

## Lepromatous Leprosy and HIV Infection

### TO THE EDITOR:

The patient is a 19-year-old heterosexual male with a history of intravenous drug abuse since the age of 15. He is a resident of Buenos Aires, Argentina.

In June 1991 the patient developed erythema nodosum leprosum (ENL). The ENL improved with corticosteroid therapy which the patient self-administered without consulting a physician. In January 1992 he was admitted to the hospital with a reactional episode characterized by ENL, some of which resembled erythema multiforme lesions, and severe bilateral ulnar neuritis with nerve enlargement, tenderness, and loss of function.

Electromyography at that point showed a severe left ulnar sensory neuropathy and a moderate right ulnar motor loss. Skin smears from cutaneous lesions and nasal scrapings were positive for acid-fast bacilli (AFB) with a bacterial index (BI) of 2+. Skin biopsies from lesions showed ENL and were positive

for AFB. The patient tested positive for HIV infection by ELISA, confirmed by Western blot. The patient's serum was negative for antigenemia. The peripheral blood CD4+ count was 1563/mm<sup>3</sup>.

The patient was treated with daily rifampin 600 mg, clofazimine 50 mg, dapsone 100 mg, and prednisolone 80 mg. After slight improvement the prednisolone was gradually tapered. When the dose reached 40 mg daily, the patient again developed neuritis with impaired neural functioning. At that point, the clofazimine was increased to 300 mg daily while the prednisolone was again gradually tapered. After 3 months the patient's prednisolone could be discontinued.

Although the antiinflammatory effect of large doses of clofazimine is well known, the drug may be even more important for a patient with reactions with leprosy and HIV infections. In these cases the possible



FIG. 1. Macules and nodules on the thigh.

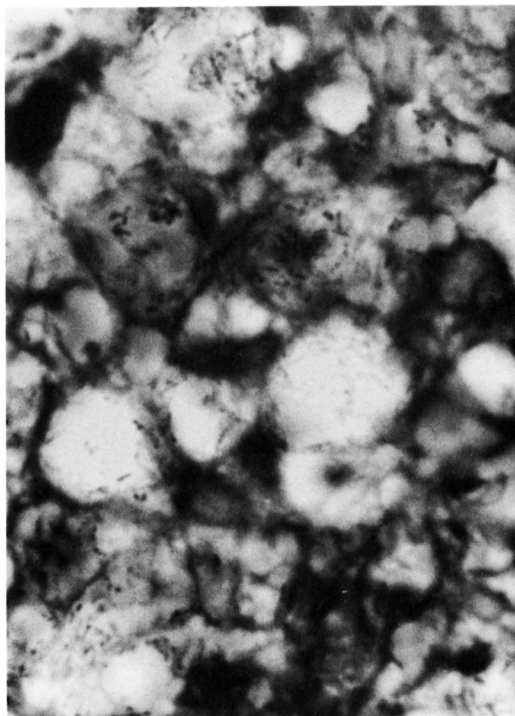


FIG. 2. Ziehl-Neelsen stain of skin biopsy showing numerous acid-fast bacilli in vacuolated cells in the dermis.

adverse effects of corticosteroid therapy on the course of the HIV infection by causing immunodeficiency may be particularly important.

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## Necrotizing Erythema Nodosum Leprosum Triggered by Cotrimoxazole?

### TO THE EDITOR:

Erythema nodosum can be the first recognizable manifestation of lepromatous leprosy, although it is most common in patients receiving specific chemotherapy (4). Patients with erythema nodosum leprosum (ENL) may present erythematous nodules over the whole body; whereas in patients with erythema nodosum from other causes the lesions are generally restricted to the lower limbs, particularly the shins. An unusual and more severe presentation is the necrotizing erythema nodosum leprosum (NENL), most commonly seen in Southeast Asia (5). Verma and Pandhi (6) recently have reported a case where NENL was the first recognized manifestation of lepromatous leprosy. We report here another case for whom NENL was the presentation of lepromatous leprosy and was possibly associated to the use of cotrimoxazole (sulfamethoxazole + trimethoprim).

A 23-year-old man sought a doctor complaining of right axillary and left inguinal lymphadenopathy and fever of acute onset. The patient was given a prescription of oral cotrimoxazole, although a conclusive diagnosis was not made. On the second day of cotrimoxazole use indurated red-bluish plaques and nodules, some of them with blisters, appeared on his upper and lower limbs. Admitted to a teaching hospital, on

physical examination he had jaundice, palpable cervical, axillary and inguinal lymph nodes, arthritis of his right knee and ulcerated necrotic plaques mostly on his arms and thighs. A presumptive diagnosis of sulfonamide-induced cutaneous necrotizing vasculitis, arthritis and hepatitis was made, and oral prednisone 40 mg per day was instituted, with much improvement in a few days.

Laboratory tests of interest were: hematocrit 27.6%, leukocytes 39,300/mm<sup>3</sup> (56% neutrophils, 33% band forms, 8% lymphocytes, 3% monocytes), platelets 425,000/mm<sup>3</sup>, ESR 124 mm/hr, direct bilirubin 4.9 mg/dl, indirect bilirubin 2.6 mg/dl, aspartate aminotransferase (SGOT) 30 U/l, alanine aminotransferase (SGPT) 28 U/l, alkaline phosphatase 223 U/l, gamma-glutamyltranspeptidase 80 U/l. A definite diagnosis was made when the result of a skin biopsy performed during the acute episode revealed an infiltrate of lymphocytes, plasma cells and histiocytes around vessels, nerves and sweat glands; countless acid-fast bacilli (AFB) were seen within foamy macrophages. Solidly staining, fragmented and granular forms of AFB were also seen on slit-skin smear examination from the earlobes, elbows and a cutaneous lesion; the bacterial index was 5+. A percutaneous liver biopsy performed during recovery re-