

# Recurrent Erythema Nodosum Leprosum Precipitated by Antileprosy Drugs

TO THE EDITOR:

Erythema nodosum leprosum (ENL), an inflammatory reaction occurring in patients with lepromatous or borderline lepromatous leprosy (3,5), presents as a group of tender dermal and/or subcutaneous nodules that arise in apparently normal skin. Although ENL occasionally develops in untreated patients (6), more than 50% of patients with lepromatous leprosy in South East Asia develop ENL within the first year of administration of sulfones (7).

Recently, a lepromatous leprosy patient [bacterial index (BI) of 5+] was referred to us with a severe ENL reaction with necrotic lesions after 4 months of multidrug therapy (MDT). A thorough clinical examination and relevant laboratory investigations were done to rule out other causes of ENL. After stopping MDT, the ENL lesions subsided. The patient, then given dapsone, rifampin and clofazimine individually, developed ENL with dapsone and rifampin while he tolerated clofazimine. In view of the recent advances in antileprosy chemotherapy, he was given individually ofloxacin, minocycline and clarithromycin, and developed ENL lesions within 24 hr of taking ofloxacin and clarithromycin. He has been taking the combination of minocycline and clofazimine for the past year but, unfortunately, neither his BI nor histopathology have shown any improvement.

Lepromatous leprosy is associated with polyclonal B-cell activation (7). Several authors have shown that circulating IgM antibodies against phenolic glycolipid-I (PGL-

I) are decreased in serum during ENL (1,4). Other authors using suction-induced blister formation on ENL lesions have shown increased levels of IgM antibodies against PGL-I in blister fluid (2). It appears that with the onset of bactericidal antileprosy therapy there is disintegration of bacilli and release of antigenic material (8). Supporting evidence includes the finding of disintegrated bacilli and foamy macrophages in ENL lesions, the deposition of immune complexes composed of IgM, IgG and complement components of the classical pathway and infiltration of polymorphonuclear leukocytes (neutrophils) (10). All of these changes show that bactericidal antileprosy drugs can precipitate ENL in susceptible individuals by the release of antigenic material, and PGL-I is likely the antigenic component of immune complexes in ENL.

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