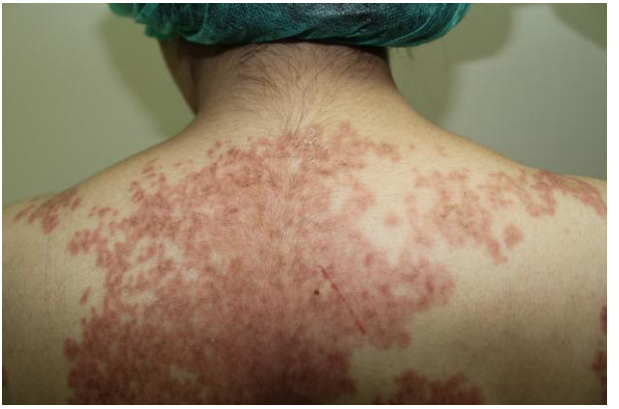
Case 17.1
A 23-year-old Thai female from Bangkok.
Chief complaint: Pruritic erythematous rash on trunk for 2 weeks.



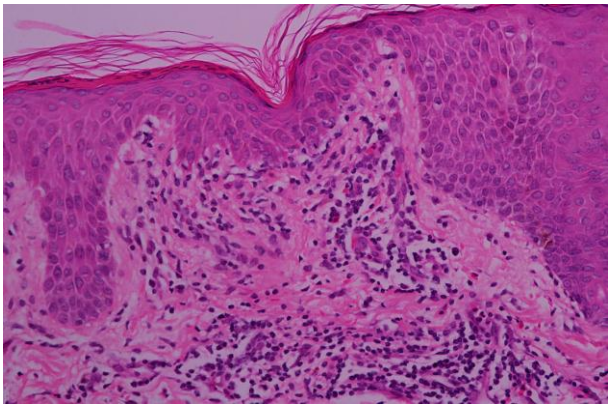
Present illness: The patient developed pruritic edematous erythematous reticulated plaques on her back, chest and pubic area for 2 weeks. The lesions resolved in small area leaving a net-like hyperpigmentation.The patient had been treated as eczema with oral anti-histamine with little improvement.

Past history
She has no underlying disease.

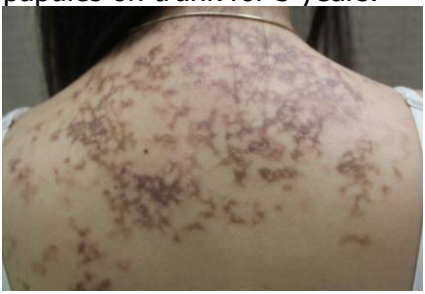
Family history
None of her family member had history of similar skin lesion.

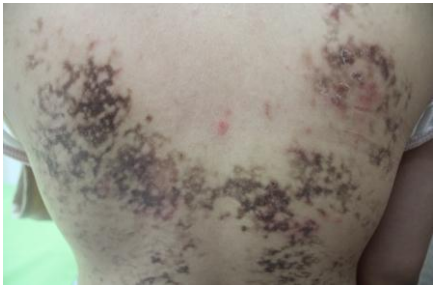
Skin examination
Multiple discrete erythematous, edematous, reticulated papules and plaques on chest, back and pubic area. The lesions healed in some area and leaving the background of reticulated hyperpigmentation

Histopathology (S13-495A, abdomen)
There are mound of parakeratosis, and epidermal hyperplasia in association with superficial and deep perivascular and focal lichenoid infiltration with lymphocytes, melanophages admixed with some neutrophils.



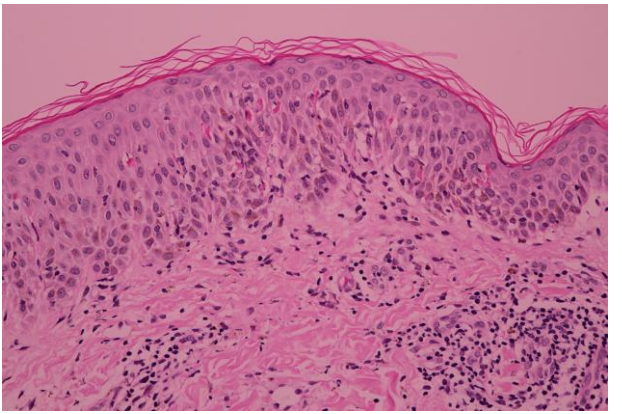
Case 17.2
A 21-year-old Thai female from Rayong province
Chief complaint: Recurrent intensely pruritic erythematous papules on trunk for 3 years.





Present illness: The patient developed recurrent severely pruritic erythematous papules on her back, chest, abdomen for 3 years. Most of lesions resolved spontaneously and leaving reticulated hyperpigmentation. The lesions were precipitated by seafood diet. The patient had been treated as eczema and post-inflammatory hyperpigmentation with oral anti-histamine, topical and systemic corticosteroid with some improvement but the lesions still recur.

Past history
She has no underlying disease and does not take any oral and topical medication prior to the eruption of skin lesions.
Family history
None of her family member had history of similar skin lesion.
Skin examination
Multiple reticulated brownish patches with some lesions shows erythematous papules on back, chest, abdomen.
Histopathology (S13-14778A, back)
There is superficial perivascular and lichenoid inflammatory-cell infiltration of lymphocytes admixed with some melanophages in association with epidermal hyperplasia and scattered necrotic keratinocytes.



Diagnosis: Prurigo pigmentosa
Treatment: Doxycycline 100 mg twice per day
Fexofenadine 60 mg twice per day

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Discussion

Prurigo pigmentosa (PP) is a recurrent inflammatory dermatosis present with severe pruritus. It was first described in 1971 by a Japanese dermatologist, Masaji Nagashima, and most patients diagnosed as PP have been reported in Japan to date.

PP usually presents in late teen to early twenties and most often appears in spring and summer. The clinical features include symmetrical and intensely pruritic erythematous urticarial papules in early lesions, with fully developed lesions turn to erythematous papules, papulovesicles and may confluent into reticulated plaques with a preferentially on trunk, back, chest, neck, shoulder, lumbosacral and abdomen[1]. The individual lesions rapidly change within days and can subside spontaneously within 1 week leaving reticulated hyperpigmented macules and patches. Pruritus is severe in early lesion but resolving lesion tended to be lesser. However, bullous PP is rarely reported[2-4].

Histopathologic findings are quite vary at the different stages. In early stage, there is superficial perivascular infiltration of neutrophils followed by scattering of neutrophils in papillary dermis. The neutrophils can pass quickly through the epidermis resulting in spongiosis, necrolytic keratinocytes and ballooning. While in fully developed lesions, lymphocytes and eosinophils predominated over neutrophils appear both in dermis which lead to patchy lichenoid pattern. Epidermis finally turn to hyperplastic and hyperpigmented. There are melanophages in papillary dermis resulting in residual hyperpigmentation in final stage of disease. However, histopathologic finding in PP is still vary and non-specific. The diagnosis of PP requires clinicopathological correlation based on typical features of PP.

The exact causes of PP and its pathogenesis are unclear. However, several factors have been suggested include ketosis[5] causing from diet, fasting (anorexia nervosa)[6], diabetes mellitus[4] and soft-drink ketosis[7], pregnancy[8], menstruation, atopic diathesis[9],

sweating, friction from clothing, Helicobacter pylori infection, and chemical allergens such as para-amino compounds, trichlorophenol, nickel[10] and chrome[11]. Some authors suggested that autoimmune also play role in PP. However, recent studies reported the relationship between PP and diet modification with recurrence of PP after restarting dietary modification.

PP should be differentiated from dermatitis herpetiformis, linear IgA bullous dermatosis, acute lupus erythematosus in early disease. While late lesions should be differentiated from confluent and reticulated papillomatosis of Gougerot and Carteaud, dyschromicum perstans, and pigmented contact dermatitis.

For the treatment, various agents were reported for treatment of PP. The most effective are oral antibiotics such as minocycline[9, 12], doxycycline[13], sulfamethoxazole, dapsone, macrolides[14] and oral retinoid[15], chemical peeling combined with light emitting diode(LED)[16] have been reported as an effective treatment for PP. While topical corticosteroids, oral steroids, antihistamine are ineffective in treating PP.

Recurrence of lesions usually developed at the same sites of primary lesions within 3-9 years. Both minocycline and doxycycline cannot prevent the recurrence of PP[1].

In our patients, we presented 2 cases of PP who have classic characteristic of PP. The first case demonstrated early disease who was treated with doxycyline since early stage of disease. After 6-month follow-up in this case has shown that patients developed minimal hyperpigmentation. This finding suggests that early treatment may have benefit by ceasing the inflammatory process resulting in reduce sequela of post-inflammatory hyperpigmentation.

References

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