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Colocalization of hypertrophic lichen planus and vitiligo: Coincidental or causal

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ABSTRACT

Several patients with vitiligo have been reported to develop lichen planus (LP) only in longstanding vitiliginous patches. Here we present a rare case of simultaneous occurrence and colocalization of hypertrophic lichen planus (LP) with vitiligo. A 36-year-old male presented with a hyperpigmented hypertrophic plaque over right leg. He simultaneously noticed gradual depigmentation of medial part of the lesion and the adjacent skin along with leucotrichia. Histology showed features of a lichenoid interface dermatitis, favouring a diagnosis of LP. We believe that the association between LP and vitiligo is more than coincidental, and a causal link must be present.
KEYWORDS: *lichen planus, hyperpigmented, vitiligo, hypertrophic plaque and leucotrichia*

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INTRODUCTION

Few patients with vitiligo have been described to develop lichen planus (LP) only in the depigmented patches. Few case reports have been published in the literature describing patients with LP colocalized in vitiliginous skin[1-3]. Here, we report a case of simultaneous occurrence and colocalization of hypertrophic LP with vitiligo, which has not yet been reported in the literature to the best of our knowledge.Given that both vitiligo and LP are frequent, affecting between 1% and 2% of the general population, it's possible that their correlation is just a coincidence and has nothing to do with one another[4-5].

CASE REPORT

A 36-year-old male presented with an asymptomatic hyperpigmented raised lesion over right leg noticed 7 months back. The lesion was initially pea-sized and gradually increased to the present size over the next 3 months. The patient simultaneously noticed gradual depigmentation of medial part of the lesion and the adjacent skin. He was prescribed topical 0.05% Clobetasol Propionate for 3 months, which resulted in peripheral and perifollicular repigmentation. However, there was minimal improvement in the raised lesion. Past and family history was insignificant. On examination, a well-defined hyperpigmented hypertrophic plaque of size 3x3cm was present over the antero-medial aspect of right leg. Depigmentation of the medial part of the plaque as well as the adjacent skin along with leucotrichia was noticed, extending around 1.5 cm beyond the medial margin of the plaque. Perifollicular repigmentation was also seen. Rest of the cutaneous examination was normal. Examination of palms, soles, nails, mucosae was within normal limits. Histopathological examination of the hypertrophic plaque revealed hyperkeratosis, acanthosis, wedge-shaped hypergranulosis, saw-toothing of rete ridges, spongiosis and band-like lymphohistiocytic infiltrate obscuring the dermo-epidermal junction. Civate bodies were focally present and pigment incontinence was noted.

RESULT AND DISCUSSION

Colocalization of LP and vitiligo has infrequently been reported in the literature. As both disorders are reported to occur in 1–2% of the general population, this association may be merely a coincidence and may not represent any common underlying pathogenesis [6-7]. However, there is some evidence supporting the hypothesis that similar immunological mechanisms may be involved in the pathogenesis of both the diseases [8-9].

Several different patterns of association between LP and vitiligo have been recognised in the literature. LP lesions have been described as confined to vitiliginous skin alone or affecting both normal and

vitiliginous skin. In most cases, vitiligo is described as the antecedent disease. Sun exposed vitiliginous skin is seen to be affected more frequently as compared to sun-exposed normally pigmented skin and non-sun-exposed vitiliginous skin [10].

Colocalization of LP within vitiliginous patches has been explained by various theories. The actinic damage theory suggests that photodamage within the vitiliginous skin causes the release of inflammatory mediators, thereby promoting the accumulation of effector T cells as seen in LP. According to this theory, LP is more likely to be seen in sun-exposed vitiliginous skin. In contrast to this theory, few cases have been reported in which LP lesions occurred only on non-sun-exposed vitiliginous areas, such as scrotum, inguinal folds, and thighs [11-12]. Another popular theory is the koebnerization theory, which suggests that cellular injury in vitiliginous skin, augmented by the effects of actinic damage, modifies the immunopathogenic mechanisms that are involved in Koebner phenomenon, resulting in LP on sun-exposed vitiliginous skin.

Colocalization may develop since both vitiligo and LP can be triggered by local trauma. Veitch et al suggested Wolf's isotopic response for colocalization of these two conditions, in which one disease may trigger a second, pathogenically unrelated skin lesion [13-14]. Another theory is that long-standing vitiligo alters the expression of antigens identified by effector T cells in LP or inactivates suppressor T cells, thereby leading to the pathophenotype of LP. G¨oktay et al have suggested that tumor necrosis factor (TNF) may be an important mediator for colocalization of these two conditions as TNF-immunoreactivity has frequently been noted in LP and enhanced levels of TNF- production from melanocytes have also been recognised in patients with vitiligo.

However, many cases have been reported in the literature that cannot readily be explained by these theories. Baran et al. described lesions of LP, affecting only the normal skin around several vitiliginous patches in the shape of a rim with sharp borders [14-15]. Wayte and Wilkinson reported a case in which widespread LP affected normal skin and spared all the long-standing vitiliginous patches, with sharp borders separating the two types of lesions. In fact, they proposed that changes in vitiliginous skin protected against lichenoid transformation [16].

Most of the cases reported in literature describe occurrence of LP over longstanding patches of vitiligo. However, in our patients, there was simultaneous occurrence and colocalization of hypertrophic LP with vitiligo, which has not yet been reported in the literature. After a thorough review of literature, we conclude that the association between LP and vitiligo is more than coincidental, and a causal link must be present. Although the exact pathophysiologic mechanism is not yet well understood, better understanding of the interrelationship between LP and vitiligo may provide significant insight into the pathogenesis of both these conditions.

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