# "Lepromatous leprosy as a presenting feature of HIV:" Diagnostic and management dilemmas

Sir,

We read with interest the article entitled "Lepromatous leprosy as a presenting feature of HIV" by Belgaumkar *et al.*[1] The authors have discussed the presentation of lepromatous leprosy (LL) as an opportunistic infection in an ART naïve patient with advanced disease and also the challenges in diagnosis and management.

The authors have diagnosed this case as LL with erythema nodosum leprosum (ENL) without considering carefully the clinical presentation and symptomatology. We feel that the clinical morphology of the lesions looks more like histoid leprosy (HL). The patient has papulonodular lesions with central depression, over the face with normal looking intervening skin, and the absence of madarosis also supports the diagnosis of HL.[2] Most of the lesions on legs are infiltrated papulonodules with few showing crusting suggestive again of HL. The presence of more than the usual number of lesions in this patient most likely indicates its possible transformation to LL, which is known to occur though not often.[3] Further, the absence of systemic features such as fever, arthralgias, and neuritis is unusual for ENL, which has acute presentation with associated systemic features. The authors also mention that *Mycobacterium leprae* and HIV act synergistically which could worsen the nerve damage, but surprisingly this patient had all the sensations intact. Enlarged nerves with no nerve function impairment (NFI) or tenderness are also more consistent with the diagnosis of HL rather than LL with ENL. HL may present with thickened nerves and the NFI may appear much later. Although the association of HL with HIV is quite rare, two cases (one presenting as immune reconstitution inflammatory syndrome) have been reported by Bumb *et al.*[4] and Sivasankari *et al.*[5] in HIV-infected patients taking highly active retroviral therapy for 9 and 11 months, respectively.

It is also stated that sensitivity and specificity of serological tests for HIV are affected in leprosy giving rise to false positive results, but the authors do not refer to performing an alternate confirmatory test. Very low CD4 count of 11 in a HIV patient represents advanced stage presenting with plethora of clinical manifestations and susceptible to various opportunistic infections, but surprisingly this patient had only history of weight loss (significant or not?) and anorexia and no laboratory evidence of any opportunistic infection.

The authors did not mention about lymphadenopathy in general physical examination, but detected abdominal lymphadenopathy on ultrasonography which was ascribed to be due to tuberculosis (TB). As lymphadenopathy can also be present in LL, severe ENL, and HIV *per se*, investigations done to confirm its tubercular etiology have not been mentioned.

Thalidomide administered for the control of ENL has dual role in HIV, i.e. helping the treatment naïve patient by reversing latency due to its immunomodulatory action[6] and also associated with T-cell activation which in turn leads to replication of HIV.[7] As leprosy and HIV are independently associated with peripheral neuropathy, further addition of thalidomide may increase the risk. Mention is also made about immunosuppression affecting the symptoms of ENL; however, it is well known that patients do continue to experience pain and fever in all stages of HIV disease. Observation about no further episode of ENL in 3 months of follow-up is not substantiated. If the initial episode was asymptomatic and diagnosed on histopathology, how will the next episode be recognized?

To summarize, the authors in our view faulted on many points: (i) a case which is clinically HL has been labeled as LL; (ii) no confirmatory test was done for the diagnosis of HIV infection and also abdominal TB, being fully aware that serology and ultrasonography alone are not confirmatory; (iii) in the absence of classical cutaneous lesions or systemic features, only histopathology suggestive of ENL cannot be the guiding principle for institution of antireaction therapy; (iv) in the scenario of HIV and leprosy coinfection, thalidomide is not a judicious choice for the control of ENL. Moreover, what parameters to monitor to decide about the end point of therapy because the diagnosis of ENL has been based on histopathology; (v) leprosy has been considered as an opportunistic infection – meaning that the degree of HIV related immunosuppression had reached a critical level making the patient susceptible for all types of infections. It is highly improbable that such a severely immunocompromised patient would survive the incubation period of leprosy, which typically lasts an average of 5 years, without receiving ART.

In view of all stated above, the described patient is a case of HL which got coinfected with HIV during the course of the disease and immunosuppression due to HIV, to some extent altered the clinical picture and course of leprosy.

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There are no conflicts of interest.

Vinod Hanumanthu, Tarun Narang, Sunil Dogra, Bhushan Kumar[1]
Department of Dermatology, Venereology and Leprology, Post Graduate Institute of Medical Education and Research, Chandigarh, [1]Department of Dermatology, Shalby Hospital Sahibzada Ajit Singh Nagar, Punjab, India

**Address for correspondence:**
Dr. Sunil Dogra,
Department of Dermatology, Venereology and Leprology, Post Graduate Institute of Medical Education and Research, Sector 12, Chandigarh, Punjab, India.
E-mail: sundogra@hotmail.com

## References

1. Belgaumkar VA, Chavan RB, Deshmukh NS, Ponathil AP. Lepromatous leprosy as a presenting feature of HIV. Indian J Sex Transm Dis AIDS

2021;42:162-5.
2. Kaur I, Dogra S, De D, Saikia UN. Histoid leprosy: A retrospective study of 40 cases from India. Br J Dermatol 2009;160:305-10.
3. Chaudhury DS, Chaudhury M, Armah K. Histoid variety of lepromatous leprosy. Lepr Rev 1971;42:203-7.
4. Bumb RA, Ghiya BC, Jakhar R, Prasad N. Histoid leprosy in an HIV positive patient taking cART. Lepr Rev 2010;81:221-3.
5. Sivasankari M, Sinha P, Sunita BS, Awasthi S. A case of histoid leprosy presenting as immune reconstitution inflammatory syndrome (IRIS) in a patient of human immunodeficiency virus (HIV) infection on highly active retroviral therapy (HAART). Indian Dermatol Online J 2021;12:441-3.
6. Vignesh R, Shankar EM. Thalidomide as a potential HIV latency reversal agent: Is it the right time to forget the ancestral sins? EBioMedicine 2017;24:20-1.
7. Vergara TR, Samer S, Santos-Oliveira JR, Giron LB, Arif MS, Silva-Freitas ML, *et al.* Thalidomide is associated with increased T cell activation and inflammation in antiretroviral-naive HIV-infected individuals in a randomised clinical trial of efficacy and safety. EBioMedicine 2017;23:59-67.



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