

Seborrhea Herpeticum: Cutaneous Herpes Simplex Virus Infection Within Infantile Seborrheic Dermatitis

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Eczema herpeticum has been well described in the setting of atopic dermatitis (AD) and other dermatoses. We present the case of a 2-month-old infant boy with cutaneous herpes simplex virus (HSV) infection within existing diffuse infantile seborrheic dermatitis. Providers should be aware that cutaneous HSV can be confined to a seborrheic distribution and may represent underlying epidermal dysfunction secondary to seborrheic dermatitis.

Practice Points

- Cutaneous herpes simplex virus may present in a seborrheic distribution within infantile seborrheic dermatitis, suggesting underlying dysfunction secondary to seborrheic dermatitis.
- Treatment of seborrhea herpeticum involves antiviral therapy to treat the secondary viral infection and gentle skin care precautions for the primary condition.

Classically, eczema herpeticum is associated with atopic dermatitis (AD), but it also has been previously reported in the setting of pemphigus vulgaris, Darier disease, ichthyosis vulgaris, burns, psoriasis, and irritant contact dermatitis.^{1,2} Descriptions of cutaneous herpes simplex virus (HSV) in the setting of seborrheic dermatitis are lacking.

Case Report

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A 2-month-old infant boy who was otherwise healthy presented to the emergency department with a new rash on the scalp. Initially there were a few clusters of small fluid-filled lesions that evolved over several days into diffuse clusters covering the scalp and extending onto the forehead and upper chest (Figure). The patient's medical history was notable for infantile seborrheic dermatitis and a family history of AD. His grandmother, who was his primary caretaker, had a recent history of herpes labialis.

Physical examination revealed numerous discrete, erythematous, and punched-out erosions diffusely on the scalp. There were fewer similar erosions on the forehead and upper chest. There were no oral or periocular lesions. There were no areas of lichenification or eczematous plaques on the remainder of the trunk or extremities. Laboratory testing was positive for HSV type 1 polymerase chain reaction and positive for HSV type 1 viral culture. Liver enzymes were elevated with alanine aminotransferase at 107 U/L (reference range, 7–52 U/L) and aspartate aminotransferase at 94 U/L (reference range, 13–39 U/L).

The patient was admitted to the hospital and was treated by the dermatology and infectious disease services. Intravenous acyclovir 60 mg/kg daily was administered for 3 days until all lesions had crusted over. On the day of discharge, the patient was transitioned to oral valacyclovir 20 mg/kg daily for 7 days with resolution. One month later he developed a recurrence that was within his existing seborrheic dermatitis. After a repeat 7-day course of oral valacyclovir 20 mg/kg daily, he was placed on prophylaxis therapy of oral acyclovir 10 mg/kg daily. Gentle skin care precautions also were recommended.

Comment

Eczema herpeticum refers to disseminated cutaneous infection with HSV types 1 or 2 in the setting of underlying dermatosis.² Although it is classically associated with AD, it has been reported in a number of other chronic skin disorders and can lead to serious complications, including hepatitis, keratoconjunctivitis, and meningitis. In those with AD who develop HSV, presentation may occur in

active dermatitis locations because of skin barrier disruption, which may lead to increased susceptibility to viral infection.³

Herpes simplex virus in a background of seborrheic dermatitis has not been well described. Although the pathogenesis of seborrheic dermatitis has not been fully reported, several gene mutations and protein deficiencies have been identified in patients and animal models that are associated with immune response or epidermal differentiation.⁴ Therefore, it is possible that, as with AD, a disruption in the skin barrier increases susceptibility to viral infection.

It also has been suggested that infantile seborrheic dermatitis and AD represent the same spectrum of disease.⁵ Given our patient's family history of AD, it is possible his presentation represents early underlying AD. Providers should be aware that cutaneous HSV can be confined to a seborrheic distribution and may represent underlying epidermal dysfunction secondary to seborrheic dermatitis.

References

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Figure 1. Seborrhea herpeticum. Fluid-filled lesions that evolved into diffuse clusters on the scalp.