Epidemiology, Treatment, and Prevention of Community-Acquired Methicillin-Resistant Staphylococcus aureus Infections

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Since first described in 1961, methicillin-resistant Staphylococcus aureus (MRSA) has become a common nosocomial pathogen. Substantial increases in MRSA infections among nonhospitalized patients are being reported. Methicillin-resistant S aureus is the most common isolate from skin and soft tissue infections in selected centers in the United States. Community-acquired MRSA strains differ from nosocomial strains in clinically relevant ways, such as in their propensity to cause skin and soft tissue infection and severe necrotizing pneumonia. Clinicians in numerous specialties, particularly primary care physicians, will likely evaluate patients presenting with community-acquired MRSA and should become familiar with the epidemiology and clinical characteristics of and evolving therapeutic and preventive strategies for this infection.


CA = community-acquired; CAP = CA pneumonia; MRSA = methicillin-resistant Staphylococcus aureus; MSSA = methicillin-sensitive S aureus; PVL = Panton-Valentine leukocidin; SCC = staphylococcal cassette cartridge; TMP-SMX = trimethoprim-sulfamethoxazole

Accumulating evidence reveals the emergence of methicillin-resistant Staphylococcus aureus (MRSA) in the community.\(^1\) Many of these community isolates are distinctly different from nosocomial strains. The emergence of these distinct MRSA isolates in the community, coined community-acquired MRSA (CA-MRSA), has become a topic of intense interest. The incidence of CA-MRSA infection seems to be increasing in the United States and worldwide. Primary care clinicians will need to be knowledgeable about the epidemiology, clinical syndromes, treatments, and preventive strategies associated with CA-MRSA.

THE CHANGING FACE OF S AUREUS

Shortly after the introduction of penicillin in the 1940s, penicillin-resistant S aureus isolates were described first in hospitals and subsequently in the community.\(^1\) Today, the vast majority of staphylococcal isolates carry plasmids encoding a penicillinase-rendering penicillin resistance. Methicillin, a penicillinase-resistant semisynthetic penicillin, was introduced in 1961. Less than 1 year later, MRSA was reported.\(^1\) Today, MRSA is a common nosocomial isolate and accounts for more than 50% of S aureus isolates from intensive care units in the United States.\(^2\) In 1982, MRSA was first reported outside of the hospital among intravenous drug users in Detroit, Mich.\(^3\) Subsequently, MRSA was described in Polynesian populations in western Australia and in pediatric populations in the southern and midwestern United States.\(^4,5\) In 1999, a report of the deaths of 4 children due to severe MRSA infections in Minnesota and North Dakota garnered much attention.\(^6\) A burgeoning body of literature continues to detail the emergence of CA-MRSA. These community isolates are composed of a heterogeneous mix of strains, some apparently well suited to survival and propagation in the community.

WHAT CONSTITUTES CA-MRSA?

There is no universally accepted definition of what constitutes CA-MRSA. Epidemiologically oriented studies base the definition on the timing of MRSA isolation in culture relative to hospital admission (ie, <24-72 hours), with or without excluding patients with established MRSA risk factors (recent hospitalization, hemodialysis, indwelling catheters, etc).\(^8\) Numerous epidemiological studies have suggested that hospitals were the primary MRSA reservoir and that hospital contact accounted for most MRSA infections in the community.\(^5\) In contrast, outbreaks and small case series of MRSA infections in the community were notable for the lack of any health care exposure among cases.\(^6,6\) These reports elucidated characteristics differentiating CA-MRSA from local nosocomial strains. Molecular analysis (eg, pulse-field gel electrophoresis analysis) revealed distinct strains compared with local nosocomial isolates.\(^7\) These distinct community strains often expressed resistance to β-lactams alone, in contrast to the multidrug-resistance pattern that typified nosocomial strains. Clinicians also noted a predilection for skin and soft tissue infection in these outbreaks.\(^8\) The discovery of a novel resistance element in CA-MRSA, in conjunction with molecular epidemiological insights, has
Methicillin resistance is mediated via a chromosomally incorporated resistance gene, meca, which confers altered binding of β-lactams to penicillin binding protein 2a. The meca gene is packaged in a cassette called the staphylococcal cassette cartridge (SCC), which aids in successful chromosomal incorporation. Until 2002, only 3 SCC types (I-III) were known, but a novel fourth type has been isolated from CA-MRSA. It is becoming clear that the smaller type IV cassette, which usually does not include multiple other resistance elements, predominates among CA-MRSA strains. Rapid molecular diagnostics are available to test for the meca gene in vitro and eventually will likely be available to directly test clinical specimens. These tests may prove useful to more rapidly diagnose MRSA infections. A recent San Francisco–based study used both epidemiological and molecular techniques to characterize CA-MRSA. The strong association between type IV cassettes and CA-MRSA was confirmed. Furthermore, the study suggested that most MRSA infections in the community were related to a growing community reservoir, not a hospital reservoir, of MRSA.

Methicillin-resistant *S aureus* infections in the community are composed of escaped nosocomial isolates (particularly among people with traditional MRSA risk factors) and novel community isolates of MRSA, the latter of which we deem to be true CA-MRSA. Regardless of origin, the growing community reservoir of MRSA poses daunting new challenges to the control and treatment of MRSA infections.

Currently, CA-MRSA sensu stricto can be distinguished by the following characteristics: (1) the lack of multidrug-resistant phenotype, (2) the presence of exotoxin virulence factors, (3) type IV SCC, and (4) molecular distinction from nosocomial strains. Only an isolate’s lack of multidrug-resistant phenotype is readily available to practicing clinicians outside of research settings. Table 1 shows representative resistance patterns among CA-MRSA compared with nosocomial strains. These susceptibility patterns are dynamic and may vary markedly by region. Already, CA-MRSA strains have encroached on health care settings to cause nosocomial outbreaks, and increasing antimicrobial resistance patterns have been observed among type IV isolates. Table 2 highlights differences between CA-MRSA and nosocomial MRSA.

Community-acquired MRSA strains tend to have associated exotoxins. The most common is the Panton-Valentine leukocidin (PVL) toxin, which is lethal to neutrophils and is associated with skin and soft tissue infections (specifically cellulitis, cutaneous abscesses, and furuncles) as well as severe necrotizing pneumonia. Historically an uncommon virulence factor (present in <5% of isolates), PVL is emerging with CA-MRSA and probably in part explains the predilection for skin and soft tissue infections. Recent in vitro work revealed that PVL-positive strains of *Staphylococcus* bind preferentially to damaged respiratory epithelium. This correlates clinically with data...

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**TABLE 1. Representative Antimicrobial Susceptibilities (%) of Community-Associated and Health Care–Associated MRSA**

<table>
<thead>
<tr>
<th>Antimicrobial</th>
<th>Community-associated</th>
<th>Health care–associated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxacillin</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>79</td>
<td>16</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>83</td>
<td>21</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>44</td>
<td>9</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>94</td>
<td>80</td>
</tr>
<tr>
<td>Rifampin</td>
<td>96</td>
<td>94</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>92</td>
<td>92</td>
</tr>
<tr>
<td>Trimethoprim-sulfamethoxazole</td>
<td>94</td>
<td>90</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

*Resistance data vary by geographic region. MRSA = methicillin-resistant *Staphylococcus aureus*.
†See Table 2 and text for treatment recommendations; susceptibility does not necessarily indicate appropriate monotherapy.
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**TABLE 2. Characteristics of CA-MRSA vs Health Care–Associated MRSA**

<table>
<thead>
<tr>
<th>CA-MRSA</th>
<th>Health care–associated MRSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>At-risk groups or conditions</td>
<td>Residents in long-term care facility, patients with diabetes mellitus, patients undergoing hemodialysis/peritoneal dialysis, prolonged hospitalization, intensive care unit admission, indwelling intravascular catheters</td>
</tr>
<tr>
<td>SCC type</td>
<td>Types I, II, and III</td>
</tr>
<tr>
<td>Antimicrobial resistance</td>
<td>Multidrug resistance, common (Table 1)</td>
</tr>
<tr>
<td>PVL toxin</td>
<td>Rare</td>
</tr>
<tr>
<td>Associated clinical syndromes</td>
<td>Nosocomial pneumonia, nosocomial- or catheter-related urinary tract infections, intravascular catheter or bloodstream infections, surgical-site infections</td>
</tr>
</tbody>
</table>

*CA = community-acquired; MRSA = methicillin-resistant *Staphylococcus aureus*; PVL = Panton-Valentine leukocidin; SCC = staphylococcal cassette cartridge.
showing that PVL-associated pneumonias are associated with prior influenzalike illnesses.\textsuperscript{22} It is postulated that PVL contributes to enhanced community fitness, perhaps through enhanced transmission from draining wounds. Other exotoxins, including that responsible for staphylococcal scalded skin syndrome, have been described in community strains.\textsuperscript{21} A recent prospective study\textsuperscript{24} suggests that CA-MRSA possesses enhanced infectivity and virulence. This report showed at least a 12-fold greater attack rate among MRSA nasally colonized soldiers compared with methicillin-sensitive \textit{S aureus} (MSSA) colonized soldiers over an 8- to 10-week period. Furthermore, PVL-positive strains were found in all patients whose illness was severe enough to require hospitalization.

**POPULATIONS AT RISK**

The incidence of CA-MRSA varies regionally, but comprehensive epidemiological studies have not been published recently. Local prevalence data and antibiograms should be updated and monitored. Attendant to this, clinicians need to culture appropriate sources (furuncles, soft tissue abscesses, etc). The incidence of CA-MRSA varies also by age and is reported consistently in younger patients than is nosocomial MRSA.\textsuperscript{15} Outbreaks have occurred in several discrete patient populations. Identified at-risk populations include children (particularly those in day care centers), soldiers, prisoners, homeless persons, intravenous drug users, and men who have sex with men.\textsuperscript{16,24-26} Certain ethnic groups also have been associated with outbreaks including Pacific Islanders, Native Americans/Alaska Natives, and Pacific and Canadian aboriginals.\textsuperscript{19} Competitive athletes, specifically those who participate in fencing, rugby, football, and wrestling, have been involved in outbreaks among high school, college, and professional teams.\textsuperscript{27} Recently, an outbreak occurred in a group of divers.\textsuperscript{28} A lack of personal hygiene and a lack of basic infection-control principles probably contribute considerably to these outbreaks.\textsuperscript{27} Patients with recent or frequent antimicrobial use or persons who were recently hospitalized, have contact with others who have skin and soft tissue infections, or live in crowded quarters are also at risk.\textsuperscript{12,25,29}

**CLINICAL SYNDROMES**

Skin and soft tissue infections and lower respiratory infections account for much of the current clinical literature about CA-MRSA. Community-acquired MRSA has been reported less frequently in endocarditis, brain abscesses, bacteremia, sinusitis, and musculoskeletal infections.\textsuperscript{30-34} Recent reports about CA-MRSA causing necrotizing fasciitis, myositis, osteomyelitis, prosthetic joint infection, and complicated parapneumonic effusions highlight the various clinical settings and syndromes with which this pathogen has been associated.\textsuperscript{35-38} Such diverse manifestations may become recognized more frequently over time as the prevalence and physician awareness of this pathogen increase.

Community-acquired MRSA should be considered in the differential diagnosis of skin and soft tissue infections, particularly among patients at risk or slow to respond to \(\beta\)-lactam therapy. Furunculosis and cutaneous skin abscesses are the most common manifestations, but simple cellulitis also can occur. Recurrent furunculosis and transmission to close contacts (family members, team mates, etc) occur frequently.\textsuperscript{39} The dermonecrotic lesions encountered may be misdiagnosed as spider bites, and such lesions should suggest CA-MRSA.\textsuperscript{40} Infections may be severe, possibly because of enhanced virulence and/or ineffective initial antimicrobial therapy. A strain causing staphylococcal scalded skin syndrome has emerged in Asia.\textsuperscript{41} Although skin and soft tissue infections account for most of the morbidity associated with CA-MRSA, mortality is exceedingly uncommon.

Reports of lethal community-acquired pneumonias (CAPs) due to CA-MRSA are mounting. \textit{S aureus} is an uncommon cause of CAP, one exception being postinfluenza infection. Two reports have detailed CA-MRSA CAP from the 2003-2004 influenza season.\textsuperscript{42,43} Combined, 19 cases were reported in the United States. All had documented or suspected influenza preceding infection. Despite primarily young, healthy hosts, mortality exceeded 25%, and survivors often had prolonged stays in intensive care units with attendant morbidity. Of 15 isolates tested, 14 expressed the PVL toxin. Clinical features of PVL-positive necrotizing pneumonias may include leukopenia, hemoptysis, influenza prodrome, concomitant furunculosis, and a fulminant and often fatal course.\textsuperscript{22}

**TREATMENT**

**SKIN AND SOFT TISSUE INFECTIONS**

When choosing an empirical antimicrobial for skin and soft tissue infections, one should consider the likelihood that MRSA is the etiologic agent, the severity of the infection, and pertinent host factors including immunologic status (eg, diabetes mellitus and human immunodeficiency virus), allergies, and factors that may impede follow-up. \(\beta\)-Lactam agents currently remain the antimicrobial of choice for most skin and soft tissue infections in many if not most parts of the country. If CA-MRSA is strongly suspected on the basis of local prevalence data or epidemiological and/or clinical clues, empirical-directed therapy is indicated. Recently, interim guidelines for the evaluation and manage-
ment of CA-MRSA skin and soft tissue infections have been posted on the Internet by the Infectious Disease Society of Washington and by Washington state and county health departments. When possible, incision and drainage should be performed, and an aerobic bacterial culture should be obtained. Culture and in vitro susceptibility results should dictate which pathogen-specific antimicrobial to use. For small (<5 cm) cutaneous abcesses with no significant surrounding cellulitis or systemic symptoms, drainage alone is sufficient, provided close follow-up is available.

Empirical antimicrobial therapy is indicated for patients with larger abcesses, cellulitis, systemic symptoms, or serious comorbidities. Moderate cases treated early and appropriately may permit successful outpatient therapy. Adult patients without serious comorbidities for whom outpatient treatment is believed appropriate can be given trimethoprim-sulfamethoxazole (TMP-SMX), minocycline, doxycycline, or clindamycin (Table 3). No randomized prospective data are available to support one oral agent over another in this setting. However, the following points should be considered when choosing an outpatient oral regimen.

Clindamycin is a bacteriostatic antimicrobial that binds to the bacterial ribosomal 50S subunit, thereby inhibiting protein synthesis. Inhibition of protein production is probably beneficial in some toxin-mediated bacterial syndromes, such as streptococcal toxic shock syndrome and necrotizing fasciitis. To date, no published data suggest similar benefits when treating PVL-positive staphylococcal strains. Numerous treatment failures have been attributed to inducible clindamycin resistance. A clue to the potential presence of such lies in the in vitro susceptibility results: any erythromycin-resistant, clindamycin-susceptible isolate for which clindamycin therapy is being considered. Some microbiology laboratories may do this routinely, others only on clinician request. Alternatives to clindamycin should be considered for inducibly resistant strains.

β-Hemolytic streptococci (groups A, B, C, and G) are another extremely common cause of skin and soft tissue infections, particularly erysipelas and cellulitis. Clindamycin is usually active against these microbes, but TMP-SMX and tetracyclines are not recommended. Accordingly, if clinical suspicion for β-hemolytic streptococci and MRSA is high (eg, high-risk patient with cellulitis), combination therapy with a penicillinase-resistant β-lactam (eg, dicloxacillin, cephalaxin) and either a tetracycline or TMP-SMX provides adequate outpatient antimicrobial coverage. Although clindamycin is usually active against β-hemolytic streptococci, bacterial isolates are cultured rarely from cellulitis; hence, MRSA strains would not likely be tested for inducible resistance. Close monitoring of the therapeutic response is indicated.

Among patients with severe skin and soft tissue infections, marked systemic symptoms, or mitigating comorbid conditions, hospitalization, parenteral antimicrobials, and appropriate surgical drainage are preferred. Vancomycin remains the initial agent of choice for serious skin infections in which MRSA is suspected until culture and sensitivity data are available to tailor therapy. After substantial clinical improvement, some patients may be able to complete their antimicrobial course with an oral agent.

Several other antimicrobials are available for treatment of CA-MRSA. Linezolid has been approved for treatment of MRSA skin and soft tissue infection, is highly bioavailable, and is active against MRSA. Expense, drug interactions, and the potential for promoting linezolid resistance make this drug undesirable for widespread outpatient use. Newer fluoroquinolones (eg, gatifloxacin, moxifloxacin, and levofloxacin) have enhanced activity against Staph-

<table>
<thead>
<tr>
<th>Antimicrobial</th>
<th>Usual dosage</th>
<th>Notes</th>
<th>TABLE 3. Potential Outpatient Oral Therapies for Mild or Moderate Skin and Soft Tissue Infections When CA-MRSA Is Suspected in Adults**&lt;sup&gt;*&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>TMP-SMX DS†</td>
<td>1 DS tablet every 12 h</td>
<td>If β-hemolytic streptococci infection also is suspected, consider β-lactam therapy as well</td>
<td></td>
</tr>
<tr>
<td>(160 mg/800 mg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minocycline</td>
<td>100 mg every 12 h</td>
<td>If β-hemolytic streptococci infection also is suspected, consider β-lactam therapy as well</td>
<td></td>
</tr>
<tr>
<td>Doxycycline</td>
<td>100 mg every 12 h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clindamycin</td>
<td>300-450 mg every 6 h</td>
<td>Request double disk diffusion test for inducible resistance if isolate is erythromycin-resistant and clindamycin-susceptible</td>
<td></td>
</tr>
</tbody>
</table>

*All furuncles and abcesses should be drained, cultured, and tested for susceptibilities; mild furuncles alone (<5 cm) do not require systemic antimicrobials. CA-MRSA = community-acquired methicillin-resistant Staphylococcus aureus; DS = double-strength; TMP-SMX = trimethoprim-sulfamethoxazole.
†Adjust dose for renal insufficiency.
ylococcus, and combined with rifampin, appear effective in diverse clinical scenarios. Increasing fluoroquinolone resistance among CA-MRSA isolates and limited experience for this indication suggest that fluoroquinolones should not be first-line agents for empirical treatment. Daptomycin and quinupristin-dalfopristin are relatively new parenteral agents, each with activity against MRSA and with Food and Drug Administration approval for skin and soft tissue infection. Each appears to be similar in efficacy to vancomycin for skin and soft tissue infection. Daptomycin is taken once daily, making it an attractive option for outpatient therapy. Cost and concern about promoting resistance weigh against its routine use over vancomycin.

Therapy duration is usually 7 to 14 days, depending on severity of the infection, therapeutic response, and host factors. One recent study suggested that 5 days' duration is equivalent to 10 days' duration for uncomplicated cellulitis, but MRSA was not suspected in this patient population. In most instances, 7 to 10 days' duration is adequate. In severe cases or in patients who are slow to respond or are vulnerable hosts, treatment for 14 days or longer may be needed.

PNEUMONIA

The potential confluence of widespread PVL-positive MRSA colonization in the community with an influenza pandemic bodes dire consequences. Therefore, although CA-MRSA pneumonia is much less common than skin and soft tissue infections, the associated morbidity, mortality, and therapeutic challenges merit discussion. It is now prudent to consider CA-MRSA as an etiology of severe CAP in the correct clinical context. Severe necrotizing pneumonia with or without hemoptysis after an influenza-like illness in high-risk patients warrants therapy directed against MRSA. Unfortunately, effective therapeutic options are limited. Vancomycin traditionally has been used, but at routine dosages, it concentrates poorly in alveoli and is known to be inferior to beta-lactam therapy for severe MSSA pneumonia. Linezolid was associated with greater clinical cure rates than vancomycin in 1 retrospective subgroup analysis of a prospective nosocomial pneumonia study. However, whether this is replicable and applicable to CA-MRSA CAP remains to be seen. Daptomycin is inactivated by surfactant and has been shown to be inferior to ceftriaxone for CAP. Thus, it is an inappropriate treatment for pneumonia. The fulminant nature of these infections is presumably related to PVL toxin. Therefore, it is unclear whether more active antimicrobials alone will translate into better clinical outcomes. New and novel therapeutic approaches are needed for this severe infection. Combination therapies (eg, vancomycin plus rifampin, TMP-SMX, or clindamycin), although unproven, merit further investigation. Intravenous immunoglobulin neutralizes PVL toxin in vitro, but the clinical relevance of this remains undefined. Patients with known or suspected CA-MRSA necrotizing pneumonia should be treated in consultation with infectious disease and critical care specialists.

PREVENTION

There is great interest in measures to abort and prevent CA-MRSA outbreaks. Education of health care providers, patients, caregivers, high-risk populations, and appropriate organizations about CA-MRSA and adherence to basic infection-control principles are key to preventive strategies. Informed physicians are in a better position to recognize, treat, and appropriately counsel patients with CA-MRSA. Data are limited about which specific control interventions may be most effective. Measures that may help prevent outbreaks are listed in Table 4. Simple personal hygiene interventions appear effective in aborting outbreaks. Daily hot showers, use of antibacterial soaps and hand sanitizer, and appropriate care and coverage of any draining lesions with clean dry bandages should be stressed to patients. Personal items that may facilitate transmission, such as towels, razors, and clothing, should not be shared. Community-acquired MRSA wound drainage is considered highly infectious, and adequate coverage of draining lesions is important. When appropriate coverage cannot be attained, athletes should be withheld from competition.

### Table 4. Suggested Measures to Limit the Spread of CA-MRSA*

<table>
<thead>
<tr>
<th>Category</th>
<th>Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personal and caregiver hygiene measures</td>
<td>Shower daily using soap and hot water</td>
</tr>
<tr>
<td></td>
<td>Wash hands frequently and/or use sanitation gels</td>
</tr>
<tr>
<td></td>
<td>Cover wounds with dry, clean dressings</td>
</tr>
<tr>
<td></td>
<td>Avoid contact with wound drainage (use gloves)</td>
</tr>
<tr>
<td></td>
<td>Avoid sharing towels, razors, clothing, personal items</td>
</tr>
<tr>
<td></td>
<td>Clean cuts and abrasions with soap and water</td>
</tr>
<tr>
<td>Environmental and organizational control measures</td>
<td>Routinely clean shared equipment (eg, wrestling mats, benches, athletic equipment, whirlpools)</td>
</tr>
<tr>
<td></td>
<td>Clean and disinfect contaminated surfaces</td>
</tr>
<tr>
<td></td>
<td>Launder contaminated clothes and/or linens in hot water with detergent or bleach</td>
</tr>
<tr>
<td></td>
<td>Limit participation in contact sports unless adequate wound coverage can be obtained</td>
</tr>
<tr>
<td></td>
<td>Use a barrier (eg, clothes, towels) to bare skin when in contact with shared equipment or surfaces (eg, sauna benches, exercise machines, massage tables)</td>
</tr>
<tr>
<td>Health care—initiated measures</td>
<td>Use antimicrobials judiciously</td>
</tr>
<tr>
<td></td>
<td>Recognize and treat CA-MRSA lesions early</td>
</tr>
<tr>
<td></td>
<td>Educate and counsel patients and caregivers about appropriate wound care</td>
</tr>
<tr>
<td></td>
<td>Consider decolonization strategies for recurrent disease or in localized outbreaks in consultation with infectious disease physicians</td>
</tr>
</tbody>
</table>

*See text for references. CA-MRSA = community-acquired methicillin-resistant Staphylococcus aureus.
COMMUNITY-ACQUIRED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS

From an organizational perspective, in addition to ensuring access to the previously mentioned interventions, targeted environmental sanitation may be helpful. For instance, appropriate laundering of towels with hot water and detergent and routine cleaning of shared equipment (e.g., wrestling mats, football pads, whirlpools, etc) should be used.

Some outbreaks have been associated with frequent or recent antimicrobial use. Limiting inappropriate antimicrobial use may be beneficial in preventing outbreaks and is recommended.

Methicillin-resistant *S. aureus* decolonization has been attempted both to abort outbreaks and to prevent disease recurrence. Various decolonization strategies have been tried, but convincing evidence of their effectiveness among outpatients with CA-MRSA is lacking. Decolonization strategies have included various combinations of systemic antimicrobials (either TMP-SMX, tetracyclines, or clindamycin each with or without rifampin), mupirocin nasal ointment, and chlorhexidine body washes, usually for 5 to 7 days. Prolonged low-dose clindamycin may prevent recurrent MSSA furunculosis but has not been investigated among CA-MRSA cases; inductive resistance may limit its effectiveness in this setting. Rifampin temporarily eradicates staphylococcal nasal carriage but should not be given as monotherapy because resistance may emerge rapidly. Mupirocin resistance is described in nosocomial MRSA, but the extent in community strains is unknown. In the absence of compelling outcome data, routine attempts at decolonization of all patients presenting with CA-MRSA is not recommended. However, in cases of recurrent disease among individuals or families or in outbreak settings among discrete patient populations, it is probably a worthwhile intervention. Currently, no one regimen can be recommended over another. In vitro antimicrobial susceptibilities should be used to aid in picking an appropriate regimen. Rifampin has numerous drug interactions that need to be considered before including it in a decolonization program.

CONCLUSIONS

Community-acquired MRSA is an emerging infectious cause of morbidity and mortality among previously healthy persons in the United States and worldwide. Accumulating evidence suggests that these heterogeneous strains are particularly suited to community survival and spread. It remains to be seen whether CA-MRSA will become the predominant staphylococcal phenotype in the community, as penicillin-resistant *S. aureus* did. If so, CA-MRSA promises to affect nearly every medical specialty. Primary care clinicians will likely see most cases manifest as skin and soft tissue infections and will need to recognize, diagnose, and appropriately treat this pathogen. Use of preventive strategies and sage counsel to patients and caregivers may prove invaluable in limiting its spread.

REFERENCES


COMMUNITY-ACQUIRED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS


COMMUNITY-ACQUIRED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS

Questions About CA-MRSA

1. Which one of the following patient populations is not associated with CA-MRSA outbreaks?
   a. Prisoners
   b. Teachers
   c. Soldiers
   d. Athletes
   e. Alaska Natives

2. Which one of the following clinical manifestations is not associated with the PVL toxin?
   a. Endocarditis
   b. Cellulitis
   c. Furunculosis
   d. Necrotizing pneumonia
   e. Cutaneous abscesses

3. Which one of the following statements about CA-MRSA therapy is true?
   a. TMP-SMX monotherapy is adequate when β-hemolytic streptococci infection also is suspected
   b. Clindamycin is proved superior to minocycline for CA-MRSA skin and soft tissue infections
   c. TMP-SMX dosing does not need to be adjusted in renal insufficiency
   d. Daptomycin is the preferred therapy against CA-MRSA CAP
   e. Clindamycin-inducible resistance has been associated with treatment failures and should be evaluated with a double disk diffusion test

4. Which one of the following antimicrobials should never be used as monotherapy against CA-MRSA because resistance may emerge rapidly?
   a. TMP-SMX
   b. Minocycline
   c. Rifampin
   d. Clindamycin
   e. Vancomycin

5. Which one of the following helps distinguish CA-MRSA from health care-associated MRSA?
   a. Penicillinase
   b. Type I SCC
   c. Type IV SCC
   d. Vancomycin resistance
   e. Linezolid resistance

Correct answers:
   1. b, 2. a, 3. e, 4. c, 5. c