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Leprosy in Children One Year of Age and Under[1]

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Reports of leprosy in infants one year of age and under are uncommon. Because of the prevailing belief that the incubation period of leprosy is perforce figured in years, diagnoses of leprosy in infants of this age group are frequently disputed. One of us (JB) recently saw two infants in Burundi in whom the clinical signs of leprosy were first observed at six months of age. This prompted us to attempt an assessment of the known prevalence of leprosy in children 12 months old or younger.

METHODS

Data for this report came from three sources: a) published reports (detailed in the Results section), b) responses to a survey by correspondence, and c) the accession files of the Leprosy Registry of the United States Armed Forces Institute of Pathology (AFIP).

Correspondence survey. We sent a letter requesting information on leprosy in children one year of age or under to 551 physicians, paramedical personnel, and others working in leprosy. The list of addressees was largely derived from the membership of the International Leprosy Association, as published in the INTERNATIONAL JOURNAL OF LEPROSY **50** (1982) 606–616. We received 188 responses: most of the responses were from addressees, but a few were from others who had learned of the survey. Only 11 letters were returned as "undeliverable." The replies came from all the major leprosy–endemic areas.

Files of Leprosy Registry at AFIP. The Leprosy Registry contains approximately 13,000 accessioned cases, with current annual additions of 1500–2000 accessions. A search of these files revealed material from only two patients one year of age or under. Both of these patients were residents of Burundi, and were detected by contact survey by Sr. M. Frades, nurse-leprologist of the Damien Foundation, directed by one of the authors (JB).

Patient #1. This six-month-old female was first seen on 3 October 1979. She had many hypopigmented macules with well-defined borders, but was otherwise healthy. The mother of this infant had been treated sporadically with sulfone for tuberculoid (TT) leprosy since 1970, and was clinically inactive in February 1979. A biopsy specimen was taken in October 1979 and evaluated at the AFIP. This specimen showed granulomatous inflammation with severely damaged nerves and rare acid-fast bacilli in the subepidermal area. We interpreted the histopathologic changes in this lesion as early active borderline tuberculoid (BT) leprosy.

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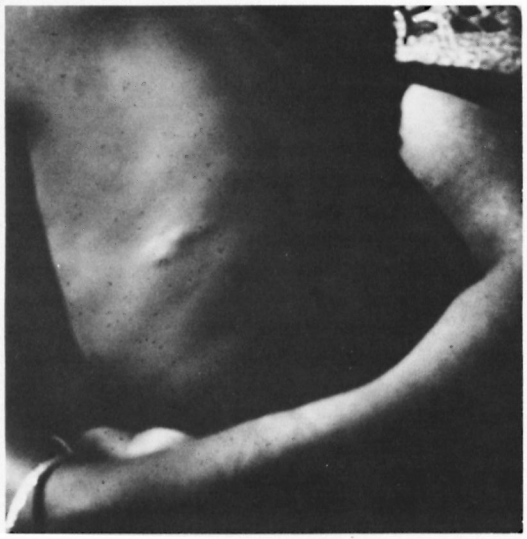

FIG. 1. Six-month-old male (patient #2) showing single infiltrated lesion with well-defined margins in left posterior axilla. Mother, who did not have leprosy, is holding the infant. (Photograph by Sr. Frades.)

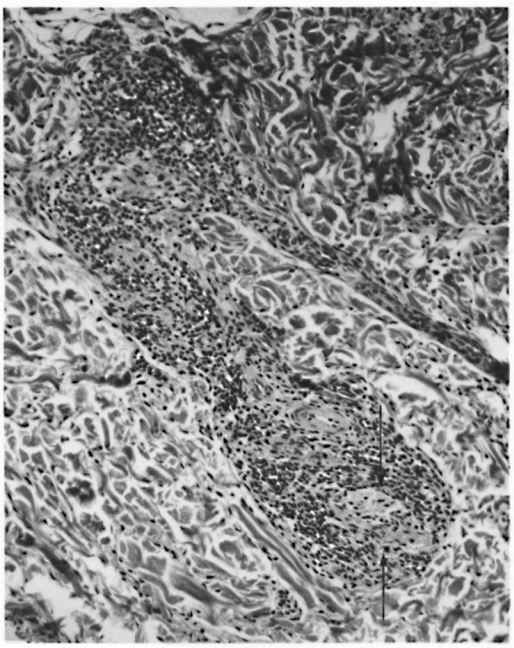

FIG. 2. Biopsy specimen from lesion shown in Figure 1. This specimen was taken eight months after the clinical photograph. Approximately 75% of the dermis was replaced by well-developed granulomas composed of epithelioid cells, occasional Langhans' giant cells, and large numbers of lymphocytes. Some of the granulomas contained fragmented dermal nerves (arrows). AFIP Neg. 84-11636 (H&E ×160).

Patient #2. This six-month-old male was first seen in July 1981 with a single, maculopapular, well-defined lesion in the left posterior axilla (Fig. 1). The mother did not have leprosy, but the father reportedly had inactive lepromatous leprosy at the time the lesion was first detected in the infant. Leprosy was suspected and early follow up was planned, but the patient did not return for examination until 1 March 1982. At that time, the patient still had only a single lesion but the size and infiltration were increased. A biopsy specimen taken at that time (14 months of age) was evaluated at the AFIP. This specimen revealed that 50% of the dermis was replaced by granulomatous inflammation. The granulomas invaded dermal nerves, and some nerves were severely damaged (Fig. 2). There were moderate numbers of acid-fast bacilli, many well stained, in nerves (Fig. 3). We interpreted the lesion as active borderline tuberculoid (BB-BT) leprosy.

RESULTS

Cases from the literature

Our review of the world literature was extensive, if not exhaustive. The earliest published description, of which we are aware, of a patient under one year of age was by Nakajo ([17]) who, in 1914, reported leprosy in a 2½-month-old Japanese female whose parents both had leprosy, probably borderline lepromatous (BL) or lepromatous (LL). The diagnosis of leprosy in the child was confirmed by biopsy. While precise classification cannot be discerned, we interpret this as borderline (BB) or lepromatous (LL). Acid-fast bacilli (AFB) were not described in the tissue sections, but tissue fluid contained many AFB. Nakajo speculated that the infant became infected from the mother's milk, by placental transfer of leprosy bacilli, or mosquito bites. The placenta and cord blood were searched but AFB were not seen. Currie (1915) ([2]) noted that there were 14 published claims of prenatal infection during the period 1890–1915, but did not give citations. We thus have not included Currie's data in our statistics. Rodriguez (1926) ([20]), in a review of 398 children of leprosy patients at Culion Leprosarium, Philippines, reported one patient and 10 suspected patients under one year of age. Three of the suspected patients later were bacteriologically positive, but specific data on these patients were not given. Mon-

testruc (1953) ([15]) studied advanced multibacillary leprosy in a three-month-old male infant of parents who did not have leprosy. The lesion appeared at two months of age. An aunt had untreated multibacillary leprosy, and was the presumed contact. Thus, the incubation period was estimated by Montestruc to be as short as two months. Dreisbach (1954) ([4]) reported macular leprosy in a seven-month-old Nigerian child. The onset of leprosy was first noted at five months of age. Information obtained by a personal (MB) conversation with Dr. Dreisbach in 1954 suggests that this child had paucibacillary (probably BT) leprosy. The mother had no evidence of leprosy but the father, who regularly cared for the infant, had far-advanced, untreated LL leprosy with marked oropharyngeal involvement. Ramu (1959) ([19]) described bacillary-positive LL leprosy in a six-month-old Indian male. Rollier (1962) ([21]) detected leprosy in two Moroccan nursing infants each one year of age. Descriptions of their histopathologic changes suggest that one had TT and the other BB leprosy. Both mothers had untreated LL leprosy. Zawahry (1963) ([24]) illustrated a one-year-old child with leprosy (sex of child is unknown). The lesions were macular, but classification is not possible. The mother had macular leprosy but, again, classification is impossible. Girdhar, *et al.* (1983) ([9]) studied a 9- to 10-month-old Indian female with BL leprosy. The earliest lesion was a small erythematous papule that initially was diagnosed as a mosquito bite. The mother had active BL leprosy with a bacillary-positive nose blow. In 1982, Duncan ([6]) described clinical leprosy in two infants seven and nine months old. Both mothers had bacillary-positive BL leprosy. Both infants had histologic evidence of leprosy; one indeterminate (I) and one TT, at 12 and 17 months of age, respectively.

In summary, there are published reports of at least 11 infants, 12 months old or younger, with proven leprosy. The mother of all but three had active leprosy. The sex of five of the infants was recorded, and four were males.

Cases from correspondence

The reported patients were divided into three categories according to the completeness of the information available: Category I = Clinical and histopathologic findings diagnostic of leprosy. Category II = Clinical findings diagnostic of leprosy, reported by workers knowledgeable and experienced in the detection of leprosy. A biopsy specimen was either not obtained or not reported. Category III = Less definite evidence than established for Category II, or information was derived from third party sources.

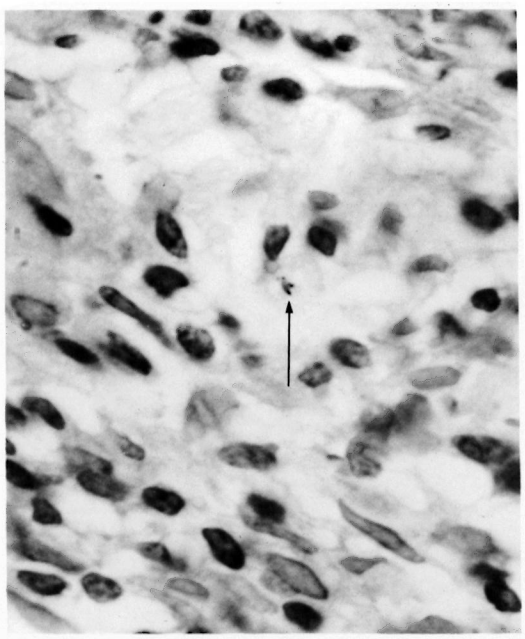

FIG. 3. A small cluster of acid-fast bacilli (arrow) in a fragment of nerve in a granuloma shown in Figure 2. AFIP Neg. 84-11642 (Fite-Faraco ×150).

A total of 40 correspondents contributed information on infants under one year of age: Category I, 8 contributors; Category II, 23 contributors, and Category III, 9 contributors.

Category I patients. Of the 11 patients in this category, there were 5 males, 1 female, and the sex was not given in 5. Ages ranged from 2½ months to 12 months. Five patients had TT, 3 BT, and 3 I leprosy. Possible contacts with leprosy were: mother = 4 (all LL), mother and father = 1 (both LL), grandfather = 1, and unknown = 5.

Category II patients. Among these 27 patients, there were: 1 male, 4 females, and the sex was unknown in the remaining 22 patients. Ages ranged from 3 months to 12 months. Classification of leprosy was distributed as follows: 4 LL, 1 BL, 7 BT, 5 TT, 4 I, and 6 unknown. Possible contacts with leprosy were: mother and father = 4 (1 BB

and 3 unknown); mother = 12 (7 LL, 1 BL, 1 BT, 1 TT, and 2 unknown); father = 3 (2 LL and 1 BB); grandfather = 2 (1 LL and 1 BT); aunt = 1 (LL); and unknown = 4.

Category III. Data are much less complete for the 40 patients in this category than for those in categories I and II. The data came from nine contributors. The age of the infants ranged from 2 months to 12 months, and sex was not given for any patient. Classification of leprosy was given for 8 patients (1 I, 1 BT, and 6 TT). The mothers of 9 infants had leprosy (4 LL, 3 BB, 1 TT, and 1 unknown), but the contacts for all of the other 31 patients were not given.

SUMMARY OF RESULTS

Category I. The combined data on all patients from the Leprosy Registry, literature, and correspondence are as follows: total patients = 19 (7 males, 4 females, 8 unknown); classification of leprosy = 4 I, 2 BL, 6 BT, 7 TT; age range was 2½ months to 12 months; possible leprosy contact = 10 mother, 1 father, 2 mother and father, 1 grandfather, 5 unknown. When the mother was the only contact named, 7 were LL (3 known to be untreated at parturition), 2 were bacillary-positive BL, and 1 had active TT. The one father had untreated advanced LL. In the two instances when both parents were named, both had lepromatous leprosy, of which at least one pair was untreated. The type of leprosy in the one grandfather contact was not indicated.

Category II. There were 32 patients in the combined literature and correspondence surveys. Sex distribution was 4 males, 4 females, and 24 unknown. Age range was 3 months to 12 months. Class distribution of leprosy was: 4 I, 6 LL, 1 BL, 9 BT, 5 TT, and 7 unknown. The mother was named as the possible contact in 13 instances: of these, there were 7 LL, 1 BL, 1 BT, 1 TT, and 3 unknown. Of the 4 fathers who were contacts, 3 were LL and 1 was BB. In six instances both parents had leprosy; classification was indicated in only one pair and both were BB. For 3 infants (2 LL and 1 BT), a grandfather was named as the contact, and for 2 infants (both LL), an aunt. Four infants had no known contact with leprosy patients.

The combined data for the total of 51 infants in categories I and II are presented in Tables 1–3. The sex of 19 patients was documented, and the ratio of males/females was 1.375 to 1. Class of leprosy was known, or estimated, for 44 patients; 36 had established forms of leprosy, while only 8 had indeterminate disease. Forty of the infants had a relative with active or inactive leprosy, and the mothers of 29 of the 51 infants may have been the source of the infection.

DISCUSSION

Precise modes of natural transmission of leprosy have not been established, but skin-to-skin contact ([12]) and nasorespiratory droplet infection ([3]) are believed to be the most common routes. Placental transmission of leprosy has long been a subject of conjecture. Recent evidence suggests, and may prove, that there is placental transmission of leprosy to the fetus, and that the development of babies of mothers with leprosy are adversely affected ([5, 8]).

The best evidence that *Mycobacterium leprae,* or antigens thereof, cross the placental barrier is the detection of *M. leprae*-specific IgA and IgM antibodies in cord blood of 30–50% of babies from lepromatous mothers ([13]). *M. leprae*-specific IgA and IgM rise in some infants of lepromatous mothers during a three-month to 24-month period following birth ([14]); two such infants developed clinical leprosy at 9 months and 17 months ([7]). There are several reports of the demonstration of intact *M. leprae* in placentas and in cord blood ([11, 23]).

There is epidemiologic evidence to suggest that leprosy may be transmissible from mothers to offspring via the placenta, but this route must be less frequent than direct contact. Rodriguez ([20]), for example, cites several studies in which immediate segregation of infants from mothers with leprosy reduced the incidence of leprosy in the infants. In his study of infants at Culion from 1915 through 1924, Rodriguez observed that segregation of infants up to only six months of age from a mother with leprosy decreased the ultimate incidence of leprosy in the infants. If transplacental infection were a common phenomenon, or were the predominant route of infection, then it would be expected that the incidence would continue to rise beyond the six-month limit. In

addition to transplacental transmission, infants may become exposed to viable *M. leprae* in the milk of untreated mothers ([18]). Our data show that 29 of 51 (57%) mothers of children who developed leprosy under one year of age had leprosy; of these only 14 (28%) had multibacillary disease, and only 5 (10%) were known to be untreated at parturition. We have no information to suggest that the mothers of any of the 22 infants whose mothers did not have leprosy had subclinical disease at parturition. Eleven (22%) of the infants probably acquired leprosy by non-maternal intrafamilial (father, grandfather, aunt) contact and 11 (22%) by unknown contacts. Thus, a minimum of 43% of the infants acquired leprosy after birth; however, all of the infants could have acquired the disease after birth. Conversely, this suggests that between 0% and 57% of the infants could have been infected before birth. Five of the mothers had active leprosy at parturition, suggesting that at least 10% of the infants were at risk of infection *in utero.*

The youngest infant without a familial contact with leprosy was 2½ months old, and a total of six infants 5 months old or younger are believed to have acquired leprosy by extraparental contact. Muir ([16]) saw a "child of six weeks with lesions scattered over the body," but did not give any details of the history of the disease. Most authorities assume that the average incubation period for leprosy is 2½ years to 3½ years, some even longer. This assumed prolonged incubation period of leprosy is frequently attributed by some to the presumed slow generation time of the leprosy bacillus, approximately 13 days as determined by the normal mouse foot pad infection assay. *M. leprae* may, however, multiply much more rapidly in a more suitable environment. For example, Hastings and Morales ([10]) estimate that the generation time for *M. leprae,* if all the bacilli were viable, could be as short as 26 hours. Hence, in highly susceptible individuals it is reasonable to assume that small inocula could multiply rapidly enough to produce a high bacterial burden and clinical disease within brief periods.

TABLE 1. *Combined (categories I and II) age and sex distribution of infants.*

Age (mo.)	Male	Female	Unknown	Total
2–5	3	2	4	9
6–12	8	5	28	41
Unknown (≤1 yr)	0	1	0	1
Totals	11	8	32	51

Because newborn infants possess *M. leprae*-specific IgM antibodies, intrauterine antigen-induced tolerance is a distinct possibility in some infants. It is known that cell-mediated immune tolerance can be induced in neonatal mice ([1]), and newborn mice may be rendered tolerant to delayed-type hypersensitivity responses to antigens of the leprosy bacillus by neonatal injection of *M. leprae* ([22]). This suggests that early intrauterine exposure to antigens of *M. leprae* could suppress the cell-mediated immune response to infection by *M. leprae* in humans, and could promote the development of leprosy in early infancy.

SUMMARY

Information obtained from a review of the literature, the United States Armed Forces Institute of Pathology files, and from a correspondence survey revealed a total of 91 infants one year of age and under in whom leprosy was diagnosed. Biopsy confirmation was available on 19 infants, and in an additional 32 patients the diagnosis of leprosy

TABLE 2. *Combined (categories I and II) age and class of leprosy distribution.*

Age (mo.)	Class of leprosy						Total
	I	LL	BL	BT	TT	Unknown	
2–5	2	2	1	1	2	1	9
6–12	6	4	2	13	10	6	41
Unknown (≤1 yr)	0	0	0	1	0	0	1
Totals	8	6	3	15	12	7	51

TABLE 3. *Combined (categories I and II) distribution by age and possible source of infection.*

Age (mo.)	Possible source of infection					Total
	Mother	Father	Both parents	Other relatives[a]	Unknown	
2–5	1	0	1	3	4	9
6–12	19	6	7	2	7	41
Unknown (≤1 yr)	1	0	0	0	0	1
Totals	21	6	8	5	11	51

[a] Grandfather = 3; aunt = 2.

was considered certain even though biopsy confirmation was not obtained. Although the mother was probably the most common source of the infection (29 infants), it was of interest to note that the father, another relative, or an unknown contact was the source of the infection in at least 43% of the infants. The youngest infant was 2–3 months old and had no known familial contact. The role of intrauterine exposure to *Mycobacterium leprae,* or to antigens of *M. leprae,* in infection and pathogenesis is discussed. The diagnosis of leprosy in infants under one year may frequently be missed or early signs disregarded because of a mistaken belief that leprosy is exceedingly rare or nonexistent in the very young.

RESUMEN

El análisis de la información obtenida de la literatura, de los expedientes del Instituto de Patología de las Fuerzas Armadas Americanas, y de una encuesta por correspondencia reveló que en 91 infantes de un año de edad y menores, se había hecho el diagnóstico de lepra. En 19 casos se pudo hacer una biopsia confirmatoria y en 32 pacientes adicionales el diagnóstico de lepra se consideró cierto aún cuando no se obtuvo la confirmación por biopsia. Aunque la madre fue la fuente más común de la infección (29 infantes), fue interesante notar que el padre, otro familiar, o un contacto no relacionado, constituyeron la fuente de infección en cuando menos 43% de los infantes. El infante más jóven tuvo 2–3 meses de edad y no tenía un contacto familiar conocido. Se discute el papel de la exposición intrauterina al *Mycobacterium leprae* o a los antígenos del mismo en la infección y en la patogénesis de la enfermedad. Debido a la idea errónea de que la lepra es extremadamente rara o inexistente en los muy jóvenes, muchas veces el diagnóstico de la misma no se intenta en los infantes menores de un año y con frecuencia los síntomas son ignorados o pasan desapercibidos.

RÉSUMÉ

On a procédé à une revue de la littérature, au dépouillement des archives de l'United States Armed Forces Institute of Pathology, et à une enquête par correspondance, pour rechercher le nombre d'enfants, âgés d'un an au maximum, chez lesquels une lèpre a été diagnostiquée. Un total de 91 enfants a été trouvé. Chez 19 enfants, le diagnostic était confirmé par biopsie; chez 32 autres enfants, le diagnostic de lèpre a été considéré comme certain, malgré l'absence de confirmation par la biopsie. La mère était probablement la source la plus commune d'infection (29 enfants); il est cependant intéressant de relever que le père, un autre parent ou même un contact inconnu a constitué la source d'infection chez au moins 43% de ces enfants. L'enfant le plus jeune était âgé de 2 à 3 mois; il n'avait aucun contact familial connu. On discute le rôle d'une exposition intra-utérine à *Mycobacterium leprae,* ou à des antigènes de *M. leprae,* dans l'infection et la pathogénèse. Le diagnostic de la lèpre chez les enfants de moins de un an peut fréquemment être méconnu; on peut négliger des signes précoces croyant à tort que la lèpre est extrêmement rare ou n'existe pas chez les enfants très jeunes.

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have spent many hours or days searching their records and preparing detailed abstracts on many of the patients included in this report. We apologize to those individuals who so generously supplied data which, after careful consideration, could not be used in this study because of criteria that we had established. Some of the information so gathered is a continuing subject of stimulating correspondence.

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The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the U.S. Department of the Army or the U.S. Department of Defense.

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