

BULLOUS IMPETIGO: A MIMICKER OF IMMUNE-MEDIATED DERMATOSES

Carolyn A. Hardin¹; James Jones¹; Laura Chachula²; James Neiner¹

¹Department of Dermatology, San Antonio Military Medical Center, San Antonio, Texas

²Task Force Sinai Dispensary, Multinational Force & Observers, Sinai Peninsula, Egypt

Abstract

The differential diagnosis of vesiculobullous lesions is immense. Some entities such as bullous impetigo may be diagnosed clinically if the patient's demographics as well as the lesion morphology and distribution present classically; however, atypical presentations may require additional laboratory studies for confirmation. We describe a non-classic case of bullous impetigo that clinically mimicked two rare immunobullous dermatoses, pemphigus foliaceus and linear IgA.

Case Report

An otherwise healthy 18 year-old African-American male in basic military training was referred to an outpatient dermatology clinic for evaluation of the acute onset of mildly pruritic blisters on the back. Examination revealed polycyclic plaques with central desquamation and light crusting, some with an intact rim of flaccid bullae, distributed diffusely on the back (Figures 1 and 2). Oral and genital mucosa demonstrated no lesions and there was no regional adenopathy. A full review of systems was negative, although the patient endorsed a mild cold several weeks prior to this eruption.

{Figure 1. Diffuse polycyclic plaques with central desquamation and light crusting, some with an intact rim of flaccid bullae.}

The differential diagnosis included atypical pityriasis rosea, bullous tinea, bullous impetigo, linear IgA bullous dermatosis, and pemphigus foliaceus. Initial diagnostic studies included potassium hydroxide preparation (KOH prep) of a representative plaque on the back, superficial wound culture of bullae fluid, 3mm lesional punch biopsy for H&E, 4mm perilesional punch biopsy for direct immunofluorescence (DIF), and serum desmoglein antibodies.

{Figure 2. A representative lesion is shown with biopsy sites demarcated. Note the intact bullae at the periphery.}

KOH prep was negative for fungal elements. Lesional punch biopsy with H&E staining demonstrated mild non-specific findings including minimal spongiosis, superficial perivascular lymphocytic infiltrate and dermal pigment incontinence; notably, the majority of the stratum corneum was absent and a few cocciform bacterial colonies were present within an intact hair follicle, without associated acantholysis. Direct immunofluorescence of perilesional skin was negative for IgG, IgA, IgM, C3, and fibrin. Culture of fluid obtained from intact bullae yielded heavy growth of methicillin-sensitive staphylococcus aureus.

On the basis of these findings the patient was diagnosed with bullous impetigo and, given the extent of disease, prescribed a 10 day course of oral cephalexin 500 mg twice daily for ten days. The patient was re-evaluated two weeks after his initial presentation in the dermatology clinic. He reported the crusts and bullae resolved within a few days of starting oral cephalexin; he successfully completed the entire course without adverse effects. On repeat examination, the primary lesions had resolved, leaving behind a striking annular pattern of post-inflammatory pigmentary alteration. Areas of central hypopigmentation and peripheral rims of hyperpigmentation remained as the shadow of the previous crusts and flaccid bullae, respectively (Figure 3).

{Figure 3. Resolution of lesions following treatment with cephalexin resulted in prominent post-inflammatory alteration. Arrowheads designate healing biopsy sites.}

Discussion

Impetigo is a common, highly contagious bacterial skin infection most frequently observed in children, although it can occur in any age group.¹ Bullous impetigo is less common than crusted (non-bullous) impetigo and almost exclusively results from infection with *Staphylococcus aureus*.¹⁻³ The carriage of *Staphylococcus* is the most important risk factor but other factors have been recognized including warm temperature, humidity, skin trauma, and participation in contact sports.¹ Bullous impetigo typically presents with localized vesiculopustules containing clear yellow fluid that later become confluent, resulting in bullae. Central collapse of bullae promotes development of thin yellow-brown crusts often with a collarette of scale. This clinical appearance is due to epidermal acantholysis related to virulence factor exfoliative toxin A which targets desmoglein 1. Desmoglein 1 is also the implicated antigen in staphylococcal scalded skin syndrome and pemphigus foliaceus.^{2,4,5}

Impetigo is generally a clinical diagnosis that classically presents in young children, located on the face or intertriginous areas. The onset of pemphigus foliaceus is commonly in middle aged adults, with crusted erosions in a seborrheic distribution. Linear IgA bullous dermatosis may present in both children and adults with annular vesicles or bullae. In children, the lesions often appear on the abdomen, thighs, and groin; whereas in adults, lesions have a predilection for the extensor extremities, face, and trunk. Thus, the case patient's demographics, morphology, and distribution prompted an expanded work-up to rule out the aforementioned immunobullous dermatoses, outlined in Table 1, below.

In certain high risk adult patient populations, bullous impetigo may suggest an underlying immunosuppressive condition, such as human immunodeficiency virus (HIV).⁶ It was postulated that this patient's participation in close quarters combat training – predisposing to moist skin, minor skin abrasions, and skin-to-skin contact – may have contributed to his development of skin disease.

Impetigo is a self-limiting disease that typically resolves without scarring in 3 to 6 weeks, but treatment is recommended due to its highly contagious nature.^{1,7} Localized disease is treated topically with empiric agents such as mupirocin that are active against both *Staphylococcus aureus* and group A *Streptococcus*.⁷ It is recommended that extensive disease, however, be treated orally with agents such as first generation cephalosporins, clindamycin, or extended spectrum penicillins.⁷ Despite the low incidence of community acquired methicillin resistant staph aureus in impetigo, bacterial cultures are recommended prior to the initiation of oral antibiotic therapy.^{3,7,8} Unlike the bullae of staphylococcal scalded skin syndrome, fluid collected from bullous impetigo may indeed grow bacterial cultures.³ Recurrent staphylococcal infections may indicate carriage in the nares, axilla, or groin, and decolonization should be considered.⁹ As systemic symptoms and regional adenopathy rarely develop during the acute infectious process, there have been rare case reports of bullous impetigo progressing to staphylococcal scalded skin syndrome following systemic dissemination of the exfoliative exotoxins.¹⁰ Luckily, the only apparent sequelae for the case patient were the post-inflammatory pigmentary changes and he was permitted to return to full duty without restriction.

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