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Editorial

Post-Herpes Zoster Granulomatous Reactions

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Editorial

Herpes zoster is caused by the reactivation of latent varicella-zoster virus (VZV) infection. Cutaneous lesions of herpes zoster present with erythematous papules and vesicles, which heal within a few weeks, occasionally leaving neuralgia.Wolf's post-herpetic isotopic response is the occurrence of new, unrelated disorders such as granulomatous reactions, malignant tumors, dysimmune reactions, and infections on the skin areas that had previously been infected by herpes viruses [1]. Healed skin following herpes zoster is not normal skin, but may become a privileged site for developing other kind of dermatosis. Several possible mechanisms such as viral, immunological, vascular, and neural etiology are proposed in this process.Recent studies have suggested that skin is the neuro-immune-endocrine organ, and indicated that the damage to peripheral sensory nerve fibers caused by VZV may have a crucial role in post-zoster skin reactions [2]. In fact, the subsequent dysregulation of neuropeptides released by involved sensory fibers might alter the local neuro-immune control accounting for local onset of immunity-related reactions [3]. Among various conditions, granuloma annulare is the representative condition [4], which is suggested to be composed of Th1 subset. Histologically, lichenoid and granulomatous features are occasionally seen following herpes zoster, including the histiocytic component of an interstitial array between collagen fibers reminiscent of interstitial granuloma annulare [5]. Also, granulomatous vasculitis and granulomatous folliculitis have been reported [6,7]. Interestingly, granulomatous lesions can be induced not confined to the previously affected sites but also beyond the herpes zoster-affected sites, as multiple daughter lesions [8], which may reflect the sustained macrophage activation. On the other hand,only several cases of post-herpes zoster scar sarcoidosis have been reported to date [9]. Scar formation following herpes zoster may induce sarcoidal granuloma. Alternatively, interferon-γ (IFN-γ) induced by viral infection favors Th1 shift, which may play a role in granuloma formation. Immunostaining of VZV antigens revealed the localization in the eccrine sweat glands [10,11], and VZV reactivation may induce the local inflammation and/or immunological reactions.Granuloma formation for VZV protein may also be relevant to sarcoid reactions.

Patients frequently suffer from persistent neuralgia following herpes zoster, whereas they also complain of itching (post-herpetic itch). Previously, tissue eosinophilia at the post-herpetic skin was reported, which clinically presented with well-circumscribed brownish plaques with raised borders [12]. Histological examination revealed a dermal perivascular infiltrate composed of lymphocytes, histiocytes, and numerous eosinophils, without granulomatous changes. In addition, several atypical lesions can be induced such as papular lesions or erythematous plaques in a reactive inflammatory process against residual zoster viral antigens, or mediated by Th2-dominant immune reactions [13]. In the blister fluids of herpes zoster infection, Th2-based cytokines such as IL-4 and IL-10 were significantly higher, whereas the levels of IL-2 and IFN-γ were decreased [14], suggesting a shift with a Th2 dominant cytokine production. IL-4 induces eotaxin [15], which may recruit eosinophils into the skin.

Post-herpetic granulomatous lesion of childhood occurrence is extremely rare. To date, only 1 pediatric case of granulomatous lesions complicating primary varicella zoster infection was reported in a patient with Rothmund-Thomson syndrome [16]. Another possible case developed a well-circumscribed brownish plaque on the right upper back, and histological features showed granulomatous reaction [17]. Vitiligo is one of the conditions of isotopic response following herpes zoster in childhood [18], suggesting that isotopic reaction can be seen in not only adults but also children.

In conclusion, granulomatous reaction might be induced by several possible mechanisms, *i.e.*i) longstanding local immune response to the VZV components, residual zoster viral antigens, or degenerated dermal components due to herpes zoster, ii) shift toward Th1 due to IFN-γ induced by viral infection, and iii) local neuro-immune dysregulation by neuropeptides released by damaged peripheral sensory nerve fibers.

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