Diffuse facial rash in a former collegiate wrestler

The patient described a history of "recurrent impetigo," which led to an uncommon diagnosis.

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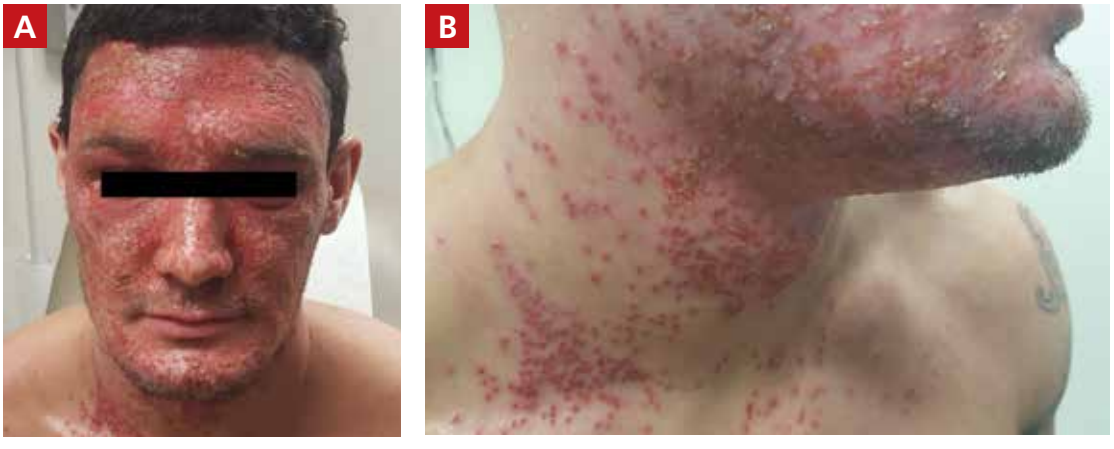
A 22-YEAR-OLD CAUCASIAN MAN with a history of atopic dermatitis (AD) was referred to our dermatology clinic for evaluation of a diffuse facial rash that had been present for the previous 7 days. The rash initially presented as erythema on the right malar cheek that rapidly spread to the entire face. Initially diagnosed as impetigo, empiric treatment with sulfamethoxazole/trimethoprim (800 mg/160 mg PO BID for 7 days), dicloxacillin (500 mg PO BID for 6 days), cephalexin (500 mg TID for 5 days), and mupirocin (2% topical cream applied TID for 6 days) failed to improve the patient's symptoms. He reported mild pain associated with facial movements.

The patient had a history of similar (but more limited) rashes, which he described as "recurrent impetigo," that began during his career as a high school and collegiate wrestler. These rashes were different from the rashes he described as his history of AD, which consisted of pruritic and erythematous skin in his antecubital and popliteal fossae. He denied any history of herpes simplex virus (HSV) infection.

A physical examination revealed numerous monomorphic, 1- to 3-mm, punched-out erosions and ulcers with overlying yellow-brown crust encompassing the patient's entire face and portions of his anterior neck. Several clustered vesicles on erythematous bases also were noted (**FIGURES 1A AND 1B**). We used a Dermablade to unroof some of the vesicles and sent the scrapings to the lab for Tzanck, direct fluorescent antibody assay (DFA), and HSV polymerase chain reaction (PCR) testing.

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

FIGURE 1
Monomorphic, punched-out erosions



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Prophylactic valacyclovir treatment at a 28-day high school wrestling camp reduced outbreak incidence of herpes gladiatorum by 89.5%.

Diagnosis: Eczema herpeticum secondary to herpes gladiatorum

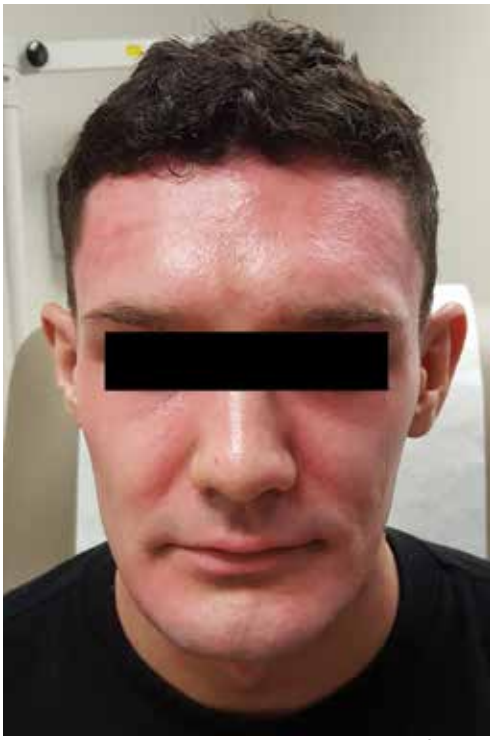
The patient's laboratory results came back and the Tzanck preparation was positive for multinucleated giant cells, and both the DFA and HSV PCR were positive for HSV infection. This, paired with the widely disseminated rash observed on examination and the patient's history of AD, was consistent with a diagnosis of eczema herpeticum (EH).

Rather than primary impetigo, the patient's self-described history of recurrent rashes was felt to represent a history of HSV outbreaks. Given his denial of prior oral or genital HSV infection, as well as the coincident onset of these outbreaks during his career as a competitive wrestler, the most likely primary infection source was direct contact with another HSV-infected wrestler.

Herpes gladiatorum refers to a primary cutaneous HSV infection contracted by an athlete through direct skin-to-skin contact with another athlete.[1] It is common in contact sports, such as rugby and wrestling, and particularly common at organized wrestling camps, where mass outbreaks are a frequent occurrence.[2] Herpes gladiatorum is so common at these camps that many recommend prophylactic valacyclovir treatment for all participants to mitigate the risk of contracting HSV. In a 2016 review, Anderson et al concluded that prophylactic valacyclovir treatment at a 28-day high school wrestling camp effectively reduced outbreak incidence by 89.5%.[2]

The lesions of herpes gladiatorum are classically limited in distribution and reflective of the areas of direct contact with infected skin, most commonly the face, neck, and arms. Our patient's history of more limited outbreaks on his face was consistent with this typical presentation. His current outbreak, however, had become much more widely disseminated, which led to the diagnosis of EH secondary to herpes gladiatorum.

FIGURE 2
Day 10 of treatment



Our patient responded well to a 10-day course of valacyclovir 1000 mg PO BID and a 7-day course of cephalexin 500 mg PO TID for coverage of bacterial superinfection.

Eczema herpeticum: Pathogenesis and diagnosis

Also known as Kaposi's varicelliform eruption, EH is a rapid, widespread cutaneous dissemination of HSV infection in areas of dermatitis or skin barrier disruption, most commonly caused by HSV-1 infection.[3] It is classically associated with AD, but also can occur in patients with impaired epidermal barrier function due to other conditions, such as burns, pemphigus vulgaris, mycosis fungoides, and Darier disease.[4] It occurs in <3% of patients with AD and is more commonly observed in infants and children with AD than adults.[5]

Clinically, the most common manifestations are discrete, monomorphic, 2- to 3-mm, punched-out erosions with hemorrhagic crusts; intact vesicles are less commonly observed.[4] Involved skin is typically painful and may be pruritic. Clinical diagnosis should be confirmed by laboratory evaluation, typically Tzanck preparation, DFA, and/or HSV PCR.

Complications and the importance of rapid treatment

The most common complication of EH is bacterial superinfection (impetigo), usually by *Staphylococcus aureus* or group A

streptococci. Signs of bacterial superinfection include weeping lesions, pustules, honey-colored/golden crusting, worsening of existing dermatitis, and failure to respond to antiviral treatment. Topical mupirocin 2% cream is generally effective for controlling limited infection. However, systemic antibiotics (cephalosporins or penicillinase-resistant penicillins) may be necessary to control widespread disease.[4] Clinical improvement should be observed within a single course of an appropriate antibiotic.

In contrast to impetigo, less common but more serious complications of EH can be life threatening. Systemic dissemination of disease is of particular importance in vulnerable populations such as pediatric and immunocompromised patients. Meningoencephalitis, secondary bacteremia, and herpes keratitis can all develop secondary to EH and incur significant morbidity and mortality.[1]

Fever, malaise, lymphadenopathy, or eye pain should prompt immediate consideration of inpatient evaluation and treatment for these potentially deadly or debilitating complications. All patients with EH distributed near the eyes should be referred to ophthalmology to rule out ocular involvement.

Immediately treat with antivirals

Due to the potential complications discussed above, a diagnosis of EH necessitates immediate treatment with oral or intravenous antiviral medication. Acyclovir, valacyclovir, or famciclovir may be used, with typical treatment courses ranging from 10 to 14 days or until all mucocutaneous lesions are healed.[4] Although typically reserved for patients with recurrent genital herpes resulting in 6 or more outbreaks annually, chronic suppressive therapy also may be considered for patients with EH who suffer from frequent or severe recurrent outbreaks.

Our patient. Given his otherwise excellent health and the absence of symptoms of potentially serious complications, our patient was treated as an outpatient with a 10-day course of valacyclovir 1000 mg PO BID. He was additionally prescribed a 7-day course of cephalexin 500 mg PO TID for coverage of bacterial superinfection. He responded well to treatment.

Ten days after his initial presentation to our clinic, his erosions and vesicles had completely cleared, and the associated erythema had significantly improved (**FIGURE 2**). Given the severity of his presentation and his history of 2 to 3 outbreaks annually, he opted to continue prophylactic valacyclovir (500 mg/d) for long-term suppression. JFP

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References

1. Shenoy R, Mostow E, Cain G. Eczema herpeticum in a wrestler. *Clin J Sport Med*. 2015;25:e18-e19.
2. Anderson BJ, McGuire DP, Reed M, et al. Prophylactic valacyclovir to prevent outbreaks of primary herpes gladiatorum at a 28-day wrestling camp: a 10-year review. *Clin J Sport Med*. 2016;26:272-278.
3. Olson J, Robles DT, Kirby P, et al. Kaposi varicelliform eruption (eczema herpeticum). *Dermatol Online J*. 2008;14:18.
4. Downing C, Mendoza N, Tyring S. Human herpesviruses. In: Bolognia JL, Schaffer JV, Cerroni L, eds. *Dermatology*. 4th ed. Philadelphia, PA: Elsevier; 2018:1400-1424.
5. Leung DY. Why is eczema herpeticum unexpectedly rare? *Antiviral Res*. 2013;98:153-157.

A diagnosis of eczema herpeticum requires immediate treatment with oral or intravenous antiviral medication.