INTERNATIONAL JOURNAL OF LEPROSY

Volume 51, Number 1
Printed in the U.S.A.

A Clinical and Immunological Study of Four Babies of Mothers with Lepromatous Leprosy, Two of Whom Developed Leprosy in Infancy[1]

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Leprosy is uncommon in children four years of age or younger, even when there is household contact with an active, infectious case. This is generally considered to indicate that the incubation period is about four years ([25]) and that placental transmission of *Mycobacterium leprae* is an exceedingly unusual event ([11, 29]). The occasional diagnosis of leprosy in children aged three years or less might indicate either very heavy exposure or infection *in utero*.

Hitherto it has been almost impossible to evaluate the possibility of placental transmission of *M. leprae*. However, it has recently been shown that, even in tuberculoid leprosy (in which the body load of *M. leprae* is small), increased levels of antimycobacterial antibody are present in patients undergoing relapse after stopping treatment ([30]). It has therefore been postulated that antibody levels could be used as diagnostic tests for leprosy before there are clinical manifestations of active disease, or when the diagnosis is uncertain ([18]).

In the present study, a series of 113 mothers with different types of leprosy were studied through pregnancy and delivery. Follow up, including regular clinical and immunological assessments of both mothers and children, continued for one to two years after delivery, and a further assessment was conducted when the children were about four years old. This paper reports the results of clinical and immunological studies in four children born to mothers with active lepromatous leprosy, two of whom almost certainly developed leprosy during the period of observation.

PATIENTS AND METHODS

One hundred thirteen women with leprosy and 27 healthy controls were studied throughout pregnancy, at delivery, and followed up with their babies during lactation. The classification of leprosy according to the scale of Ridley and Jopling ([23]) was: TT and BT = 36; BL = 42; LL = 35. Selection of women for the study was based chiefly on their willingness to participate in it.

Assessment of mothers. This included (at the start of the study) full clinical examination, examination of slit skin smears and "nose blows" for *M. leprae*, skin biopsy of an active-looking lesion, and tests of sensory and motor function. Immediately after delivery placental tissue, membranes, and the umbilical cord were collected for histological, bacteriological, and immunological studies. Samples of cord blood, maternal blood, and colostrum were obtained. During lactation, milk and blood samples were obtained, and leprosy assessments regularly repeated.

Assessment of babies (Phase I). This was carried out at birth, monthly for three months, and at approximately three-month intervals thereafter. In addition to routine examinations and measurements, blood samples were obtained (usually by scalp vein

[1] Received for publication on 14 June 1982; accepted for publication in revised form on 15 September 1982.

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puncture) every three to six months for one to two years. Special attention was paid to the skin condition. Examinations were carried out in a well lit, south facing room, using daylight when possible, with the baby completely undressed. When a baby was found to have skin lesions suspected of being leprosy, they were measured, and sensory testing attempted using a pin held at an angle of 45° to the skin and applied with sufficient pressure to indent the skin. Palpation of peripheral nerves was also carried out, and the sizes of the peripheral nerves were recorded. Three babies had punch biopsies of suspicious lesions under local anesthesia. Independent assessments were carried out by two senior leprologists.

Skin testing and vaccination/immunization. Vaccination/immunization was performed at local well baby clinics, and included BCG, smallpox, triple vaccine (DPT), and oral poliomyelitis, singly or in combination, according to availability. When the babies were nine to fifteen months old they were skin tested with A6 (purified protein of *M. leprae* grown in armadillos) and PPD; babies who were negative to PPD were given BCG vaccination and retested with PPD two months later.

Later follow up of the babies (Phase II). Skin tests with AB22 (purified protein of *M. leprae* grown in armadillos) and PPD were repeated about two years later, when the children were three to four years old, together with a full examination and slit skin smears.

Informed consent for investigation was given by all the mothers participating in the study.

Immunological methods

Quantitation of immunoglobulins (IgA, IgM, and IgG). This was carried out by single radial diffusion in gel. Due to the low concentration of IgA in cord sera, the method was modified by reducing the concentration of anti-IgA in the gel to obtain sufficient sensitivity. IgA could be quantitated in concentrations above 8×10^{-3} g/l, and could be detected in sera at concentrations between 4 and 8×10^{-3} g/l. The average concentration of IgA in normal cord sera is between 4 and 8×10^{-3} g/l, and concentrations above 8×10^{-3} g/l can indicate intrauterine infection ([14]).

Antibodies against *M. leprae* antigen 7. These were determined by radioimmunoassay (RIA) using ^{125}I-labelled *M. leprae* antigen 7 as described previously ([12]). The ^{125}I-labelled *M. leprae* antigen 7 was prepared from bacilli purified from the liver of *M. leprae*-infected armadillos by Draper's method ([3]). One hundred μl of sera diluted 1:100 and 1:1000 were mixed with 100 μl of labelled *M. leprae* antigen 7. This mixture was left for 30 min at 20°C and thereafter 1.5 ml of 1% Staphylococci (strain Cowan 1) was added. The bacterial pellet with the antibody-bound labelled *M. leprae* antigen 7 was obtained by centrifugation and counted. The result was expressed as a percentage of the maximum binding using antiserum against *M. leprae* produced in the rabbit.

IgA, IgM, and IgG anti-*M. leprae* antibody activity. These were demonstrated and quantitated by solid phase radioimmunoassay (sRIA) as previously described ([16, 17]). In short, Nunc polystyrol test tubes were coated with sonified *M. leprae*. One hundred μl of a lepromatous serum pool (LSP) diluted 10^{-1}, 10^{-2}, 10^{-3}, and 10^{-4}, or 100 μl of lepromatous serum diluted 10^{-1}, 10^{-2} and 10^{-3}, were added. The test tubes were left for 24 hr, sucked empty, washed, and 100 μl of ^{125}I-labelled and purified antihuman IgA, IgM or IgG was added. The test tubes were again left for 24 hr, sucked empty, washed, and counted in a gamma counter. The result was expressed as a percentage of the IgA, IgM, and IgG anti-*M. leprae* antibody activity in the LSP as previously described ([16]).

A standard curve was made for each set of experiments from the counts obtained from the 1×10^{-1}, 1×10^{-2}, 1×10^{-3}, and 1×10^{-4} dilutions of the LSP and the result was expressed as a percentage of the LSP calculated by the following method: One percent of LSP meant the same number of counts in a 1×10^{-3} dilution of the LSP as in a 1×10^{-1} dilution of the patient's serum; 10% of LSP meant the same number of counts in a 1×10^{-3} dilution of the LSP as in a 1×10^{-2} dilution of the patient's serum; and 100% of LSP meant the same number of counts in a 1×10^{-3} dilution of the LSP as in a 1×10^{-3} dilution of the patient's serum.

RESULTS

Assessment of infectivity of mothers

Clinical, skin smear, and biopsy results. Of the 36 mothers with BT or TT leprosy, 11 had active disease. However, in only two cases were solid-staining bacilli seen in biopsies or skin smears, and it seems very unlikely that these mothers could infect their babies.

Of the 76 mothers with lepromatous (LL or BL) leprosy, 40 showed some clinical deterioration during pregnancy or soon after delivery. In 28 cases the deterioration amounted to frank relapse probably due to the emergence of sulfone-resistant leprosy. Solid-staining bacilli were found in skin smears or biopsies in 38 cases; these patients must be considered as potentially highly infectious to their unborn children by hematogenous spread via the placenta.

Results of nose blows. Nasal mucus (nose blow) was examined in 43 patients with lepromatous leprosy, but only one was found to be positive; no solid-staining bacilli were seen. The risk of droplet transmission of leprosy after delivery seems to be slight in this group of patients.

***M. leprae* in milk.** Milk from nine mothers with highly bacilliferous leprosy was examined for acid-fast bacilli by concentration methods; all nine specimens were negative. It seems unlikely that the milk was, in this group of patients, a major source of *M. leprae* or of risk to the babies.

The placenta as a site for *M. leprae*. Routine examination of placental sections failed to demonstrate acid-fast bacilli. Even using concentration methods to search for acid-fast bacilli in placental tissue from ten of the most bacilliferous patients, the results were negative in half the cases; the other half showed very scanty acid-fast bacilli or debris. Details of placental histology will be reported elsewhere ([5]).

Clinical assessment of babies with suspected leprosy

Four babies between nine and 17 months of age developed hypopigmented lesions which were suspected to be due to leprosy. In all cases the mothers had active lepromatous leprosy. The clinical data are summarized in Table 1. Babies 2 and 3 showed strong evidence of leprosy, but Baby 1 did not have leprosy, and it was doubtful that Baby 4 had leprosy. The evidence for the diagnosis of leprosy in Babies 2 and 3 relies mainly on the histological examination (although both had sensory loss on skin testing), and this was carried out by Dr. Dennis Ridley. In neither skin biopsy were acid-fast bacilli seen, but both showed dermal nerve pathology which is almost pathognomonic of leprosy. Baby 2 showed infiltration of a neurovascular bundle in the dermis with lymphocytes and mononuclear cells, and there was swelling and proliferation of Schwann cell nuclei. Baby 3 also showed dermal nerve pathology, in that there was Schwann cell proliferation in a nerve bundle in the subcutis, but the changes were less marked than in Baby 2.

None of the four infants received antileprosy treatment (other than via the breast milk prior to weaning); but all the lesions had almost completely disappeared when the children were finally assessed two to three years after the lesions appeared. At about this time a new macule appeared in Baby 4, but it was shown to be not due to leprosy. A new lesion in Baby 3 appeared at 27 months, but the biopsy was not diagnostic of leprosy and the lesion had resolved at the last assessment.

Skin testing with *M. leprae* antigen was performed at the end of the study; Babies 1 and 4 were negative; 2 and 3, positive (all four infants were negative when first tested at nine to 15 months).

Immunological assessment of babies with suspected leprosy

Immunoglobulins. Immunoglobulin levels for cord sera of Babies 2, 3, and 4 are shown in Table 2 (cord serum from Baby 1 was not available). The median figures shown in Table 2 are medians found in cord blood from the babies of all mothers with active lepromatous leprosy. The levels of IgA tended to be slightly lower than the median figures; while IgM was in the median range. The IgG levels of these three babies, however, were higher than the median, although the IgG levels of their mothers' sera (taken at delivery) were lower than average for mothers with active lepromatous leprosy.

Antibodies against *M. leprae* antigen 7. The concentrations of antibodies against *M. leprae* antigen 7 declined in all three babies

TABLE 1. *Clinical data on four babies suspected of developing leprosy and their mothers.*

Case no.	Mother	Baby age (months)	Details of lesions (Baby)						Follow up	Skin test with *M. leprae* antigen at 3 years of age
			No.	Size (cm)	Site	Margin	Sensory loss	Biopsy		
1	LL[a] (BI = 3.5)[b] (MI = 2.8)[c]	9	2	1 × 1 1 × 1	L leg L leg	Well defined	No	Not leprosy	Clinical diagnosis "ringworm." Treated with anti-fungal ointment and resolved within 2 weeks.	Negative
2	BL[d] (BI = 1.1) (MI = 0)	17	2	0.5 × 1 1.8 × 3	Neck R leg	Well defined	Yes	Tuberculoid leprosy	Gradual fading of lesions. No sign of leprosy at examination aged 4 and 4½ years.	Positive
3	BL[e] (BI = 4.2) (MI = 4.0)	12	3	1 × 4 1 × 4 1 × 4	Back Back Back	Poorly defined	Yes	Consistent with indeterminate leprosy	Lesions began to fade after 3 months. New macule 1.4 × 1.5 on buttock seen at 23 months, biopsy "not diagnostic of leprosy." All lesions almost disappeared at 3–4 years.	Positive
4	BL[a] (BI = 3.0) (MI = 4.5)	14	1	1.5 × 1	Chest	Poorly defined	No	Not done	Lesion faded at 17 months, no abnormality seen at 39 months. New lesion 1 × 1 seen on thigh at 45 months, biopsy "nothing at all to indicate leprosy."	Negative

[a] Suspected dapsone resistance during pregnancy.
[b] BI = Bacteriologic Index.
[c] MI = Morphological Index.
[d] "Cured BT" relapsed "active BL" during pregnancy.
[e] Proven dapsone resistance during pregnancy.

TABLE 2. *Immunoglobulin levels of the cord sera of three babies and median figures of babies of all mothers with active lepromatous leprosy.*

	IgA[a]	IgM[a]	IgG[b]
A9/70 (Case No. 2)	4–8	32	10.0
A9/89 (Case No. 3)	4–8	60	9.8
A9/244 (Case No. 4)	4–8	110	12.5
Median LL-BL, BI+	9.5	54	9.5

[a] Values given as $\times 10^{-3}$ g/l.
[b] Values given as g/l.

from birth to six to eight months of age (Fig. 1) as expected from the 28 day half-life of maternal IgG in newborn babies ([15]). No rise was seen at the time the babies developed skin lesions suspicious of leprosy.

IgA, IgM, and IgG anti-*M. leprae* antibody activity. The median IgA, IgM, and IgG anti-*M. leprae* antibody activity of 26 babies of mothers with lepromatous leprosy in serial samples of serum from birth to 20 months of age are shown in Figure 2. The antibody activity is expressed as a percentage of the activity in a lepromatous serum pool (LSP). IgG antibody activity declined markedly from 25% in cord sera to 0.25% of the LSP in sera taken 12 months after birth, and thereafter showed a slight insignificant increase. IgM anti-*M. leprae* antibodies could be detected in 55% of the cord sera and the activity rose during this period to 10%. IgA antibodies could be detected in 30% of cord sera and the activity also rose steadily to 2.5% of the LSP in sera taken at about 18 months of age.

The IgA, IgM, and IgG anti-*M. leprae* antibody activities of serial serum samples from the four babies with suspected leprosy are shown in Figures 3, 4, 5 and 6. Baby 1 shows a "normal" pattern (Fig. 3) with lower IgA, IgM, and IgG anti-*M. leprae* than illustrated in Figure 2. Babies 2 and 3 show

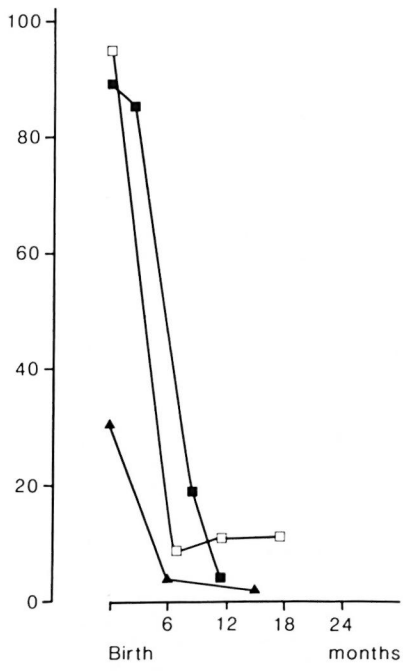

FIG. 1. The activity of antibodies against *M. leprae* antigen 7 in cord serum and repeated serum samples taken from ▲ Case 2, □ Case 3 and ■ Case 4. The activity is expressed as percentage of the maximum binding using a polyvalent rabbit anti-*M. leprae* antibody preparation.

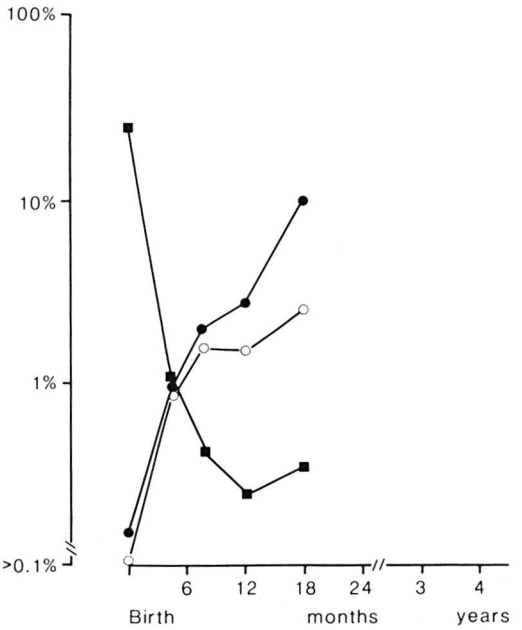

FIG. 2. Median IgA (○), IgM (●), and IgG (■) anti-*M. leprae* antibody activity in sera from 29 babies of mothers with lepromatous leprosy. The median results were calculated from all the individual results after the sera had been allocated to five groups; cord sera and sera taken around 3, 6, 12 and 18 months after birth. The results are expressed as a percentage of IgA, IgM, or IgG anti-*M. leprae* antibody activity in a lepromatous serum pool (LSP).

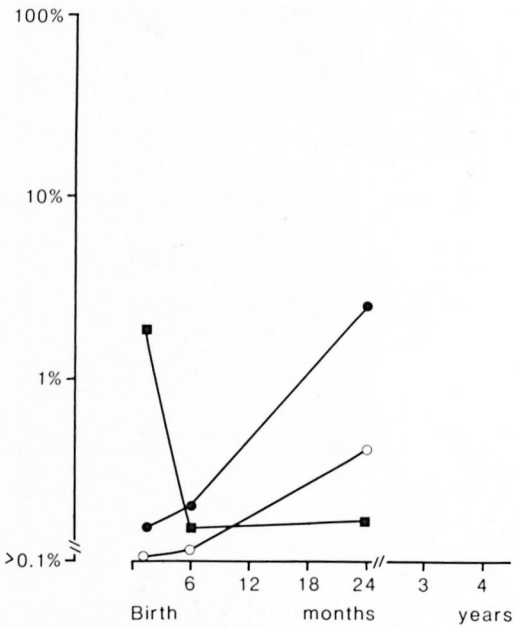

FIG. 3. IgA, IgM, and IgG anti-*M. leprae* antibody activity in serum taken one month after birth and repeated sera taken up to two years after birth from A9/36 (Case No. 1). The results are expressed as a percentage of the activity in LSP as in Figure 2.

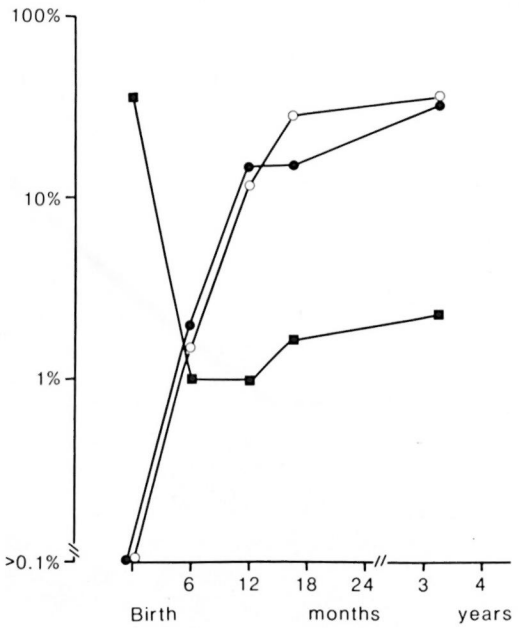

FIG. 5. IgA, IgM, and IgG anti-*M. leprae* antibody activity in cord serum and repeated sera taken up to 3¾ years after birth from Baby A9/89 (Case No. 3); otherwise as for Figure 3.

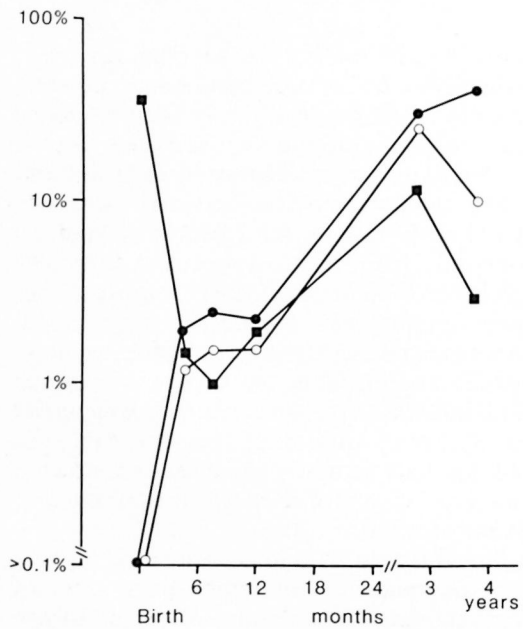

FIG. 4. IgA, IgM, and IgG anti-*M. leprae* antibody activity in cord serum and repeated serum samples taken up to four years after birth from Baby A9/70 (Case No. 2); otherwise as for Figure 3.

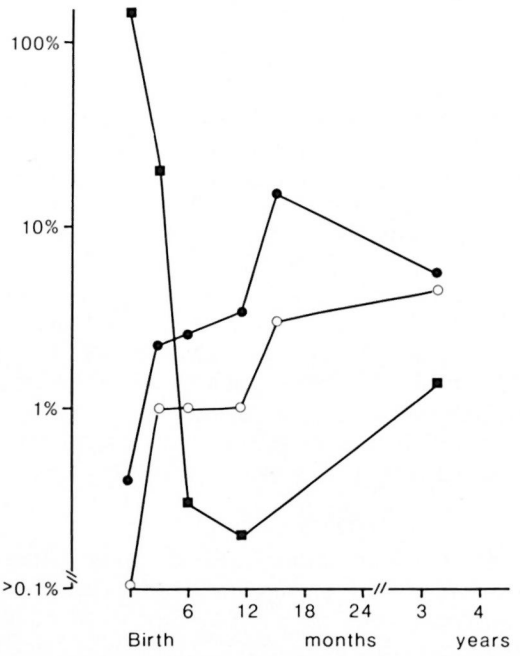

FIG. 6. IgA, IgM, and IgG anti-*M. leprae* antibody activity in cord serum and repeated sera taken 3¼ years after birth from Baby A9/244 (Case No. 4); otherwise as in Figure 2.

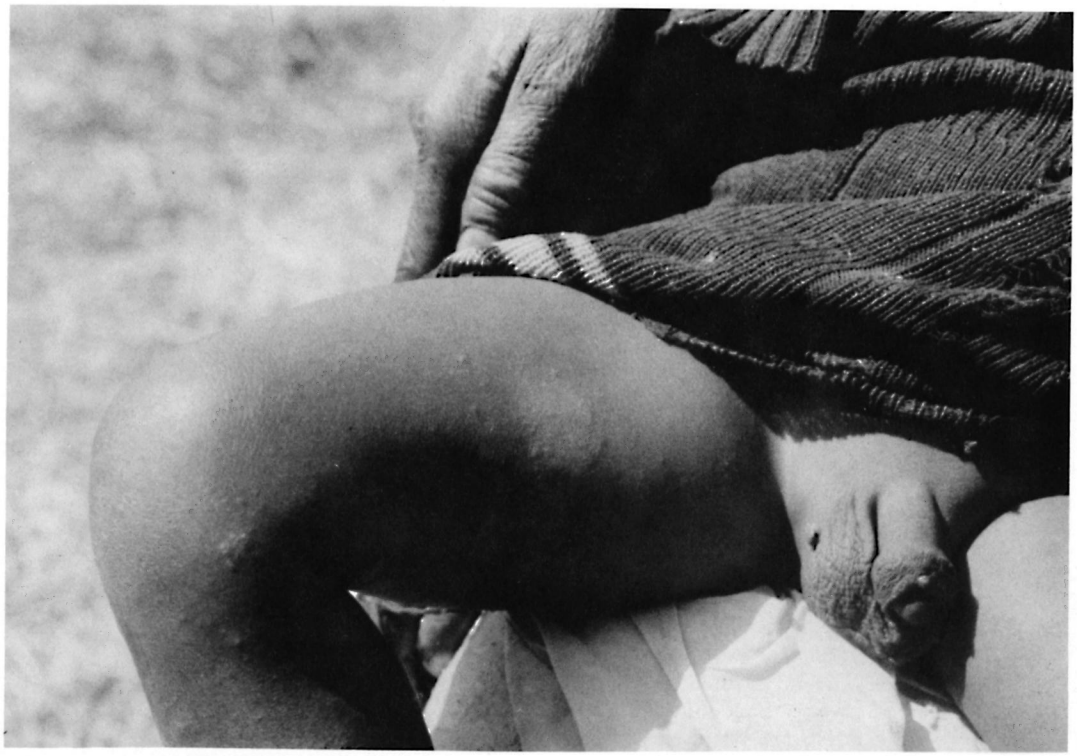

FIG. 7. Case No. 2, Baby A9/70. A hypopigmented, slightly raised, macule with a clearly defined margin, measuring 1.8 × 3.0 cm is shown on the inner aspect of the thigh. Lesions of a generalized scabies infection are also visible.

a different pattern. Both Babies 2 and 3 showed a marked rise in IgA and IgM anti-*M. leprae* antibody activity. In Baby 3 this rise was early and in Baby 2 it was somewhat slower; the IgM activity reaching 30% of LSP activity (both babies) and the IgA activity reaching 35% and 25% of LSP activity, respectively, in sera taken three years after birth. Baby 4 with unproven leprosy showed a pattern of IgA, IgM, and IgG anti-*M. leprae* activity between the pattern seen in Babies 2 and 3 and that made by the sera from all babies of mothers with lepromatous leprosy as illustrated in Figure 2.

DISCUSSION

Of the four babies studied, Baby 1 had no evidence of leprosy and almost certainly had a ringworm infection. However, in contrast, the clinical evidence that Babies 2 and 3 were suffering from leprosy when examined at the ages of 17 months and 12 months, respectively, is very strong. The lesions looked like leprosy (Fig. 7), showed some sensory impairment, and histologically were considered to be tuberculoid and indeterminate leprosy, respectively. The evidence that Baby 4 had leprosy is inconclusive.

The diagnosis of leprosy in Babies 2 and 3 was reinforced by the fact that they both developed positive skin tests to *M. leprae.* They also had a marked increase in IgA and IgM anti-*M. leprae* antibody activity on sequential studies of their sera during the first three years of life. Baby 4 had a negative skin test to *M. leprae,* but had a rise in IgA and IgM anti-*M. leprae* antibody activity on sequential testing of the sera, evidence that the baby might well have been infected *in utero.* No such rise in antibodies was seen in Baby 1.

To find two babies with proven leprosy was unexpected, and gave a prevalence of leprosy in children under two years of age whose mothers had active lepromatous leprosy of 5% (2/38). This figure is comparable with that reported by earlier workers ([9, 24]). It is likely that many such cases are missed.

The lesions are transient, and unless they are carefully sought they can easily be overlooked, even if examinations are conducted in a well-lit room ([8]).

Possible routes by which these children were infected include skin-to-skin contact, via the milk, inhalation of droplets from mothers' nasal secretions, or transplacentally. Skin-to-skin contact seems unlikely ([21]). The clothing habits in Ethiopia make such contact unusual except during breast feeding, and in any case few patients with lepromatous leprosy have bacilli on the skin surface except near the nose ([22]).

Maternal milk may be a possible source of infection for babies. While high counts of *M. leprae* in breast milk have been recorded from a few patients with active, untreated lepromatous leprosy ([21, 27]), a larger study failed to corroborate this ([24]). Using a PEG precipitation technique, Saha, *et al.* ([26]) demonstrated *M. leprae* in nine out of 12 samples of milk from lepromatous mothers. However, the nine milk samples tested in our study (from the mothers most likely to be positive) were negative for acid-fast bacilli. Our negative results may reflect the use of a less sensitive technique ([26]) or, alternatively, may be because most of our patients with active lepromatous leprosy were relapsing with rather localized lesions. Heavy breast milk infection may well require advanced disease involving the nipple and milk ducts. It seems unlikely that the babies in our study were infected in this way.

Droplet infection is now considered to be the usual route of spread of *M. leprae*. Although "casual nose blows" from these mothers were negative, it is possible that they would have been positive if taken in the early morning. However, one would expect this "normal" transmission of infection to cause leprosy with a normal incubation period. For babies aged 12 months and 17 months to develop a disease whose incubation period is usually four years or so predicates very heavy exposure, an unusual route of infection, or both.

Placental transmission meets these conditions. Patients with active lepromatous leprosy have a bacteremia of up to 10^5 *M. leprae* per ml ([4]). The placenta is highly vascular, and even minor breaches of its integrity might lead to the passage of large numbers of *M. leprae* to the fetus. Increased concentration of IgA ([14]) and demonstration of IgA and IgM anti-*M. leprae* antibody activity in cord sera ([16]) from babies of mothers with active lepromatous leprosy have already been reported from this study, indicating transfer of *M. leprae* antigens or bacilli across the placenta.

The placentae of babies in the study were examined. *M. leprae* were not observed histologically, and were present only in small numbers in homogenized and concentrated tissue. Previous studies, however, have shown *M. leprae* in both placenta and cord blood of babies born to mothers with active lepromatous leprosy ([1, 7, 20, 28]). It may be noted that in rare cases congenital tuberculosis can affect the baby but leave no signs of placental infection ([6]).

The immunological tests of the cord sera, by the demonstration of IgM anti-*M. leprae* antibodies, suggested infection had occurred *in utero* in Baby 4. However, no evidence was found, either by increased IgA or the presence of IgA or IgM anti-*M. leprae* antibodies in cord sera, for congenital infection in the two babies who later developed clinical signs of leprosy.

The most interesting results came from the solid phase radioimmunoassays for IgA, IgM, and IgG anti-*M. leprae* antibodies. There was a marked increase of IgM anti-*M. leprae* antibody activity to about 30% of LSP in sera taken at three years of age from Babies 2 and 3. These values are above the highest IgM anti-*M. leprae* antibody activity found in sera from a group of healthy nonleprosy contacts of patients with active lepromatous leprosy. This IgM activity in the two babies was also about three times higher than the median activity found in sera from all the babies of mothers with lepromatous leprosy. The rise in IgA anti-*M. leprae* antibody activity was also significantly higher than normally seen in sera taken six months to 12 months after birth from babies of both lepromatous and tuberculoid leprosy mothers ([17]). Therefore the demonstration of increased IgM anti-*M. leprae* antibody activity in the sera from the two babies who developed leprosy might be a specific indication of early *M. leprae* infection.

The results of assays for antibodies against *M. leprae* antigen 7 failed to demonstrate

increased antibody formation in Babies 2 and 3 at the time their skin lesions appeared. This is due to the limited sensitivity of the method which detects IgG antibodies chiefly to one antigenic component of the bacilli ([13]). We feel this method is poorly suited for the demonstration of IgA and IgM antibodies against *M. leprae*.

The clinical appearance of Babies 2 and 3 was that of indeterminate leprosy, although the recognition of impaired sensation in such young children suggests it was close to tuberculoid. This form of leprosy is often self-healing ([2, 10]) and, indeed, in Babies 2 and 3 the lesions rapidly and spontaneously resolved, and skin tests to *M. leprae* antigen became positive. Nevertheless, it would be unwise to assume that these children are now cured. They probably have been infected by an unusual route and possibly exposed to an unusually heavy inoculum of *M. leprae*. Baby 2 showed a decrease of IgG anti-*M. leprae* antibody activity at 4 years compared to 2½ years. This coincides with the disappearance of the skin lesions and suggests healing of leprosy. We have observed a similar decrease of IgG anti-*M. leprae* antibody activity after dapsone (DDS) treatment of patients with lepromatous leprosy ([18, 19]). No such fall was seen in Baby 3. Furthermore, it would be unwise to assume that no more cases of leprosy will occur in this group of children. Continued observation and repeated tests for some years to come is likely to bring to light more information which may greatly increase our understanding of the pathogenesis of leprosy.

SUMMARY

One hundred thirteen women and 27 healthy controls were studied throughout pregnancy, at delivery, and followed up with their babies during lactation. Thirty-eight of the mothers with lepromatous leprosy were found to have solid-staining bacilli in skin smears or biopsies, and hence were considered potentially highly infectious to their unborn children by hematogenous spread via the placenta. Two babies of mothers within this group were diagnosed as having leprosy on clinical and histological grounds. A third baby could well have had leprosy, but the case was not proven. The fourth baby did not have leprosy and, although it did have ringworm, was thus deemed to be a reasonable control. The leprosy skin lesions were first observed at a special followup clinic when the children were between the ages of 9 and 17 months.

The demonstration of IgA and IgM anti-*M. leprae* antibodies in cord sera was taken as an indication of intrauterine immunologic stimulation, and hence transplacental transmission of *M. leprae*. The two babies with proven leprosy showed an early and significant increase in serum IgA and in particular serum IgM anti-*M. leprae* antibody activity. A third baby, suspected of having leprosy but in whom the diagnosis was not proven, showed a similar but less marked increase in serum IgA and IgM activity. The fourth baby showed no such rise in anti-*M. leprae* activity. A decrease in serum IgG anti-*M. leprae* antibody activity could be demonstrated in one of the babies with leprosy after healing of the leprosy lesions, but not in the second baby.

RESUMEN

Se estudiaron 113 mujeres con lepra y 27 mujeres sanas durante su embarazo, al tiempo del parto, y durante la lactancia. Treinta y ocho de las madres con lepra lepromatosa tuvieron bacilos sólidamente teñidos en sus biopsias o extendidos linfáticos de piel y se consideraron potencialmente muy infecciosas para sus hijos por nacer dada la dispersión hematógena transplacentaria del bacilo. Dos bebés de las madres dentro de este grupo se diagnosticaron como afectados de lepra por criterios clínicos e histológicos. Un tercer bebé pareció tener lepra pero ésto no se demostró de manera definitiva. Un cuarto bebé no tuvo lepra pero sí una tiña y por ésto se estudió como control. Las lesiones leprosas en la piel de los bebés se observaron por primera vez cuando estuvieron entre los 9 y los 17 meses de edad.

La demostración de anticuerpos anti-*Mycobacterium leprae* de las clases IgA e IgM en el suero de cordón umbilical se tomó como un indicio de estimulación inmunológica intrauterina y por tanto, de la transmisión transplacental del *M. leprae*. Los dos bebés con lepra comprobada mostraron un incremento temprano y significativo en sus niveles séricos de IgA y particularmente de IgM, con actividad anti-*M. leprae*. El tercer bebé con lepra no confirmada mostró un incremento similar aunque menos marcado en sus niveles de IgA e IgM anti-*M. leprae*. El bebé con tiña no tuvo anticuerpos contra el bacilo de la lepra. En uno de los bebés con lepra se pudo observar una disminución en la IgG sérica anti-*M. leprae* después de la curación de las lesiones lepróticas; ésto no ocurrió en el segundo bebé.

RÉSUMÉ

On a étudié 113 femmes et 27 témoins en bonne santé, pendant la grossesse, lors de l'accouchement, et, avec leur nouveau-né, durant l'allaitement. On a observé que 36 des mères atteintes de lèpre lépromateuse présentaient des bacilles uniformément colorés dans les frottis cutanés ou dans les biopsies; dès lors, on a considéré que ces mères étaient éventuellement fortement infectieuses pour leurs enfants, avant la naissance, par le biais d'une transmission hématogène à travers le placenta. Chez 2 nouveau-nés de mères appartenant à ce groupe, on a diagnostiqué la lèpre, sur la base de critères cliniques et histologiques. Un troisième nouveau-né aurait bien pu avoir la lèpre, mais le cas n'a pas été confirmé. Le quatrième bébé n'avait pas la lèpre et quoiqu'il présentât une teigne, on a estimé qu'il pouvait raisonnablement être considéré comme un témoin. Les lésions cutanées de lèpre ont été observées pour la première fois, au cours d'un suivi établi spécialement, lorsque les enfants avaient entre 9 et 17 mois.

La démonstration d'anticorps IgA et IgM anti-*Mycobacterium leprae* dans le sérum du cordon a été considéré comme le signe d'une simulation immunologique intra-utérine, et dès lors d'une transmission transplacentaire de *M. leprae*. Les deux nouveau-nés souffrant d'une lèpre confirmée présentaient une augmentation précoce et significative de l'activité des anticorps IgA du sérum, et encore plus des anticorps IgM anti-*M. leprae*. Un troisième bébé suspect de lèpre, mais chez lequel le diagnostic n'avait donc pas été confirmé, révélait une augmentation semblable mais moins marquée dans l'activité des IgA et des IgM du sérum. Le quatrième enfant ne présentait pas une telle élévation dans l'activité anti-*M. leprae*. Une diminution dans l'activité des anticorps sériques IgG anti-*M. leprae* a pu être mise en évidence chez un des bébés atteint de lèpre, après guérison des lésions lépreuses, mais non chez le deuxième enfant présentant les mêmes conditions.

Acknowledgments. We thank the staff and patients of the Addis Ababa Leprosy Hospital for their help and cooperation throughout the study. We are particularly grateful to: Ato Moges Merid and Ato Wondemagengehu Mekuria and the Sisters and Staff of Ward 3 for their help with the assessments of mothers and babies and in-patient care of sick babies; Drs. H. Wheate and J. Warndorff who acted as independent assessors of babies with leprosy in Phase I of the study; Dr. J. K. Nsibambi and Dr. T. Warndorff who helped with Phase II of the study; Drs. D. S. Ridley and J. Warndorff for their expert advice on the histology of the biopsies; Dr. R. J. W. Rees who examined aliquots of placental tissue, milk, and colostrum by concentration methods for the presence of *M. leprae* and also supplied the PPD, A6 and AB22 for skin tests; Glaxo Laboratories for supplying the BCG used; Helen Bergsvik who carried out the serological tests in Oslo; and to Ato Tshome Kassahun and Woiz. Amarech Gebre Mariam who made all the baby clothes for the study.

This work was supported by grants from Ander Jahre's Fund for the Promotion of Science and the Norwegian Council for Science and the Humanities. The serological work was also supported by the Immunology of Leprosy (IMMLEP) component of the UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases.

M. E. Duncan was supported for part of the study by a research grant from the British Leprosy Relief Association (LEPRA).

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