

Lepromatous leprosy as a presenting feature of HIV

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Abstract

Various bacterial, mycobacterial and fungal opportunistic infections occur frequently in immunocompromised individuals, however, leprosy in retroviral disease is a relatively rare association. Hereby, we report a case of lepromatous leprosy that presented with clinical features mimicking other opportunistic infections and subsequently led to the diagnosis of HIV. The myriad challenges associated with the diagnosis and management of HIV–leprosy coinfection are also discussed. Thus, although uncommon, atypical cutaneous lesions in HIV-seropositive patients warrant investigation for leprosy.

Key words: HIV, lepromatous leprosy, opportunistic infections

INTRODUCTION

Leprosy patients with HIV clinically present similar to patients with leprosy in the general population. Sometimes, on starting antiretroviral treatment (ART), subclinical leprosy manifests as clinical leprosy or the preexisting leprosy worsens immune reconstitution inflammatory syndrome (IRIS).^[1] HIV-positive patients with low immunity may present with various opportunistic infections such as cutaneous cryptococcosis, deep fungal infections, extensive molluscum contagiosum, and extensive viral warts.^[2] Our case of lepromatous leprosy is reported for its rare opportunistic infection-like manifestations. Thus, leprosy can be

considered the presenting feature in this patient that led to the detection of his HIV-positive serostatus.

CASE REPORT

A 50-year-old married male presented with asymptomatic reddish raised lesions over the face, trunk and extremities for 1 month, with a history of anorexia, progressive weight loss and mild abdominal pain.

On examination, multiple erythematous to skin-coloured papules and nodules were present on

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the face, ears, trunk and extremities with a single verrucous lesion over the nose [Figures 1 and 2]. Hyperkeratotic nodules were present over the palms and soles. Both ulnar nerves were thickened and nontender. Sensations were intact.

A differential diagnosis of secondary syphilis, cryptococcosis, histoplasmosis and Hansen's disease (papulo-nodular lesions) and verruca vulgaris (wart-like lesions) was considered.

Routine blood investigations were normal. Venereal Disease Research Laboratory and *Treponema pallidum* hemagglutination assay were negative. Enzyme-linked immunosorbent assay for HIV 1 was positive. CD4 count was 11. Slit-skin smear showed a bacillary index (BI) of 5+. To confirm the diagnosis, skin biopsies from multiple sites were performed. Histopathology of all the specimens (papular, nodular and verrucous) showed perivascular foamy macrophages with polymorphs (vasculitic changes) extending up to the panniculus suggestive of lepromatous leprosy with erythema nodosum leprosum (ENL). Ziehl-Neelsen stain (Fite-Faraco modification) was positive for acid-fast bacilli (AFB) with BI of granuloma of 6+ [Figures 3 and 4]. Investigations for all other opportunistic infections were negative.

Abdominal ultrasonography revealed lymphadenopathy suggestive of abdominal tuberculosis. Mantoux test and sputum (AFB) were negative.

A final diagnosis of lepromatous leprosy with ENL and abdominal tuberculosis in an HIV-positive patient was made.

He was started on antitubercular therapy (three daily tablets of a fixed-dose combination comprising isoniazid 75 mg, rifampicin 150 mg, ethambutol 275 mg, and pyrazinamide 400 mg). In addition, WHO-multiple drug therapy (multibacillary [MB]) consisting of daily dapson 100 mg and clofazimine 50 mg along with thalidomide 100 mg BD and clofazimine 100 mg TDS (reactional dose) was prescribed. Liver function tests were monitored. ART consisting of daily TLE regimen (tenofovir 300 mg, lamivudine 300 mg and efavirenz 600 mg) was started. Currently, the patient is under regular follow-up with gradual regression of lesions and no further ENL episodes over the past 3 months.

DISCUSSION

Despite declaring elimination in December 2005, leprosy is still endemic in some parts of India.^[1] Although HIV

coinfection significantly changes the natural history of many diseases, literature does not describe a major change in the course of leprosy in people living with HIV.^[2] This is probably due to inadequate knowledge of the natural history of coinfecting patients and paucity of studies due to low incidence and long incubation period of leprosy. Some studies in Tanzania^[3] and Zambia^[4] showed a small increase in HIV prevalence in leprosy patients. However, the sensitivity and specificity of serological tests for HIV are affected in leprosy, giving rise to false-positive results.

In HIV-positive patients, as cell-mediated immunity is reduced, MB leprosy is expected to be observed more often than paucibacillary (PB). However, many studies have shown that there is no such rise in MB cases.^[3,4] The relatively long incubation period for lepromatous disease might be responsible for greater predilection for tuberculoid disease, as patients might die of AIDS-related complications before manifesting lepromatous disease.^[2]

Nearly one-third of HIV-infected individuals experience some peripheral nerve damage, manifesting as paresthesia and stiffness. Distal sensory polyneuropathy is the most common neurologic complication, although mononeuropathy (either affecting only a portion of one limb or multiple nerves in an asymmetric fashion) may occasionally occur. Notably, there is no sensory loss in HIV neuropathy.^[5] In contrast, lepra bacilli invade Schwann cells and axons, leading to demyelination and axonal degeneration, causing sensory, motor and autonomic neuropathy. Therefore, in leprosy, the typical pattern is mononeuritis multiplex with hypo/anesthesia, motor weakness and anhidrosis. HIV might act synergistically with *Mycobacterium leprae* to worsen nerve damage in dually infected persons.

Increased incidence of acute neuritis and Type 1 reaction was seen in HIV patients with MB (borderline lepromatous [BL]), but not PB leprosy, while ENL reactions were rarely reported until recently.^[2] A debatable issue is the safety of long-term steroids in immunosuppressed patients presenting with AIDS, leprosy and lepra reactions. Previous data have shown that short-term steroid therapy may be used with minimal complications in this challenging scenario.^[6] In coinfecting patients, significant clinical improvement is seen with early ART initiation, with reduction in steroid dose requirement during reactions.^[7] The use of rifampicin with protease inhibitor-based ART is problematic because of the potential for drug interactions. Rifampicin is a potent inducer of the cytochrome P450-3A4 subenzyme, responsible for the metabolism of protease

inhibitors (PIs). This may result in subtherapeutic concentrations of the PI, thus increasing the risk of treatment failure and virological resistance. However, as the dose of rifampicin is only monthly in leprosy, no such effects are seen.^[8] Often, in patients with latent leprosy, after starting ART, the immune system recovers and clinical leprosy starts to manifest, whereas in others with clinical leprosy, there is worsening of the existing lesions. This phenomenon is called IRIS.^[1]

Rarely, unusual presentations of leprosy such as granuloma annulare-like,^[9] erythema multiforme-like,^[10] Sweet's syndrome-like,^[10] systemic lupus erythematosus-like,^[11] and verrucous lesions^[12] are seen. Diagnosis of leprosy, often missed in HIV patients, is unraveled subsequent to ART initiation and IRIS.

Talhari *et al.* have reported a treatment-naïve seropositive male with neurocysticercosis who

presented with disseminated infiltrated lesions diagnosed as BL leprosy. His CD4 count was 6.^[13] Camaclang and Cubillan recently reported a similar case with disseminated verrucous papules and plaques with infiltrated papules on the face, diagnosed as lepromatous leprosy. HIV (tested due to the atypical presentation) was positive. CD4 count was 106.^[14] Our patient presented with papular, nodular and verrucous lesions, simulating opportunistic infections such as cutaneous cryptococcosis, histoplasmosis and secondary syphilis. This prompted us to investigate for HIV which tested positive. Interestingly, the patient's serostatus had remained hitherto undetected. Furthermore, there were no classical skin lesions of leprosy, clinical neuritis or sensory impairment. The only clinical clue suggestive of leprosy was thickened ulnar nerves. Another confounding feature was the lack of symptoms or signs of reaction, probably attributable to his ART-naïve advanced retroviral disease (AIDS stage) with severe T- and B-cell dysregulation and dysfunction. Diagnosis of lepromatous leprosy with ENL could be made only after bacteriological and histopathological examination. IRIS was ruled out as he was not on ART prior to the appearance of lesions (criteria were not fulfilled). The present



Figure 1: Erythematous to skin-colored nodules on the face, nose, and ear



Figure 2: Erythematous papules and nodules over both legs with crusting over some lesions

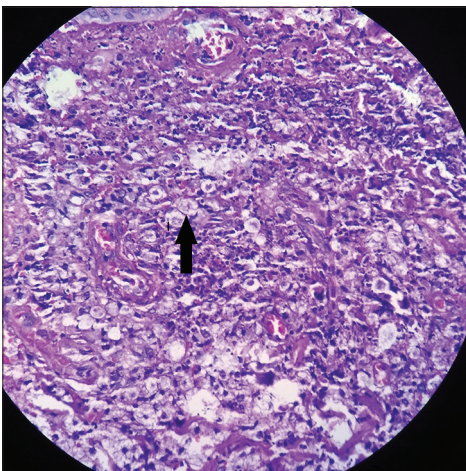


Figure 3: H and E (×40) from nodule – abundant foamy macrophages seen in the dermis, suggestive of lepromatous leprosy

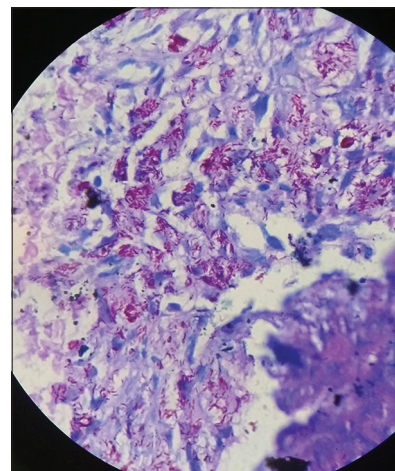


Figure 4: Large number of lepra bacilli seen on Ziehl-Neelsen stain (×40)

case as well as previously documented patients^[13,14] suggest that atypical manifestations of leprosy in HIV-positive patients could be a harbinger of progressive immunosuppression, consistent with the “opportunistic leprosy” subtype (clinical classification proposed by Talhari *et al.*).

CONCLUSION

Although the clinical presentation of leprosy in HIV is more or less similar to that of the general population, the diagnosis of leprosy might be missed in such individuals due to unusual presentations masquerading as other diseases. Therefore, atypical cutaneous lesions in HIV-seropositive patients warrant investigation for leprosy, the emerging “great imitator.”

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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