ASSOCIATION OF PEMPHIGUS VULGARIS WITH VITILIGO

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A patient had vitiligo for 10 years with development of new lesions for the last four months. Concomitant with the recent activation of vitiligo, he developed pemphigus vulgaris. Corticosteroid therapy controlled pemphigus vulgaris, and also halted the development of new vitiligo lesions. Histopathologic study revealed inflammatory infiltrate at the dermo-epidermal junction along with oasal cell layer dissolution suggesting auto-immune origin cf vitiligo.

Key words : Pemphigus vulgaris, Vitiligo, Association.

Auto-immune hypothesis for vitiligo is primarily based upon the association of vitiligo with a number of other clinical conditions believed to be of auto-immune origin.[1] We observed a patient having vitiligo who also developed pemphigus vulgaris.

Case Report

A 42-year-old male was suffering from vitiliginous lesions for the last 10 years. A few new lesions had cropped up during the last 4 months. Concomitant with the activation of vitiligo, he developed painful erosive lesions in the mouth. At the time of admission, he was also having flaccid bullous lesions over the skin for about two months.

On examination there was extensive involvement of cutaneous surface with depigmented macules. Clear flaccid bullae intermingled with raw erosive lesions over the scalp, face, neck, axillae and groins. Mouth showed painful erosive lesions. Nikolsky's sign was positive. Smear showed acantholytic cells. Skin biopsy from an early vesicle revealed a suprabasal cavity filled with acantholytic cells. H-E stained section of the skin from a vitiliginous lesion revealed absence of melanin pigment in the epidermis. Inflammatory infiltrate comprising of lymphocytes and a few histiocytes was seen at the dermo-epidermal junction along with basal cell layer dissolution (Fig. 1). No dopa positive cells were observed in the vitiliginous areas. Corticosteroid therapy, comprising 80 mg of prednisolone initially, not only helped in controlling pemphigus vulgaris but also halted the development of new vitiligo lesions.

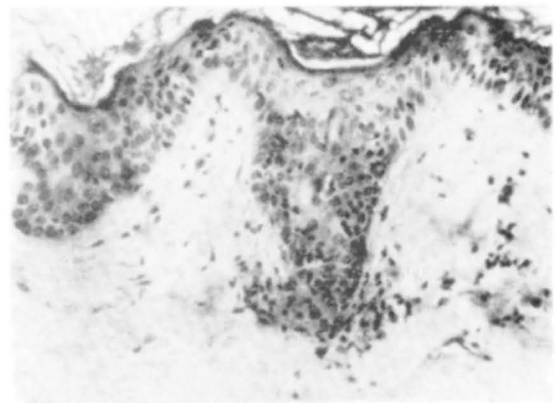

Fig. 1. Inflammatory infiltrate comprising of lymphocytes and histiocytes hugging the dermo-epidermal junction along with basal cell layer dissolution (H E x 150).

Comments

It has been suggested that some auto-immune mechanism causes destruction of melanocytes leading to vitiligo.[1] Increased association of thyroiditis,[2] pernicious anaemia,[3] Addison's disease,[4] diabetes mellitus,[5] myasthenia gravis,[6] and alopecia areata[7] has been

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documented in vitiligo patients. This hypothesis is further supported by the increased prevalence of organ-specific auto-antibodies in some of the patients having vitiligo.[8] Localised lymphyocytic infiltrate at the progressive edge of vitiligo, along with basal cell layer dissolution, also lend support to the auto-immune pathogenesis.[9] Activation of vitiligo in our patient, along with concomitant development of pemphigus vulgaris suggests that the association of these two diseases may be more than coincidental.

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