

CASE REPORT

A Case of Post-zoster Eosinophilic Dermatitis

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Post-zoster eosinophilic dermatitis is a rare disease that occurs as an isotopic response in the region of previously healed herpes zoster. We report here on a case of post-zoster eosinophilic dermatitis that occurred 13 years after an episode of herpes zoster. A 48-year-old woman presented with several pruritic, brown papules and plaques in the same dermatome where her previous episode of herpes zoster had healed. Histopathologically, there was a dermal inflammatory cell infiltration composed of abundant eosinophils, lymphocytes and histiocytes. Based on these clinicopathologic findings, the patient was diagnosed with post-zoster eosinophilic dermatitis. (*Ann Dermatol* 21(3) 274~276, 2009)

-Keywords-

Herpes zoster, Isotopic response, Post-zoster eosinophilic dermatitis

INTRODUCTION

Post-zoster eosinophilic dermatitis is one of the isotopic responses that occur in the region of healed herpes zoster. It was first described by Mitsuhashi and Kondo¹ in 1997. The lesions in the first reported case clinically appeared as sharply demarcated brown plaques. Histologically, the lesions were characterized by a dermal infiltration of eosinophils and lymphocytes without atypicality. The dermatitis resolved one month after the application of topical steroid for 2 weeks¹. To the best of our knowledge, only one case of post-zoster eosinophilic dermatitis, as an

isotopic response, has been reported in the English medical literatures¹. We report here on another case of post-zoster eosinophilic dermatitis in a 48-year-old woman with a history of herpes zoster infection.

CASE REPORT

A 48-year-old female patient presented to our department with several severely pruritic, sharply demarcated, erythematous to brown papules and plaques with fine scales on her right flank (Fig. 1). The lesions had slowly developed for the past 6 months. The patient had no history of an insect bite, trauma or medication. Her medical history included herpes zoster along the T10 dermatome of the right flank, and this had had been successfully treated with an oral antiviral agent (Acyclovir, Vacrax[®], 4,000 mg/day, 7 days) 13 years previously and she had residual scars. On laboratory examination, the complete blood cell count was within the normal limits.



Fig. 1. Several erythematous to brown papules and plaques with fine scales along the T10 dermatome on the right flank. Inset: a magnified view.

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Eosinophils accounted for 3.8% (0~5%) of the peripheral white blood cells, and the total serum IgE level was 91.2 ku/L (<100 ku/L). The erythrocyte sedimentation rate was increased (35 mm/h). The skin biopsy specimen obtained from a plaque on the right flank revealed mild acanthosis and spongiosis in the epidermis and a perivascular inflammatory cell infiltration composed of abundant eosinophils, lymphocytes and histiocytes in the dermis (Fig. 2). There was no granulomatous change. An application of a topical steroid (Dethasone ointment[®], bid) for 2 weeks resulted in the lesions clearing up; no sign of recurrence was noted 6 months after treatment.

DISCUSSION

Wolf et al.² define an isotopic response as the occurrence of a new skin disorder at the site of another unrelated and already healed skin disease. The first disease in most of

these cases is herpes zoster, and several cutaneous reactions have been described in the dermatomes affected by herpes zoster³. The second diseases may be granulomatous reactions (mainly granuloma annulare), malignancies (single tumor and leukemic or lymphomatous infiltration), immune disorders (e.g. lichen planus, allergic contact dermatitis), infections (viral, bacterial, fungal) and others (e.g. acneiform lesions, reactive perforating collagenosis)³⁻⁵. Seven cases of postherpetic isotopic reactions have currently been reported in the Korean dermatologic literature (Table 1)⁶⁻¹³.

The pathogenesis of isotopic responses is unclear. Several possibilities have been considered: a viral origin, an immunologic origin, a vascular origin and a neural origin. Concerning a viral origin, it has been suggested that either the virus is directly responsible for the second disease, or the second disease is the result of an inflammatory reaction that develops in response to residual viral

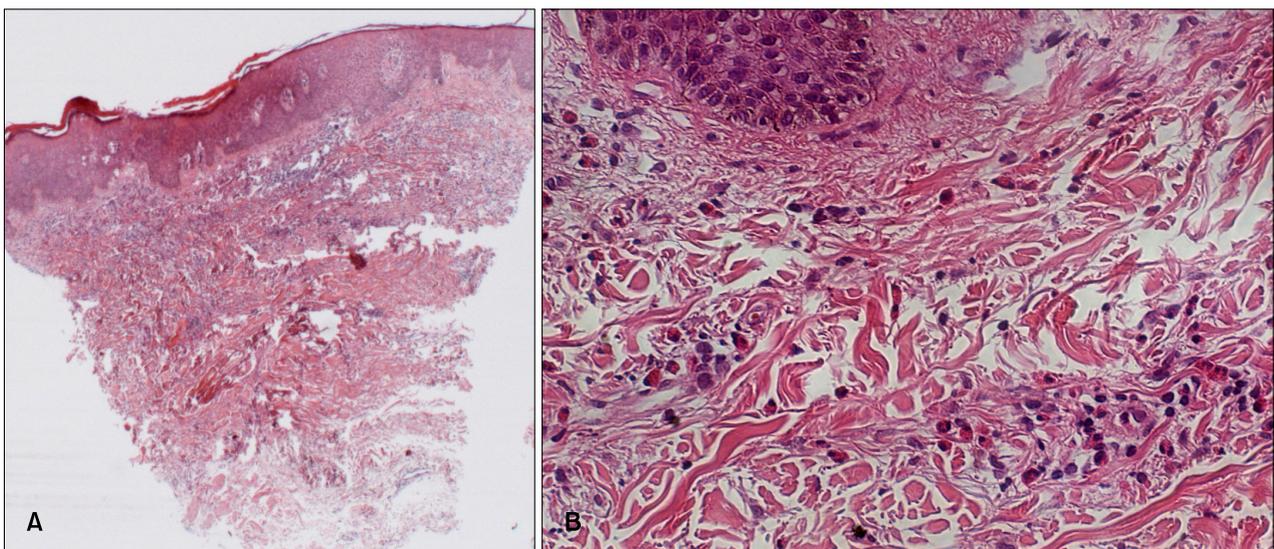


Fig. 2. (A) A biopsy specimen obtained from a representative plaque showed mild acanthosis and spongiosis in the epidermis and a perivascular inflammatory cell infiltration in the dermis (H&E, $\times 25$). (B) The dermal perivascular inflammatory cell infiltration was composed of lymphocytes and histiocytes, along with abundant eosinophils (H&E, $\times 400$).

Table 1. Summary of the postherpetic isotopic responses that were previously reported in the Korean dermatologic literatures

Case	Age (yr)/Sex	Location	Time interval*	Isotopic response	References
1	27/M	Right forehead and scalp	1 mo	Acneiform eruption	6
2	56/W	Right trunk (T9-T10)	3 mo	Post-zoster granuloma	7
3	67/F	Left neck and shoulder (C4)	1 mo	Reactive perforating collagenosis	8
4	8/M	Right chest (T2)	1 mo	Benign fibrous histiocytoma	9
5	55/F	Right neck and shoulder	2 mo	Granuloma annulare	10
6	39/M	Left buttock and lower leg	2 wk	Lichen planus	11
7	44/F	Right flank (T11)	3 mo	Lichen planus	12
8	76/M	Right chest (T4)	2 wk	Cutaneous metastatic carcinoma of lung	13
9	48/F	Right flank (T10)	13 yr	Post-zoster eosinophilic dermatosis	Our case

*Time interval between the resolution of herpes zoster and the appearance of the second cutaneous lesions

antigens or to the tissue damage. Polymerase chain reaction has been used to investigate the skin lesions for the presence of varicella-zoster virus, which has been consistently found only in the early lesions³. The development of post-zoster dermatosis may be influenced by long-lasting immunologic changes, alterations in the microcirculation or the destruction of A δ and C nerve fibers that occurs after viral infection of the skin^{4,14}.

Our patient presented with several erythematous to brown papules and plaques with fine scales on the T10 dermatome in the area of a previous zoster infection. The clinical differential diagnosis in this case included granuloma annulare and lichen planus. However, the absence of any histological granulomatous change ruled out the possibility of granuloma annulare. Lichen planus was also excluded because of the absence of the characteristic histological findings such as basal layer vacuolization, Civatte bodies and a band-like inflammatory cell infiltration. Instead, one of the most pronounced histological features in our case was a prominent eosinophil infiltration in the lesion. Thus, we needed to further exclude the possibility of drug eruption and insect bites, but the patient denied any medication history or insect bite history, and peripheral eosinophilia was not detected. Based on these clinical and histopathological findings, we diagnosed our case as post-zoster eosinophilic dermatosis. Post-zoster eosinophilic dermatosis is one of the isotopic responses, and these are clinically and histopathologically distinct from the other known dermatoses. It occurs on the region of the healed herpes zoster and it is histologically characterized by a dermal infiltration of eosinophils.

In an isotopic response, the time elapsing between the first disease and the second one widely varies, ranging from days to years¹⁴. In our patient, the delay was approximately 13 years. Until we better understand the pathogenesis of the isotopic response, it will be difficult to design targeted therapies^{3,7}. Langenberg et al.¹⁵ suggest that antiviral therapy may not be appropriate in inflammatory post-zoster conditions. Instead, topical or systemic corticosteroids are often used^{7,15,16}. Most post-zoster conditions are self-limited in nature, but these lesions should be examined histologically because of the possibility of occurrence of some malignancies such as lymphoma and Kaposi's sarcoma^{16,17}.

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