2. DUNCAN, M. E. and PEARSON, J. M. H. The association of pregnancy and leprosy. III. Erythema nodosum leprosum in pregnancy and lactation. Lepr. Rev. **55** (1984) 129–142.
3. KARAT, A. B. Long-term follow-up of clofazimine (Lamprene) in the management of reactive phases of leprosy. Lepr. Rev. **46** Suppl. (1975) 105–109.
4. MAURUS, J. N. Hansen's disease in pregnancy. Obstet. Gynecol. **52** (1978) 22–25.
5. PLOCK, H. and LEIKER, D. L. A long-term trial with clofazimine in reactive lepromatous leprosy. Lepr. Rev. **47** (1976) 25–34.
6. SCHULZ, E. J. Forty-four months' experience in the treatment of leprosy with clofazimine (Lamprene; Geigy). Lepr. Rev. **42** (1972) 178–187.
7. STEEN, J. S. M. and STAINTON-ELLIS, D. M. Rifampicin in pregnancy. Lancet **2** (1977) 604–605.
8. TUCHMANN-DUPLESSIS, H. and MERCIER-PAROL, L. Influence d'un antibiotique, la rifampcine, sur le developpement prenatal des ronguers. C.R. Acad. Sci.(d) **269** (1969) 2147–2149. As cited in: Shepard, T. H. *Catalog of Teratogenic Agents.* 6th edn. Baltimore: Johns Hopkins University Press, 1989, pp. 558–559.

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 ([9]).

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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REFERENCES

1. ANDREOLI, A., BRETT, S. J., DRAPER, P., PAYNE, S. N. and ROOK, G. A. W. Changes in circulating antibody levels to the major phenolic glycolipid during erythema nodosum leprosum in leprosy patients. Int. J. Lepr. **53** (1985) 211–216.
2. BHOOPAT, L., SCOLLARD, D. M., THREETRANONT, C., CHIEWCHANVIT, S., NELSON, D. L. AND UTAIPAT, U. Studies of human leprosy lesions in situ using suction-induced blisters: cell changes with IgM antibodies to PGL-I and interleukin-2 receptor in clinical subgroups of erythema nodosum leprosum. Asian Pac. J. Allergy Immunol. **9** (1991) 107–119.
3. BROWNE, S. G. Erythema nodosum in leprosy. J. Chron. Dis. **16** (1963) 23–30.
4. LEVIS, W. E., MEEKER, H. C., SCHULLER-LEVIS, G., SERSEN, E. and SCHWERER, B. IgM and IgG antibodies to phenolic glycolipid-I from *Mycobacterium leprae* in leprosy: insight into patient monitoring, erythema nodosum leprosum, and bacillary persistence. J. Invest. Dermatol. **86** (1986) 529–534.
5. PETTIT, J. H. S. and WATERS, M. F. R. The etiology of erythema nodosum leprosum. Int. J. Lepr. **35** (1967) 1–10.
6. REA, T. H. and LEVAN, N. E. Erythema nodosum leprosum in a general hospital. Arch. Dermatol. **3** (1975) 1575–1580.
7. REICHLIN, M., PRANIS, R. A., GELBER, R. H., REES, R. J. W., TAVERNE, J. and TURK, J. L. Correlation of euglobulin immunoglobulin G levels with erythema nodosum leprosum in lepromatous leprosy. Clin. Immunol. Immunopathol. **8** (1977) 335–344.
8. RIDLEY, M. J. and RIDLEY, D. S. The immunopathology of erythema nodosum leprosum: the role of extravascular complexes. Lepr. Rev. **54** (1983) 95–107.
9. WATERS, M. F. R., REES, R. J. W. and SUTHERLAND, I. Chemotherapeutic trials in leprosy. 5. A study of methods used in clinical trials in lepromatous leprosy. Int. J. Lepr. **35** (1967) 311–335.
10. WATERS, M. F. R., TURK, J. L. and WEMAMBU, S. N. C. Mechanisms of reaction in leprosy. Int. J. Lepr. **39** (1971) 417–428.