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-

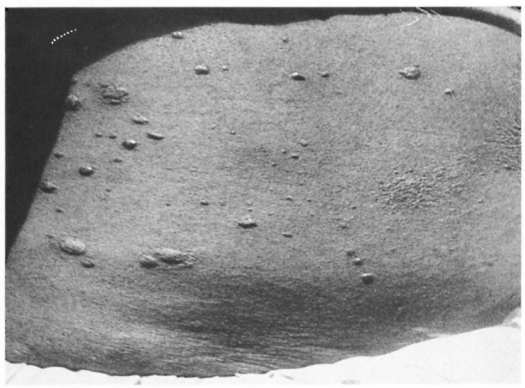

FIG. 1. Histoid lesions over the abdomen before treatment.

FIG. 2. Histoid lesions over the abdomen after 8 weeks of pefloxacin treatment.

rosy and was investigated accordingly. Her bacterial index (BI) was 6+, morphological index (MI) was 10%, and skin biopsy revealed features of histoid leprosy. Routine investigations were within normal limits.

She was put on pefloxacin 400 mg BID for a period of 8 weeks in addition to WHO-MDT. She tolerated the drugs well, but there was no clinical improvement (Fig. 2). Her BI remained 6+ as expected but even the MI did not fall to 0; it was 2%. Another skin biopsy showed the same features. (Facilities for other investigation such as mouse foot pad inoculation are not available to us.)

A study by Grosset, *et al.* ([1]) showed marked clinical improvement in patients with nodular lepromatous leprosy on treatment with pefloxacin for 8 weeks. For practical purposes, a histoid lesion is an unusually active leproma and the histoid features are lost as soon as the lesion starts to regress ([2]). Compared to this, our patient with histoid features did not respond at all clinically, bacteriologically or histologically to treatment with pefloxacin or WHO-MDT.

Does the histoid variety of leprosy differ from other nodular lepromatous leprosy with respect to the response to treatment with newer antileprosy drugs like pefloxacin? We wish to share the experiences of other workers with pefloxacin treatment in histoid leprosy and the possible pathogenesis of the response.

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