Community-associated Methicillin-resistant *Staphylococcus aureus* Skin Infections in the Tropics

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KEYWORDS

- Community-associated
- Methicillin-resistant *Staphylococcus aureus* • MRSA
- *Staphylococcus aureus* • Skin and soft tissue infection
- Tropical

Although most of the world's population lives in tropical, developing regions, there has been comparatively little research into the epidemiology of *Staphylococcus aureus* in these areas. The high burden of disease caused by malaria, tuberculosis, and human immunodeficiency virus (HIV) infection, together with a lack of diagnostic microbiology facilities and overall limitations in resource availability, impede understanding of *S aureus* infections. Available data suggest that skin and soft tissue infections (SSTI) caused by *S aureus* are extremely common, particularly in children. In addition, there are high rates of *S aureus* infection in sterile sites (invasive infection) in these same regions, and SSTI are an important source of invasive infection.[1] The understanding that strains of community-associated methicillin-resistant *S aureus* (CA-MRSA) often emerge locally raises the possibility that MRSA is also widespread among populations in the tropics. This review focuses on skin-related manifestations of CA-MRSA in tropical regions and describes what is known about the epidemiology, effects of hygiene and living conditions, diagnosis, and treatment and prevention options at the individual and community levels.

EPIDEMIOLOGY

Most staphylococcal infections involve the skin and soft tissues, and, in tropical regions, such SSTI are abundant. The prevalence of pyoderma, scabies,

ST is supported by an Australian National Health and Medical Research Council Postdoctoral fellowship (436,033). The authors have nothing to disclose.

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Dermatol Clin 29 (2011) 21–32
doi:10.1016/j.det.2010.09.005

and dermatophyte infections is high,[2] with an estimated 111 million children in developing countries having pyoderma at any one time. Studies from the 1970s found the point prevalence of pyoderma in children to be 7% in Tanzania[3,4] and up to 25% in Panama.[5] The Pacific region has a particularly high prevalence of SSTI. Recent studies in indigenous communities in tropical northern Australia found the point prevalence of pyoderma in children to range from 11% to 20%.[6] In Fiji, the prevalence of pyoderma was 26% in primary school children and 12% in infants.[7] The rates of scabies infection are similarly high, and scabies is a common antecedent for skin infection in these regions.[3,4,6–8] The most common bacterial pathogens of SSTI are *Streptococcus pyogenes* and *S aureus*, with recent studies finding recovery rates from swabs of pyoderma lesions of 29% to 80% and 57% to 80% for *S pyogenes* (group A β-hemolytic streptococci) and *S aureus*, respectively. Frequently, individual sores are infected with both pathogens.[7,9–11]

These superficial bacterial infections are not always innocuous. Complications include bacteremia[12–14] and other invasive diseases, and, for *S pyogenes*, the nonsuppurative sequelae of poststreptococcal glomerulonephritis[15] and also possibly acute rheumatic fever.[16] *S aureus* typically ranks as the third or fourth most commonly isolated bacterial pathogen in surveys of bacteremia in the developing world,[17–21] with skin infections frequently the primary source of the bacteremia.[1,22] Mortality from *S aureus* bacteremia in low-resource regions is typically high. For example, in a hospital in northeastern Thailand, the case mortality for patients with *S aureus* sepsis was 52%, causing an estimated 1% of all hospital deaths.[23]

Although *S pyogenes* remains invariably sensitive to penicillin, antibiotic management for *S aureus* depends on local rates of antimicrobial resistance. Some experts suggest that if the prevalence of CA-MRSA is greater than 10% in clinical isolates in any given population, then these populations should be considered as high prevalence and β-lactam antibiotics should not be used as empiric therapy for staphylococcal infections.[24] Most studies in tropical regions have been conducted in inpatient hospital settings where rates of methicillin resistance among all *S aureus* infections range from less than 10% to close to 50%, with most of this attributed to health care–associated (HA) MRSA strains.[25–28] Community-based studies of nasal colonization in India and Brazil found lower rates of MRSA carriage, ranging from 0% to 4%.[29–33]

However, hospital-based studies and colonization data may not reflect the epidemiology of *S aureus* in community-based SSTI. The few recent studies in tropical and developing country settings that have concentrated on SSTI in outpatients or community-based cohorts showed that CA-MRSA constituted 23% and 11% of *S aureus* recovered from pyoderma lesions in Australian indigenous[9] and Indian settings[11] respectively, and also 15% of *S aureus* recovered from SSTI in Hong Kong.[34] Several factors may be contributing to the emergence of CA-MRSA from circulating methicillin-susceptible *S aureus* (MSSA) strains in less-developed settings; these factors include high rates of secondarily infected scabies, domestic crowding, poor skin hygiene, and the ready availability and use of β-lactam antibiotics.[12,35] In support of this, detailed molecular studies have shown that strains of MSSA acquire the resistance determinant *mecA*, carried on *SCCmec*, much more frequently than was previously appreciated.[36]

Nowhere has the problem of CA-MRSA been so striking as in the United States, where CA-MRSA infections have become more common than health care–associated MRSA (HA-MRSA) infections. For example, in San Francisco, the incidence of all types of CA-MRSA infections in 2005 was 316 per 100,000 population, compared with 31 cases of HA-MRSA infections per 100,000.[37] This increase is the direct result of a prevalent strain of CA-MRSA, the USA300 strain.[38–41]

Unlike in the United States, tropical regions have observed a diversity of circulating CA-MRSA clones (**Table 1**).[9,34,42–52] Different clones are typically distinguished by established genotyping techniques such as pulsed-field gel electrophoresis or multilocus sequence typing (MLST). Using MLST, which involves sequencing the internal

Table 1
Dominant clones of CA-MRSA in tropical regions as determined by multilocus sequence typing

Region	Clone	References
East and southeast Asia	ST30, ST59, ST834	34,42–44
Australia (tropical) and Pacific islands	ST93, ST30, CC75, ST8 (USA300) in Hawaii	9,45–47
Subcontinent (India and Pakistan)	ST772	48
Africa	ST88, ST5, ST30, ST80	49–51
Latin America	ST8 (USA300)	52

Abbreviation: ST, sequence type.

fragments of 7 housekeeping genes, *S aureus* has been shown to be a species with distinct clonal lineages or clonal complexes. Many clones can be found across different continents and include clonal complexes (CC) 30, 8, 45, 15, 5, and 1. However, the proportional representation of these clones varies according to geography. For example, CC30 is common in Singapore and Hong Kong,[44,53] and CC93 is the most prevalent clone of hospital-based isolates in northern Australia.[45]

Perhaps most intriguing is a phylogenetically divergent clone called CC75, which occurs mainly in the tropics.[46] Despite the extensive characterization of *S aureus* in developed countries, CC75 has to date only been reported in the literature in northern Australia and Cambodia.[9,45,46,54] In addition, analysis of the *S aureus* multilocus sequence type (MLST) database (http://saureus.mlst.net/) reveals isolates with CC75 alleles from Malaysia, Indonesia, and, somewhat incongruously, Ireland and the Czech Republic. We have also found CC75 in Fiji.[55] CC75 is a phylogenetic outlier whose presence may be related to crowded living conditions, poor skin hygiene, and frequent skin infections.

Several virulence factors have been identified in CA-MRSA isolates,[56] most notably Panton-Valentine leukocidin (PVL). This bicomponent toxin can form pores in the cell membrane of host leukocytes. Early epidemiologic studies linked PVL with cutaneous abscesses, severe SSTI, and necrotizing pneumonia.[57,58] However, controversy exists as to the exact role of PVL. It was not associated with worse outcomes in a multicountry study of complicated SSTI,[59] nor in a Thai study of bacteremic patients.[60] There are also conflicting results regarding the role of PVL from studies using mouse models of SSTI.[61–63] Several recent independent clinical studies in Australia and New Zealand have shown a significant association between PVL and cutaneous furunculosis or skin and soft tissue abscesses.[45,64,65] PVL is also expressed frequently in MSSA isolates; approximately 50% of MSSA isolates from Africa and northern Australia harbor the *pvl* genes.[45,66] Thus, MSSA is likely to be a significant, but under-recognized, contributor to the burden of PVL disease in these settings.

In summary, evidence suggests that *S aureus*–related SSTI produces a significant burden of disease in tropical communities. Up to 25% of staphylococcal isolates are CA-MRSA, and ecological conditions favor the emergence and spread of resistance. Of concern, a considerable proportion of both CA-MRSA and MSSA strains causing SSTI harbor the PVL gene, which may cause more severe clinical manifestations.

PREDISPOSING FACTORS

Perhaps the most important predisposing factor for SSTI in tropical zones is the presence of scabies infestation (ie, epidermal infestation by the mite, *Sarcoptes scabiei*). Scabies lesions that are secondarily infected by *S pyogenes* or *S aureus* are very common in many tropical settings. For example, in Fiji, 57% of infants and 30% of children with scabies had evidence of secondary bacterial infection.[7] A reduction in the prevalence of scabies can lead to a reduction in the prevalence of impetigo. This reduction was clearly shown in Panama after mass drug administration program for scabies with permethrin cream, where the prevalence of impetigo was reduced from 32% to 2%. Similarly, in the Solomon Islands after mass drug administration with oral ivermectin, the prevalence of impetigo was reduced from 40% to 21%.[67,68] In addition, community-based treatment of scabies with permethrin in an indigenous Australian community led to a reduction in scabies prevalence from 28.8% to less than 10%. This treatment resulted in a sustained 2-year reduction in impetigo prevalence in children from 69% to less than 35%, with residual impetigo in children being less severe (fewer purulent and crusted lesions).[69]

Overcrowding, poor hygiene, limited water supply, and hot, humid weather have all been linked to SSTI in tropical zones.[4,70] These factors reflect the important role that low socioeconomic status plays; in the main, SSTI in tropical zones is a disease of poverty. Overcrowding is important because of the close personal contact required for transmission of *S pyogenes*, *S aureus*, and scabies.[71,72] Washing with water seems to play an important role in the prevention of impetigo. A randomized controlled trial of handwashing promotion and plain soap in Pakistan found a reduction in the incidence of impetigo amongst children of 34% in the intervention group.[73] Antibacterial soap did not provide an advantage compared with plain soap, suggesting that the cleansing process with water is the key factor. This conclusion was supported by a further study in an Australian aboriginal community in which access to a swimming pool was associated with a reduction in the prevalence of impetigo in children, from 62% to 18%.[74]

DIAGNOSIS

Unless there are clinical features that suggest unusual pathogens, most cases of impetigo and furunculosis are caused by *S pyogenes* or *S aureus*, or sometimes both. Knowledge of the local

antimicrobial resistance pattern of *S aureus* helps to determine whether culturing such lesions will be clinically useful. In resource-poor regions, there is likely to be limited benefit to routine culture of such lesions if rates of MRSA are low. In regions with higher rates of MRSA, clinicians must consider whether to culture infected lesions, or whether changing the empirical therapy away from β-lactams will be more effective.

Point-of-care rapid diagnostics to determine whether MRSA or PVL-producing isolates are present in lesions are now becoming available. For example, Cepheid has a SSTI kit that can detect *S aureus* and MRSA, with a reported sensitivity of 97% for MRSA in wound specimens.[75] An initial study of an immunochromatographic test for PVL that can be used with clinical specimens found the assay to have a sensitivity of 79% and specificity of 100% compared with polymerase chain reaction detection of *pvl* genes in the same *S aureus* isolates.[76] Despite these promising advances, there are currently no clinical trials to demonstrate that directing therapy against the PVL toxin improves outcome in cases of SSTI. Furthermore, the kits can be prohibitively expensive. Therefore, at this stage, such rapid diagnostics are unlikely to enter widespread clinical use, especially in the developing world.

TREATMENT OF UNCOMPLICATED SSTI CAUSED BY CA-MRSA

An important initial step in the management of staphylococcal SSTI is to recognize severe and/or complicated infection (including sepsis, toxic shock, pneumonia, and osteomyelitis). In addition, identifying certain factors that predispose to severe infection, such as extremes of age, comorbid illness (such as diabetes), malnutrition, and immunocompromised states is important. Careful clinical assessment to identify signs that suggest deeper infection or sepsis is critical. A simple algorithm for the assessment and treatment of children in the tropics who have common tropical skin infections has been developed for incorporation into the World Health Organization (WHO) Integrated Management of Childhood Illness program (**Fig. 1** and **Table 2**).[77] This algorithm, designed to assist primary health care workers, was found to be a robust tool in a validation study and is a useful guide for deciding how to proceed with the management of SSTI in children.[77]

Drainage of Abscesses

As outlined earlier, CA-MRSA frequently causes skin abscesses. In patients with small, simple abscesses (<5 cm), incision and drainage alone is usually adequate and obviates the need for antibiotic treatment.[78] However, in select situations, it may be reasonable to add an antibiotic that is active against MRSA; for example, if there is cellulitis surrounding the abscess or if adequate clinical follow-up cannot be assured.

Topical Treatments: Disinfecting Agents

In some patients with simple localized impetigo (eg, a small single lesion), the use of simple hygiene measures such as washing with clean water (±soap) often suffices as treatment by disinfecting the lesion. In addition, a variety of topical disinfecting treatments are available including hexachlorophene, chlorhexidine gluconate, povidone-iodine, and gentian violet; however, few data support their use.[79] Extensive clinical experience suggests that gentian violet is useful in simple and localized impetigo,[80] and some laboratory and clinical data suggest that gentian violet is effective against MRSA impetigo and colonization.[81,82] However, we recommend against its use in isolation in patients who have extensive impetigo, which is the most common clinical presentation in these tropical, developing countries.

Topical Treatments: Antimicrobial Agents

A Cochrane systematic review in 2004 concluded that topical treatment of localized impetigo with either mupirocin or fusidic acid is at least as effective as treatment with oral antibiotics.[83] These 2 topical agents were superior in studies comparing them with oral erythromycin, not the more routinely used agents such as cephalexin or (flu)cloxacillin. Also, the studies included in the Cochrane review were in patients with localized impetigo, not the widespread, often severe, lesions that are often seen in tropical settings. Furthermore, there is considerable risk of the rapid development of resistance for both these agents.[84–86] Other topical treatments, such as bacitracin, are either equivalent or inferior to both placebo and oral antibiotics.[83] For these reasons, we do not recommend the routine use of topical antimicrobials for treatment of impetigo in tropical regions.

Oral Antibiotics for Uncomplicated SSTI

In clinical trials for the treatment of impetigo, a consistent finding has been the high rate of clinical response to placebo (whether oral or topical); this suggests that a certain proportion of cases of impetigo will resolve without antibiotic treatment.[83] The decision to initiate oral antibiotic treatment of SSTI should be based on the extent of the disease (both in terms of number of lesions and the

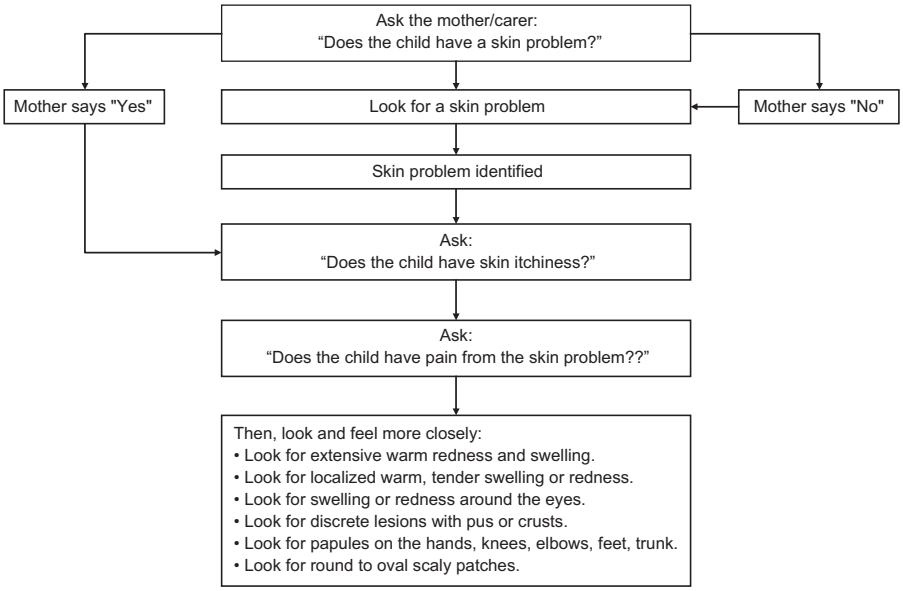

Fig. 1. Assessment algorithm for common childhood skin disorders. (*Data from* Steer AC, Tikoduadua LV, Manalac EM, et al. Validation of an integrated management of childhood illness algorithm for managing common skin conditions in Fiji. Bull World Health Organ 2009;87(3):174.)

extent of individual lesions), as well as the risk of extension of local disease and the risk of systemic bacterial invasion. In addition, there are public health considerations in trying to prevent spread to other individuals, particularly in tropical zones where poststreptococcal sequelae are common.[87] Parenteral therapy should be considered in patients with extensive disease, patients with fever, or those in whom adherence to oral therapy is likely to be poor. Intravenous therapy is indicated in patients with suspected invasive disease, as discussed later.

As outlined earlier, the most prominent pathogens that require treatment are *S pyogenes* or *S aureus*, and antibiotic choice should be directed toward both these pathogens. To date, no *S pyogenes* isolate has been found to be resistant to penicillin, and therefore a β-lactam antibiotic remains appropriate for *S pyogenes*. However, the choice of empiric antibiotic for treatment of staphylococcal SSTI is less simple and depends on local susceptibility patterns.[35] A variety of oral agents are available, including clindamycin, doxycycline, trimethoprim-sulfamethoxazole (co-trimoxazole), and linezolid. There have been no head-to-head, randomized, controlled trials of oral antibiotic therapy for CA-MRSA SSTI.

Clindamycin

In much of the United States, where the USA300 strain of CA-MRSA is most prevalent, clindamycin is commonly used for SSTI caused by CA-MRSA. Clindamycin is generally well tolerated, is well absorbed orally, with a bioavailability approaching 100%, and reaches high tissue concentrations, making it an excellent antibiotic for the treatment of soft tissue infections. Because it is a protein synthesis inhibitor, it also has the theoretic advantage of inhibiting toxin production, including PVL.[88] Its drawbacks include that it is bacteriostatic rather than bactericidal, the risk of *Clostridium difficile* infection, the need for dosing 3 to 4 times a day, poor palatability of pediatric formulations, and concerns about inducing resistance. Resistance rates to clindamycin apparently vary by strain; in the past, the USA300 strain has appeared to have been consistently susceptible to clindamycin, although recent reports suggest that this may be changing.[89,90] The situation is less clear in tropical developing regions where fewer data are available. An important caveat is the issue of inducible resistance, or macrolide-lincosamide-streptogramin B (MLSB) resistance, which is usually encoded by the *erm* gene.[91] The MLSB resistance mechanism should be tested for using the D-zone test when a staphylococcal isolate is resistant to erythromycin but susceptible to clindamycin; if the D-zone test is positive, clindamycin should not be used because of the risk of clinical failure.[92] Approximately 25% of CA-MRSA isolates in northern Australia and Hong Kong have been found to have inducible clindamycin resistance.[12,34]

Table 2
Modified WHO Integrated Management of Childhood Illness algorithm for classification of common childhood skin conditions

Sign	Diagnosis	Action
Any general danger sign (including unable to feed, vomiting all food provided, lethargy) Extensive warm redness or swelling	Very severe skin infection	Give first dose of appropriate antibiotic Refer urgently to hospital
Swelling or redness around eyes	Periorbital or orbital cellulitis	Give first dose of appropriate antibiotic Refer urgently to hospital
Localized warm tender swelling and redness	Abscess or cellulitis	Give first dose of appropriate antibiotic Refer to hospital: small abscesses (<5 cm) may be able to be drained without the need for antibiotics
Discrete sores/lesions with pus or crusts	Impetigo	If small/single impetigo lesion, consider gentian violet or simple hygiene methods such as daily cleansing with soap and clean water Otherwise, give appropriate oral antibiotic for 7 d Follow-up in 5 d
Itchiness and papules	Scabies	Provide topical permethrin skin cream Treat the whole family with permethrin cream Follow-up in 2 wk
Round to oval flat scaly patches, often itchy	Fungal infection	Give appropriate topical antifungal for 2 wk Follow-up in 2 wk
If there are not enough signs to classify in any of the above boxes OR if other signs present are not found in the above boxes	Other skin conditions	Refer to the doctor or skin clinic

Adapted from Steer AC, Tikoduadua LV, Manalac EM, et al. Validation of an integrated management of childhood illness algorithm for managing common skin conditions in Fiji. Bull World Health Organ 2009;87(3):174.

Trimethoprim-sulfamethoxazole (Co-trimoxazole)

Trimethoprim-sulfamethoxazole is also commonly used in the treatment of CA-MRSA soft tissue infection. Although it is commonly believed that *S pyogenes* is constitutively resistant to trimethoprim-sulfamethoxazole, recent data suggest that it may be effective.[93] Therefore, it may be a useful agent in tropical zones where *S pyogenes*, MSSA, and CA-MRSA are the major causes of skin infections. In a retrospective comparative study of more than 400 children with CA-MRSA SSTI, trimethoprim-sulfamethoxazole and clindamycin were found to be comparable in their effectiveness.[94] A large, randomized, controlled trial of trimethoprim-sulfamethoxazole compared with intramuscular benzathine penicillin G is currently underway in the Northern Territory of Australia (Australian and New Zealand Clinical Trials Registry ACTRN12609000858291).

Doxycycline and Linezolid

There are few data on which to base a recommendation for use of doxycycline or linezolid in the treatment of CA-MRSA SSTI. Doxycycline is inexpensive, but is contraindicated in children aged less than 8 years because of effects on dentition and bone growth. Linezolid is a protein synthesis inhibitor that has been found to be more effective than vancomycin or β-lactam antibiotics for

treatment of SSTI.[95,96] However, its use is limited by its high cost and potential for side effects such as thrombocytopenia.

TREATMENT OF SEVERE INFECTIONS CAUSED BY CA-MRSA

Invasive infections with CA-MRSA have a broad spectrum of clinical presentations, with the most common being bacteremia, severe sepsis, necrotizing pneumonia, osteomyelitis, and pyomyositis. CA-MRSA–associated necrotizing fasciitis has also been reported. Vancomycin is the mainstay of treatment of these infections, although treatment failures have been reported. Many experts recommend the addition of a protein synthesis inhibitor such as clindamycin or linezolid to decrease toxin production (including PVL and the toxic shock syndrome toxin-1). In areas where clindamycin resistance is known to be high, and where linezolid is available, it may be preferable to use linezolid initially and change to a less expensive antibiotic once susceptibility results are known. Several newer agents are also available for use against CA-MRSA, although few are used in tropical developing countries; these include daptomycin, tigecycline, and quinupristin-dalfopristin.

MANAGEMENT OF COMMUNITY OUTBREAKS

Outbreaks of CA-MRSA infections have been described in sporting teams,[97] correctional facilities,[98,99] military camps,[100,101] and some small villages.[102,103] A consistent finding is the importance of personal hygiene. Infections or carriage caused by CA-MRSA have been associated with poorer hygiene practices in correctional facilities,[98] with sharing bars of soap or towels in sporting teams,[104] and using a contaminated sauna in a rural Alaskan village.[105] General recommendations provided by the Centers for Disease Control and Prevention (CDC) Web site (http://www.cdc.gov/mrsa/prevent/index.html) to prevent transmission of *S aureus* include covering wounds; frequent hand washing; avoiding sharing personal items; washing of soiled sheets, towels, and clothes; and regular cleaning of environmental surfaces that come into contact with the skin.

Apart from interventions aimed at improving hygiene practices, some groups have attempted more aggressive case finding and decolonization of subjects found to be colonized.[102,104,106] Such an approach terminated an outbreak of PVL positive MSSA infections in a small German village.[102] All 144 members of this village were tested for colonization by nasal swab and those who were colonized with *S aureus* or had current or relapsing furuncles underwent stringent decolonization procedures together with their family members. This process involved application of nasal mupirocin 3 times daily for 5 days; daily treatment of skin and hair with an octenidine-based wash solution; antiseptic treatment of the throat with 0.1% chlorhexidine solution 3 times daily; daily disinfection of personal items with an alcohol-based antimicrobial cleanser; and daily changing and washing of towels, bedclothes, and clothing. These measures eventually ended the outbreak. The ability to carry out these interventions on a village-level scale bears witness to the impressive efforts of the public health team and investigators. However, it is doubtful that this could be accomplished in other settings, particularly those with resource limitations, inadequate facilities for bathing and washing, and overcrowded households. Our experience indicates that even the basic CDC recommendations for personal hygiene are often difficult to achieve.

Casting further doubt on the efficacy of decolonizing strategies at the community level were the results of a cluster randomized, double-blind, placebo-controlled trial of targeted mupirocin therapy in CA-MRSA–colonized soldiers.[107] In this study of 3447 soldiers, there was no statistical difference in frequency of CA-MRSA infections in soldiers found to be already colonized and then given mupirocin or placebo, and no difference in incidence of SSTI or new CA-MRSA colonizations between groups of soldiers randomized to each arm. Thus, the CDC does not currently recommend routine screening for MRSA colonization, nor for eradication of colonization in infected persons or their contacts.[108]

In a community outbreak, we would suggest interventions to improve personal and community hygiene. Such strategies have been effective in halting outbreaks in a religious community[109] and in a correctional facility.[110] If an outbreak continues unabated and resources are available, then targeted screening for colonization and subsequent eradication for cases and household contacts could be considered.

VACCINATION AGAINST *S AUREUS*

Although there has been considerable preclinical and clinical research into both active and passive immunization against *S aureus*, an effective strategy has not emerged.[111] Progress in the development of a viable active vaccine has faced several hurdles, including an incomplete understanding of pathogenesis of *S aureus* infection.

There are few clinical and epidemiologic data to support the notion that immune protection is achieved after a single vaccination against *S aureus* infection; this is particularly the case for CA-MRSA, where recurrent SSTI is common.[24] Two vaccine candidates (StaphVAX and IsdB) have reached clinical trials.[112,113] StaphVAX is a conjugate vaccine of 2 capsular polysaccharides (CP5 and CP8) that are present in many clinical *S aureus* isolates. Two phase III trials of this vaccine have been conducted in patients on hemodialysis. The first trial was promising because it showed greater than a 50% reduction in *S aureus* bacteremia in the first 40 weeks after vaccination.[112] However, the second, larger, but as yet unpublished, trial failed to show a benefit.[111] Results of a phase II trial of a vaccine using IsdB, a cell wall protein, have similarly not been published.[111] Passive immunization using antistaphylococcal antibodies has been studied in high-risk groups, particularly premature and low birth weight infants, but clinical trials in this group have failed to show a benefit in reducing bacterial infections.[114] In summary, successful immunization against *S aureus* in high-risk groups has still not been achieved, and a routinely available antistaphylococcal vaccination for the developing world remains a more distant goal.

SUMMARY

SSTI caused by *S aureus* are very common, particularly in children, in tropical regions. The proportion of *S aureus* SSTI caused by CA-MRSA varies according to region, but is up to 25% in some areas. There are diverse CA-MRSA clones, including several that harbor PVL. Key predisposing factors for staphylococcal infections are scabies infestation, overcrowding, poor hygiene, and inadequate water supplies. In the setting of a community outbreak of staphylococcal SSTI, interventions intended to improve personal and community hygiene are likely to be the most practical, effective, and achievable. Options for oral treatment of clinical infections caused by CA-MRSA include clindamycin and trimethoprim-sulfamethoxazole. Although rapid diagnostics are now available, and 2 vaccines have reached clinical trials, neither of these is likely to be of use in tropical, developing regions in the near future.

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