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Some Possible Interactions of *M. leprae* and HIV in the Peripheral Nerves

TO THE EDITOR:

Infection with the human immunodeficiency virus (HIV) has affected millions of people in the African continent ([5]) where leprosy is also endemic. The association of the acquired immunodeficiency syndrome (AIDS) with leprosy has seldom been reported ([3]), probably because of the prolonged incubation period of leprosy. A patient with AIDS who gets infected with *Mycobacterium leprae* succumbs to other (opportunistic or nonopportunistic) infections before he develops clinical leprosy. The possible interactions between the two infections have also been discussed ([6]). The possible results of the defective cell-mediated immunity due to HIV infection include conversion of subclinical leprosy cases into tuberculoid cases, downgrading of paucibacillary cases to multibacillary cases, and an increased incidence of type 2 lepra reactions ([6]).

However, interactions of two infectious agents at the level of peripheral nerves is a distinct possibility with which those involved in the care of leprosy patients must be prepared to deal. This is so because the HIV is not only lymphotrophic but also neurotrophic ([2]). In addition to the subacute encephalitis, peripheral neuropathy has also been recognized as a neurologic manifestation of HIV infection. The peripheral nerve syndromes found in patients with HIV infection include chronic inflammatory polyneuropathy and distal symmetric sensorimotor neuropathy ([4]). The etiologic role of the HIV in these nerve affections has been demonstrated by culture of the virus from the sural nerve of a patient with peripheral neuropathy ([2]) and, more recently, by the demonstration of HIV replication in infiltrating mononuclear cells in the peripheral nerves by *in situ* hybridization ([1]). Nerve biopsies in these patients revealed vasculitis and inflammatory infiltrates in the epineurium and endoneurium. What is more interesting is that this neurologic involvement could occur before or in the absence of immunodeficiency ([2]).

The implications of these findings are grave for patients with leprosy who may get infected with HIV. In addition to the immunodeficiency, they could suffer from additional nerve damage as a result of either a downgrading reaction or necrotizing vasculitis of the nerves due to the HIV infection. The deterioration of nerve function may occur while the patient is on antileprosy treatment, and it will have to be differentiated from a type 1 reaction. The management of peripheral neuropathy caused by HIV infection is far from clear. The HIV infection can also lead to Bell's palsy ([4]) which will have to be differentiated from leprous facial palsy. The investigations suggested for the evaluation of a patient with HIV-induced peripheral neuropathy include the examination of the cerebrospinal fluid, electromyography, nerve conduction velocity, and nerve biopsy ([4]). None of these

may be available in the field and, when available, may not differentiate the nerve dysfunction due to a lepra reaction from that caused by the HIV in a patient infected with both *M. leprae* and HIV. The only direct confirmation of the fact that the HIV is responsible for the neuropathy will be by the demonstration or culture of the virus.

Another problem in these patients will be the treatment of lepra reactions. Systemic corticosteroids which are often needed for the control of reactions may have an adverse effect on the course of HIV infection by causing immunodeficiency.

The possible interaction of *M. leprae* and HIV in the peripheral nerves is a distinct possibility which will require careful study and follow up of the patients who may get infected with both of these agents.

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Quantitative Relationship Between Anti-PGL-I-specific Antibody Levels and the Lepromin Reaction

To the Editor:

The discovery the that phenolic glycolipid (PGL-I) from *Mycobacterium leprae* ([3]) was a species-specific antigen ([4, 9]) was one of the most important discoveries made in the last decade in the field of mycobacterial diseases, and it stimulated the already successful search for species-specific glycolipid antigens in other important human pathogens, notably *M. tuberculosis* ([1, 2, 5]). Although many studies were devoted to the PGL-I antigen (reviewed in [2]) and reasonable correlation was established between anti-PGL-I antibody titers and bacterial loads (bacterial index, BI [7]), to our knowledge an investigation to verify if there were quantitative relationships between antibody titers and the intensity of lepromin reactions has escaped the attention of previous workers.

For the purpose of this study, sera were collected from 53 first-time leprosy patients. After the blood was collected, 0.1 ml of lepromin-H was injected intradermally and the lepromin reaction was measured as millimeters of induration 21 days later. The patients were subsequently classified according to the Ridley-Jopling scheme ([6]). The sera were tested using ELISA, and IgG and IgM levels were scored as the absorbance obtained using a 1/250 dilution; the ELISA method was as described before ([1]). The lepromin test, ELISA, and the classification of the patients according to the Ridley-Jopling scheme were done blind by independent workers.

The quantitative relationships between the anti-PGL-I antibody levels and the lepromin reaction are shown in The Figure. The data neatly divided the patient population into three major groups: a) patients with a negative lepromin test (0–2 mm induration but antibody levels above 500); b) patients with a positive lepromin test (more