

Scratching the surface of impetigo

Impetigo is a common, highly contagious, superficial skin infection that primarily affects children. Most lesions occur on the face, however, other body surfaces can also be affected. Impetigo tends to start as small blisters, which becomes filled with pus. These lesions rupture and the purulent exudate dries to form golden coloured crusts. These lesions can be very infectious. Secondary skin infections of existing skin lesions (e.g., cuts, abrasions, insect bites, chickenpox, eczema) can also occur, leading to an acute, disseminated impetigo.

Impetigo is commonly caused by *Staphylococcus aureus* bacteria, *Streptococcus pyogenes* or mixed infections. Methicillin-resistant *S aureus* (MRSA) and gentamicin-resistant *S aureus* strains have also been reported to cause impetigo. Impetigo is classified as either non-bullous (impetigo contagiosa) (about 70% of cases) or bullous types.

Signs and symptoms

Children with nonbullous impetigo commonly have multiple coalescing lesions on their face (perioral, perinasal) and extremities or in areas with a break in the natural skin defence barrier. The initial lesions are small vesicles or pustules (<2cm) that rupture and become a honey-coloured crust with a moist erythematous base. Pharyngitis is absent, but mild regional lymphadenopathy is commonly present. Nonbullous impetigo is usually a self-limiting process that may resolve within two weeks.

Bullous impetigo is considered to be less contagious than the non-bullous form. It tends to affect the face, extremities, axillae, trunk, and perianal region of neonates, but older children and adults can also be affected. The initial lesions are fragile thin-roofed, flaccid, and transparent bullae (<3cm) with a clear, yellow fluid that turns cloudy and dark yellow. Once the bullae rupture, they leave behind a rim of scale around an erythematous moist base but no crust, followed by a brown-lacquered or scalded-skin appearance, with a collarette of scale or a peripheral tube-like rim.

Bullous impetigo also differs from nonbullous impetigo in that bullous impetigo may involve the buccal mucous membranes, however regional adenopathy rarely occurs. At times, extensive lesions in infants may be associated with systemic symptoms such as fever, malaise, generalized weakness, and diarrhoea. Rarely, infants may present with signs of pneumonia, septic arthritis, or osteomyelitis.

Diagnosis

The diagnosis of impetigo is usually made on the basis of the history and physical examination. However, bacterial culture and sensitivity can be used to confirm the diagnosis and are recommended in the following scenarios:

- When MRSA is suspected
- In the presence of an impetigo outbreak
- In the presence of post-streptococcal glomerulonephritis (PSGN); in such cases, urinalysis is also necessary

Biopsy may be appropriate in doubtful or refractory cases of impetigo.

Management

Treatment of impetigo typically involves local wound care in conjunction with either a topical antibiotic or a combination of systemic and topical agents. In general, the antibiotic selection has coverage against both *S aureus* and *S pyogenes*. In areas with a high prevalence of community-acquired MRSA with susceptible isolates, children older than 8 years may take clindamycin or doxycycline in cases. Trimethoprim-sulfamethoxazole can be used in situations in which group A streptococci are unlikely.

Approach considerations

Treatment of impetigo typically involves local wound care along with antibiotic therapy. Antibiotic therapy for impetigo may be with a topical agent alone or a combination of systemic and topical agents.

Gentle cleansing, removal of the honey-coloured crusts of non-bullous impetigo using antibacterial soap and a cloth, and frequent application of wet dressings to areas affected by lesions are recommended. Good hygiene with antibacterial washes, such as chlorhexidine or sodium hypochlorite baths, may prevent the transmission of impetigo and prevent recurrences, but the efficacy of this has not been proven.

For antibiotic therapy, the chosen agent must provide coverage against both *Staphylococcus aureus* and *Streptococcus pyogenes*. The prevalence of methicillin-resistant *S aureus* (MRSA) and macrolide-resistant *Streptococcus* has changed empiric treatment options for impetigo. MRSA was responsible for 78% of all community staphylococcal-related skin and soft tissue skin infections in a multicenter US study. Fortunately it is not as common in our setting.

Topical mupirocin or fucidic acid are adequate treatment for single lesions of non-bullous impetigo or small areas of involvement. Systemic antibiotics are indicated for non-bullous impetigo with extensive involvement, in athletic teams, childcare clusters, multiple family members, or for severe bullous impetigo. The use of a penicillin or macrolide, tends to work very well in community-acquired infections. However, in the presence of CA-MRSA the use of clindamycin or doxycycline in children older than 8 years can be considered. Trimethoprim-sulfamethoxazole can be used in situations in which group A streptococci are unlikely. Immunocompromised children may need oral treatment from the start.

In patients with bullous impetigo who present to the emergency department with large areas of involvement resulting in denuded skin from ruptured bullae, management also includes intravenous fluid and antibiotics. Fluid is given at a volume and rate similar to standard volume replacement for burns.

Inpatient care is required for patients with impetigo who have widespread disease or for infants at risk of sepsis and/or dehydration due to skin loss. If inpatient care is warranted in the child with untreated impetigo, contact isolation is recommended.

Sources: Medscape, Cincinnati Children's Hospital Medical Center, Clinical article supplied by Dr Rakesh Newaj.