NONSEPTIC TARSAL DISINTEGRATION IN LEPROSY

Sir,

Bone changes in leprosy have been described as the result of direct invasion of the skeletal structures with lepromatous granulomata, or secondary infection, or as absorption related to loss of sensation. Characteristically the major joints remain intact and this is ascribed to the preservation of proprioceptors in leprosy, even in the total absence of other forms of sensation. This is completely different from syphilis, where the proprioceptors are usually lost (e.g. tabes dorsalis), and the major joints typically are affected.

This letter describes two patients with documented borderline lepromatous leprosy who also had marked loss of sensation, including joint sense.

Patient 1. A 50-year-old male patient with well treated, though still skin-smear positive, borderline lepromatous leprosy presented for treatment of a severely swollen foot. No evidence of diabetes, syphilis or rheumatoid disease was found.

Radiologically the foot was completely disorganized with total disruption of the ankle joint and the midtarsal joints with erosion of the adjacent joint surfaces. There was lysis of the navicular and cuneiform bones and the cuneiform bone with some new bone formation of a hazy nature. There was concentric absorption of some of the metatarsal bones and of two of the remaining phalanges. Aspiration of the copious fluid produced no growth on culture and this was later confirmed during surgery.

The extremity was completely anaesthetic almost to the knee, with loss of vibration and joint position sense. No evidence of past or present plantar ulceration was seen.

Patient 2. A 40-year-old female patient with well treated, though still skin-smear positive, borderline lepromatous leprosy presented with a chronically swollen foot. No evidence of diabetes, syphilis or rheumatoid disease was found.

Radiologically the talo-navicular, talo-calcaneal and calcaneo-cuboid joints were disrupted and there was new bone formation of hazy character.

The extremity was completely anaesthetic up to midcalf, with loss of vibration and joint sense. No evidence of past or present plantar ulceration was found.

Both patients had faithfully worn correctly designed shoes, which had obviously protected the feet against ulceration, but failed to compensate for loss of deep and proprioceptive sensation. Both parties required major, ablative surgery. I would be interested to hear of other cases and to know if sensory neurological testing has a predictive value in such cases?

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RIFAMPICIN MONOTHERAPY IN PAUCIBACILLARY LEPROSY

Sir,

It was interesting to read the results of treatment of paucibacillary (PB) leprosy with ten weekly doses of rifampicin (Lepr Rev 1987, 58: 349–58). However, I do not think that it is prudent to use rifampicin monotherapy even in PB leprosy patients for the following reasons:

1. The threshold of $10^6$ organisms for the natural occurrence of drug resistant mutants is applicable to Mycobacterium tuberculosis and we do not really know whether the analogy is applicable to M. Leprae.

2. The said threshold is for the drugs against which resistance develops in a stepwise fashion and not for the drugs against which it develops in a single step. The resistance of M. leprae against rifampicin develops as a single step process. It has developed earlier than that against dapsone.
3 After the unfortunate experience of dapsone monotherapy and case reports of rifampicin resistant leprosy, rifampicin monotherapy appears to be unjustified even in PB leprosy patients. If resistance to rifampicin becomes ubiquitous as has happened with dapsone, we will lose the most potent antileprosy drug available to us today.

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References


FIELD DIAGNOSIS OF EARLY LEPROSY

Sir,

I would like to comment on Dr Smith’s paper (Lepr Rev, 1987; 58: 141-8) which describes the use of a questionnaire of 20 case histories. The diagnosis of leprosy is based on the presence of at least one of the three cardinal signs of anaesthesia, thickening of peripheral nerves at the sites of predilection and the finding of acid-fast bacilli. The 20 cases reported give no details of skin-smear results and diagnoses are made in the absence of any of the cardinal signs, e.g. case history 3 and 18.

I note also that the location of the hypopigmented patches influences the diagnosis in two similar cases, when lesions are on the face (case 1) the response is ‘suspect’ while when on the buttocks (case 18) ‘affected’ is comparatively preferred.

I disagree with the diagnosis in case 1, since there is a history of contact with an infectious case ‘affected’ would be the possible correct diagnosis. I also question the diagnoses in cases 1, 8 and 12 where the sex of the child seems to influence the decision and I would disagree with the diagnosis in cases 13 and 19 which I find confusing.

However, despite my cautionary comments I do appreciate the attention that this paper gives to this much neglected area of leprosy.

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REPLY—FIELD DIAGNOSIS OF EARLY LEPROSY

Sir,

I have read Dr Kulkarni’s comments carefully and I welcome the opportunity to reply.

I wholeheartedly agree with Dr Kulkarni that this is indeed a much neglected area in leprosy and it has thus been with some trepidation that I have attempted to tackle the subject of the field diagnosis of early leprosy.

In defence of the ‘standard’ diagnoses used in the case histories I would point out that the majority of the 79 field workers who completed the questionnaire agreed with the standard