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An A Typical Case of Compress Visceral Leishmaniasis in a Patient Living with HIV: Diagnosis Error or Challenge?

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ABSTRACT

Visceral leishmaniasis, also known as kala-azar, is a vector-borne parasitic disease that can progress to a life-threatening illness in immunocompromised individuals, especially those infected with HIV.

We report the case of a patient with HIV-1, initially diagnosed through pulmonary tuberculosis and under antitubercular and antiretroviral (ARV) treatment including Tenofovir, Lamivudine, and Dolutegravir, with good virological control (viral load was undetectable) and a CD4 count of 72 cells/mm³, presenting with pancytopenia associated with splenomegaly. The initial diagnosis was likely hematopoietic tuberculosis given the negative bone marrow aspiration and leishmaniasis serology. Nine months into well-managed treatment, the patient continued to have hematological disorders along with splenomegaly and hepatomegaly. Another bone marrow biopsy revealed leishmania bodies, leading to the diagnosis of visceral leishmaniasis. Treatment with liposomal Amphotericin B was started, resulting in a favorable clinical and biological outcome.

Keywords

Visceral leishmaniasis, HIV, Diagnosis, Bone marrow, Antiretrovirals, Amphotericin B.

Introduction

Leishmaniasis is an infection caused by protozoa of the genus *Leishmania that infects the host's macrophages*. The vector is a dipteran, the sandfly. Endemic areas include the Mediterranean basin, extending from the Middle East to Central Asia, and Central and South America. The global prevalence is estimated at 12 million cases and is continually rising. There are major types of leishmaniasis: cutaneous and visceral [1].

Visceral leishmaniasis, also known as kala-azar, is fatal if untreated. It presents with symptoms indicative of sepsis, accompanied by splenomegaly and anemia. There are anthroponotic varieties caused by *Leishmania donovani* and zoonotic varieties caused by *Leishmania infantum* [2].

Visceral leishmaniasis (VL) affects both children and adults, whether immunocompetent or immunocompromised. Since the first cases of VL in AIDS during the 1980s, the classic picture of childhood VL has evolved into opportunistic VL in adults infected with HIV [3]. A new transmission cycle was then described among drug users via syringes, with concurrent transmission of HIV, HCV, and leishmaniasis [4]. The number of co-infected patients has quickly increased, with more than 1,900 cases reported in Italy, Spain, France, and Portugal in the early 2000s. Since the era of highly effective antiretrovirals, a decrease in co-infection cases has been observed [5]. In co-infection with *Leishmania*-HIV, quantitative PCR has simplified diagnostic management due to its non-invasive nature and ability to monitor parasitic load after treatment [6]. Additionally, VL has been described in new immunocompromised populations, such as organ transplant recipients, in connective tissue diseases, chronic inflammatory diseases, and with immunosuppressive treatments (corticosteroids, anti-TNFα, etc.) [4]. Diagnostic and therapeutic management of

VL in immunocompromised patients is a major issue, especially in resource-limited countries [5]. We report the observation of an HIV-positive patient with bone marrow leishmaniasis, shedding light on this issue.

Case Presentation

A 47-year-old man was treated for confirmed HIV-1 infection revealed by pulmonary tuberculosis in 2015, with an initial viro-immunological evaluation showing a viral load of 244,433 copies/ml. He was on ARV treatment with Tenofovir, Lamivudine, and Dolutegravir, which was well-managed but with persistently low CD4 counts at 72 cells/mm³. He was admitted in June 2022 to the Infectious Diseases Department for confirmed pulmonary tuberculosis associated with febrile pancytopenia, including normochromic normocytic anemia (7.6 g/dl), leukopenia (1,720) with neutropenia (640), lymphopenia (780), and thrombocytopenia (66,000), along with an inflammatory syndrome (C-reactive protein at 126) and homogeneous splenomegaly. An etiological workup was performed, including a myelogram, bone marrow biopsy (BMB), leishmaniasis serology, CMV PCR, and macrophage activation syndrome (MAS) evaluation, all of which were negative. A diagnosis of bifocal tuberculosis with confirmed pulmonary involvement and probable hematopoietic involvement was made given the clinical and biological improvement under antitubercular treatment, with anemia improving (Hb at 9.2 g/dl versus 7.6 g/dl, leukocytes 2,900 versus 1,720, and platelet count 120,000 versus 66,000).

Nine months later, the patient presented with asthenia and fever sensations. The evaluation showed pancytopenia (normochromic normocytic anemia at 7.5 g/dl, leukopenia at 1,620 with neutropenia at 840, lymphopenia at 480, and thrombocytopenia at 109,000), splenomegaly, and portal trunk dilation to 13 mm. A myelogram was performed, showing normal marrow richness with the presence of leishmania bodies (Figure 1), while leishmaniasis serology remained negative! A BMB was redone with slides for parasitological and cytopathological study, confirming the diagnosis of bone marrow leishmaniasis. The viro-immunological workup showed an undetectable viral load and CD4 count at 12 cells/mm³.

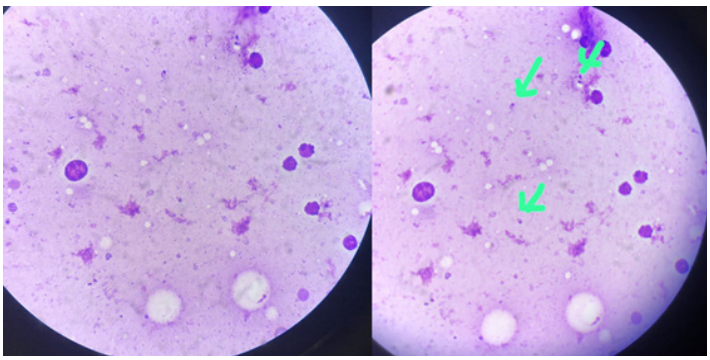

Figure 1: Leishmania bodies visualized on myelogram.

Discussion

Visceral leishmaniasis is a vector-borne parasitic infection transmitted by female sandflies, with humans being an incidental host [7]. It is caused by two *Leishmania* species: *L. donovani* and *L. infantum*, the latter being found in Morocco. Most cases occur in India and Nepal, but the disease can also occur in other geographic areas, including the Mediterranean region [8]. There is a high prevalence among immunocompromised individuals, particularly those with HIV [9], especially in Morocco where the disease sporadically evolves mainly in the northern part of the country with nearly 150 cases per year [10]. Most infections are asymptomatic or minimally symptomatic, but in a minority of patients, the progression is severe leading to symptomatic disease. Clinical and histological manifestations depend on the host's immune response, which largely determines the intensity and effectiveness of this response [11]. The main features are organomegaly, fever, cachexia, and pancytopenia. These signs are non-specific, so diagnosis requires the detection of the parasite in affected organs [12]. Bone marrow aspiration with cytological and parasitological analysis remains the key examination for confirming the diagnosis, as *Leishmania* appears as an intracellular parasite, and histological analysis may show granulomas in infected tissues in immunocompetent subjects, which may be absent in immunocompromised patients, particularly those living with HIV [13,14]. Quantitative PCR on peripheral blood is the simplest and most reliable means for confirming the diagnosis and monitoring post-treatment [6].

Our patient presented clinical symptoms (fever, splenomegaly, and weight loss) and biological symptoms (pancytopenia and inflammatory syndrome) of visceral leishmaniasis a year before the diagnosis was confirmed, with negative leishmaniasis serology, normal myelogram, and BMB, which could be related to the deep immunosuppression the patient experienced with low CD4 counts at 72 cells/mm³ despite well-managed therapy, as demonstrated by several studies showing a positivity rate of bone marrow aspiration from 60 to 80% and serology from 70% [11,15]. The progression in the absence of treatment was marked by persistent fever, asthenia, and hematological disorders, leading to the performance of sternal aspiration, which confirmed the diagnosis of visceral leishmaniasis by the presence of amastigotes of *Leishmania*. Moreover, histological examination showed an increase in macrophages containing leishmaniasis bodies in the bone marrow.

Conclusion

The diagnosis of visceral leishmaniasis in immunocompromised individuals, especially those with HIV, requires high vigilance. Despite the specificity of serological tests, diagnosis often relies on histological examination. In cases of diagnostic delay, especially in resource-limited settings, treatment should be started based on clinical and epidemiological arguments to prevent complications and ensure effective disease management.

References

1. Cossart C, Le Moal G, Garcia M, et al. Manifestations cutanées inhabituelles au cours d'une leishmaniose viscérale associée au VIH. Ann der. 2016; 143: 841-845.

2. Mc Call LI, Zhang WW, Matlashewski G. Determinants for the development of Visceral Leishmaniasis Disease. PloS Pathog. 2013; 9: e1003053.
3. Sallem I, Khibri H, Maamar M, et al. Co-infection leishmaniose viscérale – infection VIH : à propos de 5 cas. PAMJ. 2017; 4.
4. Gangneux JP, Belaz S, Robert-Gangneux F. Mise au point et actualités sur la leishmaniose viscérale méditerranéenne. Journal des Antiinfectieux. 2015; 17: 25-28.
5. Alvar J, Aparicio P, Aseffa A, et al. The relationship between leishmaniasis and AIDS: the second 10 years. Clin Microbiol Rev. 2008; 21: 334-359.
6. Mary C, Faraut F, Lascombe L, et al. Quantification of Leishmania infantum DNA by a real-time PCR assay with high sensitivity. J Clin Microbiol. 2004; 42: 5249-5255.
7. Chappuis F, Sundar S, Hailu A, et al. Visceral leishmaniasis: what are the needs for diagnosis, treatment and control? Nat Rev Microbiol. 2007; 5: 873-882.
8. Hernández-Pérez J, Yebra-Bango M, Jiménez-Martínez E, et al. Visceral leishmaniasis (kala-azar) in solid organ transplantation: report of five cases and review. Clin Infect Dis. 1999; 29: 918-921.
9. FernándezGuerreroML, Robles P, Rivas P, et al. Visceral leishmaniasis in immunocompromised patients with and without AIDS: a comparison of clinical features and prognosis. Acta Trop. 2004; 90: 11-16.
10. Zarrouk A, Kahime K, Boussaa S, et al. Ecological and epidemiological status of species of the Phlebotomus perniciosus complex (Diptera: Psychodidae, Phlebotominae) in Morocco. Parasitol Res. 2015; 115: 1045.
11. Fernandez-Guerrero ML, Pablo Robles, Pablo Rivas, et al. Visceral leishmaniasis in immunocompromised patients with and without AIDS: a comparison of clinical features and prognosis. Acta Tropica. 2004; 90: 11-16.
12. Sundar S. Diagnosis of kala-azar – an important stride. J Assoc Physicians India. 2003; 51: 753-755.
13. Moltó A, Mateo L, Lloveras N, et al. Visceral leishmaniasis and macrophagic activation syndrome in a patient with rheumatoid arthritis under treatment with adalimumab. Joint Bone Spine. 2010; 77: 271-273.
14. Zacarias DA, Nuno Rolão, Flaviane A de Pinho, et al. Causes and consequences of higher Leishmania infantum burden in patients with kala-azar: a study of 625 patients. Trop Med Int Health. 2017; 22: 679-687.
15. Berman JD. Human leishmaniasis: clinical diagnostic and chemotherapeutic developments in the last ten years. Clin Infect Dis. 1997; 24: 684-703.