

- C. G. Autoantibodies in leprosy. *Int. J. Lepr.* **47** (1979) 171–175.
11. MOTTA, C. P. The epidemiological situation in the Americas. *Lepr. Rev.* **52** Suppl. 1 (1981) 61–68.
 12. OLIVEIRA, J. S. M. A natural human model of intrinsic heart nervous system denervation: Chagas' cardiopathy. *Am. Heart. J.* **110** (1985) 1092–1098.
 13. PETRY, K. and EISEN, H. Chagas' disease: a model for the study of autoimmune diseases. *Parasitol. Today* **5** (1989) 111–116.
 14. ROSENBAUM, M. Chagasic cardiomyopathy. *Prog. Cardiovasc. Dis.* **7** (1964) 199–225.
 15. ROSSI, M. A., GONÇALVES, S. and RIBEIRO-DOS-SANTOS, R. Experimental *Trypanosoma cruzi* cardiomyopathy in BALB/c mice: the potential role of intravascular platelet aggregation in its genesis. *Am. J. Pathol.* **114** (1984) 209–216.
 16. SOARES, J. D. and JUNQUEIRA, L. F., JR. Incidência de arritmias associadas a manobra de Valsalva nas diversas formas clinicas da doenca de Chagas. *Rev. Soc. Bras. Med. Trop.* **20** Suppl. 11 (1987) 58.
 17. TANOMITZ, H. B., BURNS, E. R., KUMAR SINHA, A., KAHN, N. N., MORRIS, S. A., FACTOR, S. M., HATCHER, V. B., BILEZIKIAN, J. P., BAUM, S. G. and WITTNER, M. Enhanced platelet adherence and aggregation in Chagas' disease: a potential pathogenic mechanism for cardiomyopathy. *Am. J. Trop. Med. Hyg.* **43** (1990) 274–281.

Histoid Leprosy in Early Macular Lepromatous Leprosy; Incidental Finding or Sign of Augmented Local Immunity?

TO THE EDITOR:

Wade first coined the term "histoid leprosy" by describing a variant of leprosy clinically characterized by cutaneous and subcutaneous nodules over an apparently normal skin, and histologically by bacillary-rich lepromas primarily exhibiting single tissue elements such as fibromas and the like (⁴). The origin of histoid leprosy is yet to be elucidated, and ever since the first description by Wade, this type of leprosy has aroused interest.

We report here an untreated case of leprosy with histoid nodules occurring along with macular lesions of active lepromatous leprosy and a macular lesion with features of regressive lepromatous leprosy.

A 29-year-old black man originating from Ghana, who had been living in Italy for 2 years, presented with a history of the appearance 3 months earlier of a small nodule on his right arm which subsequently was followed by several other nodules on his limbs and face. The nodules were of various sizes (ranging up to 0.5 cm in diameter), were skin-colored, and had a shiny surface, well-defined edges, and a hard consistency.

In addition to the nodular lesions, careful examination of the skin disclosed coppery, ill-defined macules of which the patient was unaware. The macules were present on the

flanks and spared the extremities. A solitary, round, coppery macule of about 2–5 cm diameter with hyperpigmented and ill-defined margins was present on his back. His hair, eyelashes and eyebrows were preserved. General examination disclosed a subjective sensory impairment of pain over the ulnar side of the right forearm.

Skin biopsies were done on one of the nodules and on the macula on his back. Hematoxylin-eosin staining of the nodular lesion showed a pseudoencapsulated mass made up of fusiform histiocytes arranged in crisscross fashion along with polygonal and irregularly shaped histiocytes. The nodule was deep seated with borders pushing down the subcutis and pushing aside hair follicles. Fite-Faraco staining revealed numerous, well-preserved, solid-stained acid-fast bacilli (AFB) scattered either singly or in small bundles throughout the nodule. Globi were occasionally present.

Histopathology of the macular lesion on the back showed an accumulation of vacuolar histiocytes in the superficial dermis, with rare lymphocytes and scattered eosinophils. Fite-Faraco staining showed many intracellular AFB, mostly granular. A nasal swab was positive for AFB.

The diagnosis of subpolar lepromatous leprosy featuring both macular and histoid

lesions was established. He was put on a treatment regimen of rifampin (10 mg/kg/day) and ofloxacin (600 mg/day), and remarkable improvement was seen within a few months.

The peculiarity of our case is the unusual concomitance, in an untreated patient, of various elementary cutaneous lesions commonly seen in different stages of lepromatous leprosy, namely, nodular lesions featuring the classical clinical presentation of histoid leprosy. Coppery macules on the flanks, with their symmetrical distribution and ill-defined margins, represented lesions of early active lepromatous leprosy. The macule on the back, with its hyperpigmented ill-defined margins, vacuolated histiocytes and fragmented AFB, was clearly a regressive lesion.

Others already have reported several findings suggestive of augmented immune response in histoid leprosy⁽²⁾. Cell-mediated immunity has been reported to be relatively high in histoid leprosy, giving an explanation for the attempted focalization of the lesions⁽³⁾. The peculiar phenomenon of "tuberculoid contamination" in histoid leprosy⁽¹⁾, although not satisfactorily explained, would further suggest a hyperactive cellular response. Moreover, histoid nodules display a peculiar tissue response because they enlarge by internal expansion and not by outward infiltration as in ordinary allergic lepromatous leprosy. The relevance of the tissue response in modulating the clinical expression of leprosy is not a novelty but a well-established concept in the pathogenesis of leprosy. Immune areas of borderline leprosy are a typical example of a tissue response to leprosy infection which are locally different.

Similarly, a sort of varying competence in the local response can be postulated in our patient; namely, some areas remained anergic and expressed themselves through active lepromatous macules whereas others displayed a local, augmented immune response that favored regression of macrophagic infiltration in one site (macule on the back) and limitation of the infection to a few foci in others (histoid nodules).

In conclusion, the occurrence of histoid nodules in our patient reasonably can be regarded as a sign of "local augmented immunity" locally able to prevent infiltrative expansion of the infection but unable to hamper bacillary multiplication within the nodule.

—Paolo Fiallo, M.D.
Enrico Nunzi, M.D.
Paolo Betto, M.D.
E. Bonoldi, M.D.
F. Torregrossa, M.D.

Ospedale Civile di Vicenza, Italy
C.I.R.-LEP., University of Genoa
Viale Benedetto XV, 7
I-16132 Genoa, Italy

REFERENCES

1. MANSFIELD, R. D. Histoid leprosy. *Arch. Patol.* **87** (1969) 580–585.
2. SEHGAL, V. N., SRIVASTA, G. and SAHA, K. Immunological status of histoid leprosy. *Lepr. Rev.* **56** (1985) 27–33.
3. SEHGAL, V. N., SRIVASTAVA, G. and SHARMA, V. K. Immune responses in disseminated necrotizing histoid leprosy. *Int. J. Dermatol.* **27** (1988) 413–414.
4. WADE, H. W. The histoid variety of lepromatous leprosy. *Int. J. Lepr.* **31** (1963) 129–142.

Infected Trophic Ulcers and Tenderness of Posterior Tibial Nerve in Cured Leprosy Patients

TO THE EDITOR:

Tenderness of the nerves is traditionally taught as a sign of activity of the infection in leprosy⁽¹⁾. A peculiar condition arises in patients who have completed multidrug

therapy and are cured except for residual nerve damage affecting the posterior tibial nerve. These patients continue to suffer from trophic ulcers on their anesthetic feet if they