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Review article

Unraveling the enigma of spinal cord schistosomosis: clinical spectrum, diagnosis, and therapeutic insights

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ABSTRACT. Spinal cord schistosomosis is a rare but significant cause of myelopathy, ranging from asymptomatic egg deposits to severe transverse myelitis. Common in endemic regions, *S. mansoni* myelopathy presents as acute paraplegia or cauda equina syndrome. Diagnosis involves identifying ova in stool, urine, or rectal biopsy, accompanied by peripheral eosinophilia. Treatment includes praziquantel and corticosteroids, with laminectomy reserved for specific cases. Prognosis varies, with around one-third recovering completely, one-third with residual deficits, and one-third remaining unchanged or deteriorating, particularly in necrotic myelitis. Recognition in endemic areas is critical for timely intervention and improved outcomes in this potentially treatable myelopathy.

Keywords: spine, spinal cord, schistosomosis, bilharziosis, parasite

Introduction

Schistosomosis, also known as bilharziosis, stands as a parasitic helminthic infection induced by blood-dwelling platy helminth flukes attributed to the genus *Schistosoma* [1]. This pervasive global public health concern and prevalent tropical parasitic affliction affect approximately 300 million individuals worldwide. Of this afflicted population, 60% exhibit symptomatic manifestations, while 10% grapple with severe forms of the disease [2]. Despite notable strides in control measures, efficacious antischistosomal medications, and enhancements in socioeconomic conditions and health education, the persistent menace of schistosomosis endures. Contributing factors to its continual expansion encompass the industrialization of developing nations, population migrations, and the propagation of the disease through travel, particularly in tourism-dense endemic areas [3].

Within the spectrum of schistosomosis, neuroschistosomosis emerges as a distinctive entity denoting the infiltration of the central nervous system (CNS) by *Schistosoma* species. The neurological involvement in this context is further subcategorized into cerebral and spinal manifestations, the latter being the focal point of our comprehensive review [4]. Neuroschistosomosis, despite its severity, contends with being a neglected and under-recognized disorder. The prognosis hinges significantly on early detection and the institution of aggressive therapeutic interventions [5]. This review aims to elucidate the intricacies of spinal involvement in this parasitic infection, delving into its pathophysiology, clinical presentations, diagnostic modalities, and treatment strategies.

History

Schistosomosis, an enduring ailment, has woven its historical tapestry in tandem with the journey of humankind. Evidential threads extracted from ancient Egyptian papyri and Assyrian medical texts firmly establish the antiquity of this parasitic affliction. Moreover, biblical passages poignantly allude to an epidemic reminiscent of schistosomosis, with its pervasive impact extending to Mesopotamia, a depiction framed as a "curse". In the annals of the modern era, the formal delineation of schistosomosis (bilharziosis) in humans can be traced back to the pivotal year of 1851. Theodor Maximilian Bilharz, conducting pioneering work at Kasr-al Aini hospital in Cairo, Egypt, authored the inaugural description of this malady. His meticulous observations marked a crucial milestone, casting a spotlight on a disease that had hitherto eluded

Table 1. Milestones in *Schistosoma* development

Year	Milestone and discovery
7000 BC	In the bible, there are passages ascribable to a curse that could resemble schistosomosis. Joshua ordered to kill each inhabitant of Jericho apart from Rahab and her family. The deficit was believed to be associated with infected well water.
5800–4000 BC	Based on studies conducted on human skeleton remains, in Tell Zeiden in Syria, demonstrated the evidence of a terminal spined schistosomes from the pelvic sediment.
2494–2345 BC	Schistosomosis has spread to Egypt as a result of the fifth dynasty of Pharaohs.
1500 BC	The AAA disease was mentioned in several Egyptian medical papyri describing a disease characterized by discharge from penis. The disease is most likely schistosomosis.
1580	Prospero Alpini, Italian physician and botanist, noticed an oddly high incidence of hematuria in Egypt.
1798	Renault, a French physician with Napoleon's campaign, described Egypt as the only country where men menstruate. He also described the symptoms of the disease and examined the bladder from soldiers.
1847	Yoshino Fuji, Japanese physician working in Numakuma county, reported signs of Katayama syndrome in Hiroshima prefecture.
1851	Discovery of *Schistosoma* by a German parasitologist (Theodor Maximilian Bilharz). He found a trematode inside the mesenteric vein of young Egyptians.
1858	The word schistosomiasis was proposed by David Weinland because of the male worm morphology. It comes from the union of two Greek words "schistos" that means "split" and "soma" that mean "body".
1859	Thomas Spencer Cobbled named *Bilharzia* as a generic term for the parasite and discovered *Bilharzia magna* in a west African monkey.
1897	Robert Thomson Leiper, Scottish physician, understood the complete cycle of *Schistosoma* species with the recognition of snails as intermediate host.
1902	Patrick Manson discovered the spread of schistosomosis in central America.
1904	Fujiro Katsurada, Japanese professor of pathology, described the hepatic involvement in schistosomosis. He examined a cat in Yamanashi prefectures and found 32 parasites, including 5 pairs, in the portal vein. He also named a new species (*S. japonicum*) and noted this species eggs in the faeces of his patients.
1907	Luigi Sampon proposed a new species as *S. mansoni*, named after Patrick Manson as the parasite responsible for the lateral spined eggs.
1910	Marc Armand Ruffer found calcified eggs in the kidneys of Egyptian mummies of the 20th dynasty (1250 BC).
1915	Robert Thomson Leiper distinguished between *S. mansoni* and *S. haematobium* by their morphology, egg type, and snail host.
1990	Using enzyme-linked immunosorbent assay, Deelder et al. detected *Schistosoma* circulating anodic antigen in cheek, gut, and shin of Egyptian mummies (known to be infected with *S. haematobium*.
2014	Molecular biology techniques started to be used to confirm schistosomosis in mummies utilizing PCR primers. These techniques are suitable for direct detection of ancient DNA.

comprehensive understanding. This historical genesis not only reflects the longstanding coexistence of schistosomosis with humanity but also underscores the continual quest for unraveling its complexities across the epochs [6]. Table 1 describes milestones in *Schistosoma* development.

Epidemiology

Schistosomosis, ranking as the second most prevalent human parasitic infection of significant public health concern and disease burden, closely follows malaria in global impact [7]. This insidious ailment contributes to a staggering 70 million disability-adjusted life years, maintaining its prominence as a formidable public health challenge, particularly in underdeveloped nations [8]. Its occurrence is intricately linked to factors such as poor hygiene, poverty, and substandard housing conditions. Recognizing its profound implications, international entities, including the United Nations International Children's Emergency Fund (UNICEF), United Nations Development Programme (UNDP), the World Bank, and the World Health Organization (WHO), have collectively directed efforts toward its control [9].

WHO estimates underscore the magnitude of schistosomosis, affecting around 300 million individuals globally, with an additional 800 million at risk of infection. Its endemic presence spans 76 countries, with a notable concentration in Africa, where 46 nations bear the burden, hosting 94% of all schistosomosis infections and exposing 85% of the world's population to potential risks. The annual mortality toll, currently standing at 250,000 cases, may indeed be conservative. Furthermore, schistosomosis's linkage with heightened horizontal transmission of the Human Immunodeficiency Virus (HIV) in endemic regions adds another layer of complexity to its public health impact [10].

With an age-related pattern characterized by a convex curve, schistosomosis exhibits an increasing prevalence in childhood, reaching a zenith in adolescence, and gradually waning in adulthood [11]. Remarkably, less than 5% of the afflicted population progresses to manifest CNS symptoms, with cerebral complications overshadowing spinal manifestations. Autopsy findings in endemic regions disclose *Schistosoma* species in up to 28% of examined brains, underlining the pervasiveness of this parasitic intrusion into neurological tissues. Spinal schistosomosis, accounting for 5.6–50% of admissions due to non-traumatic spinal cord myelopathy in endemic areas, further emphasizes its

Table 2. Characteristics of the most common *Schistosoma* species

Species	Location	Geographical distribution	Egg characteristics
S. mansoni	Intestinal	Africa, the Middle East, the Caribbean, Brazil, Venezuela, Suriname	Large (114–180 μm long by 45–70 μm width) with prominent lateral spine near the posterior end
S. haematobium	Urogenital	Africa, the Middle East, Corsica (France)	Large (110–170 μm long by 40–70 μm wide) with a conspicuous terminal spine
S. japonicum	Intestinal	China, Indonesia, the Philippines	Large (70–100 μm long by 55–64 μm wide). It is more rounded than other species with small and less conspicuous spine
S. mekongi	Intestinal	Several districts of Colombia and the Ioa, People Democratic Republic	Small (50–80 μm long by 40–65 μm wide). Similar to *S. japonicum* regarding other characteristics
S. intercalatum	Intestinal	Rain forest areas of central Africa, west of Africa	Similar to *S. haematobium* but longer with central bulge and a large terminal spine
S. guineensis	Intestinal	Rain forests of central Africa	Similar to *S. haematobium* and *S. intercalatum* with central bulge and a large terminal spine

clinical significance [12].

Neuroschistosomosis, when symptomatic, emerges as a severe disorder necessitating prompt diagnosis and intervention. Predominantly attributed to *S. japonicum* (cerebral), *S. mansoni* (spinal), and *S. haematobium*, reported cases underscore the critical importance of targeted management strategies in mitigating the impact of this parasitic neurological affliction [13].

Parasite

Human infections by *Schistosoma* involve six distinct species: *S. mansoni*, *S. haematobium*, *S. japonicum*, *S. intercalatum*, *S. mekongi*, and *S. guineensis*. Of these, *S. mansoni*, *S. haematobium*, and *S. japonicum* stand as the most extensively distributed, each asserting its presence in diverse geographical regions (Table 2). Within endemic zones, a noteworthy phenomenon unfolds, wherein individuals may concurrently harbor multiple *Schistosoma* species, leading to heightened infection intensities compared to those with singular infections. This nuanced understanding of co-infections contributes to the intricate dynamics of schistosomal parasitism and underscores the need for targeted approaches in regions characterized by multiple endemic species [14].

Life cycle

The life cycle of *Schistosoma* species unfolds with intricacies marked by distinct features, including sexual dimorphism, the absence of a muscular pharynx, and the production of non-operculated eggs. Human infection occurs when an individual encounters contaminated freshwater, allowing the parasite to penetrate the skin. Freshwater snails, specifically *Pulinuss* species, *Biomphalaria* species, and *Oncomelania* species, serve as intermediate hosts, releasing numerous infective schistosomal larvae into the freshwater environment. Following sexual reproduction within snails, thousands of larvae, now cercariae, are prompted for release into water upon exposure to light. These motile cercariae, characterized by a forked tail and measuring 0.1–0.2 mm in length, employ proteolytic enzymes and mechanical activity to penetrate human skin within 12–24 hours after emerging from the snail [15].

Upon skin penetration, the cercaria undergoes a rapid transformation into a schistosomulum, subsequently invading the lymphatic system and entering nearby veins. Passively carried by the bloodstream to the right heart, the schistosomulum traverses pulmonary capillaries and lungs before breaking out and re-entering the bloodstream. Transported to the liver through the portal system and splanchnic vasculature, the schistosomulae mature into a mating pair of female and male worms, each measuring 1–2 cm in length and 0.3–0.6 mm in width. Adult parasites, feeding on blood, exhibit a unique gynaecophoric canal in the muscular male worm, which envelops and transports the slender female against the blood flow to the hepatic portal vein and its branches surrounding the intestine [16].

In the case of *S. mansoni* and *S. japonicum*, worms migrate to distal mesenteric veins to lay eggs crossing the intestinal wall, eventually reaching faeces. *S. mansoni* prefers inferior mesenteric vein tributaries, while *S. japonicum* favors superior mesenteric vein tributaries. Conversely, *S. haematobium* occupies bladder veins for egg laying in urine. Egg deposition, commencing within 25–30 days, results in excretion through stool (or urine), leading to hatching in freshwater. The miracidium, a ciliated larva, actively swims to penetrate the tegument of a snail intermediate host. Through asexual multiplication, hundreds of cercariae emerge within 4–6 weeks [17].

In spinal schistosomosis, eggs laid in the hepatic portal system travel through the valveless paravertebral venous plexus of Batson to reach the lower spinal cord and diffuse throughout the central nervous system via normal cerebrospinal fluid circulation. Alternatively, adult worms may, more rarely, migrate directly to the CNS, thus completing the intricate life cycle of *Schistosoma* species [18].

Pathogenesis

In spinal cord schistosomosis, the pivotal pathogenic role is attributed not to the worms themselves but to the retained eggs, initiating a robust inflammatory response that constitutes the primary pathology. This response, characterized by a delayed hypersensitivity reaction of the host, is orchestrated by immunogenic glycoproteins and proteolytic enzymes actively secreted by miracidia within the eggs. Remarkably, these miracidia can mature over a span of several days, remaining viable for up to three weeks. The resultant antigens, originating from both live and dead eggs, trigger a

cascade of events, inducing eosinophil-mediated inflammation and the formation of granulomas. These necrotic-exudative granulomas reach their peak size approximately one week after the deposition of eggs. Within this intricate immunologic milieu, CD4+ T-helper cells specific to schistosome ova antigens play a crucial role, contributing to the development of periovular granulomas. The cumulative impact of thousands of eggs and the substantial granuloma, situated within the spinal cord, elucidates the clinical manifestations observed in spinal cord schistosomosis. The mass effects generated by this inflammatory response underscore the pathophysiological mechanisms governing the neurological findings, ultimately centering around eosinophil-mediated inflammation, cellular immune reactions surrounding the eggs, and the formation of granulomas. In essence, the intricate interplay between the host's immune system and schistosomal eggs within the spinal cord precipitates the distinctive clinical features observed in spinal cord schistosomosis [19].

Pathology

The gold standard for diagnosing spinal cord schistosomosis involves biopsy of nervous tissue, followed by light microscopy to discern the presence of granulomas or schistosomal eggs (Figure 1). However, this procedure is often circumvented due to the inherent risks and potential complications associated with its invasiveness. Histopathological examination of spinal cord tissues reveals schistosomal granulomas in diverse evolutive phases. Periovular granulomas, a hallmark of the pathological landscape, exhibit a necrotic center containing either individual eggs or large clusters surrounded by epithelioid cells, giant cells, and lymphocytes. This core is further encapsulated by an outer layer comprising plasma cells, eosinophils, and fibroblasts. Notably, histological analysis may reveal additional features such as perivascular cuffing or vasculitis, venous thrombosis, and hemorrhages. The lower thoracic and lumbosacral regions of the spinal cord emerge as the most common sites for intramedullary granulomas. Additionally, granulomas may manifest on the spinal cord's surface and the roots of the cauda equina. The comprehensive histopathological examination thus provides crucial insights into the nuanced nature of the pathological changes associated with spinal cord schistosomosis [20].

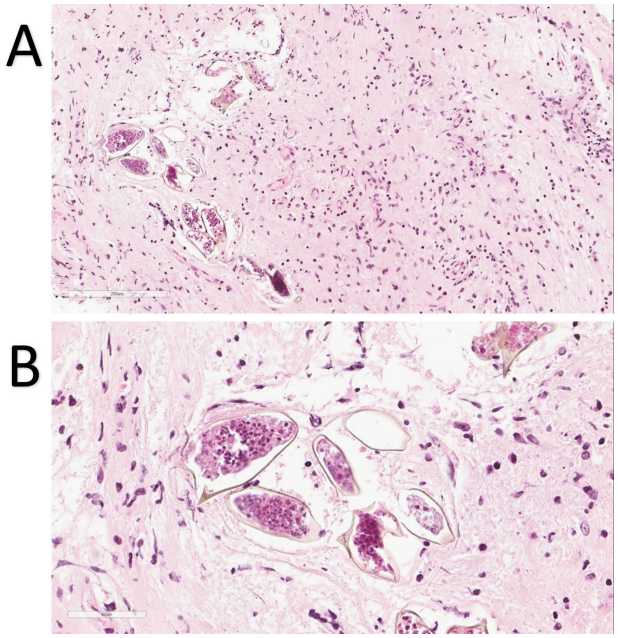

Figure 1. A. Intermediate power view of the hematoxylin-and-eosin-stained section from the biopsy depicting the deposition of multiple Schistosoma eggs (left-side of the photomicrograph) within a reactive spinal cord parenchyma. B. High-power view further showing the details of the Schistosoma eggs; some of which with a lateral spine

Clinical features

Spinal schistosomosis emerges as a significant etiology of acute myelopathy in regions endemic to the disease. Often overlooked, especially in the pediatric population, it warrants consideration in the differential diagnosis of acute paraplegia. Predominantly attributed to *S. mansoni* and *S. haematobium*, with sporadic instances involving *S. japonicum*, this condition predominantly affects children and young adults following freshwater exposure. The onset of spinal cord dysfunction varies, manifesting anywhere from days to years after exposure. Typically occurring in the early stages of infection, spinal schistosomosis presents with minimal systemic symptoms and lacks clinical evidence of hepato-splenic involvement, making diagnosis challenging until spinal cord symptoms become apparent [21].

Clinically, four distinct forms have been delineated: myelitic, granulomatous (pseudo-tumoral), radicular or myeloradicular, and vascular (syndrome of anterior spinal artery occlusion) [22].

The granulomatous form, characterized by an intense immunological reaction around eggs,

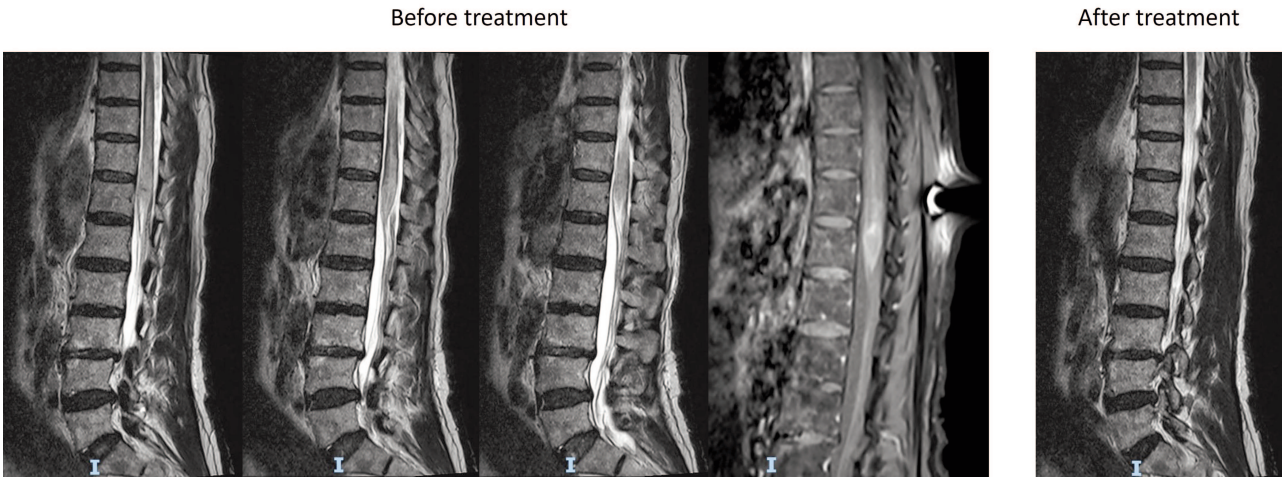

Figure 2. MRI of the whole spine showing a swollen lower dorsal region with abnormal low T1 high T2 signal intensity opposite to the level of D12 up to the level of D10 (left images before treatment) and a repeat MRI of the whole spine showing regressive course of the cord expansion and abnormal high T2 signal seen at the distal 3 segments of the spinal cord (right image after treatment)

Figure 3. MRI of the whole spine showing intramedullary high signal intensity involving the spinal cord from the level of T5 until the conus medullaris with edematous and enlarged spinal cord associated with with nodular and patchy enhancement involving distal spinal cord from T11 level including the conus medullaris and the cauda equina nerve roots. The images are T1-weighted (left column), T2-weighted (middle column), and contrast-enhanced T1-weighted (right column)

induces gliotic and fibrotic changes, leading to the formation of intrathecal granulomatous masses. These changes may occur either intradurally or extradurally, with the lower thoracic region being the most commonly affected segment, likely due to heightened systemic-vertebral anastomosis. It typically presents as a subacute expansive tumor-like lesion [21,22].

The myelitic form, non-granulomatous in nature, manifests as acute and progressive inflammatory myelitis with arachnoiditis, primarily affecting the lower thoracic spinal cord, conus medullaris, and spinal roots. Clinical features include asymmetric lower limb weakness, sensory symptoms, and sphincter dysfunction [21,22].

The radicular form, often accompanying the granulomatous form and arachnoiditis, presents with radicular pain and may progress to chronic asymmetric myeloradiculopathy. Isolated radicular involvement is considered exceptional [21,22].

The vascular form, resulting from spinal artery occlusion, is exceedingly rare, with limited reports in the literature. Patients may present with spinal cord infarction [21,22]. In essence, the clinical spectrum of spinal schistosomosis encompasses diverse manifestations, emphasizing the importance of recognizing its varied presentations for accurate diagnosis and timely intervention [21,22].

Differential diagnosis

The differential diagnosis of spinal schistosomosis encompasses a range of infectious

and noninfectious causes. Among infectious etiologies, considerations include tuberculosis, paragonomiosis, echinococcosis, syphilis, dracunculosis, and infection with human T-cell lymphoma virus type 1. Noninfectious causes that warrant consideration in the differential diagnosis encompass trauma, neoplasms, such as Burkitt's lymphoma, idiopathic adhesive arachnoiditis, and lathyrism, induced by the heavy consumption of the chick-ling pea. Accurate differentiation among these various conditions is crucial for timely and targeted therapeutic interventions in cases presenting with symptoms resembling spinal schistosomosis [23].

Diagnosis

Diagnosing spinal cord schistosomosis presents challenges due to nonspecific neurological symptoms at onset and the absence of detectable eosinophilia. High antibody levels, while indicative of exposure to *Schistosoma* species, do not confirm the schistosomal etiology of neuroschistosomosis and may yield serological cross-reactions with other helminths [24].

Magnetic resonance imaging of the spinal cord is a pivotal diagnostic tool, offering high sensitivity to detect abnormalities. Common findings include swelling, particularly in the lower thoracic segment and conus medullaris, observed as hyperintense T2-weighted images and equal or hypointense T1-weighted images. Gadolinium administration may reveal irregular contrast-enhancing patches and linear radicular enhancement of nerve roots, with rare reports of cyst-like lesions and signs of arachnoiditis [24,25] (Figure 2,3).

Direct parasitological methods, visualizing *Schistosoma* eggs in stool or urine samples, remain the standard for testing, yet challenges exist in timing, sample collection, and laboratory expertise. Full blood count often reveals eosinophilia following freshwater exposure. Cerebrospinal fluid analysis may show pleocytosis, increased protein, raised IgG index, and positive Enzyme Linked Immunosorbent Assay (ELISA) test for detection of antibodies to schistosomes [24].

Schistosomal serology, though not universally accepted, includes tests like Falcon assay screening test, ELISA, fluorescent microscopy immunoassay, and immunoblot testing, detecting specific antibodies against *Schistosoma* antigens. Urine-based polymerase chain reaction assays offer a sensitive and specific method in endemic areas [24].

A definitive diagnosis, however, necessitates spinal cord biopsy, revealing *Schistosoma* ova in various stages, inflammatory reactions, granuloma formation, and demyelination near the ova. Despite these diagnostic approaches, the complexity of spinal cord schistosomosis underscores the need for a comprehensive evaluation considering clinical, radiological, and parasitological aspects [24].

Treatment

The treatment approach for spinal schistosomosis involves a combination of medical therapy, corticosteroids, and, in certain cases, surgical intervention. Early diagnosis is crucial to prevent severe disability [5,13,21].

Praziquantel, an oral drug, is a cornerstone of medical therapy, exhibiting high effectiveness and safety. Administered at doses ranging from 40–60 mg/kg/day in two divided doses, the treatment duration varies from 1–14 days. Re-examination of feces and urine one month post-treatment assesses efficacy, especially in regions with potential resistance [5,13,21].

Artemether, an antimalarial drug, is effective against schistosomulae during the initial weeks of life. Some experts recommend a combination of praziquantel and artemether for their synergistic action against adult worms. Oxamniquine is another schistosomicidal drug effective against S. mansoni with a dose of 30 mg/kg daily for 2 days [5,13,21].

Corticosteroids, like intravenous methylprednisolone and oral prednisone, accompany medical therapy to address acute allergic reactions, mass effects, and hypersensitivity linked to the death of adult worms. Intravenous methylprednisolone at a dose of 15–20 mg/kg (maximum dose 1 gram) over 5 to 7 days followed by oral prednisone at a dose of 1.5–2 mg/kg per day for 3 weeks have been suggested. Other authors use oral prednisone at a dose of 1–1.5 mg/kg per day for 3 weeks followed by progressive and gradual reduction [5,13,21].

Antiepileptic drugs, such as levetiracetam or sodium valproate, may be considered if needed for long-term therapy without interfering with praziquantel metabolism [5,13,21].

Surgical intervention, including decompressive laminectomy, mass excision, and root liberation, is reserved for cases unresponsive to medical therapy. A therapeutic trial with praziquantel and corticosteroids is advised in suspected cases even

without direct diagnostic proof [5,13,21].

In severe cases with disabling paraplegia, multidisciplinary rehabilitation is essential, incorporating wheelchair devices, intermittent bladder catheterization, prevention of pressure sources and venous thrombosis, re-education for intestinal and sexual dysfunction, and treatment for spasticity and pain. The comprehensive treatment strategy underscores the importance of timely and integrated management to mitigate the impact of spinal schistosomosis [5,13,21].

Prognosis

After treatment with praziquantel and corticosteroids, clinical improvement in spinal schistosomosis is typically rapid, often manifesting within days, though delayed responses may occur over several weeks. A poor response could be attributed to irreversible structural cord damage or delayed diagnosis and treatment. Prognostically, around one-third of patients experience complete recovery, another third recover with residual deficits, while the remaining third either remain unchanged or deteriorate. Notably, patients with necrotic myelitis generally have a less favorable prognosis. Regular follow-up assessments are crucial to monitor outcomes and address any evolving clinical considerations [21,26].

Conclusion

Spinal cord schistosomosis, though rare, represents a significant and potentially treatable etiology of myelopathy. Its spectrum ranges from asymptomatic egg deposits to severe acute transverse myelitis featuring hemorrhage and necrosis. Clinical presentations commonly include acute paraplegia and cauda equina syndrome, particularly in *S. mansoni* myelopathy. The preferred treatment comprises a combination of praziquantel and corticosteroids. This therapeutic approach should be considered for individuals residing in endemic areas, especially when accompanied by peripheral eosinophilia and identification of ova in stool, urine, or rectal biopsy. Laminectomy is reserved for cases where biopsy and decompression are deemed necessary. Vigilant consideration of spinal schistosomosis in endemic regions, along with timely and comprehensive management, is pivotal for achieving optimal outcomes in affected individuals.

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