

A CASE OF PSORIASIS ON VITILIGINOUS AREAS

VİTİLİGOLU ALAN ÜZERİNDE GELİŞEN BİR PSÖRIAZİS OLGUSU

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SUMMARY: We present a case of psoriasis on vitiliginous areas. The loss of melanocytes on psoriatic lesions, any psoriatic lesions on normal skin in our patient with vitiligo, and the effectiveness of ultraviolet light on psoriasis indicated that melanocyte may play a role in epidermal regulation in psoriasis.

Key Words: Psoriasis, Vitiligo, Autoimmunity.

INTRODUCTION

Vitiligo is a disorder characterized by the loss of melanocytes from the epidermis, and is associated with autoimmune diseases such as thyroid disease, diabetes mellitus, alopecia areata, pernicious anemia, and autoimmune polyglandular syndrome. Vitiligo is related to organ-specific autoantibodies such as parietal, adrenal, and thyroid-(related) antibodies (1), and antimelanocyte antibodies such as antityrosinase, and antityrosinase related proteins 1 and 2 antibodies (2). These antibodies indicate vitiligo to be an autoimmune disease.

PATIENT

A 50-year-old caucasian man, with a 7-year history of vitiligo and a 5-year history of chronic psoriasis was given topical treatment. Psoriatic lesions appeared sharply demarcated in the vitiliginous skin. There was no history of trauma nor any family history of carrying hepatitis-B. His complete blood cell count and urine analysis

ÖZET: Biz, vitiligo alanı üzerinde gelişmiş bir psöriazis olgusu sunmaktayız. Psöriatik lezyonlarda melanosit yokluğu, vitiligolu alanlar dışındaki normal deride psöriatik lezyonların olmaması ve psöriaziste ultraviyolenin etkinliği; melanositlerin, psöriaziste epidermal regülasyonda rol oynayabileceğini düşündürmektedir.

Anahtar Kelimeler: Psöriazis; Vitiligo; Otoimmünite.

were normal or negative. The thyroid was palpable. Antithyroid peroxidase and thyroglobulin rates were high. Both lobes were heterogeneous and larger than normal on thyroid ultrasound. Based on these results we made a diagnosis of Hashimoto's thyroiditis.

On physical examination, large depigmented areas on the skin of the elbow, chin, hand dorsum and scrotum were observed (Fig. 1 and 2). Papulosquamous eruptions were confined to the nonpigmented areas and psoriatic lesions were sharply limited to the areas of vitiligo. Subungual hyperkeratosis and pitting on the nail plates were observed. He had no arthritis. The treatment given, oral PUVA therapy twice a week, is still being applied.

Histopathologic findings were consistent with psoriasis and vitiligo. There was no specific staining with S-100.



Fig. 1: Psoriasis and vitiliginous areas, on his arms.



Fig. 2: Psoriasis on vitiliginous areas, on his scrotum and glans penis.

DISCUSSION

Vitiligo is a depigmenting disorder due to the loss of melanocytes from the epidermis. Direct contact between cutaneous free nerve endings and epidermal melanocytes has been demonstrated in vitiligo. The discovery of a wide range of neuropeptides in the skin and the demonstration that some of them are able to regulate melanocyte differentiation like melanogenesis and dendricity have given more strength to the neural hypothesis. An increased immunoreactivity of neuropeptide Y (NPY) or an altered balance of nerve growth factor receptors and calcitonin gene-related peptide have been observed in vitiliginous skin (2). The association with autoimmune diseases such as Graves' disease, diabetes, pernicious anemia and alopecia

areata suggests a possible autoimmune cause (3). Immunologic phenomena offer some evidence in support of a relationship between vitiligo and psoriasis (4). El Mofty and El Mofty's (5) 22 patients had autoimmune vitiligo. Similarly there is also increasing evidence of a role of immunologic abnormalities in the pathogenesis of psoriasis (6). The demonstration of Ig G in the stratum corneum, Ig G and complement-bearing cells in the upper dermis, anti-Ig G activity on lymphocyte membranes, circulating immune complexes, increased levels of Ig G antiglobulins in patients with psoriatic arthritis, decreased numbers of circulating T cells, and decreased suppressor T cell activity in patients with psoriasis lend support to this theory (4).

Psoriasis has been associated with other autoimmune diseases such as arthritis and lupus erythematosus (7). Powell and Dicken reviewed the cases of 95 patients with both diseases seen at the Mayo Clinic. They noted no increased frequency of psoriasis or vitiligo in these patients but they did find a greater than expected incidence of autoimmune disorders and arthritis (12). Our patient had Hashimoto's thyroiditis. De Moragas noted a lack of melanin in lesional psoriasis. The resistance of psoriasis to the pigmented skin in vitiligo patients and therapy of psoriasis with ultraviolet light may indicate that the melanocyte is capable of epidermal regulation (8).

Some authors (10,11) have emphasized that because psoriasis started in areas of vitiligo or was confined to areas of vitiligo (8) the two diseases could be pathogenetically related or that vitiliginous skin is predisposed to developing psoriasis; however, Chapman's case report (9) seems to refute this idea. Menter et al. present a case in which there developed guttate psoriasis with lesions restricted solely to areas of vitiligo (13). However, our patient had psoriasis limited to areas of vitiligo. Recently, Julian and Bowers stated that the strict anatomical coexistence of vitiligo and psoriasis would suggest a common causal mechanism and a variety of theories have been postulated; but these remain speculative (14). There is no further information about this condition.

The incidence of vitiligo with psoriasis may be greater than is known in the literature. Although a variety of theories have been

postulated about anatomical coexistence, even more investigations may be necessary to determine the concurrence of vitiligo and psoriasis.

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