ORIGINAL CONTRIBUTIONS

HISTOPATHOLOGICAL STUDY OF DIFFUSE IDIOPATHIC MELANODERMA

N G Jogaikar, Mohan B Gharpuray, Vinay Kulkarni and K R Chandvania

Histopathological study of clinically involved as well as normal skin in 30 cases of diffuse idiopathic melanoderma revealed increased melanin pigmentation of the basal cells in all the cases, liquefactive degeneration of basal cells in 20 cases, epidermal atrophy in 17 cases, melanophages in the upper dermis in 26 cases, and a perivascular or diffuse lymphohistiocytic infiltrate in 28 cases. There were distinct clinical and histopathological differences from lichen planus.

Key words : Diffuse idiopathic melanoderma, Histopathology.

Disorders of hyperpigmentation are one of the commonest and the most perplexing problems in dermatology because many a times the aetiology remains obscure despite detailed investigations. Also, as there is no satisfactory treatment, it becomes more vexing and troublesome for the patient as well as the dermatologist treating it. We undertook histopathological study of diffuse melanoderma to understand the aetiopathology of this common perplexing problem.

Materials and Methods

Thirty patients having diffuse hyperpigmentation of skin involving not less than 30% of the body area were included in the study. The patients were of the age groups from 9 years to 68 years and of both sexes in equal proportions, and had lesions mainly in the central parts of the body. These patients were selected after a thorough dermatological and systemic examination. Patients with hyperpigmentary disorders associated with systemic disturbances or psychiatric disorders or with a history of drugs or external applications were excluded. Routine investigations for hemogram, urine analysis and stools examination were all within normal limits.

The patients were randomly divided into two groups. In all patients from both the groups, a biopsy was taken from the clinically involved skin. The other biopsy for comparison was taken in the first group consisting of 18 patients from peri-lesional normal-looking skin. In the remaining 12 patients, this second biopsy was taken from the normal skin atleast 15 cm away from the margins of the lesions. Two sections were selected from each biopsy. One was stained with routine haematoxyline and eosin (H & E) stain and the other was stained with acid orcein Giemsa (AOG) stain. The latter was used for better visualization of melanin as suggested by Pinkus and Mehregan.[1]

Results

Histopathological changes in the biopsies from the lesions in both the groups were similar, while those in the peri-lesional normal skin and the distant normal skin differed remarkably.

In the involved skin, the most remarkable change in the epidermis was increased melanin pigmentation of the basal layer. This was

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uniform and was seen in all the 30 (100%) cases. The next characteristic change was liquefactive degeneration of the basal cell layer observed in 20 (66.6%) cases. Epidermal atrophy with flattening of the rete ridges was seen in 17 (56.6%) cases. One third i.e. 10 cases showed epidermal hyperplasia. Hyperkeratosis was seen in 4 (13.3%) cases and granular layer hyperplasia in 1 case only. The most prominent dermal feature was the presence of abundant melanophages in the upper dermis seen in 26 (86.6%) cases. Infiltrate in the dermis was present in 28 (93.3%) cases. It was purely lymphocytic in 22 (73.6%) cases and lymphohistiocytic in 6 (20%) cases. The infiltrate was distributed either perivascularly or it was scattered in the dermis. A band-like infiltrate was seen in 11 (33.3%) cases.

Clinically uninvolved skin immediately adjacent to the lesions showed histopathological changes seen in the involved skin almost in similar percentages. On the other hand, the normal skin distant from the lesions did not show any such similarities.

Comments

The most characteristic change observed in all the cases was the increased melanin pigment in the intact basal cells (Figs. 1 & 2). The other important histopathological change observed in the lesional skin was liquefactive degeneration of the basal cell layer with pigment incontinence indicated by abundant melanophages in the upper dermis. A lymphocytic or lymphohistiocytic infiltrate was seen in the dermis. This was band-like in only 33% cases, while in the remaining cases it was diffuse and perivascular. In the epidermis, prickle cell layer atrophy and flattening of the rete ridges predominated over elongation and/or saw-toothing of the rete ridges. Hypergranulosis was not observed and only 4 (13.3%) cases showed hyperkeratosis.

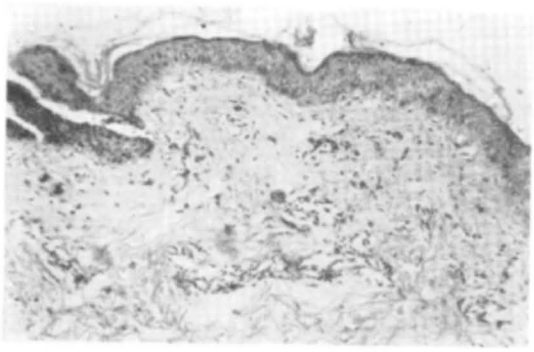

Fig. 1. Histopathology of lesional skin (H and E stain).

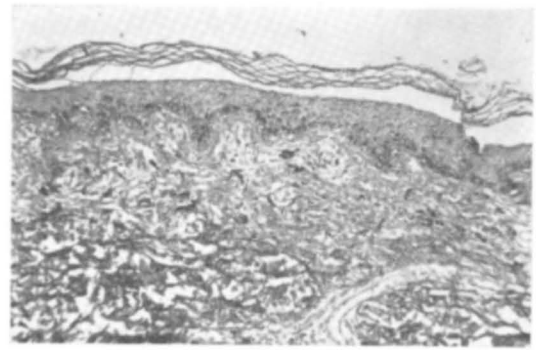

Fig. 2. Histopathology of lesional skin (AOG stain).

According to Lever,[2] the most important characteristics of lichen planus (LP) are : (a) hyperkeratosis, (b) focal hypergranulosis, (c) irregular acanthosis, (d) damage to the basal cell layer, and (e) a band-like dermal infiltrate in close approximation of the epidermis. In our series, hyperkratosis was seen in 13.3% cases, hypergranulosis in 3.3% cases, damage to basal cell layer in 66% cases and band-like infiltrate in the dermis in 33% cases. Thus, there is a little, if any, correlation between the histopathology of diffuse idiopathic melanoderma (DIM) and LP.

Histopathological findings in our cases also show certain definitive variations from the findings reported by Bhutani et al.[3] Thus,

presence of epidermal atrophy, absence of hypergranulosis and absence of colloid bodies observed by us was not reported by them. On the background of their findings they had coined the term lichen planus pigmentosus. The word lichen however generally implies a raised papular lesion. In addition there are many clinical and histopathological differences between DIM and LP. Hence, in our opinion, the term used by Bhutani et al,[3] appears to be inappropriate for cases of diffuse macular pigmentation. The term used by Shima,[4] Pigmentio macularis perstans or persistant macular pigmentation (PMP) appears more befitting.

Further, we observed histopathological changes similar to those in the lesional skin, in the normal-looking skin immediately adjacent to the lesions. This suggests that the normal-looking skin around the lesions does contain the disease process at a microscopic level. This may also explain the continuous peripheral spread of the melanoderma. To the best of our knowledge, LP like changes are not seen in the histopathology of the surrounding normal skin in LP.

Our findings are more akin to the histopathological findings in lichenoid tissue reaction (LTR) than LP. Weedon[5] lays emphasis on the basal cell degeneration, pigment incontinence and diffuse lymphohistiocytic infiltrate in LTR. These features were seen in majority of our cases. Therefore DIM appears to be a type of LTR with an additional finding of increased melanin pigment in the basal cell layer. We support the term persistant macular pigmentation due to lichenoid tissue reaction to cases of DIM.

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