

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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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

facial palsy; 5 had unilateral and 1 patient had bilateral facial palsy.

None of the patients had evidence of optic neuritis, nystagmus, palsy of any other cranial nerve, tremor, ataxia, paralysis of ocular movements, abnormality of gait or alteration of position, or vibration sense. There was no evidence of high intracranial pressure in any of the patients.

Myokymia needs to be differentiated from blepharospasm, an involuntary recurrent spasm of both eyelids that occurs in elderly persons as an isolated phenomenon with varying degrees of spasm of other facial muscles (⁵). Since all of our patients were 50 years or older, this possibility had to be excluded. However, there was no frank spasm or closure of eyelids as seen in blepharospasm. Moreover, 50% of our patients were unaware of their myokymic movements. Only six patients had associated facial palsy and none had facial contracture. Myokymia was restricted to the periorbital muscles and did not extend to the facial muscles in any patient. There was no correlation of myokymia with the duration of leprosy or facial palsy and its duration, however all patients had multibacillary disease.

The absence of tremor, optic neuritis, paralysis of cranial nerves other than the VIIth, paralysis of limbs, dysarthria, and internuclear ophthalmoplegia (INO) ruled out the possibility of multiple sclerosis in our patients (¹).

The possibility of pontine tumors or vascular events was ruled out in view of the absence of high intracranial pressure and the lack of paralysis of the Vth, VIth, and VIIth cranial nerves (⁶).

Whether the occurrence of myokymia is a manifestation of incipient facial nerve damage or some unidentified central ner-

vous system changes brought about by leprosy, or just a chance association, is a matter of conjecture. To the best of our knowledge, this is the first report of its kind in the literature.

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Reduced Fingertip Blood Flow and Peripheral Dysautonomia in Leprosy Patients in Iranian Azerbaijan and in Maharashtra, India

TO THE EDITOR:

Damage to the somatic motor and sensory pathways in the nerves is well recognized in leprosy neuropathy but autonomic function can also be disturbed (⁴). Recently,

we have described a sensitive technique for the measurement of fingertip blood flow by laser Doppler flowmetry, and have used the method to assess the competence of skin sympathetic (vasomotor) reflexes control-