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Leprosy in children: correlation of clinical, histopathological, bacteriological and immunological parameters

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Summary The study of leprosy in children has indicated an incidence of 10% amongst leprosy patients attending the clinic. The duration of the disease was usually less than 2 years. The expression of leprosy in this group was either a macule and/or a plaque. Classification was, therefore, different and conforms to indeterminate (I), borderline tuberculoid (BT) and borderline (BB) leprosy. Only occasionally were other clinical variants seen. The bacteriology was largely unproductive by slit-skin smears. The lepromin (Mitsuda) responses were positive in BT and unpredictable in BB patients. Epicutaneous responses to sensitization with dinitrochlorobenzene (DNCB) paralleled responses to lepromin. Microscopic pathology was of very little help. The correlation of these parameters was only 50–60% indicating that the diagnosis of leprosy should primarily be based on clinical features.

Introduction

Leprosy is a well-conceived entity in children.[1,3] The reports[3] of ever increasing numbers of children afflicted with the disease reflect that a sizeable population in certain vulnerable areas has an age at onset of leprosy between the ages 0–14 years. In India about 15% of cases belong to this age group.[4] It is generally believed that indeterminate, tuberculoid tuberculoid (TT), borderline tuberculoid (BT), and borderline borderline (BB) groups are frequently recorded amongst children,[5,7] whereas borderline lepromatous (BL) and lepromatous lepromatous (LL) are only occasionally encountered. This appears to be a paradoxical situation in children and runs counter to the concept that 'immune responses' are either negligible or poorly developed in young children.[8] Hence it is worthwhile to comprehend this phenomenon through clinical, bacteriological, histopathological and immunological parameters as prescribed by Ridley & Jopling.[9]

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Patients and methods

Twenty-five children were amongst a total of 250 fresh leprosy patients attending the Urban Leprosy Centre of our Institution, during the National Leprosy Eradication Programme from August 1987 to July 1988. They were classified on the Ridley & Jopling scale[9] with some modifications.[10] They were subjected to skin biopsy for histopathological study. Slit-skin smear examination was done to determine bacteriological status.

Lepromin test was done using 0·1 ml of lepromin A containing 40 million bacilli per ml. The early (Fernandez) response was read after 48 hours and the late (Mitsuda) after 4 weeks. The Fernadez response was graded as negative, doubtful (±), 1+, 2+, 3+ depending upon reaction sizes of less than 5 mm, 5–10 mm, 10–15 mm, 15–20 mm or more than 20 mm respectively. The Mitsuda response was graded as negative, doubtful (±), 1+, 2+ or 3+ depending upon reaction sizes of 0, 1–3 mm, 4–6 mm, 7–10 mm, or more than 10 mm, respectively.

DNCB (Epicutaneous sensitization with dinitrochlorobenzene) testing was done according to WHO.[11] DNCB solution in acetone was used in 2 strengths—a sensitizer dose of 2000 μg and a challenge dose of 50 μg. Solutions were placed on the skin through firmly held steel rings above and below the cubital fossa. In the absence of a reaction at 15 days, a rechallenge with 50 μg was given in a similar way and test read after 48 hr. The response was graded as negative, 1+, 2+, 3+ or 4+. Twelve age- and sex-matched healthy children formed controls.

Observations

Children comprised about 10% of the leprosy cases diagnosed over a 12-month period in our Institute. Their distribution according to age and sex is shown in Table 1. The mean age was 10·6 years, while the duration of the disease varied from 2 months to 10 years, with a mean of 1·8 years. The majority (84%) of them had a disease duration of less than 2 years. Sixteen of them had borderline tuberculoid (BT) and 6 borderline borderline (BB) leprosy. The three other patients were classified as borderline lepromatous (BL), indeterminate (I) and polyneuritic (P) leprosy. Macules occurred in 14 children and plaques in 5 children. Both the lesions were present in the other 5 children. Well-formed granulomas in tissue sections were seen in 13 children, whereas a scattered infiltrate comprising lymphocytes and histiocytes was seen in the rest. In the single case of BL, acid-fast bacilli on slit-skin smear examination were demonstrated (Table 2).

Fernandez response was doubtful (±) in 3 borderline tuberculoid and 1 borderline borderline whereas it was negative in rest of them.

It is apparent from Table 2 that the late (Mitsuda) reaction depicted varying positivity from BT to BB leprosy. Moreover, there was no difference in sensitization with DNCB in the BT group and

Table 1. Age and sex distribution

	Males		Females	
Age group (Years)	No. of cases	%	No. of cases	%
0–5	1	6·25	1	11·11
6–10	5	31·25	3	33·33
11–14	10	62·25	5	55·55
	16	100·00	9	100·00

Table 2. Correlation of various parameters

No.	Clinical Diagnosis	Histopatho-logical	Bacterial index	Lepromin (Mitsuda)
5	BT	BT	0	1+
3	BT	BT	0	2+
1	BT	BT	0	3+
3	BT	NS	0	1+
2	BT	NS	0	2+
1	BT	NS	0	±
3	BB	BB	0	±
2	BB	NS	0	1+
1	BB	NS	0	±
1	BL	BL	5+	±

NS, non-specific

Table 3. Response to DNCB

Leprosy groups	No.	Grading of response				
		–	1+	2+	3+	4+
BT	16	1	3	5	2	5
BB	6	3	–	2	–	1
BL	1	1	–	–	–	–
I	1	–	–	1	–	–
P	1	–	–	1	–	–
Total	25	5	3	9	2	6
Controls	12	1	1	2	4	4

Table 4. Correlation of lepromin (Mitsuda) and DNCB response

Lepromin response	No.	DNCB response				
		–	1+	2+	3+	4+
–	0	–	–	–	–	–
±	6	4	–	2	–	–
1+	11	1	1	4	–	5
2+	7	–	1	3	2	1
3+	1	–	1	–	–	–

controls (Table 3). In the BB group, on the other hand, only three children could be sensitized—a response which is significantly poor when compared to BT and controls. ($p < 0{\cdot}05$). The correlation between lepromin (Mitsuda) and DNCB sensitization is shown in Table 4. The correlation of various parameters namely clinical, histopathological, bacteriological and lepromin (Mitsuda) is shown in Table 2. It is evident that a clinical–histopathological correlation was seen in 9 BT, 3 BB and 1 BL patient.

Discussion

It is well-established that children are more susceptible to acquiring leprosy than adults.[12] However, the spectrum of the disease among them is different. It is characterized by macules and/or plaques present more often on exposed areas.[7] Macule is a predominant lesion. Accordingly, the histopathology also reflects a non-specific picture in a large number of patients. This aspect has been well-documented in our study. As the formation of a granuloma is indicative of effective build up of immunity,[13] it follows that in children immunity was not as effective as in adults.

The Fernandez response to lepromin indicates a pre-existing hypersensitivity to either *M. leprae* or other cross-reacting Mycobacteria.[14] It appears that negative or doubtful responses in children could be due to inadequate pre-exposure to such organisms or to the inability of the immune system to respond to such exposure. The response pattern of the Mitsuda reaction was almost similar to that of adults in our study. A positive response in the indeterminate and the polyneuritic patients indicates their place nearer to the tuberculoid end of the spectrum. The impairment of DNCB responses in conformity with negative or doubtful Mitsuda reactions in BB and BL leprosy indicates the parallel impedement of specific and non-specific CMI.

A correlation of various parameters namely clinical, histopathological and lepromin (Mitsuda) responses was seen in only 56·7% of BT children. In the clinically BB group only 50% conformed to the histopathological picture of BB. Moreover, slit-skin smears failed to demonstrate acid-fast bacilli in any of them. Such discordance among various parameters has earlier been reported.[15 18] In one double-blind study,[15] such correlation was found to be only 44%. Our study, therefore, reiterates that clinical criteria should be the mainstay in the diagnosis of leprosy in children.

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