

# Impetigo in children: a clinical guide and treatment options

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## Abstract

Impetigo is a contagious, superficial bacterial infection of the skin, most frequently encountered in children. Causative organisms are almost always *Staphylococcus aureus* or streptococci, or a combination of the two. Predisposing factors are nasal and perineal colonisation, overcrowding, poor personal hygiene, minor skin trauma and pre-existing skin diseases with disrupted skin barrier function, like eczema. Infection is mainly acquired through contact with sufferers or nasal carriers. Treatment should be given to avoid spread of the disease, and to minimise the risk of infecting others. Although the majority of cases of impetigo are self-limiting, under certain circumstances complications like toxic shock syndrome, staphylococcal osteomyelitis, septic arthritis and pneumonia can occur. Furthermore, certain strains of group A  $\beta$ -haemolytic streptococci causing impetigo may result in poststreptococcal glomerulonephritis, just like streptococcal throat infections can result in rheumatic fever in children, but the pathogenesis remains poorly understood. It appears to be due to abnormal immune response or hypersensitivity to streptococcal antigens.

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## Introduction

Impetigo is a common bacterial skin infection in children. Causative organisms are almost always *Staphylococcus aureus* or streptococci, or a combination of the two. Infection is usually acquired by skin-to-skin contact, or from contact with nasal carriers of these organisms. Impetigo is typically classified as primary or secondary. Primary impetigo occurs following a bacterial invasion of previously normal skin. In secondary impetigo, infection occurs secondary to some other underlying skin disease that disrupts the skin barrier function, such as atopic eczema, hence the term impetiginised eczema.

## Discussion

Impetigo is almost always caused by *Staphylococcus aureus* or streptococci, or a combination of the two.<sup>1</sup> Susceptibility to these infections depends on host immune factors, as well as virulence of the organisms. There are two clinical types, impetigo contagiosa and bullous impetigo.<sup>2</sup>

### Impetigo contagiosa

This is the most common form. Usually caused by *Staphylococcus aureus*, streptococci or both, depending on geographical variations, with the streptococcal form being more prevalent in warmer climates. The early lesion of impetigo contagiosa is a very thin-walled vesicle that develops on an erythematous base.

The vesicles rupture rapidly, and as a result, they are seldom seen. The exuding serum dries to form brownish crusts with a characteristic honey colour (Figure 1). In some cases, there may be a purulent discharge (Figure 2). Satellite lesions occur in the vicinity due to autoinoculation.<sup>3</sup> The crusts eventually dry, separate and disappear, leaving an area of erythema that heals without scarring.

The most common area is the face, especially around the mouth and nose. Most children are nasal carriers of the causative organisms. The trunk and limbs can also be affected. Lesions are usually localised. In severe cases, there may be regional lymphadenitis with fever and other constitutional symptoms. Widespread impetigo contagiosa is most common in the setting of secondarily infected atopic eczema.

### Bullous impetigo

Bullous impetigo is almost always caused by *Staphylococcus aureus*. The organisms can readily be cultured from blister fluid. Staphylococally produced epidermolytic toxin has been recovered from the blister fluid in some cases, and it is accepted as the basis for the bullae formation. Bullous impetigo is usually sporadic but clusters of cases may occur in families, and larger outbreaks are occasionally seen in institutions.<sup>1</sup>

In bullous impetigo, the bullae are less rapidly ruptured. They become much larger and may last for two to three



Figure 1. Impetigo contagiosa around the nose and mouth in a child. Note the crusting and honey-coloured exudates.



Figure 2. Impetigo contagiosa. Note the purulent exudation.

days. When bullae rupture, yellow crusts with oozing result. A pathognomonic finding is a “collarette” of scale surrounding the blister roof at the periphery of the ruptured lesions. Lesions may look circinate due to central healing and peripheral extension.<sup>1</sup> They may continue to enlarge, forming polycyclic patterns.<sup>3</sup> Bullae may occur anywhere and may be widely and irregularly distributed. Although impetigo is mainly a clinical diagnosis, it is worthwhile to get a pus swab from the lesions or exudates to confirm the diagnosis. Microscopy, culture and sensitivity are also necessary to guide the choice of antibiotic agents for treatment.<sup>4</sup> This will also help to identify cases of community-associated methicillin-resistant *Staphylococcus aureus* (MRSA).

## Treatment

For mild, localised infections a topical antibiotic alone may be enough. In both staphylococcal and streptococcal impetigo, mupirocin ointment has shown good results.<sup>5</sup> Fucidic acid is also effective against both organisms.<sup>5</sup> Other topical antibiotics such as bacitracin and neomycin are less effective.<sup>5</sup>

Disadvantages of topical therapy relate to compliance problems for extensive disease, and failure to eradicate organisms on uninvolved skin or in respiratory tract reservoirs, which is particularly important in clinical settings of epidemic impetigo.<sup>3</sup>

In widespread or severe cases where systemic therapy is required, cloxacillin, amoxicillin/clavulanate and cephalexin are drugs of choice, but patterns of resistance must be considered. Erythromycin is less effective as resistance against it is rising.<sup>5</sup> Penicillin VK is not appropriate oral therapy for impetigo.<sup>3,5</sup> For community-associated MRSA, drugs like vancomycin, clindamycin or linezolid should be prescribed. However, resistance of community-associated MRSA to some of these agents does occur.<sup>6,7</sup>

## Complications

In untreated cases, the disease may spread to other areas of the body.

- Staphylococcal impetigo with strains producing toxic shock syndrome toxin-1 (TSST-1) may lead to toxic shock syndrome in children.
- Staphylococcal osteomyelitis, septic arthritis and pneumonia can occur, but such a severe disease progression is usually limited to children with acquired or inherited immune deficiencies.
- Certain nephritogenic strains of group A  $\beta$ -haemolytic streptococci may cause poststreptococcal glomerulonephritis, possibly by way of antigenic mimicry.

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