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^3 (56% neutrophils, 33% band forms, 8% lymphocytes, 3% monocytes), platelets 425,000/mm^3, 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, gamaglutamyltranspeptidase 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-

vealed only hydropic degeneration of the hepatocytes; AFB were not seen on a Ziehl-Neelsen stained preparation.

After a follow up of 3 months, the patient has had a favorable outcome under multidrug therapy and is still tapering the dose of prednisone.

In this patient the initial diagnosis was an untoward reaction to sulfonamides manifested as liver damage, cutaneous vasculitis and a serum sickness syndrome, which has been reported to occur 2 hr to 3 days after the start of therapy ([1]). The initial impression was corroborated by the favorable response to corticosteroids. Although leprosy was thought of initially, the diagnosis was somewhat surprising to the attending physicians.

ENL has several different clinical manifestations, apparently associated to ethnic factors, that are nevertheless histologically similar ([4]). The release of mycobacterial antigens from the macrophages is required for the formation and deposition of immune complexes that form the immunopathological basis of the erythema nodosum, but the intrinsic mechanism is not completely understood ([2]).

Erythema nodosum was the first recognizable manifestation of leprosy in this patient, and was temporally related to the use of cotrimoxazole. Since sulfonamides have been shown to be (poorly) active against *Mycobacterium leprae,* and actually were used in the past for the treatment of leprosy ([3]), we question whether NENL in this case was triggered by the destruction of *M. leprae* induced by cotrimoxazole.

Drugs with a bactericidal effect on *M. leprae* that are not used for the treatment of leprosy, such as cotrimoxazole, or that are mostly used for other purposes, such as the fluorquinolones, can theoretically trigger ENL in patients with undiagnosed lepromatous leprosy. Therefore, leprosy must be remembered as a differential diagnosis of erythema nodosum with or without vasculitis in patients taking these drugs.

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Pefloxacin in Histoid Leprosy

To the Editor:

A 26-year-old female was diagnosed as having leprosy 6 months ago and was put on antileprosy treatment. She developed shiny nodular lesions over the body while on treatment. When she was first seen by us she had well demarcated, shiny, papular and nodular, nontender lesions over the face, extensors of the forearms, back and abdomen (Fig. 1). The surrounding skin was apparently unaffected. All of the nerves of predilection were thick but not tender. She was diagnosed clinically as a case of histoid lep-