Case Letter

Rapid-onset lichen simplex chronicus after a superinfected herpes simplex virus eruption

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Dear Editors,

Lichen simplex chronicus (LSC) is a cutaneous condition that results from continuous scratching of a localized area of intractable pruritus. LSC is typically chronic, with known stimuli including xerosis and atopic dermatitis. Rarely has LSC been reported to develop rapidly after herpes zoster (Akyol et al., 2000). Herein, we describe LSC developing 3 weeks after a superinfected herpes simplex virus type 2 (HSV-2) eruption.

A 56-year-old woman presented for an itchy eruption on the lower back recurring monthly over 1 year. Her past eruptions and the associated pruritus had resolved with short courses of valacyclovir without the need for antipruritics. She reported that the current episode was more pruritic, painful, and exudative than usual. Physical examination revealed a large cluster of punched-out erosions with scalloped borders and purulent drainage on the superior gluteal cleft. Viral and bacterial cultures of the lesion were taken, and the patient was treated with 7 days of valacyclovir 500 mg twice daily and mupirocin 2% ointment three times daily.

At a follow-up visit 1 week later, the patient reported worsening pruritus in conjunction with the lesion developing an overlying crust. The viral culture tested positive for HSV-2, and the bacterial culture grew methicillin-resistant Staphylococcus aureus and Pseudomonas aeruginosa. The patient was treated with 7 days of doxycycline 100 mg twice daily and gentamicin 0.1% ointment three times daily.

At a follow-up visit 2 weeks later, the patient reported that the pruritus had intensified even further, resulting in persistent scratching of the area. Upon examination, there was a large, depigmented, hypertrophic plaque following the outline of the previous HSV-2 eruption (Fig. 1). Skin biopsy was diagnostic of LSC (Fig. 2); immunostains for HSV-1/2 were negative. Treatment with triamcinolone acetonide 0.1% ointment twice daily for 1 week significantly improved the patient’s pruritus.

LSC is an unusual sequela of confirmed herpesvirus eruptions that, to our knowledge, has been previously reported only twice (Akyol et al., 2000; Gerritsen et al., 1998). In these cases, the patients had herpes zoster, and LSC was hypothesized to have resulted from subacute herpetic neuralgia (SHN; Akyol et al., 2000) or postherpetic neuralgia (PHN; Gerritsen et al., 1998). SHN is a potential complication of varicella zoster virus (VZV) reactivation and defined as pain, burning, and/or itching that persists after healing of the herpes zoster eruption but resolves within 4 months. On the other hand, PHN refers to symptoms that persist beyond 4 months after the initial eruption. Neither SHN nor PHN is traditionally associated with other herpesviruses, such as HSV (Gonzales, 1992; Ooi and Zawar, 2011). This difference is likely explained by the observation that HSV reactivation in the dorsal nerve root remains confined to the affected neuron, whereas VZV reactivation involves inflammation and necrosis of multiple neurons. The cumulative damage in herpes zoster can lead to hyperac-
tivity of nerve fibers, clinically manifesting as SHN or PHN (Ikoma et al., 2006).

In our case, bacterial superinfection may also have contributed to localized tissue destruction, resulting in SHN, which then led to LSC. Interestingly, both in this report and one other report (Gerritsen et al., 1998), LSC developed 3 weeks after the initial herpesvirus eruption, suggesting that HSV and VZV may induce LSC faster than other stimuli that follow the typical chronic timeline.

Our case is important for dermatologists to appreciate because rapid-onset LSC in the context of HSV-associated SHN or PHN is rarely reported. In addition to topical corticosteroids, therapies for HSV-associated neuralgia could include those standardly used for PHN, including topical lidocaine, topical capsaicin, and gabapentin. Early consideration of these therapies may help prevent sequelae, such as the LSC in this case.

Conflicts of interest
None.

Funding
None.

Study approval
N/A.

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