Localized Chronic Urticaria at the Site of Healed Herpes Zoster

Sir,

Isotopic response is defined as the occurrence of a new, unrelated disease at the same site as a previous disease, commonly herpes zoster. Various diseases have been reported in terms of isotopic response associated with herpes zoster, but the development of localized chronic urticaria is very uncommon. Herein, we described a rare case of localized chronic urticaria presenting as pruritic multiple erythematous weals at the site of healed herpes zoster.

CASE REPORT

A 58-year-old Korean woman presented in May 1995 with multiple grouped ulcers with crusts and hypertrophic scars on the left side of her abdomen (T9 – T10 dermatomes). She said she had been treated at a private dermatological clinic under the diagnosis of herpes zoster for 3 weeks. She complained of severe pain of the involved dermatomes of the skin.

Systemic analgesics and intralesional nerve block with triamcinolones and lidocaine were used for the treatment of post-herpetic neuralgia for over a year. Epidural block was carried out twice, but the pain was persistent. During the course of treatment, the pain showed only mild alleviation without remarkable improvement. The involved skin healed with a hypertrophic scar.

In July 1996 she complained of localized pruritic erythematous weals and papules at the site previously affected by herpes zoster (Fig. 1). The histopathological findings of the weal consisted of dermal oedema and mild perivascular infiltrates.

The localized urticaria did not respond to antihistamine treatment and it was persistent locally at the dermatomes without spreading or generalization. No topical treatment was used. Also postherpetic neuralgia did not improve and it did not show any notable changes in severity after the development of localized urticaria.

DISCUSSION

The aetiology of isotopic response is considered multifactorial (1); viral, immunological, neural and vascular. In this case, we considered the inter-related actions of the nervous system and the immune system. The localization of the urticaria to the previous lesion of herpes zoster without generalization suggests interaction of the nervous system and the immune system. In addition, the persistent postherpetic pain suggests the association of damaged nerve fibres in the development of localized urticaria. It is known that various neuropeptides from sensory nerve fibres in the skin contribute to the occurrence of urticaria (2). Although nerves may not be directly involved in the pathogenesis of secondary disease arising in herpetic scars, the nervous system may indirectly influence the immune system.

Another possible explanation in our case is the role of increased numbers of mast cells in the hypertrophic scar of a previous zoster. We counted mast cells in toluidine blue-stained section of the urticarial weal and scar. The mast cell count was 34/10 high power field (HPF, ×400) in the weal and 48/10 HPF in the scar. Differences in the distribution of mast cell were also noted. In the weal, mast cells were more frequent in the upper dermis, but in the scar, they showed a more even distribution. Although it may be possible that the increased numbers of mast cells in hypertrophic scar have influenced the development of localized urticaria, we could not find any definite evidence for this.

In our patient, localized urticaria developed 1 year after the herpes zoster infection. The interval between the first disease and the isotopic second disease was variable from days to decades. These observations suggest that skin remembers its previous injuries no matter how normal it looks (3).

REFERENCES


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