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The Possible Site Of Trauma In Leucodermic/Vitiligenous Skin

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ABSTRACT

The histological, histochemical and ultrastructural changes in leucodermic skins are compared with those of normal human skins in order to find out if the former represents a traumatized skin. There were no significant differences in the histochemical features between the normal and leucodermic human skins. Ultrastructural studies however revealed that the melanocytes in the affected areas were undergoing degenerative changes such as loss of melanosomes, structural alterations in melanosomes and the presence of wide intracellular spaces. It is, therefore, suggested that in leucoderma, there occurs cellular traumatization rather than traumatisation at the organ level.

INTRODUCTION

Histologic response to repeated trauma of the skin includes increase in skin thickness, increase in stratum corneum layers, increase in the diameter of the hair follicles and an increase in the number of blood vessels and nerves[1]. Melanin is known to protect the skin from radiation damage[2,3]. However, it is not known whether its absence, as in the case of leucoderma/vitiligo, results in traumatization of the depigmented skin. Behl[4], on the other hand, suggests that trauma or local irritation may result in depigmentation of the normal skin. The present studies were therefore conducted in order to find out the site of trauma in leucodermic skin.

MATERIALS AND METHODS

A. *Histochemistry.* Leucodermic and corresponding normal human skins were obtained by a punch biospy (4 mm) after local anesthesia. They were immediately blotted free of blood and 10μ sections were cut on a freezing microtome. The sections were then stained for alkaline phosphatase activity[5], which selectively stains the blood vessels and capillaries in the skin[1].

B. *E. M. Studies.* The leucodermic and corresponding normal skins were obtained by a 4 mm punch biospy after local anesthesia from 4 individuals from the region of the forearm. The depigmented lesions, new and old (2,8,15 and 20 years of duration) were used for this study. All biospy materials were fixed in glutaraldehyde, post fixed in osmium

tetroxide and embedded in epon or epon-araldite mixtures. The sections were stained with uranyl acetate and Reynolds lead citrate and observed in Phillips 300 or AEI EM 6 electron microscopes.

RESULTS

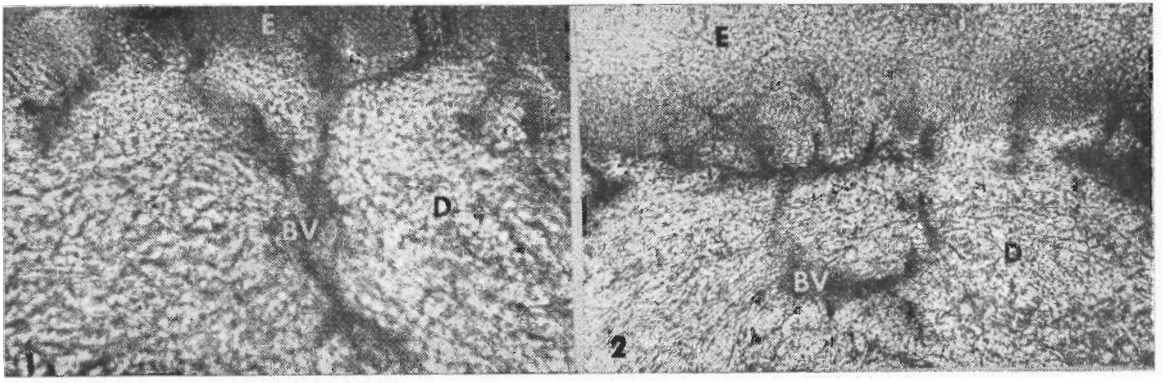
A. *Histochemical.* As can be seen from Figs. 1 to 4, the distribution of alkaline phosphatase activity was found to be similar in normal and leucodermic skins. Since the alkaline phosphatase activity localisation in the skins is known to be indicative of the distribution of blood vessels and capillaries[1] we may infer that the blood vascular system is similarly organized in normal and leucodermic skins.

B. *E. M. Studies.* The distribution of the melanosomes in the keratinocytes and melanocytes of the normal skin was essentially the same as described by earlier workers[7,8]. The melanocytes contained mostly stage III and stage IV melanosomes. In the keratinocytes the fully mature melanosomes were dispersed as discrete units (Fig. 5). The average long and transverse axis of the melanosomes were 0.73μ and 0.28μ respectively. In the 2 and 8 years old leucodermic lesions, however, a number of melanocytes undergoing degenerative changes were found (Figs. 6 to 7).These degenerative changes included the presence of wide intracellular spaces, a highly indented nucleus (Fig. 6), clumping of mitochondria and loss of cristae in mitochondria (Fig. 7). In the 15 and 20 year old lesions no regressing melanocytes could be observed.

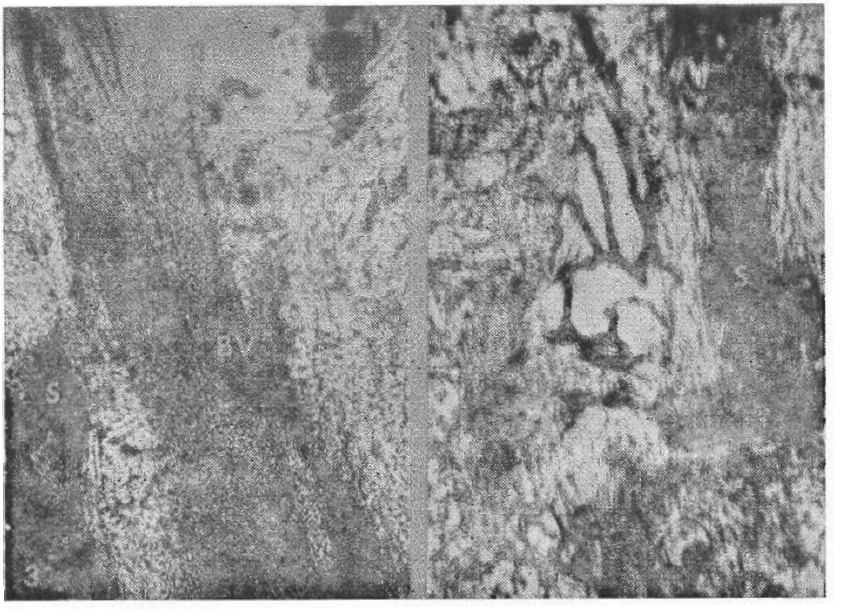
DISCUSSION

Routine histological observations of the leucodernic skins revealed that there were no anatomical changes in the skin. There was neither an increase in the diameter of the skin and hair follicles nor there was an increase in the stratum corneum layers. Since the alkaline phosphatase activity in leucodermic skin was same as in normal skin, leucodermic skin does not seem to represent traumatized skin[1,6].

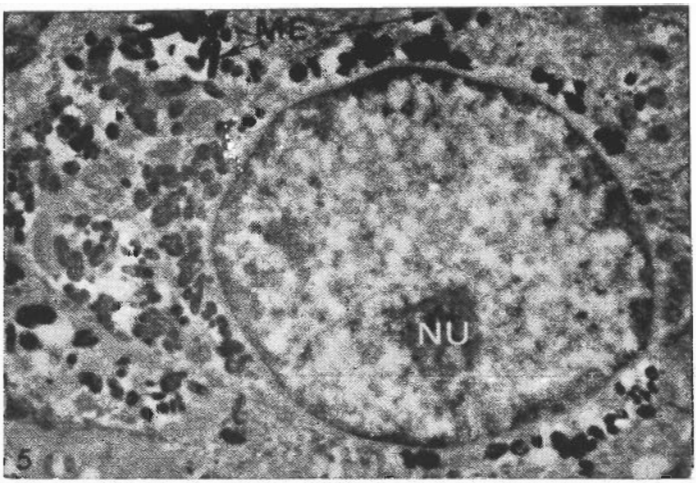
A number of biologically active phenols have been shown to be potent depigmenting agents[9]. Based on the hypothesis that vitiligo could be a phenomenon similar to chemically induced leucoderma a number of workers have tried to find out the mechanism of depigmentation of the skin by these compounds[8-10]. Chavin [11] has shown that neither the skin nor the melanocytes have any special affinity to hydroquinone, a potent depigmenting agent. Spleen, intestine, skeletal muscle, gall bladder, liver & kidney show almost the same affinity to this compound as the integument[11]. Electron microscopic observations of the phenol treated depigmenting skin have revealed that the melanocytes were undergoing netcrotic changes[12]. In the present study, similar degenerative changes in the melanocytes have been observed in the leucodermic lesions (Fig. 6 and 7). Since the leucodermic urine and blood manifests increased concentrations of phenols [13,14], it is suggested that the endogenous as well as exogenous phenols cause traumatic changes only in those cells which have a high capacity to oxidize phenols. Since only the melanocytes in the integument have a high capacity to oxidize phenols, the degenerative changes occur in these cells alone. It is, therefore, suggested that in leucoderma/vitiligo there occurs cellular traumatization rather than traumatization at organ level.



Figs. 1 and 2 : Normal and leucodermic human skins respectively showing the blood vessels and capillaries as revealed by alkaline phosphatase activity. Note the intense anastomorsis of the blood vessels × 150. BV = blood vessels, D = dermis, E = epidermis and S = sebaceous glands.



Figs. 3 and 4 : Showing the rich net work of blood vessels and capillaries in the hair follicle of normal and leucodermic skin respectively. The darkly stained body is the highly alkaline phosphatase positive sebaceous glands × 150. Abbreviations same as in Figs. 1 and 2.



Figs. 5 *to* 7 : Electron micrographs of normal and leucodermic skins to show the ultrastructural changes in depigmenting skins. ME - melanosomes, MS - Mitochondria, Nu - nucleolus, V - vaculole.

Fig. 5 : Normal human skin showing the distribution of melanosomes in the keratinocytes. Note that the melanosomes are distributed as discrete units. The nucleus shows a regular outline with electron dense chromatin present along the inner nuclear membrane × 7,700.

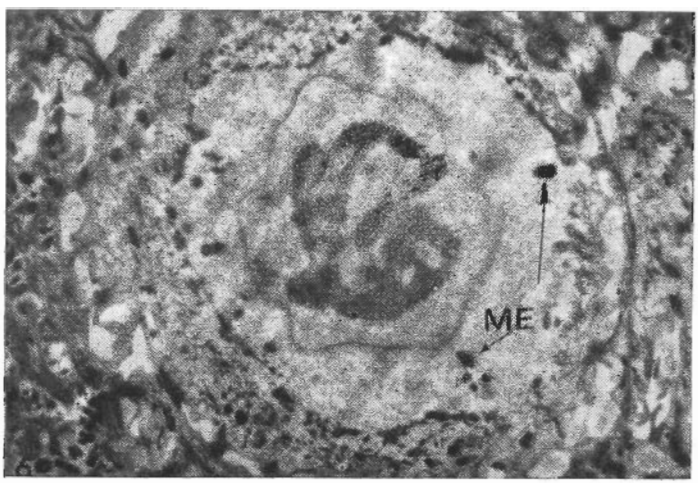


Fig. 6 : An∝ - dendritic cell in the basal layer of 11 years old leucodermic skin. The nucleus is highly indented and the electron dense chromatin is clumped in the centre. The cytoplasm appears almost empty and there are very few melanosomes × 7,700.

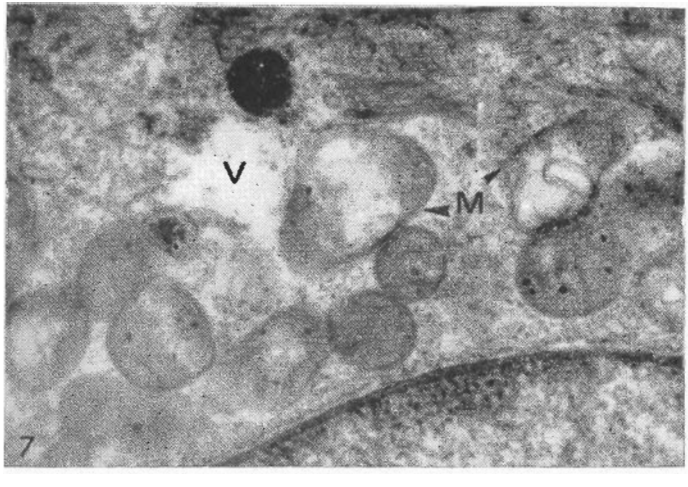


Fig. 7 : Higher magnification of an ∝ -dendritic cell from a 2year old leucodermic lesion showing the degenerative changes in the mitochondria and in general cell cytoplasm × 27,000.

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