São Paulo Medical Journal
Revista Paulista de Medicina

Review Article

Sérgio Cimerman
Benjamin Cimerman
David Salomão Lewi

Enteric parasites and AIDS

Instituto de Infectologia Emílio Ribas and Univerisidade Federal de São Paulo/ Escola Paulista de Medicina, São Paulo, Brazil

ABSTRACT

OBJECTIVE: To report on the importance of intestinal parasites in patients with AIDS, showing relevant data in the medical literature, with special emphasis on epidemiology, diagnosis and treatment of enteroparasitosis, especially cryptosporidiasis, isosporiasis, microsporidiasis and strongyloidiasis.
DESIGN: Narrative review.
KEY WORDS: HIV. AIDS. Parasitosis.

INTRODUCTION

Since the first AIDS cases were described, a high prevalence of gastrointestinal alterations has been reported, especially diarrhea associated with parasitosis.

This became more evident when the appearance of a syndrome named "Slim Disease", characterized by an intense weight loss accompanied by chronic diarrhea, prolonged fever and diffuse muscle weakness, was observed in Africa, especially in Uganda.[1,2,3] Studies conducted in Zaire and Uganda have shown the presence of some pathogenic agents responsible for the "Slim Disease", such as *Isospora, Cryptosporidium, Salmonella, Shigella* and *Campylobacter* species, amounting to a prevalence of 60 to 80%.[4] "Slim Disease" has been observed in advanced stages of HIV infection. The expression "Wasting Syndrome" was adopted in substitution by WHO in 1988 on the basis of criteria laid down by the CDC.[2]

Thus, where as infections in the gastrointestinal tract play a critical role in AIDS pathogenesis and diarrheic diseases assume a prominent role, reaching a rate of up to 50% in developed countries, in developing countries there have been reports of incidence of up to 95%, as in Haiti and

the African continent.[5]

Amongst the causes of diarrhea in developing countries, those of a parasitic origin are prominent in patients with AIDS.

Opportunistic infections caused by intestinal parasites also vary according to the geographical area and the endemic levels in each location.

The progressive decline of immunological and mucous defense mechanisms predisposes patients to early, intermediary and late gastrointestinal manifestations of HIV infection.[6] At later stages of the disease, the alterations in non-specific defense mechanisms in the production of Ig A and the reduction in local immune cell response also progress, thus increasing the susceptibility to a number of intestinal opportunistic pathogens, among which *Cryptosporidium parvum*, *Isospora belli* and Microsporidia species are the most prominent.[7]

After the emergence of AIDS, these parasites, until then known solely in veterinary medicine, were no longer considered as commensal organisms and are nowadays recognized as opportunistic pathogens common to these patients. Infections by these agents constitute a major secondary aggravating factor of the disease, often responsible for worsening the general health conditions, due to manifestations of diarrhea which are difficult to control, sometimes resulting in the death of the patient.

CRYPTOSPORIDIASIS

Cryptosporidiasis, a disease caused by an intracellular protozoan named *Cryptosporidium sp*, was described for the first time in 1907 by Tyzzer. This parasite was considered a commensal up to 1975, when it was identified as the cause of diarrhea in animals.[8]

The first manifestation of cryptosporidiasis was reported by Nine et al. in 1976, and the disease became a major concern when the notification of the first 21 patients was given, 14 of whom died of chronic diarrhea caused by *Cryptosporidium sp* infection.[9]

It is worth noting that outbreaks of epidemic linked to contaminated water are of extreme importance for the dissemination of this parasite such as the 1993 Milwaukee (USA) cryptosporidiasis outbreak, in which 403,000 human cases were reported, thereby becoming a relevant public health concern.[10-12] After this outbreak, it was recommended that immunocompromised patients should be extremely careful with water, foodstuffs and contact with animals.

Cryptosporidiasis is distributed over all continents. In Haiti and the African continent the prevalence is 50%,[13] while in the USA it ranges from 3 to 4%.[14]

In Brazil, due to the continental size of the country, the rates of incidence vary with the location. In the city of São Paulo, for instance, reports have referred to levels around 12.1 to 24.44%.[15-17] However, the latest epidemiological report on Brazil, dated February 1998, shows a cryptosporidiasis rate of only 3.5%.[18]

Cryptosporidiasis in AIDS patients usually causes chronic, bulky and intermittent diarrhea, with liquid non-bloody stools, accompanied by pain and abdominal colic, and a noticeable loss of weight can be observed.[8]

Asymptomatic cases are rarely described, occurring mostly in developing countries with patients showing milder immunodeficiency.[19,20]

Extraintestinal manifestations have been clearly described in the literature, especially in the gall bladder, biliary ducts and pancreas, leading to conditions such as papillary stenosis, sclerosing cholangitis and acalculous cholecystitis. The respiratory tract can also be affected with manifestations of chronic bronchitis.[21,22]

The intensity and duration of diarrhea in cryptosporidiosis cases is closely associated with the CD4+ T cell counts. This is well demonstrated in a classic study on HIV-infected patients with CD4+ cell counts higher than 180/mm^3 who displayed clinical healing over a period of four weeks, while 87% of the patients presenting more severe immunosuppression, with CD4+ counts lower than 140/mm^3, presented persistent and hard-to-control diarrhea.[23]

Laboratory diagnosis of cryptosporidiosis is easy to accomplish, using special staining techniques such as the Kinyoun (modified Ziehl

Neelsen) and auramine-rhodamine methods, via the detection of reddish-stained *Cryptosporidium* oocysts.[24] More sophisticated diagnostic techniques using monoclonal antibodies are already available, such as ELISA and immunofluorescence, with high sensitivity and specificity.[25-27] The treatment is controversial, with rehydration via an appropriate liquid balance and the maintenance of the patient's nutritional condition being recommended. There should be careful monitoring and administration of antidiarrheic drugs when needed.[28] Several treatments are available but they have not shown significant clinical efficiency. Studies have been conducted with hyperimmune bovine colostrum, letrazuril and diclazuril (veterinary medicine drugs), spiramicyn and more recently azithromicyn, paromomycin, octreotide and roxithromycin.[29-31]

ISOSPORIASIS

Isospora belli is a coccidium described for the first time in 1915 by Woodcock and later by Wenyon in 1923.[32] It is found in tropical and subtropical areas and is endemic in South America, Africa and in Southern Asia,[33] with an occurrence rate of 15% in Haiti,[33] 0.2% in the USA[34] and 1.8% in Brazil.[18]

Higher rates of isosporiasis in Brazil have been reported in AIDS patients living in Santos and in São Paulo, with a prevalence of 9.9% and 6.67%, respectively.[17,35] The lower prevalence of Isosporiasis may be ascribed to the secondary prophylaxis for pneumocystosis through administration of sulfamethoxazole-trimethoprim during the course of AIDS, since *Isospora belli* is sensitive to this therapy.[36]

The diarrheic condition is also noteworthy and is accompanied by fever, intestinal colic, anorexia, abdominal pain, loss of weight and peripheral eosinophilia.[37]

Isosporiasis can also show extraintestinal dissemination features, affecting the mesenteric, periaortic, mediastinal and tracheobronchial lymph nodes.[38,39] It may also be related to biliary disease, causing manifestations of acalculous cholecystitis.[37]

Isospora belli differs morphologically from *Cryptosporidium sp* not only because of its intrinsic morphology (elliptical oocyst measuring 22 x 15 µm in diameter, containing two sporocysts with four sporozooites), but also for the intracellular location in the absorptive cell, while *Cryptosporidium* is restricted to the brush borders, immediately under the apical membrane of absorptive cells.[40]

Laboratory diagnosis is carried out in the same way as for *Cryptosporidium parvum,* using the Kinyoun and auramine-rhodamine techniques,[41] although common processes such as Faust's are often enough for the diagnosis. The special coloration thus becomes a further diagnostic element for finding the parasite.

The therapeutic recommendation for isosporiasis is the administration of sulfamethoxazole-trimethoprim for 10 days, followed by prophylaxis for a further three weeks. This leads to a reduction in the number of discharges and the recovery of body weight.[36] In recurrent situations or in non-responding patients it is necessary to administer other drugs such as pyrimethamine, in isolation or in association with sulfadiazine,[42] roxithromycin[43] and metronidazole.[44] Drugs such as tetracycline, ampicillin, nitrofurantoin, quinacrine and furazolidone have already been used but showed no therapeutic success.[34]

MICROSPORIDIASIS

The third major group of intestinal pathogens to be reported are the microsporidia, which are strictly intracellular protozoa, spore-producing and with great variety of genera and species. They have widespread distribution and over 400 cases of patients with co-infection of microsporidia and HIV have been reported. The major etiologic agent is *Enterocytozoon bieneusi*.[45]

The first description of intestinal microsporidiasis in an HIV-positive patient occurred in France.[46] The first description of this condition in Brazil dates back to 1993, with cases having occurring in Rio de Janeiro, São Paulo and Ceará.[47-50]

The world prevalence of intestinal microsporidiasis ranges between 7 and 50%.[51] In Rio de Janeiro a recent study applying the direct

technique for detection of microsporidian spores in the stool samples of 140 HIV-positive patients showed that the rate of prevalence of intestinal microsporidiasis was 17.86%.[52] The fact that these cases have only recently appeared can be explained by the diagnostic difficulty, as electronic microscopy is required for confirmation of the presence of the parasite. In contrast, in the city of São Paulo only 1.3% of microsporidian spores were shown up in patients with AIDS and chronic diarrhea via the modified trichrome staining method.[52A]

Optical microscopy methods, such as the chromotrope method, were then proposed whereby spores would appear as oval-shaped and stained pink or pale red.[53] This technique has been improved and named Gram-chromotrope. Fecal sample smears are stained in Gram's stain, followed by a chromotrope solution.[54]

Chronic diarrhea and/or biliary disease manifestations in HIV-infected individuals with CD4+ T cells of between 50 and 100/mm^3 may suggest microsporidiosis. Transmission is unknown to date, but there have been reports of congenital transmission and via inhalation of airborne spores.[51]

Regarding treatment, albendazole has been the most promising drug for handling intestinal microsporidiasis.[55-57]

GIARDIASIS AND AMEBIASIS

The protozoans *Giardia lamblia* and *Entamoeba histolytica* are important causes of acute diarrhea in homosexual males, even for those that are not HIV-positive. Together with *Campylobacter*, *Salmonella, Shiguella* and *Yersinia* they cause high frequencies of enteritis, colitis and proctitis.[58]

Statistical data found in the literature are rather divergent, but the incidence of these protozoans amongst the homosexual populations of large cities, such as New York, Los Angeles, San Francisco and Toronto is quite high.[59]

The prevalence of *Entamoeba histolytica* in HIV-negative homosexual patients in the USA ranges between 21 and 32%, and is around 12% in the United Kingdom.[60]

A study performed in Los Angeles with 100 HIV-positive patients, mostly homosexuals, showed a prevalence rate of 55% for giardiasis and 3% for amoebiasis.[61]

In France, studies focused upon the prevalence of intestinal parasitosis showed a high frequency of protozoan species in AIDS patients, and the rates of *Giardia lamblia* and *Entamoeba histolytica* were 5.8% and 2%, respectively[62].

Previous studies demonstrated that giardiasis incidence does not differ amongst HIV-positive and negative patient populations.[63]

Infection with *Giardia lamblia* and HIV amongst homosexual males is correlated with enteritis or enterocolitis, but no histological study of the colonic mucosa has yet been carried out.[64]

In Brazil, examination of 771 fecal samples from AIDS patients living in São Paulo, performed under the program for the control and prevention of AIDS, have shown rates of 5.18% and 8.49% of amebiasis and giardiasis, respectively.[15] In a recent study in the city of São Paulo analyzing patients with more severe immunodeficiency, *E. histolytica was not observed but G. lamblia* cysts were seen in about 27% of the examined patients.[17]

Despite this high prevalence, classical protozoa such as *Giardia Lamblia* and *Entamoeba histolytica* are less frequent as causes of severe illnesses in HIV-infected patients, when compared with Microsporidia, *Isospora belli* and *Cryptosporidium parvum* and they are not considered as opportunistic infections in AIDS.

Amebiasis may present with invasive characteristics, but this has rarely been reported in the literature.[65]

More recently, in Recife, a study was conducted to evaluate invasive amebiasis in AIDS patients. Seventy four patients were examined, out of which 54 had diarrhea but *Entamoeba histolytica* was found in only one patient (1,3%) and practically an absence of invasive amoebiasis.[66]

STRONGYLOIDIASIS

Among the helminths in association with AIDS, there is no doubt that the most important pathogen is *Strongyloides stercoralis*, which was

first described in 1876 by Normand, after necropsy of patients with diarrhea in from Cochin-China, then an autonomous region of China.[67]

This geohelminth presents its major effects in immunodepressed patients, leading to the dissemination of the infection. This occurs in transplanted patients, individuals presenting malnutrition and patients submitted to prolonged use of corticosteroids, suffering from leukemia, lymphomas or AIDS.[68,69]

Despite the possibility of dissemination of this helminth in HIV-positive patients, only 14 cases had been reported by 1994 in the international literature.[70]

A case of massive infection by *Strongyloides stercoralis* in AIDS patients in São Paulo was recently reported, presenting the nematode even in sputum samples.[71]

The clinical syndrome of disseminated strongyloidiasis is characterized by gastrointestinal signs and symptoms, such as nausea, vomiting, meteorism, anorexia and diarrhea, alternating with periods of intestinal constipation and secondary infections. Respiratory symptoms such as dyspnea, hemoptysis, coughing, or manifestations of asthma and even extensive pneumonia, may be observed. At the same time, the patient can present fever, cachexia and loss of weight.[72, 73]

In immunosuppressed patients, self-infestation is speeded up and a large number of larvae are released, causing the dissemination of the infection.[74]

In Brazil, the prevalence of strongyloidiasis in HIV-positive patients is around 4 to 15%, in comparison with 1.4% in the general population.[15,17,69]

Laboratory diagnosis is based upon the Baerman-Moraes or Rugai-Mattos-Brisola methods, presenting as their basic principle the thermo and hydrotropism of *Strongyloides stercoralis* larvae seen in stool samples.[67]

Strongyloides stercoralis larvae may also be detected in aspirated duodenal material, sputum, broncho-alveolar lavage, cervicovaginal cytology, cerebrospinal fluid and gastric cytology.[74]

Generally, the search for *Strongyloides stercoralis,* even in isolated samples, shows a positivity of 30% in immunocompromised individuals with AIDS. When three or more samples are taken, this positivity reaches levels as high as 80%.[67]

Thiabendazole is considered the drug of choice for treating strongyloidiasis, in immunosuppressed individuals. The rates of eradication by this anti-helminth drug can reach levels as high as 90%.[67,68,72] Other chemotherapy agents that may be used include albendazole and cambendazole.

In *Strongyloides stercoralis* hyperinfection cases, treatment with thiabendazole may be used for prolonged periods, followed by maintenance doses. Often, eradication is difficult.[70,71] Another option in these cases is the administration of ivermectin, a drug used for onchocerciasis, with healing rates around 94%.[70,75,76] This drug is not yet available in Brazil.

OTHER PARASITES

Other helminths such as *Ascaris lumbricoides* (3.52%), *Trichiuris trichiura* (4.14%), *Ancylostomidae* (2.69%), *Enterobius vermicularis* (0.21%), *Schistosoma mansoni* (1.66%), *Taenia sp* (0.21%) and *Hymenolepis nana* (10.41%) have shown lesser frequency and importance in AIDS patients.[15]

Several other intestinal parasitoses may lead to aggravation and / or dissemination of the course of AIDS. Protozoa such as *Cyclospora sp* and *Blastocystis hominis* have arisen, which may also cause major diarrhea manifestations.

Blastocystis hominis is a potentially pathogenic parasite, more predominant in male homosexuals than in other population groups, and its occurrence is not higher in individuals suffering from digestive disturbances.[77] It has been isolated in human feces with a frequency ranging from 1 to 60% in individuals from different parts of the world and exposure to contaminated water contributes to the increase of this parasitosis.[78]

The association of *Blastocystis hominis* with immunodepression has been well-documented, chiefly in diabetic and leukemic patients.[79,80] In AIDS, *Blastocystis hominis* leads to chronic diarrhea manifestations; six cases in Bahia (Brazil)

were recently reported with parasite eradication in the stool samples after specific treatment.[81]

Blastocystis infection is common in immunocompromised hosts and it may be diagnosed through conventional techniques such as the Lutz and Faust methods. There are reports in the literature that show trichrome stains and Ficol concentrations as alternatives for finding this parasite.[82,83]

There is some controversy about the appropriateness of treating this enteroparasitosis. When treatment is chosen, metronidazole is used.[84]

Cyclospora sp was initially isolated in 1870 by Eimer. The first report on its association with AIDS dates back to March 1989, in a male patient with chronic diarrhea.[85]

Studies on the prevalence of cyclosporiasis show very low rates, not higher than 1% in developed countries.[86,87]

Outbreaks of cyclosporiasis due to the consumption of raspberries imported from Guatemala occurred recently: 1465 cases were reported from 20 states of the USA.[88] Other outbreaks were reported in Virginia (USA), due to contamination of fresh basil, causing gastrointestinal disease.[89]

The diagnosis of infection by *Cyclospora cayatenensis* is based upon the detection of oocysts in fecal samples, via optical microscopy. *Cyclospora cayatenensis* differs from *Cryptosporidium* in its large size.[90] Due to the morphological similarities and lack of knowledge on how to differentiate between these two species, diagnosis of cyclosporidiosis is seldom accomplished.[33] It is essential to determine the sporulation of Cyclospora, which may be done using 2.5% potassium dichromate.

The low frequency of this protozoan may once again be explained by the use of sulfamethoxazole-trimethoprim administration as a prophylactic treatment against *Pneumocystis carinii* in AIDS patients presenting CD4+ cell counts lower than $200/mm^3$.[91,92]

CONCLUSION

It can be noted that parasitosis presents a very significant interface with AIDS, and this has been the object of studies by many authors. Investigations on the etiology of diarrhea in HIV-infected patients indicate parasitosis as the cause in up to 40% of patients.

REFERENCES

1. Kanradt T, Niese D, Vogel F. Slim disease (AIDS). Lancet 1985;2:1425.
2. Mhiri C, Bélec L, Di Constanzo B, et al. The slim disease in African patients with AIDS. Trans R Soc Trop Med Hyg 1992;86:303-6.
3. Grunfeld C, Schanbelan M. The wasting syndrome: pathophysiology and treatment. In: Broder S, Merigan Jr TC, Bolognesi D. Textbook of Aids Medicine. 2nd ed. New York: Williams & Williams; 1994:637-49.
4. Bolinger RC, Quinn TC. Tropical diseases in the HIV-infected traveler. In: Broder S, Merigan Jr, Bolognesi D. Textbook of Aids Medicine, 2ed. New York: Williams & Williams; 1994:311-22.
5. Smith PD. Diarréia infecciosa nos pacientes com AIDS. Clin Gastroenterol Am Norte 1993;3:569-84.
6. McGowan I, Chalmers A, Smith GR, Jewell D. Advances in mucosal immunology. Gastroenterol Clin North Am 1997;26:145-73.
7. Soares RLS, Camillo-Coura L, Magalhães LF, et al. Isosporíase como causa freqüente de diarréia crônica em pacientes com AIDS em nosso meio. Ann Acad Nac Med 1996;156:24-5.
8. Soave R, Armstrong D. Cryptosporidium and cryptosporidiosis. Rev Infect Dis 1986;8:1012-23.
9. Center for Disease Control. Cryptosporidiosis: assessment of chemotherapy of males with acquired immune deficiency syndrome (AIDS). Morb Mortal Wkly Rep 1982;31:589-92.
10. MacKenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of Cryptosporidium infection transmitted through the public water supply. N Engl J Med 1995;331:161-7.
11. Goldstein ST, Juranek DD, Ravenholt O, et al. Cryptosporidiosis: an outbreak associated with drinking water despite state-of-the-art water treatment. Ann Intern Med 1996;124:459-68.
12. Widmer G, Carraway M, Tzipori S. Water-borne cryptosporidium: a perspective from the USA. Parasitol Today 1996;12:286-90.
13. Colebunders R, Francis H, Mann JM, et al. Persistent diarrhea, strongly associated with HIV infection in Kinshasa, Zaire. Am J Gastroenterol 1987;82:859-64.
14. Smith, PD, Lane HC, Gill VJ, et al. Intestinal infections in patients with the acquired immunodeficiency syndrome (AIDS). Ann Intern Med 1988;108:328-33.
15. Dias RMDS, Pinto WP, Chieffi PP, et al. Enteroparasitoses em pacientes acometidos pela síndrome de imunodeficiência adquirida (AIDS/SIDA). Rev Inst Adolfo Lutz 1988;48:63-7.
16. Rodrigues JLN, Leser P, Silva TMJ, et al. Prevalência da criptosporidiose na síndrome diarréica do paciente HIV-positivo. Rev Assoc Med Brasil 1991;37:79-84.
17. Cimerman S. Prevalência das parasitoses intestinais em pacientes portadores da síndrome da imunodeficiência adquirida. Master's Dissertation in Infectious and Parasitic Diseases Unit, Universidade Federal do Estado de São Paulo/Escola Paulista de Medicina. São Paulo; 1998:125.
18. Brasil, Ministério da Saúde. Programa Nacional de DST/AIDS. Associação Brasileira Interdisciplinar de AIDS/(ABIA). Bol Epidemiol (AIDS) 1988;11:1-41.
19. Baxby D, Hart CA. The incidence of cryptosporidiosis: a two year prospective survey in a children's hospital. J Hyg (London) 1986;96:107-11.
20. Moreno A, Gattel JM, Mensa J, et al. Incidencia de enteropatógenos en pacientes con infección por el vírus de la immunodeficiencia humana. Med Clin (Barc.) 1993;102:205-8.
21. Salvador Grande F, Saizmonzón L, De La Torre P, et al. Colecistitis

alitiasica y cryptosporidiasis intestinal: associación frecuente en pacientes VIH. Rev Esp Enferm Dig 1995;87:593-6.

22. Vakil NB, Schwartz SM, Buggy BP, et al. Biliary cryptosporidiosis in HIV-infected people after the waterborne outbreak of cryptosporidiosis in Milwaukee. N Engl J Med 1996;334:19-23.
23. Flannigan T, Whalen C, Turner J, et al. Cryptosporidium infection and CD4 counts. Ann Intern Med 1992;116:840-2.
24. Casemore DP, Armstrong M, Sands RL. Laboratory diagnosis of cryptosporidiosis. J Clin Pathol 1985;38:1337-41.
25. Alles AJ, Waldron MA, Sierra LS, et al. Prospective comparison of direct immunofluorescence and conventional staining methods for detection of Giardia and Cryptosporidium spp. in human fecal specimens. J Clin Microbiol 1995;33:1632-4.
26. Parisi MT, Tierno Jr PM. Evaluation of new rapid commercial enzyme immunoassay for detection of Cryptosporidium oocysts in untreated stool specimens. J Clin Microbiol 1995;33:1963-5.
27. Marshall MM, Naumovitz D, Ortega Y, et al. Waterborne protozoan pathogens. Clin Microbiol Rev 1997;10:67-85.
28. Hoepelman IM. Human cryptosporidiosis. Int. J STD & AIDS 1996;7:28-33.
29. Martins CAP, Guerrant RL. Cryptosporidium and cryptosporidiosis. Parasitol Today 1995;11:434-6.
30. Hashmey R, Smith NH, Cron S, et al. Cryptosporidiosis in Houston, Texas: a report of 95 cases. Medicine 1997;78:118-39.
31. Uip DE, Lima AL, Amato VS, et al. Roxithromycin treatment for diarrhea caused by Cryptosporidium spp. in patients with Aids. J Antimicrob Chemother 1998;41(suppl B):93-7
32. Wenyon CM. Coccidiosis of cats and dogs and the status of the Isospora of man. Ann Trop Med Parasitol 1923;17:231-76.
33. Soave R, Johnson WD. Cryptosporidium and Isospora belli infections. J Infect Dis 1988;157:225-9.
34. De Hovitz JA, Pape JW, Boncy M, et al. Clinical manifestations and therapy of Isospora belli infection in patients with the acquired immunodeficiency syndrome. N Engl J Med 1986;315:87-90.
35. Sauda FC, Zamarioli LA, Ebner Filho W, et al. Prevalence of Cryptosporidium sp and Isospora belli among AIDS patients attending Santos reference center for Aids, São Paulo, Brazil. J Parasitol 1993;79:454-6.
36. Pape JW, Verdier RI, Johnson Jr WD. Treatment and prophylaxis of Isospora belli infection in patients with the acquired immunodeficiency syndrome. N Engl J Med 1989;320:1044-7.
37. Benator DA, French AL, Beaudet LM, et al. Isospora belli infection associated with acalculous cholecystitis in a patient with AIDS. Ann Intern Med 1994;121:663-4.
38. Restrepo C, Macher AM, Radany EH. Disseminated extraintestinal isosporiasis in a patient with acquired immune deficiency syndrome. Am J Clin Pathol 1987:87:536-42.
39. Michiels JF, Hofman P, Bernard E, et al. Intestinal and extraintestinal Isospora belli infection in an AIDS patient: a second case report. Pathol Res Pract 1994;190:1089-93.
40. Marcial-Seone MA, Serrano-Olmo J. Intestinal infection with Isospora belli. P R Health Sci J 1995;14:137-40.
41. Lindsay DS, Bubey JP, Blagbrun BL. Biology of Isospora spp. from humans, non-human primates, and domestic animals. Clin Microbiol Rev 1997;10:19-34.
42. Ebrahimzadeh A, Bottone EJ. Persistent diarrhea caused by Isospora belli: therapeutic response to pyrimethamine and sulfadiazine. Diagn Microbiol Infect Dis 1996;26:87-9.
43. Musey KL, Chidiac C, Beacauire G, et al. Effectiveness of roxithromycin for treating Isospora belli infection. J Infect Dis 1988;158:646.
44. Forthall D, Guest SS. Isospora belli enteritis in three homosexual men. Am J Trop Med 1984;116:840-2.
45. Bryan RT, Weber R. Microscoporidia: emerging pathogens in immunodeficient persons. Arch Pathol Lab Med 1993;117:1243-5.
46. Desportes I, Charpentier Y, Galian A, et al. Occurrence of a new microsporidian: Enterocytozoon bieneusi n.g. n.s, in the enterocytes of human patient with AIDS. J Protozool 1985;32:250-4.
47. Hirschfeld MPM, Cury AE, Moura H, et al. Identificação de microsporidia em 5 pacientes com AIDS no município de São Paulo. Rev Bras Parasitol Vet 1993;2:95.
48. Moura H, Cardoso RR, Veloso VG, et al. Microsporidia e diarréia crônica em AIDS: identificação de enterocytozoon em 2 pacientes no Rio de Janeiro. In: Congresso da Sociedade Brasileira de Medicina Tropical – Resumos, Fortaleza, 1993;180.
49. Brasil P, Sodré FC, Cuzzi T, et al. Microsporidiose intestinal: estudo de 6 pacientes HIV-positivos no Rio de Janeiro. Rev Soc Bras Med Trop 1994;27(suppl1):145.
50. Wuhib T, Silva TMJ, Newman RD. Cryptosporidial and microsporidial infections in human immunodeficiency virus-infected patients in northeastern Brazil. J Infect Dis 1994;170:490-7.
51. Weber R, Bryan RT. Microsporidial infections in immunodeficient and immunocompetent patients. Clin Infect Dis 1994;19:517-22.
52. Sodré FC. Microsporidiosis humanas: desenvolvimento de métodos para diagnóstico morfológico das microsporidioses intestinais em pacientes HIV-positivos no município do Rio de Janeiro. Master Dissertation, Fundação Instituto Oswaldo Cruz. Rio de Janeiro; 1996.
52. De Paula Amato JG. Aplicação de método modificado de coloração tricrômica, em fezes diarréicas de infectados pelo HIV, para pesquisa de microsporídios. Master dissertation. Faculdade de Medicina da Universidade de São Paulo;1998:74.
53. Weber R, Bryan RT, Ownen RL, et al. Improved light-microscopical detection of microsporidia spores in stool and duodenal aspirates. N Engl J Med 1992;326:161-6.
54. Moura H, Da Silva JL, Sodre FC, et al. Gram-chromotrope: a new technique that enhances detection of microsporidial spores in clinical samples. J Eukaryot Microbiol 1996;43:94S-95S.
55. Blanshard C, Ellis DS, Tovey DG, et al. Treatment of intestinal microsporidiosis with albendazole in patients with AIDS. AIDS 1992;6:331-3.
56. Dietrich DT. Lew EA, Kotler DP, et al. Treatment with albendazol for intestinal disease due to Enterocytozoon bieneusi in patient with AIDS. J Infect Dis 1994;169:178-83.
57. Molina JM, Oksenhendler E, Beauvais B, et al. Disseminated microsporidiosis due to Septata intestinalis in patients with AIDS: clinical features and response to albendazole therapy. J Infect Dis 1995;171:245-9.
58. Cunningham AL, Grohman GS, Harkness J, et al. Gastrointestinal viral infections in homosexual men who were symptomatic and seropositive for human deficiency virus. J Infect Dis 1988;157:386-91.
59. Markell EK, Havens RF, Kuritsubo RA, et al. Intestinal protozoa in homosexual men of the San Francisco Bay area: prevalence and correlates of infection. Am J Trop Med Hyg 1984;33:239-45.
60. Allason-Jones E, Mindell A, Sargeaunt P, et al. Entamoeba histolytica as a commensal intestinal parasite in homosexual men. N England J Med 1986;315:353-6.
61. Esfandiari A, Jordan WC, Brown CP. Prevalence of enteric parasitic infection among HIV-infected attendees of an inner city AIDS clinic. Cell Mol Biol 1995;41:519-23.
62. Cotte L, Rabodorina M, Piens MP, et al. Prevalence in intestinal protozoans in French patients infected with HIV. J Acquir Immune Defic Syndr 1993;6:1024-9.
63. Mendez OC, Szmulewicz G, Menghi C, et al. Comparacion de indices de infestaciones por enteroparasitos entre poblaciones HIV- positivas y negativas. Medicina (Buenos Aires) 1994;54:307-10.
64. Guerin JM, Hoang C, Galian A, et al. Severe diarrhea in a patient with AIDS. J Am Med Assoc 1986;256:591.
65. Jessurun J, Barrón-Rodríguez LP, Fernández-Tinoco G, et al. The

prevalence of invasive amebiasis is not increased in patients with AIDS. AIDS 1992;6:307-9.

66. Alencar LCA, Magalhães V, Melo VM, et al. Ausência de amebíase invasiva em aidéticos homossexuais masculinos, em Recife. Rev Soc Bras Med Trop 1996;29:319-22.
67. Mahmoud AAF. Strongyloidiasis. Clin Infect Dis 1996;23:949-53.
68. Devault Jr GA, King JW, Rohr MS, et al. Opportunistic infections with Strongyloides stercoralis in renal transplantation. Rev Infect Dis 1990;12:653-71.
69. Dias RMDS, Mangini ACS, Torres DMAGV, et al. Ocorrência de Strongyloides stercoralis em pacientes portadores da síndrome de imunodeficiência adquirida (AIDS). Rev Inst Med Trop 1992;34:15-7.
70. Celedon JC, Mathur-Wagh V, Fox J, et al. Systemic strongiloidiasis in patients infected with the human immunodeficiency virus. Medicine 1994;73:256-63.
71. Levi GC, Kallás EG, Leite KRM. Disseminated Strongyloides stercoralis infection in an AIDS patient: the role of suppressive therapy. Braz J Infect Dis 1997;1:48-51.
72. Wong B. Parasitic diseases in immunocompromised hosts. Am J Med 1984;76:479-85.
73. Del Carpio D, Rodríguez D, Vildósola H. Síndrome de hiperinfección por Strongyloides stercoralis en una paciente con infección por el vírus de la immunodeficiencia humana (VIH): reporte de un caso y revisión de la literatura. Rev Gastroenterol Peru 1995;15:290-5.
74. Sarangarajan R, Belmonte AH, Tchertkoff V. Strongyloides stercoralis hyperinfection diagnosed by gastric cytology in an AIDS patients. AIDS 1997;11:394-5.
75. Ferreira MS, Nishioka SA, Borges AS, et al. Strongyloidiasis and infection due to human immunodeficiency virus: 25 cases at a Brazilian teaching hospital, including seven cases of hyperinfection syndrome. Clin Inf Dis 1999;28:154-55
76. Torres JR, Isturiz R, Murillo J, et al. Efficacy of ivermectin in the treatment of strongyloidiasis complicating AIDS. Clin Inf Dis 1993;17:900-2.
77. Stenzel DJ, Boreham PFL. Blastocystis hominis revisited. Clin Microbiol Rev 1996;9:563-84.
78. Jones TC. Blastomycys hominis: more than 70 years of debate regarding pathogenicity. Braz J Infect Dis 1997;1:102-3.
79. El Masry NA, Bassily S, Farid Z, et al. Potential clinical significance of Blastocystis hominis in Egypt. Trans R Soc Trop Med Hyg 1990;84:695.
80. Garavelli PL, Soaglione L, Libanore M, et al. Blastocystis: a new disease in patients with leukemia. Haematologica 1991;76:80.
81. Brites C, Barberino MG, Bastos MA, Silva N. Blastocystis hominis as a potential cause of diarrhea in AIDS patients: a report of six cases in Bahia, Brazil. Braz J Infect Dis 1997;1:91-4.
82. Lee MJ. Pathogenicity of Blastomycia hominis. J Clin Microbiol 1991;29:2089.
83. Zaman V. The diagnosis of Blastocystis hominis in human feces. J Infect 1996;33:15-6.
84. Garavelli PL, Libanore M. Blastocystis in immunodeficiency diseases. Rev Infect Dis 1990;12:158.
85. Hart AS, Ridinger MT, Oundarajan R, et al. Novel organism associated with chronic diarrhea in AIDS. Lancet 1990;335:169-70.
86. Brenan MK, McPherson DW, Palmer J, et al. Cyclosporiasis: a new cause a diarrhea. Can Med Assoc J 1996;155:1293-6.
87. Soave R. Cyclospora: an overview. Clin Infect Dis 1996;23:429-37.
88. Herwaldt BL, Ackers ML, Cyclospora Working Group. An outbreak in 1996 of cyclosporiasis associated with imported raspberries. N Engl J Med 1997;336:1548-56.
89. Centers for Disease Control and Prevention. Outbreak of cyclosporiasis, northern Virginia, Washington, D.C., Baltimore, Maryland, Metropolitan Area, 1997. Morb Mortal Wkly Rep 1997;46:689-91.
90. Mannheimer SB, Soave R. Protozoal infections in patients with AIDS. Infect Dis Clin North Am 1994;8:483-97.
91. Pape JW, Verdier RI, Boncy M, et al. Cyclospora infection in adults infected with HIV: clinical manifestations, treatment and prophylaxis. Ann Intern Med 1994;121:654-7.
92. Goodgame RW. Understanding intestinal spore-forming protozoa: cryptosporidia, microsporidia, isospora, and cyclospora. Ann Intern Med 1996;124:429-41.

Sérgio Cimerman - MD. Instituto de Infectologia Emílio Ribas and Escola Paulista de Medicina, Univerisidade Federal de São Paulo. São Paulo, Brazil.
Benjamin Cimerman - Parasitology Department, University of Mogi das Cruzes. Mogi das Cruzes, Brazil.
David Salomão Lewi - MD, PhD. Infectious and Parasitic Diseases Unit, Universidade Federal de São Paulo / Escola Paulista de Medicina. São Paulo, Brazil.

Sources of funding: Not declared
Conflict of interest: Not declared
Last received: 19 March 1999
Accepted: 7 April 1999
Address for correspondence:
Sérgio Cimerman
Alameda Jauaperí, 890 - Apto. 111
São Paulo / SP - Brasil - CEP 04523-014
E-mail: sc@mandic.com.br

RESUMO

OBJETIVO: Relatar a importância das parasitoses intestinais em pacientes com Aids, mostrando dados relevantes na literatura médica, com enfoque em especial, abordando a epidemiologia, diagnóstico e tratamento das enteroparasitoses, principalmente da criptosporidíase, isosporíase, microsporidíase e estrongiloidíase. **TIPO DE ESTUDO:** Revisão narrativa.
PALAVRAS-CHAVE: HIV. AIDS. Parasitoses.