

THE CONCEPT AND LIMITS OF BORDERLINE LEPROSY¹

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Before commencing the study of the form or "group" of leprosy termed "borderline," it is necessary to establish certain principles.

1: As I stated at the International Leprosy Congress held in Havana in 1948, and subsequently (¹¹), nature takes no heed of the rigid mould of our classifications, and between the extreme, typical cases of the polar forms—lepromatous and tuberculoid—there is a continuous chain of intermediate forms. Thus, if at some theoretical moment in the evolution of leprosy we draw a horizontal line with typically lepromatous and typically tuberculoid cases at the two ends, then in the middle we shall find a series which become continually less tuberculoid or less lepromatous, and finally a series of cases in which the clinical, immunologic, bacteriologic and histopathologic characteristics of both polar forms will be mixed to a varying extent. These are the borderline cases, ones which are intermediate between the two polar forms on a level above the simple macular forms.

2. It should never be forgotten, although unfortunately many authors have disregarded this fact in defending their points of view, that in the patient leprosy, like any other disease and like human life itself, is undergoing continuous change. During one period of its evolution, varying in duration, the polar characteristics are not firmly fixed and mutation may occur from one polar form toward or to another, and, much more frequently, from the initial indeterminate group to one or the other of the polar forms. It is in one of these phases of instability that the borderline cases appear.

These unstable forms were first described by Wade and Rodriguez (²⁵) as a mutation from tuberculoid which occurs after "relapse" in a tuberculoid case and as a result of repeated reactional episodes. This statement does not contradict the fact that, in other cases, the subsequent course followed by the disease in patients with indeterminate leprosy is more or less settled from the outset: cases in which the bacilli are present in numbers and the lepromin reaction is negative develop into the lepromatous form, if treatment does not cure them beforehand, and those which are bacteriologically negative and give a strongly positive lepromin reaction change into tuberculoid cases, with very few exceptions.

¹This paper is based on one presented at the Symposium on Borderline Leprosy, held in Rio de Janeiro in March 1960. It represents the author's personal views, and should not be regarded as an official opinion of the World Health Organization.

Finally, at the end of a certain time cases exist which are fixed permanently in a polar form and no changes of type will occur in these. This fixed point in the evolution of the disease is reached earlier and with greater constancy in the lepromatous form, which is more stable than the tuberculoid, the latter undergoing mutation more frequently; nevertheless, there are certain tuberculoid cases just as fixed and unchangeable as the lepromatous ones.

3. The failure on the part of non-Iberian and non-Latin-American leprologists, or at least some of them, to understand the Ibero-American classification is due to two facts:

(a) The Ibero-American doctrine is more than a classification; it is a whole physio-pathologic doctrine or theory to explain the course of leprosy, and we unduly confuse both names. It is logical and even obligatory for us to wish to adapt the classification to the theory, and this can be done. However, classification cannot be based on four different criteria, although generally all of these coincide in the same patient. This objection has frequently been raised at our international congresses. Since the time of the Havana Congress, the following order of priority has been used for the criteria of classification: “. . . in diminishing order of availability, (1) clinical, (2) bacteriologic, (3) immunologic, and (4) histopathologic.” A leprologist as outstanding as Wade has vigorously emphasized that: “the accepted primary criterion of classification is the clinical one—including, of course, the bacteriological findings in smears. Immunology (the Mitsuda reaction) and histopathology are contributory, but secondary.” (As Wade (²¹) expressed it in discussing the less familiar forms of leprosy, “an individual worker will see only what he is prepared to recognize.”) So as to acquire a better understanding of the different viewpoints I have endeavored, during the course of recent years, to get well-known leprologists in Africa and India to show me their clinical cases, in order to make clearer to me the views expressed in their publications.

HISTORY OF THE BORDERLINE FORM OR GROUP

It would take too long—and is probably unnecessary—to make an exhaustive analysis of all the literature dealing with this problem. Nevertheless, I deem it useful to mention the main papers on the subject so as to analyze the points of view, sometimes extremely varied, of the authors who have tried to clarify this problem, which most clinicians find difficulty in understanding.

Synonyms.—It is useful to recall the terms used to designate this group of cases, i.e., borderline, intermediate, transitional, *limitante* or *limitrofe*, *fronteirico*, N?C (CN?L), and dimorphous. In his first study on reactions in tuberculoid leprosy in 1934, Wade (¹⁹) pointed out that some of these cases had undergone a transformation into lepromatous cases (called nodular by Davison, from whom Wade obtained his mate-

rial); the study concerned South African patients. Subsequently, in collaboration with Lowe (²⁴), he studied the classification of patients in the leprosarium at Purulia, India, and described cases of this type as a subgroup of the lepromatous form. Still later, Lowe (^{13, 14}) designated this same type—i.e., cases of tuberculoid reaction or reactional tuberculoid leprosy which undergo a change into the lepromatous form—by the symbol N?C. The word “borderline” appeared for the first time in the paper by Wade and Rodriguez (²⁴) in 1940, with the title “Borderline Tuberculoid Leprosy.” It dealt with tuberculoid patients who had suffered relapses and, at least apparently, had undergone mutation to lepromatous cases. Subsequently, in 1941, Wade (²⁰) distinguished two stages in this process of mutation: tuberculoid relapse as the initial phase, and a more advanced stage of borderline tuberculoid leprosy.

It should be stressed that, so far, only the mutation of certain forms of tuberculoid leprosy has been given the name of borderline leprosy. Later on, I shall make it clear that there are also cases which are borderline from the outset, i.e., have initial symptoms corresponding to this group.

Cochrane (⁴), also in 1940, described cases of this group which he termed “intermediate.” Their fundamental difference from true tuberculoid cases was the negativity of the lepromin reaction.

To illustrate how difficult this question is, as well-known an expert as Cochrane has repeatedly tried to give different terms to describe these cases, in successive publications. These were recently analyzed by Wade (²¹), so that they can be briefly reviewed here.

The “intermediate” form of 1940 reappeared in 1946 (⁵) under the name of “uncharacteristic” or “borderline.” Cochrane then recognized four varieties of this type (already no longer considered as a group): atypical tuberculoid, true intermediate, sarcoidal, and atypical lepromatous.

In 1948, he (⁶) again changed the name of this form of leprosy and proposed the creation of the “atypical” leprosy group.

In 1949 (⁷), Cochrane suggested the name “dimorphous,” ascribed to Khanolkar, to designate this type which has the clinical and histologic characteristics of lepromas and leprids at one and the same time.

In 1951, in a communication to the Third Pan-American Conference of Leprology held in Buenos Aires (⁸), he returned to his first name of “intermediate.”

In 1953, at the VI International Congress of Leprology, held in Madrid, Khanolkar and Cochrane (¹²) presented a paper in which they defined the characteristics of their “dimorphous macular” form, and also spoke of the existence of a “dimorphous neural” form, which will not be considered in this article. The Classification Committee of that congress agreed to regard “dimorphous” as synonymous with borderline, but parenthetically, and with retention of “B” as the type symbol.²

From this time onward a certain group of leprologists, influenced by Cochrane and neglecting the classic and fundamental work of the Brazilian school of leprology, have regarded the macular dimorphous

²This was inadvertently changed about by the translator of the Spanish version of that report, but since the committee wrote its report in English, that one is to be taken as official.

leprosy group as having been firmly established. Thus, we are faced with two groups of data which will be analyzed separately.

GENUINE BORDERLINE GROUP

The clinical descriptions of borderline leprosy in the literature do not seem very clear, and despite tables of differential characteristics between this form and the reactional tuberculoid form, we find difficulty in seeing any clear difference between the two clinical types.

The initial paper by Wade and Rodriguez tells of three cases of relapse of initially tuberculoid cases. Sometimes the relapse preserves the initial tuberculoid features (first phase or stage of this state or condition), while in other cases it takes on a different aspect, appearing to evolve towards the lepromatous type although, according to the authors, this change is only rarely complete. Clinically the main lesions of the cases reported were described as tumid plaques of reactional nature, outwardly diffusing into the surrounding normal skin in the manner of lepromatous infiltrations, but which may contain, or be aligned along, areas of normal-appearing skin which were sites of former tuberculoid lesions which had healed; and at the edges of such areas the elevated infiltration ends abruptly.

Photographs published in a recent paper by Wade (²³) illustrates such a case. It is one of a Filipino girl who exhibited a lesion covering most of the left cheek, extending onto the nose and involving both eyelids, and up over the temple area and back to the ear. It was a smooth-surfaced reddish infiltrate, obviously of reactional nature, and it was centered by a large immune area against which the infiltrate abutted abruptly. The most important distinctive feature of this lesion is that around most of the outer edge the infiltrate thins off to the level of the normal skin, although it still appears to be elevated along a part of the lower border below the nose down past the corner of the mouth (see Figs. 1 and 2).

In the same paper, Wade published two photographs of typical, very marked reactional tuberculoid cases, and he affirmed—referring to the second case, which had an extensive large plaque on the arm centered by a large immune area — that “elevation is quite as distinct and marked at the outer edge of the plaque, where it would be thin and diffusing off if the condition were borderline.”

The first WHO Expert Committee on Leprosy (²⁶) recognized the existence of the borderline group, and, after dealing with the reactional tuberculoid condition, restricted itself to the following description:

A malign form, very unstable, almost always very strongly positive on bacteriological examination, generally negative to lepromin. This form frequently arises from the tuberculoid form as a result of repeated reactions, and sometimes evolves to the lepromatous form.

Thus only three characteristics of this group are mentioned: first, that bacteriologic examination is *almost always* strongly positive;

secondly, that the lepromin test is *generally* negative, and thirdly, that it *sometimes* evolves to the lepromatous form. It should be stressed that the description of these characteristics is qualified to some extent by using the terms "almost always" and "generally," so as to leave open the possibility that the bacteriologic examination may be negative, or the Mitsuda reaction positive.

As is seen below, the recommendations on the subject of the Sixth International Congress of Leprology (¹⁵), in which uses the same words "almost always" and "generally," are also used, is equally careful. The original wording (including a separate paragraph on reactions in these cases) is as follows:

A malign form, very unstable; almost always strongly positive on bacteriological examination; the lepromin reaction generally negative. Such cases may arise from the tuberculoid type as a result of repeated reactions, and sometimes they evolve to the lepromatous type. The nasal mucosa often remains bacteriologically negative even when the skin lesions are strongly positive.

The skin lesions are usually seen as plaques, bands, nodules, etc., with a regional distribution similar to that of lepromatous leprosy, except for conspicuous asymmetry. The ear lobes are likely to present the appearance of lepromatous infiltration. The lesions frequently have a soft or succulent appearance, and peripherally they slope away from the centre and do not present the clear-cut, well-defined margins seen in the tuberculoid type; they are therefore liable to be mistaken for lepromas. The surface of the lesions is generally smooth, with a shiny appearance and a violaceous hue; sometimes (in light skins) with a brownish (sepia) background.

In reactional borderline cases, the lesions show extreme oedema, erythema and desquamation. The reaction frequently extends to nerves, and marked nerve pain and dysfunction develop. The skin lesions, during this phase, may ulcerate superficially, or sometimes widely and deeply; and the skin is acutely tender. Bacteriologically the lesions are strongly positive. The lepromin reaction is usually negative.

In this description no reference is made anywhere to the general symptoms which frequently accompany these cases, and I believe that, in some respects, it is less clear than the original description by Wade and Rodriguez.

Here it is necessary to make note of the condition called "reactional tuberculoid leprosy." This was first distinguished as a distinct entity by de Souza Campos, in 1940 (¹⁶). In 1944, Bechelli, Rotberg and Maurano (²) again called attention to the differences between reactional tuberculoid leprosy proper ("a primary and sole manifestation of the disease or one following uncomplicated neuromacular lesions, appearing as an exanthematic outbreak") and the "reactivation of pre-existing tuberculoid lesions," as they describe the tuberculoid reactivation condition.

In 1954, de Souza Campos and Rath de Souza (¹⁷), in a paper written at the suggestion of the First Expert Committee on Leprosy, endeavored to clarify the differences between tuberculoid reaction, reactional tuberculoid leprosy and borderline leprosy. This paper stresses a fact which is only too often forgotten, namely, the differences—in my opinion fundamental—between tuberculoid leprosy during reactivation and

reactional tuberculoid leprosy. In the former, already existing (generally minor) tuberculoid lesions become acutely aggravated, while the latter is a clearly differentiated clinical form; as the authors vividly put it: "it is not an intercurrent condition."

The differences between reactional tuberculoid leprosy and borderline leprosy appear much more confused. For example, Cochrane in 1940 (⁴) summarized them as follows:

TUBERCULOID CASES

Lesions: Raised, erythematous and infiltrated, with sharply defined edges which remain distinct, even when they extend.

Reactive stage: Considerable tenderness of lesions. Febrile period, if any, of short duration. Emaciation seldom marked and patient rarely so ill as to need hospitalization.

Bacilli: Lesions positive, usually becoming negative within six months.

Lepromin: Reaction positive.

INDETERMINATE CASES

Raised erythematous lesions with a more succulent appearance; edges, while infiltrated, tend to be less well defined.

During reactive stage, marked burning and tenderness of lesions, which may ulcerate. With fever, sometimes prolonged, and emaciation, patient may appear extremely ill, and often requires hospitalization.

Positive, not becoming negative in less than 9 to 18 months.

Reaction negative.

This comparative description only shows differences of degree which are difficult to appreciate, apart from the statement that in the tuberculoid form the Mitsuda test is always positive while in the intermediate form it is negative.

The same applies, more or less, to the article by Souza Campos and Rath de Souza referred to (¹⁷), from which the tabulation shown in Table 1 is taken (not including the section on ordinary lepra reaction, ENL).

As can be seen, most of the differential characteristics are more ones of degree than of fundamental differences. I should like to stress the statements that the reactional tuberculoid cases, when they suffer relapses, have a tendency to become borderline, and that the borderline cases, when they have a tendency to regress, return to the reactional tuberculoid characteristics.

Over a number of years, during repeated trips in different parts of Africa and South-East Asia, I have endeavored to collect observations on clinical cases which I felt might be of borderline nature, and, whenever possible, photographs as well as biopsy specimens of those lesions which appeared most lepromatous in nature. I discussed with different leprologists the cases they diagnosed as borderline, in order better to understand their interpretation of this group.

I have had occasion to review with Dr. J. Convit, of Caracas, Venezuela, his collection of borderline cases (¹⁰). He agreed that, in many instances, neither clinical nor histopathologic examination could establish the differential diagnosis between reactional tuberculoid and borderline, but that the only typical feature of borderline cases was their capacity to take on blue coloration in lesions or parts of lesions

TABLE 1.—Main features of "tuberculoid reactivation," "reactional tuberculoid leprosy," and "the borderline condition" (de Souza Campos and Rath de Souza).

	Tuberculoid reactivation	Reactional tuberculoid leprosy (RTL)	The borderline condition
Origin	Slow onset. Intercurrent; appears in the chronic evolution of T leprosy.	Acute onset. Secondary, in macular cases or, rarely, circinate T in reactivation. May be initial manifestation.	Acute onset. Usually secondary to RTL after repeated relapses; may be initial manifestation of the disease.
Eruptive type	Pre-existing lesions present activated borders, infiltrated, enlarged, reddish; new lesions may appear. Never abrupt outbreak as in RTL.	Polymorphous lesions (tubercles, nodules, plaques, etc.), reddish or wine-colored tumid and succulent. Peculiar features of location.	Same aspect as RTL; color tends ferruginous; external borders tend to fade away, inner often abrupt; infiltration less accentuated.
General phenomena	Absent.	Rarely acute phenomena; usually sub-acute (sub-febrile, moderate joint pains, etc.); sometimes entirely absent.	Almost always present (fever, headache, severe arthritis, etc.); patients frequently confined to bed.
Evolution	Subsides, the case returning to chronic evolution, frequently with increased lesions. With relapse, may evolve to RTL.	The acute phase subsided, usual tendency to clinical cure. With relapse, tendency to become borderline.	The acute phase subsided, lepromatous aspect frequently assumed. Rarely, involution produces or restores RTL characteristics.
Bacteriology	Persistently negative. Development of positivity usually coincides with tendency to change to RTL.	Frequently positive while acute (bacilli numerous, globi absent); tends negative with regression.	Usually positive before and during the outbreak (globi). When negative, tendency to regression to RTL.
Lepromin reaction	Almost always positive, of greater or lesser intensity.	Nonreactive while acute and B+; positive if without bacilli, increasing with regression.	Practically always and permanently negative. Rare positive reactions of slight intensity.
Structure	Classical tuberculoid with acute inflammatory phenomena: vascular dilatation, endothelial swelling, edematous dispersion, etc. No lipids; usually no bacilli.	Less typical tuberculoid, plus other changes of (2), the edema intra- and extracellular with consequent changes. Bacilli almost always found, but lipids absent.	Intermediate between RTL and lepromatous, Bacilli always abundant (globi); re lipids, not yet well studied.

that had become lepromatous after intravenous injections of methylene blue, those cases showing a blue color being borderline, and those without any change in color being reactional tuberculoid.

On only one occasion was I fortunate enough to obtain a biopsy specimen showing a mixed lepromatous and tuberculoid structure in the same section, although other more fortunate leprologists have come across several cases of this nature, and Cochrane showed me in his laboratory various specimens concerning whose mixed structure there could be no doubt. The remaining cases, after histologic examination made without seeing the patient or his photograph, by capable histologists such as Rodriguez Puchol in my laboratory in Madrid and J. M. M. Fernandez in Rosario, Argentina, were labelled unhesitatingly as reactional tuberculoid, apart from a very few which were lepromatous.

It is not my intention to make any special reference to the histologic aspects. I simply wish to say that before a diagnosis of borderline is accepted histologically, the patient should show both the lepromatous and tuberculoid structure in the same lesion or in different lesions. I believe it unjustified to base this histologic diagnosis on differences of detail, such as the presence of a free subepidermal band in a case with a typical tuberculoid structure. Furthermore, after full discussion with Wade, I recognize that, as recently stated by Alonso and Azulay (¹), on many occasions it is impossible to confirm the diagnosis of borderline histologically in indisputable clinical cases, perhaps because we have not made a sufficient number of biopsies and, in addition, because the histologic structure may change during the evolution of the disease process.

My initial statement was that between the typical polar forms, lepromatous and tuberculoid, there is a continuous series of transitional cases, so that the borderline group neighbors on the lepromatous type and must have, at the other extreme, a frontier with the tuberculoid type. In this connection I shall mention three characteristics of the borderline group, accepted by all authors:

1. Although the borderline group cases may be, exceptionally, primary, or borderline from the outset, they generally originate through a mutation of tuberculoid cases, starting from a tuberculoid relapse (Wade and Rodriguez) or through a mutation after one or more outbreaks of reactional tuberculoid (de Souza Campos and Rath de Souza).

2. The instability of the borderline group, which can regress to the reactional tuberculoid form or—more rarely—develop into the lepromatous type, was stressed in the first report of the WHO Expert Committee on Leprosy and by the Sixth International Congress of Leprology.

3. Its close relationship to the reactional tuberculoid form, which occasionally makes differential clinical diagnosis of these two forms of leprosy impossible, the frequent evolution of the reactional tuberculoid

cases to borderline forms, the less frequent involution of borderline cases to the reactional tuberculoid forms—all this shows an extraordinary relationship between the two forms of the disease.

On the other hand, the frontiers between the reactional tuberculoid form and “tuberculoid reactivation” seem to me to be clearer and easier to establish, without neglecting the possibility of the change of a reactivated tuberculoid case into a reactional tuberculoid or borderline case, generally after a relapse.

In my view, then, it would be more logical to establish what might be termed the “tuberculoid frontier” of the borderline group between the reactional tuberculoid and tuberculoid reactivation forms, and to consider a borderline or intermediate group (I reject the term “dimorphous”) with two clinical varieties having rather indefinite boundaries, one more tuberculoid including the former reactional tuberculoid type, and the other closer to the lepromatous type, the genuine borderline group. The group would thus have the following form:

Intermediate or borderline group / Relapsed tuberculoid and reactional tuberculoid variety
 \ Borderline variety (close to the L type)

In a symposium on borderline leprosy held in Rio de Janeiro in March 1960, de Souza Campos, the author who first distinguished reac-



FIGS. 1 and 2. Early borderline leprosy in a young Filipino girl, occurring as a relapse reaction 10 years after the appearance of the original tuberculoid lesion on the cheek. That lesion is now represented by an “immune area” of normal appearance, against which the infiltration of the new lesion abuts abruptly, while it thins off outwardly over most of its periphery. [From Wade (23).]



FIG. 3. A young Indonesian girl, October 14, 1959, with a typical reactional tuberculoid lesion covering the left half of the face, from forehead to chin and including the nose. On the right cheek is a lesion which was perhaps the initial one.

FIG. 4. The same patient as in Fig. 3, on February 1, 1960, after 3½ months of DDS treatment. The infiltration of the lesions has subsided.

tional tuberculoid leprosy, proposed, exactly as I did, the formation of one group only for reactional tuberculoid and borderline.

To illustrate the different gradations of the clinical aspect of the borderline group, we may start with two photographs published in the paper by Wade (²³) (Figs. 1 and 2). These represent a typical example of a borderline case most closely related to the tuberculoid type on the frontier of tuberculoid leprosy.

The first picture of the present report (Fig. 3) shows the lesion of Nani, a young Indonesian girl who, on October 14, 1959, presented a typical reactional tuberculoid leprosy lesion covering the left half of the face including both eyelids, and the whole of the surface of the chin and the nose extending to the right cheek. The lesion is a heavy, smooth infiltrated plaque, reddish-brown in color. On the right cheek exists another lesion with well-defined outer edges, perhaps the initial tuberculoid lesion, also in reaction. The Mitsuda reaction was doubtful; bacteriology, negative; histology showed a tuberculoid reactional picture.

Three and one-half months afterwards, on February 1, 1960, during



FIG. 5. Reactional tuberculoid lesions in an Indonesian girl, with two centrally healing plaques on the chest. The outer edges of these lesions are less well defined than the inner edges, although the latter are not sharp. Plaques on the face, more infiltrated in the centers than outwardly. A less marked lesion on the right arm above the bend of the elbow.

which time the patient had been under DDS treatment, the lesions were no longer infiltrated and the reddish-brown color had almost disappeared (Fig. 4).

Fig. 5 shows the lesions of Masnati, an Indonesian girl, with two tuberculoid plaques on the chest which show healing centers. The outer edges are not well defined; the internal edges are more clearly defined than the external ones, but they are not as sharp as in Wade's case. On the face there are several plaques, reddish-brown, smooth, more infiltrated centrally than at the edges. Bacteriology, scanty bacilli; Mitsuda reaction, negative; histology, tuberculoid reactional picture.

Figures 6 and 7 are of Halima, a young Indonesian girl, with a typical tuberculoid plaque in reaction on the trunk, and an exanthematic outbreak of plaques and nodules on the face, eyebrows, arms and hands, with a few on the chest. The plaques are reddish-brown, smooth, and generally much more infiltrated in the center than at the edges, but the edges of the elevated lesions are clearly defined. On the cheek the plaques are confluent, with a tendency to join together in polycyclic form. The earlobes are much infiltrated and have a lepromatous aspect. Bacteriology, a few bacilli in the earlobes; Mitsuda reaction, doubtful; histology, tuberculoid reaction by biopsy of the earlobes.

With prednisolone treatment and DDS, the lesions cleared up in a



FIGS. 6 and 7. A young Indonesian girl with a typical tuberculoid plaque in reaction on the trunk, and a widely disseminated exanthematic outbreak of plaques and nodules elsewhere, especially on the face, arms and hands.

FIG. 8. The same patient as in FIGS. 6 and 7, 3½ months later, after treatment with prednisolone and DDS. The lesions have almost disappeared.





FIG. 9. A borderline case with very marked lesions of the face closely related to lepromatous leprosy, in an African patient at the Westfort Institution, Pretoria. (Seen with Davison.)

few weeks. Fig. 8, taken on February 1, 1960 (three and one-half months later) shows that the lesions had almost disappeared.

Figure 9 shows a borderline case with very marked lesions most closely related to the lepromatous type of leprosy. It is of an African patient with plenty of nodules of lepromatous aspect, certain plaques with sharp and elevated edges, and metastatic lesions spread over the whole body. This patient was observed with Davison in Westfort Institution, Pretoria, in 1959. Bacteriology, numerous bacilli (no globi).

Figures 10 and 11 show an example of an unusually marked, but typical, tuberculoid reactivation. The irregular, annular plaques show well-defined and sharp outer edges, especially the one on the front of left arm. The central immune areas are also limited by very sharp edges. Not all of the existing lesions were involved in the reaction; on the back of the left arm and forearm are definite minor tuberculoid lesions the quiescence of which is in marked contrast to the other lesions. The external edges on the left arm are typical tuberculoid, but in some plaques on the trunk the immune areas are limited by very sharp edges better defined than the external edges of the same plaques.

DIMORPHOUS MACULAR LEPROSY

It has already been mentioned that the first attempt by Cochrane, together with Khanolkar (¹²), to establish the dimorphous macular



FIGS. 10 and 11. An example of marked but typical tuberculoid reactivation.

group was at the Madrid congress. Recently, Browne (³) described 62 cases of dimorphous macular leprosy seen at the Yalisombo leprosarium in the Belgian Congo. These represented 10 per cent of the hospitalized cases, and such cases were also found in 3.2 per cent of the outpatients. I would emphasize that the number of cases diagnosed by Browne as indeterminate was only 3.5 per cent of the total number of patients, both inpatients and outpatients.

The clinical description of Browne's cases can be summarized as follows: They commence with a hypochromic single macule with well defined edges, more or less anesthetic and occasionally slightly erythematous. This initial lesion remains stationary for some months, in exceptional cases for some years, and then an exanthematic outbreak of small lesions appears, described first as hypochromic erythematous macules, but later as hyperemic and infiltrated.

It is curious to note the bacteriologic findings described step by step by Browne. The initial lesion may at first be bacteriologically negative or show few bacilli; furthermore, bacilli are not found in either the nasal mucosa or the earlobes. Immediately before the outbreak of secondary lesions, numerous bacilli can be seen, including globi; while at the time of the outbreak abundant bacilli are found, and frequently globi, in all the secondary lesions and almost always in the nasal mucosa and earlobes.

This description would seem to duplicate that given in 1941 by Souza Lima and Alayon (¹⁸) in their classic monograph on "incharacteristic" lesions, when they describe the abrupt lepromatous transformation of such lesions. To make the similarity even more complete, Browne men-

tions, as do the Brazilian authors, that immediately before the outbreak a temporary regression of the initial hypopigmented macular lesion is often seen.

My firm impression that this "dimorphous" macular form, of which I have seen many cases so labelled by followers of Cochrane, is merely the transformation phase of an initial indeterminate lesion was strengthened on reading the chapter on classification in Cochrane's latest book (⁹). The author states that what he calls "dimorphous" is neither a group nor a type, as these terms are used in formal classification, but a phase or stage through which pass most leprosy cases which do not remain with a fixed indeterminate morphology or undergo regression, either spontaneous or following treatment.

This is clearly specified in the diagram in Fig. 77 of Cochrane's book, and would appear to follow from the classification tables (p. 158), although I must confess that I fail to understand the advantages of the system suggested, comprising both a detailed classification and one for workers in mass campaigns.

There is one very suggestive detail in the classification proposed for field work. In it the dimorphous macular type and the reactional tuberculoid group or form are deleted. The first, since it exists according to Cochrane and his followers, should be included in the indeterminate group, while the reactional tuberculoid type, since its existence is also recognized in the previous table, will doubtless be included in the borderline group, i.e., what is finally advised here.

THE INDETERMINATE GROUP

In conclusion, I shall make a brief reference to the indeterminate group. One of the main differences between the ideas of the American school and those of the Indian leprologists is the special position of the maculoanesthetic form. I merely point out that in the description by the first WHO Expert Committee on Leprosy, unstable indeterminate lesions and other more stable maculoanesthetic lesions were included in the indeterminate group. Clearly, a large number of these lesions have a slightly tuberculoid structure and the lepromin reaction is positive.

As a result, at the Madrid Congress (¹⁵) the extent of the indeterminate group was reduced and maculoanesthetic cases were included in the tuberculoid group as a macular tuberculoid variety. From a strictly scientific viewpoint this is certainly justified; on the other hand, a large number of leprologists work under conditions in which it is impossible to carry out systematic lepromin tests or histologic studies of their cases. It is also certain that some maculoanesthetic cases do not show a tuberculoid structure, at least in a single biopsy, and that in them the Mitusda reaction is practically negative. Nevertheless, from the clinical viewpoint, they are both identical.

If the clinical criterion were to be regarded as the most important one for classification, would not an end be put to much sterile and purely academic discussion, by returning to the concept of an indeterminate group proposed by the Havana congress? All clinicians would agree in accepting a macular lesion group which would include the varieties indicated, and which might be subdivided following more thorough immunologic and histopathologic studies.

CONCLUSIONS

1. Borderline leprosy comprises a group of cases intermediate between the tuberculoid and lepromatous types which, in exceptional circumstances, may evolve to the latter form. It is an unstable group which responds relatively well to treatment and whose clinical symptoms are influenced favorably by combined sulfone, thiosemicarbazone, and corticoid derivative medication.

It includes two varieties, one closer to the tuberculoid type, namely, the reactional tuberculoid form or variety, and another closer to the lepromatous type, to which some cases show remarkable clinical similarity, this being the genuine borderline variety or form.

Clinically it is characterized by raised lesions, in the form of plaques or bands and nodules, reddish or wine-colored, sometimes grayish, which occasionally are similar to those of tuberculoid reaction, while others, particularly in certain parts such as the earlobes, resemble the lepromatous type. The general state of health may be affected. Such cases are almost always bacteriologically positive, more intensely so the nearer the case is to the lepromatous type. The lepromin reaction is usually negative; in a few cases it is positive, although never intensely so.

2. The so-called dimorphous macular form (used by some authors) is admittedly neither a group nor a type. It is said to be a usual stage or phase in the evolution of indeterminate cases into the lepromatous type. It would be better and more comprehensible for the majority of leprologists to conserve, for this stage in the evolution of indeterminate cases, the term "indeterminate" and to suppress the term "dimorphous macular."

CONCLUSIONES

1. La lepra limítrofe comprende un grupo de casos intermedios entre las formas tuberculoidea y lepromatosa, que, en circunstancias excepcionales, pueden evolucionar hacia la última forma. Trátase de un grupo inestable que responde bien al tratamiento y cuyos síntomas clínicos son afectados favorablemente por una medicación combinada de sulfona, tiosemicarbazona y derivados corticoideos.

Abarca el grupo dos variedades: una más próxima a la forma tuberculoidea, o sea la forma o variedad tuberculoidea reactiva, y la otra, más próxima a la forma lepromatosa, a la que algunos casos muestran notable semejanza clínica, siendo ésta la verdadera variedad o forma limítrofe.

Clínicamente, caracterízase por lesiones elevadas en forma de placas o franjas y nódulos, rojizas o de color vinoso, grises algunas veces, que de vez cuando son similares a

las de la reacción tuberculoidea, mientras que otras, en particular en ciertos sitios tales como los lóbulos de las orejas, se parecen a la forma lepromatosa. Esos casos son casi siempre positivos bacteriológicamente, más intensamente mientras más se aproxima el caso a la forma lepromatosa. La reacción de la lepromina suele ser negativa; en algunos casos es positiva, pero nunca intensamente.

2. La llamada forma macular dimorfa (término usado por algunos autores) no es reconocidamente ni grupo ni forma. Dicen que es una etapa o fase habitual en la evolución de los casos indeterminados hacia la forma lepromatosa. Sería mejor y más comprensible que la mayoría de los leprologos retuvieran para esta etapa de la evolución de los casos indeterminados, el término de "indeterminado y suprimieran el de "macular dimorfa."

CONCLUSIONS

1. La lèpre border-line comprend un groupe de conditions intermédiaires entre les types tuberculoïde et lépromateux qui, dans des conditions exceptionnelles, peut évoluer vers ce dernier type. Il s'agit d'un groupe instable qui répond relativement bien au traitement, et dont les symptômes cliniques sont favorablement influencés par une thérapeutique combinant les sulfones, la thiosemicarbazone et les corticostéroïdes.

Ce groupe englobe deux variétés, dont l'une se rapproche du type tuberculoïde, c'est la forme ou variété tuberculoïde réactionnelle, et dont l'autre se rapproche du type lépromateux, avec lequel elle peut montrer, dans certains cas, une remarquable similitude clinique. C'est cette dernière variété qui est communément considérée comme border-line.

Cliniquement, la lèpre border-line est caractérisée par des lésions surélevées, plaques, bandes ou nodules, de couleur rougeâtre ou vineuse, quelquefois tendant vers le gris. Ces lésions peuvent, à l'occasion, être semblables à celles notées dans la réaction tuberculoïde, alors que d'autres, surtout en certains endroits tels que les lobules de l'oreille, ressemblent au type lépromateux. L'état général être affecté. De tels cas sont presque toujours bactériologiquement positifs, et se rapprochent d'autant plus de la lèpre lépromateuse que la bactériologie est positive. La réaction à la lepromine est habituellement négative; dans un petit nombre de cas elle peut toutefois être positive, jamais fortement cependant.

2. La forme dite dimorphe maculaire, ainsi nommée par certains, n'est considérée ni comme un groupe ni comme un type. Elle passe pour être un stade habituel, une phase dans la transformation des cas indéterminés en type lépromateux. Il serait préférable, et plus compréhensible pour la majorité des leprologues, de conserver le terme "indéterminé" pour cet épisode de l'évolution des cas indéterminés, et de biffer le terme "dimorphe maculaire."

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