# Histoid Variety of Lepromatous Leprosy\*

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Histoid lesions seen in 15 patients at Ankaful Leprosarium, Ghana, over a period of 7 years are described. Some of the earlier observations made by other workers on this interesting variety have been confirmed. The significance of this type of lesion, as far as it is known at present, is discussed.

#### Introduction

The histoid variety of lepromatous leprosy was first described by Wade in 1963. In the latter part of the same year, Wade sent collections of sections from histoid lepromatous cases to various centres and our interest in this interesting variety which was aroused at that time has been maintained. Since 1964, we have seen 15 patients with the histoid type of lesion, and in this report we present a few salient observations, some of which have earlier been mentioned by other workers, on this interesting variety of lepromatous leprosy.

#### Materials for Study

The 15 cases mentioned above occurred in a total patient population of 960 seen since 1964 at Ankaful Leprosarium. All 15 cases were confirmed by histopathological and bacteriological studies.

Of these, 9 were relapsed cases, while the remaining 6 had not previously received any specific antileprosy treatment. It should be noted here that while these 15 cases conformed to the characteristic features of histoid lepromatous leprosy, 10 more cases showed only early histoid features either clinically or histopathologically or in both.

In 3 of the latter 10 cases the clinical features underwent regression, while in the remaining 7 cases the subsequent biopsies showed only isolated areas of early histoid features which were overrun by the general picture of lepromatous pathology. These were taken to be stages, caught in the initial transformation into the variety. For the purposes of this report these 10 cases are not included.

#### CLINICAL FEATURES

The essential clinical features noted could be divided into 4 main types: (1) Subcutaneous nodules—these are separate nodules which are not tender. They grow primarily by expansion and tend to expand upwards and to become attached to the dermis. (2) Deeply fixed cutaneous nodules. These are derived from the subcutaneous nodules. Their tendency is to soften, rupture and ulcerate

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on the top, and finally heal with superficial scarring. (3) Superficially placed cutaneous nodules. These are primarily cutaneous lesions, are protuberant and pearl-like in appearance, and occasionally become pedunculated. (4) Histoid plaques or pads. These are seen over bony prominences, especially around the elbows and knees.

#### CHARACTERISTICS OF HISTOID LESIONS

- (a) Many young histoid nodules were found to be transient in nature (Rodriguez, 1969).
- (b) In long-standing cases the different varieties of lesions mentioned above were seen in the same patient. This was particularly observed in relapsed cases (see Fig. 1).
- (c) In the majority of our cases, characteristic lepromatous infiltration was largely limited to the face and ears, leaving the back and extremities as areas prone to the formation of histoid nodules. In some cases the infiltration was minimal and the isolated histoid lepromata looked seemingly innocent.
- (d) Involvement of the nasal mucosa was far less than would be expected (Price and Fitzherbert, 1966) and there was no involvement of the eye except for the formation of pannus in one case.
- (e) Trophic disturbances in the limbs, such as dyskeratosis, atrophy of the nails, and hyperpigmentation following chronic venous and lymphatic stasis, were minimal in the histoid cases.

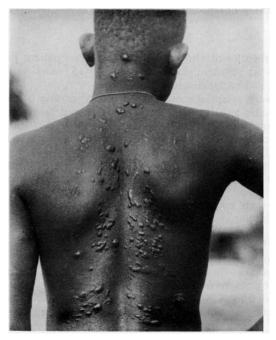


Fig. 1. Subcutaneous nodules, deeply fixed cutaneous nodules, superficial cutaneous nodules and plaques in a lepromatous patient.

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(f) Erythema nodosum leprosum, the frequently seen subacute reaction in lepromatous leprosy, was not seen in any of the histoid cases.

#### BACTERIOLOGY AND HISTOPATHOLOGY

It was observed in skin smears that the large number of leprosy bacilli were grouped in dense "bundles", and that the "globus" formation was rarely seen. Also the average size of the bacilli was observed to be larger than usual. Wade (1963) commented that the absence of "globus" formation may perhaps be explained on the ground of some local peculiarity of metabolism of the bacilli in the environments obtained, whereby they fail to produce the glial substance essential to the formation of the globus which occurs in the ordinary leproma.

The typical histoid nodule presents histopathologically a picture of intertwined whorls and bands of spindle-shaped cells, together with areas which are entirely composed of large, round histiocytes massed with bacilli (see photomicrographs Figs 2 and 3). These constitute what Wade (1963) described as "histoid habitus". In Wade's modification of Fite stain, the bacilli show deep purple staining which in places gives the appearance of an amorphous mass of pigment completely overlapping the identity of the cells in which they are contained. The cellular

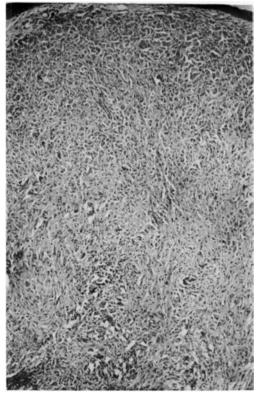


Fig. 2. Lepromatous leprosy. Section of the skin shows predominantly histiocytic infiltration. Histiocytes show a downward pattern of distribution which gives a whorled appearance. (H & E  $\times$  60)

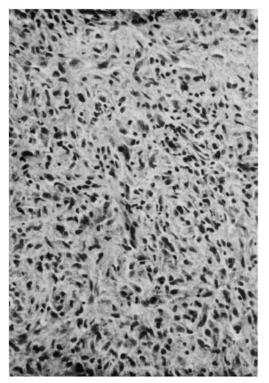


Fig. 3. Lepromatous leprosy. Section of the dermis showing histiocytes distributed in whorled fashion. (H & E  $\times$  300)

infiltrate itself seems to expand and push out the collagen matrix to form a pseudocapsule. These nodules are well vascularized but contain no nerve twigs. The interesting feature which Wade described as "contaminating tuberculoid foci" was observed in some of our cases where in isolated areas the epithelioid cells and lymphocytes constituted a small focus within the predominantly histiocytic infiltration.

It was observed that these areas contained few or no bacilli in Wade-Fite staining. The other interesting feature, which was mentioned by Wade (1963) and has been confirmed in the present study, is the alteration in the stainability of the bacilli in such "contaminating foci". When sections were cut from a paraffin block the bacilli in the sections, made a few years later out of these blocks, showed a "defatting" effect and were found to be smaller and fewer in numbers. The exact mechanism of this is not understood.

### SIGNIFICANCE OF HISTOID LESIONS

The appearance of histoid lesions certainly indicates a highly active lepromatous process. This type of lesion is associated with drug resistance against dapsone (Pettit *et al.*, 1966). It is thought that this type of lesion is caused by a

mutant variety of *Myco. leprae* (Rodriguez, 1969). We do not know details of this supposed mutation-for example, whether it is a genetic or a phenotypic adaptation, or both. The fact that histoid leproma are seen in relapsed cases, especially in a post-sulphone period, indicates that an increased incidence of such lesions poses a considerable epidemiological problem. What is the mechanism of formation of such single tissue-element infiltrate composed entirely of, spindle-cell tissue or histiocytes? These lesions are not at all ascribable to the "fibrosis" of ordinary lepromata (Wade, 1963) and they are very different from the inflammatory granulomata seen in the ordinary lepromatous case.

Lever (1961) holds that in the skin the adventitial cells around a blood vessel, which normally develop into fibroblasts, may under pathological conditions, produce histiocytes. Petit *et al.* (1966) conjecture that in leprosy an acute increase in the number of bacilli might cause these adventitial cells to act as host-cells for bacilli, retaining some fibroblastic properties though not producing any large amount of collagen. Such cells differ somewhat from the normal reticulo-endothelial host cell and so modify the attributes of the granuloma.

The significance of the "contaminating tuberculoid foci" in the histoid lesions is not entirely clear. Melamed *et al.* (1964) view this as evidence of a transitional phase borderline reaction in its evolution to lepromatous pathology. Pettit *et al.* (1966) consider that these contaminating foci as examples of tissue reactivity which could be elicited even in a pure lepromatous (LL) lesion.

The histoid variety of a lepromatous lesion can be easily missed or misdiagnosed, especially where the luscious shiny isolated nodules are not accompanied by discernible infiltration elsewhere in the body. To pathologists, these nodules may masquerade as nodular subepidermal fibrosis, xanthoma, fibrosarcoma, or keloid. The diagnosis can of course be clarified by acid-fast staining and Mallory's aniline blue staining for collagen. To the clinician, especially in West Africa, histoid lesions of lepromatous leprosy may bear a resemblance to an onchocercal nodule, keloid, warts, Kaposi's sarcoma, or neurofibroma.

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