Kaposi Varicelliform Eruption of Mpox in a Peeling Sunburn

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PRACTICE **POINTS**

- Desquamation can be associated with dissemination and higher severity course in the setting of mpox (monkeypox) viral infection.
- Antiviral treatment with tecovirimat is warranted in those with severe mpox infection or those at risk of severe infection including skin barrier disruption.
- Kaposi varicelliform–like eruptions can happen in the setting of barrier disruption from peeling sunburns, atopic dermatitis, severe acne, and other dermatologic conditions.

To the Editor:

The recent global mpox (monkeypox) outbreak that started in May 2022 has distinctive risk factors, clinical features, and patient attributes that can portend dissemination of infection. We report a case of Kaposi varicelliform eruption (KVE) over a peeling sunburn after mpox infection. Dermatologists should recognize cutaneous risk factors for dissemination of mpox.

A 35-year-old man who was otherwise healthy presented with a papulopustular eruption that began on the shoulders in an area that had been sunburned 24 to 48 hours earlier. He experienced fever (temperature, 38.6 °C)[101.5 °F]), chills, malaise, and the appearance of a painful penile ulcer. He reported a recent male sexual partner a week prior to the eruption during travel to eastern Asia and a subsequent male partner in the United States 5 days prior to eruption. Physical examination revealed a peeling sunburn with sharp clothing demarcation. Locations with the most notable desquamation—the superior shoulders, dorsal arms, upper chest, and ventral thighs—positively correlated with the highest density of scattered, discrete, erythematous-based pustules and pink papules, some with crusted umbilication (Figures 1 and 2). Lesions spared sun-protected locations except a punctate painful ulcer on the buccal mucosa and a tender well-demarcated ulcer with elevated borders on the ventral penile shaft. HIV antigen/antibody testing was negative; syphilis antibody testing was positive due to a prior infection 1 year earlier with titers down to 1:1. A penile ulcer swab did not detect herpes simplex virus types 1/2 DNA. Pharyngeal, penile, and rectal swabs were negative for chlamydia or gonorrhea DNA. A polymerase chain reaction assay of a pustule was positive for orthopoxvirus, and the Centers for Disease Control and Prevention confirmed *Monkeypox virus*. On day 12, a penile ulcer biopsy was nonspecific with dense mixed inflammation; immunohistochemical stains for *Treponema pallidum* and herpes simplex virus types 1/2 were negative. Consideration was given to starting antiviral treatment with tecovirimat, which is approved by the US Food and Drug Administration for smallpox caused by variola virus, through the Centers for Disease Control and Prevention expanded access protocol, but the patient's symptoms and lesions cleared quickly without intervention. The patient's recent sexual contact in the United States later tested positive for mpox.

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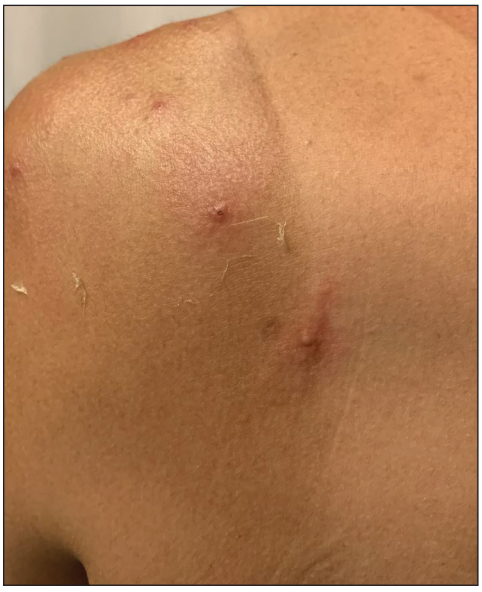

FIGURE 1. Left shoulder with pustule and pink umbilicated hemorrhagic crusted papules on an erythematous base overlying a background of superficially exfoliating hyperpigmentation with sharp cutoff of sun-protected skin below the shirt.

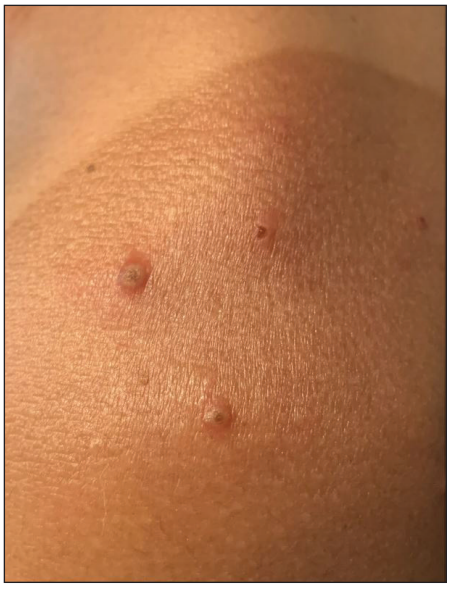

FIGURE 2. Centrally umbilicated crusted papules on the left shoulder overlying hyperpigmented sun-exposed skin, sparing sun-protected skin.

Given that the density of our patient's mpox lesions positively correlated with areas of peeling sunburn with rapid spread during the period of desquamation, he was diagnosed with KVE due to mpox in the setting of a peeling sunburn.

The recent mpox outbreak began in May 2022, and within 3 months there were more than 31,000 confirmed mpox cases worldwide, with more than 11,000 of those cases within the United States across 49 states and Puerto Rico.[1] Gay, bisexual, and other men who have sex with men have constituted the majority of cases. Although prior outbreaks have exhibited cases of classic mpox lesions, the current cases are clinically distinctive from classic mpox due to prevalent orogenital involvement and generalized symptoms that often are mild, nonexistent, or can occur after the cutaneous lesions.[2]

Although most current cases of mpox have been mildly symptomatic, several patients have been ill enough to require hospital admission, including patients with severe anogenital ulcerative lesions or bacterial superinfection.[3] Antiviral treatment with tecovirimat may be warranted for patients with severe disease or those at risk of becoming severe due to immunosuppression, pregnancy/breastfeeding, complications (as determined by the provider), younger age (ie, pediatric patients), or skin barrier disruption. Dermatologists play a particularly important role in identifying cutaneous risk factors that may indicate progression of infection (eg, atopic dermatitis, severe acne, intertrigo, Darier disease). Kaposi varicelliform eruption is the phenomenon where a more typically localized vesicular infection is disseminated to areas with a defective skin barrier.[2] Eczema herpeticum refers to the most common type of KVE due to herpes simplex virus, but other known etiologies of KVE include coxsackievirus A16, vaccinia virus, varicella-zoster virus, and smallpox.[2] Although classic mpox previously had only the theoretical potential to lead to a secondary KVE, we expect the literature to evolve as cases spread, with one recent report of eczema monkeypoxicum in the setting of atopic dermatitis.[4]

At the time of publication, mpox cases have notably dropped globally due to public health interventions; however, mpox infections are ongoing in areas previously identified as nonendemic. Given the distinctive risk factors and clinical presentations of this most recent outbreak, clinicians will need to be adept at identifying not only infection but also risk for dissemination, including skin barrier disruption.

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