The Presence of *M. leprae* in Human Milk

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In the pathogenesis of leprosy inoculation or inoculation through the skin is still thought to be the chief method by which the bacillus enters the body. Evidence for entrance by the respiratory and alimentary portals is believed to be lacking. For example, Spickett referring to the paper of Weddell and Palmer on the pathogenesis of leprosy wrote:—

‘The evidence of Weddell and Palmer does appear to establish that *M. leprae* may be disseminated throughout the body by the circulation, and this being so one of the major problems connected with a respiratory or alimentary portal of entry is solved. However, this does not in any sense provide evidence that such portals of entry are used and it is difficult to see how the type of evidence that Weddell and Palmer have produced can provide such evidence.’

Again, Badger writes:—

‘Occasionally one sees reference to possible indirect contact transmission through food, clothing, insect vectors, etc. Concrete and definite information relative to such transmission is lacking.’ (Italics—mine.)

In view of these remarks, the following patient will be of interest.

In May this year a Nepali woman, aged 22 years, came into my consulting room carrying her 6 months old baby. She complained of swelling of the face and ‘pins and needles’ sensation in hands and feet for one year, developing when she was about 3 months pregnant. This was her first child. My clinical notes are as follows:—

**Skin**

The skin of the face is slightly erythematous and shows a diffuse, generalised infiltration. Both ear lobes are swollen. There is a broad based erythematous, shiny nodule about 1 cm across situated on the chin to the right of the mid-line. The skin of the chest, back and front, and of the neck shows multitudes of small, slightly hypopigmented coalescing macules symmetrically distributed.

**Nerves**

<table>
<thead>
<tr>
<th>Nerve</th>
<th>R</th>
<th>L</th>
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<tbody>
<tr>
<td>Auricular</td>
<td>+2</td>
<td>—</td>
</tr>
<tr>
<td>Ulnar</td>
<td>+1</td>
<td>—</td>
</tr>
<tr>
<td>Radial</td>
<td>+1</td>
<td>+1</td>
</tr>
<tr>
<td>Peroneal</td>
<td>+1</td>
<td>+2</td>
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**Anesthesia**

No obvious impairment of tactile and pain sensations in the extremities. Thermal sense not tested.

**Slit Skin Scrapings**

One from each ear lobe. Both showed massive infection with acid fast bacilli; that is, innumerable bacilli and numbers of globi in every field. At least 50% of the bacilli were judged to be in good well-staining solid rods.

**Diagnosis**

Lepromatous leprosy in very active stage.

**Therapy**

The patient was started on DDS 30 mgm. per week and given 3 months’ supply as she had come from a considerable distance.

Later, it occurred to me that I had let slip the opportunity to examine the patient's breast milk. However, 2 months later she turned up again bringing her baby.

**Examination of Breast Milk**

Both breasts were fairly full. The nipples were normal and there appeared to be no cracks or fissures. A small stream of milk was ejected.

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from the left nipple by slight digital pressure. Then a few more drops were gently expressed, one being caught on a freshly cleaned laboratory slide. This one drop was distributed over almost the whole slide by means of a platinum loop previously sterilised by making it red hot in a spirit flame. The slide was fixed and stained in the usual way for AFB.

Using a Vickers binocular microscope equipped with a movable stage, a thorough search of the slide was made. A group of 12 well stained shortened rods was soon found, and the remainder of a total of 118 acid fast bacilli were counted by searching approximately 2,650 fields, occupying about 2/3 of the whole smear, thus averaging 1 bacillus in 22 fields.

An experienced Japanese bacteriologist, Dr. Ivawamura, a member of our hospital staff, checked some of my findings. There could be no doubt at all in our minds that the bacilli we had seen in this woman’s milk were indeed M. leprae. The same arrangements or formations of bacilli were seen in the milk as can be seen in skin smears from active lepromatous leprosy. For example, bacilli lying side by side like cigars in a box, or a cluster of 12 well stained shortened rods, and pairs of slightly shortened bacilli lying parallel with a little gap between each pair. Others were found singly—full length, well stained, solid rods. Three which were judged to be solid rods were lying head to head in the shape of a letter ‘Y’ each one well stained. Fragmented bacilli were also very characteristic.

Details of the bacillary count are given as follows:

<table>
<thead>
<tr>
<th>Percentage</th>
<th>Description</th>
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<tbody>
<tr>
<td>92.7%</td>
<td>11 solid rods</td>
</tr>
<tr>
<td>99.7%</td>
<td>82 fragmented bacilli</td>
</tr>
<tr>
<td>19.5%</td>
<td>23 short rods</td>
</tr>
<tr>
<td>10.6%</td>
<td>2 granules</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>118</strong></td>
</tr>
</tbody>
</table>

Control

Just to check that my staining technique was not at fault, I prepared another slide of the milk of a healthy lactating woman. I used the same set of stains, the same solution of 10% sulphuric acid. None of these had been changed or replenished since preparing the slide from the lepromatous woman’s milk. A search of 1,500 fields (approx.) was negative. I was, however, impressed with the numbers of white cells present, which Dr. Ivawamura identified as lymphocytes and plasma cells. This led to a discussion about bacilli in breast milk, and there emerged the comparison between the breast milk of a woman suffering from miliary tuberculosis and that of a woman suffering from lepromatous leprosy, referred to later in this paper.

DISCUSSION

It seems clear that if ever this woman’s child develops leprosy the main portal of entry will have been the alimentary canal. Skin-to-skin contact or the mother’s clothes wrapped about the child may play their part in transmitting the disease, but, by comparison with the breast milk, it would seem to be a very minor part. In about a minuscule amount of its mother’s milk at least 118 acid fast bacilli were present. This could, perhaps, mean that in a 4 ounce feed approximately 2 million bacilli are present. Whether or not this is so, it seems certain that this child is consuming countless bacilli.

This finding lends significance to bacilli which might get trapped in the nasal mucosa. One way by which their deeper penetration into the body could occur would be by secretions from the naso-pharynx passing into the alimentary canal.

Thus, in the light of Professor Weddell’s findings, supported by the evidence given above, a mental picture emerges. Some of these bacilli are probably finding their way from this child’s alimentary canal into the blood stream. They will then pass to some of his Schwann cells. Here they will lie dormant during the so-called ‘silent’ or ‘latent’ period. In another paper on the pathogenesis of leprosy, Professor Weddell has likened the Schwann cell to a ‘refrigerator’. At the end of the incubation period or period of ‘refrigeration’, the bacilli emerge. Then, according to the degree of tissue resistance, leprosy could develop in one of its forms. At length it decides to display itself: pins
and needles sensation, an area of hypoesthesia, an enlarged cutaneous nerve, a patch, or an area of generalized diffuse erythematous infiltration, such as the skin of the face.

This picture may be a faulty one, but in the light of the findings and evidence already referred to, there seems to be enough truth in it to highlight the tremendous importance of preventative treatment, especially in the case of children of lepromatous mothers. My own line as regards this mother and her child will be to:

1. Treat the mother vigorously with DDS.
2. Find out if the child has a natural immunity to tuberculosis. If not, to give BCG inoculation, repeating, if necessary, at regular intervals in order to maintain a positive Mantoux reaction.
3. Give regular prophylactic doses of DDS to the child.
   If at all possible, to continue both these lines of preventative treatment to the child until well past adolescence.

We have been following these lines of attack in a nearby Government leprosy settlement for several years. They are suggested by Browne. In the light of the evidence given in this paper, prolonged preventative treatment is emphasised.

As regards the mother of this child, it seems that a specific comparison can be made between the breast milk of a woman suffering from miliary tuberculosis and that of a woman suffering from lepromatous leprosy. This comparison is made on the basis of 2 established facts:

1. That _M. leprae_ can be seen in the lumen of blood vessels of persons with very active lepromatous leprosy. I have 4 biopsies of my patients which demonstrate this.
2. That _M. tuberculosis_ have been found in the breast milk of women suffering from miliary tuberculosis, and that this finding has been reported in Japanese medical journals.

Therefore, it can be said that, just as the _M. tuberculosis_ can pass in the blood stream to the mammary gland and occasionally be found in the breast milk of a woman suffering from miliary tuberculosis, so it is possible for the _M. leprae_ to pass in the blood stream to the milk of this woman who is suffering from very active lepromatous leprosy.

It is hoped that workers in the field of leprosy will check on this finding by examination of the milk of every lactating woman suffering from lepromatous leprosy that they come across, especially those whose skin smears show both a high bacillary and a high morphological index.

Further investigation must be made to show if this is an occasional or a constant happening in lactating women suffering from lepromatous leprosy.

In lactating women suffering from other forms of leprosy which clinically and histopathologically could be shown to lie between the lepromatous end of the spectrum and the borderline zone, one could postulate that the bacilli, if present at all, would be increasingly difficult to find in the milk, by reason of their fewness, the nearer the patient approaches the borderline zone.

**SUMMARY**

Attention is drawn to the view held by leprologists that there is a lack of evidence to show that the alimentary canal may be a portal of entry for the _M. leprae_. Findings which appear to be definite evidence are submitted. The bearing of this evidence on the importance of preventative treatment is emphasised. Leprologists are urged to check the evidence given.

**ACKNOWLEDGEMENT**

I wish to express my thanks to Dr. Iwawura for checking the microscope findings, and for drawing my attention to the relevant facts in tuberculosis.
REFERENCES
4. WEDDELL. "Pathogenesis of Leprosy", Ciba Foundation.

CONTINUATION NOTES

SUMMARY OF PAST NOTES
2nd May, 1967. Date of first visit to O.Ps, when she brought her 6 months old baby. Found to be suffering from very active lepromatous leprosy—one year history since she became pregnant. M.I. 50-60%, B.I. +4. Started on DDS 30 mgm. per week and given 3 months' supply. Unfortunately did not think to examine the breast milk on this occasion.

8th July, 1967. Date of second visit to O.Ps. A total dose of DDS 270 mgm. had been taken since first visit. Breast milk examined, 118 acid fast bacilli of which 9.2% were solid rods, were present in approximately one minim of breast milk. Had I examined the milk at the first visit before any treatment had been given, it is highly probable that the number of bacilli and proportion of solid rods would have been much higher. Prescribed DDS 60 mgm. per week.

CONTINUATION NOTE
29th July, 1967. Date of third visit. Since the second visit a further 180 mgm. of DDS had been taken.

REPEAT EXAMINATION OF BREAST MILK: Three milk smears made as follows—

1. An ordinary smear, fixed and stained in the usual way using 10% sulphuric acid to decolourise. 1,500 microscopic fields searched. I could find no acid fast bacilli. I did find 4 irregularly stained very short fragmented acid fast organisms—each consisting of 2 granules, or having the appearance of a very short bipolar rod broken in the middle. These might have been bacillary debris.

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