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CASE REPORT

HIV and leprosy in a 27-year-old Filipino male: A case report

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ABSTRACT

INTRODUCTION Hansen's disease or leprosy is a chronic infectious disease caused by *Mycobacterium leprae* associated with inflammation that may damage the skin and peripheral nerves. In countries where leprosy is still endemic, an increasing prevalence of human immunodeficiency virus (HIV) can be seen, hence increasing the possibility of HIV-leprosy co-infection. Hansen's disease, if not treated promptly, can cause scars and deformities associated with leprosy reaction. Immunosuppressive drugs like corticosteroids used in the treatment of leprosy reaction may put the patient at risk of opportunistic infections.

CASE REPORT This is a case of a 27-year-old Filipino male with HIV-leprosy co-infection, who manifested with erythema nodosum leprosum reaction, treated with tapering dose of oral corticosteroids and multidrug therapy (MDT) for multibacillary leprosy showing good response to treatment after 5 months without recurrence of reaction. The use of chronic oral corticosteroids, despite its immunosuppressive effects, has been beneficial in the management of reactions in this patient with HIV-leprosy co-infection.

CONCLUSION Considering that both Hansen's disease and HIV directly affects T helper CD4+ lymphocytes in its pathogenesis, there seems to be little to no alteration in the course of patients with HIV-leprosy co-infection. Hence, treatment of HIV-leprosy co-infection does not differ from that of a seronegative leprosy patient. This case highlights the occurrence of erythema nodosum leprosum reaction in HIV-leprosy co-infection and the need for immunosuppressive drugs to control reaction and prevent nerve damage. Close monitoring is imperative to weigh the risk-benefit ratio of medications given to patients with HIV-leprosy co-infection.

KEYWORDS Leprosy reaction, Hansen's disease, Human immunodeficiency virus

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INTRODUCTION

Co-infection with HIV has been a challenge in managing many infectious diseases, particularly mycobacterial diseases. Most patients reported to have HIV-leprosy co-infection had paucibacillary leprosy, with exacerbation of lesions during a course of immune reconstitution inflammatory syndrome (IRIS) associated with initiation of highly active antiretroviral treatment (HAART). Rarely, multibacillary lepromatous leprosy exists in the setting of HIV.[1]

There is limited data on the course of leprosy in HIV co-infected patients, hence this case report aims to describe the manifestations of lepromatous leprosy in an HIV co-infected patient and the satisfactory prognosis with the use of chronic corticosteroids in the management of leprosy reactions in the immunocompromised.

CASE REPORT

A 27-year-old male, diagnosed case of HIV since February 2018, who was already receiving efavirenz 600mg/emtricitabine 200mg/tenofovir 300mg for 75 weeks, with an improved CD4 count of 406 after 6 months of HAART (baseline CD4 count 124) and a viral load of <34 copies/mL consulted at our institution. He initially presented with a few hypopigmented, hypoesthetic plaques on the elbows 11 months prior to consulting. Lesions increased in size and number, evolving into multiple erythematous papules, plaques, and nodules on the face, ears, and extremities associated with hypoesthesia. The patient was previously seen by a physician and was given betamethasone valerate ointment and prednisone 20mg/day for 1 week with minimal improvement. The persistence of lesions prompted consult at our institution. Past medical history showed late latent syphilis treated with benzathine penicillin G 2.4M units intramuscular injection for 3 doses in 2018. Family history was unremarkable. He is currently in a monogamous sexual relationship

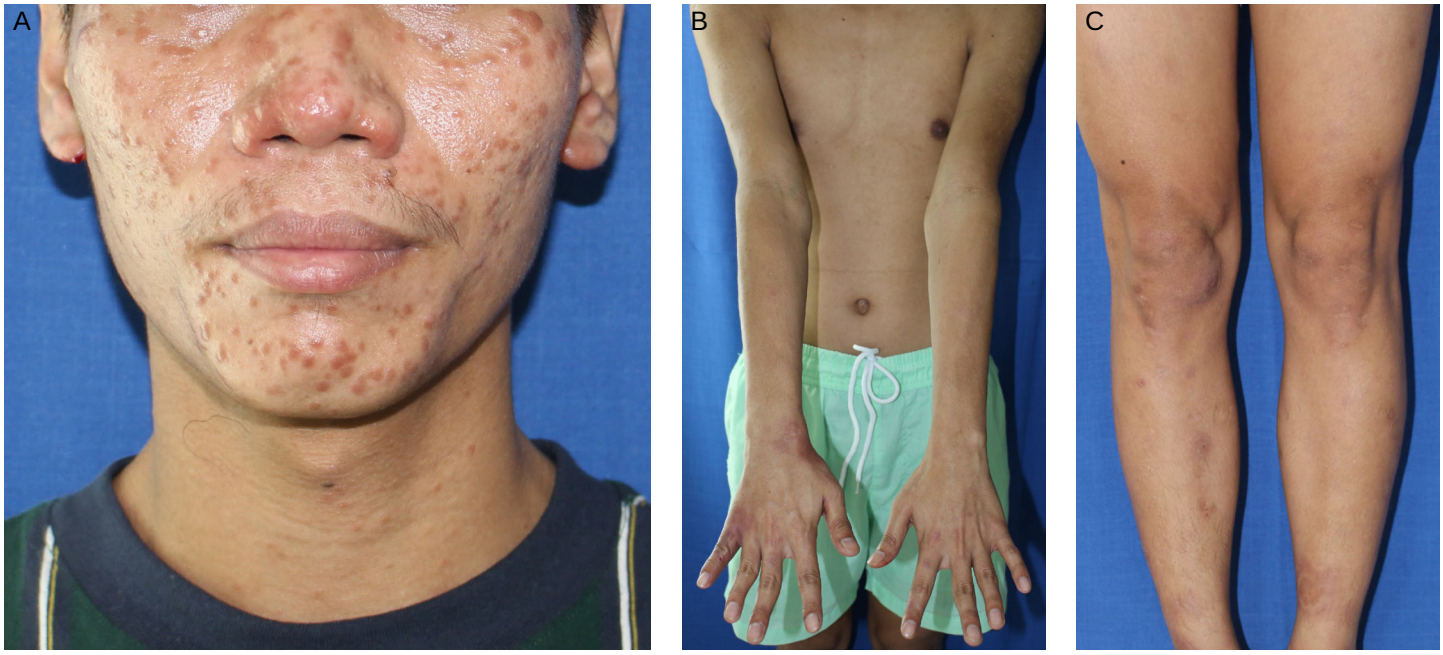

Figure 1. A to C. Patient on first consultation presenting with multiple, well-defined, erythematous papules, plaques and nodules on the face, ears, and extremities.

with a male partner and works as a call center agent.

On physical examination, there were multiple, well-defined, round to irregularly-shaped erythematous papules, plaques, and nodules that range in size from a few millimeters up to 2 to 3 centimeters in diameter on the face, ears, and extremities (Figure 1). There were no palpable peripheral nerves. Caloric testing revealed 0% sensation on cold and hot temperatures, and 50% sensory deficit on soft touch and pinprick on lesional skin. A 4-mm skin punch biopsy stained with H&E revealed mild thinning of the epidermis, subepidermal grenz zone, and a dense, nodular, granulomatous inflammatory infiltrate of foamy histiocytes, and many lymphocytes, surrounding adnexal structures and extending to the deep dermis (Figure 2). Histopathological diagnosis was Hansen's disease, lepromatous leprosy. A slit skin smear revealed a Bacillary Index (BI) of 1.83+ with all *M. leprae* bacilli in solid form. Chest radiograph, complete blood count, urinalysis, G6PD assay, kidney, and liver function tests, as well as the rest of the metabolic parameters were normal. The patient was then started on multidrug therapy-multibacillary regimen (MDT-MB) blister pack every 28 days. However, after the 3rd cycle, due to unavailability of MDT blister pack, the patient was switched to second-line drugs rifampicin 600mg/tab once a month, ofloxacin 400mg/tab once daily, and clarithromycin 500mg/tab once daily, but had poor adherence and subsequently lost to follow-up.

Five months after initiating MDT-MB, with good compliance with HAART for 107 weeks, the patient came in for a follow-up with a noted elevation of old lesions and appearance of new plaques and nodules with sizes ranging from a few centimeters up to 6 centimeters in diameter, on the face, arms and bilateral anterior legs. This was accompanied by fever, arthralgia, and myalgia. Partial clawing of the right hand was also noted (Figure 3). A diagnosis of Hansen's disease lepromatous leprosy, in reaction, was made and the patient was resumed on 4th cycle of MDT-MB blister pack and was given prednisone 40mg/day for 2 weeks, with gradual tapering in decrements of 10mg/day every 2 weeks until the oral corticosteroid is discontinued. The patient has been compliant with medications and had complet-

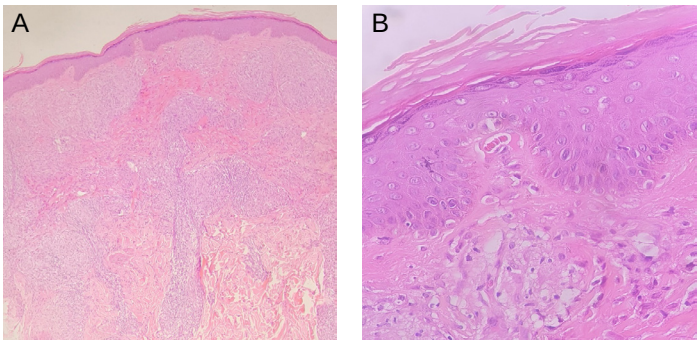

Figure 2. A. Epidermis showed mild thinning and a subepidermal clear grenz zone (H&E, 10x). **B.** A nodular, granulomatous inflammatory infiltrate composed of foamy histiocytes and lymphocytes is observed on the dermis (H&E, 40x).

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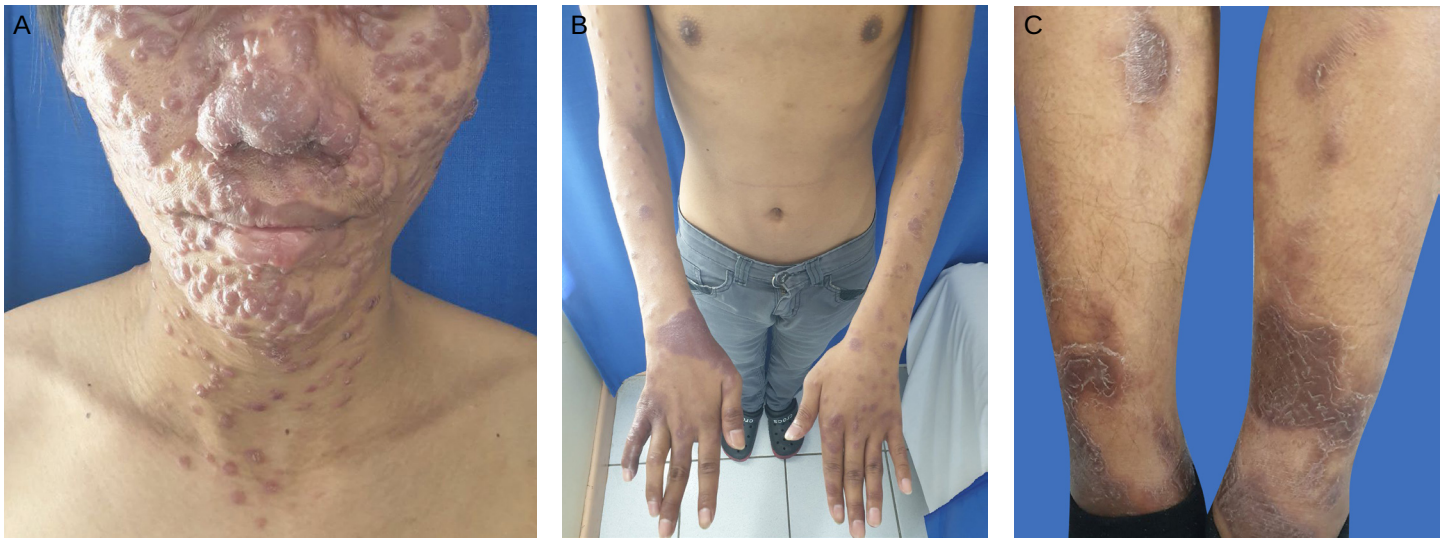

Figure 3. A to C. Patient in leprosy reaction showing multiple, well-defined, erythematous to hyperpigmented papules, plaques and nodules on the face, ears, neck, upper and lower extremities.

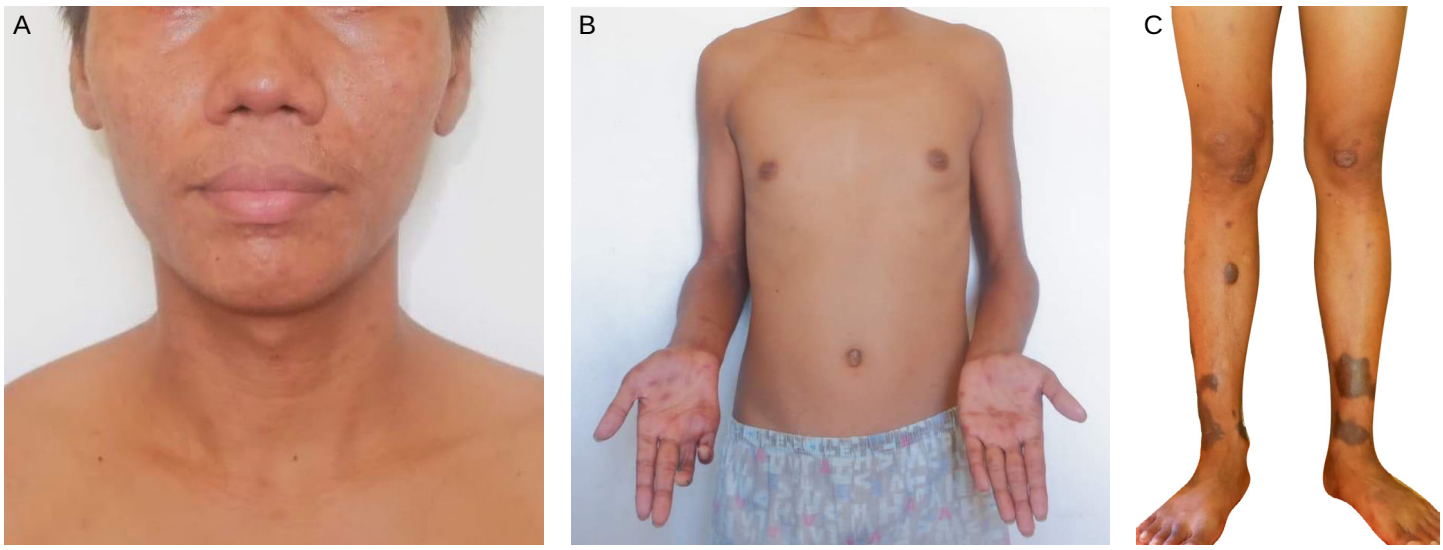

Figure 4. A to C. Patient after completion of 7 cycles of MDT-MB blister pack and 10 weeks of tapered dose of prednisone, with no recurrence of reaction, showing multiple, well-defined, erythematous to hyperpigmented patches, plaques and nodules on the face, neck, palms, upper and lower extremities.

ed 7 cycles of MDT-MB blister pack with no noted recurrence of reaction (Figure 4). Our plan is to complete 12 cycles of MDT-MB blister packs for our patient.

DISCUSSION

HIV and Hansen's disease are diseases that affect cell-mediated immunity. It was initially expected that the decrease in CD4+ cells seen in HIV patients would result in an increase in susceptibility to leprosy, just as in *mycobacterium tuberculosis*. But studies have found that both diseases exist independently, and that HIV does not significantly affect the pathogenesis of neural or skin lesions.[2,3] It is however possible that all types of leprosy can occur in co-infected patients, with the predominance of paucibacillary cases in most published reports. In a study by Deps et al., HIV patients with low CD4+ count had borderline tuberculoid lesions with well-formed granuloma and normal numbers of CD4 cells.[4] On the other hand, patients with lepromatous leprosy had loose inflammatory infiltrates comprised of macrophages and a small number of CD8+ lymphocytes.[1,2]

Several reports have suggested that initiation of HAART

has been associated with the occurrence of reversal reaction and IRIS-associated reversal reaction, with the diagnosis of leprosy increasingly being reported in this new clinical syndrome. IRIS is the paradoxical worsening in clinical status after starting HAART, attributable to an increase in production and redistribution of CD4+ cells, and increased immune responses to other opportunistic pathogens due to inhibition of regulatory T cell function in suppressing pro-inflammatory cytokines responsible for the focal and systemic signs of inflammation.[1,3,5] Lockwood and Lambert proposed a defining criteria for the correct identification of leprosy IRIS, which includes the following: (1) leprosy and/or leprosy reaction presenting within six months of starting HAART; (2) an advanced HIV infection; (3) low CD4+ count (<200 cells/mm3) before initiating HAART; (4) CD4+ count increasing after starting HAART.[1,2] Our patient presented with an advanced HIV infection, with baseline CD4+ count of 124 improved to 406 after 6 months of starting HAART. Although the patient did not meet the diagnostic criteria initially proposed by the authors, with lepra reaction occurring after 107 weeks of starting HAART, a few leprosy IRIS cases presented with other timings. This can be explained by considerable individual variation in the reconstitution of both CD4+ and memory CD4+ T cells, which may persist up to 48 weeks of starting HAART. Hence, they revised a new classification identifying four possible situations of Leprosy IRIS in HIV patients. Particularly, they identified a group of HIV patients on HAART who were later diagnosed with leprosy and developed lepra reactions later after starting MDT.[4]

Our patient presented with type 2 erythema nodosum leprosum reaction, with sign of deformity after being non-compliant with the MDT regimen. Data have suggested that over-all peripheral nerve damage is higher in HIV leprosy co-infection than patients with leprosy alone, especially in multibacillary forms. This greater damage was largely attributable to HIV disease, but not related to neurotoxicity of HAART.[6]

In a clinical cohort study by Pires et al., they followed up patients for a period of at least two years for the occurrence of leprosy reactions and have found reactions to be more common among seronegative leprosy patients, with type 1 reversal reactions being predominant in both groups. This occurrence is associated with the borderline clinical forms, since these are immunologically unstable forms.[1] In the same study, the occurrence of type 2 ENL reaction was seen in the borderline lepromatous leprosy patients, and in patients who were severely immunosuppressed for a prolonged period. They have treated reactions in leprosy-HIV co-infected patients with the use of systemic corticosteroids using the same dose as patients without HIV, with a preference for prednisone at 1mg/kg for Type 1 reactions, acute neuritis and some cases of Type 2 ENL reactions with report of satisfactory improvement. Majority of patients in this study received 50mg/day of prednisone in accordance to weight, with gradual tapering every 15-20 days, decreasing to a dose of 20mg, then 5mg until drug is fully withdrawn. Results showed that most patients had a single episode of reactional states within 2 years of follow up, with reaction episodes of moderate intensity and more severe complications in patients presenting with type 2 ENL reactions. All co-infected patients were also found to have shorter periods of leprosy reaction, averaging at 2 months as compared to seronegative patients.[1] This was also consistent with similar cohort studies showing no adverse effects from treating leprosy reactions in HIV-leprosy co-infection with the usual dosage of prednisone with good response to treatment.[2] Although chronic corticosteroids may render HIV patients more susceptible to infections, the balanced benefit of treatment of reactions in preventing nerve damage and disability is paramount in the disease process, and hence, a closer monitoring for these patients should be observed.

CONCLUSION

Considering that both Hansen's disease and HIV directly affects T helper CD4+ lymphocytes in its pathogenesis, there seems to be no or little alteration in the course of patients with HIV-leprosy co-infection. Hence, treatment of HIV-leprosy co-infection does not differ from that of a seronegative leprosy patient. This can also be explained by the positive response of our patient with the standard MDT therapy for leprosy and antiretroviral for HIV, as both conditions are treated as separate entities. This case highlights the occurrence of ENL reaction in HIV-leprosy co-infection and the need for immunosuppressive drugs to control reaction and prevent nerve damage. The importance of close monitoring to screen for opportunistic infections is warranted while taking his medications.

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