Prurigo Pigmentosa Caused by Sweating

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Prurigo pigmentosa is a peculiar pruritic pigmented dermatosis characterized by the sudden appearance of reddish papules accompanied by severe pruritus. The etiology still remains unknown but environmental factors may play a role, in which physical trauma or friction from wet clothing induces the skin lesions. It has a seasonal preference for spring to summer when there is increased sweating. We report a case of prurigo pigmentosa which may be triggered by sweating and cured with minocycline.(Ann Dermatol 13(3) 167~170, 2001).

Key Words : Prurigo pigmentosa, Sweating

Prurigo pigmentosa(PP) is an inflammatory dermatosis of distinct entity first described by Nagashima et al.in 1971[1]. It is a recurrent pruritic eruption characterized by erythematous papules that coalesce to form a reticulated, mottled pattern. Although more than 300 cases have been reported in Japan[2], it seems to be rare elsewhere[4]. In Korea, 10 cases[3] have been reported and all cases were treated with dapsone. Although systemic conditions, such as diabetes mellitus, pregnancy, fasting, and dieting, were associated with prurigo pigmentosa, suggesting involvement of endogenous factors in pathogenesis, exogenous factors, such as friction from clothing, have been suggested as possible triggers. Herein we report a case of prurigo pigmentosa that occurred in winter after sweating. We consider sweating may be a triggering factor in prurigo pigmentosa.

CASE REPORT

A 20-year-old woman presented with reticulate erythematous papules and plaques with scattered focuses of brown pigmentation on her back for 3 years of duration. She complained of severe itching and recurrence of the same lesion[5]. The lesions appeared suddenly in summer and disappeared 3 months later spontaneously without leaving pigmentation but recurred in the next summer. At this time, the lesion occurred in winter for the first time and had not subside during the last 10 months. Therapy with systemic and topical steroid in another hospital improved the itching but not the skin lesion[5]. She claimed that the lesion occurred after she was exposed to a new working environment where it was hot and induced heavy sweating.

On inspection, there were patchy or gross reticular brown pigmentation with clusters of miliary sized papules around the pigmented area forming edematous erythematous plaques(Fig. 1 and 2). Her family and medical history were non-contributory. The patient was not taking any systemic medications at the time of onset of the eruption and denied exposure to any industrial chemicals. But she said that she had a bad habit of skipping meals after leaving home to work. Routine laboratory evaluation revealed no abnormalities and a KOH smear of the upper back was also negative for fungus. Topical application of corticosteroids and oral antihistamine for 1 week was done for a preliminary diagnosis of a variant of confluent and reticulate papillomatosis(CRP) and xerotic eczema with no appreciable benefits.

Biopsy specimens showed parakeratosis, spongiform edema, scattered eosinophilic apoptotic ker-

Received October 2, 2000.
Accepted for publication May 4, 2001.
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Annals of Dermatology
Vol. 13, No. 3, July 2001

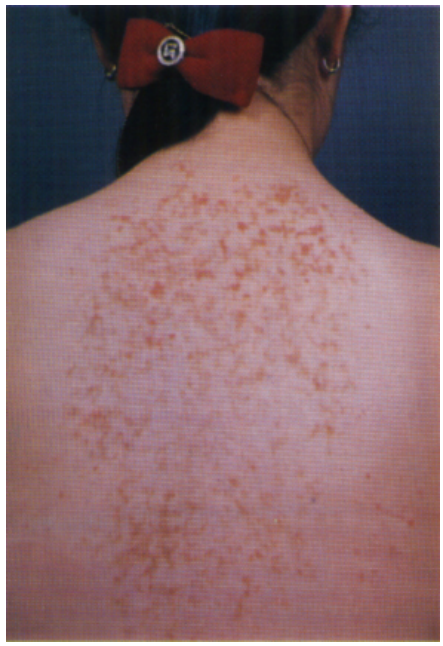

Fig. 1. The lesions were distributed on the back.

atinocytes and exocytosis in the epidermis, damage of the basal cell layer, and perivascular lymphohistiocytic infiltration in the dermis(Fig. 3). Oral Acyclovir 125mg bid for 5 days for a presumed diagnosis of erythema multiforme also failed to respond. The skin lesions were more aggravated. After review of literatures, we considered the possibility of prurigo pigmentosa and started minocyclin 200mg daily for 7days and resulted in improvements of erythematous skin eruptions leaving brownish pigmentations.

DISCUSSION

This case was diagnosed as prurigo pigmentosa based on the following findings; 1) the characteristic skin rashes; 2) biopsy results showing the nonspecific, but consistent findings; 3) dramatic improvement with minocycline therapy.

Prurigo pigmentosa(PP) is a recurrent pruritic eruption characterized by sudden onset of erythematous papules that coalesce to form a reticulated, mottled pattern. They may evolve into urticarial plaques and may have scales, but no formation of vesicles or bullae is generally found except a few cases of bullous PP[5] associated with diabetes mellitus. The rashes are usually symmetrically distributed on the trunk and nape[6]; however, there are reports of other locations on the breast, limbs, abdomen, antecubital fossa, lumbosacral region, face or forehead[7]. Coalesced red papules disappear in days or a week leaving a mottled, reticulated hyperpigmentation with a characteristic marble-like appearance. The lesions usually occur in summer and are more common in young females, particularly in adolescence, but may occasionally develop in males or older ages. The total course may be 6 months to 8 years[6]. Clinically PP may be confused with lichen pigmentosus, prurigo melanotica, pigmented contact dermatitis, pruritic chronic multiformis, confluent and reticulated papillomatosis, urticaria pigmentosa or erythema dyschromicum perstans[6]. Our case manifests the characteristics of PP (young female, sudden onset, recurrence, reticulated pattern, marked itching). But the cause of

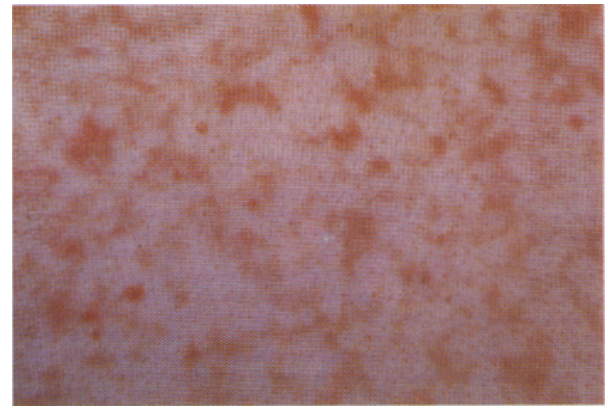

Fig. 2. A closer view reveals the intermingling of erythematous papules and reticulate hyperpigmentation.

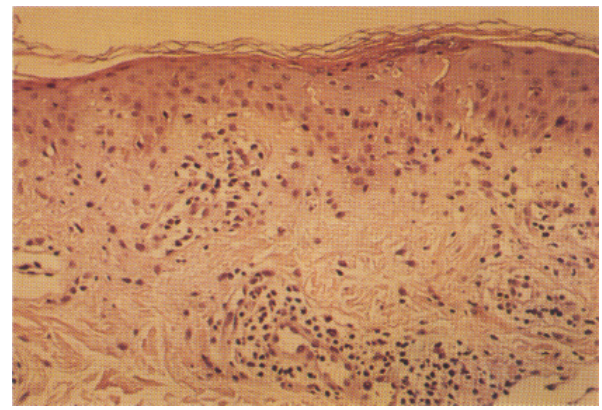

Fig. 3. Skin biopsy of lesion site(H&E stain, ×100): a superficial perivenular lymphocytic infiltration with multifocal basal cell damages and exocytosis.

recurrence during winter presumed that she works in a high temperature environment.

The cause of PP is unknown. Nagashima[6] speculated that some environmental contamination might play a role, more specifically physical trauma or friction from the clothing. Contact allergic reaction to p-amino compounds[7], similar in chemical structure to sulfonamides, have also been suspected. Other possible causes include application of trichlorophenol on the skin[8], intake of a bismuth-subsalicylate containing antacid[9], and the use of acupuncture[10]. Miyachi et al[11] described that oxygen intermediates produced by infiltrated cells are involved in the pathogenesis of the lichenoid tissue reaction of PP. Recently some reports have described PP associated with ketosis, fasting, dieting and insulin-dependent diabetes mellitus[13-15]. They speculated that surplus ketone bodies may remain around the blood vessels and give rise to perivascular inflammatory reaction and these inflammatory reactions may be a trigger in the pathogenesis[15]. In our case, no exogenous cause could be identified except that sweating could be a triggering factor. Also we tried to reveal endogenous factor, such as relationship between skipping a meal and skin lesion, but failed.

Histopathologically PP displays a lichenoid tissue reaction. Other characteristics of the papular lesion are elongation of rete ridges, intercellular and intracellular edema, exocytosis, basal cell liquefaction degeneration, papillary dermal edema, superficial blood vessel dilatation, and a mild perivascular lymphohistiocytic infiltration. Electronmicroscopic observation shows injury of the basal cells and severe damage of the lower epidermal cells suggesting that prurigo pigmentosa is a tissue reaction similar to lichen planus[16]. Teraki et al[17] found by immunohistochemical studies that intercellular adhesion molecule 1(ICAM-1)and HLA-DR were intensely expressed by keratinocytes in erythematous papules and focal prolonged expression of ICAM-1 and HLA-DR were observed in pigmented areas. CD4+ cells predominated in the dermal infiltrate, while CD8+ cells adhered to the epidermis much like a lichenoid tissue reaction in dermatoses such as fixed drug eruption, erythema multiforme and graft-versus-host disease. The persistence of expression of ICAM-1 might be due to continuing secretion of cytokines by lymphocytes or keratinocytes and could explain the recurrent rashes that are localized to these sites[17].

Although dapsone and sulphamethoxazole are effective for prurigo pigmentosa, minocycline is of help in the treatment of prurigo pigmentosa and safer than dapsone and sulphamethoxazole[18,19]. In comparison, the response to topical or oral corticosteroid or oral antihistamine is poor. Dapsone or sulfonamides might exert its anti-inflammatory effects by suppressing oxygen intermediates induced by an unidentified allergen, which ultimately protects the tissue from injury, by the hydroxyl radicals[3]. Minocycline is a semi-synthetic tetracycline known to have anti-inflammatory properties and inhibit neutrophil chemotaxis and mitogenic response of lymphocytes. Minocycline may have similar anti-inflammatory effect to dapsone; i.e. an inhibition of oxygen intermediates in inflammation[18].

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