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
new bone formation at the first metatarso-phalangeal joint and periosteal new bone formation of the shafts of the second, third and fourth metatarsal bones with sclerosis. These changes were interpreted as the result of chronic forefoot ulceration, probably with infective changes of the first metatarso-phalangeal joint, while the changes of the metatarsal shafts are considered to be due to toxic damage from the ulceration. In addition there was erosion and lysis of the distal and proximal phalanx and of the metatarso-phalangeal joint of the great toe. These were considered to be fresh, progressive changes, undoubtedly provoked by the administration of corticosteroids.

Case 2. A borderline tuberculoid patient in reversal reaction received corticosteroids in spite of chronic midfoot ulceration, which was considered indolent and inactive. Within 2 weeks of the introduction of corticosteroids, the foot became swollen with discharge of pus from the ulcer and several sinuses. Surgical consultation was requested. The radiogram showed two sclerotic remnants of metatarsal shafts, indicating longstanding bone changes from loss of sensation. Otherwise, the picture was one of violently spreading infection throughout the whole foot, including the ankle joint.

Eventually both patients required major, ablative surgery, midfoot amputation, respectively, below the knee amputation under heavy antibiotic cover.

Ideally no patient with any evidence of secondary infection, even the so-called chronic, inactive ulceration, should receive corticosteroids, but since plantar ulceration is such a common feature of leprosy, this is obviously an impossible demand.

However, all patients who are considered for corticosteroids should be carefully examined and watched for secondary infection. If possible, surgical consultation and intervention should be requested before corticosteroid treatment is instituted.

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LEPROMA OF THE METAPHYSIS

Sir,

A 20-year-old Ethiopian with slit-skin smears positive for acid-fast bacilli presented with a clinical diagnosis of polar lepromatous leprosy and an acute, hard swelling of the right elbow region.

The radiogram was interpreted as an osteoclastoma. A drill biopsy was taken to confirm this but unexpectedly the tumour was found to be a lepromatous granuloma, containing many acid-fast bacilli, both intact and broken and also many globi. Under continued treatment for leprosy the tumour regressed as did the skin manifestations.

Lepromata of cancellous bone are well-known, particularly of the fingers in relation to the proximal interphalangeal joints. So far only one report of leproma of cortical bone has appeared1 and it is of interest that in both cases the identical misdiagnosis was made initially.

Leproma of bone should be suspected whenever a cystic lesion is found in a bacillary patient. Since the leproma can be expected to regress under medical treatment, no specific treatment is indicated, except support of the region to avoid collapse of the bone, until satisfactory healing has taken place.

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Reference