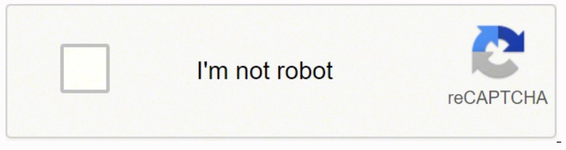

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Schistosomiasis mansoni pdf

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Involvement of Central Nervous System in the Schistosomiasis

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The involvement of the central nervous system (CNS) by schistosomes may or may not determine clinical manifestations. When symptomatic, neuroschistosomiasis (NS) is one of the most severe presentations of schistosomal infection. Considering the symptomatic form, cerebral involvement is almost always due to Schistosoma japonicum *and the spinal cord disease, caused by* S. mansoni *or* S. haematobium. *Available evidence suggests that NS depends basically on the presence of parasite eggs in the nervous tissue and on the host immune response. The patients with cerebral NS usually have the clinical manifestations of increased intracranial pressure associated with focal neurological signs; and those with schistosomal myeloradiculopathy (SMR) present rapidly progressing symptoms of myelitis involving the lower cord, usually in association with the involvement of the cauda equina roots. The diagnosis of cerebral NS is established by biopsy of the nervous tissue and SMR is usually diagnosed according to a clinical criterion. Antischistosomal drugs, corticosteroids and surgery are the resources available for treating NS. The outcome is variable and is better in cerebral disease.*

Key words: schistosomiasis - neuroschistosomiasis - central nervous system/parasitology - *Schistosoma mansoni* - *Schistosoma japonicum* - *Schistosoma haematobium*

ETIOLOGY AND EPIDEMIOLOGY

The term neuroschistosomiasis (NS) refers to the symptomatic or asymptomatic involvements of the central nervous system (CNS) by schistosomes. When associated with clinical symptoms, it is one of the most severe presentations of schistosomal infection. NS can be caused by *Schistosoma japonicum*, *S. mansoni*, and *S. haematobium*. Considering the symptomatic form, the last two species are almost always associated with a myeloradicular syndrome and the first species, with cerebral disease.

Symptomatic cerebral NS has been recorded in about 2-4% of individuals infected with *S. japonicum* (Watt et al. 1986). On the other hand, this form of presentation is very rare in association with the other two species. Schistosomal myeloradiculopathy (SMR) is less frequent than cerebral disease. There are around 500 cases reported since the description of the entity in 1930. Although SMR is considered a rare form of NS, its prevalence is unknown and some authors believe that this entity has been underdiagnosed (Joubert et al. 1990, Haribhai et al. 1991, Ferrari 1997, Silva et al. 2003). This possibility is reinforced by the greater number of cases published as knowledge of the disease spreads and by the relatively large number of patients seen by some investigators during a short time (Asano 1992, Ferrari 1997, Peregrino et al. 2002). *S. mansoni* is the species responsible for the great majority of the reported cases of SMR (Ferrari 1999).

As demonstrated by necropsy studies (Scrimgeour & Gajdusek 1985, Gonçalves et al. 1995), asymptomatic deposition of schistosomal eggs in the more highly vascular cerebral structures is more frequent than the symptomatic forms of NS.

PATHOGENESIS

Several aspects of the pathogenesis of NS are unknown, although available evidence suggests that the lesions seen in the CNS depend basically on the peesence of parasite eggs in the nervous tissue and on the host immune response. The eggs can reach the CNS at any time of the infection; however, in the great majority of the cases associated with neurological symptoms, the involvement of the CNS occurs during the evolution of the infection to its chronic phase or concomitantly with the less severe chronic forms (i. e., intestinal and hepatointestinal forms – for *S. mansoni* and *S. japonicum*, and urinary forms without obstructive uropathy – for *S. haematobium*). On the other hand, asymptomatic NS is much more common in association with the more severe chronic forms of *S. mansoni* and *S. haematobium* infection (i. e., hepatosplenic and cardiopulmonary forms for the first species and obstructive uropathy for the latter) (Pittella 1991, 1997, Ferrari 1999).

It is believed that in symptomatic NS the eggs reach the CNS through retrograde venous flow into the Batson vertebral epidural venous plexus, which connects the portal venous system and venae cavae to the spinal cord and cerebral veins. This route permits either anomalous migration of the adult worms to sites close to the CNS followed by in situ oviposition, or massive embolization of eggs from the portal mesenteric-pelvic system. The small round eggs of *S. japonicum* travel all this way and reach the brain; on the other hand, *S. mansoni* and *S. haematobium* eggs, which are larger and bear protruding spines, are retained in the lower spinal cord. Once deposited in the nervous tissue, the mature embryo secretes and excretes antigenic and immunogenic substances that account for the periovular granulomatous reaction. A large

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A STUDY OF THE POTENTIAL THERAPEUTIC EFFECT OF GINGER (*ZINGIBER OFFICINALE*) LOADED NANOPARTICLES ON MURINE SCHISTOSOMIASIS *MANSONI*

By
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Abstract

Chemotherapy is the most widely advocated method of antischistosomal control. Repeated chemotherapy has resulted in the emergence of drug-resistant schistosome strains. In the last few years, such resistance has drawn the attention to alternative drugs especially from natural sources (ginger). Nanoparticles have received special attention because they act as potent drug delivery systems. This study evaluated the antischistosomal effect of ginger extract loaded on chitosan nanoparticles on *Schistosoma mansoni* experimentally infected mice. The present study was conducted on sixty eight female BALB/C mice. Mice were exposed to 80±10 cercariae per mouse and divided into 3 main groups;(G1) negative control, (G2) positive control, G3) infected /treated, either by ginger extract (G3a), chitosan nanoparticles (G3b), praziquantel (G3c) or ginger extract loaded on chitosan nanoparticles (G3d). All groups were evaluated by parasitological and biochemical parameters. The results showed that worm burden and the egg density in liver were significantly reduced with P value<0.001 in G3d. The alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were significantly decreased in group G3d with p value<0.001 which indicated recovery of the liver tissue.

Key words: Ginger, Praziquantel, Chitosan nanoparticles, *Schistosoma mansoni*

Introduction

Schistosomiasis control strategy is based on the treatment of infected patients with selected drug as praziquantel (Mostafa *et al*, 2011). For centuries, ginger has been used in traditional medicine for respiratory disorders, stroke, hypercholesterolaemia, and schistosomiasis (Ali *et al*, 2008; Islam *et al*, 2008). It has antibacterial, antifungal, antioxidant, and anti-inflammatory effects and it increases the phagocytic activity and disease resistance against pathogens (Imtiyaz *et al*, 2013; El-Sayed *et al*, 2015). Unfortunately, the long term worldwide application of praziquantel coupled with the discovery of praziquantel-tolerant schistosome has triggered concern over the development of drug-resistant *Schistosoma* strains (Appiah *et al*, 2000).

Few investigations were done upon the antihelminthic activity of ginger and its constituents and showed that both crude powder and aqueous extract of dried ginger showed anti-helminthic activity in sheep (Iqbal *et al*, 2006). The nanotechnology embraces promise for medication and nutrition, as materials at the nanometer dimension exhibit novel properties different from those of isolated atoms and bulk material (Isaac *et al*, 2013).

Given the previous layout, the current work studied the anti-schistosomal effect of ginger extract loaded on chitosan nanoparticles on *Schistosoma mansoni* experimentally infected mice.

Material and Methods

Experimental design: The study was carried out on sixty eight female BALB/ C mice, 6-8 weeks old (18-20g),divided into 3 main groups;(G1) 8 mice uninfected untreated, (G2) 12 infected untreated mice, (G3) infected/treated groups subdivided into, (G3a) 12 mice treated by ginger extract, (G3b) 12 mice treated by chitosan nanoparticles, (G3c) 12 mice treated by praziquantel and (G3d) 12 mice treated by ginger extract loaded on chitosan nanoparticles.

Mice infection: Mice infected with 80±10 *S. mansoni* cercaria via subcutaneous route (Peters and Warren, 1969). Mice and cercariae were purchased from the Schistosome

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Case Report

Cellular immunodeficiency related to chronic dermatophytosis in a patient with *Schistosoma mansoni* infection: can schistosomiasis induce immunodeficiency?

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Abstract:

Here, we describe a case of hepatosplenic schistosomiasis that progressed to widespread persistent dermatophytosis. Significant T and B lymphocytopenia was confirmed. T-cell deficit is associated with increased susceptibility to fungal infections of skin and mucous membranes. The accumulation of a large amount of blood cells in the spleen could have played a crucial role in the development of lymphocytopenia in the present case. Alternatively, the schistosomiasis-induced increase in prostaglandin E2 levels could have inhibited the production of interferon-γ, a cytokine fundamental to fungal resistance. This case shows the potential of hepatosplenic schistosomiasis to impair the immune response.

Keywords: Schistosomiasis. Immunodeficiency. Chronic dermatophytosis.

INTRODUCTION

Schistosomiasis is a tropical disease caused by the flatworm *Schistosoma mansoni*. Specific freshwater snails (*Biomphalaria spp*) act as intermediate hosts, and human contact with water contaminated with larvae (cercariae) leads to infection. Immunopathological reactions against Schistosoma eggs trapped in tissues lead to inflammatory and intestinal obstructive disease, hepatosplenic inflammation, and liver fibrosis. Portal hypertension leads ultimately to hypersplenism and cytopenias, mainly thrombocytopenia and neutropenia. Schistosomiasis affects more than 200 million people worldwide: 120 million are symptomatic and 20 million have severe disease[1]. Despite the severity and impact of schistosomiasis on public health, the literature contains no reports relating schistosomiasis to any form of immunodeficiency. Here, we report the case of a patient with hepatosplenic schistosomiasis who developed lymphocytopenia associated with chronic dermatophytosis, indicating secondary immunodeficiency.

CASE REPORT

A 37-year-old man born in the rural area of the State of Minas Gerais, Brazil, to non-consanguineous parents of Caucasian and African ascendancy, showed normal neuropsychomotor development and no major diseases up to the age of 21 years. At this age, extensive and chronic dermatophytosis developed on his trunk, legs, and feet, and in the gluteal, inguinal, and perineal regions (**Figure 1**).

There was multiple circular, oval, and circinate lesions, confluent with scaly and hypochromic centers, with a 1-cm average diameter. A large, elevated, and erythematous lesion with a scaly center lesion that covered almost the entire upper back was particularly suggestive of a dermatophytosis. These lesions occurred intermittently, but 6 years prior, when the patient was 31 years old, the lesions became persistent, more extensive, and refractory to topical treatments. Abdominal enlargement occurred 4 years prior. His visited the Infectious Diseases Clinic, where a hepatosplenic form of *Schistosoma mansoni* infection was diagnosed. Treatment was initiated, and because of the uncommon pattern of the dermatophytosis, he was referred to the Primary Immunodeficiency Outpatient Unit for investigation of possible immunodeficiency. The patient had no history of sinusitis, pneumonia, cutaneous infection, meningitis, mucocutaneous or systemic candidiasis, chronic diarrhea, or other infections. His birthplace is endemic for *S. mansoni*. He had two brothers, one of whom died because of complications of *S. mansoni* infection, while the other is healthy. His uncle was also diagnosed with hepatosplenic schistosomiasis. During our initial investigation, dermatophytosis was confirmed by direct exam of the scraped skin from three different regions, which indicated the presence of multiple branched and septate

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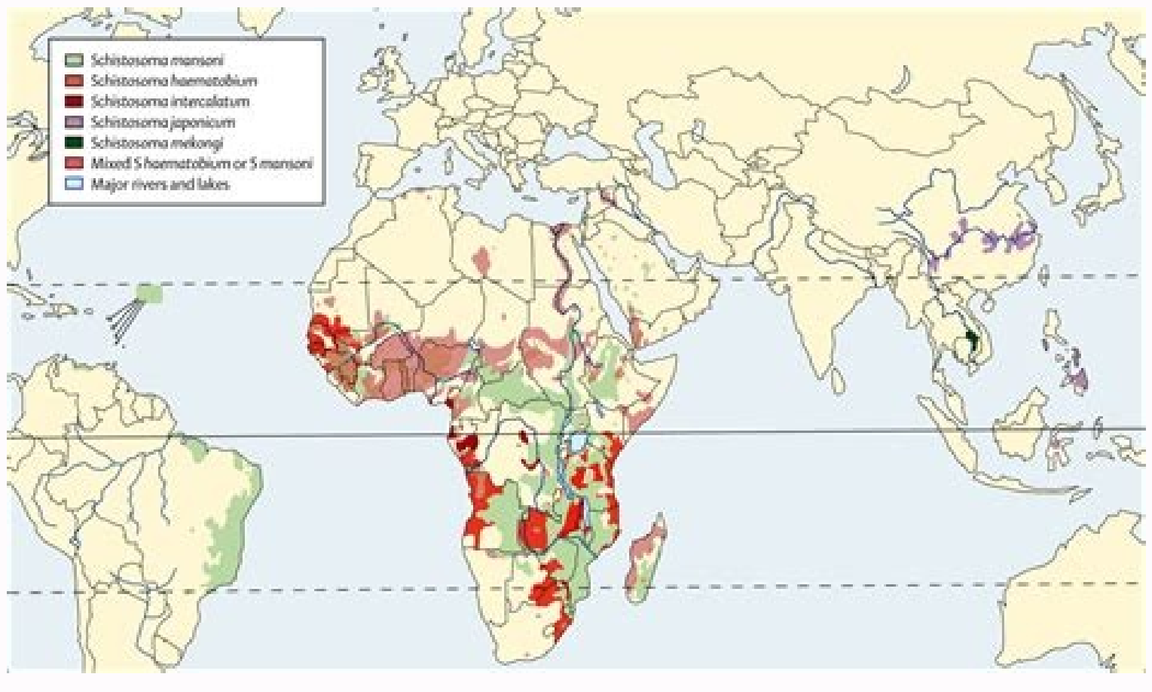

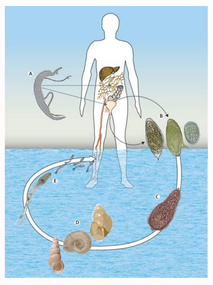

Schistosoma mansoni treatment. Schistosomiasis mansoni symptoms. Schistosoma mansoni eggs. Schistosomiasis mansoni life cycle. Schistosomiasis mansoni and haematobium. Schistosomiasis mansoni sign. Schistosoma mansoni hosts. Schistosoma mansoni test.

Mansoni (43, 44). Proteinases and associated genes of parasitic helminths. Schistosoma Mansoni: cytochemistry and morphology of the Golgi gastrodermal system. Schistosomiasis is estimated to cause only 280,000 deaths in sub-Saharan Africa (1.2). Digestive tract The digestive tract of S. References 1. Some ââ host proteins (hemoglobin, immunoglubolin (IG) G, and serum albumin) have been identified as worm antioxidants (superoxide dismutase (zolla) and thioredoxin) and ââ proteins . Prichard Rk, Basanez Mg, Bancain BA, McCarthy Js, Garcia HH, Yang GJ, et al. However, when SMCB1 Ã is incubated with the proteinase-inhibitor prior to immunization, protection levels decrease significantly, emphasizing the importance of the activity peptidase in protective potential. First, treating infected mice with cysteine protease inhibitors not only reduces a significantly worm burden, but also inhibits egg production by females. The authors suggest that blocking antibodies impact on nutrient autation by both GUT and a integument (36). Gut-protected prostheses Schistosome prostheses are involved in a wide range of essential processes such as invasion, migration, feeding, reproduction, activation and evasion of the immune system [reviewed in Ref. Morales me, Rinaldi G, GN, GN, Kines KJ, Tort JF, Brindley PJ. DOI: 10.1186 / 1471-2172-11-56 PubMed Abstract | Published full text | Full text by Crossref | Google Scholar 56. Hum Vaccin Immunother (2014) 10 (2): 399 Ã¢ â¥ "409. Several peptidases are used in the treatment and acquisition nutrients provided by the host blood (26 "28). Cysteine protease inhibitors block the degradation of schistosoma hemoglobin in vitro and reduce the weight of the worm and in vivo egg production. The antigens are listed under the position they have been identified in italics. 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Full text PUBMED | Full text of Crossref | Google Scholar 51. J Parasitolo (1968) 54 (1): 15 Ã ¢ â,¬ "27. Doi: 10.1136 / bmj.d2651 Crossref Full Text | Google Scholar Transtional Profiling of the Oesophageal Gland Schistosome Male Worm Region Mansoni. Schistosome SOD has also been detected in the proteomic analyzes of the vomiting and the transcriptional analysis of the digestive tract worm (23, 24, 29). Furthermore, several studies confirm the presence of proteases in gastrodermis from immunocythochemistry and underlines the roles of the endopettidase of the Cysteine, of the Catepsin L, of the Catepsin D, of the Catepsin B, of the asparaginyl endopeptidase and of metalloprotease in the hemoglobin and whey of degradation and the processing of albumin (45 "48). Bloodworking and nutrient absorption are fundamental to schistosomes survival and direct or indirect interruption of these processes can represent a realistic strategy for vaccine development. Quantification of clinical morbilitus associated with schistosome infection in sub-Saharan Africa. In addition to being a promising target of the vaccine, SMCB1 also acts as adjuvant, since the protection provided by immunization with other proteins increases, such as the 3-phosphate dehydrogenase glyceraldeide dhydrogenase and multiple antigen peptide, from

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