TUBERCULOID RELAPSE IN LEPROMATOUS LEPROSY

Sir,

Waters & Ridley[1] recently reported 6 patients who had been lepromatous and who, after many years of chemotherapy and bacteriological negativity, were found on relapse to have upgraded to borderline-tuberculoid (BT) leprosy. In a related article,[2] they reported the development of weakly positive lepromin reactions in some lepromatous patients treated for more than 22 years; a control group of BL patients remained lepromin negative, when less than 20 years of treatment had been given.

We have recently seen tuberculoid relapses in 2 previously lepromatous patients, from western Uganda, 7 and 12 years after initial diagnosis. They had had multiple-drug regimens and had never shown signs of reaction. Condensed histories of both cases are given below.

Case 1

A.M., female, aged 20, presented in 1979 with a 2-year history of multiple skin lesions. No nerves were enlarged and she had no disability. Smears showed a BI of 4 at all sites and an MI of 20%. She was classified clinically as BL, but no biopsy was done. She was treated for 10 years with dapsone and clofazimine, and also had rifampicin (1500 mg) as an annual dose in 6 of the 10 years. Follow-up smears were negative after 1983 and treatment was stopped in 1989. Attendance has been good throughout and she remained without any disability.

After stopping treatment for 10 months she presented with about 20 new lesions, suggestive of borderline-tuberculoid leprosy. Skin smears of the lesions were negative and the biopsy was reported as follows:

> The biopsy shows a borderline-tuberculoid leprosy with dermal nerve disruption; no acid-fast bacilli were seen in multiple sections.

Several family members and neighbours have had leprosy, so a reinfection is a possibility. There is no clinical sign of a reaction and otherwise she remains well. MDT (WHO—2-year regimen) is in progress. A recent HIV test was negative.

Case 2

E.N., male, aged 29, presented in 1984 with a 1-year history of ill-defined macular skin lesions; he had some infiltration of the ear-lobes and his skin smear had an average BI of 2·8 (highest 5) and an MI of 2%. One patch was well-defined with marked loss of sensation, suggesting that he was down-grading from BT to BL or LLs. He had no disability and no biopsy was done.

He was treated with dapsone and clofazimine for 5 years, and also had 6 doses of rifampicin (1500 mg) at 12-monthly intervals. Smears were negative after 1985; his attendance was good and he had no disability.

After stopping treatment for 13 months he presented with about 25 new lesions, typical of borderline-tuberculoid leprosy. The smear was negative and a biopsy was reported as follows:

> 'The histology shows a borderline-tuberculoid leprosy with no evident acid-fast bacilli.'

He has had no sign of a reaction and is currently making good progress on MDT (WHO—2-year regimen). There is no disability and he is HIV negative.

Discussion

Both cases were clinically BL when first treated, supported by the fact that the smears became negative quickly. It is unclear what the new lesions are due to, but DDS-resistance is not a factor, because 3 drugs were used from the beginning; HIV infection, widespread in the area, is also excluded in these cases.

Bacterial relapse due to persisters or reinfection, and delayed hypersensitivity reaction are all possibilities, none being excluded by the biopsy. Waters & Ridley[1] comment that BT lesions could represent a late reversal reaction, not necessarily a relapse, in a BL patient who had taken 12 years of DDS. On the other hand, neither of our cases had any clinical features of a reversal reaction, that is, the lesions were uninflamed and there was no pain or neurological deficit.

In one of our cases there was evidence of recent down-grading on initial presentation and it is perhaps not surprising that silent (or symptomatic) up-grading occurs quite rapidly in this situation. However, it remains the case that both these patients have moved from being near-lepromatous to being tuberculoid, without an overt reaction, in approximately half the time found by Waters & Ridley.

50 Abbots Way
Newcastle, Staffs, UK
P. SAUNDERSON

POB 4008
Kampala, Uganda
K. WADDELL

References

[1] Waters MFR, Ridley DS. Tuberculoid relapse in lepromatous leprosy. *Lepr Rev*, 1990; **61:** 353–65.

[2] Waters MFR, Ridley DS., Lucas SB. Positive Mitsuda lepromin reactions in long-term treated lepromatous leprosy. *Lepr Rev*, 1990; **61:** 347–52.