

SCHISTOSOMIASIS: PREDISPOSING CAUSE FOR THE FORMATION OF HEPATIC ABSCESES? CASE REPORT

Vasco Carvalho Pedrosa de LIMA (1) & Fernando Cotait MALUF (2)

SUMMARY

An adult patient with chronic schistosomiasis from an endemic area, complained about a seven day fever, along with jaundice and lumbar backache on the right side. Image exams showed multiple pyogenic liver abscesses. All the classic etiologies were discarded through clinical, radiological and laboratorial criteria. Schistosomiasis can cause pylephlebitis as a complication, along with immunosuppression, granulomatous reaction with central lobular liver necrosis and a greater risk of infection. The authors suggest that schistosomiasis in its chronic form may be the predisposing cause of multiple pyogenic liver abscesses, especially in endemic areas.

KEYWORDS: Schistosomiasis; Hepatic abscess; Liver abscess.

INTRODUCTION

The pyogenic hepatic abscess is an uncommon affection with reported prevalence, between 0.013% to 0.59%^{8,11}.

Although rarely found, the hepatic abscess has been studied since the classic Greek physicians' time. Hippocrates wondered whether the prognosis of hepatitis abscesses could be different, depending on its drainage aspects. But only in 1836 was this disease clinically and pathogenically described. Waller, in 1846, helped to understand the etiology of the pyogenic hepatic abscess when he described a case of appendicitis complicated with pylephlebitis¹⁶. In 1938 OCHSNER, studying 575 cases in the literature, noticed that, in 34% of them, complicated appendicitis⁸ was the etiology. From then on, several papers have tried to better characterize the predisposing factors, causative agents, diagnostic methods and treatment⁶.

Most pyogenic hepatic abscesses are single, but multiple abscesses are common. The predisposing cause is known in 76% (single), and 100% the cases of multiple abscesses⁷. Diseases of the biliary tract, portal bacteremia from intra-abdominal septic foci, such as diverticulitis or appendicitis; systemic bacteremia; hepatic trauma; direct extensions from a contiguous site of infection; infected hepatic metastasis; granulomatous chronic disease; leukemias; deficiency of the complement system components are known predisposing factors for the formation of pyogenic hepatic abscesses^{2,6,7}. The verified mortality spans from 15% to 100%^{7,12}. This difference could be explained by several prognostic factors - etiology, characteristics of the abscesses and patient - and the different clinical and surgical management approaches⁶.

In this paper, a case of chronic schistosomiasis

(1) Physician, Infectious Disease Unit, Hospital Emilio Ribas, SP, Brasil

Clinical Professor of Medicine, Division of Infectious Diseases, Faculdade de Ciências Médicas da Santa Casa de São Paulo, Brasil

(2) Intern in Infectious Diseases, Faculdade de Ciências Médicas da Santa Casa de São Paulo, Brasil

Correspondence to: Hospital Emilio Ribas, Diretoria de Apoio Técnico, Av. Dr. Arnaldo, 165, 01246-000, São Paulo, SP, Brasil

(*Schistosoma mansoni*), presenting with multiple pyogenic hepatic abscesses, is discussed. However, none of the classical predisposing factors was found. The association of possible susceptibility to *Staphylococcus aureus* infection, multiple pyogenic hepatic abscess, and chronic schistosomiasis was not found in the literature, either.

CASE REPORT

A 47-year-old white male patient, from Andirá, north of Paraná (endemic zone of schistosomiasis), was admitted to the Instituto de Infectologia "Emílio Ribas"/SP, complaining of fever, associated to jaundice and a seven days' duration lumbar pain on the right side. He had been living in São Paulo for more than twenty years had therefore no chance of infections during this time and had no history of chronic alcoholism. The admission physical exam showed a 10 cm tumor, in the left claviculo-external region, painful to palpation, fibroelastic, no fluctuation, hyperemic with local increase of temperature; two dental elements showed coronary fracture and probable endodontal involvement. The liver edge was palpable 6 cm below the right costal margin and the abdomen showed no tenderness to superficial or deep palpation. Routine WBC count showed leukocytosis (22.900/mm³), with a shift to the left (myeloblasts = 7%), hemoglobin = 14.8g%, hematocrit = 40%, albumin = 3g%, serum bilirubin (total) = 14mg%, bilirubin (direct) = 9.8 mg%, bilirubin (indirect) = 4.2 mg%, serum alkaline phosphatase = 135 I.U., SGOT = 186 I.U., SGPT = 158 I.U. (aminotransferases four times above higher normal values), negative serology for A, B and C hepatitis, stool examination showed *Schistosoma mansoni* eggs and blood culture was positive for *Staphylococcus aureus*.

Ultrasonography showed hepatomegaly and a rounded 5.5 cm heterogeneous image in hepatic segment VII; portal vein, gallbladder and biliary tree were within normal standards. Later, computerized tomography scan showed a low attenuation area and other multiple small loci, also with low attenuation, in the posterior segment of the right hepatic lobe (specially hepatic segment VII) (Fig. 1); loss of perinephric fat shadow in the upper pole of the right-sided kidney close to the liver; pleural effusion and right-sided pulmonary disease.

The day following admission, the patient experien-

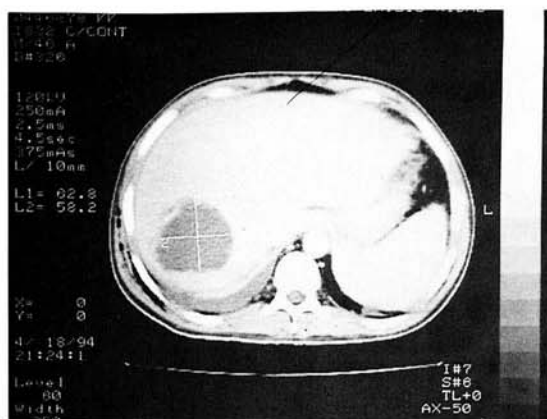


Fig. 1 - Computerized tomography scan showing a low attenuation area and other multiple small loci in the posterior segment of the right hepatic lobe.

ced respiratory distress. Chest X-ray showed patchy consolidation in the lower right-sided lobe. Three days following admission, the first percutaneous drainage of the largest abscess cavity was performed, and 500 ml of bloody-purulent fluid was removed, and culture of this fluid revealed *Staphylococcus aureus* with the same antibiotic sensitivity to those isolated on blood culture. Ten days following admission, a second percutaneous drainage of the same abscess was performed, due to its increase in size (11.1 cm). About 300 ml of bloody-purulent fluid was removed, with no bacterial growth. The patient's conditions improved gradually after the second percutaneous drainage, dyspnea diminished, and abdominal pain and tenderness have disappeared. Control ultrasonography and computerized tomography showed reduction in size of the larger hepatic abscess and the smaller loci. A third percutaneous drainage was performed seven days after the second one, and 100 ml of bloody-purulent fluid was removed. Its culture showed no bacterial growth. On the 35th day after admission, a liver biopsy was performed showing: slight enlargement of the portal triad, portal fibrosis, mononuclear infiltrate and Kupffer cells hyperplasia, and hepatocytes generally preserved. On the 45th day after admission, the patient was discharged from the hospital. During the hospitalization period, he received initially, penicillin G, gentamicin and metronidazole, all IV administered; and, after culture of the fluid removed from the abscess, and the blood culture, oxacillin, also IV administered, for four weeks, then orally, for two more weeks. The antibiotic change was due to *Staphylococcus aureus* sensitivity to oxacillin and resistance to the initial antimicrobial chemotherapy.

DISCUSSION

The pyogenic hepatic abscess is a rare disease^{6,7}, however, it has high morbidity and mortality rates⁷. Most abscesses are single, but multiple abscesses are common, like the ones reported in this case. The multiple hepatic abscesses are usually found in males^{8,12}, onset may be at any age, but most often occurs between 3 and 81, mean age 47 years-old, pertinent to the observed age of the reported patient⁷.

Clinical features are: fever, followed by abdominal pain, weight loss (10kg or more, in three months or less), nausea, vomiting and anorexia^{6,7}. Less frequently, diarrhea, abdominal distention, pruritus, dyspnea and prolonged hiccup attacks^{6,7}. Physical examination reveals hepatomegaly (64%), fever (55%)¹¹, abdominal tenderness to palpation (55%), jaundice (32%) and signs of pulmonary involvement (18%)⁷. Laboratory features are: anemia (77%) with hematocrit < 35%, leukocytosis (73%) with a shift to the left (50%), elevated alkaline phosphatase (55%), serum bilirubin > 2mg% (41%)¹², aspartate aminotransferase > 100 U.I. (39%), albumin < 3g% (36%)^{7,14}. Blood culture and culture from the pus removed from the hepatic abscess are positive in 38% of the cases⁷. This patient presented several of the symptoms, signs and laboratory exams usually found in medical literature.

The biliary tract infections represent the main causes of the formation of multiple pyogenic hepatic abscesses, followed by portal bacteremia from an intra-abdominal site (appendicitis, diverticulitis, colic perforation, pancreatic abscess, splenic abscess, and perinephric abscess), bacteremia, hepatic diseases, chronic alcoholism, diabetes mellitus, hepatic trauma (blunt, nonpenetrating specially), direct hepatic infections caused by extensions from a contiguous site of infection, immune diseases (specially in children), leukemias and extra-hepatic infected metastasis^{2,6,7,14}. The cause is typically obvious in multiple pyogenic hepatic abscesses, and detected in 100% of the cases, all of them listed above⁷. This patient, however, clinically, laboratorially and radiologically, did not present any of the classical causes related in the medical literature.

Chronic schistosomiasis, in the hepato-intestinal form presents with pylephlebitis as a complication in some cases⁵. It has been shown that necrotic areas in the liver, caused by a primary or metastatic neoplasm could be infected by bacteria, the same occurring in

central lobular necrosis, caused by hepatic granuloma⁵, which was not observed in our case. It was also shown that chronic schistosomiasis patients present: serum factors that inhibit the thymic lymphoid cells (T cells)^{1,9,13}, mononuclear cells that suppress lymphocytic activity¹⁵ and antibodies against IgE e IgG^{3,4,10}. Two cases were found in the literature of patients that came from endemic areas for *Schistosoma mansoni* and presented with pyogenic abscesses caused by *S. aureus* and acute schistosomiasis⁵. Acute schistosomiasis differs from the chronic disease in immunopathological aspects. The incidence of *Staphylococcus aureus* as a causative agent of multiple pyogenic hepatic abscesses is about 5%⁷.

A experimental test was designed to confirm schistosomiasis as a predisposing factor to the formation of pyogenic hepatic abscesses: 16 albino rats were inoculated with the *Schistosoma mansoni*'s cercaria (70); sixty days later, the same animals were inoculated (IV route) with *Staphylococcus aureus* (2 x 100.000/ml) isolated from a patient with bacteremia. Ten to fourteen days later, 5 rats presented multiple pyogenic hepatic abscesses. The *Staphylococcus aureus* was isolated in cultures from the pyogenic fluid removed from the hepatic abscesses in these 5 cases⁵.

Based on this report, we suggest the hypothesis that schistosomiasis, in its chronic form, could be related to the formation of pyogenic hepatic abscesses, representing therefore, a possible predisposing cause, specially in subjects who lived in endemic areas. This association was not found by the authors in medical literature. Maybe this finding is related to the rarity of the event, or to the fact that a great number of international journals come from countries in which the schistosomiasis incidence in the population is not high enough, or even inexistent.

RESUMO

Esquistossomose: causa predisponente para a formação de abscessos hepáticos?

Paciente adulto, natural de região endêmica para esquistossomose e portador crônico da doença, apresentava queixa de febre há sete dias, associada à icterícia e dor lombar em região direita. Os exames radiológicos mostraram abscessos hepáticos piogênicos múltiplos, cuja causa predisponente é conhecida, segundo trabalhos da literatura, em 100%

dos casos. Através de parâmetros clínicos, laboratoriais e radiológicos todas as etiologias clássicas foram afastadas. Sabe-se que a esquistossomose pode provocar, como complicação, a pyleflebite, além de depressão imunológica e reação granulomatosa com necrose lobular central e maior risco de infecção. Os autores deste relato de caso sugerem ser a esquistossomose, na sua forma crônica, causa predisponente para formação de abscessos hepáticos piogênicos múltiplos, principalmente em regiões endêmicas.

REFERENCES

1. COLLEY, D. G.; HIENY, S. E.; BARTHOLOMEW, R. K. & COOK, J. A. - Immune responses during human schistosomiasis mansoni. III. Regulatory effect of patient sera on human lymphocyte blastogenic responses to schistosome antigen preparations. **Amer. J. trop. Med. Hyg.**, 26: 917-925, 1977.
2. GARTY, B. - Z.; CONLEY, M. E.; DOUGLAS, S. D. & KOLSKI, G. B. - Recurrent infections and staphylococcal liver abscess in a child with Clr deficiency. **J. Allergy clin. Immunol.**, 80: 631-635, 1987.
3. HIATT, R. A.; SOTOMAYOR, Z. R.; SANCHEZ, G.; ZOMBRANA, M. & KNIGHT, W. B. - Factors in pathogenesis of acute schistosomiasis mansoni. **J. infect. Dis.**, 139: 659-666, 1979.
4. HOFSTETTER, M.; POINDEXTER, R. W.; RUIZ-TIBEN, E. & OTTESEN, E. A. - Modulation of the host response in human schistosomiasis. III. Blocking antibodies specifically inhibit immediate hypersensitivity responses to parasite antigens. **Immunology**, 46: 777-785, 1982.
5. LAMBERTUCCI, J. R.; TEIXEIRA, R.; NAVARRO, M. M. M.; COELHO, P. M. Z. & FERREIRA, M. D. - Liver abscess and schistosomiasis. A new association. **Rev. Soc. bras. Med. trop.**, 23: 239-240, 1990.
6. McDONALD, A. P. & HOWARD, R. J. - Pyogenic liver abscess. **Wld. J. Surg.**, 4: 369-376, 1980.
7. McDONALD, M. I.; COREY, G. R.; GALLIS, H. A. & DURACK, D. T. - Single and multiple pyogenic liver abscesses. Natural history, diagnosis and treatment, with emphasis on percutaneous drainage. **Medicine** (Baltimore), 63: 291-302, 1984.
8. OCHSNER, A.; DEBAKEY, M. & MURRAY, S. - Pyogenic abscesses of the liver. II. An analysis of forty-seven cases with review of the literature. **Amer. J. Surg.**, 40: 292-319, 1938.
9. OTTESEN, E. A.; POINDEXTER, R. W. & HUSSAIN, R. - Detection, quantitation and specificity of antiparasite IgE antibodies in human schistosomiasis mansoni. **Amer. J. trop. Med. Hyg.**, 30: 1228-1337, 1981.
10. OTTESEN, E. A. & POINDEXTER, R. W. - Modulation of the host response in human schistosomiasis. II. Humoral factors with inhibit lymphocyte proliferative responses to parasite antigens. **Amer. J. trop. Med. Hyg.**, 29: 592-597, 1980.
11. PITT, H. A. & ZUIDEMA, G. D. - Factors influencing mortality in the treatment of pyogenic abscesses. **Surg. Gynec. Obstet.**, 140: 228-234, 1975.
12. RAMBO, W. M. & BLACK, H. C. - Intrahepatic abscess. **Amer. Surg.**, 35: 144-148, 1969.
13. ROCKLIN, R. E.; BROWN, A. P.; WARREN, K. S. et al. - Factors that modify the cellular-immune response in patients infected by *Schistosoma mansoni*. **J. Immunol.**, 125: 1916-1923, 1980.
14. RUBIN, R. H.; SWARTZ, M. N. & MALT, R. - Hepatic abscess: changes in clinical, bacteriologic and therapeutic aspects. **Amer. J. Med.**, 57: 601-610, 1974.
15. TODD, C. W.; GOODGAME, R. W. & COLLEY, D. G. - Immune responses during human schistosomiasis mansoni. V. Suppression of schistosome antigen-specific lymphocyte blastogenesis by adherent/phagocytic cells. **J. Immunol.**, 122: 1440-1446, 1979.
16. WALLER, 1846 apud LOISSON, E. - Des suppurations intra et perihépatiques d'origine typhlo-appendiculaire. **Rev. Chir. (Paris)**, 21: 522, 1900.

Recebido para publicação em 17/08/1994.

Aceito para publicação em 02/06/1995.