A NOTE ON TUBERCULOID CHANGES IN LEPROSY AS SEEN IN INDIA

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The articles and correspondence regarding tuberculoid changes in leprosy that have appeared in the International Journal of Leprosy have been of much interest to workers in India. Since very many cases with such changes are seen here, especially in this part of the country, it is desired to offer certain comments on the matter, based on my own personal experience of this type of lesion.

During the several years that I was at Dichpali (in Hyderabad, South India) I saw only a few such cases, and I remember taking a photograph and making a biopsy examination of only one lesion of this variety. In that specimen I found a few bacilli. I had seen photographs and read some publications of the Calcutta workers concerning similar lesions, but I did not give the matter very close attention for lack of clinical material.

On coming to Calcutta, however, I found that patients with tuberculoid lesions formed about 50 per cent of all who come to this clinic. Muir explained the difference between Calcutta and Dichpali in this respect on the ground that the treatment center here attracted many early cases which would never have gone to an institution such as Dichpali. He thought that tuberculoid lesions were probably quite common all over India, but that they were often not detected as being due to leprosy. I do not feel that this is the whole truth. In Dichpali a certain amount of outpatient and village work was done, and we also had many early cases in the hospital. If tuberculoid lesions had been very common they could not possibly have failed to attract our attention.

Workers in Calcutta (Henderson [1, 2], Muir and Chatterji [7] and Lowe [5]) have made careful studies of these lesions, but only some of the findings have yet been published. In general our clinical findings are in agreement with those of Wade ([8]), and his

clinical description corresponds very closely with the condition as we see it in our patients. However, we find that, especially in the smaller lesions, the thickening and erythema are often not confined to the margin but involve the whole lesion. When such cases are kept under observation the healing at the center and spreading at the margin are often observed. Another point is that the infiltration in some of our cases is extreme, and much more marked than is shown in many of his photographs, and when these markedly infiltrated lesions are very numerous, and when as they commonly do, they occur on the face, the case may very closely simulate a C3 case of leprosy (Plate 38, Figs. 1 and 2). A third point in diagnosis of doubtful cases, and often an important one, is the great frequency with which we find thickened cutaneous nerves leading to the macules.

Our histological findings are similar to those of Wade; we find that the granuloma consists largely of epithelioid cells. However, from the nature of the granuloma alone we find it impossible to distinguish these lesions from ordinary lepromatous lesions. It is only the occurrence of giant cells, and the focal arrangement and distribution of the granuloma, that make the tuberculoid nature of the change apparent.

There are three points in connection with the pathology of these lesions in which our findings differ from Wade's. The first of these is in regard to the deep extension of the changes. Muir and Chatterji (7), by excision of the whole thickness of the skin together with the cutaneous nerve supply, found that the appearance in sections of round tuberculoid foci in the corium is really an artefact, that these foci are really transverse sections of branching granulomatous cords which run deep into the skin and are continuous with the lesions in the cutaneous nerve branches. This granuloma in the nerve branches is of exactly the same nature as that of the corium, showing tuberculoid changes and giant cells.

Secondly, as a logical conclusion from these observations, Muir and Chatterji considered that the tuberculoid lesion is essentially a granulomatous change arising in the neighborhood of the nerve ramifications in the skin and spreading upwards along the nerve branches. Jeanselme (3) has made similar observations. He says:

> The nerve ramifications in the skin are particularly involved. In certain cases the changes often appear to be entirely confined to their neighborhood. By special staining it has been found possible to demonstrate nerve fibers in the tuberculoid foci in the skin.

Thirdly, we differ from Wade on the matter of bacteriological contents of these lesions. Henderson, and Muir and Chatterji, originally found that smears and sections usually showed no acid-fast bacilli and the latter writers suggested that the lesions might be caused by some other form of the leprosy organism. However, further experience has modified this view. By using special staining methods (6) we have been able to demonstrate a few acid-fast bacilli in over 70 per cent of 100 specimens of biopsy material taken from tuberculoid lesions. This fact, together with the results of the leprolin test seen in patients showing tuberculoid lesions, suggests that these lesions are caused by a markedly enhanced power of the tissues to respond to a small number of bacilli, and also that the marked tissue response probably destroys most of the bacilli. Nevertheless, acid-fast bacilli can usually be found, nearly always in the cutaneous nerve branches and often in the corium. It is interesting to observe that the bacilli are very rarely found either inside or in the neighborhood of the giant cells, but they are more often found at the edge of the foci of granulomatous change. Nevertheless, I have once observed bacilli in a giant cell. A *camera lucida* drawing of this specimen is shown in Plate 38, Fig. 5; this was the only time that this observation was made in several hundreds of such sections examined.

We find that a clear-cut differentiation between tuberculoid and other lesions is not always easy. In addition to the typical lesions already described we get some that show very few giant cells and a considerable number of bacilli, and a few that show no giant cells whatever and many bacilli, yet all of these lesions appear clinically the same and show a similar distribution of the granulomatous change in the skin.

Our observations regarding the position of the tuberculoid change in the evolution of the disease are as follows: Commonly the first visible lesion is a tuberculoid macule, one single small macule being occasionally seen as the only evidence of the disease. Figure 3 is from a photograph of such a lesion, the only one found on a patient who came to our clinic very recently. It was excised, and sections showed typical tuberculoid changes. After examining several sections we found in one of them two acid-fast bacilli in the granulomatous area in the corium, and six in a nerve twig. Fig. 4 also shows a typical tuberculoid macule occurring on the back. The part marked was excised and sectioned, and a few acid-fast bacilli were found in the corium and many in the deep cords.

Frequently, however, several such macules appear more or less simultaneously, and thickening of the cutaneous nerves is soon observed. Sometimes the initial macule or macules may not have the tuberculoid appearance, and may show only depigmentation and sensory changes; in such cases the nerve thickening is often so slight as to be undetected. Then, for some reason not often apparent, the lesions and the nerves supplying them become thick and infiltrated, and biopsy examination shows the typical tuberculoid change. The infiltration usually subsides after a time, particularly in the center of the macule, but it may recur, and a gradual radial spread at the margin is often observed. Sometimes the tuberculoid lesion forms the center of a spreading leprotic infiltration of the skin, but when this becomes at all marked the infiltration of the original lesion often subsides and it remains as a depressed area in the middle of a typically leprotic lesion teeming with bacilli.

Generally speaking, we find that the existence of tuberculoid lesions (as also of marked cutaneous nerve thickening and nerve abscess which often accompany them) appears to be incompatible with the simultaneous existence of the leprotic infiltration in any marked degree. If the case becomes a C2 or C3 case the tuberculoid lesions disappear. On the whole, however, the existence of tuberculoid lesions is evidence of high resistance to leprosy, and we find that in comparatively few patients showing these lesions does the disease progress to the cutaneous type.

The question arises as to whether we in Calcutta are dealing with a local peculiarity of the disease, or whether such cases are common in other parts of India and in other countries. A survey of the literature of the subject of the tuberculoid condition in leprosy (which is extensive, for I have traced over fifty references to it) shows that such lesions are apparently much more common in some countries than in others. They are commonly seen in Japan, North India, and parts of Africa, but seldom in most other countries. However, there seem to be records of such lesions in practically every country in which leprosy exists, even in Norway where it is said to be very rare; Lie (4) reported having observed three cases. It is interesting to observe that when such cases occur in countries where they are rarely seen there often seems to be considerable difficulty in diagnosis, and I have seen several records of such patients being shown for diagnosis before dermatological societies where there was much discussion as to whether the condition was leprosy or tuber-

culosis of the skin. The clinical descriptions and the photographs which have sometimes accompanied these reports make it quite clear to us who see them so commonly that they were cases of tuberculoid leprosy.

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DESCRIPTION OF PLATE

PLATE 38

FIG. 1. Tuberculoid lesions of face, with marked thickening and erythema, simulating cutaneous-type leprosy. No bacilli found in smears, but a few in sections.

FIG. 2. A large, thick, indurated erythematous macule involving the forehead and nose, with marked keratosis; anesthesia complete. Biopsy was not done, but clinically the case is a typical one of marked tuberculoid lesions of the face, confirmed by negative bacteriological findings in smears.

FIG. 3. A small tuberculoid macule, the only lesion found on the patient. Histologically tuberculoid. A very few acid-fast bacilli were found in sections.

FIG. 4. A typical tuberculoid macule on the back. The part marked was excised, and numerous bacilli were found in sections.

FIG. 5. A camera lucida drawing of a giant cell in a tuberculoid lesion, containing several acid-fast bacilli.

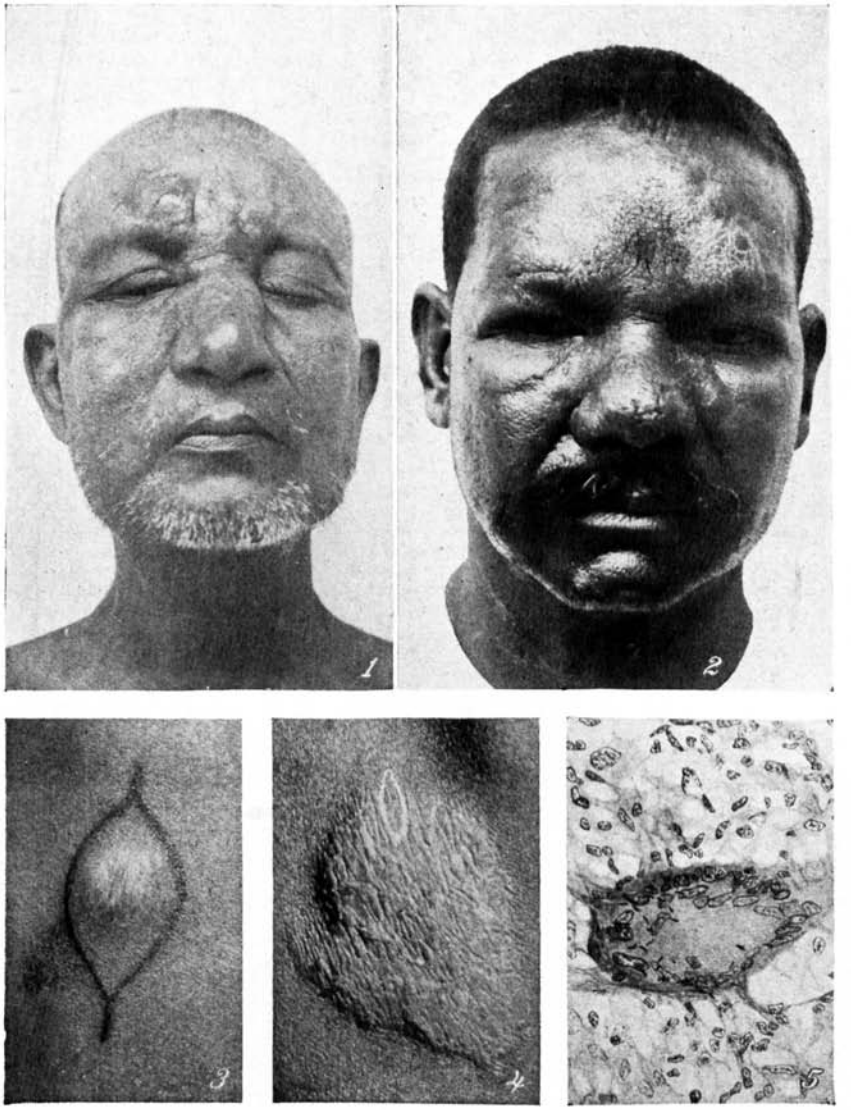

PLATE 38