



Visceral Leishmaniasis–Leprosy Co-infection in a Patient Living with HIV: A Case Study

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Abstract

Leprosy and visceral leishmaniasis are two chronic infectious diseases resulting from intracellular pathogens whose clinical expression is largely the result of host cell-mediated immunity. Yet the concomitant occurrence of them in patients living with HIV remains exceedingly rare. This gives substantial diagnostic and therapeutic difficulties. The work studies a case of a 26-year-old Moroccan man, with HIV infection. Since August 2023 he has been receiving ART triple therapy as well as cotrimoxazole prophylaxis. He presents with a four-month history of progressive, diffuse, infiltrating papulo-nodular cutaneous lesions surrounding the body such as the face, trunk, and limbs, associated with leonine facies, eyebrow depilation, ulcerated nodules, and ENT manifestations. Laboratory workup showed pancytopenia with aregenerative anemia. Bone marrow aspirate showed significant hypoplasia of the three hematopoietic lineages, with significant intra- and extramacrophagic infiltration by *Leishmania spp.* amastigotes. Simultaneously, a skin biopsy showed histological appearance compatible with lepromatous leprosy; and Ziehl-Neelsen staining of nasal mucus suggested many acid-fast bacilli clustered in the form of globi, consistent with *Mycobacterium leprae*. It was also noted there to be two opportunistic intracellular pathogens as co-infection carriers of leprosy in such a severely immunocompromised host. It underscores the imperative to expand diagnostic reasoning in the face of diffuse dermatological and hematological presentations in persons living with HIV, and particularly in tropical or endemic settings and suggests the need for close interdisciplinary collaboration to maximize the recovery detection and management of these rare correlations.

Keywords : Leprosy; HIV; Co-Infection; Visceral Leishmaniasis; Case Study

1. Introduction

Leprosy is a chronic infectious disease caused by *Mycobacterium leprae*. It primarily affects the skin, mucous membranes, eyes, and peripheral nervous system. Its symptoms depend largely on the host's cell-mediated immune response. According to World Health Organization (WHO) definitions, a person with leprosy's clinical signs and symptoms and the need for specific therapeutic management should be seen as having the disease diagnosis [1–3]. Despite WHO-mandated elimination goals, leprosy remains a global pandemic in several regions. Since 2021, there are new cases, with Brazil, India and Indonesia collectively being responsible for over 79% of global cases in 2023 [2,4–6].

Unlike tuberculosis- and atypical mycobacterial infections, which become substantially more frequent with the HIV epidemic, the association between leprosy and HIV has not taken on, in terms of incidence, a central public health role in leprosy-endemic countries. But this correlation poses difficulties for diagnosis and treatment [7], especially with the risk of leprosy reactions and immune reconstitution inflammatory syndrome after receiving antiretroviral therapy.

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Visceral leishmaniasis is a systemic parasitic infection due to the presence of flagellated protozoa of the genus *Leishmania*. In people living with HIV, it is one of the leading opportunistic parasitic infections after toxoplasmosis and cryptococcosis. Immunosuppression promotes an atypical tendency, sometimes severe forms and complicates clinical interpretation when multiple opportunistic infections coexist [8,9]. The present study reports upon the observation of a patient living with HIV receiving antiretroviral triple therapy who had visceral leishmaniasis and lepromatous leprosy detected concurrently.

2. Materials and Methods

2.1. Study Type and Setting

This is a descriptive study of the single clinical observation type (case report), reporting the case of a patient living with HIV in whom visceral leishmaniasis and lepromatous leprosy were diagnosed concomitantly.

Data were collected retrospectively from the patient's medical record. They included anamnestic elements (medical history, mode of revelation of HIV infection, current treatments), clinical features (dermatological, ENT, neurological, and general signs), as well as the results of paraclinical investigations.

Patient anonymity was preserved in accordance with the principles of the Declaration of Helsinki.

- Biological and Morphological Investigations
- The diagnostic workup was based on the following examinations:
- Complete blood count: full blood count and reticulocyte count, to investigate involvement of hematopoietic lineages.
- Bone marrow aspirate: cytological examination of a bone marrow sample, intended to investigate pancytopenia and to identify amastigote forms of *Leishmania* spp. in intra- and extramacrophagic locations.
- Skin biopsy: histopathological analysis of a lesional fragment in search of features suggestive of leprosy.
- Bacterioscopic examination of nasal mucus: detection of acid-fast bacilli following Ziehl-Neelsen staining, with microscopic examination under oil immersion and morphological characterization (length, width, grouping into globi).

3. Results

The case of this report describes a 26-year-old male, single, residing in Tinghir, who presented for HIV infection diagnosed in August 2023 with varicella meningitis and *Pneumocystis pneumonia*. The patient was commenced on antiretroviral triple therapy with cotrimoxazole prophylaxis. Diffuse, chronic, infiltrated cutaneous lesions were present. Symptoms began four months prior with the presence of infiltrated papulo-nodular lesions, initially localized primarily to facial features e.g., eyebrow ridges, earlobes, nose, and then gradually to trunk, back, and limbs over time. The course was a progressive worsening, the lesions becoming edematous, infiltrated, and painful and ENT manifestations consisted of nasal obstruction, nasal voice, and rhinorrhea.

The dermatological examination revealed papulo-nodular lesions and infiltrated, erythematous, symmetrical, and bilateral plaques present in the entire body region. The face was observed to have a leonine facies appearance, with deep wrinkles, cutaneous thickening of the forehead, cheeks, chin, and earlobes. Nodules of ulceration were observed, especially in the lower extremities. Eyebrow depilation, keratosis pilaris, and severe cutaneous xerosis were also noted. There were no neurological, ocular, or osteoarticular findings. The general course of the disease occurred in a context of apyrexia however worsening condition. Laboratory workup showed pancytopenia with normochromic, normocytic, regenerative anemia: hemoglobin 8.3 g/dL, leukocytes 1,560/mm³, platelets 72,000/mm³, and reticulocytes 40,000/mm³.

A thorough examination of the bone marrow revealed a markedly hypocellular marrow with major hypoplasia of all three hematopoietic lineages, due to substantial infiltration by amastigote forms of *Leishmania* spp., in both intra- and extramacrophagic positions. In this setting, a skin biopsy showed histological findings compatible with leprosy, probably of the lepromatous form. Nasal mucus samples were also taken. After Ziehl-Neelsen staining, a microscopic examination under oil immersion confirmed that many non-motile acid-fast bacilli were present as red rods measuring 1–8 µm long and 0.3 µm wide, rounded in their extremities and clustered into globi, compatible with *Mycobacterium leprae*.

4. Discussion

This case demonstrates an exceptional association of visceral leishmaniasis with leprosy in a severely immunocompromised HIV-infected patient. The presence of these two tropical diseases with intracellular tropism within the same immunosuppression context is rarely described. The same scenario was reported by Aquino et al. in 2020 in an HIV-positive patient presenting with severe visceral leishmaniasis during treatment for a leprosy reaction of the erythema nodosum leprosum type. What's more, this observation highlights not only the possibility of coexistence but also the challenge in making conclusions when diagnosing patients who are immunocompromised [8].

Visceral leishmaniasis is much more common in HIV-infected people than in patients who are immunocompetent. If the CD4 number falls below $200/\text{mm}^3$ then it increases the tendency toward this disease. The clinical manifestation of this disease in patients with co-infections is atypical, parasite load elevated, and relapses common, characterized by compromised immune response and activation of viral replication [8,10]. Leishmania in its systemic, visceral, typical form invades the main sites of bone marrow, spleen, and liver. But particularly in people who are HIV-positive, unusual cutaneous features are increasingly being described. These lesions may develop during disease progression or represent the mode of disease revelation [10]. In relation to leprosy, the published evidence supports that infection with HIV does not increase the incidence of leprosy in a systemic population. Nevertheless, it changes the immunologic milieu, can induce multibacillary or atypical forms, and dramatically elevate the risk of leprosy events, especially after starting antiretroviral therapy. These reactions, albeit sometimes severe, may be considered as an immune reconstitution inflammatory syndrome based on the framework of inflammation [11,12].

Leonine facies, diffuse infiltration of cutaneous lesions, the involvement of the earlobes, and the multiple acid-fast bacilli in the nasal mucus all strongly suggested lepromatous leprosy for the patient. The pancytopenia, in turn, encouraged bone marrow examination which led to the diagnosis of visceral leishmaniasis. Considering the diffuse dermatological manifestations and the hematological involvement of the case of a patient living with HIV, expanded diagnostic reasoning is suggested to include multiple concurrent opportunistic or tropical infections, as this presentation emphasizes. It is also a reminder of the value of an integrated approach that integrates infectious diseases, dermatology, anatomical pathology, parasitology, and microbiology.

Therapeutically, the management of these co-infections is still rather complicated. Multidrug therapy is still the gold standard treatment for leprosy given the frequent risk of leprosy reactions and the need for ongoing monitoring of the disease. Visceral leishmaniasis, by contrast, needs specific therapy, typically liposomal amphotericin B in HIV-positive patients, with the possibility of secondary prophylaxis, because of the high risk for relapse [11].

5. Conclusion

This work highlights an uncommon co-infection with visceral leishmaniasis and leprosy in a patient living with HIV. This demonstrates the critical need to maintain a high level of clinical suspicion in a tropical context and to develop a multimodal diagnostic strategy for atypical presentations in immunocompromised patients.

Further emphasis is given to the importance of the cooperation between clinicians, dermatologists, infectious disease specialists, anatomical pathologists, and microbiologists to maximize the identification and management of these rare associations. More in-depth studies would be useful to provide greater insights into the true frequency of such co-infections, their pathophysiological interactions, and the most appropriate therapeutic strategies in resource-limited settings.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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