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Leprosy and AIDS in the Amazon Basin

To The Editor:

The state of Amazonas in Brazil is a hyperendemic area of leprosy. Although there has been a control program in action for about the last 20 years, the prevalence rate and the detection rate were 39.4 per 10,000 and 67 per 100,000 inhabitants in 1994, respectively. The first case of AIDS in the state was diagnosed in 1986. Although there is a low incidence rate of AIDS in the state of Amazonas (9.5/100,000), 63% of the cases were diagnosed within the last 3 years (Ministerio da Saude do Brasil. Boletim Epidemiologico D.S.T./AIDS, Brasilia, 1995. Ano VII). This shows an increased trend of AIDS in the region. Despite an existing possibility of an interaction between *Mycobacterium leprae* and the HIV infection([1–3, 6, 7]), few clinical reports have been written and the effects of this co-infection have not yet been defined([4, 5]).

In this letter, the clinical aspects and progression of four patients who were identified as having leprosy and HIV infection [HIV 1-HIV 2 antibodies by enzyme immunoassay (Genelavia-Sanofi, France, and immunofluorescence)], one of them with AIDS which was identified by the presence of Kaposi's sarcoma, are described.

Case 1. JCLN, a 25-year-old married male, presented with a hypochromic lesion on the left arm, was skin-smear negative, intradermal reaction was Mitsuda positive and a histopathological examination of the lesion showed tuberculoid infiltrate. This led to the diagnosis of the tuberculoid form of leprosy in October of 1992. A drug combination treatment, including ofloxacin, was given to the patient who had agreed to take part in a double-blind trial for a period of 6 months. The patient took the treatment regularly and had no side effects or leprosy reactions. A routine serologic exam showed a positive result for HIV on 27 October 1993. Only after that did the patient say that he had known he was infected with HIV since 1991 but that he had ignored the fact and had not taken any preventative measures. The leprosy lesion has disappeared, and a general clinical exam and laboratory exams have not shown any abnormalities.

Case 2. VNA, a 27-year-old, male homosexual hairdresser. A diagnosis of borderline lepromatous leprosy was made on 15 September 1994. The patient presented with skin infiltration and disseminated plaques. The first skin-smear exam showed a bacterial index (BI) of 3.2 with 1% of intact

bacilli. Multidrug therapy (MDT-WHO) was started, and a month later the patient presented with a type 1 reaction which responded well to prednisone. In February 1995, a nodule-like lesion, reddish-purple and shiny in appearance, developed on the sole of the patient's right foot. Histopathological examination of the lesion was compatible with Kaposi's sarcoma. A serologic exam showed a positive result for HIV in March 1995. At present, the leprosy lesions are decreasing in size and number, and the patient is still taking MDT-WHO and AZT.

Case 3. RLS, a 27-year-old, married male was diagnosed as having lepromatous leprosy on 3 October 1978, having presented with anesthesia in the lower limbs, ulnar clawing in the left hand, and ulnar and common peroneal nerve enlargement. He had a positive skin smear, and histopathological examination showed histocytes with vacuolated cytoplasm with acid-fast bacilli. Treatment with sulfone was introduced but changed to MDT-WHO in June of 1986. After 2 years, the patient was released from treatment. Because his wife was detected as having HIV, he was advised to have the test which proved seropositive in March of 1995. At present, he has lost weight and has an ulcer on the sole of his foot.

Case 4. RM, a 30-year-old, male homosexual had anesthetic tuberculoid plaques on the thoracic region and the neck and left arm, a negative skin smear, a positive Mitsuda reaction, and histopathology of the lesion showed tuberculoid infiltrate. The patient was diagnosed as having tuberculoid leprosy, and MDT-WHO was started. HIV was detected in a routine serologic exam. At the moment, the patient has only residual patches and his general clinical and laboratory examinations are normal.

The high prevalence of leprosy and the increased trend of HIV infection in the Amazon region suggest that the co-existence of these two infections is not a rare event. However, there is a lack of epidemiological studies evaluating the prevalence of HIV in leprosy patients in the region. The clinical evolution or the response to treatment of the leprosy patients described above have not changed from those without HIV infection. However, two of them have not yet completed treatment. Only one patient had type 1 reaction and he responded satisfactorily to prednisone. Epidemiological studies are necessary to better understand the prevalence of the co-infection (leprosy and AIDS), and the progression of existing and new cases will need to be closely monitored. A probable hypothesis is that in the Amazon region these two infections are occurring in different social classes, which would explain the low incidence of sufferers of co-existing leprosy and HIV.

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TT Leprosy: Does It Indicate Percutaneous Infection?

TO THE EDITOR:

The subepidermal zone (SEZ) is a narrow strip of dermis which lies just below the basal layer of the epidermis. Histological changes in the SEZ find mention even in the early literature, and the SEZ is regarded as a differentiating point between tuberculoid and lepromatous leprosy. A free SEZ is also know as the band of Unna, who first stated that this zone is free of bacilli when compared to the lepromatous granuloma ([12]). Complete obliteration of the SEZ by epithelioid cell granuloma is exclusively a feature of tuberculoid (TT) leprosy as per the Ridley-Jopling classification ([7]). Hence, this piece of histologic change isolates TT from almost all other types of leprosy. While we have some convincing explanation about the free grenz zone of lepromatous leprosy ([4]), the significance of its obliteration in TT leprosy is not adequately explained. Evolution of a granuloma in the most superficial part of the dermis makes more sense as a starting point of the granuloma and, more importantly, might indicate the cutaneous route of infection.

Although inhalation of bacilli-rich droplets is at present regarded as the most common mode of entry by leprosy bacilli ([3]), infection through the skin, as advocated in earlier studies ([1, 2, 11]), has not been discarded. Can the evolution of TT leprosy be attributed to the percutaneous entry of the bacilli? Will the route by which the antigen gains access to the biological system decide the nature of the immune response? An important study by Ridley ([5]) states that granuloma in the SEZ and the epidermis is found in cases with high resistance which detects and mounts an immune attack at the site of entry of *Mycobacterium leprae*. Now there are pieces of evidence which attribute an antigen-processing function to the epidermal Langerhans' cells in many delayed-type hypersensitivity (DTH)-mediated diseases, including leprosy ([10]). These mononuclear phagocytes are known to process and transport *M. leprae* antigens through the subepidermal lymphatics to regional lymph nodes and lymphocytes on the route for an effective immune response ([8, 9]).

On the basis of these observations, one is tempted to interpret the pathogenesis of TT and other types of leprosy mainly around the route of infection. If the organism is detected by the antigen-processing cells, there is an effective immune response, resulting in a localized TT leprosy. If this check system is bypassed due to entry through routes other than the skin, an entirely different course of the disease may ensue. An epithelioid cell granuloma, including that of TT, is a manifestation of DTH. We cannot think of its evolution without a phase of acute inflammation. Type 1 reaction involving the SEZ and the epidermis appears to represent this acute state. In other words, TT cases with involvement of both the SEZ and the epidermis may have started out with a type 1 reaction although they are now immunologically stable, i.e., TT patients may have started out as borderline. This also may explain the occurrence of type 1 reaction, which is a strong borderline feature, in some TT cases which as a whole are considered immunologically stable. The apparent low lymphocyte density and the lack of focalization (Fig. 1) were due to massive inflammatory edema. These reacting granulomas probably indicate a recent sensitization and a persisting antigenic stimulation. As the acute phase subsides, the granuloma attains the compact histology usually described for TT leprosy. It is logical to suppose that in TT leprosy DTH follows an