ONLINE EXCLUSIVE

Vesicular eruption in a 2-year-old boy

A bath with scented soap prompted a flare of the boy's eczema. Days later, he was hospitalized with diffuse erosions covering 90% of his body. What was the cause?

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A 2-YEAR-OLD BOY with atopic dermatitis developed a flare of his eczema after having a bath with mint-scented soap. His mother treated the flare with over-the-counter topical hydrocortisone cream. Two to 3 days later, he developed grouped vesicles on the right side of his neck. Three days after that, he developed a painful generalized vesicular eruption all over his body.

The boy was admitted to a hospital for supportive care and empiric antibiotics, but was discharged when no bacterial infection was found. The patient's mother was instructed to follow up with his primary care provider in the next 2 weeks.

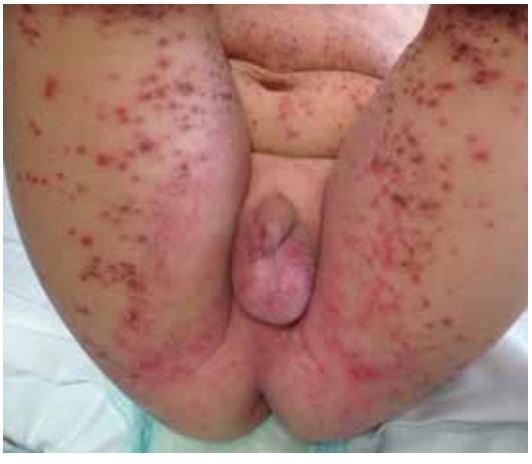
Three days after his hospitalization, the eruption on the young boy's body spread and he was uncomfortable. He was brought to our hospital's pediatric clinic, where physicians examined him and decided to transfer him to the university hospital for further evaluation.

On exam, the boy was afebrile, but uncomfortable and irritable. Diffuse heme-crusted and punched-out erosions covered about 90% of his body (**FIGURE**). His mucous membranes were not involved. Underneath the heme-crusted erosions, there were lichenified pink plaques on the antecubital fossae, popliteal fossae, periocular face, and buttocks. The patient's right dorsal foot had a small vesicle; all other vesicles on his body had crusted over.

The patient's family indicated that the child had received the varicella vaccine without incident at 12 months of age. He had no history of travel, no contact with sick individuals, and no exposure to pets or other animals.

- WHAT IS YOUR DIAGNOSIS?
- HOW WOULD YOU TREAT THIS PATIENT?

FIGURE
Heme-crusted erosions covered nearly all of the boy's body



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Eczema herpeticum occurs more often in infants and children than in adults, and is a potentially life-threatening emergency.

Diagnosis: Eczema herpeticum

Eczema herpeticum (EH) was suspected based on the appearance of the lesions. A Tzanck smear came back positive for multinucleated giant cells and a herpes simplex virus (HSV) amplified probe came back positive for HSV-1—confirming the diagnosis.

EH—also known as Kaposi varicelliform eruption—is a superficial generalized viral infection (typically caused by HSV-1; HSV-2 is less common). The infection commonly occurs in patients with underlying atopic dermatitis, but may also occur in those with Darier disease, pemphigus, burns, and other conditions that disrupt the skin barrier. Other viruses, such as Coxsackie virus, can also cause EH. Eczema vaccinatum is a variant that may occur after smallpox vaccination.[1] EH occurs more often in infants and children than in adults,[2] and is a potentially life-threatening dermatologic emergency.

Who's at risk? Patients with underlying chronic skin conditions such as eczema may have impaired cell-mediated immunity, making them more susceptible to a viral infection like EH.[1] In addition, treatment of underlying chronic skin conditions with immunosuppressive therapies often increases susceptibility to superimposed infection.[1] (In this case, the patient's parents had treated an eczema flare with a topical hydrocortisone cream.) Lastly, increased risk may be associated with mutations in the gene encoding filaggrin.[2]

Areas affected. EH typically appears in areas of pre-existing dermatitis as monomorphic, discrete, 2- to 3-mm, punched-out, heme-crusted erosions with scalloped borders.[2] The erosions initially appear as vesicles or pustules, which may appear concurrently with the erosions. The erosions can coalesce to form larger lesions.[3] Fever, malaise, and lymphadenopathy may also be present.[2,3]

4 factors differentiate EH from other conditions

The differential for eczema herpeticum includes impetigo, bullous impetigo, shingles, chicken pox, scabies, pustular psoriasis, bullous pemphigoid, drug hypersensitivity reactions, and exacerbation of a primary dermatosis or skin condition.[1,4]

EH may be differentiated from these by its location, its development in the setting of pre-existing dermatitis, its response to antiviral medications, and the results of laboratory testing. Because of the vast differential, physicians must maintain a high index of suspicion for EH, particularly when a patient with a pre-existing skin condition presents with acute onset cutaneous pain.[3]

Perform a Tzanck smear to diagnose the underlying infection

If EH is suspected, treatment must be initiated immediately.[3] (In our patient's case, he was started on intravenous acyclovir 10 mg/kg every 8 hours.)

Once treatment is underway, a Tzanck smear of the vesicle base can be performed at the patient's bedside to narrow the cause of the infection to HSV or varicella zoster virus (VZV). Multinucleated giant keratinocytes (as in our patient's case) are diagnostic for one of the herpes viruses; concurrent inflammatory cells are also to be expected in an inflammatory skin condition but by themselves are not diagnostic of herpes.

If available in the laboratory, direct fluorescent antibody testing can differentiate between HSV and VZV. Alternatively, a nucleic acid amplified probe test may be used to provide a quick and specific result. The most specific test is a viral culture, but it lacks sensitivity and usually requires 2 to 5 days for results.[2] A bacterial skin swab and blood culture should also be considered to direct antibiotic therapy if superinfection has occurred.

Antivirals and antibiotics should be given until lesions heal

Patients with EH should be admitted to the hospital for at least 24 to 48 hours of intravenous acyclovir.[4] Antivirals—oral or intravenous—should be given for 10 to 14 days or until all mucocutaneous lesions are healed. Recommended dosing for acyclovir is 15 mg/kg (up to 400 mg) by mouth 3 to 5 times per day or, if severe, 5 mg/kg (if ≥12 years of age) to 10 mg/kg (if <12 years of age) intravenously every 8 hours.[2] Patients should also receive a 3- to 6-month suppressive course of oral acyclovir, valacyclovir, or famciclovir.[4]

Intravenous antibiotics should also be

considered, pending the results of bacterial skin swabs and a blood culture, as the skin of patients with atopic dermatitis is colonized with *staphylococcus* 90% of the time.[4]

Potential complications. Bacterial sepsis resulting from superinfection and disseminated HSV, although extremely rare, is the main cause of death associated with EH.[3] One case in the literature described a 43-year-old woman with extensive EH superimposed on atopic dermatitis, disseminated HSV, and *Pseudomonas aeruginosa* septicemia. Despite treatment with intravenous acyclovir and antibiotics in a burn center intensive care unit, the patient experienced septic shock and disseminated intravascular coagulation with progression to multiorgan failure and death.[3]

Our patient's antiviral regimen was transitioned to a 14-day course of oral acyclovir, which he completed. Topical steroids and an immunosuppressant (tacrolimus ointment) were applied concurrently. He was subsequently prescribed a 6-month suppressive course of acyclovir and was scheduled for follow-up at an outpatient dermatology clinic to discuss resuming therapy for atopic dermatitis. JFP

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