Eczema Herpeticum and Impetigo: What's the Common Underlying Problem?

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Disruption in the epidermal barrier creates susceptibility to bacterial and viral colonization that can range in severity from benign to dermatologic emergency.

Eczema herpeticum. Figure 1 is a 17-year-old male with atopy and a history of childhood eczema who presents with acute onset of punched out lesions on the lower lip, buccal mucosa, and the skin of the neck extending down to his right shoulder. Some lesions on the lower lip had a golden crusted appearance. A few days before presenting to the clinic, the patient experienced fever with body aches that lasted for three days. He noticed sores on his lips and was treated with docosanol topical cream. Lymphadenopathy subsequently developed along with a rash starting at the patient’s lower lip and extending down his neck. Neglected, untreated eczema was noted on the neck flexors and elbow flexors. The punched out lesions were secondary to eczema herpeticum, a skin dissemination of herpes. The yellow golden crusts were secondary to impetigo, a bacterial infection. Oral valacyclovir, topical mupirocin, and triamcinolone ointment 0.1% were prescribed, and contact contagion precautions were discussed with the patient. On follow-up at day two, the punched out lesions had healed and systemic complaints resolved, but golden crusts were still evident, consistent with persistent impetigo, which was treated with systemic Keflex. Three days later, all lesions were healing and no new lesions were visible. The areas with eczema improved as well.
Nonbullous impetigo. **Figure 2** is an 8-year-old boy with history of eczema first documented at age 5 years and treated in the past with topical steroids. One week ago, he was diagnosed with a strep throat infection and given an oral antibiotic. At that time a new rash and “eczema” appeared on the right cheek. These “raw” red areas were consistent with nonbullous impetigo. A topical mid-potency steroid ointment and mupirocin ointment were added to his treatments and his rash resolved within 48 hours.

**Question 1:** What is the common underlying pathophysiology that results in the two skin diseases (herpes viral and impetigo bacterial infections) seen here?

A. Genetic immunosuppression  
B. Skin barrier dysfunction  
C. Local skin immunosuppression by topical steroid use  
D. Skin colonization by aggressive bacteria or virus  
E. HIV infection

**Answer:** B. Skin barrier dysfunction

**Question 2:** Why would a topical steroid be applied to skin with herpes or bacterial infection?

A. To prevent scarring  
B. To decrease itch  
C. To repair the skin barrier  
D. To decrease erythema  
E. To avoid allergic contact dermatitis to mupirocin

**Answer:** C. To repair the skin barrier

**Discussion**

An intact, healthy epidermal barrier is the body’s best defense against harmful microbes, allergens, and irritants. A significantly disrupted skin barrier has been associated with increased susceptibility to microbial colonization with bacteria and viruses as well as with allergic sensitization to any chemical exposures.\(^1\) Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by recurrent eczematous lesions and changing clinical presentations; a defective skin barrier plays a major role in pathogenesis of the condition.\(^2\) Loss of function mutation in the skin barrier gene filaggrin (FLG) is responsible for defects in the protective function of epidermal barrier and is one of the strongest predictors of AD.\(^3\) FLG deficiency results in increased transepidermal water loss and decreases acidity of the stratum corneum, both of which create favorable conditions.
for Staphylococcus aureus to express proteins involved in colonization and immune invasion.\(^4\,^5\) Additionally, lack of FLG has been shown to confer a high risk for AD eczema herpeticum—a dangerous disseminated cutaneous herpes simplex virus-1 (HSV-1) infection.\(^6\) The bacteria and viruses that colonize in eczema are not specifically virulent, but in the setting of a defective skin barrier are given the opportunity to grow. Local use of topical steroids or immune modulators (eg, tacrolimus ointment), can cause local immunosuppression and possibly lead to local skin infection. However, in the context of eczema, use of these agents combined with anti-infectives is often essential for complete clearance of the lesions.

**Eczema herpeticum (EH),** also known as Kaposi’s varicelliform eruption, is a disseminated cutaneous HSV-1 infection that can arise in patients with conditions affecting the integrity of the skin barrier. EH most commonly occurs among infants and children with atopic dermatitis, but it can also be seen in other skin conditions where barrier dysfunction is seen, such as burns, pemphigus, ichthyosis, mycosis fungoides, Darier disease, and Sezary syndrome.\(^7\) Additionally, EH can be triggered by trauma to the skin or cosmetic procedures like skin peels, dermabrasion, and laser procedures where skin integrity is compromised.\(^8\) The infection most often presents with monomorphic punched out lesions over the underlying diseased skin, but can extend into the areas of normal skin.\(^9\) These punched out lesions may persist until antiviral treatment is instituted. If treated early, they tend to heal within 2 to 4 weeks and rarely cause scarring. EH can be seen during a primary herpes infection or as a complication of recurrent outbreaks. Associated fever and lymphadenopathy have been reported. It is considered a dermatologic emergency, since it can be complicated by bacterial superinfection (as in the case above), sepsis, and death. EH is treated with oral antiviral therapy. Individuals are contact-contagious until lesions dry up. Infected fomites should be cleaned with virucidal solution to prevent further spread of infection.\(^10\)

Diagnosis of EH is usually clinical, although it can be confirmed by viral culture, direct fluorescent antibody stain, or Tzank smear. Bacterial cultures are also suggested when superimposed impetigo is suspected.\(^9\)

**Impetigo** is a common, superficial bacterial skin infection that typically affects children younger than age 6 years but may also affect adolescents and adults. Impetigo is often associated with a skin barrier dysfunction such as trauma to the skin, insect bites, chickenpox, burns, and as in the case presented here, atopic dermatitis. The disrupted skin barrier allows for the growth of S. aureus and Streptococcus pyogenes, the two causative agents. The most common sites of infection are the face, especially the area around the mouth, nose, and neck. Impetigo is highly contagious, spreading through person-to-person contact as well as through fomites. Adults often acquire impetigo through close contact with infected children.\(^7\) The infection is most prevalent in geographic areas with high humidity and most cases are seen during the summer and fall.\(^7\) To reduce the chances of spreading the infection affected children should use clean towels, have linen changed daily, wear different clothes daily, and refrain from school and sports contact until the crusts dry out.\(^11\) Many schools and athletic organizations have regulations that restrict athletic involvement of infected individuals; these include at least 3 days of topical treatment for localized eruptions or topical treatment combined with oral antibiotic for more widespread infection; no new eruptions 48 hours before athletic participation; and proper drying of all lesions before returning to practice.\(^10\)

**Non-bullous impetigo** presents as erosions that begin as fragile vesicles that rupture and leave lesions with what is often described as “honey-colored crust.”\(^7\) Untreated impetigo usually heals in 2 to 4 weeks. Most patients recover without any scarring, however, there can be slight hypopigmentation seen in children with darker skin tone. Impetigo is usually diagnosed clinically. Bacterial culture can confirm the diagnosis. Treatment involves cleansing the lesions and using moist soaks to remove the overlying crusts. Antibiotic ointment can be applied to the affected areas. Systemic antibiotics are prescribed to treat more extensive infections.\(^7\,^12\) Treatment of the underlying skin barrier dysfunction, with a topical steroid or immune modulator, is important to optimize healing.

**References:**


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