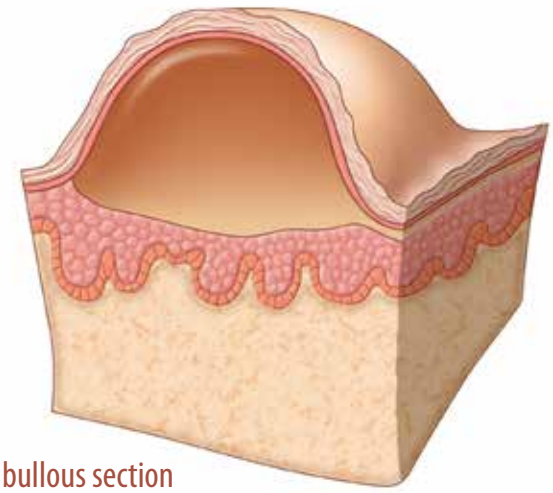
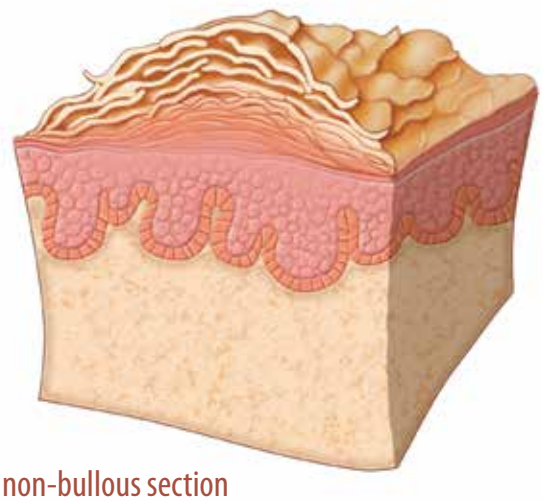
CASE STUDY

A Moustache for a Good Cause

Pre-test CME Quiz



bullous section



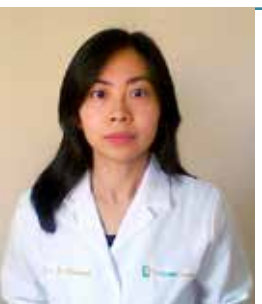
non-bullous section

ABSTRACT

Impetigo is a gram-positive bacterial infection of the superficial layers of the epidermis. There are two forms of impetigo: bullous and nonbullous. Diagnosis of impetigo is usually based solely on the history and clinical presentation. Culture and sensitivity results can help the physician choose appropriate antibiotic therapy. Treatment of impetigo typically involves local wound care, along with antibiotic therapy, either topical alone or in conjunction with systemic therapy. For mild or localized cases, topical mupirocin or topical fusidic acid applied 2 to 3 times daily for 7 to 10 days are adequate treatment. Systemic antibiotics are indicated for widespread, complicated, or severe cases associated with systemic manifestations of impetigo. Beta-lactam antibiotics remain an appropriate initial empiric choice, with coverage against both Staphylococcus aureus and Streptococcus pyogenes. For patients with recurrent impetigo or Staphylococcus aureus nasal carriers, topical mupirocin cream or ointment can be applied inside the nostrils 3 times daily for 5 days each month to reduce colonization in the nose.

KEYWORDS: Impetigo, Staphylococcus aureus, Group A beta hemolytic streptococci Bullous impetigo, Nonbullous impetigo

A 19-year-old male presents with a 4-day history of erythematous plaques covered with much thickened yellowish crusting on the moustache area. This patient grew a moustache for the first time in support of a November men's health fundraising event. His symptoms started after he dyed his moustache 4 days ago. His 7 housemates all participated in the same event and dyed their moustaches. None of them developed the same symptoms. The patient was initially treated with



ABOUT THE AUTHOR

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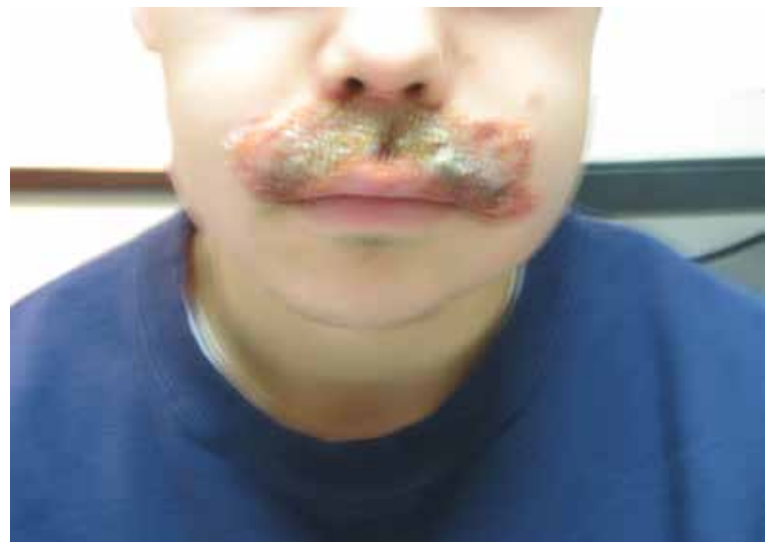
a 7-day course of prednisone 50mg once daily for the diagnosis of allergic reaction, but the symptoms got progressively worse.

What is your diagnosis?

Impetigo is a gram-positive bacterial infection of the superficial layers of the epidermis. It is the most common bacterial skin infection and accounts for approximately 10% of skin problems observed in the pediatric population.[1] It tends to affect skin on the face or extremities that has been disrupted by traumas.[1] There are two forms of impetigo: bullous and nonbullous.

Nonbullous impetigo is the more common form, constituting approximately 70% of cases. It is caused by *Staphylococcus aureus* (*S aureus*), group A beta hemolytic streptococci (GABHS, also known as Streptococcus pyogenes), or a combination of both. Most infections begin as a streptococcal infection, but staphylococci replace the streptococci over time. Methicillin-resistant S aureus (MRSA), which can be hospital or community acquired, is an increasingly common cause of impetigo,[2] especially the nonbullous form. Nonbullous impetigo begins with a single erythematous macule that rapidly evolves into a vesicle or pustule and ruptures. The released serous contents then dry, leaving a crusted and honey-colored exudate over the erosion. Spread of impetigo occurs with contiguous extension or to distal areas through inoculation

Figure 1: Impetigo



from scratching.[3] Nonbullous impetigo is more contagious than the bullous type.[4] The face and extremities are most commonly affected, but skin on any part of the body can be infected by impetigo. Lesions are usually asymptomatic, with occasional pruritus. Erythema or edema is usually not present. Regional adenopathy is common.

Bullous impetigo is a toxin-mediated erythroderma in which there is a loss of cell adhesion in the superficial dermis, causing blisters and skin sloughing by cleaving of the granular cell layer of the epidermis. The blisters quickly appear, spontaneously rupture, and drain so that only the remnants are seen at the time of presentation. It may affect intact skin and is caused almost exclusively by *S aureus*.[3,4]

Intact skin is usually resistant to colonization or infection by *S aureus* or GABHS. These bacteria are introduced from the environment and transient colonization can be facilitated by factors such as high temperature or humidity, disruption of skin or preexisting cutaneous disease (e.g.

scratching, varicella, herpes simplex, scabies, thermal burns, surgery, trauma, radiation therapy, insect bites), young age, or recent antibiotic treatment. Immunosuppression by medications (eg, systemic corticosteroids or chemotherapy), systemic diseases (eg, HIV infection, diabetes mellitus), intravenous drug abuse, and dialysis encourage bacterial growth. After initial infection, new lesions may be seen in areas with no apparent break in the skin. Upon close examination, these lesions usually demonstrate some underlying physical damage however.

Approximately 30% of the population is colonized in the nares by *S aureus*; approximately 10% of individuals in the perineum and, more uncommonly, in the axillae, pharynx, and hands. Patients with atopic dermatitis or other inflammatory skin conditions more commonly have skin colonized by S aureus. Studies have shown a 60-90% *S aureus* colonization rate in patients with atopic dermatitis. Individuals who are permanent carriers serve as reservoirs of the infection for other people. Some individuals colonized by *S aureus* experience recurrent episodes of impetigo.

Diagnosis of impetigo is usually based solely on the history and clinical presentation. Bacterial culture and sensitivity are recommended to identify possible MRSA,[2] if an outbreak of impetigo has occurred, or if acute poststreptococcal glomerulonephritis (APSGN) is present.[3] Gram stain and bacterial culture of the fresh exudate underneath the scab may be obtained. On Gram stain, the presence of gram-positive cocci in chains indicates *Streptococcus pyogenes*; gram-positive cocci in clusters indicate S aureus. Culture and sensitivity results can help the physician choose appropriate antibiotic therapy. A bacterial culture of the nares may be obtained to establish whether a patient is a *S aureus* carrier. If the nares culture is negative and the patient has recurrent episodes of impetigo, bacterial cultures should be obtained from the axillae, pharynx, and perineum.

Alternative diagnostic possibilities are key to consider in recurrent cases or those that do not respond to treatment. Tinea and herpetic impetigo are common conditions that mimic impetigo.[5,6,7,8]

Patients who developed impetigo have a antistreptolysin O (ASO) serologic response. Antideoxyribonuclease B (anti-DNase B) and antihyaluronidase (AH) titers are useful to establish a recent streptococcal skin infection in the differential diagnosis of APSGN, because only 51% of patients with impetigo-associated APSGN develop an increased ASO titer.

A potassium hydroxide wet mount may be performed to exclude bullous dermatophyte infection. A Tzanck preparation or viral culture may be performed to exclude herpes simplex infection.

Impetigo usually resolve within 7-10 days with appropriate treatment. Cultures should be performed

SUMMARY OF KEY POINTS

Impetigo is the most common bacterial skin infection and accounts for approximately 10% of skin problems observed in the pediatric population.

There are two forms of impetigo: bullous and nonbullous.

Nonbullous impetigo is the more common form of impetigo, constituting approximately 70% of cases. It is caused by *Staphylococcus aureus*, group A beta hemolytic streptococci, or a combination of both.

Bullous impetigo is a toxin-mediated erythroderma, causing blisters and skin sloughing by cleaving of the granular cell layer of the epidermis. It is caused almost exclusively by *S aureus*.

Diagnosis of impetigo is usually based solely on the history and clinical presentation. Bacterial culture and sensitivity are recommended to identify possible MRSA, if an outbreak of impetigo has occurred, or if acute poststreptococcal glomerulonephritis (APSGN) is present.

Treatment of impetigo typically involves local wound care, along with antibiotic therapy, either topical alone or in conjunction with systemic therapy.

Post-test CME Quiz

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to rule out resistant organisms in persistent cases.[8] Cellulitis, lymphangitis, and suppurative lymphadenitis may occur in as many as 10% of patients with impetigo. Scarring is unusual, but postinflammatory hyperpigmentation or hypopigmentation may occur. Untreated lesions of nonbullous impetigo may rarely progress to ecthyma, a deep dermal infection, after which subsequent scarring can occur. Staphylococcal scalded skin syndrome can result if the exfoliative toxins are absorbed into bloodstream. APSGN is a rare complication of nonbullous impetigo from nephritogenic strains of GABHS (for example, serotypes 49, 55, 57, 59), with an annual incidence of less than 1 case per 1,000,000 population in developed countries.[9,10,11] Treatment of impetigo with systemic antibiotics does not prevent the development of APSGN, most likely because activation of the immune response precedes antibiotic treatment. Other complications of impetigo include scarlet fever, erysipelas, guttate psoriasis, pneumonia, osteomyelitis, septic arthritis, bacterial endocarditis, and septicemia.[1,12]

Treatment of impetigo typically involves local wound care, along with antibiotic therapy, either topical alone or in conjunction with systemic therapy.

Gentle cleansing, removal of the honey-colored crusts using antibacterial soap, and regular application of wet dressings to affected areas are

recommended. For mild or localized cases, topical mupirocin or topical fusidic acid applied 2 to 3 times daily for 7 to 10 days are adequate treatment. Unfortunately, S aureus and MRSA resistance to mupirocin has emerged at 5-10%.[13,14] High resistance rates of 32.5-50% have been reported with the use of fusidic acid.[15,16,17,18,19]

Systemic antibiotics are indicated for widespread, complicated, or severe cases associated with systemic manifestations of impetigo. Beta-lactam antibiotics (for example, cephalexin, amoxicillin-clavulanate, cloxacillin) remain an appropriate initial empiric choice, with coverage against both Staphylococcus aureus and Streptococcus pyogenes. Erythromycin and clindamycin are alternatives in patients with penicillin allergy. If MRSA is suspected, alternative antibiotics include clindamycin, trimethoprim-sulfamethoxazole, and vancomycin.[20]

For patients with recurrent impetigo or *S aureus* nasal carriers, topical mupirocin cream or ointment can be applied inside the nostrils 3 times daily for 5 days each month to reduce colonization in the nose.

All of the tables and photos are original.

No competing financial interests exist in preparation of this case study.

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