Akibatnya, terjadi radikulopati, mielopati, peningkatan tekanan intrakranial, dan sekuel klinis. Setiap spesies menunjukkan kecenderungan infeksi atopik dan gejala klinis yang berbeda. Gejala neurologis paling umum yang disebabkan oleh infeksi *Schistosoma mansoni* (*S. mansoni)* atau *S. haematobium* adalah mielopati, sementara ensefalitis berat lebih sering terjadi pada infeksi *S. japonicum*. Gejala ensefalopati dapat berupa sakit kepala, gangguan penglihatan, delirium, kejang, keterbatasan motorik, dan ataksia. Kelainan tulang belakang dapat menyebabkan nyeri punggung, rasa tidak nyaman yang menjalar ke tungkai bawah, kelemahan otot, hilangnya sensasi, dan gangguan berkemih. Analisis histopatologi yang menunjukkan granuloma dan telur *Schistosoma* diperlukan untuk diagnosis pasti. Penanganan saat ini mencakup prosedur bedah, steroid, dan obat schistosomisida; kortikosteroid diberikan pada tahap akut, dan praziquantel digunakan setelah cacing betina mulai bertelur. Jika intervensi medis gagal meredakan kompresi atau degenerasi medula, pembedahan dapat dipertimbangkan dalam kasus tertentu. Semakin dini diagnosis ditegakkan dan terapi yang tepat dimulai, semakin baik prognosis pasien.

Kata Kunci: Otak; *Schistosoma*; Schistosomiasis; Tulang Belakang; Penyakit Tropis.

INTRODUCTION

The COVID-19 pandemic and efforts to control it in 2021 resulted in a reduced focus on the part of governments and healthcare providers, particularly on the availability of interventions and treatments for neglected tropical diseases (NTDs), including schistosomiasis.[1–3] Schistosomiasis (bilharzia) is a disease caused by blood trematode worms of the genus *Schistosoma, characterized as both acute and chronic.*[4] Schistosomiasis is prevalent in tropical and subtropical regions, especially in poor communities that do not have access to safe drinking water and adequate sanitation.[5] World Health Organization (WHO) reports that schistosomiasis in all its forms has been transmitted in 78 countries, of which 51 are endemic countries requiring preventive chemotherapy (**Figure 1**).[6]

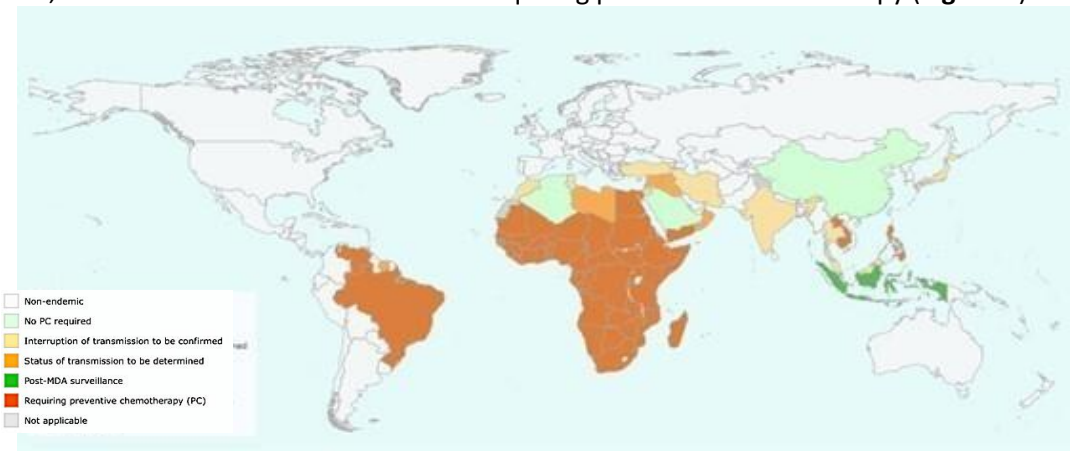

Figure 1. Map of the geographical distribution of schistosomiasis. (Source: https://www.who.int/data/gho/data/themes/topics/schistosomiasis)

It is estimated that around 251.4 million people required preventive treatment for schistosomiasis in 2021, and more than 75 million people have received treatment.[6] There are five species of *Schistosoma* known to infect humans that are distributed across a wide geographic range.[6,7] Infections with *S. japonicum, S. mansoni, S. intercalatum,* and *S. mekongi* have been linked to protracted intestinal and liver fibrosis. In contrast, chronic infections of *S. haematobium* are prone to the

formation of fibrosis, narrowing, and a calcified bladder.[8,9] Ectopic eggs of the species *Schistosoma* migrate to various organs and give rise to clinical manifestations based on the affected organs, such as the skin, brain, muscles, eyes, adrenal glands, and genitourinary system. Granulomas can form in the fallopian tubes, ovaries, and uterus.[9]

The term "neuro-schistosomiasis" refers to schistosomiasis of brain tissue, which is the most severe clinical form of *Schistosoma* infection and can arise either symptomatically or asymptomatically due to the presence of schistosomes.[10] Neuro-schistosomiasis cases have been found in soldiers and workers serving in schistosomiasis endemic areas, as well as in tourists who rarely visit these areas.[11] *Schistosoma japonicum, S. mansoni, and S. haematobium* are common species that cause neuroschistosomiasis. Infection of *S. japonicum* typically leads to acute encephalitis involving the cerebral cortex, basal ganglia, subcortical white matter, or inner capsule. Infections with *S. mansoni* or *S. haematobium* can cause neurological manifestations, particularly myelopathy (acute transverse myelitis and subacute myeloradiculopathy), linked to necrotic inflammation in the lumbar-sacral area.[13,14]

Methods

A literature search was performed between September and October 2023 using the following keywords: "schistosomiasis" OR "*Schistosoma*" OR "infection AND brain AND *Schistosoma*" OR "schistosomiasis AND brain" OR "schistosomiasis AND nervous system AND central" OR "schistosomiasis AND spinal" OR "schistosomiasis AND bone and back." Literature was gathered from electronic databases, including PubMed, Scopus, and Google Scholar, as well as the bibliographies of relevant publications. The search was limited to freely accessible full-text articles in English, but no systematic methodology was applied to the search and selection process.

RESULTS

Life Cycle

Schistosoma has a complex life cycle (**Figure 2**), involving freshwater snails as intermediate hosts and higher vertebrates, such as mammals, as definitive hosts, where *Schistosoma* lives in the bloodstream of these mammals.[15] Click or tap here to enter text. Male and female *Schistosoma* worms mate, with the female residing in the male's ventral gynaecophoric canal. Female *S. mansoni* lays 100–300 eggs daily, *S. haematobium* lays 20–200, and *S. japonicum* lays 500–3,500. Egg shape and location help differentiate species, as they accumulate in the target organ's capillaries.[16] Highly antigenic eggs migrate through the intestinal wall or bladder and are excreted in feces or urine.[16,17]

Once expelled into freshwater, eggs hatch into miracidia within ten days, which infect specific snail intermediate hosts, transforming into sporocysts and later into cercariae. The cercariae, capable of free-swimming for up to 72 hours, must find and penetrate a definitive mammalian host within 12–24 hours to continue the life cycle.[16,17] Each *Schistosoma* species has a distinct snail host in endemic regions; for example, *Oncomelania hupensis* hosts *S. japonicum*, while *Biomphalaria* species host *S. mansoni*. Cercariae penetrate intact skin using suckers and proteolytic enzymes, shed their tails, and migrate via the lymphatic system to the lungs, where they develop immune-resistant schistosomula. These schistosomula travel to the liver's portal vessels, mature into adult worms, and form pairs before migrating to their target organs, completing the life cycle.[18] This complex parasitic process underpins the persistence of schistosomiasis in endemic regions.

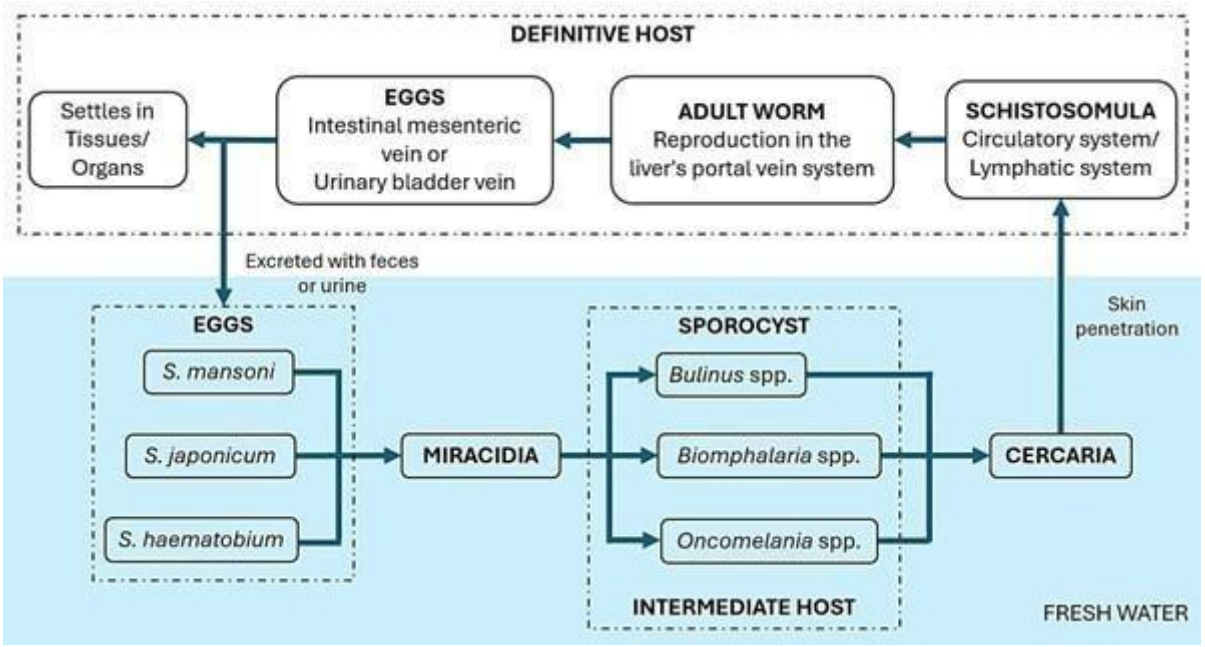

Figure 2. Life cycle of the worm *Schistosoma* spp.

Pathogenesis

After the worms develop into adults, they reproduce and produce eggs. *Schistosoma* can enter the central nervous system. After maturation and reproduction, *Schistosoma* can invade the central nervous system (CNS). Eggs must enter Batson's epidural venous plexus, linking the portal vein and vena cava systems to the spinal cord and cerebral veins, to induce CNS symptoms. Through this route, mature worms may deposit eggs near the CNS or embolize large numbers

from mesenteric vessels. The smaller, spherical eggs of *S. japonicum* can reach the CNS, while the larger eggs of *S. mansoni* and *S. haematobium* are typically confined to the lower vertebral cord.[13,16,19] In chronic hepatosplenic and cardiopulmonary schistosomiasis, venous or arterial invasion mechanisms can result in CNS implication without symptoms.[13,20] . The rare detachment of eggs and the absence of a peri-ovular inflammatory response associated with severe chronic infections result in the lack of symptoms.[20]

Schistosomiasis pathology primarily arises from the intense inflammatory response to trapped eggs, not the worms themselves. Eggs contain miracidia, which can mature into adults within 5–20 days. Chronic disease is driven by host immune reactions and granulomatous responses to antigens, particularly glycoproteins, secreted by miracidia. Granulomas predominantly form in areas where eggs accumulate, *specifically* in the intestines and liver for S. mansoni and *S. japonicum, and* in the genitourinary tract for S. haematobium . [19,21] Granulomas may form in tissues such as the brain, muscles, skin, lungs, and adrenal glands. Perioval granulomas consist of fibroblasts, eosinophils, and plasma cells, with necrotic centers containing egg clusters surrounded by giant cells, lymphocytes, and epithelioid cells. While single eggs are reabsorbed, tissue damage can lead to the development of fibrosis. Chronic infections lead to the formation of calcified eggs and excessive extracellular matrix deposition, resulting from granulomatous inflammation. Immune responses dynamically regulate collagen deposition, crosslinking, contraction, and reabsorption.[21]

Clinical Manifestations

Schistosomiasis of the cerebral

Brain involvement in schistosomiasis is more frequent with *S. japonicum* infections (4–28%) than with other *Schistosoma* species. The primary neurological manifestations are diffuse encephalopathy and seizures. The smaller eggs of *S. japonicum* have a higher likelihood of penetrating the brain.[19] Granulomatous lesions and increased intracranial pressure in the subcortical white matter, cortex, internal capsule, or basal ganglia commonly manifest as neurological disturbances, including fever, headache, nystagmus, speech difficulties, and motor weakness. One possible outcome of high pressure in the brain is papilledema. Neurological symptoms typical of cerebral complications due to infection *S. japonicum* are seizures, both focal and generalized. Brain granulomas caused by *Schistosoma* can trigger partial motor seizures and secondary generalized tonic-clonic seizures. In the absence of systemic infection, partial motor seizures may serve as an early and sole indicator of neuroschistosomiasis.[22] Brain infection-related complications of *S. haematobium* or *S. mansoni* typically affect the brain cortex, cerebellum, and leptomeninges.[23]

Pseudo-tumoral encephalic schistosomiasis (PES), primarily caused by *S. japonicum*, occurs predominantly in individuals aged 10–40 from endemic regions without other schistosomiasis manifestations. While all brain lobes may be affected, the cerebellum, occipital lobe, and frontal lobe are most commonly involved. PES may also present extracerebral lesions on the dura mater's inner surface. Neurological symptoms result from slow-growing brain lesions, edema, and mass effects, leading to increased intracranial pressure, intracranial hypertension, or hydrocephalus. Common symptoms include headaches, motor and sensory disorders, visual disturbances, mental status changes, vertigo, speech disorders, vomiting, and ataxia, with cerebral nerve paralysis and meningeal signs being rare. Diagnosis relies on identifying eggs or granulomas via biopsy, as other diagnostic methods are nonspecific.[24]

Schistosomiasis of the spine

The predominant form of neuroschistosomiasis, spinal cord schistosomiasis (SCS), is primarily caused by *Schistosoma mansoni* and typically occurs without other symptoms of infection.[10,13]

However, previous reports mentioned that SCS occurred during *acute toxemic schistosomiasis* (ATS), immediately afterwards, or simultaneously with hepatosplenic types. SCS is more common in children, adolescents, and young adults than in older people. The clinical manifestations of SCS are classified into three forms: [1] *Medullary form*, primarily involving the spinal cord; [2] *Myeloradicular form*, affecting both the spinal cord and nerve roots; [3] *Conus-cauda equina syndrome*, which is predominantly involved in the *conus* or *cauda equina*. Medullary schistosomiasis typically presents with symmetrical sensorimotor anomalies, rapid progression, and severe weakness. In contrast, conus-cauda equina syndrome progresses more slowly, with asymmetrical sensorimotor changes and milder weakness. The myeloradicular type, the most common form, presents intermediate features and is primarily caused by *S. mansoni* and *S. haematobium.*[25]

Other symptoms in neuroschistosomiasis

Immunocompromised individuals with schistosomiasis may develop cerebral vasculitis, mediated by eosinophil-induced toxicity. During the acute stage, schistosomula have not matured, and eggs are undetectable, as *Schistosoma* begins egg production 1–2 months post- infection. Severe cases, such as *S. mansoni* infestations, can result in hyper-eosinophilic syndrome, leading to ischemic infarctions and chronic endomyocardial fibrosis.[26] Granulomatous inflammation around *Schistosoma* eggs may cause cerebral arterial necrosis, resulting in subacute hematomas, cerebellar hematomas, or subarachnoid hemorrhage. These neurological complications highlight the severe impact of schistosomiasis on the central nervous system.[27]

Diagnosis

Conventional microscopic detection

The parasitological detection of *Schistosoma* eggs in urine or feces is crucial for diagnosing active schistosomiasis, with the Kato-Katz method and the miracidia hatching technique (MHT) considered the "gold standard," particularly in endemic areas. The Kato-Katz method, using 25–50 mg of stool, is valued for its simplicity, cost-effectiveness, and ability to identify species and estimate worm burden. However, it is less effective in detecting low-intensity infections, especially in non-endemic or low-prevalence areas.[28]

The Helmintex test, which employs paramagnetic beads, offers higher sensitivity than the Kato-Katz method but requires further evaluation for cost-effectiveness and field applicability. For superior diagnostic accuracy, rectal biopsy with an ovogram, which has a sensitivity of 95–100%, is often preferred for diagnosing active schistosomiasis.[29,30]

Radiology features of neuroschistosomiasis

Diagnosis of neuroschistosomiasis by neuroimaging using *computerized tomography* scan (CT) and *magnetic resonance imaging* (MRI) can provide effective results. Additionally, CT and MRI can assess the severity of the disease process and its complications in the target organ.[31] CT images in neuroschistosomiasis are associated with inflammatory reactions and granuloma formation due to egg deposition in the brain and spinal cord, generally showing mass lesions with hyperdense centers surrounded by edematous shadows or calcification, and hypodense areas with varying contrast enhancements. Granulomatous lesions are also associated with secondary bleeding.[32]

MRI is effective in diagnosing cerebral and spinal schistosomiasis, revealing masses with scattered or clustered nodular "mud-like" enhancements in cortical or subcortical areas. Spinal schistosomiasis typically presents as lesions accompanied by spinal edema, conus medullaris involvement, cauda equina involvement, and irregular thickening of the cauda equina nerve roots.

MRI also detects spinal cord compression.[24,32] The MRI images of neuroschistosomiasis typically show enlargement of the spinal cord, especially in the ventral area, due to the formation of intramedullary Schistosoma granulomas. These granulomas may appear as unevenly nodular, multiple lesions resembling beaded strands, diverse, highly intense lesions with ill-defined boundaries, or moderate extensions of the distally located medulla that are iso-intense compared to the medulla oblongata.[14] Spinal cord atrophy can be found in longstanding cases.[22]

Immunology detection

Immunological methods for detecting *Schistosoma* antibodies are highly sensitive, cost- effective, and simple, making them useful for early screening and surveillance in endemic areas. These methods, including ELISA, IHA, and immunofluorescence, detect IgG, IgM, or IgE antibodies against soluble egg or adult worm antigens. While serology is beneficial for diagnosing travelers and patients without eggs, such as those with Katayama syndrome, it is less specific compared to fecal screening due to cross-reactivity with other worms [28,30]

Molecular technique

Nucleic acid detection, particularly PCR, offers superior sensitivity and specificity for diagnosing schistosomiasis, effectively detecting *Schistosoma* DNA in various samples, including feces, urine, blood, and environmental specimens. Blood-based PCR is promising for the diagnosis of acute schistosomiasis, while real-time PCR can estimate the burden of *S. mansoni* infection. However, these methods require costly infrastructure and rigorous validation for broader application in control programs.[29,33,34]

Treatment

Praziquantel

A pyrazinoisoquinoline derivative, praziquantel, is a secure schistosomicidal, very potent, and effective oral drug against every adult worm of the *Schistosoma* species.[34] Praziquantel has been widely used since its development in the mid-20th century due to its safety and efficacy. Due to currently no vaccine for immunization, praziquantel has become the backbone of schistosomicidal. The precise mode of action of praziquantel remains undetermined, and while the calcium-ions pathway of *Schistosoma* was recently proposed as a molecular target, the data is yet inconclusive.[35]

Artemether

Artemether and artesunate, derived from *Artemisia annua*, were first identified as anti-*schistosoma*l agents in the 1980s, particularly against *S. japonicum*. These artemisinin derivatives, initially used for malaria, are effective against juvenile *Schistosoma* within the first three weeks of infection. Administering artemether biweekly effectively targets and eliminates schistosomula in humans and animals.[35]

Corticosteroid

For encephalopathy schistosomiasis, prednisone (1.5–2.0 mg/kg daily for three weeks) combined with praziquantel is recommended during the egg-laying period to reduce CNS granulomatous inflammation. However, no randomized trials have confirmed the effects of corticosteroids on the spinal cord. Praziquantel is not advised during acute infection due to its inability to target immature schistosomula (3–21 days). Corticosteroids remain the preferred treatment, while the potential use of artemether, alone or with corticosteroids, for neurologic complications requires further study.[25,36,37]

Disease Prevention

Research on schistosomiasis vaccines, including *S. mansoni* smFABP and DNA-based approaches, shows promising progress. While praziquantel lacks preventive effects, artemether offers prophylactic potential when taken biweekly, effectively targeting all major *Schistosoma* species.[8] In addition to vaccination and alternative medicine, there are several essential factors in efforts to control schistosomiasis, especially in endemic areas[6]: (1) Provide population-based preventive chemotherapy; (2) Ensure a safe water supply; (3) Health education for improved water sanitation; (4) Avoiding urine or stool contaminated with schistosome is a prerequisite; (5) Eradicating snails reduces worm transmission and adds further control. Visitors to endemic regions should be aware of potential exposure to freshwater larvae. Prompt treatment is recommended upon clinical suspicion or confirmed diagnosis of schistosomiasis to reduce disease burden. Topical lotions containing N, N-diethyl-m-toluamide effectively kill *Schistosoma* cercariae with minimal risk to humans.[38]

CONCLUSION

Neuro-schistosomiasis, with symptoms of *Schistosoma* involvement in the CNS, is a serious condition. Despite increasing reports of the disease in endemic areas and among tourists, it remains underdiagnosed. *Schistosoma* infestations can result in harm to the central nervous system and spinal cord. The stage of infection and the clinical form have a significant impact on the etiology, clinical presentation, and prognosis. Reducing irreversible neurological consequences and improving clinical outcomes requires the immediate identification and intervention of these conditions. To effectively treat neuroschistosomiasis, the best treatment is to combine targeted anti-*Schistosoma* treatment with rapid surgical debridement. Early diagnosis, accompanied by prompt and appropriate treatment, can significantly improve a patient's prognosis.

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CONFLICT OF INTEREST

All authors declare that they have no conflict of interest concerning the submitted manuscript.

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