

Community Associated Methicillin-Resistant *Staphylococcus Aureus* in HIV-Infected Patients

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Abstract

Until recently, methicillin-resistant *Staphylococcus aureus* was considered an uncommon community pathogen, almost exclusively associated with healthcare exposure. Over the last decade, however, methicillin-resistant *S. aureus* infection, particularly skin and soft tissue infection, has emerged in healthy individuals with no traditional risk factors for its acquisition. Several risk factors, including certain lifestyle behaviors, have been associated with community-acquired methicillin-resistant *S. aureus* colonization and infection. Regardless of other concurrent risk factors, HIV-infected patients have an increased risk for acquiring this pathogen. This article summarizes the current knowledge regarding associated risk factors, clinical manifestations, and management of community-acquired methicillin-resistant *Staphylococcus aureus* infections in HIV-infected patients. (AIDS Rev. 2010;12:153-63)

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Key words

HIV. Community-acquired methicillin-resistant *Staphylococcus aureus*. Bacterial infections. MRSA.

Introduction

Until recently, methicillin-resistant *Staphylococcus aureus* (MRSA) was considered a nosocomial pathogen. Infections caused by MRSA were almost exclusively associated with healthcare exposure such as recent hospitalization, intensive care unit stay, prolonged antimicrobial therapy, surgery, residence in long-term care facilities, dialysis, or presence of medical devices. In the last decade, however, MRSA has also appeared as an emergent causal agent of infection in the otherwise healthy population and in individuals with underlying conditions, but without traditional risk

factors for healthcare-associated MRSA infection¹⁻³. Methicillin-resistant *S. aureus* colonizing or infecting outpatients who have not been exposed to the established risk factors has been called community-acquired or community-associated MRSA (CA-MRSA)⁴.

Despite the growing prevalence of MRSA in hospitals, these strains have been uncommon in the community. There is molecular evidence that MRSA strains have evolved in the community, are well adapted to survive there, and show genetic features distinctive from MRSA strains acquired in hospitals^{5,6}. Two main clones, USA400 and USA300 (belonging to ST1 and ST8 by multi-locus sequence typing, respectively), are reported to be responsible for most CA-MRSA infections occurring in the USA^{5,7-9}. On other continents, the most frequent STs of CA-MRSA are ST30 for the Southwest Pacific clone, and ST80 for the European clone^{6,10}, although the ST8 (USA300) and ST30 clones have also been extensively described in Europe⁹⁻¹². The *mecA* gene, required for expression of methicillin resistance, is carried by a genetic element known as the staphylococcal cassette chromosome *mec* (SCC*mec*)¹³. Most CA-MRSA show a SCC*mec* type-IV structure (and less commonly, types V, VI, and VII), which differs from the structures

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encoding methicillin resistance in hospital-acquired MRSA⁹. However, in recent years, SCC*mec* type IV has also been found in nosocomial MRSA isolates, mainly in Europe¹⁴. The SCC*mec* types associated with CA-MRSA are smaller and more transmissible than those related with hospital-acquired MRSA^{9,15}. Another significant difference is that almost all CA-MRSA isolates contain genes encoding Panton-Valentine leukocidin (PVL), a cytotoxin related to leukocyte destruction and tissue necrosis¹⁶⁻¹⁸. Although the exact role of PVL in severe infection is unclear, it is usually considered as a marker for community MRSA acquisition.

Transmission of nosocomial MRSA strains is common within the healthcare setting, but there is little spread between household contacts. On the other hand, CA-MRSA occurs in clusters, spreading through family members and community associates¹⁹.

Initial reports of CA-MRSA, mainly skin and soft tissue infections and often presenting as outbreaks, were described in selected populations whose common characteristic is close person-to-person contact, such as prisoners or correctional facility inmates^{20,21}, men who have sex with men²², athletes²³, young children in day-care facilities³, military personnel^{24,25}, aborigine populations^{26,27}, and injection drug users^{2,28,29}.

In the last decade, the prevalence of CA-MRSA infection has increased, and this pathogen has become an important cause of skin and soft-tissue infection in the general population in the USA and Europe. In recent years, the CA-MRSA USA300 genotype has been reported as the most common cause of community-occurring staphylococcal skin and soft-tissue infection in patients admitted to emergency departments in several urban areas in the USA^{30,31}. Although the prevalence in other parts of the world is not yet as high as in the USA, an important worldwide spread of CA-MRSA clones has been reported during recent years^{6,10-12}.

Epidemiology of community-acquired methicillin-resistant *Staphylococcus aureus* infection in HIV-infected patients

Before the emergence of the CA-MRSA epidemic, HIV infection was associated with an increased risk of *S. aureus* infection that was mainly related to intravenous drug use, skin lesions, presence of an intravascular catheter, or advanced immunosuppression^{32,33}.

As the relevance of CA-MRSA infection has progressively increased in the general population over the last years, this trend has also been observed in HIV-infected patients^{34,35}. Furthermore, the incidence of CA-MRSA

infection in HIV patients appears to be significantly higher than in the general population. Several recent cohort studies have documented the impact of CA-MRSA infection in the HIV-infected population. In a large cohort study of all patients seen in an urban HIV clinic in the USA from 1993 through 2005, Crum-Cianflone, et al. reported that 7% of patients developed CA-MRSA infection (90% of them skin and soft tissue infections). This incidence was 18-fold higher than in the general population and it has significantly increased since 2003³⁶. In another recent study in both community and hospital settings in the USA, Popovich, et al. noted that the incidence of CA-MRSA skin and soft tissue infections in HIV-infected patients significantly increased from 411 cases per 100,000 HIV-infected patients during 2000 to 2003, to 1,474 cases per 100,000 HIV-infected patients during 2004 to 2007, and this incidence was > 6-fold higher than it was in HIV-negative individuals³⁷. In this study, a > 4-fold increase in skin and soft tissue infections between periods was also reported in hospitalized HIV-infected patients and it was in addition to, not replacement of, methicillin-sensitive *S. aureus* (MSSA). In a cohort of 4,607 patients attended in an urban outpatient HIV clinic in the USA, Burkey, et al. observed an increase in the incidence of MRSA bacteremia from 5.3 to 11.9 /patients-year between 2000 and 2004. In another clinic-based study, Mathews, et al. reported 126 CA-MRSA infections in 3,455 HIV-infected individuals between 2000 and 2003, with a 6.2-fold increase from the beginning to the end of the study period. Emergence of CA-MRSA has also been noted in children and young adults with HIV infection³⁸ (Fig. 1). Almost all the studies focusing on this issue come from the USA. Some of the studies performed in Europe have not shown a similar trend³⁹.

With the extended use of HAART, the number of hospital admissions related to opportunistic infectious disease in HIV patients has significantly declined, and non-AIDS-related comorbidities have become the most common cause of hospitalization. In this context, MRSA infection is reported to be one of the emerging causes of non-AIDS-related hospitalizations in this population⁴⁰.

Risk factors for community-acquired methicillin-resistant *Staphylococcus aureus* colonization and infection in HIV-infected patients

In several cohort studies, HIV infection itself has been identified as an independent risk factor for CA-MRSA colonization or infection. Therefore, regardless of the

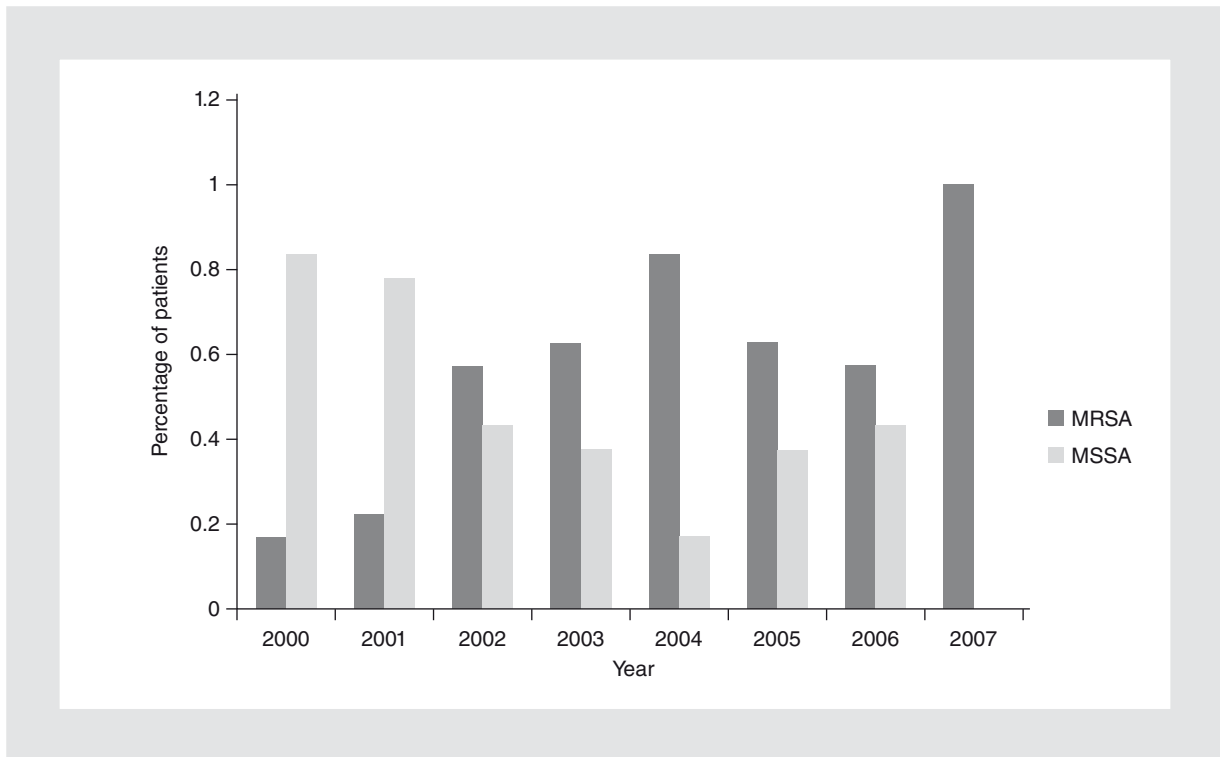


Figure 1. Increasing proportion of community-acquired methicillin-resistant *Staphylococcus aureus* infections among children and young adults with HIV infection (adapted from Srinivasan, et al.³⁸). MRSA: methicillin-resistant *Staphylococcus aureus*; MSSA: methicillin-sensitive *Staphylococcus aureus*.

presence of other high-risk factors, such as male-male sex or injection drug use, HIV-infected persons are more likely to be colonized or have clinically significant CA-MRSA infection compared to the HIV-negative population^{37,41-46}. In any case, the risk of CA-MRSA colonization or infection increases in accordance with the number of risk factors present⁴³.

Risk factors independent of HIV infection

Certain risk behaviors related to HIV acquisition have also been associated with CA-MRSA colonization and infection. Thus, some HIV-infected patients may be exposed to several risk factors for CA-MRSA infection, which could explain, in part, its high incidence in this population.

The association between male-male sex and the risk of MRSA infection has been well documented. Diep, et al. conducted a large observational study including all patients who had been treated for MRSA infection at nine of the ten medical centers in San Francisco and in a primary care organization in Boston between 2004 and 2006. The incidence of CA-MRSA infection was found to be significantly higher in men who have sex with

men (MSM)⁴¹. This study also focused on a subset of patients seen for CA-MRSA infection in an HIV clinic, and the authors observed that male-male sex was a risk factor for CA-MRSA infection regardless of HIV infection.

In other studies restricted to the HIV-infected population, a higher incidence of CA-MRSA infection in MSM has also been noted⁴⁷. More than 30% of CA-MRSA skin and soft tissue infections in HIV-infected MSM involve the buttocks, genitals, and perineum. Several factors that could explain person-to-person CA-MRSA spread in this population have been described, such as close skin-to-skin contact with someone with skin or soft tissue infection, multiple sexual partners, prior sexually transmitted infection, and other high-risk sex behaviors^{36,46,48}. Moreover, public hot tub or sauna use has also been related to acquiring CA-MRSA infection; hence, indirect mechanisms of transmission are also possible^{46,48}. Nonetheless, other studies have found a lower prevalence and no clear risk factors for CA-MRSA colonization and infection in HIV-infected MSM⁴⁹.

Injection drug use (IDU) is associated with a higher risk of *S. aureus* infection⁵⁰ in both HIV-infected and noninfected patients. During the last decade, with emergence of CA-MRSA infection, injection drug users

Table 1. Risk factors associated with community-acquired methicillin-resistant *Staphylococcus aureus* infections in HIV-infected patients

Study	Design	Year	Risk factors	Infection
Diep, et al.	Retrospective	2004-2006	MSM Prior MRSA infection Prior clindamycin use	SSTI
Crum-Cianflone, et al.	Retrospective	1993 -2005	Low CD4 cell count High plasma HIV viral load Recent use of β -lactam antibiotics History of syphilis	90% SSTI
Mathews, et al.	Retrospective Clinic-based cohort	2000- 2003	MSM IDU CD4 cell count < 50/ μ l High plasma HIV viral load Absence of TMP-SMX prophylaxis	SSTI
Lee, et al.	Retrospective case-control	2002-2003	Prior hospitalization High-risk sex behavior History of STD Prior ciprofloxacin use Absence of TMP-SMX prophylaxis	Skin infections
Trinh, et al.	Retrospective	2004-2006	Immune status and plasma HIV viral load not associated with increased risk	SSTI
Srinivasan, et al.	Retrospective	2000-2007	No different risk factors than HA-MRSA	SSTI Eye infections
Skiest, et al.	Prospective	2003-2004	Prior hospitalization Recent antibiotic use	SSTI
Burkey, et al.	Retrospective	2000-2004	Low CD4 cell count Detectable plasma HIV viral load History of IDU End-stage renal disease	Bacteremia
Szumowski, et al.	Prospective	2005-2007	History of MRSA infection or colonization Antibiotic use prior 6 months High-risk sex behavior History of STD Illicit drug use No association with: CD4 cell count < 200/ μ l HAART receipt TMP-SMX prophylaxis	SSTI
Popovich, et al.	Retrospective	2000-2007	Alternative housing (subsidized housing, shelters, substance-abuse centers) Location of residence No association with: CD4 cell count	SSTI

CA-MRSA: community-acquired methicillin-resistant *Staphylococcus aureus*; HA-MRSA: hospital-acquired methicillin-resistant *Staphylococcus aureus*; IDU: injection drug user; MSM: men who have sex with men; SSTI: skin and soft tissue infections; STD: sexually transmitted disease; TMP-SMX: trimethoprim-sulfamethoxazole.

appear to be a selected population with an increased risk of CA-MRSA colonization and infection^{2,28,51,52}. In addition, IDU has been described as an independent risk factor for CA-MRSA bacteremia in HIV patients⁵³.

Risk factors related with HIV infection

Impaired cellular immunity is a hallmark of HIV infection, but HIV patients, particularly those with advanced

infection, may also have inadequate humoral immunity and abnormal chemotaxis, phagocytosis, and bactericidal activity, resulting in increased susceptibility to bacterial pathogens, including *S. aureus*⁵⁴⁻⁵⁷. The role of HIV-related immunosuppression as a risk factor for CA-MRSA acquisition has been assessed in various studies, but the results are controversial (Table 1). Several authors have found an association between low CD4+ T lymphocyte count (< 200/ μ l) and a higher

risk of both asymptomatic colonization^{45,58,59} and clinically significant CA-MRSA infection^{36,47,53}. In contrast, this association has not been confirmed in other studies^{37,46,48,60,61}. It is likely that other overlapping risk factors could have overshadowed the influence of immunosuppression in some cases. Most studies have focused on selected HIV-infected populations with a high prevalence of other risk factors for CA-MRSA acquisition, such as MSM. Furthermore, the use of trimethoprim-sulfamethoxazole (TMP-SMX), which is given to severely immunosuppressed patients as prophylaxis against opportunistic infections, has been identified as protective for CA-MRSA acquisition^{47,48,58} and it could lead to underestimation of the effect of immunosuppression on the risk of developing CA-MRSA infection. In any case, although CA-MRSA infection is also commonly reported in HIV-infected patients with high CD4 cell counts receiving antiretroviral therapy, some studies have indicated that the most severely immunosuppressed HIV patients may have an increased risk for CA-MRSA; hence, further studies are needed to clarify the influence of immune status on the risk of developing CA-MRSA infection.

The effect of plasma viral load and HAART are also controversial. High plasma HIV viral load has been associated with an increased risk of CA-MRSA infection in some studies^{36,47,53}, and HAART use has been found to be protective⁵⁹, but these findings are not consistent with those of other studies^{45,46,48,60}. Of note, these data come from observational studies and some of them involve selected populations with other concurrent risk factors.

Other risk factors

Other risk factors previously found to be related to healthcare-associated MRSA in HIV-infected patients have been also described in association with CA-MRSA. Prior hospitalization, antimicrobial use within the previous 6-12 months, invasive procedures, and a history of previous MRSA or MSSA infection have also been associated with an increased risk of acquiring CA-MRSA infection in the HIV population^{33,35,48}.

Asymptomatic colonization and risk of community-acquired methicillin-resistant *Staphylococcus aureus* infection

Staphylococcus aureus is both a commensal and pathogenic agent. The anterior nares are the main reservoir of *S. aureus*, but other sites can be colonized.

The association between skin and mucosal colonization and invasive *S. aureus* infection has been well documented in the general population and in HIV patients⁶³⁻⁶⁵. Other colonization sites are also reported to increase the risk of *S. aureus* infection⁶⁶. As has been described for MSSA and healthcare-associated MRSA, nasal carriers of CA-MRSA also have an increased risk of skin infection, and nasal carriage of this microorganism can even increase the risk of skin infection in household members who are not MRSA carriers⁶⁷.

Several studies have shown that HIV-infected individuals have an increased risk of CA-MRSA nasal colonization^{43,58} and that nasal carriage is a risk factor for CA-MRSA infection in these patients^{46,59,60} (Table 2). Furthermore, the well-documented transmission of CA-MRSA in both MSM and heterosexual couples has reinforced the idea that other sites of colonization could play a role in CA-MRSA transmission and subsequent infection^{46,52,68-70}.

The high prevalence of asymptomatic CA-MRSA colonization in the HIV-infected population may contribute to the risk of person-to-person transmission and infection.

Clinical presentation

The most common clinical presentation of CA-MRSA infection is skin and soft-tissue infection such as furuncle, abscess (commonly in multiple locations), wound infection, and cellulitis. Community-acquired MRSA isolates containing PVL genes have been associated with recurrent and occasionally severe primary skin infections as well as necrotizing fasciitis and myositis⁷¹⁻⁷³. However, it is not clear whether CA-MRSA is more virulent than MSSA^{38,59,74,75}.

Community-acquired MRSA infection of the respiratory tract is less common, but severe necrotizing pneumonia has been reported in patients infected with PVL-positive CA-MRSA⁷⁶⁻⁷⁸. Other clinical presentations include bloodstream infection⁷⁹ and rare localizations secondary to bacteremia, such as endocarditis⁸⁰, mycotic aneurysm⁸¹, arthritis, osteomyelitis⁸²⁻⁸⁴, infections of the eye and orbit, epidural abscess, meningitis and brain abscess⁸⁵⁻⁸⁸, and severe sepsis⁸⁹.

In HIV-infected patients, the clinical presentation of CA-MRSA infection does not significantly differ from that of HIV-negative persons. As in the general population, the most common infections are skin and soft tissue infections, but other forms such as bacteremia⁵³, endocarditis⁹⁰, and other serious infections have also been described⁹¹ (Fig. 2).

Table 2. Risk factors associated with community-acquired methicillin-resistant *Staphylococcus aureus* colonization in HIV infected patients

Study	Design	Year	Risk factors
Seybold, et al.	Prospective	2006-2007	CD4 cell count < 200/ μ l
Genizal, et al.	Prospective	2005	CD4 cell count < 200/ μ l Prior MRSA or MSSA infection Prior hospitalization Protective: recent use of TMP-SMX No association with: HIV risk factor incarceration
Ramsetty, et al.	Retrospective	2002-2007	CD4 cell count < 200/ μ l Antibiotic exposure during last year

CA-MRSA: community-acquired methicillin-resistant *Staphylococcus aureus*; MSSA: methicillin sensitive *Staphylococcus aureus*; TMP-SMX: trimethoprim-sulfamethoxazole.



Figure 2. Community-acquired methicillin-resistant *Staphylococcus aureus* skin abscess in a 41 year-old-man with HIV infection. He was severely immunosuppressed (CD4 cell count 153/ μ l) although plasma viral load was undetectable (< 40 copies/ml). There were no other risk factors for CA-MRSA. He had lesions in several locations. MRSA: methicillin-resistant *Staphylococcus aureus*; CA: community acquired.

Skin and soft tissue infections caused by CA-MRSA are not more severe in HIV-positive than HIV-negative cases and they are usually diagnosed and treated in the outpatient setting. However, a higher frequency of recurrent infection has been described in the HIV population⁹²⁻⁹⁵. Although some authors have reported higher hospitalization rates for CA-MRSA skin and soft tissue infections in HIV-positive than HIV-negative cases, this fact does not seem attributable to a different clinical presentation⁴⁴. Some anatomic sites of infection are more common in certain HIV patient subgroups, such as skin infection in the buttocks and genital area, commonly described in MSM⁴⁶⁻⁴⁸.

Management

Since CA-MRSA has emerged as a common causal agent of skin and soft tissue infections in HIV-infected patients in the community setting, clinicians should now consider MRSA coverage when empirical treatment is administered for skin and soft tissue infections or other infections in which CA-MRSA could be implicated.

In contrast with healthcare-associated MRSA strains, CA-MRSA isolates usually remain broadly susceptible to several non- β -lactam antibacterial agents. Sensitivity to clindamycin, TMP-SMX, doxycycline, vancomycin, and rifampin is generally described; thus, these are good options for treating CA-MRSA infection^{31,42,48,51}. Increasing rates of ciprofloxacin resistance have been reported in CA-MRSA isolates, and these agents are not recommended as empirical treatment³¹. There have been some reports of USA300 isolates resistant to several or multiple antimicrobial agents^{7,22}, but this situation remains rare.

Treatment of skin and soft tissue infections

Antimicrobials such as clindamycin, doxycycline, and TMP-SMX are recommended for empirical treatment of CA-MRSA skin and soft tissue infections, although only some of them have been evaluated or compared in randomized clinical trials^{96,97}.

Usually, TMP-SMX is active against most CA-MRSA isolates and is the most commonly used agent for treating skin and soft tissue infections. It seems that TMP-SMX is the most appropriate empirical antibiotic for suspected MRSA skin and soft tissue infections, especially in settings where clindamycin resistance is common.

However, oral TMP-SMX may be insufficient in cases of serious CA-MRSA infection apart from skin and soft tissue infections⁹⁵.

Clindamycin is a commonly used and effective therapy for uncomplicated CA-MRSA skin and soft tissue infections. However, an increasing incidence of CA-MRSA strains resistant to clindamycin have been described⁹⁸ and several studies in HIV-infected patients have shown high rates of clindamycin-resistant CA-MRSA isolates causing skin and soft tissue infection^{38,44,52,60}. Inducible clindamycin resistance has to be ruled out by performing a D-test⁹⁹.

Tetracyclines have also been used to treat MRSA infections⁹⁷. Recent studies, however, have found increasing resistance to tetracyclines^{22,98}.

The newer drug linezolid is a bacteriostatic agent that can be given orally and is approved for treating MRSA skin and soft tissue infections.

The choice of empirical therapy in the management of CA-MRSA skin and soft tissue infections depends on the severity, and clinicians must distinguish between uncomplicated and severe or complicated skin and soft tissue infection. Drainage has been described as the most important outcome factor in the treatment of skin and soft tissue infection caused by CA-MRSA, and antimicrobial therapy may not be necessary for small, uncomplicated skin abscesses with no signs of systemic infection¹⁰⁰⁻¹⁰². In other cases, particularly when there is invasive or systemic disease, other comorbidities, or immunosuppression, antibiotic use is recommended.

Treatment of severe and invasive infections

Vancomycin has been considered the treatment of choice in severe CA-MRSA infection requiring intravenous therapy. However, new drugs, such as daptomycin, linezolid, or tigecycline may also be good options for some patients, particularly those with severe presentations¹⁰³.

Daptomycin is a bactericidal drug approved for treatment of *S. aureus*, including MRSA skin and soft tissue infection bacteremia and right-sided endocarditis. Daptomycin has shown similar efficacy to vancomycin in treatment of patients with complicated skin and soft tissue infections due to MRSA and patients with *S. aureus* bacteremia^{104,105}. However, daptomycin should not be used for the treatment of MRSA pneumonia because the drug is inactivated by pulmonary surfactant causing clinical failures¹⁰⁶.

Linezolid can be administered either orally or intravenously to patients with MRSA infections. Linezolid

has shown in a randomized trial similar cure rates as vancomycin in patients with presumed MRSA infections including skin and soft tissue infections and pneumonia¹⁰⁷. However, its efficacy in treatment of MRSA bacteremia has not been well established. Thus, linezolid is approved for treatment of MRSA skin and soft tissue infections and pneumonia, but it is not approved for bacteremia and endocarditis.

Tigecycline is another of the new agents that can be used for parenteral treatment of MRSA skin and soft tissue infection¹⁰⁸.

In general, in cases of bacteremia or severe disease with sepsis and multiorgan failure, daptomycin is usually indicated, except for respiratory tract infection, in which linezolid is preferred. However, few comparative studies evaluating these therapies are available and no studies have been focused specifically on HIV-infected patients.

Given the association between toxin-producing CA-MRSA and severe infection, antimicrobial agents that inhibit PVL production could prove beneficial in the treatment of these infections¹⁰⁹. Combined therapy including antibiotics interfering with bacterial protein synthesis, such as clindamycin or linezolid, may be particularly valuable for severe infection when viable organisms are expected for several days^{109,110}.

The currently available agents for the treatment of CA-MRSA infection are summarized in table 3.

Surveillance and prevention

Although the incidence of CA-MRSA infection is higher in HIV-infected patients than the general population, there are no specific guidelines regarding surveillance and management of asymptomatic carriers. Nevertheless, physicians attending HIV-infected patients should routinely ask about skin lesions and be aware of the possibility of CA-MRSA acquisition, especially when infection presents as an abscess, is recurrent, and when similar lesions are present in close contacts.

There is no evidence supporting an indication for treatment with nasal mupirocin or skin antiseptics such as chlorhexidine to decontaminate HIV-infected patients with skin and soft tissue infections caused by CA-MRSA. However, decontamination may be indicated in recurrent skin and soft tissue infection or when transmission from a known contact is well documented. When control of CA-MRSA transmission within a cohort is attempted, it is unclear whether all members or only CA-MRSA nasal carriers should be treated. In any

Table 3. Treatment of community-acquired methicillin-resistant *Staphylococcus aureus* infections

Agent	Indications	Dose
TMP-SMX	SSTI*	160/800 mg po BID
Clindamycin	SSTI*	300 mg po TID
Doxycycline or minocycline	SSTI*	100 mg po BID
Ciprofloxacin or levofloxacin	SSTI*	500 mg po BID (ciprofloxacin) or 750 mg po every 24 h (levofloxacin)
Vancomycin	Complicated SSTI Pneumonia Bacteremia Invasive infections	1 g IV every 12 h
Linezolid	Complicated SSTI Pneumonia Invasive infections	600 mg po or IV BID
Daptomycin	Complicated SSTI Bacteremia Invasive infections	6 mg/kg IV every 24 h†
Tigecycline	Complicated SSTI Pneumonia Bacteremia Invasive infections	Loading dose 100 mg IV followed by 50 mg IV every 12 h

CA-MRSA: community acquired methicillin resistant *Staphylococcus aureus*; SSTI: skin and soft tissue infections; TMP-SMX: trimethoprim-sulfamethoxazole; po: orally; IV: intravenously; BID: twice daily; TID: thrice daily.

*Complicated abscesses and osteomyelitis: rifampin 600 mg po every 24 hours can be associated; †Experts suggest higher doses for bacteremia.

case, it is important that decontamination be performed simultaneously in all members to avoid antimicrobial resistance. The use of systemic antibiotics is reserved for patients with active skin and soft tissue infection¹¹¹.

Recommendations to prevent CA-MRSA transmission to other contacts in HIV-infected patients do not differ from those used in other risk populations. Usually, CA-MRSA is transmitted through skin-to-skin contact with a person colonized or infected by CA-MRSA, or by contact with contaminated objects or surfaces. Survival of MRSA in an inanimate environment can be long, and its presence in environments close to patients with active skin and soft tissue infection has been described. The recommendations to prevent person-to-person transmission are summarized in table 4¹¹².

Conclusions

HIV-infected patients have a higher risk of acquiring CA-MRSA infection compared with HIV-negative individuals. Other risk conditions associated with CA-MRSA acquisition may also be prevalent in certain subgroups of HIV patients. The incidence of CA-MRSA

Table 4. Recommendations to prevent person-to-person transmission of community-acquired methicillin-resistant *Staphylococcus aureus*¹¹²

- Keep wounds covered with clean dry bandages.
- Clean hands with soap and water or alcohol-based gel. Clean hands immediately after contact with infected skin.
- Maintain good general hygiene.
- Do not share items that may become contaminated such as towels, clothing, bar soap, razors.
- Launder clothing that has come in contact with wound drainage after each use.
- Clean equipment and other environmental surfaces with which multiple individuals have skin contact with an over-the-counter detergent/disinfectant active against *Staphylococcus aureus*.
- In a gym, steam room, or locker room use a clean towel when sitting on benches.
- Avoid direct contact with other people's wounds or bandages; wash hands thoroughly with soap after any such contact.

Adapted from Gorwitz, et al.

infection, particularly skin and soft tissue infection, has significantly increased in the HIV-infected population over the last years. Therefore, this pathogen should be taken into account in the choice of appropriate treatment in HIV patients with serious community-acquired infection in which *S. aureus* is suspected.

Although CA-MRSA infection has been reported independently of immune status or HAART use, the risk appears to be higher in severely immunosuppressed patients.

Currently, there are no specific recommendations for prevention of CA-MRSA transmission in the HIV-infected population, but these patients are commonly exposed to the healthcare environment; thus, measures to prevent in-hospital CA-MRSA spread should be implemented.

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