Implications of interleukin-17 in psoriatic lesions as Koebner phenomenon caused by recurrent occupational burns

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Abstract

The Koebner phenomenon (KP) is the emergence of new lesions in an uninvolved skin area caused by different types of stimulations, including mechanical stress, chemical stress, trauma, or injury. KP affects patients with certain skin diseases and is frequently observed in patients with psoriasis. We report the case of a 43-year-old obese male welder who developed psoriatic lesions only in areas of repeated burns due to his occupation. He was repeatedly exposed to mild burns in his anterior neck and the periorbital region as he was welding without shield protection. Subsequently, erythema appeared in the same region. Skin appearance and skin biopsy suggested psoriasis vulgaris (PV), and immunohistochemical analysis of anti-interleukin (IL)-17, a crucial element in the development of PV, showed the positive-stained cells. The anti-IL-17 staining was prominent around the thickened epidermis as psoriatic lesions. IL-17 produced by T helper 17 cells stimulates keratinized cells and promotes chemokine secretion involved in neutrophil migration. Our case showed that patients, even without a history of PV, may have a risk of developing KP and PV via the enhanced production of IL-17 locally in the repeated burn area. No recurrence of skin symptoms was observed when the patient used a fully defensive shield during welding.

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Introduction

Koebner phenomenon (KP) has been reported to occur in various diseases including psoriasis and some autoimmune vesicular diseases.[1,2] It has been described as the emergence of psoriatic lesions in uninvolved skin areas after trauma in approximately 25-30% of psoriatic patients.[1-3] Many triggers such as tattooing, radiation, viral infections, and surgical incisions have been reported to develop KP.[1] We report a case of a welder who developed psoriatic lesions only in areas repeatedly exposed to mild occupational burns.

Case Report

A 43-year-old obese male working as a welder, with a high body mass index of 26.5, without a history of psoriasis vulgaris (PV) has repeatedly suffered from erythema with scaling on his unshielded neck and face during welding for more than 10 years. Scale erythema was observed on bilateral eyebrows, superior eyelids, temples, and the right anterolateral region of the neck (Figure 1a and b). Skin biopsy from the erythematous lesion on his neck revealed hyperkeratosis and parakeratosis of the epidermis, regular elongation of epidermal rete ridge, thinning or loss of the granular layer, and intracorneal micro-abscesses (Figure 1c and d). The immunohistochemical analysis of the anti-interleukin (IL)-17 antibody showed the positive-stained cells similar to previous reports, that were prominent around the thickened epidermis (Figure 1e).[4,5] Notably, IL-17 produced by T helper 17 cells stimulates keratinized cells and promotes chemokine secretions involved in neutrophil migration.[4] The patient was diagnosed with PV by clinical manifestations and histopathological examination. With topical application of maxacalcitol and dexamethasone valerate, the erythema had improved by the 2-month follow-up point (Figure 1f and g). Using a fully defensive shield over the face during welding prevented relapse of the lesions, and the patient has been in remission for 19 months.

Discussion

This case developed psoriatic lesions selectively in areas of repeated minor occupational burns, suggesting that the burns may have caused KP and PV. Initially, KP was reported as the emergence of psoriatic lesions after skin trauma in the uninvolved skin of psoriatic patients.[1] Recently, the definition was extended to include lesions emerging after trauma in people without pre-existing skin disease.[2] KP is generally developed in 10 to 20 days after irritation, but some cases have been reported in a minimum of 3 days and a maximum of 2 years.[3]

Although the mechanism of KP has not been fully elucidated, its involvement in epidermal cell damage and skin inflammation has been suggested in the pathophysiology.[6] It has been reported that inflammatory mediators, including IL-17 produced by gamma-delta (γδ) T-cells, are involved in inducing the development of new psoriatic lesions.[2,7] Moreover, γδ T-cells are activated to enhance IL-17 production during the inflammation and the healing response after burn injury.[8,9] We think that changes in the cytokine milieu in the burn area may have triggered KP. Guttate psoriasis and pityriasis rubra pilaris were reportedly caused by KP in burn areas.[6,10] To clarify the mechanism of cytokine interactions related to KP in the burn area and the induced skin diseases, future accumulation of cases is required.

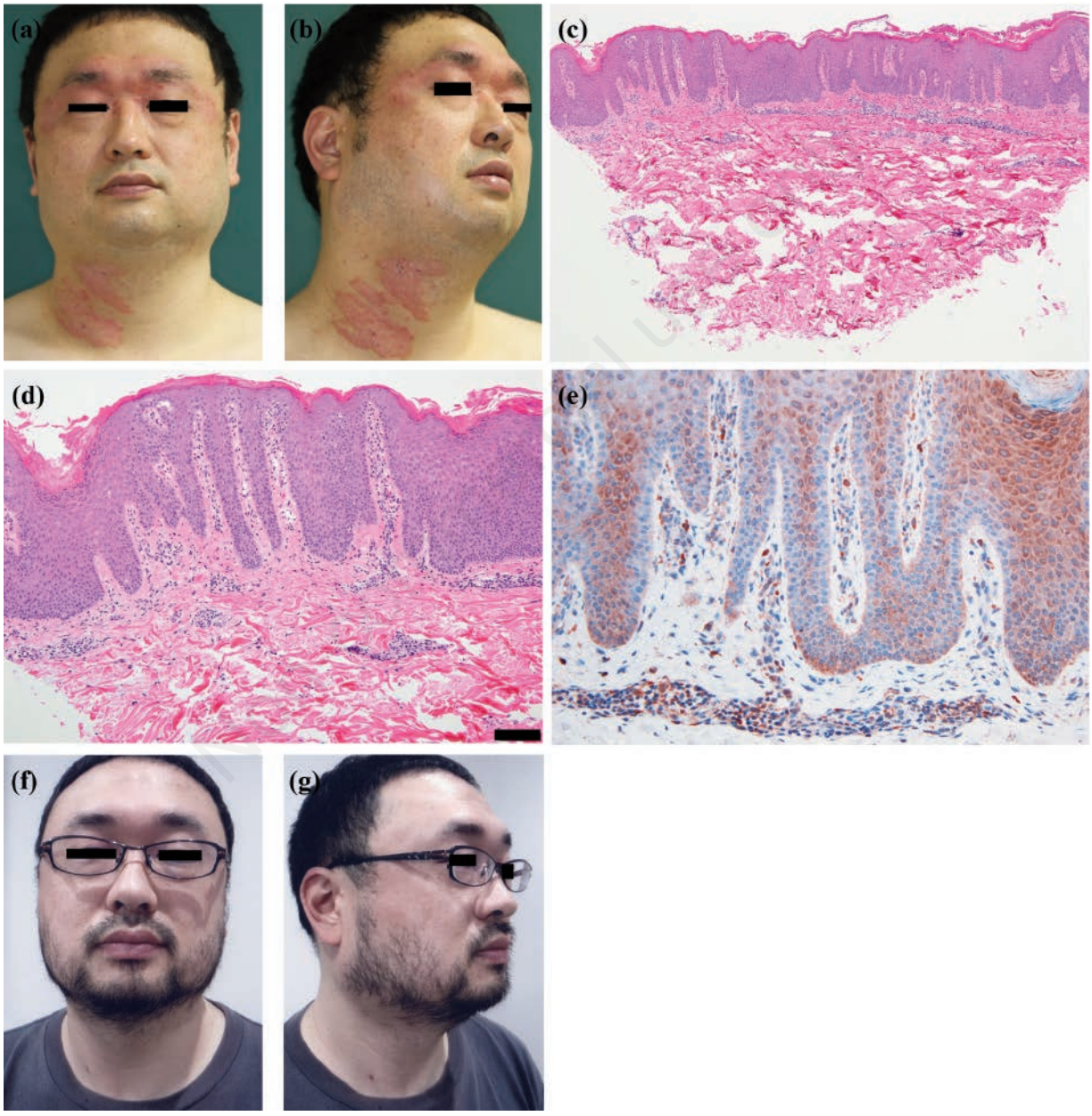

Figure 1. Clinical and histopathological features of the patient. a,b) Erythema on both upper eyelids, temples, and neck before the treatment; c,d) skin biopsy of the skin lesions on the patient's neck showing hyperkeratosis and parakeratosis of the epidermis, regular elongation of epidermal rete ridge, thinning of the granular layer, and intracorneal micro-abscess. Scale bar=100 μm; e) the immunohistochemical analysis of anti-interleukin (IL)-17; rabbit polyclonal to IL-17 (Abcam, ab79056), showed the positive-stained cells in the skin; f,g) improvement in skin lesions 2 months after the treatment.

Conclusions

Our case showed that the patient, even without a history of PV, may have a risk to develop PV by enhanced production of IL-17 locally in the repeated burn area. PV, a chronic inflammatory disease, is associated with complications such as obesity, diabetes mellitus, metabolic syndrome, hypertension, and dyslipidemia.[11] In this patient, obesity is the only risk factor for developing PV. We speculate that PV was caused by the changes in the cytokine milieu in the skin following repeated trauma in a patient with predisposing factors for PV, even without a history of psoriasis. This is a rare case of PV occurring only in specific areas of repeated mild burns.

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