CLASSIFICATION OF TREATED LEPROSY PATIENTS IN THE ABSENCE OF ADEQUATE RECORDS

Sir,

Facing the problem of having in our treatment register several hundred patients who had a history of anti-leprosy treatment prior to registration with us but whose original classification and signs of leprosy were unrecorded, we wished to evaluate the accuracy of our classifications. We hoped to develop for our paramedical workers guidelines for classifying such patients more accurately.

We took a random sample of 56 patients from those who remained on treatment because their most likely classification was considered to be LL leprosy. This classification was made 7–9 years after their beginning treatment with us, on the basis of either repeatedly positive slit skin smears or clinical findings conventionally considered as residual indicators of LL leprosy, namely: madarosis; nasal collapse; pattern of anaesthesia; pattern of digit loss; signs of old papules, nodules, infiltration or ENL; history. Where the only evidence of LL leprosy was clinical, indicators from at least two categories were required.

These 56 patients were tested with lepromin (Mitsuda H, supplied by courtesy of Dr R J W Rees) on the volar side of the left forearm 15 cm proximal to the most distal wrist crease. Eleven patients whose first slit skin smear following registration had shown a BI greater than 1+ are excluded from the short analysis presented here.

For the remaining 45 patients, Mitsuda reactions (read at 26–36 days after injection) were as follows: 27 (60%) negative (no induration detectable); 8 (18%) doubtful (induration 1–3 mm); 10 (22%) positive (induration > 3 mm).

The following clinical findings had a useful predictive value for a negative Mitsuda reaction:

		Total no. of patients with this sign	Predictive value of this sign for complete absence of detectable cell-mediated immunity against <i>Mycobacterium leprae</i> (within this group) (%)	No. of patients with this sign and little or no cell-mediated immunity (Mitsuda reaction 1–3 mm)
	Definite flattening			
	of nasal bridge	9	67	3 (33%)
2	Anaesthesia on nose	6	67	1 (17%)
3	'Lepromatous face'—			
	flattened nasal bridge			
	+ some loss of eyebrows			
	+ earlobe abnormality			
	suggesting old infiltration or papules	4	75	1 (25%)
4	Definite anaesthesia			\ /0/
	of both cornea	4	75	1 (25%)
5	Anetoderma suggestive			(/ 0/
_	of old papules/nodules			
	(but excluding ears)	12	75	3 (25%)
6	Definite loss eyebrows	12	75	2 (17%)
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7	signs no. 4, 5, 6	6	83	1 (17%)
	nodules consistent with leprosy lesions	16	75	3 (19%)

Surprisingly, the pattern of anaesthesia, the pattern of loss of digits and signs of previous thickening of (or nodules in) the earlobes showed no correlation with Mitsuda reaction sizes.

Thus it would seem that on clinical grounds alone without the help of lepromin testing, it is difficult to correctly identify previously treated and inactive lepromatous patients with no detectable cell-mediated immunity. Since this is a group which in the past might have had to receive lifelong dapsone monotherapy and now should perhaps be considered to require triple therapy before discharge or lifelong surveillance without treatment and whose identification therefore is desirable, our findings might be of interest to some of your readers.

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