Psoriasis coxsackium

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INTRODUCTION

In 1887, Moritz Kaposi coined the phrase "Kaposi's varicelliform eruption" defining a viral-induced rash in patients with an underlying dermatosis.[1] The most classic presentation is when patients with atopic dermatitis develop eczema herpeticum secondary to herpes simplex virus; however, other viruses have also been implicated, including coxsackievirus A16.[2] There are sparse reports of Kaposi varicelliform eruption in patients with psoriasis, and none in association with coxsackievirus.[3] Herein, we report what is possibly the first known case of psoriasis complicated by coxsackievirus, which we termed "psoriasis coxsackium."

Abbreviation used:

HFMD: hand-foot-and-mouth disease

CASE REPORT

A 32-year-old woman with a 25-year history of chronic plaque psoriasis presented with a vesiculopustular rash. Her psoriasis had been well-controlled with topical treatment and phototherapy. At her last clinic visit 2 months before presentation, psoriasis was limited to <5% total body surface area, primarily involving the extensor surfaces of the extremities (including elbows and knees). Three days before presentation, she developed fever and a sore throat. The following day, she developed scattered vesicles on the extensor aspects of the extremities. The fever and sore throat resolved 2 days after onset; however, the rash continued to develop. She presented with mildly erythematous, minimally scaly papules and plaques on the extensor surfaces of the arms and legs (Fig 1). Within these plaques, scattered 3- to 5-mm vesicles, few pustules, and crusted erosions were observed. A few erythematous papules were visualized on the palms and fingertips. No oral lesions were observed. She complained of early pruritus and subsequent burning pain. Sick contacts included her daughter, who developed fever on the same day as the patient, a rash 1 week later, and who was subsequently diagnosed with hand-foot-and-mouth disease (HFMD).

Two punch biopsies were performed on the right arm. Histopathologically, the biopsy showed robust spongiotic and interface dermatitis with foci of dyskeratosis and a focus of papillary epidermal necrosis with an evolving intraepidermal pustule (Fig 2). There were also subtle histopathologic features, such as psoriasiform hyperplasia, absence of a granular layer, and dilated blood vessels in edematous dermal papillae, suggestive of background psoriasis. Direct immunofluorescence and immunohistochemical stains for herpes simplex virus 1, herpes simplex virus 2, varicella-zoster virus, and parvovirus were negative. A respiratory virus polymerase chain reaction panel was positive for human rhinovirus/enterovirus. A lesional polymerase chain reaction test result for enterovirus from a vesicle within a psoriasis plaque on the extensor aspect of her arm was also positive. Utilizing clinicopathologic correlation, the diagnosis of psoriasis coxsackium was made. Clobetasol 0.05% ointment was prescribed to use twice daily for up to 2 weeks for symptomatic control. On follow-up in the clinic one month later, the patient demonstrated a complete clearance of her rash.

DISCUSSION

HFMD is an enterovirus-mediated illness, characteristically causing a papulovesicular exanthem and oral enanthem. Although more common in children, HFMD has recently been reported more frequently in

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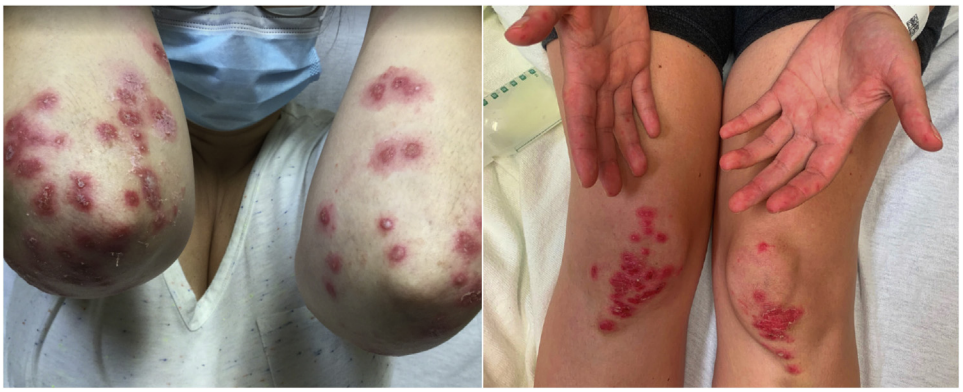

Fig 1. Erythematous, minimally scaly papules and plaques on the extensor parts of the patient's arms and legs. Within these plaques, scattered 3- to 5-mm vesicles, few pustules, and crusted erosions were observed. A few erythematous papules were observed on the palms and fingertips.

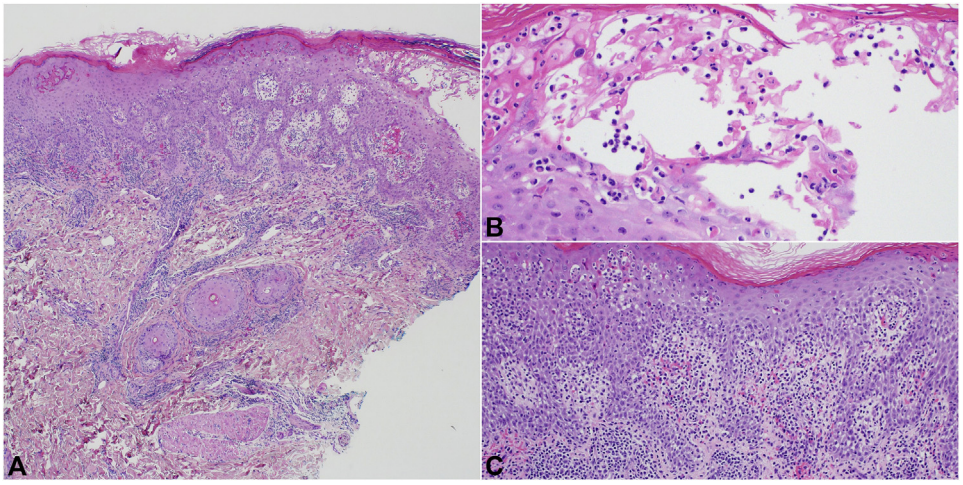

Fig 2. Hematoxylin-eosin staining with robust spongiotic and interface dermatitis with a focus of papillary epidermal necrosis with an evolving intraepidermal pustule (**A**), shown in higher power in (**B**), and foci of dyskeratosis (**C**). (**A**-**C**, Hematoxylin-eosin stain; original magnification: **A,** ×40; **B,** ×200; and **C,** ×100.)

adults. HFMD classically involves the oral mucosa and acral surfaces; yet, our patient had involvement within her psoriasis plaques. This begs the question of what led our patient to develop psoriasis coxsackium.

Psoriasis is a chronic inflammatory disease of the skin, in which the immune system plays an integral role. Raposo et al[4] found that antiviral genes are upregulated in patients with psoriasis as compared with patients with atopic dermatitis; however, this finding was only present in the skin, not the bloodstream. There is also enhanced production of antimicrobial peptides in psoriasis as compared with atopic dermatitis.[5] However, all antimicrobial peptide classes have antibacterial properties, but only a few classes have antiviral properties.[6] This may explain why HFMD developed in our patient, and subsequently Kaposi varicelliform eruption, after a high-risk household exposure. Additionally, we speculated as to an alternative pathogenesis, including koebnerization of the psoriasis after HFMD. This pathogenesis has been reported in the literature; a patient developed psoriasis papules on the sites of previous HFMD blisters.[7] However, it is unlikely, because our patient presented with psoriasiform change on the extensor surfaces—a common location for psoriasis, not HFMD. No psoriasiform rash was observed on her hands. Additionally, there was epidermal necrosis at the biopsied site, a feature that we would not expect in psoriasis. Another recent article described guttate psoriasis following a HFMD episode in a patient with no previous history of psoriasis; the authors proposed that this may have represented immune

system activation following infection in a genetically predisposed individual.[8]

This represents to our knowledge, the first reported case of psoriasis coxsackium. Although this had an indolent course, it remains a rare clinical presentation to consider when managing psoriasis flares.

Conflicts of interest

None disclosed.

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