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CLINICAL CASE REPORT

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Developing Shingles-Induced Koebner Phenomenon in a Patient With Psoriasis

A Case Report

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Abstract: Both shingles and psoriasis are common cutaneous diseases. About 25% of the psoriatic patients develop Koebner phenomenon (KP) after various injuries, and in rare instance, KP may occur at the site of healed or healing shingles.

We report a 30-year-old man with 7-month history of scalp psoriasis who developed KP at the areas of developing shingles. Cutaneous examination revealed scaly erythematous papules and plaques located on the scalp and forehead, and groups of clustered erythematous papules with silver scales in the dermatome distributed on the right side of chest wall the prior herpes zoster lesions involved. After removal of the scales on the papules, underlying bleeding points were present.

The lesions on chest had good response to anti-psoriatic therapies, as the lesions on scalp did. After a year of follow-up, recurrent psoriasis occurred, but the lesions were located only on the scalp, and the areas of prior occurrence of shingles, because of which we considered diagnosis of recurrent psoriasis rather than relapsing KP for the chest lesions.

Not only the healing and healed shingles can trigger KP in psoriasis, but also the developing shingles can cause psoriatic KP at the site of herpes zoster lesions.

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Abbreviations: HZ = herpes zoster, KP = Koebner phenomenon.

INTRODUCTION

Shingles, also called herpes zoster (HZ), is a common viral disease. Psoriasis is another common, chronic relapsing and remitting inflammatory disease that involves the skin and joints with an overall prevalence of 2% to 3% of the world's population.[1,2] Koebner phenomenon (KP), also called isomorphic response, is initially referred to the formation of psoriasiform lesions after cutaneous trauma on healthy skin areas of psoriatic patients, and now is extended to the instances that the people who had pre-existing dermatosis develop lesions after trauma or injury.[1–5] About 25% of the patients with psoriasis develop KP after various traumatic injuries.[1,2] Only a few of psoriatic KP following shingles have been reported in literature,[3,4,6–10] and the KP lesions always occur at healing or healed HZ eruptions with a latent period of 1 week[6] to 4 months[7] from the occurrence of shingles in this condition. Herein, we report a psoriatic patient in whom KP is occurring at the site of developing HZ lesions, and to our knowledge, no similar description has been reported before.

CASE PRESENTATION

A 30-year-old man was referred with 3-day history of clustered scaly papules on the right chest wall. The patient had episodes of groups of erythematous papules with shooting pain distributed on his right side of chest alone in the dermatome 9 days ago, which quickly turned into vesicles within 2 days. He was diagnosed as HZ and started on treatment of valaciclovir on the 4th day after the appearance of painful papules, resulting in rapid pain relief after being treated for 2 days. On the 6th day of the course of disease, the patient found that the vesicles began subsiding and became scaly papules rapidly in situ. No new areas were involved except the site of prior HZ lesions. He had no trauma or localized application of medications before the painful papules appeared. The patient had 7-month history of scalp psoriasis showing good response to topical application of calcipotriol ointment, but he used the medicine irregularly resulting in recurrent lesions sometimes. He never had KP lesions, and any other associations, since the onset of psoriasis. His family history was also unremarkable.

Cutaneous examination revealed that scaly erythematous papules and plaques located on the scalp and forehead (Figure 1A), and groups of clustered erythematous papules with silver scales in the dermatome distributed on the right side of chest wall where the prior HZ lesions occurred (Figure 1B, C). After removal of the scales on papules, underlying bleeding points were present. No other kinds of lesions were observed elsewhere including limbs, nails, and the trunk except the site where the prior HZ eruptions involved. The patient was diagnosed as KP of psoriasis, and was treated with topical application of clobetasol propionate and calcipotriol compounds, which resulted in excellent response after a week of treatment (Figure 1D). On 1 year of follow-up, the patient had occasional recurrence of psoriasis when the treatment was stopped; no new zosteriform eruptions reoccurred. Interestingly, the recurrent lesions occurred only on the scalp and

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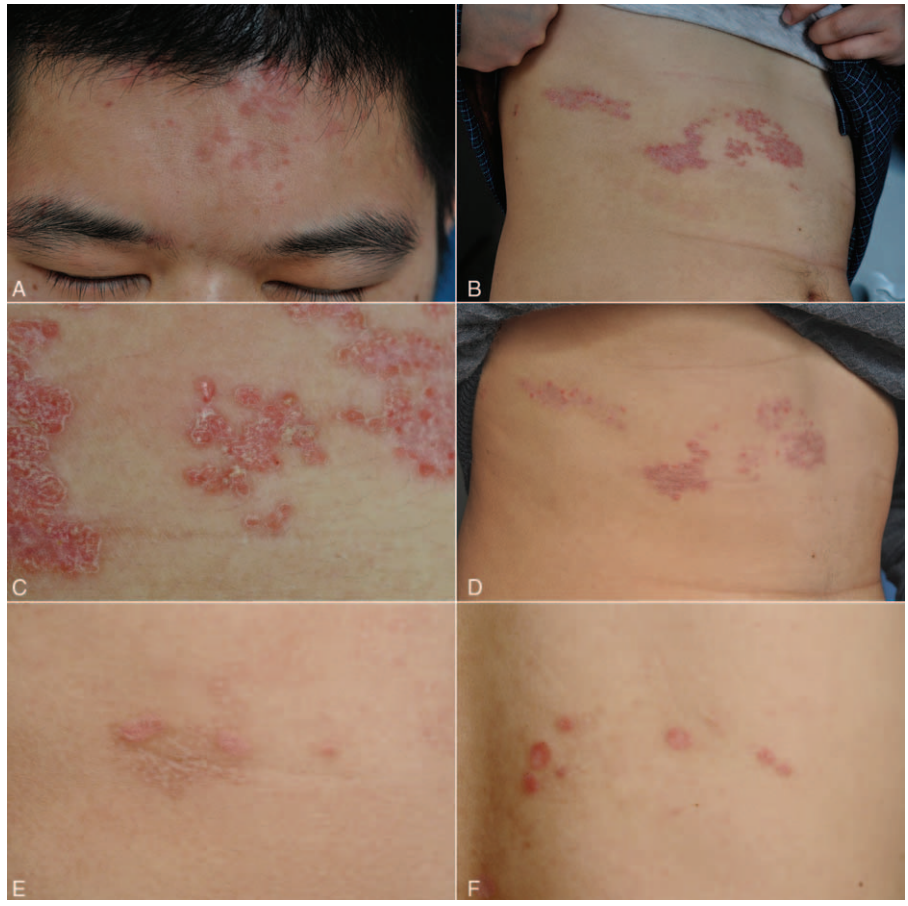

FIGURE 1. (A) Scaly erythema localized on the scalp and forehead. (B, C) Groups of clustered papules with silver scales in the dermatome distributed on the right side of chest wall where the previous developing HZ involved. (D) The scaly papules relived markedly after 1 week of topical application of clobetasol propionate and calcipotriol compounds. (E,F) The recurrent psoriatic lesions on the areas of the previous shingle on 7 months of follow-up.

the areas of prior shingles each time (Figure 1E,F). Histology from thoracic lesion showed regular epidermal hyperplasia with test-tube-shaped rete ridges, thinning over some derma papilae, acanthosis, parakeratosis, and lack of granular layer (Figure 2).

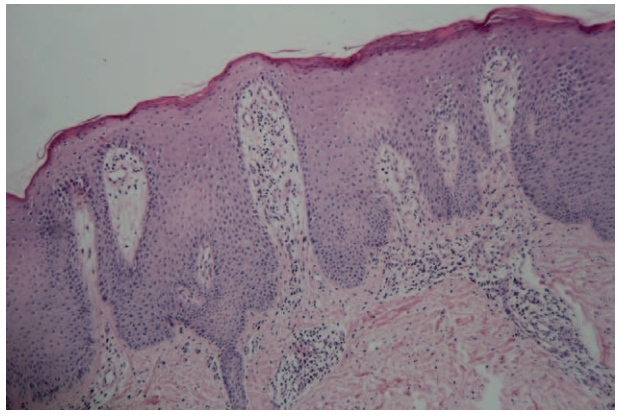

FIGURE 2. Histology shows regular epidermal hyperplasia with test-tube-shaped rete ridges, thinning over some derma papilae, acanthosis, parakeratosis, and lack of granular layer.

DISCUSSION

The present patient had groups of painful papules and vesicles distributed in the dermatome on unilateral chest wall; HZ can be diagnosed clinically although without laboratory support. Generally, the natural history of HZ is that its initial lesions appear as erythematous papules, which turn into vesicle within 12 to 24 hours. New lesions may appear within 3 to 7 days. The vesicles of HZ progress into pustules in about 3 days and form scabs over the next 7 to 10 days.[11,12] The inflammatory lesions within the involved dermatome after HZ have been rarely described, but the lesions are present as flat topped lesions or annular papules, and usually appear within a month with the absence of scales. It was found in the present patient that his blisters became scaly papules on the 6th day during the course; we considered that the scaly papules formed during developing stage of HZ rather than recovering time. Based on the psoriasis history and the present clinical features: the developing painful papules and vesicles became scaly papules with underlying bleeding points, which showed excellent response to topical application of clobetasol propionate and calcipotriol compounds, we considered that the present scaly papules are KP of psoriasis subsequent to developing HZ rather than to healing HZ lesions themselves, although slight scarring may occur in patient with HZ at the site the blisters have been.

The diseases tending to koebnerization include psoriasis, vitiligo, lichen planus, Darier disease, bullous dermatoses, and so on.[1–5] The provoking factors for KP include trauma, burns, friction, insect bites, surgical incision, as well as dermatoses and therapeutics, such as HZ, syphilis, and ultraviolet B treatment,[1–5] and even the cupping therapy, a kind of traditional Chinese medicine.[13] Season, disease severity, trauma on skin, scar tissue, and emotional stress can also trigger KP in patient with psoriasis.[2] KP can develop in any anatomic site.[2] The duration from injury to occurrence of KP may range from 3 days to years, but in general, it is between 10 and 20 days.[2] Only a few cases have described their KP lesions of psoriasis at the site of healed or healing HZ as well as varicella involved.[3,4,6–10] In the present patient, KP eruptions occurred at the areas of developing HZ lesions with only 6-day interval from the painful chest and 4 days from the appearance of vesicles, which, to our knowledge, has never been reported before. Other interesting entity is that, on a year of follow-up, the recurrent psoriatic lesions located only on the scalp and the healed HZ areas. As the recurrent thoracic lesions occurred synchronously with the scalp's each time, we considered them to be relapsing psoriasis rather than recurrently psoriatic KP on healed HZ. However, the reason why the recurrent psoriasis was localized to the areas mentioned above was unknown.

Although KP is a common condition, its pathogenesis is not fully understood yet, but may involve cytokines, stress proteins, adhesion molecules, and autoantigens.[2] It is considered that nerve growth factor, tumor necrosis factor-α, substance P as well as interleukin-1 play important roles in the pathomechanism of koebnerization in psoriasis.[2–5] The occurrence of psoriatic koebnerization at the site of shingles and varicella was considered to be mediated by neuropeptides such as substance P.[2] Substance P, an endogenous neuropeptide being potentiated by viral infection and acting as a chemomediator of nociceptive impulse from periphery to central nervous system, plays an important role in the HZ-associated pain. Viral infection can potentiate the effect of substance P by decreasing the degradation of its breakdown enzyme. Substance P has also been demonstrated to be increased in psoriatic lesions.[14] We speculate that substance P may play an important role in the present KP, although the exact mechanisms remain unknown. Interestingly, we soon met another typical shingles patient with prior severe psoriatic lesions over his whole body, but neither his prior shingles involved areas nor other areas occurred KP during 6 months of follow-up. It suggests that KP occurs at the site of HZ lesions in not all the psoriatic patients with shingles. As a matter of fact, although both psoriasis and shingles are common diseases, few of psoriatic KP subsequent to HZ have been reported. It is still unclear why such a presentation does not occur more often.

Koebner lesions should be treated in the same way as the associated dermatosis,[15] as our present patient did. The present patient was notable because the psoriatic KP lesions occurred at the site of developing HZ, and the recurrent psoriasis localized only on the primarily areas and healed shingles areas.

CONCLUSIONS

Both psoriasis and shingles are common cutaneous diseases. About 25% of the patients with psoriasis develop KP after various traumatic injuries. Not only healing and healed shingles can cause KP in psoriasis, but also developing shingles can cause such a condition at the site of HZ lesions. The therapies for psoriasis can also be used to treat Koebner lesions of psoriasis.

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