Management of common bacterial infections of the skin

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Purpose of review

Bacterial skin infections commonly encountered in the community include impetigo, folliculitis/furunculosis, simple abscesses, erysipelas and other nonnecrotizing cellulitis. The review focuses on recent epidemiological, bacteriological and therapeutic advances.

Recent findings

Impetigo and erysipelas occur in about 20 and 1 person/1000/year, respectively. Main risk factors for erysipelas are toe-web intertrigo and lymphedema. The true incidence of furunculosis is unknown, whereas outbreaks in small communities are reported worldwide. *Staphylococcus aureus* is the predominant pathogen for impetigo and furunculosis, and methicillin-resistant strains play a growing role in both diseases. Erysipelas are mainly caused by streptococci, whereas local complications (i.e. abscesses or blisters) may be due to staphylococci, including methicillin-resistant strains in involved geographic areas. Recent trends for treating impetigo and furunculosis predate community-acquired methicillin-resistant *S. aureus*. For outbreaks of furunculosis, stringent decolonization measures are showing promise, whereas there is no validated therapeutic regimen for chronic furunculosis. Current trends for erysipelas involve ambulatory treatments and reduced duration of antibiotics.

Summary

Despite better epidemiological or bacteriological knowledge of common bacterial skin infections, the exact role of methicillin-resistant staphylococci needs regular surveys in involved geographic areas. Antibiotic treatment must be active on staphylococci and, to a lesser degree, on streptococci.

Keywords

cellulitis, erysipelas, folliculitis, furunculosis, impetigo, skin abscess

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Introduction

The objective of this paper is to provide a critical review of recent literature on 'common bacterial skin infections', which include impetigo, folliculitis (including furuncles and furunculosis), simple abscesses, erysipelas and other nonnecrotizing dermal hypodermal infections (cellulitis). These are generally of mild to modest severity and can be easily treated. Rather than a systematic review, it is a personal and biased choice on those particular skin disorders. The review will include neither the field of necrotizing soft tissue infections, particularly necrotizing fasciitis, which are actually rare disorders, nor 'diabetic foot' infections, as both raise specific questions in terms of clinical presentation, diagnosis and management.

Impetigo, the most common skin infection in children throughout the world, consists of superficial, nonfollicular pustules that are mostly caused by *Staphylococcus aureus* or β-hemolytic streptococci. Furuncles ('boils') are infections of the hair follicle, frequently caused by *S. aureus*, in which suppuration extends to the deep dermis, where a small abscess develops. Furuncles differ from folliculitis, in which inflammation is more superficial and pus is present within the epidermis [1].

Erysipelas is an acute, superficial, nonnecrotizing dermal/hypodermal infection that is mainly caused by streptococci [2]. The definitive diagnosis is based on clinical findings that usually include a sharply demarcated shiny erythematosus plaque of sudden onset associated with pain, swelling and fever. Other nonnecrotizing bacterial dermal/hypodermal infections, often named 'cellulitis' in the literature, are acute spreading infections of the skin, extending more deeply than erysipelas to involve the subcutaneous tissues. Associated regional lymphadenopathy and lymphatic streaking are inconstant, and local complications (abscesses, necrosis) more frequent than in

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erysipelas. Petechiae and ecchymoses with frequent bullae may develop in inflamed skin resulting in hemorrhagic cellulitis [3].

Epidemiology and risk factors

Despite their high frequency, there are limited data on the incidence and fluctuations of common bacterial infections of the skin such as impetigo, erysipelas or nonnecrotizing cellulitis. Impetigo is a skin infection that is common throughout the world and occurs most frequently among economically disadvantaged children in tropical or subtropical regions, but it is also quite frequent in northern climates during the summer months. In a study in The Netherlands [4], the annual incidence of impetigo varied from 0.017 events per person-year in 1987 to 0.021 events per person-year in 2001. Another study from Britain [5], providing data collected from a sentinel general practice network over the years 1999–2003, gave a mean total incidence of 18.7 events per week per 100 000 inhabitants, corresponding to 0.01 events per person-year. In this study, incidence was highest in children under 5 years followed by that for children 5–14 years; the incidence then decreased rapidly over the age range 15–44 years and was a minimum for the elderly (65 years and older). Male and female incidence rates reported in children were virtually the same. Such figures were confirmed in Norway with a reported incidence rate of impetigo of 0.017 events per person-year in an island community in the years 2001–2005 [6]. Epidemics were identified that always started during summer with incidence rates in these epidemics between 0.045 and 0.099 events per person-year [6]. In one study, the temporal incidences of impetigo and insect bite lagged by around 5 weeks, suggesting that there is an association between the episode incidence rates of impetigo and insect bite and with air temperature, and that improved management of insect bites through the use of antiseptic treatments might contribute to reducing the impact of this condition [5].

Erysipelas affects predominantly adult patients in the sixth or seventh decade and is located on the lower limb in more than 80% of cases. A female predominance exists, except in young patients. A recent Dutch study [7] showed an incidence of about 2/1000/year when both erysipelas and cellulitis affecting the leg were considered. Very similarly, the incidence of lower-extremity cellulitis in Olmsted County (Minnesota) was estimated at 199 per 100 000 person-years [8]. These figures were partly confirmed in a community-based study conducted in Belgium that showed an age-standardized incidence of erysipelas increasing from 1.88 to 2.49/1000/year in the period from 1994 to 2004 [9]. In this report, a seasonality of the disease (i.e. erysipelas occurred more frequently in summer and less frequently in winter) was suggested, but not demonstrated [9]. Predisposing factors are now well identified for erysipelas or cellulitis of the leg. Since the first case–control study by Dupuy *et al.* in 1999 [10], several reports using a similar approach have confirmed that they mostly include loco-regional factors, i.e. disruption of the cutaneous barrier (leg ulcer, wound, fissured toe-web intertrigo, pressure ulcer), lymphedema, chronic edema or local surgical operations (lymph node dissection, saphenectomy). Toe-web intertrigo appears to be a major portal of entry whether due or not to dermatophytes; its self-reporting, however, is low in a recent report, suggesting that attempts to reduce the risk of recurrence by treatment of toe-web intertrigo may actually fail [11]. Although clinically recognizable lymphedema is a particularly strong risk factor, any edema is both a risk factor for cellulitis/erysipelas [10] and a consequence [11] of the disease. General factors such as obesity or a history of prior cellulitis are less important and diabetes does not appear to be a risk factor for erysipelas. Risk factors for bacteremia in patients with limb cellulitis were searched for in a retrospective Spanish study [12]. Microorganisms were isolated in blood cultures in 57 of 308 (18.5%) cases; surprisingly, they were mostly nongroup A β-hemolytic streptococci and Gram-negative bacteria, and factors associated with bacteremia were absence of previous antibiotic treatment, presence of at least two comorbid factors, length of illness less than 2 days and proximal limb involvement [12].

Although it is likely that folliculitis is more common in individuals with particular diseases (organ transplant recipients, diabetic patients, genetic diseases for example trisomy, etc.), there are to date no consistent data on the prevalence or incidence of either folliculitis or furunculosis in the community. On the other hand, some individuals have repeated attacks of furunculosis. Apart from children with abnormal systemic host responses, for most of these patients the only identifiable predisposing factor is the carriage of *S. aureus*, especially in the anterior nares. Knowing that the prevalence of nasal staphylococcal colonization in the general population is approximately 20–40%, it is usually unclear why some carriers develop recurrent skin infections and others not. For years, outbreaks of furunculosis caused by methicillin (oxacillin)-susceptible *S. aureus* (MSSA) [13••], as well as by methicillin (oxacillin)-resistant *S. aureus* (MRSA) [14], have been regularly reported in the literature. These outbreaks may occur in small communities including families and other settings involving close personal contact (e.g. prisons), especially when skin injury is common (e.g. sports teams). Inadequate personal hygiene and exposure to other individuals with furuncles are important predisposing factors in these settings. In a recent study in Chicago, risk factors for skin and soft tissue community-acquired (CA)-MRSA infections (mainly

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abscesses) were incarceration, African–American race/ethnicity and residence at a group of geographically proximate public housing complexes, whereas older age was inversely related and, interestingly, clonal CA-MRSA infections seem to occur in addition to, not in place of, MSSA infections [15].

Which organisms are responsible for common bacterial skin infections?

Impetigo consists of discrete purulent lesions that are nearly always caused by β-hemolytic streptococci and/or *S. aureus*. Whereas, in the past, nonbullous lesions were usually caused by streptococci, most cases are now caused by staphylococci, either alone or in combination with streptococci. Streptococci isolated from lesions are primarily group A organisms, but occasionally other serogroups (such as C and G) are responsible. In a recent study in Norway where swabs were taken from 255 of 334 patients with impetigo, *S. aureus* was isolated from 79% (201/255) of these cases; in this report, *S. aureus* was the causal agent more frequently in epidemic as compared with nonepidemic periods, whereas resistance to fusidic acid was significantly higher (up to 84%) in epidemic periods [6]. Resistance to the antibiotic fusidic acid in the European strains of *S. aureus* causing impetigo has increased in recent years. This results from clonal expansion of a strain which carries the fusidic acid resistance determinant *fusB* on its chromosome [16]. Molecular typing recently revealed that this European clone was ST123, *spa* type t171 and *agr* type IV, and therefore unrelated to earlier *fusB*+ strains that were prevalent in the UK during the 1970s [16]. Bullous impetigo is caused by strains of *S. aureus* that produce exfoliative toxins that cause the loss of keratinocyte cell-to-cell adhesion resulting in cleavage in the superficial epidermis. Those staphylococcal exfoliative toxins (three isoforms, i.e. ETA, ETB and ETD) blister the superficial epidermis by hydrolyzing a single peptide bond, Glu381–Gly382, located between extracellular domains 3 and 4 of human desmoglein 1, a desmosomal intercellular adhesion molecule that is also the target antigen of autoantibodies in pemphigus foliaceus, an autoimmune blistering disease. Although, however, bullous impetigo is invariably considered to be a staphylococcal disease, there are also very rare cases of streptococcal bullous impetigo [17]. MRSA is a major nosocomial pathogen that may also cause impetigo [14,18]. In a recent French study [19•], bacteriological samples prospectively collected from 121 patients presenting with furuncles or impetigo showed that MRSA accounted for four of 64 (6%) positive skin cultures, confirming another recent bacteriological survey [20]. Results also showed that exfoliative toxin genes were present in 10 of 10 (100%) and 12 of 21 (57%) bullous and nonbullous impetigo isolates, respectively, suggesting that both forms of impetigo may be actually associated with exfoliative toxins [19•].

Erysipelas is most commonly caused by β-hemolytic streptococci of group A, less so by group B, C or G streptococci and rarely by staphylococci [1,2,21]. Bulla formation is considered as a relatively severe but frequent local complication of the disease. In a small retrospective series of patients with bullous erysipelas, *S. aureus* was frequently identified in bullous lesions; however, it was impossible to assess whether *S. aureus* is the true pathogen of bullous erysipelas or merely a contaminant [22]. Although most cases of erysipelas are caused by β-hemolytic streptococci, many other bacteria can produce nonnecrotizing cellulitis, which can often occur in particular circumstances, e.g. *Pasteurella multocida* following cat or dog bites, *Aeromonas hydrophila* following immersion in fresh water, *Vibrio* species after saltwater exposure or *Haemophilus influenzae* in periorbital cellulitis in children. An emerging problem is the increasing prevalence of skin infections caused by CA-MRSA, which is responsible for at least half of the cases of cellulitis with purulent exudates in involved geographic areas [18], especially in children [23,24]. Those community strains cause infections in patients lacking typical risk factors (hospital admission, long-term care facility residence); they are often susceptible to non-β-lactam antibiotics, including vancomycin, trimethoprim–sulfamethoxazole (TMP-SMX), rifampicin, clindamycin and gentamicin [14,23,24].

From the SENTRY program monitoring skin and subcutaneous tissue infections over a 7-year period (1998–2004), *S. aureus* was the most predominant pathogen, ranked first in all geographic regions [25•]. *S. aureus* is also the major pathogen isolated from furuncles, furunculosis and superficial skin abscesses [1,13••]. The severity of those *S. aureus*-induced infections is determined by the presence of virulence factors, including Panton–Valentine leukocidin, a leukocytolytic toxin associated with severe cutaneous infections and highly lethal necrotizing pneumonia, which is encoded for by the gene *lukS–lukF* [13••]. In a recent study, Panton–Valentine leukocidin genes were present in 13 of 31 (42%) isolates from furuncles and were associated with epidemic furunculosis [19•]. Nasal carriage of *S. aureus* was found in 58% of patients overall and was associated with chronic furunculosis, but not with simple furuncles (88 vs. 29%) [19•]. These data strongly suggested that Panton–Valentine leukocidin is mostly associated with epidemic furunculosis and *S. aureus* nasal carriage associated with the chronicity of furuncles. As previously noticed, outbreaks of epidemic furunculosis due to CA-MRSA in young, otherwise healthy people have been particularly noteworthy, and in many US cities MRSA now represents the most common pathogen isolated in the emergency

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department from patients with skin and soft tissue infections [14,18,26]. Bacterial endocarditis in patients with CA-MRSA furunculosis is an emerging threat. In five previously healthy patients who presented with endocarditis after developing furunculosis due to CA-MRSA, blood culture isolates were found to be *PVL* gene positive and carried the type IV SCCmec element, and pulse field gel electrophoresis confirmed that the skin isolate was identical to the isolate cultured from the patient's blood [27]. On the other hand, a considerable variation in the MRSA rate in skin and subcutaneous tissue infections was noted between countries and continents, with the overall rate highest in North America (36%) compared with Latin America (29%) and Europe (23%) in the SENTRY study [25•]. Unfortunately, however, the true prevalence of MRSA causing furuncles or furunculosis is not known due to the lack of adequate, community-based, large, prospective epidemiological studies.

Which treatment for impetigo?

As *S. aureus* currently accounts for almost all cases of bullous impetigo, as well as for a majority of nonbullous infections, penicillinase-resistant penicillins or first-generation cephalosporins are now preferred, although impetigo caused by MRSA is increasing in frequency [14]. A Cochrane review of interventions for impetigo in 2004 identified only 12 studies of good quality from 57 trials including 3533 patients [28]. This systematic review concluded that: topical antibiotics showed better cure rates than placebo; between mupirocin and fusidic acid, neither topical antibiotic was superior; topical mupirocin was superior to oral erythromycin; topical and oral antibiotics did not show different cure rates, nor did most trials comparing oral antibiotics; and oral penicillin was not as effective as other antibiotics (cloxacillin, erythromycin). Since that time, there was no consistent data capable of modifying our strategy for treating impetigo. The decision of how to treat impetigo still depends on the number of lesions, their location and the need to limit spread of infection to other individuals [1]. The best topical antibiotics are mupirocin and fusidic acid (not available in the US), although resistance has been described [28]; other agents such as bacitracin and neomycin are considerably less effective and are not recommended. Retapamulin is the first agent of the pleuromutilin class formulated as a topical antibacterial for treating skin infections. A recent randomized, observer-blinded, noninferiority, phase III study in 519 adult and pediatric patients compared the efficacy and safety of retapamulin ointment, 1%, with sodium fusidate ointment in impetigo [29•]. Retapamulin and sodium fusidate showed comparable clinical efficacies (per-protocol population: 99.1 and 94.0%, respectively; difference: 5.1%, 95% confidence interval: 1.1–9.0%, $P = 0.003$) and bacteriological efficacies were similar. Success rates in the small numbers of sodium fusidate-, methicillin- and mupirocin-resistant *S. aureus* were satisfactory for retapamulin. Retapamulin thus appears to be an interesting new treatment option for impetigo, with efficacy against *S. aureus* resistant to existing therapies. Whereas it is demonstrated that simple local care (including cleansing with soap and water, removal of crusts and wet dressings) is useful for treating impetigo [30] and that handwashing with daily bathing also prevents impetigo in children [31], there is little evidence about the disinfecting measures [28].

Patients who have numerous lesions or who are not responding to topical agents should receive oral antimicrobials effective against both *S. aureus* and *S. pyogenes*. Current US guidelines predate the widespread occurrences of CA-MRSA [1], and usually recommend oxacillin, cephalexin, new macrolides (instead of erythromycin) and amoxicillin/clavulanic acid, all orally. The evidence used in developing current guidelines has, however, important limitations. Further studies including superiority outcome studies, placebo-controlled studies, measurement of time to resolution or other novel approaches are therefore needed to resolve these treatment dilemmas. Finally, glomerulonephritis following streptococcal infection may be a complication of impetigo caused by certain strains of *S. pyogenes*, but is quite rare in developed countries (less than one case per 1 000 000 population per year); to date, there is no data demonstrating that treatment of impetigo prevents this severe complication [1,28].

Which treatment for simple abscesses of the skin and furunculosis?

Effective treatment of simple skin abscesses includes incision, thorough evacuation of the pus, probing the cavity to break up loculations and simply covering the surgical site with a dry dressing, whereas Gram stain, culture and systemic antibiotics are usually not necessary [1,32]. Unusual exceptions include multiple lesions, cutaneous gangrene, severely impaired host defenses, extensive surrounding cellulitis or sepsis [1]. If MSSA infection is known or suspected, the oral agents recommended include clindamycin, dicloxacillin, cephalexin, doxycycline, minocycline and TMP-SMX. In geographic areas where CA-MRSA infections now predominate in patients with skin abscesses, agents recommended for MRSA should be used for this indication, including clindamycin, doxycycline, minocycline, TMP-SMX and linezolid in more severe cases [1,23,24]. Interestingly, in a recent study including 227 skin and soft tissue MRSA infections, the empirical use of TMP-SMX was associated with increased odds of clinical resolution [33].

Chronic furunculosis is difficult to treat and to date there is no convincing data to recommend a validated

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therapeutic strategy. Apart from ensuring personal hygiene, the management consists of long-term treatment, sometimes sequential, with topical and systemic antibiotics. A common method of controlling recurrent furunculosis is eradication of staphylococcal carriage. In patients with documented nasal colonization, the application of mupirocin or fusidic acid ointment twice daily in the anterior nares for the first 5 days each month reduces recurrences by around 50% [1]. Recently, a course of rifampicin (450–600 mg daily for 10 days) was also shown to eradicate the carrier state in the majority of cases and to prevent recurrences [34]. Low-dose azithromycin (500 mg weekly for 3 months) or clindamycin (150 mg daily for 3 months) may decrease subsequent furunculosis episodes by approximately 80% [1,35]. For outbreaks of furunculosis or small cutaneous abscesses, stringent decolonization measures using mupirocin nasal ointment and disinfecting wash solution in affected patients and their families were recently shown to be very effective. This epidemiological intervention led to cessation of the outbreak of furunculosis due to MSSA strains positive for the *lukS–lukF* gene in a German village with a long-term success (20 weeks) in this community setting [13••].

Treatment of erysipelas and nonnecrotizing cellulitis

Recent trends in the management of erysipelas and nonnecrotizing cellulitis concerning ambulatory treatments, reduced duration of antibiotics and the role of new antistaphylococcal treatments have been very recently reviewed in *Current Opinion in Infectious Diseases* [36]. Since then, only a few papers have been published that may be likely to modify our current management of these common infections [37,38•,39•,40]. Indeed, most patients with erysipelas and nonnecrotizing cellulitis do not require hospitalization [1,41], as illustrated by a recent Dutch study [7] that showed that less than 10% of patients were actually hospitalized. Contrasting with these figures, in the study by Corwin *et al.* [42], only about one-third of patients from a total of 558 eligible patients presenting at hospital for intravenous treatment of cellulitis were considered suitable for home treatment whereas 12% of patients randomized to intravenous home treatment required secondary transfer to hospital. In the literature, the main reasons for primary hospitalization are the severity of general or local signs and symptoms, suspicion of sepsis, old age and comorbidities [1,2,43]; however, true criteria of primary or secondary hospitalization still remain to be defined by adequate prospective studies performed in both in and outpatients. There is a recent trend to avoid hospitalization by promoting intravenous treatments at home for economic reasons [42,43]. In a prospective randomized controlled trial enrolling 200 patients with cellulitis randomized in the emergency department to receive intravenous antibiotics (2 g of cephazolin twice daily) either in hospital or at home, the two treatment groups did not differ significantly for the primary outcome of days to no advancement of cellulitis (mean 1.5 days) [42].

Therapy for erysipelas should include an antibiotic active against streptococci. Penicillin, given either parenterally or orally depending on clinical severity, is the treatment of choice for classical erysipelas [1,2]. Patients with erysipelas without local or general severity signs can be treated orally by amoxicillin (3–4.5 g daily) for 10–14 days, usually as outpatients [2]. A randomized study comparing treatment for 5 or 10 days with oral levofloxacin suggested that the duration of treatment can be shortened [44]. The latter result should, however, be considered with caution since at day 5, 34 of 121 patients were not randomized for reasons including insufficient improvement, and levofloxacin is not a 'gold standard' for treatment of erysipelas and nonnecrotizing cellulitis, especially in geographic areas where MRSA is not predominant. Pristinamycin (3 g daily; only available in France and Belgium) [45] or clindamycin (300 mg 3 times daily) [1] may also be used orally in penicillin-allergic patients. Severe forms usually require intravenous antibiotics, traditionally delivered in hospitals in most countries [36]. In hospitalized patients, parenteral penicillin G (12–20 MU daily), amoxicillin (3–6 g daily) [2] or cefazolin (4 g daily) [1] can be used initially in severe cases until apyrexia, followed by oral amoxicillin [2].

In cases of cellulitis with collection or penetrating trauma, an antibiotic agent also effective against *S. aureus* should be preferred (dicloxacillin, cephalexin, clindamycin) [1,36]. In geographic areas with high rates of CA-MRSA, the use of clindamycin alone or a combination of a β-lactam plus TMP-SMX for noncomplicated cellulitis was proposed [18]. The threshold at which drugs active against MRSA, such as clindamycin and TMP-SMX, should be incorporated into empiric therapy of uncomplicated cellulitis still remains to be determined, however. A decision analysis of the empiric treatment of cellulitis showed that cephalexin was the most cost-effective therapy at current estimated MRSA levels in the US, whereas TMP-SMX is unlikely to be cost-effective for the treatment of simple cellulitis (or erysipelas) [40].

Recurrence is the main complication of erysipelas [37,38•,39•]; it occurs in about 20% of cases. Measures to reduce recurrences of erysipelas include treatment of any predisposing factor such as toe-web intertrigo or wound, or reducing any underlying edema by compressive stockings or pneumatic pressure pumps [2]. If frequent infections occur despite such measures, prophylactic antibiotics appear reasonable. Options include

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intramuscular benzathine penicillin injections (1.2–2.4 MU every 3 weeks) or oral therapy with twice-daily doses of either 250 mg of erythromycin, 1 g of pristinamycin or 1 g of penicillin V [1,2,37,38•,46]. The estimated rate of recurrence under prophylactic penicillin therapy was estimated to be between 6 and 26% at 1 year from recent studies, the most frequent reason for failure being the lack of compliance [37,38•,46].

Conclusion

Today, there is still a need for epidemiological/bacteriological surveys in the community for 'superficial' pyodermas (i.e. impetigo and furunculosis) in order to evaluate more precisely the burden of CA-MRSA skin infections, especially in European countries, and to further target adequate preventive measures. Concerning erysipelas and other nonnecrotizing cellulitis, further studies evaluating home therapy, either intravenously or orally, and shorter regimens are still mandatory for these common, mainly streptococcal, diseases.

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Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 200–201).

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