

# COINCIDENCE OF VITILIGO AND DIGITATE PARAPSORIASIS

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## ABSTRACT

A 73-year-old male patient with coexisting vitiligo and digitate parapsoriasis is described. Vitiligo was present for more than 40 years when parapsoriasis appeared. Immunohistochemical staining revealed in both biopsies taken from vitiliginous and parapsoriatic lesions multiple CD4+ lymphocytes in perivascular and periadnexal arrangement. A slight epidermotropism was also expressed. There were only a few CD8+ lymphocytes (without any epidermotropism) and no CD19+ cells. On the basis of their observation and the study of the literature the authors concluded that an etiologic association of these two conditions was probable.

## KEY WORDS

*vitiligo, digitate parapsoriasis, immunohistochemical staining, case report*

## CASE REPORT

73-year-old man with consistent vitiligo on his trunk, hands and feet since 1945 is presented. In 1990 he noted a nonitching exanthema on his trunk and upper arms which hardly responded to phototherapy.

On his first presentation in our clinic, we saw on the trunk and inner sides of the upper arms multiple brown-reddish macules in a digitate arrangement and with a pseudoatrophic surface.

Routine histological examination of a biopsy from one of the erythematous lesions on the trunk showed a perivascular lymphocytic infiltration in the upper dermis invading also the epidermis.

There were no significant histological findings in a biopsy taken from a vitiligo lesion on the trunk, despite a loss of melanin. In the H&E stain a very mild invasion of some lymphocytes into the epidermis was seen.

Immunohistochemical staining with the monoclonal antibodies recognizing CD4 (T helper cells), CD8 (T suppressor cells) and CD19 (B lymphocytes) in both biopsies (parapsoriasis and vitiligo) revealed multiple CD4+ lymphocytes perivascular and periadnexal with a slight epidermotropism, but there were only a few CD8+ lymphocytes (without any epidermotropism). No CD19+ cells could be detected.

*The paper is dedicated to professor Siegfried Borelli at the occasion of his 70<sup>th</sup> birthday*

In immunohistochemical staining we saw no differences between vitiligo area and parapsoriasis area.

## DISCUSSION

Vitiligo is an autoimmune disease with disturbance or destruction of melanocytes. The etiology is unknown but there is an association with other autoimmune diseases. Autoantibodies to normal melanocytes in the serum (1), an increased ratio of T helper to T suppressor cells with high levels of CD4+ lymphocytes (2,3) and an increase of T cytotoxic lymphocytes, T activated lymphocytes and B lymphocytes in peripheral blood of these patients were reported (4).

Immunohistological investigations of the inflammatory infiltrate in vitiligo-like depigmentation revealed a predominance of CD8+ cytotoxic/suppressor lymphocytes (5). A decrease of the CD45RA+ subset and an increase of the circulating HLA-DR+ cells, indicating the presence of activated peripheral T-cells was found (6). These reports suggest that cell-mediated immunity especially T-cell dysregulation may play a role in the pathogenesis of vitiligo.

Small plaque parapsoriasis (guttate parapsoriasis/digitate dermatosis) is a chronic inflammatory dermatosis characterized by a typical distribution, absence of symptoms and failure to respond to treatment. It is regarded to be one of the many clinical manifestations of mycosis fungoides (7). In the dermal infiltrate of atrophic (large-plaque) parapsoriasis an increased ratio of T-helper to T-suppressor cells, varying from 10:1 to 2:1 could be revealed (8). In mycosis fungoides and large-plaque parapsoriasis most T-cells express the CD4+ (helper/inducer) phenotype lack, Leu-8 antigen and often Leu-9 antigen (9).

The deficiency of Leu-8 positive cells seems not to be specific for mycosis fungoides and was also found in several reactive skin disorders (10).

The discordant expression of pan-T- and majority-T-cell antigens and T-cell subset antigens showing

the absence of antigen expression by epidermal, but not inflammatory skin lesions was pointed out as an important diagnostic feature (11).

Cutaneous T cell lymphoma, in whom the neoplastic cells expressed the CD8 suppressor T cell phenotype instead of the more common CD4 helper T cell phenotype can obviously be rapidly progressive (associated with a CD2- CD7+ phenotype) or chronic (associated with a CD2+ CD7- phenotype) (12).

The most CD8+ cells in CD3+ CD4+ mycosis fungoides skin are of T-cell rather than NK-cell differentiation lacking expression of cytotoxic and suppressor-associated markers (13).

Co-existing vitiligo and small-plaque parapsoriasis was previously reported in a 50-year-old woman (14). In a parapsoriasis patch within the vitiligo area of the abdomen a subepidermal cell infiltrate was seen showing about 90% CD3+, about 90% CD4+, about 25-30% CD8+ and about 20% CD1+ cells. Authors thought that there may exist an association between the two conditions (14). In our patient we also found an increased ratio of T helper to T suppressor cells in the dermal infiltrate of parapsoriasis and vitiligo lesions.

Hypopigmented mycosis fungoides in dark-skinned patients of African or Asian origin has been reported (15). Ultrastructural studies have shown degeneration of melanocytes within some of these lesions.

Langerhans cells seem to play an important role in the pathogenesis of vitiligo (16) and in early mycosis fungoides (17).

Our patient was of Caucasian origin and vitiligo developed a long time before parapsoriasis. So we don't think we have a case of hypopigmented mycosis fungoides. But we cannot exclude the possibility of vitiligo having promoted parapsoriasis based on a similar autoimmune disorder. Vitiligo is a relatively common disease with a reported prevalence between 0.5 and 1% in Europe, and small-plaque parapsoriasis isn't a very rare dermatosis, so a certain coincidence of both seems to be likely.

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