A 19-year-old man with a history of atopic dermatitis that was managed with mild topical steroids was transferred from a hospital for the management of possible Stevens-Johnson syndrome or toxic epidermal necrolysis. Two weeks prior to admission he reported that he had a high-risk sexual encounter and subsequently developed a rash on his groin as well as dysuria. He was seen by a physician and was treated with intramuscular penicillin for presumed gonorrhea. Several days later the eruption spread to his entire body. He was admitted to an outside hospital with fever and facial lesions that had an oozing purulent discharge. He was treated with vancomycin and levofloxacin and was transferred to our burn unit for debridement. He had crusted purulent vesicles and bullae involving his bilateral arms, face, groin, and penis. Wound scrapings of one vesicle were obtained.
Kaposi varicelliform eruption, often referred to as eczema herpeticum (EH), is a disseminated infection of herpes simplex virus type 1 or type 2. Although most cases typically begin as a labial infection in herpes simplex virus type 1, our patient presented with a less common development of EH following a primary genital infection. Eczema herpeticum has been reported to occur in numerous conditions but most commonly is associated with atopic dermatitis (AD). Patients with AD who have been predisposed to EH have been shown to have deficiencies in both the innate and adaptive immune system including decreased levels of cathelicidin and type I interferon.

Kaposi varicelliform eruption typically presents as a disseminated eruption of monomorphic vesicles and bullae accompanied by fever, malaise, and lymphadenopathy. The morphology of the lesions can vary, and in our patient, the diagnosis was complicated by chancroid-like lesions (Figure 1). The differential diagnosis of EH includes widespread impetigo, Stevens-Johnson syndrome, contact dermatitis, or varicella. A Tzanck test (Figure 2) of the vesicles can help with the diagnosis of EH and direct fluorescent antibody testing also can be used to confirm a diagnosis of EH. Our patient had positive direct fluorescent antibody testing to herpes simplex virus type 2 and quickly responded to intravenous acyclovir dosed at 10 mg/kg every 8 hours. Within days he started feeling better and medication was switched to 400 mg 3 times daily until lesions cleared.

Prompt diagnosis and treatment with acyclovir or other antiviral agents is essential. Despite the attention that the National Institutes of Health has devoted to AD and herpes simplex virus superinfections, reports of EH that were misdiagnosed still are frequent and may lead to days of inappropriate treatment; moreover, the delay in diagnosis can result in complications including keratitis with resultant permanent visual impairment or death.

Practitioners always should be mindful of a history of atopy when evaluating a patient with a widespread eruption such as EH. Utilization of simple bedside tests such as a Tzanck test can help to narrow a seemingly broad differential diagnosis. Our case is an example of how correct diagnosis and treatment by a dermatologist can help to promote the avoidance of unnecessary intervention and reduce patient morbidity.

**Figure 1.** Crusted purulent vesicles and bullae involving the neck (A) and groin (B).

**Figure 2.** Epidermal cells showing typical intranuclear inclusions (H&E, original magnification ×40).
REFERENCES


