

# Superficial skin infections and the use of topical and systemic antibiotics in general practice

M H Motswaledi, MBChB, MMed(Derm), FCDerm(SA)

Department of Dermatology, Sefako Makgatho University

Correspondence to: Dr M.H Motswaledi, e-mail: motswaledi1@webmail.co.za

Keywords: superficial skin infections, topical and systemic antibiotics, impetigo, erysipelas, cellulitis, ecthyma, furuncles, carbuncles, subcutaneous abscesses

## Abstract

Superficial bacterial infections of the skin are very common. With the increasing burden of human immunodeficiency virus (HIV), this is likely to worsen. Examples of such infections include impetigo, erysipelas, cellulitis, ecthyma, furuncles, carbuncles and subcutaneous abscesses. Common causative organisms are staphylococci and streptococci. Generally, *Staphylococcus aureus* infections tend to spread locally, causing abscesses and carbuncles, while streptococci are apt to spread along tissue planes, and give rise to either cellulitis or erysipelas. However, this is not always the case. These infections cause a significant morbidity, and have to be diagnosed and treated promptly. Some result in serious complications.

Republished with permission: *S Afr Pharm J* 2017;84(1):37-41

Prof Nurs Today 2018;22(4):15-20

## Impetigo

This is the most common bacterial infection of the skin, and is usually seen in children. Almost always, it is caused by *Staphylococcus aureus* or streptococci, or a combination of the two.<sup>1</sup> The source of these infections is mainly nasal or perianal colonisation, and infection is acquired by skin-to-skin contact, or from contact with nasal carriers.

### There are two types of impetigo:

- Impetigo contagiosa, which presents as crusted lesions and characteristic honey-coloured exudates. The most affected areas are the face, especially the areas around the nose and mouth (see Figure 1).



Figure 1: Impetigo contagiosa in a child

- Bullous impetigo is usually caused by *Staphylococcus aureus*. It presents with blisters that contain a turbid fluid. These blisters rupture and leave a "collarette" of scale surrounding the blister roof at the periphery of the ruptured lesions (see Figure 2). Some lesions may enlarge to form polycyclic patterns<sup>2</sup> (see Figure 3). They occur anywhere, and may be widely and irregularly distributed.



Figure 2: Bullous impetigo. Note the "collarette" scale at the periphery of the lesion



Figure 3: Bullous impetigo. Note the polycyclic pattern

Mild and localised disease can be treated with either mupirocin ointment, or fucidic acid ointment. Mupirocin is highly bactericidal to all common, primary skin pathogens. It induces a rapid bacteriological and clinical response that is equal to, or surpasses that of other topical and oral agents. The clinical efficacy of mupirocin has been proven in a large number of studies,<sup>3</sup> and in particular, in the management of impetigo and furunculosis.

Systemic antibiotics such as cloxacillin are reserved to treat severe disease. Mupirocin nasal ointment can be used to eradicate persistent nasal carriage. Vancomycin, clindamycin and linezolid are used for cases of methicillin-resistant *Staphylococcus aureus*.

## Ecthyma

Ecthyma is a pyogenic infection of the skin. It is characterised by the formation of adherent crusts, beneath which ulceration occurs. The aetiology is similar to that of impetigo. Some cases are caused by *Streptococcus pyogenes*, some by *Staphylococcus aureus*, and others by both. This means that the infection may be synergistic.<sup>4</sup> Ecthyma occurs in both children and adults. Predisposing factors are poor hygiene, malnutrition, immunosuppression and skin trauma.

Lesions start as pustules, or small blisters, on an erythematous base. These rupture to form hard crusts of dried exudates. The crusts fall off, leaving purulent irregular ulcers (see Figure 4). Commonly affected areas are the buttocks, thighs and legs, but the ulcers may appear anywhere on the body.



**Figure 4:** Ecthyma in a child. Note the purulent irregular ulcers

To treat ecthyma, the underlying predisposing factor needs to be addressed, and antibiotics such as cloxacillin prescribed against both *Staphylococcus aureus* and *Streptococcus pyogenes*. Topical therapy with mupirocin ointment should also be given.

Ecthyma gangrenosum is a rare variant, usually associated with pseudomonas infection.<sup>5</sup> It usually occurs in neutropenic, or immunocompromised patients with pseudomonas septicaemia.<sup>5,6</sup> There are reports of organisms

other than pseudomonas causing ecthyma gangrenosum.<sup>5,6</sup> A typical (ecthyma gangrenosum) lesion is a bulla which ruptures, leaving a deep ulcer with a necrotic base and well demarcated edges. It typically heals with scarring.

Management of ecthyma gangrenosum involves identification of any underlying immune deficiency, debridement, and the giving of intravenous antibiotics. Current recommendations advocate the use of aminoglycosides like amikacin or gentamycin, and anti-pseudomonal  $\beta$ -lactam agents such as piperacillin (or tazocin) and ceftazidime.<sup>6</sup> Quinolones (i.e. ciprofloxacin) also have anti-pseudomonal properties.

## Erysipelas

This is a bacterial infection of the dermis and upper subcutaneous tissue. It presents with a well-defined, raised edge, an indication of more superficial dermal involvement. Erysipelas is usually a streptococcal disease. *Staphylococcus aureus* is occasionally isolated from lesions of erysipelas.<sup>4</sup>

Clinically, erysipelas present as an erythematous swelling with a raised, well defined edge. In severe cases, blistering can occur. Erysipelas start abruptly with acute systemic symptoms that may become severe with fever. Legs are the most commonly affected site, and an obvious wound can usually be identified as a possible site of inoculation. Erysipelas affects the face too, but here, the traumatic site of entry is generally not as well detected. Specimens for bacteriological examination should be collected from blister fluid or ulcerated surfaces. Blood cultures should also be taken.

In mild cases, treatment of oral monotherapy with cloxacillin is usually adequate, as it is bactericidal to both streptococci and staphylococci.<sup>4</sup> Clarythromycin may be given in penicillin-allergic patients. For severe cases with possible complications, intravenous treatment with benzylpenicillin is indicated.

## Cellulitis

Cellulitis is an acute, subacute or chronic inflammation of loose connective tissue, but generally the term applies to the inflammation of subcutaneous tissue in which an infective, generally bacterial cause is proven or assumed.<sup>4</sup> The causative organism is *Staphylococcus aureus* either alone, or in combination with streptococci. *Haemophilus influenzae* type b is an important cause of facial cellulitis in young children up to the age of two years, but rarely causes cellulitis in adults.<sup>7</sup>

Other bacteria are occasionally implicated in cellulitis in specific immunosuppression or exposure settings. These include *Streptococcus pneumoniae* (**present in** the sinuses), *Aeromonas hydrophila* (complicates injuries that have been contaminated by soil), *Pasturella multocida* (inoculated by animal bites) and *Pseudomonas aeruginosa* (causing gangrenous cellulitis and ecthyma gangrenosum).<sup>8</sup>



**Figure 5:** Cellulitis on the cheek. The site of inoculation is a small traumatic injury, indicated by the tip of the arrow

Cellulitis may be caused by *Acinetobacter calcoaceticus* and *Staphylococcus epidermis*, as well as *Bacteroides fragilis* and *Yersinia enterocolitica*.<sup>9</sup> A case of anaerobic cellulitis produced by a clindamycin-resistant *Clostridium perfringes* has also been reported.<sup>10</sup> Cellulitis can occur following infection of a surgical wound, ulceration or traumatic skin injury. Other predisposing factors are diabetes mellitus, immunosuppression, and venous or lymphatic insufficiency.

Clinically, cellulitis presents as an erythematous swelling with diffuse poorly demarcated edges (see Figure 5), as opposed to erysipelas which has well-defined edges. Severe cellulitis may blister and progress to dermal necrosis. Lymphangitis and lymphadenopathy can result, while fever and malaise occur frequently. Periorbital cellulitis is particularly common in children. It usually follows trauma to the eyelids, which are then infected by streptococci or staphylococci.

A clinical assessment should guide empiric treatment as to the likely pathogenic organism. If monotherapy is required, oral cloxacillin is sufficient. Clarythromycin can be given to penicillin-sensitive patients. Initial treatment must cover both streptococci and staphylococci. *Haemophilis influenzae* should be borne in mind when treating facial infections in young children. Severe cases will require intravenous treatment with cloxacillin or benzylpenicillin.

Since the aetiology of cellulitis is broad, and in case an unusual organism is implicated, active attempts to identify the causative organism are necessary. Treatment should be given for five to ten days, but for longer periods if there are complications such as fasciitis, myositis, subcutaneous abscesses and septicaemia. In recurrent cases, long-term penicillin can be given orally to prevent attacks.

Recurrent episodes of cellulitis have been associated with post-cellulitic oedema. Oedema is a risk factor for recurrent disease.

### Furuncles and carbuncles

A furuncle is a hair follicle abscess, resulting from infection with *Staphylococcus aureus*. In contrast to superficial

*Staphylococcal folliculitis*, furuncles are deep-seated. They are rare in childhood, but common in adults. The infecting strain of *Staphylococcus aureus* is usually present in the nostrils or the perineum. This implies that repeated and heavy inoculation of chronic carriers may result in the development of furunculosis.<sup>4</sup> Furunculosis is common in the HIV setting.

A furuncle starts as a small follicular inflammation, later becoming an abscess, which then ruptures and heals, leaving permanent scars. Lesions can be single or multiple, and tend to appear in crops (see Figure 6). Fever and constitutional symptoms may be present. Cheek lesions can develop into cavernous sinus thrombosis, although this is rare.<sup>4</sup>

Treatment of mild localised lesions with mupirocin ointment is usually adequate. When severe disease with multiple lesions is a factor, for example in immunocompromised patients, systemic treatment with cloxacillin may be necessary.

Carbuncles result when a group of contiguous hair follicles become deeply infected with *Staphylococcus aureus*. The condition is characterised by intense inflammatory changes in the surrounding connective tissues, as well as the subcutaneous fat. A carbuncle starts as a small painful erythematous nodule that is dome-shaped and tender to the touch. Inflammatory changes and necrosis of the intervening skin may occur. There may be high fever and malaise. Carbuncles are treated with cloxacillin or other penicillinase-resistant antibiotics. In some cases, incision and drainage may be necessary.



**Figure 6:** Furunculosis in an elderly diabetic patient

### Staphylococcal scalded skin syndrome

Staphylococcal scalded skin syndrome is a generalised, confluent, erythematous superficial exfoliation that commonly occurs in neonates and young children, following infection by certain strains of toxin-producing *Staphylococcus aureus*. It is rare in adults. Predisposing factors include immunosuppression, renal failure and malignancy.

Outbreaks in nurseries have been reported.<sup>11</sup> The staphylococcal infection usually originates at a distant focus, like the pharynx or nose. Septicaemia or cutaneous infection may also be the cause. Other primary infection foci from the urinary tract, umbilicus and conjunctivae can also lead to Staphylococcal scalded skin syndrome. The associated strain of *Staphylococcus* releases exfoliatin, an epidermolytic toxin that causes skin desquamation. The disease starts abruptly with fever, skin tenderness and erythema, sparing palms, soles and mucous membranes. This differentiates it from toxic epidermal necrolysis, which is drug-induced. The erythema in Staphylococcal scalded skin syndrome is often accentuated in flexural and periorificial areas.

Generalised exfoliation follows within a few hours or days, leaving raw, painful areas. Pus swabs from the skin lesions usually yield staphylococci. This is because the desquamation is mediated by toxins which are disseminated haematogenously. The same toxins are present in bullous impetigo which may be regarded as a localised form of Staphylococcal scalded skin syndrome. If it can be identified, the Staphylococci can be isolated from the original infection site.

Early administration of intravenous antibiotics, such as cloxacillin or a cephalosporin, results in a good recovery within seven to ten days. Intravenous penicillinase-resistant, anti-staphylococcal antibiotics, supportive skin care, as well as fluid and electrolyte management in the setting of disrupted skin barrier function, will ensure rapid recovery.<sup>2</sup> In children, where there is usually no underlying predisposing factor, the prognosis is good.<sup>12,13</sup>

Staphylococcal scalded skin syndrome epidemics in neonatal nurseries can occur. Typically, the source of such outbreaks are nursery or maternity staff infected with, or colonised by epidermolytic toxin-producing *Staphylococcus aureus*. Identification of health care workers who are colonised by, or infected with toxigenic strains of *Staphylococcus aureus*, is an integral part of managing these outbreaks. Infection control measures, including strict enforcement of chlorhexidine hand washing, oral antibiotic therapy for infected health care workers, and the application of mupirocin nasal ointment for the eradication of persistent nasal carriage, are usually adequate.<sup>12,14</sup>

## Sponsorship and conflict of interest

The author received no sponsorship from any source, nor was there any reported conflict of interest.

## References

1. Hay RJ, Adriaans BM. Bacterial infections. In: Champion RH, Burton JL, Burns DA and Breathnach SM, eds. *Rook/Wilkinson/Ebling Textbook of Dermatology*. 6<sup>th</sup> ed. Blackwell Science; 1998; p1097-1197.
2. Resnick SD. Staphylococcal and streptococcal skin infections: pyodermas and toxin-mediated syndromes. In: Harper J, Oranje A and Prose N, eds. *Textbook of Pediatric Dermatology*, Vol 1. Blackwell Science; 1998; p. 369-383.
3. Atherton DJ. The clinical efficacy of mupirocin in the treatment of primary skin infections. In: Bork K, editor. *The clinical efficacy of mupirocin in the treatment of primary and secondary skin infections*. International Clinical Practice Series. Royal Tunbridge Wells Medical, 1996; p. 25-26.
4. Hay RJ, Adriaans BM. Bacterial infections. In: Burns T, Breathnach S, Cox N and Griffiths C, eds. *Rook's textbook of Dermatology*, 8<sup>th</sup> ed. Wiley-Blackwell; 2010;30.1-30.82.
5. Tsuchiyama K, Okuyama R, Ogawa E, et al. Ecthyma gangrenosum with citrobacter freundii infection. *J Eur Acad Dermatol Venereol*. 2009;23(6):709-710. Epub 2008.
6. Goolamali SI, Fogo A, Killian L, et al. Ecthyma gangrenosum: an important feature of pseudomonal sepsis in a previously well child. *Clin Exp Dermatol*. 2009;34(5):e180-e182. Epub 2009.
7. Pedler SJ, Hawkey PM. Cellulitis in children caused by *Haemophilus influenzae* type b. *J Infect*. 1983;6:269-272.
8. Sevinsky LD, Vieceus C, Ballesteros DO, et al. Ecthyma gangrenosum: a cutaneous manifestation of *Pseudomonas aeruginosa* sepsis. *J Am Acad Dermatol*. 1993;29(1):104-106.
9. Krogstad P, Mendelman PM, Millar VL, et al. Clinical and microbiologic characteristics of cutaneous infection with *Yersinia enterocolitica*. *J Infect Dis*. 1992;165:740-743.
10. Khanna N. Clindamycin-resistant *clostridium perfringens* cellulitis. *J Tissue Viability*. 2008;17:95-97.
11. Curran JP, Al-Sahili FL. Neonatal staphylococcal scalded skin syndrome: massive outbreak due to an unusual phage type. *Paediatrics*. 1980;66(2):285-290.
12. Martinez A, Harper J. Staphylococcal scalded skin syndrome. In: Lebowitz MG, Haymann WR, Berth-Jones J and Coulson E, editors. *Treatment of skin disease, comprehensive therapeutic strategies*. 2<sup>nd</sup> ed. New York: Mosby-Elsevier, 2007; p. 620-621.
13. Berk DR, Bayliss SJ. MRSA, Staphylococcal scalded skin syndrome and other cutaneous bacterial emergencies. *Paediatric Annals*. 2010;39(10):627-633.
14. Williford PM. Antimicrobial wound management in the emergency department. An educational supplement. Opportunities for mupirocin calcium cream in the emergency department. *J Emerg Med*. 1999;17(1):213-220. References