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Childhood leprosy in an urban clinic, Hyderabad, India: clinical presentation and the role of household contacts

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Summary A retrospective case note study was done of children below the age of 14 years who attended Dhoolpet Leprosy Research Centre (DLRC) over the decade 1990–1999. The aim of the study was to describe the pattern of clinical presentation, the role of household or near neighbour contacts and the incidence of neuritis and reactions. In all, 3118 leprosy patients were registered during this period, of whom 306 were children [182 (60%) male]; 95 children had a single patch, 159 had five or fewer than five patches and 37 had multiple patches. The youngest case detected was 9 months old. The spectrum of leprosy in these children was: TT 62 (20·3%); BT 203 (66·3%); BB 3 (1%); BL 23 (7·5%); LL 5 (1·6%) and PNL 10 (3·3%). Twenty-nine cases (9·4%) were smear positive. Ninety-one children (29·7%) developed a reaction, 86 type I and five type II. A history of contact was present in 119 (38·8%) cases, family contact in 113 (95%) and other than family in six (5%). Classification of the contact was available in only 60 patients. Among the contacts of the index case, 21 (35%) suffered from PB leprosy and 39 (65%) from MB leprosy. All contacts were from the immediate family. This study shows that childhood leprosy cases continue to present in significant numbers to this outpatient clinic. There is a high level of family contact with leprosy in these cases, strengthening the strategy of screening children in leprosy-affected households. The high incidence of reactions and nerve damage in children emphasizes the importance of early detection and treatment.

Introduction

Children in leprosy endemic areas are exposed to infection by *Mycobacterium leprae*. A study in 1978 among school children in endemic area shows the prevalence rate to be 10–17/1000.[1] About 17·13% of all the leprosy cases in India are children below 15 years of age.[2,17]

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Studies have suggested that the presence of a household or neighbourhood contact of leprosy increases the risk of infection and development of leprosy,[3] with an increased risk with a multibacillary leprosy contact.[4,11,13]

This study was carried out to document the pattern of family contact with leprosy among children and to describe the clinical presentation and evolution of the disease in children affected by leprosy.

Materials and methods

The case records of children who were below 14 years of age at registration at Dhoolpet Leprosy Research Centre (DLRC), during the period from January 1990 to December 1999 were analysed. The children were either brought to the clinic by their parents or relatives, or occasionally referred from other health facilities. The age, mode of presentation, number of skin patches, involvement of nerves, skin smear status and occurrence of reactions were noted. The cases were classified according to the Ridley-Jopling classification.

A detailed contact history with leprosy was taken with reference to location and type of contact. A 'family contact' was defined as a person suffering from leprosy in the immediate family; like parents, siblings, and grandparents living in the same house. The cases in the neighbourhood were defined as 'other than family contact' and these were people living in the immediate neighbourhood.

Nerve involvement was defined as clinical thickening of the involved nerve and was graded as 1+, 2+ and 3+ depending on the clinical judgement of the examiner. Pain and tenderness of the nerves was also recorded. The records for muscle and sensory testing were not consistent and complete.

Reversal or type I reaction was defined as the sudden appearance of erythematous and raised patches in pre-existing or new skin lesions. When patients developed erythematous tender subcutaneous nodules with associated systemic features, this was designated an ENL or type II reaction. Neuritis was defined as deterioration in sensory and/or motor function.

Results

In all, 3118 leprosy cases were registered at DLRC during the 10-year period, and 306 children with leprosy were detected. Of these, 182 (60%) were male.

Almost an equal number of cases were distributed in the age groups 6–10 and 11–14 years (Table 1) and they together constituted 94·1% of the children with only 5·8% being <5 years of age. The youngest case detected was a 9 month old child with BT leprosy. The Ridley-Jopling classification of these patients was: TT 62 (20·3%); BT 203 (66·3%); BB 3 (1%), BL 23 (7·5%), LL 5 (1·6%) and PNL 10 (3·3%). Eight patients were kept under observation since the lesions were not typical; five of these were reclassified as TT and three as BT during follow-up.

The distribution of patches is given in Table 2. One hundred and eight children had a single patch, 148 had five or fewer than five patches and 39 had multiple patches. The pattern of nerve involvement is shown in Table 3. Single trunk involvement was found in 61 children; two nerves in 57 children, three nerves were involved in 21 children and more than three nerves were involved in 47 cases.

Table 1. Age distribution with spectrum of leprosy

Classification		<1 year	1–5 years	6–10 years	11–14 years	No. of cases %
TT		9	35	18	62	20·3
BT	1	8	91	103	203	66·3
BB			2	1	3	1
BL			10	13	23	7·5
LL			1	4	5	1·6
PNL			3	7	10	3·3
Total	1 (0·3%)	17 (5·5%)	142 (46·4%)	146 (47·7%)	306	100

Table 2. Number of patches

Classification	Single	5 and <5 patches	>5 patches
TT	60	2	–
BT	48	142	13
BB	–	1	2
BL	–	2	20
LL	–	1	4
Total	108	148	39

Presenting in reaction/or with neuritis was common and occurred in 79% of the children who had reactions. A small proportion (8.8%) had reaction after completing MDT (Table 4). Ninety-one (29·7%) children developed reactions (Table 5); five went into type II and 86 into type I reaction. Among the type 1 reactions 12 had reversal reaction (RR) alone, 15 had RR with neuritis and 59 had neuritis alone. Of the 12 cases of reversal reaction, all improved without any recurrence. One child had both type I and type II reaction. Although numerically the BT patients ($n = 67$) contributed the greatest number of reactions and nerve damage, a

Table 3. Pattern of nerve involvement

I. Truncal nerve involvement			
Name	Unilateral	Bilateral	Total
Ulnar	86	61	147
Median	22	10	32
Lateral popliteal	44	43	87
Post. tibial	39	27	66
Facial	2	–	2
II. Cutaneous nerve involvement			
Name	Unilateral	Bilateral	Total
Great auricular	5	11	16
Radial cutaneous nerve	33	16	49

Table 4. Time of incidence of reactions

Timing of reaction	Reversal reaction		Reversal reaction with neuritis		Neuritis
At diagnosis	8	11	50	3	72 (79)
Within 6 months of starting treatment		–	1	5	6 (6·7)
After >6 months of starting treatment		1	1	1	2 5 (5·5)
After RFT	3	2	3		8 (8·8)
Total	12	15	59	5	91 (100)

higher proportion of BL (52%), LL (80%) and PNL (40%) patients developed these complications.

Twenty-nine children (9·4%) were skin smear positive and 277 (90·6%) were smear negative. Based on smear status and number of skin lesions, 254 children were started on PB-MDT, 39 on MB-MDT and five children on ROM therapy after it was introduced in the centre in 1998.

Table 6 gives the history of contact for the childhood leprosy cases. A history of contact was present in 119 (38·8%) cases of which, family contact was present in 113 (95%) and other than family in six (5%). Classification of contacts (Table 7) was available in only 60 patients; among them 21 (35%) suffered from PB leprosy and 39 (65%) from MB leprosy. All of these contacts were from the immediate family.

Discussion

DLRC is an urban leprosy clinic with self reporting and referred patients. The compilation of retrospective data has given us some new insights into childhood leprosy.

The preponderance of male to female children of 60%: 40% is similar to the observations made in earlier studies.[4–7,12] The maximum number of cases was in the age group of 6–10 and 11–14 years, with only 5·8% of the cases being less than 6 years. This emphasizes the importance of screening preschool and younger children systematically to detect the disease earlier.

In this study, the majority of lesions were on exposed parts of the body, face, limbs,

Table 5. Classification of leprosy and reactions/neuritis

Classification of leprosy	Reversal reaction		Reversal reaction + neuritis		Neuritis alone	Type ENL
TT	1	1	2	–	4 (4·4%)	4/62 (6·45%)
BT	7	13	47	–	67 (73·6%)	67/203 (33%)
BL	4	1	4	3	12 (13·2%)	12/23 (52%)
LL	–	–	2	2	4 (4·4%)	4/5 (80%)
PNL	–	–	4	–	4 (4·4%)	4/40 (40%)
Total	12 (13%)	15 (16·5%)	59 (65%)	5 (5·5%)	91 (100%)	–

Table 6. Contact history

Type of contact	No. of children	Percentage
Family contact	113	95
Other than family contact	6	5
Total	119	100

forearms and lower limb, whereas other investigators have observed an increased incidence of single lesions on the gluteal region.[8]

In our study, there were 39 children (12·7%) with more than five patches. Of these, 29 (75%) were smear positive and only eight were smear negative (25%). The Ganapathi study, however, found a high proportion (92%) of smear negative children with more than five lesions, who were said to have the potential of developing fully fledged MB disease. Our percentage of smear positive cases is higher than that reported in earlier studies (5·6–6·1%).[1,4,9] This difference could be because their studies are survey based.

The 9-month baby with BT leprosy was the child of a case of LL on MB MDT. In view of her pregnancy, she had stopped taking treatment for 6 months and restarted MDT after delivery. Clofazamine pigmentation was present all over the child's body and in the mother's expressed breast milk. The child developed a large hypopigmented patch on the buttock extending to the thigh.

Data on classification of the contacts were not available in all the patients. From the available data in the present study, 95% of the contacts were from within the family, with 65% being MB contacts and 35% being PB contacts. However, our contact data should be interpreted cautiously, since DLRC is a self-referral clinic. Thus contacts will be aware of the possibility of leprosy and parents are more likely to bring a child with a suspicious lesion to a clinic they are familiar with. Van Beers *et al.* have shown that the risk of a person developing leprosy is 4 times higher when there is a leprosy contact in the neighbourhood. This risk is increased to 9 times if the contact case is within the immediate household.[3] The highest risk of developing leprosy is associated with the presence of a multibacillary patient in the family.[3,7,11,12] The risk of developing of leprosy from a PB contact in the family was similar to the risk from a multibacillary case in the neighbourhood.[3] We also found a significant number of PB contacts (35%) in children with leprosy. This indicates that both types of leprosy and the distance to it are the important contributing factors for the risk of developing leprosy.[3,16] Wu *et al.* found an increasing trend in household cases in a survey done over 45 years, suggesting a decline of infection from other sources in the community.[10]

Table 7. Available data on classification of contact

Type of contact	No. of children	Percentage
PB contacts	21	35
MB contacts	39	65
Total	60	100

Neuritis and the possibility of ensuing deformities in childhood leprosy is a compounded tragedy.[14,15] The high incidence of neuritis in this cohort is striking; neuritis accounted for 65% of reactions, and 24·2% of the whole cohort had neuritis. In most of these children, neuritis was present at the time of diagnosis. This emphasizes the importance of careful neurological examination at the time of diagnosis and appropriate use of steroid in children to prevent further nerve damage.

In conclusion, childhood leprosy forms a significant group in an urban clinic setting and familial contacts probably have a significant role in their development. Contact screening strategies need to stress this aspect.

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References

[1] Jayam S, Sekhar K, Ramasekhar K, Ganapathi R. Childhood leprosy. A study in an urban slum. *Ind Pediatr*, 1978; **15:** 375–377.
[2] Report of Independent Evaluation of National Leprosy Eradication Programme, 27th March to 17th April, 27. (Leprosy Division) Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India, New Delhi, 2000.
[3] Van Beers SM, Hatta M, Klatser PR. Patient contact is the major determinant in incident leprosy: implications for future control. *Int J Lepr Other Mycobact Dis,* 1999; **67:** 119–128.
[4] Prasad PVS. Childhood leprosy in a rural hospital. *Ind J Pediatr*, 1998; **65:** 751–754.
[5] [5]Kumar A, Mathur YC, Rao P. Leprosy in childhood in rural and urban areas of Hyderabad. *Ind Pediatr*, 1977; **9:** 337–338.
[6] Dayal R, Hashmi NA, Mathur PP, Prasad R. Leprosy in childhood. *Ind Pediatr*, 1990; **27:** 170–180.
[7] Kaur I, Kaur S, Sharma VK, Kumar B. Childhood leprosy in northern India. *Pediatr Dermatol,* 1991; **8:** 21–24.
[8] [8]Ganapati R, Naik SS, Pandya SS. Childhood leprosy study of prevalence rates and clinical aspects through surveys in Bombay. *Lepr Ind*, 1976; **48:** 645–660.
[9] Selvaseker A, Geetha AJ, Nisha K *et al.* Childhood leprosy in an endemic area. *Lepr Rev*, 1999; **70:** 21–27.
[10] Wu XS, Ning Y, Shi L *et al.* An epidemiological analysis of leprosy from 1951–96 in Sichuan. *Ind J Lepr*, 2000; **72:** 215–226.
[11] De Matos HJ, Duppre N, Alvim MF *et al.* Leprosy epidemiology in a cohort of household contacts in Rio de Janeiro (1987–1991). [Article in Portugese]. *Cad Saude Publica,* 1999; **15:** 533–542.
[12] Dayal R, Paliwal AK, Prasad R *et al.* A clinico-bacteriological profile of leprosy in children. *Ind Pediatr*, 1989; **26:** 122–128.
[13] George R, Rao PS, Mathai R, Jacob M. Intrafamilial transmission of leprosy in Vellore Town, India. *Int J Lepr Other Mycobact Dis*, 1993; **61:** 550–555
[14] Hammond PJ, Sundar Rao PSS. The tragedy of deformity in childhood leprosy. *Lepr Rev*, 1999; **70:** 217–219.
[15] Nadkarni NJ, Grugni A, Kini MS, Balakrishnan M. Childhood leprosy in Bombay: a clinical–epidemiological study. *Ind J Lepr*, 1988; **60:** 173–188.
[16] Suite M, Edinborough NB, Lewis M, Tollefson J. A survey to determine the prevalence of leprosy in a community in east Trinidad. *Lepr Rev*, 1994; **65:** 122–129.
[17] Kant L, Mukherji D. Childhood leprosy. *Ind Pediatr*, 1987; **24:** 105–107.