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.

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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 histioid 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

do not take proper care of them. These patients, when they present with recurring trophic ulcers, demand antileprosy therapy thinking that their disease is still active. When one palpates the posterior tibial nerve in a patient with an infected trophic ulcer on the sole of the foot, tenderness is often elicited. However, this tenderness subsides when the patient is treated with an appropriate antimicrobial for the secondary infection in the ulcer. This is most probably due to the fact that the tenderness elicited while palpating the nerve is due to the inflamed lymphatics closely related to the nerve behind the medial malleolus. The inguinal lymph nodes are also enlarged and tender in these patients and subside after antimicrobial therapy. It is well known that the lymphatics draining the deeper tissues of the foot accompany the posterior tibial vessels which are closely related to the nerve ([2]).

We suggest that for a patient who has completed multidrug therapy for leprosy and then presents with an infected trophic ulcer on the sole, the tenderness of the posterior tibial nerve should be interpreted only after the secondary infection in the ulcer is controlled. Otherwise this tenderness may be misinterpreted as a sign of activity of leprosy or a relapse or a delayed reversal reaction. This could lead to an unnecessary institution of antileprosy therapy.

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REFERENCES

1. COCHRANE, R. G. Complicating conditions due to leprosy. In: *Leprosy in Theory and Practice.* 2nd edn. Cockrane, R. G. and Davey, T. F., eds. Bristol: John Wright and Sons Ltd, 1964, pp. 331–343.
2. WILLIAMS, P. L. and WARWICK, R. Angiology. In: *Gray's Anatomy.* 36th edn. Williams, P. L. and Warwick, R., eds. Edinburgh: Churchill Livingstone, 1980, pp. 622–800.

Myokymia in Leprosy Patients

TO THE EDITOR:

Myokymia is the fine, fibrillary, involuntary movement of the eyelids and periorbital muscles which gradually involves other muscles of the face ([3, 5]). Disorders such as multiple sclerosis, brain stem vascular injury, or pontine neoplasms are important causes of myokymia ([1, 2, 6]). The majority of myokymia cases are associated with facial palsy and spastic facial contracture ([2, 6]). We recently observed this peculiar involuntary movement restricted to the periorbital muscles in 20 leprosy patients.

While clinically assessing the inmates of two different leprosy colonies in North India, in the leprosy clinic at our institute, 20 patients out of a total of 58 examined were found to have involuntary continuous movements of the periorbital muscles. This disorder was diagnosed as myokymia.

A detailed history of the duration of leprosy, myokymia, facial palsy, nausea, vomiting, dimness of vision and diplopia was obtained. A thorough clinical examination with special emphasis on testing of the facial (VIIth), oculomotor (IIIrd), trochlear (IVth), trigeminal (Vth) and abducent (VIth) cranial nerves was carried out. Other neurological examinations included assessments for higher functions, speech, motor and sensory functions, joint, position and vibration sense. An ophthalmoscopic examination was carried out in every patient.

In all 20 individuals the myokymia was bilateral; 12 patients were males, 8 were females. Their age range was 50 to 82 years (mean 59 years) and duration of their disease varied from 12 to 55 years (mean 36.15 years). The duration of myokymia in 10 patients who were aware of it varied from 6 months to 5 years (mean 3.2 years), while the duration of facial palsy ranged from 10 to 25 years (mean 14.5 years). Fourteen out of 20 patients had polar lepromatous leprosy (LL_P); 6 had subpolar lepromatous disease (LL_S). Only 6 patients had associated