STUDY OF HISTOPATHOLOGY AND MELANOGENIC ACTIVITY IN VITILIGO

KRISHNENDRA VERMA,* R. C. SARIN † AND B. R. PRABHAKAR ‡

Summary

In 30 cases skin biopsy from vitiliginous areas were studied for dopa reaction and in routine sections with H & E. Dopa reaction was completely absent in 24 cases. Epidermal changes were present in 23 cases, and dermal changes in 27 cases. Epidermal changes were hyperkeratosis, thinning of stratum malpighii, spongiosis and dissolution of the basal cell layer. Dermal changes were round cell infiltration in the upper dermis as well as hyalinisation, odema and fragmentation of collagen.

Recent renewed interest in the histopathology of vitiliginous skin has revealed many interesting features. In epidermis, besides absent or diminished dopa reaction,[1,2,3,4] thinning which could be correlated with the phase of the disease has been demonstrated[4]. In dermis in addition to scattered mononuclear infiltration[2,5,6,7,8,9], vesicle formation at dermo-epidermal junction[2] and mononuclear hugging at junction of pigmented and vitiliginous skin[9] have been reported. In this paper is presented results of histopathological study on 30 cases of vitiligo.

Material and Methods

Thirty untreated patients suffering from vitiligo were s lected at random from Skin Out-patient Department of Medical College / S. G. T. B. Hospital, Amritsar who were seen during the years 1977 and 1978. A detailed record on their history and clinical examination was made. Skin biopsy was taken in such a manner that a vitiliginous lesion with adjoining normal skin could be studied. The biopsy material was divided into two parts each of which contained a vitiliginous area and adjacent normal skin. One part of the material was subjected to dopa oxidase reaction as per technique described by Becker, Praver and Thatcher[10]. The other part was processed in routine manner and stained with haematoxylin and eosin.

Observations

(a) Epidermal changes

Dopa reaction was completely absent in vitiliginous areas in 24 cases and was present in low intensity in 6 cases. In one case activity in low intensity was present only at the junctional area between the vitiliginous and the normally pigmented skin.

In the hematoxylin and eosin stained sections lack of melanin pigment was apparent in the vitiliginous skin of all patients. In 17 cases the demarcation between normal pigmented and vitiligin-

* Post-graduate Student in Skin & S. T. D. Department.

† Professor & Head, Skin & S. T. D. Department.

‡ Professor & Head, Patholcgy Department.

Medical College/S.G.T.B. Hospital, Amritsar.

Received for publication on 22—2—1979.

ous areas was fairly sharp. Other epidermal changes were observed in 23 cases and these are shown in Table 1.

TABLE 1
Showing epidermal changes

Epidermal changes	No. of cases	Percentage
Hyperkeratosis	19	63.3
Keratotic plugging	4	13.3
Thinned stratum malpighii	18	60.0
Spongiosis	3	10.0
Dissolution of basal layer	3	10.0
No abnormality (Except lack of melanin)	7	23.3

Spongiosis was present in basal cells in one and in stratum malpighii cells in 2 cases and in one of the latter dissolution of basal cells was also seen. The other two cases with basal cell dissolution showed hyperkeratosis and thinning of stratum malpighii.

(b) Dermal changes :

Changes were observed mainly in the upper part in 27 cases in the vitiliginous areas as shown in Table 2.

TABLE 2
Showing various dermal changes

Change		No. of cases	Percentage
Round cell infiltration		26	86.7
Sub epidermal	20		
Perivascular	18		
Periadenaxal	8		
Collagen abnormalities		22	73.3
Collagen hyalinised/ oedematous	22		
Collagen fragmented	5		

Round cell infiltrate was present in upper dermis in small scattered groups of mononuclear cells mainly comprised of small lymphocytes. At places the infiltrates were seen hugging the epidermis. In two cases the infiltrate was seen breaking through the basal layer into the epidermis. The collagen fibres were hyalinised, loose or oedematous in 22 cases and fragmented in 5 of them.

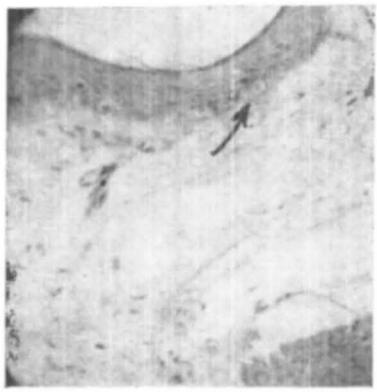

Fig 1 Showing loose dermal connective tissue, clear cell change and disappearance of some basal cells. Minimal round cell infiltration is also seen in the dermal connective tissue (H & E × 200)

Discussion

As has been reported by various workers[1,2,3,4] in this series too, majority of cases showed absence of dopa reaction in the vitiliginous skin. Few cases revealed mildly positive reaction. On H & E staining lack of pigmentation was seen in all cases and thinning

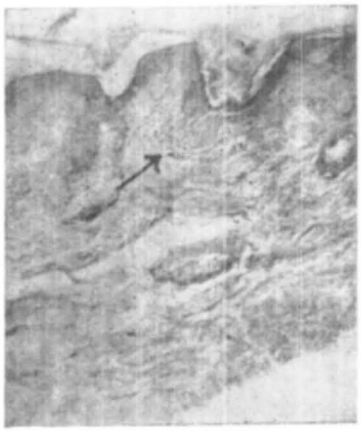

Fig.2 Showing scattered infiltrate at places hugging the basal layer of the epidermis. Collagen is hyalinised and fragmented. (H & E × 50)

of dermis specially of stratum malpighi in 60% cases. Whereas Gopinathan[2] did not observe any change in the thickness of epidermis, Behl and Pradhan[4] noted a relative thinning and flattening of the epidermis in vitiliginous areas. The latter workers further observed that with improvement of lesion the thickness of epidermis including the rete ridges tended to become normal.

Gopinathan[2] had observed small vesicles at the epidermodermal junction at the borders of some of the early vitiligo lesions. In the present series basal cell dissolution was present in 3 cases (10%).

In dermis in the upper part of the corium, cellular infiltrate consisting of small lymphocytes and histiocytes has been recorded by many earlier workers[2,5,6,7,8,9]. Behl and Pradhan[9] studied 48 cases histologically and detected at borders between vitiliginous and normal skin collection of these mononuclear cells at dermo-epidermal junction and at places in close proximity to basal layer and even at times intraepidermally. In the present series round cell infiltration in upper part of dermis was observed in 26 cases (86 7%). This infiltrate was seen in small groups and comprised of small lymphocytes and histiocytes. At places the infiltrate was observed to be hugging the epidermis. In two cases the infiltrate was seen to break through the basal layer into the epidermis. In addition, collagen was found to be oedematous, hyalinised or fragmented in 22 cases. In the absence of any history suggestive of application of any irritant or clinically detectable dermatitis, these findings, namely presence of infiltrate, changes in the collagen and in few cases dissolution of the basal cell layer, there is reason to suspect an immune mechanism operating probably in a very mild smouldering intensity perhaps comparable to mechanism operating in 'discoid lupus erythematosus'. Behl and Pradhan[9] had likewise concluded from findings in their cases that the inflammatory infiltrate indicated involvement of an immune process.

References

1. Jarrett A and Szabo: The pathological varieties of vitiligo and their response to treatment with meladinine, Br Derm, 68: 313, 1956.
2. Gopinathan T: A study of the lesions of vitiligo, Arch Derm, 91: 397, 1965.
3. Lever WF and Lever GS: Histopathology of the skin, JB Lippincott Co, Philadelphia Ed 5 p. 20, 1975.
4. Behl PN and Pradhan BK: Histomorphological spectrum of changes in three clinical stages of vitiligo, Active Quiescent and Improving, Ind J Derm Ven Lep, 43: 304, 1977.
5. Becker SW and Obermayer ME: A case for diagnosis (Vitiligo? Atrophic Lichen Planus?) Arch Derm Syph, 36: 216, 1937.
6. Buckly WR and Lobitz WC Jr: Vitiligo with raised inflammatory border, Arch Derm Syph, 67: 316, 1953.
7. Shukla RC: Vitiligo with raised border. J Ind Med Ass, 33: 16, 1959.
8. Dutta AK: Studies on vitiligo with special reference to neural concept, Ind J Derm, 43: 190, 1977.
9. Behl PN and Pradhan BK: Feature of mononuclear hugging in vitiligo, Ind J Derm Ven Lepr, 44: 66, 1978.
10. Becker, Praver and Thatcher (1935): Dopa oxidase reaction, cited by Culing C F A, Handbook of histopathological techniques, 2nd Ed, Butterworth, London, p 282, 1963.