

HIV Seroprevalence in Leprosy Patients

TO THE EDITOR:

Several human immunodeficiency virus (HIV) serological studies on contact populations living in areas with different leprosy case-detection rates have been reported (¹). HIV infection has been shown to be strongly associated with the development of active tuberculosis and diseases caused by other mycobacteria (²), but its association with leprosy is much less clear. HIV-positive patients in the tropics do not live long enough in states of severe immunodepression to develop infections like leprosy. This may be due to the prolonged incubation period of leprosy, which may be more than a decade, and the clinical course, which may evolve over years. In contrast, it is common to note a reactivation of latent, virulent *Mycobacterium tuberculosis* infection with its high morbidity.

AIDS is known to be prevalent in certain leprosy-endemic areas. Infection with HIV leads to a profound drop in the helper (CD4) T-lymphocyte count and function (³). The effect of this is a lowering of resistance to a wide range of opportunistic and other infections. Leprosy takes a long time to develop and patients may die from other causes resulting from HIV infection before leprosy becomes clinically apparent. The effect of a further depression of host resistance due to HIV infection has been expected to lead to a shifting of the clinical

spectrum of leprosy, downgrading toward the lepromatous pole. Lepromatous leprosy may make the patient more susceptible to HIV, since leprosy also presents with a depression in the cell-mediated immunity (CMI) to *M. leprae*. Therefore, it is essential to detect HIV infection in areas where leprosy is endemic for a better understanding of the risk of dissemination of this mycobacterial disease in the community.

The present study was undertaken to find out the prevalence of HIV-1 infection in the leprosy population attending the Outpatient Department (OPD) of the Central JALMA Institute for Leprosy (CJIL), Agra, India. The leprosy patients attending the OPD of the CJIL were classified according to the criteria described by Ridley and Jopling. Every third patient who attended the OPD from April 1989 to March 1993 was selected for the study and screened for HIV serology. The mean age of the patients was 34.5 years and ranged from 16 to 53 years. Blood samples (5 ml each) from a total of 4025 leprosy patients have been tested for the presence of HIV antibodies.

Sera were separated and stored at -20°C until use. Out of the 4025 patients, 30, 141, 1888, 409, 600, 751 and 200 were classified as indeterminate (I), tuberculoid (TT), borderline tuberculoid (BT), mid-borderline (BB), borderline lepromatous (BL), lepromatous (LL) and neuritic (N) leprosy, respectively. Enzyme immunoassay (EIA)

kits of Wellcozyme HIV recombinant (Wellcome Diagnostics, Dartford, U.K.) provided by the Indian Council of Medical Research (ICMR) and the National AIDS Control Organization (NACO) were used. Each sample was subjected to ELISA by two different kits and a rapid test before confirmation by Western blotting which was carried out at the National Institute of Communicable Diseases, Delhi, India. Out of 4025 patients, 8 were found to be strongly positive by the Wellcozyme HIV recombinant and UBI HIV-1/2 EIA kits. These reactive samples were evaluated by the Western blot technique, and 5 of the 8 ELISA-positive samples showed strong antibody reaction to all the major gene products of HIV-1. Therefore, the HIV seroprevalence was only 1.24 per 1000 (5/4025) in this leprosy patient population. Three of the five HIV-positive patients had TT/BT leprosy (1 TT and 2 BT) and two were BL/LL (1 BL and 1 LL).

In the present study, we report the relative risk of developing HIV-1 infection among leprosy patients in an endemic area. In all, 4025 patients with histologically proven leprosy were tested for the presence of HIV-1 antibodies using the Wellcozyme EIA kit. All positive samples underwent confirmatory testing using the Western blot technique. The very low percentage of HIV infection among the leprosy patients suggests that HIV does not pose a serious problem in already *M. leprae*-specific immunosuppressed subjects. The clinical presentation and disability grade of HIV-1-infected patients were similar to that of patients without HIV-1 infection. Our results indicate that HIV-1 infection is more associated with TT/BT leprosy and does not contribute to any association with a more serious clinical presentation of leprosy. The present study showed HIV-1 positivity of only 1.24 per 1000 which is far less than any of the prevalence figures which has been reported with other at-risk groups. This very low prevalence of HIV-1 infection is not playing any role in already *M. leprae*-specific immunosuppressed subjects.

Although many studies reported more seropositivity in lepromatous patients than in tuberculoid patients⁽⁴⁾, in our study no clear association between these groups was noted. A similar nonassociation between

BL/LL leprosy and HIV-1 has also been noted by various other groups⁽⁶⁾. Further, several authors have accounted for the progression of the disease after HIV-1 infection⁽⁵⁾. However, in the present study, on follow up none of the I, TT/BT or N patients with HIV-1 infection progressed into a more severe form (BL/LL) of the disease.

Our results indicated that HIV-1 infection does not contribute in any way for precipitation of serious forms of leprosy. We also conclude from our study of this OPD population that leprosy is not a risk factor for developing HIV-1 infection.

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