Los Angeles Pediatric Society E-Newsletter
Volume 2 No. 3 September 2015

DERMATOLOGY CLINIC

What's Your Diagnosis?

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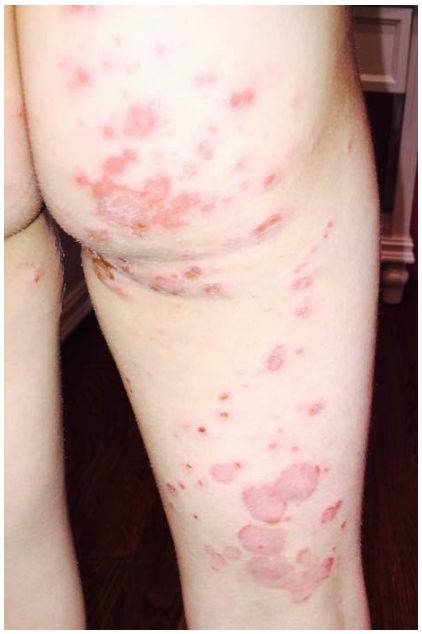
Spreading Bullae in a 7 year old Girl

Case:

A 7 year old, previously healthy, Caucasian female presents with pink, itchy, round patches, honey-crusted, weeping papules and fluid-filled bullae on her legs and buttocks. The rash began with a few, raw, excoriated, itchy pink bumps on her right buttock and upper thigh, that her parents assumed were severely scratched mosquito-bites. Within 1 week, she developed blisters and crusted papules that spread to her waist, down her right leg, and to her left leg. She feels well. She has no fevers, leg swelling, abdominal pain, tea-colored urine or recent strep pharyngitis. She has no history of gluten-insensitivity. She has no history of allergies, hives, respiratory distress or anaphylaxis. She has no personal history of herpes simplex virus (HSV) infection, but her mom does get intermittent cold sores on her lip.

What is your diagnosis?

1. Dermatitis Herpetiformis
2. Linear IgA Bullous Disease
3. Bullous Impetigo
4. HSV-infection
5. Staph Scalded Skin Syndrome



Answer: 3 – Bullous Impetigo

Impetigo is a common skin infection seen in infants and children. It is caused by either *Staph aureus* (*S. aureus*) or *Streptococci*, primarily group A *B*-hemolytic streptococcus (GABHS). It is highly contagious. It is spread by either direct contact with weeping, crusted lesions or by hand contact and respiratory spread to susceptible individuals by *S. aureus* carriers. Patients are generally well, with no fevers, malaise, pain or coryza. Risk factors for infection in children, include broken skin barrier secondary to eczema, excoriated bug bites or open sores due to minor trauma; direct contact with other infected children at home, school and sports, including shared equipment; and nasal or perineal carriage of *S. aureus* in a parent or caregiver. Up to 40% of healthy adults and up to 80% of adults with atopic dermatitis are asymptomatic carriers. *S. aureus* is transmitted by both hand and droplet transmission onto broken skin. Most cases resolve quickly and uneventfully with topical or oral antibiotics. Potential complications can include: sepsis, pneumonia, septic arthritis, osteomyelitis, lymphadenitis, guttate psoriasis, scarlet fever and post-streptococcal glomerulonephritis.

There are 2 classic presentations of impetigo: non-bullous (crusted) impetigo, and the less common bullous version. Non-bullous impetigo accounts for over 70% of cases. It starts as a small pink bump or pustule on the face, neck, hand or extremity that develops into a red-rimmed, thin-walled blister. This blister quickly ruptures, and the cloudy, yellowish fluid dries into a honey-colored crust. This crust is infectious. The lesions are spread by auto-inoculation (scratching, clothing, towels) or to others (touching, shared towels, equipment.) In the past, non-bullous impetigo was almost always due to GABHS infection; today, it is more commonly due to *S. aureus* infection. In contrast, bullous impetigo is almost always associated with *S. aureus*. It presents as flimsy, fluid-filled blisters, or shallow sores surrounded by the edges of an open blister. The lesions can be tender, and are more commonly found on the legs and diaper area, but may also be seen on the face, hands and arms. The bullae are caused by the local release of *S. aureus*-produced exfoliative toxins, ETA and ETB. Technically, bullous impetigo is a localized, mild version of Staphylococcal Scalded Skin Syndrome, seen in neonates and young children.

Diagnosis is made clinically. Skin swabs for bacterial (and sometimes viral) cultures with antibiotic sensitivities are generally reserved for recurrent or severe, treatment-resistant cases.

Treatment of impetigo will depend on severity of presentation. Without intervention, the lesions will persist and spread for weeks. Local wound care, including gentle cleansing, removal of crusts and/or drainage of bullae may help dry the lesions and prevent further spread of the bacteria. Topical antibiotics can be sufficient for milder, localized disease caused by *S. aureus*. For more extensive cases, and for infection with GABHS, oral antibiotics that cover both *S. aureus* and GABHS are recommended. For recurrent cases, consider topical treatment for intranasal *S. aureus* carriage. For severe, recalcitrant cases, consider bacterial culture and sensitivity testing for possible CA-MRSA infection or regional antibiotic resistance.

Impetigo due to cutaneous GABHS is not associated with development of post-streptococcal rheumatic fever. However, post-strep scarlet fever and acute glomerulonephritis (AGN) can occur, albeit rare. Similar to strep pharyngitis, only specific nephrogenic strains of GABHS can cause AGN, and systemic treatment of cutaneous GABHS infection cannot prevent it from occurring.

Differential Diagnoses:

Dermatitis Herpetiformis (DH) is the cutaneous manifestation of celiac disease, an autoimmune bullous disease in which IgA antibodies are produced against gliadin-bound epidermal transglutaminase in the skin (akin to circulating tissue transglutaminase antibodies in celiac disease). Patients develop symmetrically distributed, itchy, grouped pink papules that often appear crusted and bloody from scratching. The rash is classically found on the posterior neck, forearms/elbows, knees and buttocks. Approximately 20% of patients with DH have gastrointestinal symptoms. Diagnosis requires biopsy. Treatment begins with a gluten-free diet.

Linear IgA Bullous (LABD) is another IgA-mediated autoimmune disease, in which 'jewel-like', grouped and annular patterns of blisters form on the trunk, buttock, legs and sometimes face. In children, it can resolve spontaneously within 2-4 years. Diagnosis requires biopsy. Treatment involves oral anti-inflammatories, including dapsone.

Herpes simplex virus (HSV) can cause painful, grouped blisters that turn into erosions or shallow ulcerations with scalloped borders and hemorrhagic crusts. Widespread eruptions can occur in susceptible patients, including those with lowered immune systems and those with skin conditions that create a broken skin barrier: atopic dermatitis, burns. Initial lesions may present with fever, malaise, lymphadenopathy and pain. Treatment for extensive lesions includes oral anti-viral medications.

Staphylococcal Scalded Skin Syndrome (SSSS) is, technically, a more widespread version of bullous impetigo. Seen in neonates, infants and young children, it is a blistering disease caused by a *S. aureus*-produced exfoliative toxin (ETA or ETB) that cleaves desmoglein 1, an adhesion molecule in the epidermis, creating thin-walled blisters and skin sloughing. It is transmitted by asymptomatic adult carriers, or seeded from other staph infections, including pneumonia, septic arthritis and endocarditis. It usually begins with conjunctivitis or a few crusts on the perioral, perineal or umbilical areas. Patients then develop fever, lethargy, irritability and poor feeding, as a prodrome to widespread skin redness, followed by the eruption of thin-walled blisters that burst and leave behind sore, peeled skin. There is a predilection for the face and neck, especially the skin folds. It spares the mucosal membranes. Diagnosis is confirmed by isolation of *S. aureus* from the skin, nares or conjunctivae. The blister fluid itself is sterile, since the blisters are caused by the exotoxin. Skin biopsy is necessary sometimes. Treatment includes anti-staph antibiotics. Older children with mild disease may be treated at home; younger children with more severe disease require hospitalization.

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