



## RESEARCH ARTICLE

### DIFFUSE LEPROMATOUS LEPROSY IN AN HIV-POSITIVE PATIENT

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#### Abstract

Leprosy is a disease known since ancient times. It is caused by *Mycobacterium leprae*. The skin, peripheral nerves, eyes, and mucous membranes of the upper respiratory tract are especially affected. Lepromatous leprosy is a clinical entity that has become rare worldwide today with the introduction of polydrug therapy, resulting in a dramatic decline in overall prevalence. In epidemic settings, atypical manifestations should be known because they are a source of disease transmission. Lepromatous leprosy rarely occurs as part of an immune deficiency. We report a case of diffuse leprosy in an HIV-positive patient.

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#### Introduction:-

Leprosy or Hansen's disease is a disease known since ancient times. Historical sources and paleopathology have uncovered two ancient foci in Egypt and India. It is caused by *Mycobacterium leprae* (ML). The skin, peripheral nerves, eyes, and mucous membranes of the upper respiratory tract are especially affected. Lepromatous leprosy is a clinical entity that has become rare worldwide today with the introduction of polydrug therapy, resulting in a dramatic decline in overall prevalence. We report a case of diffuse leprosy in an HIV-positive patient.

#### Case Report:-

A 41-year-old single patient was treated for retrovirus infection for 4 years and had skin lesions evolving for 3 and a half years, which were painless and non-pruritic and diffused on the face, trunk, and limbs. The general condition was not good, and there was no fever. A general examination revealed a hemodynamically, respiratory stable, and afebrile patient. Dermatologic examination revealed multiple firm, shiny papules, and nodules, brown, purple, and copper in color, symmetrical, bilateral, painless, normoesthetic, smooth surface, multiple, variable in size, located on healthy skin on the face, ears, thorax, back, limbs, and palms. We also noticed rough and warty patches on the back of the hands and achromatic, brown, and purple patches on the back. (Fig. 1) Peripheral nerve palpation and sensory and motor examination were normal, and the rest of the physical examination was normal. Skin biopsies of nodules and verrucous plaques revealed that the dermis was heavily occupied by a diffuse inflammatory infiltrate with peri-sweat and perivascular reinforcement. This infiltrate consisted mainly of histiocytes with pale cytoplasm, had the appearance of Virchow's foam cells, and contained numerous Hansen's bacillus grouped in globi, confirming the diagnosis of high-grade bacterial leprosy. (Fig. 2, 3, 4)

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The blood test revealed a CD4 count of 350, a reduced viral load, and negative liver and syphilis serology. Other checks are normal. multibacillary polydrug therapy was prescribed, in combination with rifampicin, clofazimine, and dapsone, while maintaining triple antiretroviral therapy.

### Discussion:-

Leprosy is a disease known since ancient times. It is a contagious disease caused by *Mycobacterium leprae*. The bacillus reservoir is the patient, and *Bacillus leprae* is excreted through the nasal mucosa and skin into wounds or ulcers. [3] In 2005, the World Health Organization announced the elimination of leprosy as a public health problem. By then, the prevalence of leprosy in the world's population should be lower than 1/10,000. This elimination has not yet been achieved, although the global prevalence has declined: from 8.4/10,000 in 1966 to less than 1/10,000 by the end of 2000. [4] The number of new cases detected is declining: 17,396 in 2020, 202,185 in 2019, and 244,796 in 2009. The 2020 numbers are much lower than in previous years, likely due to a reduction in case detection and reporting during the COVID-19 pandemic. Although the reduction observed in 2020 has nothing to do with real epidemiological developments, these figures demonstrate that there are still significant stocks of Hansen's bacillus in leprosy-endemic countries, especially in South-East Asia, especially since they are present in all endemic countries, in some countries, the proportion of new cases of contagious multibacillary leprosy (MB) is high, exceeding 90%. [4.7]

If the prevalence is  $\geq 0.5/1000$ , the entire population is at risk of developing leprosy. After prolonged exposure to *Mycobacterium leprae*, the vast majority develop effective immunity without clinical manifestations, while less than 5% develop a wide range of clinical manifestations, including the five forms of leprosy, indeterminate leprosy, or Only transient clinical form of leprosy, purely cutaneous. Thus, Ridley and Jopling (1962) [8] classified leprosy into five types: TT, LL, BB, BL, and BT (no type I). Thus, the different forms of leprosy are determined by the underlying immune response to *M. leprae*. [8]

Tuberculoid Leprosy (TT) patients exhibit a strong cellular response to *M. leprae*, limiting disease to a few well-defined skin lesions and nerve trunks. The lesions are infiltrated with interferon-gamma (IFN- $\gamma$ )-secreting CD4+ T cells, resulting in well-defined granulomas containing epithelioid cells and multinucleated giant cells around the cutaneous nerves. [9]

Patients in leprosy poles (LL) do not have specific cellular immunity. Uncontrolled bacterial proliferation with numerous lesions and inflammatory infiltration of the skin and nerves. The dermis contained bacilli-filled foamy macrophages but few CD4+ and CD8+ lymphocytes, with disorganized granulomas. There were high antibody titers against PGL-1 and *M. leprae*-specific antigens. [10] Most patients have intermediate forms of BT, BB, and BL. These forms are characterized by a gradual regression of cellular responses from BT to BL, which is associated with increased cellular burden, more frequent skin and nerve damage, and higher antibody titers. [11] Leprosy is a poly bacillary form that typically presents with noninflammatory lesions, hypopigmented macules, and normal-appearing erythematous papular nodules with a progressive appearance, as was the case in our patient. Diffuse infiltrates of the skin are less common and usually follow the disseminated plaque phase. On the face, it begins at the arch of the brow, the bridge of the nose, and the ears. Eyebrow hair loss is often associated with it. The hands and feet are chubby. After a few years of evolution, we end up with a lion-like face. This superficial infiltration is often associated with leprosy tumors, which makes the diagnosis possible. Nerve damage was found alongside these skin signs. [12] However, the present patient's clinical presentation in the form of verrucous plaques associated with lepromatous is rare. In 2014 there were only 25 cases of verrucous leprosy. [13,14] Within 5 months of the introduction of antiretroviral drugs, HIV-infected patients were 18 times more likely to develop leprosy than untreated patients, as was the case in our patients. Thus, leprosy can manifest as inflammatory immune reconstitution syndrome (IRIS). This type of IRIS was defined as leprosy and/or type 1 or type 2 reactive status in severely immunocompromised HIV-infected patients (CD4 < 200/mm<sup>3</sup>) within 6 months of the introduction of antiretroviral drugs, combined with classic IRIS Criteria (>1 log reduction in viral load and CD4 increase). [15,16,17]

CD4 T lymphocytes, the main target cells of HIV infection, are normal in numbers in biopsies of leprosy lesions. The dermis contains bacilli-filled foamy macrophages, but few CD4+ and CD8+ lymphocytes, and no tissue granulomas, so the risk of HIV-infected cell recruitment is lower compared with tuberculous granulomas. This paradox has only been studied in HIV paucibacillary leprosy co-infected patients, so similar results cannot be

extrapolated due to the lack of studies in this patient group. [18,19] The poorer cell-mediated immune response in leprosy is secondary to low peripheral CD4 counts, reflecting a parallel, albeit independent, process. [20,21]

It can be hypothesized that leprosy in HIV patients is associated with very low peripheral CD4 counts, suggesting strong immunosuppression. This type of co-infection is rare, possibly due to the long incubation period of leprosy, since by the time HIV is diagnosed, patients may have died of opportunistic infections associated with the AIDS stage before lepromatous leprosy developed. Thus, the lack of information in medical research may also contribute to the underdiagnosis of leprosy in HIV-positive patients. [2,22,23]

### Conclusion:-

HIV testing for leprosy should be considered in patients with atypical skin findings such as verrucous lesions. Physicians must be cautious in ordering HIV screening tests in all suspected cases, whether they are atypical or not. The independent course of HIV and leprosy and the maintenance of local T cell-mediated immune responses may explain the response to combined chemotherapy in HIV and leprosy co-infected patients. HIV is treated with highly active antiretroviral therapy and leprosy with combined chemotherapy. The therapeutic goal of these two distinct diseases is to address the patient's cell-mediated immune deficiency, which appears to be the main cause of infection in both diseases.





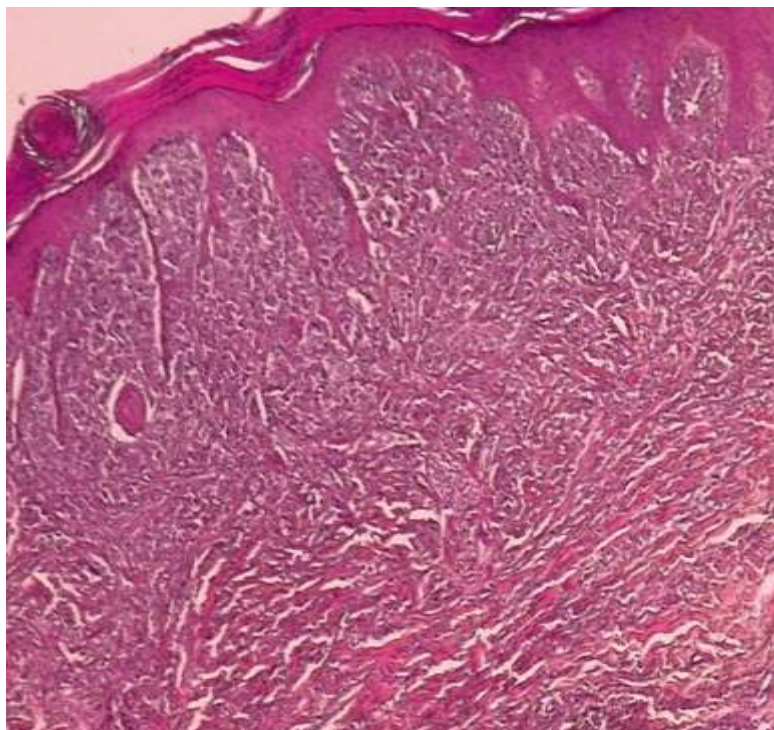
**Figure 1:** Lepromatous leprosy in an HIV-positive patient

a, b: Diffuse lepromas of the face and ears

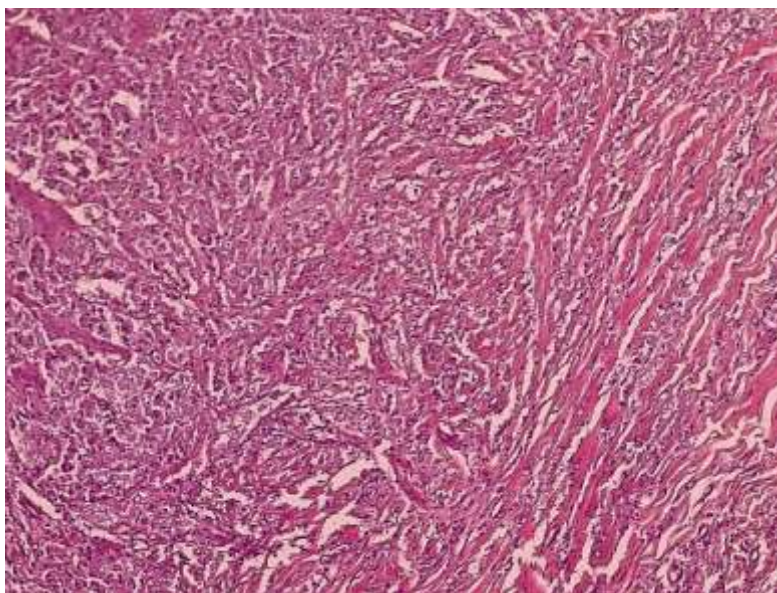
c, d: Lepromas and rough and verrucous patches of the hands

e, f: Dyschromic macules and pigmented papules of the trunk and back

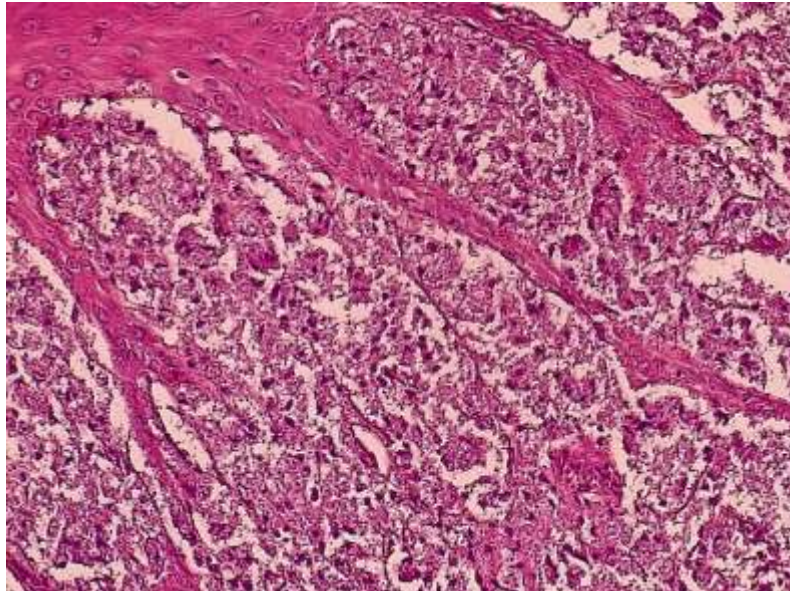




**Figure 2:** The dermis is massively occupied by a diffuse inflammatory infiltrate of histiocytic appearance (HE x 4)



**Figure 3:** Histiocytes show pale cytoplasm taking on the appearance of Virchow's foam cells (HE x 10)



**Figure 4:** Virchow's cells containing very many Hansen's bacilli grouped in globi (HE x 40)

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