

# Community-Associated Methicillin-Resistant *Staphylococcus aureus*: Review of an Emerging Public Health Concern

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## ABSTRACT

Methicillin-resistant *Staphylococcus aureus* (MRSA) traditionally has been recognized as a virulent pathogen affiliated with health care institutions. However, community-associated strains of MRSA (CA-MRSA) have emerged over the past several years in young, healthy patients without significant health care contact. These isolates carry a distinct molecular makeup and lack the multidrug resistance pattern harbored by health care strains. CA-MRSA predominantly induces skin and soft tissue infections, though the presence of unique virulence factors may cause potentially lethal necrotizing pneumonia and other invasive infections. In response to this growing public health concern, clinicians must learn to identify risk factors for CA-MRSA, treat infections with judicious use of antimicrobial agents, and facilitate prevention strategies to limit transmission.

## INTRODUCTION

For the past 4 decades, methicillin-resistant *Staphylococcus aureus* (MRSA) has been one of the most widely known multidrug-resistant organisms in health care institutions. This pathogen has become nearly endemic in intensive care units; data from the US National Nosocomial Infection Surveillance system for 2004 showed that 61% of all *S aureus* isolates in this setting harbored methicillin resistance.<sup>1</sup> In conjunction with this trend, a negative impact on patient outcomes and increased economic burden on the health care system has become evident.

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Since its initial identification in 1961, many risk factors for health care-associated MRSA (HA-MRSA) infection have been recognized. These include prolonged hospitalizations, intensive care unit stays, use of invasive procedures and catheters, dialysis, other medical comorbidities, and frequent antibiotic exposure.<sup>2</sup> Over the past decade, however, community outbreaks of MRSA infections have been reported with increasing frequency in young persons without health care exposures or associated risk factors. The report of 4 pediatric fatalities from fulminant community-associated MRSA (CA-MRSA) infections in 1997-1999 alerted the medical community to the virulence of these organisms.<sup>3</sup> In most cases, CA-MRSA strains have shown a predilection to cause skin and soft tissue infections. As the incidence rises in the community setting, it is essential for primary care physicians and specialists to recognize and treat potential CA-MRSA infections appropriately. This review will discuss the epidemiology, clinical manifestations, treatment, and prevention of CA-MRSA infection.

## DEFINITION, GENETICS, AND ORIGIN OF CA-MRSA

When MRSA infections initially emerged in the community, most strains were thought to represent organisms that had spread from health care institutions. Early epidemiologic studies attempted to distinguish CA-MRSA from HA-MRSA infections as those cultured within 24-72 hours after hospital admission.<sup>4</sup> In these studies, most patients labeled with community-associated infections had previous health care exposures and associated risk factors. Patients without health care-associated risk factors were identified as having "true" community-acquired MRSA, though this was often difficult to determine on clinical grounds alone. As epidemiologic studies accrued and began to employ molecular typing techniques, how-

ever, the existence of a novel group of true CA-MRSA strains became evident. These strains hold different genotypic and phenotypic characteristics than traditional HA-MRSA isolates.

Beta-lactam and methicillin resistance in all *S aureus* strains is mediated by production of the altered penicillin binding protein 2a, encoded by the *mecA* gene. This gene is carried on a mobile chromosomal element called the staphylococcal cassette cartridge (SCC*mec*), of which there are 5 known types. HA-MRSA strains harbor SCC*mec* types II and III, which are relatively large and carry many other resistance determinants. In contrast, CA-MRSA strains predominantly carry a smaller SCC*mec* type IV element, which usually contains *mecA* as the only resistance gene.<sup>5</sup> Due to the relatively small size of SCC*mec* type IV and lack of other resistance genes, CA-MRSA strains do not confer a multidrug resistance pattern like HA-MRSA. Instead, they are susceptible to most antimicrobial agents, with the exception of beta-lactams and erythromycin.<sup>6,7</sup>

Compared to HA-MRSA, of which there are only a handful of known clones, CA-MRSA comprises a large number of heterogeneous strains well adapted to survive and spread in the community. The diversity of CA-MRSA strains is likely attributable to the small size of the SCC*mec* type IV element, which increases the ease of horizontal transfer among various genomic backgrounds. Most authorities believe that acquisition of the SCC*mec* type IV element by the numerous community-based methicillin-susceptible *S aureus* (MSSA) strains is responsible for the rise of CA-MRSA.<sup>8</sup> The SCC*mec* type IV elements may have originated from other commensal staphylococcal species, such as coagulase-negative staphylococci. A significant proportion of *Staphylococcus epidermidis* isolates from the 1970s contained the SCC*mec* type IV element, while it has been appreciably found in *S aureus* strains for only the past decade.<sup>9</sup>

In the clinical setting, without routine molecular typing, clinicians must rely on the patient history (ie, absence of health care-associated risk factors) and lack of multidrug resistance as the most reliable indicators of CA-MRSA infection. However, reliance on these criteria should not be universal. Recent reports in the literature have described transmission of CA-MRSA strains within acute care settings. One study revealed 8 women from a postpartum unit who subsequently developed skin and soft tissue infections caused by CA-MRSA strains.<sup>10</sup> In addition, a recent study identified a subset of CA-MRSA strains (approximately 14%) that expectedly carried the SCC*mec* type IV genotype, but showed a broad multidrug resistance.<sup>11</sup>

## EPIDEMIOLOGY

The prevalence of CA-MRSA in the general population is known to be increasing and extends worldwide. It is probably underreported because submission of cutaneous abscess collections for culture has not been a routine practice. Due to diverse definitions of CA-MRSA in epidemiologic studies, the reported prevalence has varied widely. There is also probably considerable geographic variation. The prevalence in Wisconsin is not yet known, although molecular typing on isolates from the northern portion of the state has confirmed that CA-MRSA is widespread.<sup>12</sup> Hospital-based studies reveal that CA-MRSA may constitute nearly 5% of all hospitalized patients with MRSA infections.<sup>4</sup> Long-term care facilities have generally been considered reservoirs for HA-MRSA, but a recent study from a San Francisco facility showed that nearly half of all MRSA isolates were identified as CA-MRSA strains.<sup>13</sup> In 2001-2002, general population-based surveillance studies were conducted in metropolitan areas of Atlanta, Baltimore, and in Minnesota. Analysis of nearly 13,000 MRSA isolates from these centers showed that 8%-20% were community-associated. Overall, the annual incidence of CA-MRSA infection was estimated at 18-26 persons per 100,000 of the general population.<sup>6</sup>

Risk factors for acquisition of community-associated MRSA remains understudied, though certain populations have demonstrated greater risk. Table 1 provides a comparison of risk factors between HA-MRSA and CA-MRSA strains. Outbreaks of CA-MRSA have occurred more consistently in younger adults and children, especially those <2 years.<sup>6</sup> Specific ethnic groups have been reported more frequently in outbreaks, such as native Americans,<sup>12</sup> Alaskan natives, and Pacific Islanders.<sup>14</sup>

Persons in crowded conditions may also be at higher risk, especially if personal hygiene is lacking. Outbreaks have been described in correctional facility inmates, military recruits,<sup>15</sup> men who have sex with men, and users of intravenous drugs.<sup>14</sup> Competitive athletic participants at all levels have been particularly susceptible to skin and soft tissue infections caused by CA-MRSA, especially in football, wrestling, and rugby.<sup>16</sup> It is presumed that a compromised skin barrier, in conjunction with close personal or contaminated surface contact, leads to the greater risk of acquisition.

## CLINICAL MANIFESTATIONS

Both health care- and community-associated MRSA are well known for their virulence and propensity to cause a wide array of clinical syndromes. Unlike HA-

**Table 1.** Comparison of Risk Factors Associated with HA-MRSA and CA-MRSA Infections<sup>17</sup>

HA-MRSA Infections	CA-MRSA Infections
Older age: elderly	Younger age: children, young adults
Prolonged hospitalization	Certain ethnic groups: Native American, Alaskan natives, Pacific Islanders
Intensive care unit admission	Persons in crowded living conditions
Hemodialysis	Correctional facility inmates
Indwelling lines and catheters	Users of intravenous drugs
Invasive procedures	Men who have sex with men
Comorbid medical conditions	Athletes in contact sports
Institutionalization (nursing home)	Persons exposed to frequent antibiotic use
	Persons exposed to MRSA close contacts

HA-MRSA = health care-associated methicillin-resistant *Staphylococcus aureus*; CA-MRSA = community-associated methicillin-resistant *Staphylococcus aureus*.

MRSA strains, however, the predominant presentation of CA-MRSA strains are skin and soft tissue infections. Invasive CA-MRSA infections such as necrotizing pneumonia, though less common, have a more dramatic and morbid clinical course.

The presence of several different virulence factors in CA-MRSA strains, many of which are newly described, may account for this divergence of clinical syndromes. Of greatest significance are the Panton-Valentine leukocidin (PVL) genes. Although historically uncommon and found in less than 5% of all *S aureus* strains, they are identified in over 93% and 85% of CA-MRSA strains causing furunculosis and necrotizing pneumonia, respectively.<sup>18</sup> Despite their high prevalence in correlation with SCC*mec* type IV isolates, these genes are genetically unassociated with this element. The PVL genes encode a pore-forming cytotoxin that preferentially targets leukocytes and erythrocytes. In turn, this induces an intense inflammatory cascade and enzymatic tissue necrosis. This pathogenesis is likely responsible for the more aggressive nature and higher morbidity associated with PVL positive strains in comparison to PVL negative isolates.

Skin and soft tissue infections account for over 75% of infections caused by CA-MRSA,<sup>7</sup> and up to 90% of those described in the pediatric literature.<sup>19</sup> The majority of these infections include cutaneous abscesses and furuncles, as well as cellulites.<sup>20</sup> Skin lesions may classically appear necrotic and be misdiagnosed as “spider bites” before progression to more invasive infection.

Impetigo and folliculitis occur less commonly, and rare cases of scalded skin syndrome have been caused by infection with strains harboring an exfoliative toxin.<sup>21</sup> Invasion of these skin infections to deeper soft tissues is rare, although infection of pre-existing wounds with CA-MRSA is becoming more prevalent. Progression of skin and soft tissue infections to bacteremia and septic shock has occurred only rarely.

Although community-acquired pneumonia is a relatively infrequent manifestation of CA-MRSA infection, it carries a very high morbidity. Unlike HA-MRSA pneumonia, CA-MRSA strains produce necrotic foci and abscesses in the lung parenchyma, a finding largely attributable to the PVL cytotoxin. Most patients exhibit high fevers and hemoptysis, and many have concomitant furunculosis. Progression to septic shock and acute respiratory distress syndrome is usually rapid and inevitable.<sup>22</sup> The acquisition of CA-MRSA pneumonia in patients recently afflicted with an influenza-like illness may be especially catastrophic. Although 2 small studies identified only 19 such cases in the 2003-2004 influenza season, a mortality rate exceeding 25% was seen in this cohort of otherwise young, previously healthy persons.<sup>14</sup> This development will become a greater threat as the prevalence of CA-MRSA increases.

## MANAGEMENT

The Wisconsin Division of Public Health has recently issued guidelines for the management of CA-MRSA skin and soft tissue infections.<sup>17</sup> Most of these cutaneous infections are of mild to moderate severity, and can be managed in the outpatient setting. Incision and drainage should be the initial step in the management of abscesses and furunculosis. Purulent material should be collected and sent for aerobic culture. For cases of mild illness—in which the patient is afebrile, has a relatively small abscess (less than 5 cm), and lacks other medical comorbidities—incision and drainage with or without topical antibiotics may be a sufficient and definitive therapy.<sup>23</sup> Patients with a moderate illness—especially with fever, stable comorbidities (eg, diabetes mellitus), or cellulitis greater than 5 cm in diameter—should additionally receive antimicrobial therapy. Antibiotics may be considered for the initial treatment if incision and drainage is not possible, but the rapid tissue destruction caused by CA-MRSA strains often leads to development of abscesses despite appropriate therapy. Thus, close follow-up is always warranted, and incision and drainage may eventually become necessary.

When determining appropriate antibiotic coverage for any characteristic skin or soft tissue infection in

**Table 2.** Selected Antimicrobial Agents for CA-MRSA Skin and Soft Tissue Infections

Antimicrobial Agent*	% Susceptible†	Dosage (Adult)‡	Notes
TMP-sulfa DS (160 mg/800 mg)	90%-95%	1 DS tab every 8-12 hours	Does not cover Group A streptococcus
Doxycycline	90%-95%	200 mg load, then 100 mg tab every 12 hours	Does not cover Group A streptococcus
Clindamycin	70%-80%	300 mg tab every 6 hours	Indicated only if negative "D test," or isolate sensitive also erythromycin

CA-MRSA = community-associated methicillin-resistant *Staphylococcus aureus*; TMP-sulfa = trimethoprim-sulfamethoxazole; DS = double strength.

\*Duration of therapy: 7-14 days. Should be accompanied by incision and drainage whenever possible.

† Percent CA-MRSA strains susceptible to corresponding antimicrobial agent.<sup>6,7,17</sup>

‡ Use weight-adjusted dosing guidelines for pediatric patients.

clinical practice, an assessment for risk factors affiliated with CA-MRSA and local prevalence data should be utilized. Aside from the at-risk populations described previously, other clinical clues may suggest underlying CA-MRSA infection. Patients with suspected close contact or exposure to CA-MRSA, recurrent skin disease, recent and frequent antibiotic use, or disease poorly responsive to beta-lactam therapy may be considered at higher risk.<sup>17</sup> With these factors, treatment should favor coverage of MRSA over MSSA, especially if the local prevalence of CA-MRSA is >15%.<sup>24</sup>

It is likely that a majority of CA-MRSA skin and soft tissue infections are initially inappropriately treated with a traditional first-line beta-lactam agent. Due to lack of multidrug resistance in community-associated strains, however, several other oral antibiotics are available. Table 2 shows the preferred agents, though antimicrobial susceptibility patterns may vary by region. Trimethoprim-sulfamethoxazole and doxycycline are acceptable agents, with susceptibility rates around 90%-95% in CA-MRSA strains.<sup>6,7</sup> Despite potentially limited tissue penetration, they are usually effective against CA-MRSA with associated incision and drainage. It should be noted that neither of these antimicrobials cover Group A streptococcus, a common cause of erysipelas and cellulitis.

Clindamycin is more broadly effective against gram-positive organisms, but has a susceptibility rate of only 70%-80% in CA-MRSA isolates.<sup>6,7,17</sup> There is also growing concern over "inducible" clindamycin resistance, an occurrence noted increasingly in erythromycin-resistant, clindamycin-sensitive strains.<sup>25</sup> In these isolates, resistance to clindamycin is induced by mechanisms that mediate erythromycin resistance (ie, efflux pumps, ribosomal methylation). To assess for inducible clindamycin resistance in the laboratory, all erythromycin-resistant, clindamycin-sensitive strains should be analyzed with the double disk diffusion test (D test).<sup>26</sup>

This is done only at clinician request in some laboratories, but others are now performing this routinely. Unfortunately, this results in a delay of 24-48 hours in susceptibility reporting. While clindamycin is acceptable for isolates susceptible to both erythromycin and clindamycin, other therapy should be considered for inducibly resistant strains.

Patients with severe presentations of skin and soft tissue or other invasive CA-MRSA infections should be hospitalized and receive intravenous antibiotic therapy. If CA-MRSA is suspected, a non-beta-lactam antimicrobial effective against MRSA, such as vancomycin, should be started empirically while cultures are pending. Other newer second-line intravenous options for skin and soft tissue infections include linezolid, quinupristin-dalfopristin, daptomycin, and tigecycline. Linezolid is also available in oral formulation, but should be limited for outpatient use due to expense and availability of acceptable alternatives.

### ERADICATION OF MRSA COLONIZATION

For decades, many efforts have been undertaken to eradicate *S aureus* and MRSA colonization, especially in health care settings. Patients with *S aureus* nasal colonization show a greater likelihood for subsequent infection, and the risk with MRSA is even higher than MSSA. Several studies have demonstrated a 10-fold increased risk of infection after recent nasal acquisition with MRSA over MSSA.<sup>27,28</sup> Left alone, colonization may last for days, months, or even years.<sup>15</sup>

In health care settings, topical intranasal mupirocin has been shown to be effective for short-term eradication of nasal colonization, but a high rate of recolonization and potential for resistance with long-term use has been demonstrated.<sup>29</sup> Thus, the practice of MRSA screening and decolonization in health care settings should not be routine, and benefits may be limited only to specific scenarios. The Wisconsin Division of Public

**Table 3.** Key Distinguishing Features between Health Care-Associated MRSA and Community-Associated MRSA\*

	HA-MRSA	CA-MRSA
Health care contact	Yes	No
Mean age at infection	Older: elderly	Younger: children and young adults
Skin and soft tissue infections	35%	75%
Resistance gene	SCCmec types I, II, III	SCCmec types IV, V
Antibiotic resistance	Multidrug resistance	Widely susceptible, except beta-lactams and erythromycin
PVL toxin gene	Rare (5%)	Frequent (>95%)

HA-MRSA = health care-associated methicillin-resistant *Staphylococcus aureus*; CA-MRSA = community-associated methicillin-resistant *Staphylococcus aureus*; SCCmec = Staphylococcal cassette cartridge with *mecA* gene; PVL = Panton-Valentine Leukocidin.

\*Adapted with permission from Wisconsin Division of Public Health, Barry C Fox, MD.

Health guidelines provide a protocol for the eradication of health care-associated MRSA colonization.<sup>17</sup>

There has been minimal evidence to support the use of mupirocin for decolonization of CA-MRSA in the community, and no clear screening recommendations exist. Candidates may include patients with recurrent CA-MRSA skin and soft tissue infections, or high-risk household contacts of patients with skin and soft tissue infections.<sup>17</sup> Prevention of transmission in these settings is of greater importance than strategies for eradicating colonization.

### PREVENTION OF TRANSMISSION

It has been widely known that direct contact via the hands or gloves of health care workers is the major mode for transmission of HA-MRSA and other multidrug-resistant organisms in health care institutions.<sup>30</sup> Thus, infection control in these settings has focused on contact precautions and strict hand washing with alcohol-based cleansers.<sup>31</sup> These policies should be applied to the ambulatory care setting to prevent further transmission of both HA-MRSA and CA-MRSA strains.

In the community, prevention of CA-MRSA transmission is particularly dependent on several measures. Crowded environments, such as close household contacts, correctional facilities, sports locker rooms, health clubs, daycares, and homeless shelters, should be viewed as high-risk venues for transmission. Clinicians should strive for early detection of CA-MRSA in skin and soft tissue infections, and report cases to the local health department.<sup>17</sup>

Close household contacts of patients with CA-MRSA infections should be attuned to maintaining personal hygiene and a clean environment. As in health care institutions, hand washing is essential, and wound changes are to be performed with disposable gloves. In addition, personal items such as towels and razors should not be shared, and unnecessary shaving avoided. Linens should be changed frequently and cleaned in hot water.<sup>14,17</sup>

Persons using health clubs, sports participants, and coaches must have proper education on these guidelines as well. Coaches and athletic trainers should be trained in first aid for wounds and recognize those that are potentially infected. If wounds cannot be adequately covered, exclusion of athletes from participation should be considered.<sup>17</sup> At the same time, coaches should encourage athletes to report skin lesions. Adequate hygiene must also be emphasized in the locker room, and shared equipment be regularly cleaned at health clubs.<sup>14,17</sup> The Wisconsin Division of Public Health guidelines contain patient education materials on the prevention of CA-MRSA transmission.<sup>17</sup>

### CONCLUSION

The emergence of CA-MRSA is becoming a growing public health concern. Primary care physicians will be at the frontlines in managing skin and soft tissue infections caused by CA-MRSA, and must be aware of the differences between health care- and community-associated strains. The major comparisons are summarized in Table 3. With the utilization of molecular typing techniques, much progress in the classification of these novel community-associated strains has been made over the past several years. These methods have enabled the identification of CA-MRSA by the SCCmec type IV genotype and lack of multidrug resistance. However, as the prevalence continues to rise, the epidemiology likely will continue to change, and more prospective studies on clinical risk factors for acquisition will be needed.

To limit the continuing emergence of CA-MRSA, clinicians must continue to use antimicrobial therapy judiciously and use incision and drainage whenever possible. Although eradication of MRSA nasal colonization would appear initially attractive to reduce the risk of subsequent infection, routine use of intranasal mupirocin is not recommended at this time and should be applied only to specific circumstances. Similar to infection control measures in health care institutions, prevention of transmission in the community setting through educating patients and community organizations will likely prove a greater benefit.

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