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Guidelines for the diagnosis and treatment of schistosomal myeloradiculopathy

Orientações sobre o diagnóstico e tratamento da mielorradiculopatia associada à esquistossomose

José Roberto Lambertucci[1], Luciana Cristina dos Santos Silva[1] and Ronaldo Santos do Amaral[2]

ABSTRACT

Schistosomal myeloradiculopathy is the most severe and disabling ectopic form of Schistosoma mansoni *infection. The prevalence of SMR in centres in Brazil and Africa that specialise in attending patients with non traumatic myelopathy is around 5%. The initial signs and symptoms of the disease include lumbar and/or lower limb pain, paraparesis, urinary and intestinal dysfunctions, and impotence in men. The cerebrospinal fluid of SMR patients shows an increase in protein concentration and in the number of mononuclear cells in 90% of cases; eosinophils have been reported in 40%. The use of magnetic resonance imaging is particularly valuable in the diagnosis of Schistosomal myeloradiculopathy. The exclusion of other myelopathies and systemic diseases remains mandatory. Early diagnosis and treatment with steroids and schistosomicides provide a cure for most patients, whilst delayed treatment can result in irreversible physical disabilities or death. To improve awareness concerning Schistosomal myeloradiculopathy amongst public health professionals, and to facilitate the control of the disease, the Brazilian Ministry of Health has launched a program of education and control of this ectopic form of schistosomiasis. The present paper reviews current methods for the diagnosis of SMR and outlines protocols for treatment of the disease.*

Key-words: *Schistosomiasis. Neuroschistosomiasis. Myeloradiculopathy. Steroids. Praziquantel.*

RESUMO

A mielorradiculopatia esquistossomótica é a forma ectópica mais grave da infecção pelo Schistosoma mansoni*. A prevalência da mielorradiculopatia esquistossomótica em centros médicos no Brasil e em África, especializados no atendimento de pacientes com mielopatia, encontra-se em torno de 5%. Os sintomas e sinais iniciais da doença incluem: dor lombar e/ou dor em membros inferiores, paraparesia, disfunções urinária e intestinal, e impotência no homem. A análise do líqüor destes pacientes revela aumento na concentração de proteínas e no número de células mononucleares em 90% dos casos; a presença de eosinófilos foi documentada em 40%. O uso rotineiro da ressonância magnética tornou-se obrigatório na definição diagnóstica. A exclusão de outras mielopatias e doenças sistêmicas é mandatória. O diagnóstico precoce e o tratamento com corticoesteróides e esquistossomicidas curam a maioria dos pacientes, enquanto o atraso em iniciar o tratamento resulta em seqüelas irreversíveis ou morte. Para melhorar a percepção da importância da mielorradiculopatia associada à esquistossomose, o Ministério da Saúde do Brasil lançou programa de controle dessa forma ectópica da esquistossomose. Nesta revisão, descrevem-se os métodos diagnósticos atuais para o diagnóstico e os protocolos para o tratamento da doença.*

Palavras-chaves: *Esquistossomose. Neuroesquistossomose. Mielorradiculopatia. Esteróides. Praziquantel.*

Schistosomal myeloradiculopathy (SMR) is the most severe and disabling ectopic form of *Schistosoma mansoni* infection. In order to prevent serious and irreversible lesions, particularly, in young and productive individuals, early diagnosis and subsequent treatment of SMR are critical. The diagnosis of SMR is based upon: (i) the presentation of neurological symptoms and signs resulting from lesions of the spinal cord; (ii) the demonstration of schistosomal infection using microscopy and serological techniques, and (iii) the exclusion of other causes of myelopathy. The disease is characterised by a triad consisting of lumbar/lower limb pain, alterations in motor function (paraparesis) and/or altered sensitivity in the lower limbs, and urinary dysfunction. The presentation of such symptoms should alert health personnel to the possible emergence of SMR.

Since the diagnosis of SMR may be inferred from clinical and laboratory tests, and the treatment is essentially clinical,

1. Clínica de Doenças Infecciosas e Parasitárias, Faculdade de Medicina da Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil. 2. Secretaria de Vigilância em Saúde, Ministério da Saúde, Brasília, DF, Brazil.
Financial support: CNPq (Conselho Nacional de Pesquisas) and the Secretaria de Vigilância em Saúde, Ministry of Health of Brazil.
Address to: José Roberto Lambertucci. Faculdade de Medicina/UFMG, Av. Alfredo Balena 190, 30130-100 Belo Horizonte, MG, Brazil.
email: lamber@uai.com.br
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accessibility to specific propaedeutics and a greater awareness concerning the disease could prevent unnecessary surgical laminectomy. Moreover, the advent of novel diagnostic techniques, particularly magnetic resonance imaging (MRI) of the spinal cord, has facilitated the diagnosis of SMR and, consequently, the number of identified cases has increased considerably[1 3 14].

Despite such advances, however, the prevalence of SMR in endemic areas is underestimated. Furthermore, the epidemiological importance of the disease is obscured by a number of factors including: (i) the disease is not regularly notified to the appropriate health authorities, (ii) the clinical symptoms are often confusing, and (iii) access to expensive methods of diagnosis is limited. Such obstructing factors conceal the actual reality and may result in serious consequences for individual sufferers.

In Brazil, SMR is caused by *Schistosoma mansoni* and it is estimated that around 6 million people are infected. In an attempt to manage the situation, the Brazilian Ministry of Health launched a Program of Control of Schistosomal Mieloradiculopathy under the auspices of the General Coordination of Transmittable Diseases. As part of the program, a guide entitled "*Guidelines for the Epidemiological Assessment and Control of Schistosomal Myeloradiculopathy*" was prepared (a Portuguese version may be accessed by internet: http://portal.saude.gov.br/portal/arquivos/pdf/guia_mielo_esquisto.pdf. The purpose of this document was to call the attention of health professionals to the epidemiology, diagnosis and treatment of SMR, and to provide scientific evidence in support of the augmentation of public funds for the control of the disease. In furtherance of the aims of the Control Program, the present paper reviews current methods for the diagnosis of SMR and outlines protocols for diagnosis and treatment of the disease.

EPIDEMIOLOGY

The three main species of schistosomes: *Schistosoma mansoni*, *Schistosoma haematobium* and *Schistosoma japonicum* can affect the central nervous system (CNS). The deposition of eggs in the nervous tissue provokes an inflammatory response that can be asymptomatic or symptomatic. Infections with *Schistosoma mansoni* and *Schistosoma haematobium* produce myeloradicular damage, whilst infection with *Schistosoma japonicum* results in lesions in the brain.

The first review of ectopic schistosomiasis was published by Faust[5] who reported that 8 of the 82 cases that had been described at that time presented signs of spinal cord lesions, and that 3 of these patients were infected with *Schistosoma mansoni.* Significantly, this author stressed the importance of attracting medical attention to the problem and of the need to establish an accurate diagnosis, principally because of the high number of people affected and the serious consequences of the disease. Following this report, a number of other publications emphasised the importance of the association between SMR and infection with *Schistosoma mansoni*. Scrimgeour and Gajdusek[16] pondered that schistosomiasis may be the cause of paraplegia in areas endemic for the disease. They also claimed that schistosomal infection was responsible for 1% of all non-traumatic paraplegia in Tanzania and was the presumed cause in 5% of other cases. Spina-França and co-workers[20] studied a population of 353 patients who had received care for non-traumatic and non-tumoural myelopathies in Brazil, and found that the prevalence of SMR was 5.6%. Carod-Artal and co-workers[3] reported the same prevalence of SMR amongst patients presenting inflammatory myelopathies at the Sarah Kubitscheck hospital in Brasília, the capital of Brazil.

Therefore, a significant number of myelopathies of unknown aetiology occurring in areas endemic for *Schistosoma mansoni* may be related to SMR. Currently, however, despite an increase in the number of scientific reports, the prevalence of SMR in such endemic areas remains unknown and it is suspected that many cases are not officially notified and that morbidity is seriously underestimated[4].

Schistosomal myeloradiculopathy apparently affects individuals of all ages, and cases have been reported in patients between 1 and 68 years old with the average age being 26 years[18]. The incidence of SMR is higher amongst males (69 to 83% of reported cases) than females, and this has been explained by man's higher occupational exposure. It is important to emphasise that the occurrence of SMR does not depend on the level of parasite load as in other severe forms of schistosomiasis, including the pulmonary and hepatosplenic forms. In contrast, the number of eggs per gram of faeces released by SMR patients is generally small. Since carriers of *Schistosoma mansoni* can readily move from endemic to non-endemic areas, the extension of the Program of Control to the whole country is justified.

PATHOGENESIS OF SCHISTOSOMAL MYELORADICULOPATHY

The mechanism of development of SMR is still unclear, although it is accepted that the inflammatory response of the host to eggs of the parasite that are trapped in the CNS is the determinant factor for the emergence of lesions[18]. An autoimmune process, activated by *Schistosoma* infection and leading to vasculitis and cerebral ischemia, is believed to contribute to the progress of the disease, but this hypothesis has yet to be confirmed.

The inflammatory response to schistosome eggs varies in intensity and ranges from the presentation of negligible clinical signs to the occurrence of granulomas or expanded masses in the CNS. Asymptomatic deposition of eggs of *Schistosoma mansoni* in the encephalic and medullar tissues is more common than the symptomatic form of the disease. The occurrence of eggs in the spinal cord in the absence of any inflammatory reaction has been confirmed by post mortem investigations. Myeloradiculopathy occurs with higher frequency in the acute and chronic intestinal forms of the disease[6 8].

The worms and eggs may be transported within the retrograde venous flow in the valveless Batson's vertebral venous plexus, which connects the portal venous system and the inferior vena cava to the spinal veins. Thus, the eggs reach the CNS either by *in situ* oviposition or by an embolus process. Such migration

mechanism may explain the high incidence of SMR-patients affected by inflammation of the lumbosacral region. Autopsy studies have shown that *Schistosoma japonicum* eggs are often found in the brain, whilst those of *Schistosoma mansoni* are found in the lower zones of the vertebral venous plexus.

CLINICAL EVALUATION

Schistosomal myeloradiculopathy may emerge in individuals lacking a previous clinical history or diagnosis of schistosomiasis, or it may appear many years after the intestinal manifestations of the disease. Rarely, patients with the hepatosplenic form may also present this complication of schistosomiasis[18,19].

During the early stages of infection, SMR sufferers frequently complain of lumbar pain (97.5% of cases), altered sensitivity of the lower limbs (97.5%) and urinary dysfunction (96.2%), and this is followed by lower limbs weakness and sexual impotence in men[14] . Within 15 days, the initial acute or subacute manifestations of the disease may evolve into a more complex clinical condition that is characterised by a series of neurological symptoms (Figure 1). Table 1 lists the neurological symptoms associated with fully developed SMR. Occasionally, the disease may develop more slowly and take months or even years to emerge. In some patients the lumbar/lower limb pain diminishes and finally disappears as other symptoms become more perceptible, whilst in other cases there is a spontaneous clinical recovery with recurring neurological manifestations.

Some studies have suggested that increased intra-abdominal pressure caused by physical exertion may trigger the development of SMR. Neurological examination shows that lesions are frequently located in the low thoracic and lumbosacral regions, the medullary cone and the cauda equina. Isolated schistosomal lesions may also appear in the cervical spinal cord[2,19]. Paraplegia together with muscular flaccidity and lack of reflexes, urinary retention and loss of sensitivity to touch, heat and pain are predominant when the spinal cone and the cauda equina are affected. Spasticity, alteration of superficial sensitivity and urinary incontinency occur when the higher portions of the spinal cord are affected. Thus, the most frequent abnormalities found during neurological examination are bilateral paraparesis and reduced/eliminated deep reflexes. Asymmetric alterations of motor or tactile functions resulting from injured spinal nerve roots are always highly suggestive of SMR.

Professionals working in emergency units, urologists, paediatricians, general clinicians and physiotherapists must be particularly alert to the possibility of SMR since they are often contacted by carriers of *Schistosoma* at the initial stages of the disease when neurological examination is normal but the characteristic clinical prodromic triad (lumbar/lower limb pain, paraparesis, and urinary dysfunction) may be present.

LABORATORY TESTS

Cerebrospinal fluid. Analysis of the CSF of SMR patients reveals normal glucose levels together with non-specific alterations including a slight to moderate increase in protein content (161.4 ± 191.9mg/dL on average) in 95% of cases, pleocytosis with a preponderance of lymphocytes (91.9 ± 113.8 cells/mm^3) in 91%, presence of eosinophils in 41-90%, and increased gamma globulin levels in 76%[9]. According to Andrade Filho and co-workers[1], the cellular content of CSF may be reduced as the clinical picture improves, but increased levels of protein remain in 66% of cases.

Table 1 - Principal symptoms associated with schistosomal myeloradiculopathy and their approximate frequencies.

Symptoms	Estimated percentage of cases
Lumbar and/or lower limb pain	97
Weakness of the lower limbs	90
Anaesthesia/hypoesthesia of the lower limbs	98
Paraparesis of the lower limbs	97
Bladder dysfunction	96
Intestinal dysfunction	90
Erectile dysfunction	74

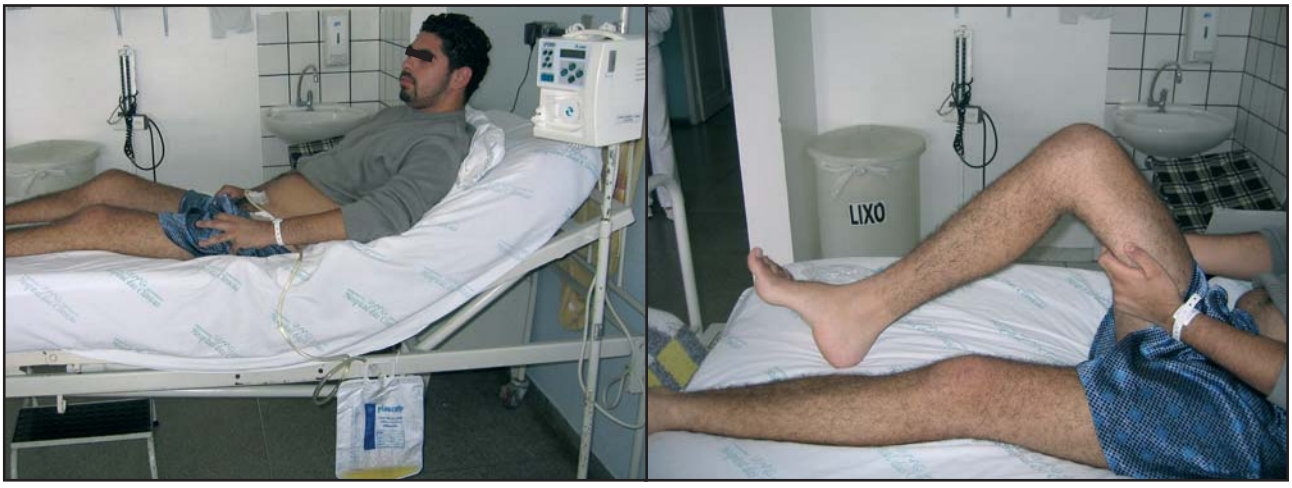


Figure 1 - A 33-year-old patient with schistosomal mieloradiculopathy. His disease presented with paraplegia, urinary and fecal retention. On the left, a cystostomy was necessary in order to drain urine from the urinary bladder. On the right, there is atrophy of the muscles of the lower limbs and he moves his right leg with the help of his hands.

Through the use of enzyme-linked immunosorbent assays (ELISA), indirect immunofluorescence (IIF) assays or haemagglutination tests, anti-*Schistosoma* antibodies can be identified in the CSF of 80-90% of SMR patients. Positive serology is considered to be reliable evidence of *Schistosoma* infection, as demonstrated by Livramento and co-workers[9] who confirmed the association between CSF syndrome (pleocytosis, mononuclear leukocytes and hyperproteinorrhachia) and positive immune reactions. In addition, in a study conducted between 1988 and 2002, involving 377 SMR-suspected patients, Moreno-Carvalho et al[10] concluded that *Schistosoma*-positive haemagglutination and IIF tests, associated with an increased number of inflammatory cells (> 4 cells/mm^3), hyperproteinorrhachia (> 40mg/dL) and the presence of eosinophils, are associated with a high probability of SMR.

The behaviour of cytokines and chemokines in the CSF of patients with SMR is under evaluation[21].

Imaging techniques. Myelography and myelotomography are abnormal in 63% of SMR patients. Such alterations are characterised by an increase in the diameter of the spinal cord, partial or complete obstruction of the vertebral canal, and thickening of the nerve roots of the cauda equina. The granulomatous form of the disease is most frequently diagnosed using these imaging methods since it is characterised by a dilation of the spinal cord[15 18 22]. Atrophy of the spinal cord may be observed in patients with long term illness.

Magnetic resonance imaging is a more accurate method that reveals abnormalities in the spinal cord in practically all SMR cases including those that cannot be detected by myelography and myelotomography (Figure 2). The use of this technique has been reported in one prospective and two retrospective studies [11 15 19] as well as in case reports. The main alterations revealed by MRI are: enlargement of the spinal cord in T1-weighted images, and hyperintensity of signal on T2-weighted images in the affected region indicating an increase in water content (oedema). In T1-weighted sequences heterogeneous diffuse granular enhancement (impregnation) following gadolinium injection is observed in most cases. Although MRI is a very sensitive method for the evaluation of SMR, the diagnostic alterations are not specific to this disease.

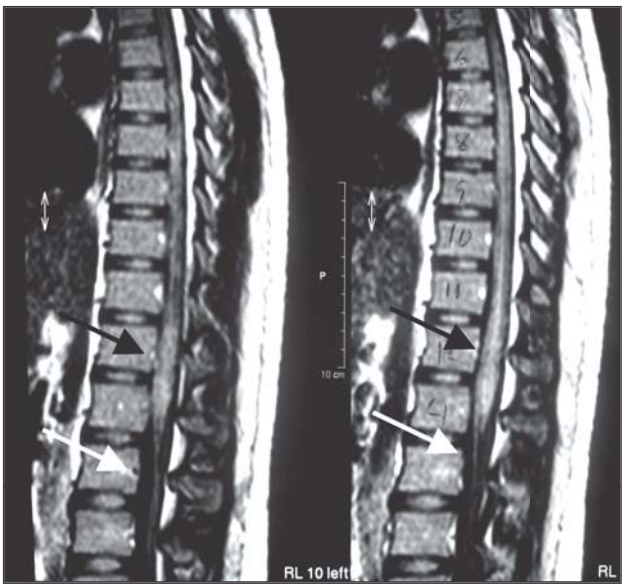


Figure 2 - Magnetic resonance imaging of the patient in Figure 1a. Black arrows point to a dilated* conus medullaris. *This is a T1-weighted image with a diffuse granular impregnation of the spinal cord after endovenous gadolinium injection (whitish areas of the spinal cord). There is also impregnation of contrast of the* cauda equina *(white arrows).

Following treatment with schistosomicides and corticosteroids, the alterations observed by MRI disappear as the clinical condition of the patient improves. Occasionally, the neurological symptoms may return after stopping treatment, but as soon as treatment is re-started the signs and symptoms of SMR disappear.

Histological and immunological analyses. Exposure to *Schistosoma mansoni* can be established in most suspected SMR-patients by the presence of eggs in faecal, urine and/or tissue (rectal and urinary bladder mucosa, liver, skin and spinal cord fragments) samples. In other cases, infection can be determined by indirect immunological techniques that reveal the presence of anti-*Schistosoma* antibodies. Epidemiological information is also quite helpful (for example, a patient who comes from an endemic area or travelled to an area known to be endemic for shistosomiasis should be investigated more carefully). Positive anti-*Schistosoma* serology, however, does not constitute a definite evidence of *Schistosoma mansoni* infection because cross-reaction with other antigens, in particular those from other helminth infections, has been described. In addition, it is impossible to distinguish an active from an extinguished infection based on serologic results, since antibodies remain in the blood for long time after the individual is cured of schistosomiasis[12].

DIFFERENTIAL DIAGNOSIS

Differential diagnosis is required because other diseases and conditions present symptoms similar to SMR, and these include viral (acquired human immunodeficiency syndrome, human T-cell lymphotropic virus type 1, herpes simplex, hepatitis B and C viruses) or bacterial (syphilis, medullar abscesses, tuberculosis) myelitis, herniated lumbar disc, multiple sclerosis, medullar trauma, tumours, vitamin B12 deficiency, antiphospholipid syndrome, diabetic or autoimmune vasculitis, syringomyelia and neurocysticercosis. Moreover, when submitted to certain conditions such as radiation therapy or intrathecal injections, individuals may exhibit neurological symptoms similar to those of SMR (Table 2).

CLINICAL PROTOCOL FOR THE DIAGNOSIS OF SCHISTOSOMAL MYELORADICULOPATHY

The adoption of the following criteria may facilitate the presumptive diagnosis of SMR: (i) clinical evidence of neurological medullar lesions, (ii) evidence of exposure to *Schistosoma*, (iii) demonstration of CSF abnormalities, and (iv) exclusion of other illnesses. The complete protocol is summarised in Figure 3.

The limited access to expensive tests, helpful in the exclusion of other causes of myelitis, is one of the greatest problems encountered in the diagnosis of SMR. Biopsy of the spinal cord and

Table 2 - Differential diagnosis of schistosomal myeloradiculopathy and associated laboratory and imaging tests*.

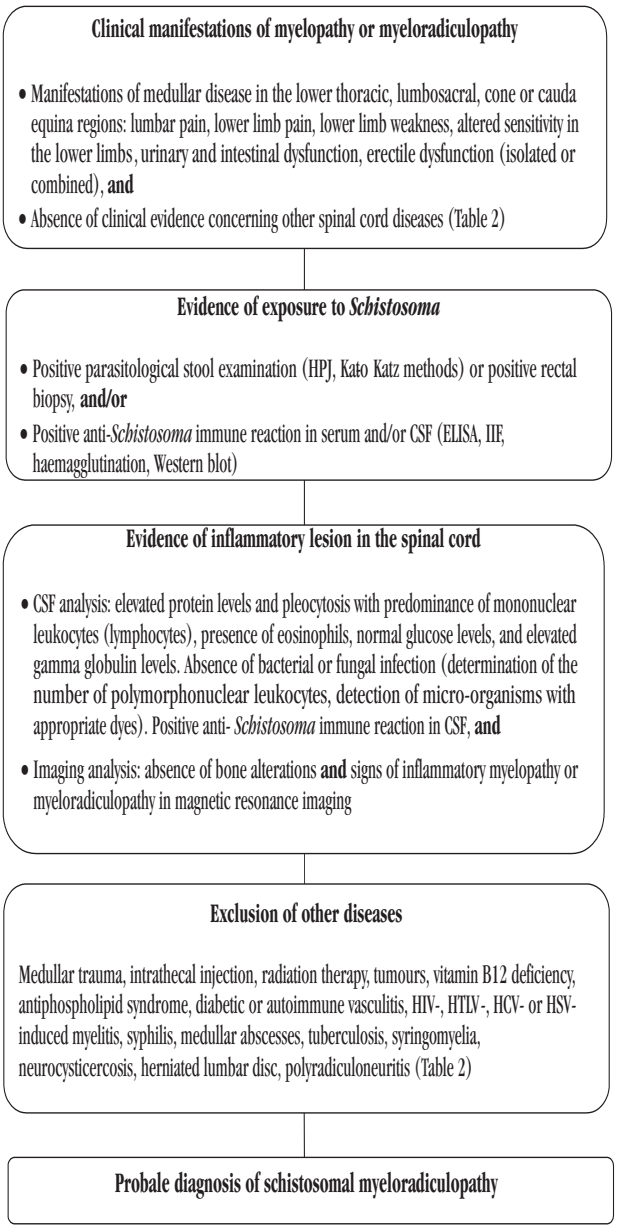
Differential Diagnosis	Laboratory and imaging techniques
Polyradiculoneuritis	CSF analysis, electroneuromyography
Hernia of the lumbosacral discs	MRI, electroneuromyography
Medullar trauma	radiography, tomography, MRI
Intrathecal injection of contrast medium or chemotherapy	clinical history
Radiotherapy	clinical history, diagnosis of neoplasia
Tumour	clinical history, tomography, MRI, bone scintillography , ultrasound of abdomen, radiography of thorax
Myelopathy induced by vitamin B12 deficiency	Vitamin B12 analysis, haemogram
Antiphospholipid syndrome	Anticardiolipin and lupic anticoagulant antibody analysis
Diabetic vasculitis	Clinical history, glycaemia, glycated haemoglobin analysis
Autoimmune vasculitis	Clinical history, ANA, ANCA
HIV-induced myelitis	Serum anti-HIV analysis
HTLV-1-induced myelitis	Serum anti-HTLV-1 analysis
HSV-induced myelitis	Serum anti-HSV (IgG, IgM) analysis
Syphilis	VDRL, FTA-abs
Medullar abscesses	Radiography, tomography, MRI, haemogram, reactive protein C, CSF analysis
Tuberculosis	Clinical history, culture and smear for bacteria, PCR of CSF, PPD, chest x-ray, x-ray of the spine and MRI
Hepatitis B and C	HBsAg, anti-HBs, total anti-HBc, anti-HCV analysis
Multiple sclerosis	MRI of brain and medulla, CSF analysis
Neurocysticercosis	MRI of brain and medulla
Syringomyelia	MRI of brain and medulla

*ANA: antinuclear antibody, ANCA: antineutrophil cytoplasm antibody, CSF: cerebrospinal fluid, FTA-abs: fluorescent treponemal antibody absorption, HIV: human immunodeficiency virus, HTLV: human T-cell lymphotropic virus, HCV: hepatitis C virus, HSV: herpes simplex virus, MRI: magnetic resonance imaging, PCR: polymerase chain reaction, PPD: purified protein derivative (tuberculosis skin test), VDRL: venereal disease research laboratory test.

demonstration of eggs of *Schistosoma mansoni* in the nervous tissue is the gold standard in the diagnosis of SMR. Biopsy is, however, an invasive procedure with complications that are far too serious to be used to diagnose a disease whose treatment is essentially clinical and presents a favourable prognosis. Sometimes spinal cord biopsy is the only way to diagnose schistosomiasis when, for instance, *Schistosoma mansoni* eggs cannot be found in faecal material or by rectal biopsy[17].

PROTOCOL FOR THE TREATMENT OF SCHISTOSOMAL MYELORADICULOPATHY

Schistosomal myeloradiculopathy therapy involves the use of schistosomicidal drugs, corticosteroids and/or surgery. Figure 4 summarises the complete protocol for the treatment of SMR. The



HPJ: Hoffman, Pons e Janer, CSF: cerebral spinal fluid, ELISA: enzyme linked immunosorbent assay, IIF: indirect immunofluorescence, MRI: magnetic resonance imaging, HIV: Human immunodeficiency virus, HTLV: Human T-cell Lymphotropic Virus, HCV: hepatitis C virus, HSV: virus herpes simples.

Figure 3 - Clinical protocol for the diagnosis of schistosomal myeloradiculopathy.

best response to treatment is achieved by using combinations of both schistosomicides and corticosteroids (Figure 5)[7 8 13 14 19]. By destroying the adult worm, schistosomicidal drugs interrupt the production of eggs and as a result eliminate the inflammatory reaction in the CNS. The corticosteroids reduce the inflammatory reactions in the areas surrounding the eggs that are responsible for the compression and destruction of nervous tissue. Whilst the duration of corticotherapy cannot be precisely defined, the benefit of its application has been fully established. Amelioration of symptoms has been observed immediately after administration of corticosteroids, although the discontinuity of treatment before six months may result in recurrence of the neurological manifestations. Surgical approaches should be reserved for patients suffering from acute paraplegia and obstruction of CSF

Treatment of schistosomal myeloradiculopathy

Prophylaxis of possible therapeutic complications

- **Ivermectin** 200μg/kg, body weight, single oral dose (patient > 5 years old) or **albendazol** 400mg a day, oral dose, during 3 days (children 2 to 5 years old), for treatment of possible strongyloidiasis
- **Ranitidine, cimetidine** or **omeprazol** for prophylaxis of gastroduodenal lesions following corticotherapy

Specific treatment of schistosomal myeloradiculopathy

- **Praziquantel** 50mg/kg, body weight (adults) or 60mg/kg, body weight (children < 15years old), divided into two oral doses at 4 h intervals for the treatment of schistosomiasis
- **Corticotherapy (prednisone)** 1mg/kg, body weight, single oral dose in the morning, during 6 months preceded (or not) by anti-inflammatory pulse therapy with **methylprednisolone** 15mg/kg, body weight per day, maximum daily dose of 1g, endovenous injection, during 5 days. Prednisone must be removed slowly, at the end of treatment, in order to avoid suprarenal insufficiency Removal of drug before the 6 month period is recommended only when patients are fully recovered from the neurological symptoms

Multidisciplinary approach

- Intermittent urinary bladder catheterism in the case of urinary retention
- Early prophylaxis, diagnosis and treatment of urinary infection
- Motor physiotherapy
- Prophylaxis and care regarding decubitus ulcers
- Psychotherapy and occupational therapy

Figure 4 - Protocol for the treatment of schistosomal myeloradiculopathy.

Figure 5 - The same patient 13 months later.

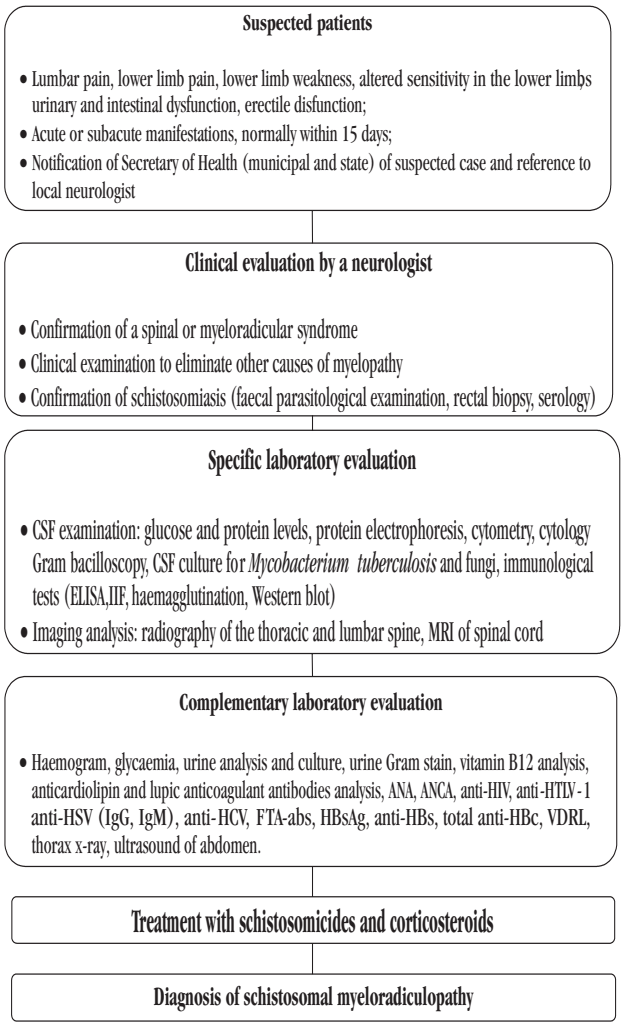
flow, as well as for those who do not respond to conventional treatment[2]. Even though surgery involves minimal manipulation (decompression, release of nerve roots and biopsy), patients often suffer undesirable consequences. For this reason, the decision to use surgery for diagnostic purposes should be made judiciously.

Ninety five percent of SMR patients that do not receive treatment either do not recover clinically or die. However, there is evidence that even delayed treatment (commencing up to 12 months after the beginning of symptoms) can result in the amelioration of the neurological problems associated with the disease[19].

Schistosomal myeloradiculopathy therapy is a multidisciplinary task and involves the participation of various professionals including nurses, physiotherapists, general clinicians, neurologists, psychologists and occupational therapists. Physiotherapy, for example, is indispensable for patients with impaired motor functions and must be maintained even after the termination of corticotherapy. Urinary infections are also very common and must be carefully investigated. Nurses must be attentive to the development of bed sores in patients confined to bed for long periods.

EPIDEMIOLOGICAL ASSESSMENT AND CONTROL OF SCHISTOSOMAL MYELORADICULOPATHY

Doctors or local health officers must notify the Secretary of Health, at the municipal and state level, of the existence of individuals presenting the prodromic triad. Additionally, parasitological examinations of faecal material by Hoffman, Pons and Janer or Kato-Katz methods should be requested. Patients should then be redirected to the regional neurologist who will carry out a clinical diagnosis of the disease and request the appropriate set of tests (CSF analysis, lumbar x-ray, computed tomography, MRI) depending on the resources available locally. If any difficulties arise in proceeding according to this protocol, the Centro de Treinamento e Referência em Doenças Infecciosas e Parasitárias (CTR-DIP) in Belo Horizonte (MG, Brazil) must be contacted. SMR patients are to be considered medical urgencies since diagnosis and treatment must be delivered as soon as possible in order to avoid irreversible neurological lesions and to increase the chances of a complete cure. Figure 6 summarises the protocol for the epidemiological control of SMR.



ANA: antinuclear antibody, ANCA: antineutrophil cytoplasm antibody, CSF: cerebrospinal fluid, FTA-abs: fluorescent treponemal antibody absorption, HIV: human immunodeficiency virus, HTLV-1: human T-cell lymphotropic virus type 1, HCV: hepatitis C virus, HSV: herpes simplex virus, MRI: magnetic resonance imaging

Figure 6 - Protocol for the epidemiological assessment and control of schistosomal myeloradiculopathy.

Schistosomal myeloradiculopathy is not classified under any specific international code (International Classification of Diseases - ICD). Schistosomal myeloradiculopathy patients treated in hospitals and outpatient clinics may thus be registered with a number of different codes including B65.1 (schistosomiasis caused by *Schistosoma mansoni* - intestinal schistosomiasis), G04 (encephalitis, myelitis and encephalomyelitis), and G54 (root and nerve plexus dysfunction). Such unintentional misinformation explains, partially, why SMR is a sub-notified disease. It is, therefore, important that SMR receives its own classification code, so, that the real prevalence of the disease can be determined. The development of strategies for controlling the disease and, ultimately, the efficient application of governmental funds, depend upon this essential epidemiological information.

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