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A case of atopic dermatitis and erythema multiforme

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Key words

Atopic dermatitis, eczema herpeticum, herpes simplex virus, erythema multiforme

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Summary

We report a case of a two-year-old boy with atopic dermatitis treated with antibiotics for pharyngitis and acute otitis media and subsequently developed targetoid and ulcerated blister mucocutaneous lesions. Diagnostic workup revealed eczema herpeticum and HSV viremia. To our knowledge, this is the first reported case of a patient with atopic dermatitis presenting with erythema multiforme likely secondary to eczema herpeticum and HSV viremia.

Introduction

A two-year-old male with well-controlled atopic dermatitis (AD) presented to his pediatrician for fever and sore throat. The patient was diagnosed with pharyngitis and acute otitis media, and he was prescribed amoxicillin. The next day, the patient developed a skin rash and gingival bleeding in addition to his intermittent fever until he was seen again by his pediatrician four days later. Due to concern for a drug reaction, amoxicillin was changed to azithromycin. Two days thereafter, he presented to his Allergy clinic for routine follow-up visit of his AD. The child was noted to be irritable and drooling, with mild gingival bleeding along the dental conjunction. He showed no signs of stomatitis or impending respiratory compromise. He also had a generalized papular targetoid rash (Fig. 1A) and several erythematous lesions with central ulceration on his fingers (Fig. 1B). In addition, the patient had three mildly denuded epidermal breakdown lesions measuring one by two millimeters around his anal mucosa. Due to concern for erythema multiforme major possibly secondary to antibiotic use, as well as significant decreased oral intake the past few days from mouth pain, the patient was admitted to the hospital for close observation, intravenous rehydration, and workup for the etiology of his symptoms.

Upon admission to the hospital, the patient's azithromycin was discontinued, and intravenous corticosteroid, methylprednisolone at 1 mg/kg/day, was started. His blister lesions on the fingers were initially thought to be targetoid lesions of erythema multiforme major. However, due to his underlying atopic dermatitis, which is a risk factor for herpes simplex virus (HSV) infection (1), and the frequent association of HSV with erythema multiforme (2), HSV PCR was obtained from his venous blood and a finger blister. His fever resolved in two days and his rash continued to improve on methyprednisolone. Both his blood and finger blister HSV

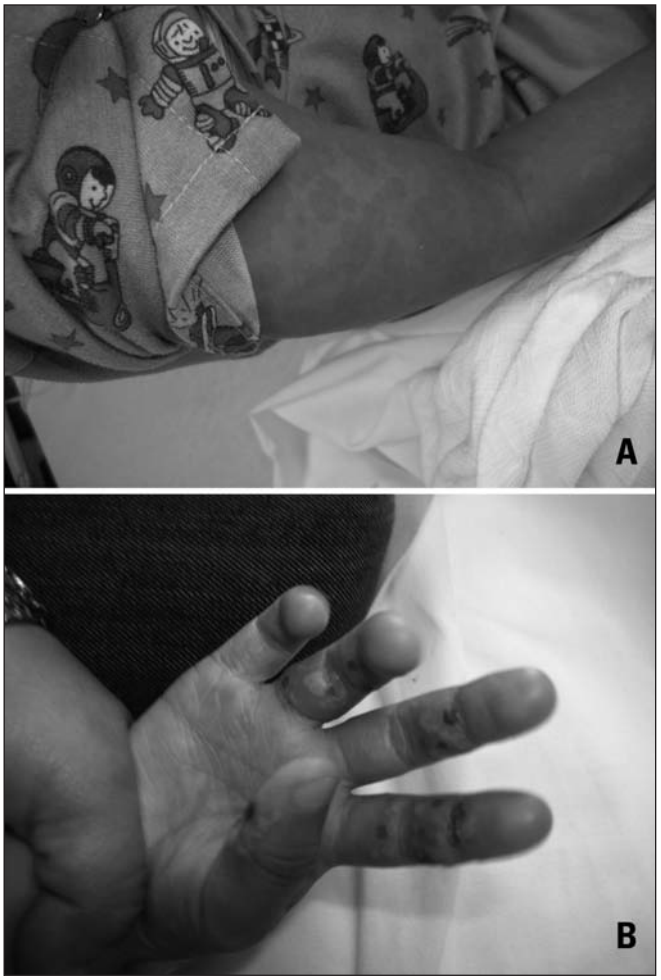
PCR came back positive on the third day of hospitalization. Thereafter, he was started on four days of intravenous acyclovir before transitioning to another six days of oral therapy. His ophthalmic exam and other work-up including mycoplasma IgM were within normal range. All of his symptoms improved by the day of hospital discharge.

Discussion

Patients with AD are known to have skin barrier defects and reduced antimicrobial peptide production that predispose them to develop eczema herpeticum (3, 4). For example, filaggrin is a monomeric protein that plays an important role in establishing appropriate barrier function and water retention in the stratified epithelium; filaggrin deficiency is associated with atopy, recurrent skin infections, and eczema herpeticum (3). Additionally, claudin-1 (CLDN1) is a tight junction adhesive protein that regulates the passage of water, solutes, and viral particles through the stratum granulosum layer of the epidermis (5). Human keratinocytes with knockdown expression of CLDN1 via siRNA demonstrate increased in vitro susceptibility to HSV-1 infectivity, and specific single nucleotide polymormphisms in the CLDN1 gene loci have been linked to the European American population with a history of confirmed eczema herpeticum diagnosis (5). Furthermore, the level of expression for antimicrobial peptides, such as cathelicidin, is significantly reduced in patients with eczema herpeticum, and cathelicidin knockout (Cnlp-/-) mice exhibit impaired antiviral killing activity and a greater risk for developing HSV infection (4). These recent research findings demonstrate that AD patients are highly vulnerable to complications from HSV via multiple mechanisms.

To our knowledge, this is the first reported case of a patient with atopic dermatitis presenting with concurrent erythema multiforme and eczema herpeticum. Given that our patient has received amoxicillin several times in the past without any adverse reaction, the erythema multiforme was likely to be secondary to HSV rather than the antibiotic use. The eczematous lesions on his fingers possibly served as the entry point for the HSV and led to viremia and erythema multiforme. Our case illustrates the need for maintaining a high index of suspicion and vigilance for HSV infection when erythema multiforme is seen, especially for patients with atopic dermatitis. Early recognition of this risk may prevent potential grave consequences of HSV septicemia.

Figure 1 - A) Patient's macular rash with central clearing. B) Blisters with central ulceration on patient's palm.



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