

CASE REPORTS

Colonic leishmaniasis in a patient with HIV: a case report

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ABSTRACT

Background: To describe an unusual clinical presentation of visceral leishmaniasis affecting the colon.

Case report: We report the case of an HIV-positive patient with visceral leishmaniasis. We describe the clinical case, the procedures performed, the treatment provided and the patient's evolution. A comparative table of previously reported similar cases is shown.

Discussion: Visceral leishmaniasis with intestinal involvement is an uncommon process. Nevertheless, this possibility should be taken into consideration in the differential diagnosis of immunosuppressed patients with symptoms of diarrhea, as a favorable prognosis depends on early diagnosis and appropriate treatment.

Key words: Visceral leishmaniasis. Colonic involvement. HIV.

INTRODUCTION

Leishmaniasis is a disease of the mononuclear phagocyte system. It is caused by a parasite of the species *Leishmania* and transmitted through the bite of the *Phlebotomus* fly, which is endemic in several regions of the world, including the Mediterranean area. In Spain, the most common clinical form encountered is visceral leishmaniasis, which is usually associated with the clinical fever, pancytopenia and hepatosplenomegaly triad (1).

According to previous reports, visceral leishmaniasis is more common in individuals presenting cellular immune deficiency. There is evidence that HIV infection is a risk factor associated with visceral leishmaniasis (2). In addition, HIV-*Leishmania* coinfection is an emerging disease.

Very few cases of colonic *Leishmania* have been described (Table I). We present the case of a patient with HIV and colitis due to *Leishmania* infection.

CASE REPORT

A male patient, aged 49 years, was admitted in October 2014 with abdominal pain and diarrhea. He had a clinical history of stage 3 HIV infection diagnosed in 2005, and chronic HCV infection with ultrasound findings of portal hypertension. He was a heavy smoker (continuing to the present) and had been consuming 100 g of alcohol daily until 2013. He was receiving antiretroviral treatment with abacavir-lamivudine-lopinavir-ritonavir (ABC + 3TC + LPV + RTV), but compliance was poor.

In the days prior to admission, the patient had abdominal cramping and 7-8 diarrheal movements per day, without pathological products, together with a recorded fever of up to 38 °C and general malaise.

Physical examination revealed poor general condition, and a large hyperpigmented lesion on the trunk, in the form of confluent plates, together with furuncle-type lesions with a white centre on both hands, hepatomegaly (6 cm), and moderate splenomegaly.

The only notable findings in the blood analysis were Hb 11.4 g/dl, 3,000/μl leukocytes, 92,000/μl platelets, GGT 119 U/l, FA 122 U/l and C-reactive protein 24.2 mg/L. Abdominal ultrasound examination revealed hepatosplenomegaly, hepatic steatosis and hepatic perihilar adenopathy.

In view of this clinical status, the patient was admitted to hospital, and treatment was started with serum, a strict diet and the reinitiation of antiretroviral treatment, with tenofovir-emtricitabine-raltegravir (TDF-FTC-RAL). A second blood test revealed CD4 44 cells/mm³ and HIV viral load 80,000 copies/mL without resistance to antiretrovirals. CMV viral load was negative. Stool culture was negative, and no fecal parasites were observed; the *Clostridium difficile* toxin test was negative. Blood and urine cultures were both negative. The patient developed acute

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Table I. Reported cases of visceral leishmaniasis with colon involvement

Case	Reference	Age	Immune status	Main symptom	Affected area	Treatment	Result
1	Vargas AM, et al. (7)	61	Immunocompromised: tacrolimus and prednisone by liver transplantation	Diarrhea Asthenia	Colon	Anfotericina B liposomal 5 mg/kg/d 5 days	-
2	Jimeno A, et al. (8)	71	Immunocompromised: azathioprine by ulcerative rectosigmoiditis	Constitutional syndrome Fever	Colon	Anfotericina B liposomal 3 mg/kg/d Days 1-5, 10, 17, 24, 31	Good response
3	Jawhar NM (4)	27	HIV+	Chronic diarrhea	Duodenum Colon	Sodium antimony gluconate	No follow-up
4	Araujo SA, et al. (9)	17	Immunocompromised: tacrolimus and prednisone by liver transplantation	Chronic diarrhea Lost weight	Terminal ileum Colon	Amphotericin B Deoxycholate (impaired renal function) Liposomal amphotericin 3 mg/kg/d 7 days	Good response
5	Hicks L, et al. (10)	24	Immunocompetent	Chronic diarrhea Lost weight	SB Colon Rectum	Liposomal amphotericin 3 mg/kg/d Days 1-5, 10, 21	Good response
6	Baba CS, et al. (6)	35	Immunocompetent	Chronic diarrhea	Colon	Amphotericin B Deoxycholate 50 mg/d 20 days	Good response
7	Marcas A, et al. (11)	-	HIV+	-	Colon	-	-
8	Sebastián JJ, et al. (12)	23	HIV+	Chronic diarrhea	Colon	Amphotericin B	Bad response (no more data)
9	Lemaistre AI, et al. (13)	-	HIV+	-	Colon	-	-
10	Diego J, et al. (14)	-	HIV+	-	SB Colon	-	-
11	Duval JL, et al. (15)	-	HIV+	-	Colon	-	-

SB: Small bowel; HIV: Human immunodeficiency virus.

renal failure by tenofovir, which was substituted by abacavir, with good results.

Thoracic-abdominal-pelvic CT revealed a homogeneous hepatosplenomegaly, with an 18 cm spleen, together with small adenopathies in the chest and abdomen. Bronchoscopy revealed diffuse inflammatory signs, mainly in the left bronchial tree; there were no tumor cells in the bronchial aspirate and the AFB (acid fast bacilli) smear was negative. Colonoscopy to the cecum revealed no lesions. Several random biopsies were taken from the ileum, the cecum, and the colon. The pathology analysis indicated ileal mucosa and of the large intestine with numerous intracellular microorganisms in the histiocyte cytoplasm, in the lamina propria, identified as corresponding to *Leishmania*, although no granulomas were observed (Fig. 1). Further biopsies were performed, of skin from the trunk and the back of the hands, and leishmaniasis was confirmed.

In view of these findings, consistent with visceral and cutaneous leishmaniasis, treatment was initiated with liposomal amphotericin B at a dose of 5 mg/kg/day for 5 days. The diarrheal symptoms and fever disappeared, and oral tolerance was good. During subsequent outpatient treatment to consolidate this recovery, a dose of 200 mg IV was given on days 10, 17, 24, 31 and 38, with good clinical and analytical response to date.

DISCUSSION

Most cases of *Leishmania* in HIV-positive patients appear in advanced stages of the disease, presenting fewer than 200 CD4 cells/mm³ lymphocytes (3). Organomegaly, which is a common clinical manifestation in immunocompetent patients, is usually absent in HIV-positive persons.

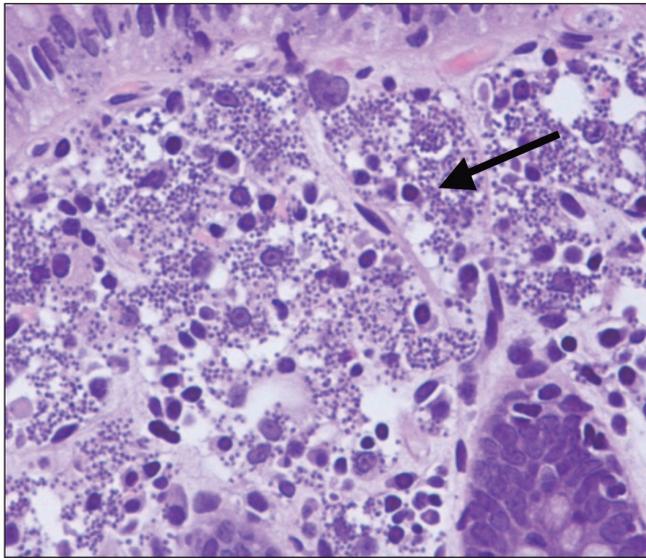


Fig. 1. Colon mucosa with occupation of the lamina propria by numerous histiocytes containing abundant, intracellular, round, and uniform amastigotes of *Leishmania* (hematoxylin-eosin stain).

Cytopenias are more common in immunocompromised patients, together with a higher rate of negative serology and increased disease relapse after treatment for *Leishmania* (3,4).

Atypical locations of visceral disease are more common in HIV patients, especially those with $CD4 < 50$ cells/mm³ and immunocompromised subjects (5). It can affect any part of the body, including gastrointestinal tract, peritoneum, lungs, pleural space or skin (2).

Leishmania can invade any part of the digestive tract, whether asymptotically or manifested as esophageal symptoms, epigastric pain, diarrhea or rectal discomfort. It should be noted that these symptoms may also be caused by other coinfections such as CMV or *Candida* (1). In this respect, the duodenum is the digestive tract location that is most commonly reported (4). The colon is rarely affected; the cases published (most of which include the presence of diarrhea) are summarized in table I.

Intestinal involvement and the consequent malabsorption in visceral leishmaniasis infection are more common in coinfecting patients. The precise mechanism by which this malabsorption takes place remains to be established but a multifactorial mechanism is known to be involved (6).

Endoscopic examination of these patients is usually prompted by epigastric pain and diarrhea. Results are irregular but usually nonspecific, with mild inflammatory alterations and atrophic mucosa. However, in over 50% of

the cases considered there are no macroscopic alterations. This was the case in the present study, in which the biopsy analysis was necessary to confirm the diagnosis (4).

In conclusion, the possibility of infection by *Leishmania* should always be considered in immunosuppressed patients, particularly those who are HIV-positive and present symptoms of diarrhea, and especially if, in addition, they develop skin lesions and cytopaenia, as occurred in the case presented here. The pathologist should also take into account the potential presence of this parasite when examining a sample from the colon of an immunocompromised patient.

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