A 15-MONTH-OLD African American boy with a history of atopic dermatitis since the age of 2 months was brought to the emergency department by his mother for an exacerbation of his chronic dermatitis. He had been regularly observed by his primary care physician and dermatologist, and the dermatitis was managed with topical corticosteroids and emollients. Over the 5 days prior to admission, he developed worsening pruritus, increased weeping lesions, irritability, and fever. He had no history of chickenpox; however, he had received a live, attenuated varicella vaccine (Varivax; Merck & Co, Inc, West Point, Pa) 5 days before admission. He had had contact with a visitor with “cold sores” 2 months earlier. On physical examination the child was irritable, uncomfortable, and constantly scratching. Rectal temperature was 40°C. Punched out erosions with an erythematous base were confluent on the face and more discrete on the trunk and upper extremities. Hemorrhagic and golden-colored crusting were evident and numerous excoriations were seen (Figure 1). Two erosions were noted on the soft palate mucosa, and the conjunctivae were normal. Shotty cervical, axillary, and inguinal lymphadenopathy was noted. A specimen for Tzanck testing was prepared by scraping the base of 1 of the facial erosions and staining the cellular material with Wright stain. Numerous multinucleated giant cells were noted on microscopic examination (Figure 2). Viral and bacterial skin cultures and bacterial blood cultures were obtained.
Eczema Herpeticum

Diagnosis and Discussion

Eczema herpeticum (EH), also known by its eponym Kaposi varicelliform eruption, is a potentially fatal, widespread cutaneous infection caused by human herpesvirus 1 (HHV-1). Eczema herpeticum is characterized clinically by clusters of umbilicated vesicles and eventually pustules in areas of previously normal skin. The most common predisposing cutaneous disorder is atopic dermatitis. Other less commonly reported risk factors for developing EH include Wiskott-Aldrich syndrome, Darier disease, pemphigus foliaceus, benign familial chronic pemphigus, and chronic irritant contact dermatitis. Cases that arise from healing second-degree burns, autografted skin, and staphylococcal scalded skin syndrome have been reported.

Abnormal skin is the virus’ portal of entry, but the eruption rapidly spreads to previously normal and uninvolved skin. The vesicles evolve into pustules and then the classic punched out erosions, which may become confluent with subsequent hemorrhagic crusting. Patients are typically febrile and appear to have had a toxic reaction. Visceral dissemination of HHV-1 and subsequent mortality have been estimated at 1% to 9%. Secondary bacterial infection often complicates the course of illness and contributes to morbidity and mortality. The most common secondary pathogen is Staphylococcus aureus.

The incidence of EH is highest in children younger than 3 years, with an equal male-female ratio. Approximately 3% of all children younger than 5 years have atopic dermatitis and are at risk of developing EH. Restriction fragment length polymorphism studies have shown that HHV-1 strains of the F35 genotype are more often associated with EH than other HHV-1 genotypes. Children with atopic dermatitis who have a number of circulating natural killer cells and decreased interleukin (IL)-2 and increased IL-4 levels are more susceptible to EH.

Herein, we report the occurrence of EH 5 days after vaccination with a live, attenuated varicella vaccine. Initial evaluations raised concern whether the clinical findings were related to this. Results of the Tzanck test revealed multinucleated giant cells; however, this test cannot distinguish between HHV-1 or varicella-zoster infection. The HHV-1 infection was ultimately confirmed by conducting a viral culture. Bacterial cultures taken from the skin grew S aureus. Our patient promptly recovered after a parenteral injection of acyclovir and antistaphylococcal therapy (cephalexin oral suspension).

Neither review of all of the literature nor postmarketing surveillance by the manufacturer (Charles A. Baechler, PhD, written communication, Merck & Co, Inc, July 25, 1997) has revealed any cases of EH after vaccination with the varicella vaccine. Adverse reactions to live, attenuated varicella vaccine include an injection site varicellalike rash with a median number of 2 lesions and a more generalized varicellalike rash with a median number of 5 lesions with the peak occurrence happening 5 to 26 days after vaccination.

Pediatric patients with chronic cutaneous disorders are at increased risk of developing severe varicella infections, and active immunization with the varicella vaccine is not contraindicated. A high clinical suspicion for EH should be maintained in children with atopic dermatitis who have a vesicular eruption, even in the setting of recent varicella vaccination.

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Corresponding author: Amir Bajoghli, MD, Department of Dermatology and Pediatrics, Boston University School of Medicine, 609 Albany St, Boston, MA 02118.

REFERENCES