Letters to the Editor

HOW TO DETECT LEPROSY IN SOME PATIENTS WITH ONLY LOCALIZED SENSORY LOSS

Sir,

Now and then, particularly in endemic areas, we are confronted with a situation where in the presence of only symptoms, a definitive diagnosis or exclusion of active infection with leprosy remains unfulfilled. Three such cases presented here highlight this point.

Case No. 1

A 16-year-old boy from the state of West Bengal came with sensory loss over the left foot of 6 months' duration. On examination there was loss of fine touch, thermal and pin-prick sensations on the dorsum of the left foot extending to the posterior and lateral parts of the lower third of the leg including the heel. Regional and sensory nerves coursing the area were not thickened or tender. No other lesions were seen on the entire skin surface and there was no history of leprosy in the family. Biopsy of the skin from the dorsum of the foot and the left sural nerve were reported normal after examining multiple sections for acid-fast bacilli (AFB).

Case Nos 2 and 3

Two men from the state of Bihar aged 21 years and 42 years sought advice for numbness over the lateral part of the dorsum of the right hand of 6 months' and 9 months' duration respectively. Previous consultation with the neurologist had disclosed no abnormality. As in the first case, the sensory deficit was on the dorsal aspect of the thumb, index finger and extended downwards along the lateral one-third of the right hand to the wrist. The rest of the skin appeared normal. A biopsy of the cutaneous branch of the radial nerve which courses over the lateral border of the wrist was done. Histopathology showed no evidence of leprosy.

Comment

The cardinal signs that justify a diagnosis of leprosy are: (1) clinical features of nerve involvement like loss or impairment of sensation on a skin patch or an extremity, and thickened or tender nerve(s); and (2) demonstration of *Mycobacterium leprae* in the skin by slit-skin skin smears which will reveal acid-fast bacilli. The clinical and routine laboratory criteria for the classification of leprosy across the spectrum from tuberculoid to lepromatous, along with the inclusion of the indeterminate variety has definitely helped workers to comprehend the severity of the disease and satisfactorily monitor drug therapy. The 3 patients seen here from highly endemic areas of the country had only localized sensory loss. The histopathology of the nerves showed no evidence of leprosy. This makes it difficult to decide whether to give the benefit of therapy or withhold it. Eminent workers with lifelong experience in leprosy have faced such problems\(^1,2\) and have suggested that a thorough histological study for the presence of perineural infiltrate and/or AFB in the biopsy
specimen should be undertaken, in the absence of which the patients had to be followed up because it was felt that no one should be unnecessarily branded as having leprosy.

These patients may represent the early stage of the disease, or it is also possible for the infection to have been arrested by the body’s immune mechanisms. Since the former requires therapy it is vital to develop tests to distinguish these two conditions and this is not possible with the investigations currently in vogue. The limited use of serological studies in endemic areas using both *Mycobacterium leprae*-specific monoclonal antibody and *M. leprae*-specific antigen has been evaluated and found useful in identifying the multibacillary cases, but in the rest there was slender or no correlation between the presence of disease and seropositivity. The relevant differential diagnoses in our patients include pressure neuropathies like carpal tunnel syndrome, peripheral neuritis caused by the ingestion of drugs containing heavy metals, traumatic neuropathy, and metabolic causes like diabetes mellitus and amyloidosis. There was no evidence to suspect any of these conditions.

Leprosy is primarily a neural disease and future research must aim at developing a test that can detect the lurking AFB or its antigenic components in the biopsy from the peripheral nerve(s) in the early stages of infection. Till this is achieved our advice to medical and paramedical field workers must be to send such patients to the nearest centre for appropriate investigations and, in the absence of any confirmatory evidence, they must be followed up in their respective places to note whether they develop other signs of the disease.

References


REPLY: THE USE OF XYLENE (XYLOL) IN MEDICAL LABORATORIES

Sir,

I was interested in Dr A C McDougall’s comments and questions concerning the deterioration of acid-fast colouration of mycobacteria during the examination of smears (*Lepr Rev*, 1989, 60: 67).

In my experience *Mycobacterium leprae* in smears is irregular in its retention of acid-fast stain. On occasion the colouration may withstand immersion oil, xylene or mountant, but at other times it unaccountably fades. This unpredictability applies to all forms of bacilli, solid, fragmented and granular, and to bacilli from different sites in the same patient. For this reason I would recommend that a leprosy smear be examined as soon as it is stained; it can be left fixed and unstained until a suitable time.

Once the colouration is lost the only sure way to restore it is to re-stain the smear as for a tissue section, but using a slightly modified technique as follows. Immerse the smear in xylene for 15 min, rehydrate, stain in cold carbol fuchsin for 15 min, dry in air, apply a drop of turpentine (pinene) to one side of the smear and tilt the slide so that the turpentine flows over the smear, differentiate immediately without drying in 25% acetic acid, wash in water, dry, counterstain and examine. The staining and bacterial morphology are restored.