FIELD DETECTION OF EARLY NEURITIS IN LEPROSY

Sir,


After our patients' hands have been checked for muscle weakness, the examiner runs forefinger and thumb of each hand over the patient's little fingers simultaneously comparing the stickiness of the two fingers.

This delicate test gives a reliable indication—sometimes the earliest sign—of ulnar neuritis. If positive, relative dryness will be found on one side due to early diminution of sweating. This should be recorded immediately and followed up by a full functional check-up, and of course steps taken to prevent progression of the neuritis.

A similar test could be applied to the feet. While stroking the foot in Fritschi's last procedure, a mental note is made of the relative dryness of the two sides. In this case dryness of the sole could indicate an early tibial palsy.

Once again the dryness, when found, is recorded and appropriate action taken.

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[Our reproduction of figure 6 on page 175 of Dr Fritschi's publication was perhaps somewhat short of optimum quality; to check the sensory area of the posterior tibial nerve, the examining finger should stroke the lateral part of the sole of the foot. The lateral border of the foot is supplied by the sural nerve. Editor.]

THALIDOMIDE IN ERYTHEMA NODOSUM LEPROSUM (ENL)

Sir,

An adult male patient from Bosé, Ikom Local Government Area, Cross River State, Nigeria, presented himself to us at Moniaya Leprosy Hospital, on 20 March 1985. He reported that the first lesion had appeared on the right cheek in 1984, about one year before. At the time of reporting he had erythematous nodules on the upper and lower limbs. Both ulnar nerves were enlarged and tender. He was classified as having borderline lepromatous (BL) leprosy.

Smears taken on 21 March 1985, were reported as follows: +1, +4, +2, +1. He was started on multidrug therapy (MDT) with rifampicin, dapsone and clofazimine. On 9 May 1985, he suffered from a severe reversal reaction, and because of severe neuritis, he was given steroid therapy. On 24 May 1985 it was reported that the lesions were still active. They were on the thighs and lower limbs. On 21 June smears were repeated: −, −, +, +, +, −. (6 sites).

I saw him for the first time on 22 January 1986. He was suffering from an ENL reaction. For four days he had tender nodules on the arms and legs, and he complained of severe pain around the left elbow. On examination I found impaired function of the left ulnar nerve (3/5), the left median nerve (4/5) and the left radial nerve (4/5). I started him on another course of steroids, i.e. six months of dexamethasone, beginning with 4 mg daily and reducing the dose monthly. Because of the severe pain the left arm was splinted and he was given analgesics. Both ulnar nerves were enlarged and tender, graded 1/3 (right) and 0 (left), (normal = 3)

On 26 February he was having agonizing pain. I made a diagnosis of ulnar nerve abscess and took him to theatre for incision of the nerve sheath, eased the pain; the nerve has not been tender since.

On 6 April he suffered from another ENL reaction. He had pyrexia and ulcerating nodules on the arms and legs. He was given increased doses of lamprene, chloroquine and panadol, and the
Letters to The Editor

steroids were increased to maximum dosage again. There was little improvement. A week later he was still suffering from the severe ENL reaction. Bullae appeared and we wondered if this could have been due to any of the drugs. Eventually the reaction subsided, only to be followed by another a month later.

At this stage the right ulnar nerve function became markedly impaired. Impaired function of both median nerves and both peroneal nerves became apparent. He developed marked sensory loss in the feet. He continued to suffer much pain and was subject to repeated episodes of ENL.

To try to locate an underlying cause for the repeated ENL reactions, a few laboratory tests were done: Hb, 10/g; thick drop, malaria—nil/filaria—nil; stool and urine, normal.

He continued to suffer intensely until a limited supply of thalidamide was obtained and on 23 June he began a course: 200 mg BD for 7 days; then 100 mg BD for 7 days; and then 100 mg daily for 7 days.

On 5 July a full review was carried out. The pain had almost completely gone. The skin was clear of nodules. He was feeling very much better. At this stage he was on thalidamide 100 mg daily and the steroids were being reduced.

He was receiving intensive physiotherapy. He was a cooperative patient and diligently worked at the exercises. His main complaint at that time was stiffness of the hands. By now there was marked impairment of function of both ulnar nerves, the right median nerve and the left peroneal nerve. He also has insensitive feet. This impairment of function seemed to happen under our very eyes and there seemed so little we could do for him in the face of the repeated ENL reactions.

However we have no doubt about the remarkable effect that the thalidamide had. It resulted in immediate relief and heralded the beginning of the slow healing process. Unfortunately, as we reduced the thalidamide, ENL nodules reappeared and so we increased the dose again. Then our supply ran out. Three weeks later he had another ENL reaction. It was not quite as bad as previous episodes but he did have pyrexia and painful nodules. Now he is back on thalidamide and we plan to continue this treatment for a few months. He is now free from complaint.

I would be most interested in any comments, criticisms on our management, or advice that anyone would care to give, for I would certainly not like to see another patient suffer in this way.

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CORTICOSTEROID-INDUCED ACTIVATION OF CHRONIC ULCERATION IN LEPROSY

Sir,

The immune suppressive effect of corticosteroids is well known and widely used in the management of reversal reactions in leprosy. The danger of provoking spread of tuberculosis by such medication is commonly recognized, but the potential dangers from plantar ulcers, without frank discharge of pus or other signs of active infection, are frequently ignored. That this can have disastrous results is illustrated by two cases seen at ALERT, Addis Ababa, in neither of whom was there any evidence of active, progressive infection.

Case 1. A borderline lepromatous patient in reversal reaction received corticosteroids in spite of chronic forefoot ulceration, which was considered indolent and inactive. Two weeks after the start of corticosteroid treatment the right foot became swollen and pus seeped from the previously inactive ulcer. Surgical consultation was requested. The radiogram showed bunching of the dorsal surfaces of talus and of naviculare, but no plantar displacement of the latter. There was capsular