Tender Honey-Colored Lesion With "Stuck-on" Crust

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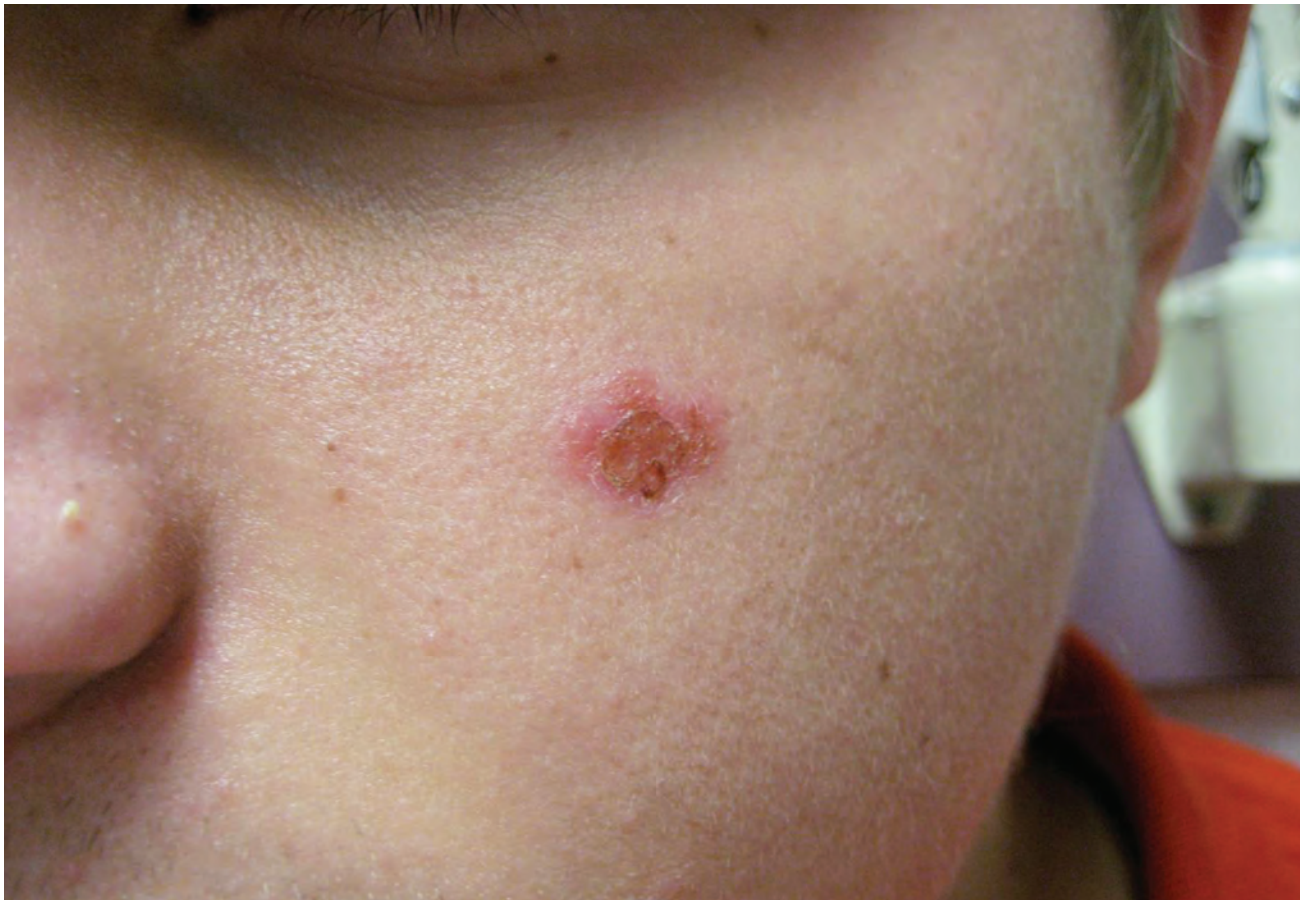

HISTORY

A 5-year-old boy presented with a lesion on his left cheek. It began a week earlier as a small erythematous macule that later became excoriated and had been enlarging. The originally mildly pruritic lesion had become tender.

Past health unremarkable. No personal or family history of skin problems.

PHYSICAL EXAMINATION

An erythematous, eroded plaque was observed on the left cheek. A yellow-brown "stuck-on" crust covered the superficial erosion. Remaining examination findings normal.

WHAT'S YOUR DIAGNOSIS?

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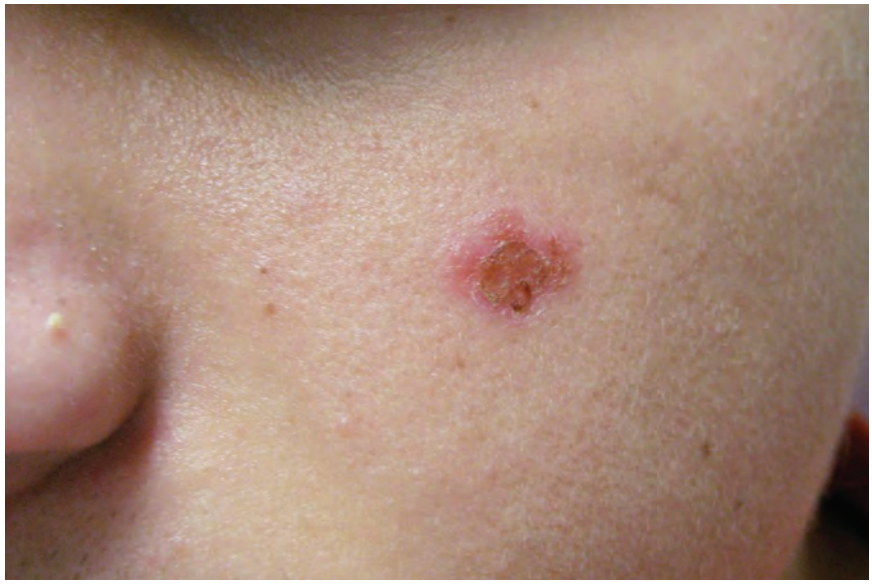
ANSWER: Impetigo



Impetigo is a superficial, highly contagious bacterial infection characterized by a localized inflamed and infected epidermis.[1] It is the most common bacterial skin infection in children and accounts for approximately 10% of all skin problems in children.[2] The condition was first described by Fox in 1864 as "circular, umbilicated quasi-bullous spots which increase centrifugally, and become covered by yellow flat crusts which cover over superficial ulceration."[3]

Of the 2 classic forms of impetigo—nonbullous impetigo (also known as impetigo contagiosa or crusted impetigo) and bullous impetigo—nonbullous impetigo, as illustrated in the present case, accounts for more than 70% of cases.[2]

EPIDEMIOLOGY

The annual incidence of impetigo is estimated to be 2.8% in children up to 4 years of age and 1.6% in those aged 5 to 15 years.[4] Thereafter, the incidence decreases rapidly with age.[5] Nonbullous impetigo most frequently occurs in children aged 2 to 5 years, while bullous impetigo occurs primarily in newborns and infants.[6-8]

Impetigo is transmitted to other persons mainly by direct contact and to oneself by autoinoculation; it can also be spread by fomites.[4,9] Bullous impetigo tends to be less contagious than nonbullous impetigo.[4,6] There is no difference in prevalence of infection in male or female patients.[5] Impetigo is more common in hot humid climates or during the summer months in temperate areas.[4,7]

Predisposing factors include poor hygiene, crowded living conditions, skin abrasions, minor trauma, insect bites, burns, atopic dermatitis, diabetes mellitus, intravenous drug abuse, and immunodeficiency.[7,9] Children with nasal *Staphylococcus aureus* colonization are at increased risk for impetigo.[9]

ETIOLOGY AND PATHOGENESIS

S aureus causes nonbullous impetigo in about 70% of cases; the second most common cause is group A β-hemolytic streptococci.[1,9] Pathogenic strains of *S aureus* almost always cause bullous impetigo.[1,5,6] Approximately 80% of *S aureus* are from phage group 2.[2] This group of organisms produces exfoliative toxins specific for desmoglein 1.[9] Hydrolysis of the amino-terminal extracellular domains of desmoglein 1 by staphylococcal exfoliative toxins results in cleavage within the stratum granulosum, which leads to bulla formation.[10]

CLINICAL MANIFESTATIONS

Nonbullous impetigo typically begins as a small, 2- to 4-mm, erythematous macule that soon becomes vesicular.[4,11] The vesicle then ruptures, leaving an exudate with a characteristic yellowish brown or honey-colored "stuck-on" crust over the superficial erosion.[11] Removal of the crust results in the reaccumulation of fresh exudate. Satellite lesions typically appear in the vicinity as a result of spread by autoinoculation.[4] Coales-

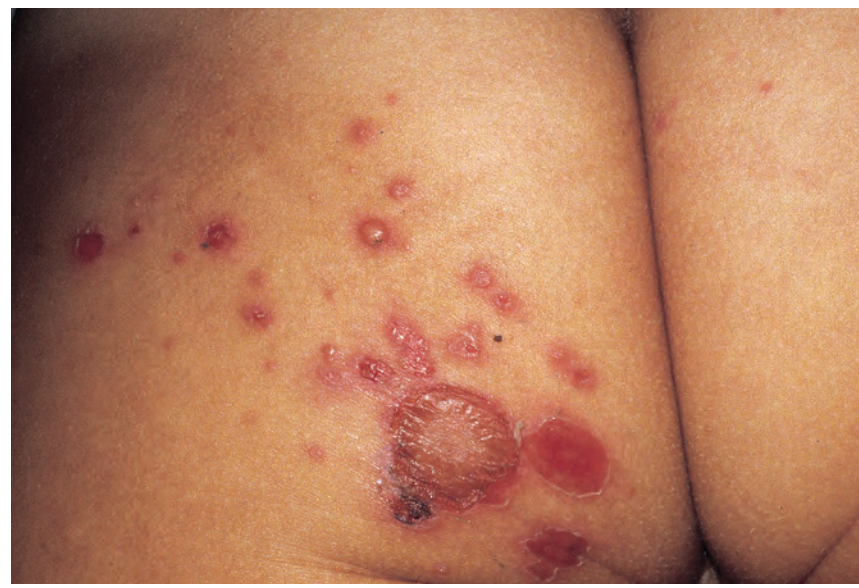


Figure – The superficial, red-based ulcerations on the left buttock of a 3-year-old girl are characteristic of bullous impetigo. *(Photo courtesy of Robert P. Blereau, MD.)*

cence of lesions produces a wider area of involvement. There is little erythema associated with the lesion.[12]

Nonbullous impetigo most commonly occurs on the face, followed by the extremities.[9] Constitutional symptoms, such as fever, malaise, and anorexia, are generally absent.[2] Mild pruritus or pain may be noted. Regional lymphadenopathy may be present.[7]

The initial lesion of bullous impetigo is a faint red macule that rapidly develops into a distinct vesicle and then a flaccid, painless bulla.[1] The bulla is sharply demarcated without surrounding erythema and measures 1 to 5 cm in diameter. The fluid may be clear or turbid. Rupture of the bulla usually reveals a moist, erythematous base that dries to form a shiny lacquer-like appearance (**Figure**).

A pathognomonic finding is a narrow rim of scale at the edge of the ruptured lesion.[2,4] Sites of predilection include the face and moist, intertriginous areas, such as the diaper area, axillae, and neck fold.[2,4,9] Fever may be present but regional lymphadenopathy is uncommon.[13] The Nikolsky sign is characteristically negative.

DIAGNOSIS

The diagnosis is mainly clinical. Consider Gram stain and culture of exudates from the lesion when the diagnosis is in doubt, when the patient appears ill, or when methicillin-resistant *S aureus* (MRSA) is suspected. Culture may also be necessary during outbreaks of poststreptococcal glomerulonephritis to identify patients with nephrogenic strains of group A β-hemolytic streptococci.[4]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of nonbullous impetigo includes herpes simplex infection, varicella, insect bites, seborrheic dermatitis, atopic dermatitis, contact dermatitis, candidiasis, tinea corporis, erythema annulare centrifugum, and scabies—all of which may become impetiginized.[2,4,11] On the other hand, the differential diagnosis of bullous impetigo includes bullous insect bites, thermal burns, allergic contact dermatitis, epidermolysis bullosa, bullous mastocytosis, Stevens-Johnson syndrome, bullous erythema multiforme, bullous tinea, bullous pemphigoid, subcorneal pustular dermatosis, and pemphigus vulgaris.[2,4,11]

COMPLICATIONS

Most cases resolve without sequelae in 2 to 3 weeks.[4] Once healed, no scarring results.[12] In about 1% to 5% of patients with nonbullous impetigo caused by nephrogenic strains (serotypes M49, M55, M57, M59) of group A β-hemolytic streptococci, acute poststreptococcal glomerulonephritis develops 2 to 3 weeks later.[4,14] Guttate psoriasis and scarlet fever may follow nonbullous impetigo caused by group A β-hemolytic streptococci.[2] Rheumatic fever does not occur as a result of streptococcal skin infection. Cellulitis, lymphangitis, and suppurative lymphadenitis may complicate nonbullous impetigo but rarely follow bullous impetigo.[2]

Potential but rare complications of either nonbullous impetigo or bullous impetigo include septic arthritis, osteomyelitis, necrotizing fasciitis, septicemia, and pneumonia; these complications occur mainly in immunocompromised persons.[2,6]

MANAGEMENT

The aims of treatment are to relieve discomfort, improve cosmetic appearance, and accelerate clinical cure thereby preventing recurrence and spread of infection in the patient and to others.[4] Local care includes cleansing and gentle debridement of crusts with warm soaks. Patients should be reminded not to scratch or touch the scabs. The importance of frequent hand washing and meticulous hygiene cannot be overemphasized.

It has been shown that topical antibiotics are significantly better than disinfecting treatments, such as bleach baths.[15] For mild infections, such as in this patient, a topical antibiotic efficacious against gram-positive bacteria, especially *S aureus* and group A β-hemolytic streptococci, is the preferred first-line therapy.[5,13,15] The topical antibiotic agents mupirocin and fusidic acid, which is not available in the United States, have similar efficacy and are applied 3 times a day.[12,15] Topical retapamulin, a derivative of the antibacterial pleuromutilin, applied twice daily is another treatment option.[5,8]

Severe, widespread, or recurrent infections require oral antimicrobials effective against *S aureus* and group A β-hemolytic streptococci. The medications of choice include dicloxacillin, amoxicillin/clavulanic acid, clarithromycin, azithromycin, and cephalosporins (such as cephalexin).[2,7,9] In communities with a high prevalence of MRSA, clindamycin, trimethoprim/sulfamethoxazole, and fluoroquinolones are appropriate.[9] The course of treatment is 7 to 10 days. ■

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