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MRSA broadens its reach

Communities, not just hospitals, are breeding grounds for staphylococcal infections.

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Methicillin-resistant *Staphylococcus aureus* (MRSA) is a common hospital- or healthcare-acquired pathogen that has presented prevention and treatment challenges for healthcare providers for well over 40 years. Historically, MRSA was found almost exclusively in patients with certain risk factors: hospitalization, invasive procedures or devices, or chronic conditions such as kidney failure. But just as hospital clones of *S. aureus* adapted to their environment by developing resistance factors to commonly used antibiotics, the character of community strains of *S. aureus* has also evolved. In the mid-1990s, healthcare providers began seeing MRSA in patients with no identifiable risk factors. Otherwise healthy children and adults were treated, and sometimes hospitalized, with infections where MRSA was identified as the offending pathogen.[1]

The "new" MRSA was clearly different from the hospital- or healthcare-acquired MRSA (HA-MRSA) and, with time, earned its distinction as "community-acquired MRSA" (CA-MRSA). The differences between the community-acquired infection and the hospital variety are quite fascinating, and sometimes frightening, as cases that go undetected can have dire consequences. With MRSA on the rise, the clinical management of patients with *S. aureus* infections must adapt in both communities and hospitals.

HA-MRSA vs. CA-MRSA

S. aureus is a common bacterial pathogen that can produce a variety of diseases from superficial skin infections, such as boils and cellulitis, to invasive life-threatening conditions, including sepsis and endocarditis.[2] Methicillin was introduced in the late 1950s to treat staphylococcal infections, and it wasn't long before the first MRSA bacterium was described (1961) in a hospitalized patient in the United Kingdom. From that time, the incidence of MRSA rose steadily, and by the 1980s MRSA was endemic in U.S. hospitals. Today, the prevalence of MRSA in hospitals ranges from 40% to 70%, depending upon geographical location—it is the most common antibiotic-resistant pathogen that causes hospital-acquired infections.[3] HA-MRSA can best be described as multi-resistant to antibiotics and is associated with risk factors that include history of hospitalization, surgery and dialysis; residence in a long-term-care facility; and presence or history of an indwelling device, such as a urinary catheter, gastrostomy tube, or tracheostomy tube.

When healthcare providers first detected MRSA in patients lacking the usual healthcare-associated risk factors (in the mid- 1990s), they believed it was simply an HA-MRSA clone from the hospital environment that had invaded the community. Further microbiologic and genetic inspection revealed that community strains are quite different from hospital clones. The conclusion from extensive genetic studies is that there are clearly two types of organisms.[4] Where HA-MRSA is resistant to most classes of antibiotics and seen in patients with identifiable risk factors associated with healthcare environments, CA-MRSA is seen in otherwise healthy patients and, although more sensitive to antibiotics, can be more virulent and invasive.[5]

Like methicillin-susceptible *S. aureus*, CA-MRSA causes a spectrum of diseases, of which skin and soft-tissue infections (SSTIs) are the most frequently encountered.[6] The most commonly reported SSTIs are furuncles (abscessed hair follicles or boils), carbuncles, and abscesses. Infected skin lesions are often mistaken for spider bites by both patients and healthcare providers because of their raised, red, and necrotic appearance. Most SSTIs can be treated successfully in the outpatient setting, but severe, deep soft-tissue abscesses may require hospitalization and treatment with parenteral antibiotics, surgical incision, and drainage.

More severe infections from CA-MRSA do occur, including necrotizing fasciitis, necrotizing pneumonia and empyema, sepsis syndrome, osteomyelitis, bac-

How do patients acquire CA-MRSA?

Because the epidemiology of CA-MRSA is still being rigorously investigated, risk factors for the acquisition of CA-MRSA aren't yet well defined, but may include:

- high prevalence of MRSA in the local community
- history of MRSA infection or colonization
- close contact with someone known to be infected or colonized with MRSA
- crowded living conditions (homeless shelters, military barracks)
- incarceration
- participation in competitive sports where skin injury, close contact, and sharing of equipment and personal items occurs
- skin or soft-tissue infection not responsive to beta-lactam antibiotics
- children under 2 years of age
- males with a history of having sex with other men
- shaving of body hair.

Source: Los Angeles County Department of Health Services. Guidelines for Reducing the Spread of Staph/CAMRSA in Non-Healthcare Settings. September 2004. Available at: http://lapublichealth. org/acd/docs/MRSA/MRSA_Guideline_12_20_04.pdf. Accessed December 1, 2007.

teremia, pyomyositis, purpura fulminans, and disseminated infections with septic emboli. In most cases, severe disease is a result from complications of a previous SSTI or viral respiratory infection; however, healthy individuals with no history of prior infections or risk factors do suffer from life-threatening illnesses. (See "How do patients acquire CA-MRSA?")

Almost a decade ago, an article reported the deaths of four pediatric patients in Minnesota and North Dakota from necrotizing pneumonia caused by CA-MRSA.[7] Although the presence of CA-MRSA strains was well documented by 1999, this report placed emphasis on the potential severity of community strains of MRSA and how they were genetically different from hospital strains. The overriding message: the emergence of this pathogen shouldn't be taken lightly.

CA-MRSA has distinct differences in terms of resistance and virulence and, despite being more sensitive to antibiotics, the organisms appear to spread with more ease and cause more skin infections. One way that researchers are able to differentiate CA-MRSA from HA-MRSA is based on the type of mobile genetic element, known as the staphylococcal chromosomal cassette *mec* (SCC*mec*), that confers resistance to different antibiotic classes.[3] There are several identifiable types of SCC*mec* elements—types I, II, III, and V are large in size and found in HA-MRSA; type IV is found in CA-MRSA and is much smaller than the other SCC*mec* types, which may explain why community strains are susceptible to more classes of antibiotics and transfer more easily between *S. aureus* strains.

In addition to the SCC*mec* type IV element, almost all strains of CA-MRSA have the Panton-Valentine Leukocidin (PVL) gene that accounts for its virulence and invasiveness. The PVL gene, rarely found in HA-MRSA isolates, allows for the production of a cytotoxin that causes tissue necrosis and renders neutrophils ineffective.

The presence of the PVL gene has been identified in strains of CA-MRSA associated with primary skin infections, severe necrotizing pneumonia, and osteomyelitis. The exact role that PVL plays in the disease process is still being investigated.

Identifying CA-MRSA

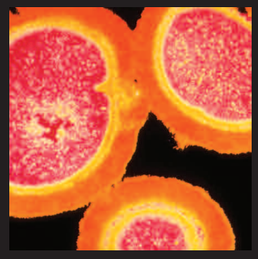
The increased presence of MRSA in the community, most importantly, requires a shift in the approach to the management of suspected or confirmed staphylococcal infections. For SSTIs such as furuncles and abscesses that are compatible with *S. aureus* infection, MRSA needs to be considered. Complaint of a spider bite should also increase the suspicion for MRSA.

Material for culture and antimicrobial susceptibility testing should be obtained whenever possible, and is strongly recommended for patients with abscesses and purulent skin lesions, with signs and symptoms of systemic infection and in association with a suspected outbreak or cluster. Susceptibility results are important to determine the usefulness of beta-lactam antibiotics and verify that antimicrobial therapy is well matched with the infecting organism. Susceptibility testing should include a "D-zone" test for clindamycin resistance in MRSA isolates, as some areas in the United States have CA-MRSA isolates resistant to clindamycin.

Treatment

Initial empirical treatment needs to be based on: the prevalence of MRSA in the local community, the presence or absence of risk factors for HA-MRSA, and the severity and type of infection.[8] The incidence of MRSA can vary geographically. For mild cases of SSTI, incision, drainage, and wound care (without oral antimicrobials) are sufficient. The decision to include antimicrobial therapy is based on: the severity and aggressiveness of the SSTI, signs and symptoms of systemic infection, patient comorbidities, extremes of patient age, lack of improvement to initial treatment with incision, and difficulty with drainage and location of the abscess (such as association with septic phlebitis or major vessels).

Initial therapy with a beta-lactam antibiotic may be adequate if MRSA isn't suspected.[9] Some experts suggest that empiric therapy should be changed from a beta-lactam antibiotic if greater than 10% to 15% of the community *S. aureus* isolates are MRSA. Alternatives to beta-lactams for outpatient treatments of SSTIs, when MRSA is suspected, are trimethoprim-sulfamethoxazole (TMP-SMX), tetracyclines (minocycline or doxycycline), clindamycin, rifampin (in combination with other agents), and linezolid. Fluoroquinolones (ciprofloxacin and levofloxacin) and macrolides (erythromycin, clarithromycin, and azithromycin) shouldn't be selected (because resistance has already been established) or widespread use will potentially lead to the rapid development of resistance to these agents. In all cases, patients must be monitored closely for their responses to the selected therapies, and antimicrobials may be adjusted based on culture and susceptibility results.



Emerging mupirocin resistance is also a concern that lends caution to the widespread use of the antibiotic.

One notable drawback of clindamycin use—and for that matter, antibiotics in general—is the risk for *Clostridium difficile*-associated disease (CDAD). Like MRSA, CDAD is considered to be a healthcare-associated infection, and risk factors include prior antibiotic use and hospitalization. Similarly, children and adults without prior healthcare association are developing CDAD in their communities. Antibiotic use needs to be based on necessity to prevent the development of resistance and other adverse effects.

Patients need to be instructed to return if: no improvement is seen within 48 hours, signs and symptoms of systemic infection develop, or local symptoms worsen. If at all possible, a follow-up visit within 48 hours should be made to assess the progress and response of treatment.

Although rare, patients' SSTIs sometimes progress into life-threatening, invasive processes such as necrotizing fasciitis, necrotizing pneumonia, or sepsis.[10] In these cases, hospitalization is indicated. Treatment—with empiric, broad-spectrum, parenteral antibiotics that are active against MRSA (such as vancomycin)—is required. Antibiotics are similarly tailored based on identification and susceptibility results.

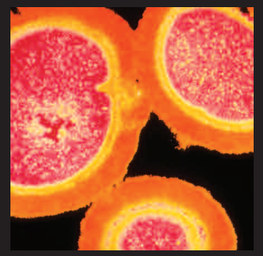
MRSA colonization

Patients infected with MRSA often become colonized with the organism after their infections have been treated and resolved. Patients colonized with MRSA continue to harbor the organism as part of their "normal" flora, but its presence doesn't disrupt any of the body's functions. Although colonizing organisms can often exist in harmony with healthy body systems, the potential impact of a colonized individual is fully highlighted in the case of MRSA. MRSA-colonized patients have a greater chance of developing infection in the future and also serve as sources for cross-transmission of the organism to other patients, healthcare personnel, family members, and others in close contact.[1] If indeed the organism is passed on to another individual, he or she may also become colonized and, in turn, has a greater chance of developing infection and/or may serve as a potential carrier and source for further cross-transmission. The impact of colonization is compounded as the organism is passed from person to person, oftentimes left undetected and unrecognized until disease develops.

Patients most frequently become colonized with MRSA in their nares. Because colonization poses a greater risk for development of repeated infections, decolonization may be considered to prevent future infections.[8] In some instances, applying a course of topical mupirocin (Bactroban) to the anterior nares may reduce nasal colonization by CA-MRSA. Decolonization may be considered in extreme cases for patients with recurrent infections or in circumstances in which there is evidence of ongoing transmission in a defined cohort, such as a household or a hospital unit. Methods to eradicate MRSA colonization using a combination of topical mupirocin alone, or in combination with oral antibiotics and antiseptic body wash, have proven successful. These regimens are sometimes prescribed for patients colonized with MRSA prior to undergoing cardiovascular or orthopedic surgery where MRSA surgical-site infections can be devastating.

With the lack of hard evidence that decolonization measures are effective in the long term, routine decolonization of all patients with MRSA is not recommended. There are several limiting factors to decolonization practices that dampen the long-term success of the therapy.

In addition to the need for follow-up cultures to ensure eradication of MRSA, there is no guarantee that an individual won't become recolonized with the same, or another, strain. Emerging mupirocin resistance is also a concern that lends caution to the widespread use of the antibiotic. Decolonization should not be attempted until standard prevention measures have been reinforced and deemed unsuccessful.



Controlling MRSA requires a combination of strategies.

Spreading CA-MRSA

In certain circumstances, healthcare personnel may be implicated in the transmission of MRSA. Treatment with mupirocin is indicated if they are linked epidemiologically to transmission. Healthcare personnel colonized with MRSA, but not symptomatic or linked epidemiologically to an outbreak or transmission, don't require decolonization.[9]

CA-MRSA is transmitted primarily by contact with an infected or colonized individual or items contaminated by an infected person. An all-too-familiar scenario in hospitals can also play out in communities—hands come in contact with individuals, environmental surfaces or items that are infected, colonized or otherwise contaminated with MRSA. If the hands are not washed or decontaminated after contact, they become very effective vehicles of transmission. Other factors for community transmission include skin-to-skin contact, crowded conditions, and poor hygiene.

CA-MRSA has become notorious for causing outbreaks of suppurative skin infections among members of groups such as sports teams, prison inmates, military recruits, day care attendees, homosexual men, and injection-drug users. A closer look at outbreaks among athletes—from college and high school football players and wrestlers to members of fencing clubs—illustrates some of the factors that contribute to person-to-person transmission.[11]

The first is preexisting skin damage. Athletes often receive skin trauma and abrasions through which skin pathogens can easily gain entry. In addition to direct trauma, protective clothing can be hot and cause skin chafing that results in open wounds and lacerations. Players have also reported that they aren't always diligent with covering open skin abrasions. The second is direct contact with another individual's skin. MRSA can easily be transmitted through the kind of direct skin-to-skin contact characteristic of wrestling and football. The third factor is the use of shared equipment and other personal items (such as, balms and lubricants) that are not cleaned between player use.

Even though CA-MRSA isolates have their roots firmly planted in the community, and are easily distinguishable from HA-MRSA, they make their presence known in the hospital setting as well. Patients with more severe cases of CA-MRSA need to be hospitalized—introducing the strains into the hospital environment. The risk of transmission clearly exists in hospitals as there have been several documented outbreaks of CA-MRSA in the inpatient setting.[12] Therefore, the same respect for HA-MRSA must be assigned to CA-MRSA in hospitalized patients.

The fight against CA-MRSA

Historically, the practices employed to prevent and control MRSA centered on scrupulous compliance with hand hygiene, isolation, and environmental de-contamination. The success of these measures has been somewhat dismal, and more aggressive techniques to control—and in many cases, eliminate—MRSA have emerged in the wake of continued outbreaks and ongoing evidence of cross-transmission in hospital environments.[13] Although experts haven't reached a consensus, and studies to evaluate the effectiveness of additional measures continue, researchers commonly agree that controlling MRSA requires a combination of strategies.

As a starting point, standard precautions and contact precautions are recommended for all patients with open or draining wounds that aren't contained within bandages, or for patients with suspected or known infections of MRSA (community- or hospital-acquired). Patients should be placed in private rooms or grouped with cohorts. Gloves and gowns should be used for all contact with patients or their environments, and removed prior to exiting the rooms. After wound care and dressing changes, gloves should be changed before going on to other tasks. Hand hygiene should be done before and after gloving. Surgical masks, although not routinely needed, may be indicated in certain circumstances, such as during the suctioning of respiratory secretions identified with MRSA. All environmental surfaces and equipment should be considered potentially contaminated and routinely cleaned with an approved disinfectant. Non-critical patient-care equipment should be dedicated to patients' rooms.

In the hospital setting, control of CA-MRSA may benefit from the more aggressive strategies currently being employed to reduce HA-MRSA. Because persons colonized are at greater risk for developing infection and serve as a reservoir for potential transmission, some experts believe that identification and isolation of infected and colonized patients is paramount to reducing the incidence of MRSA. Many organizations have initiated protocols to collect active surveillance cultures (ASC) from patients on admission to hospitals or to certain units. This provides an opportunity to identify potential carriers of MRSA and utilize isolation and/or barrier precautions to prevent cross-contamination.

Screening programs consume vital resources, and therefore, most are targeted at high-risk populations and settings. High-risk populations may include transfers from long-term-care facilities, patients with a history of MRSA infection, and patients with kidney disease. High-risk settings may include intensive care, burn, bone marrow/stem cell transplant and oncology units. Sampling the anterior nares is generally sufficient for ASC. To increase yield, samples from the throat, endotracheal tube aspirate, percutaneous gastrostomy sites and perirectal or perineal area may be considered. Patients are then placed in isolation, or full-barrier precautions (gowns and gloves for every contact) are used, until testing results are received. Repeat cultures to determine a change in colonization status is often indicated.

The Healthcare Infection Control Practices Advisory Committee (HICPAC) released a landmark publication in 2006 on "Management of Multidrug-Resistant Organisms in Healthcare Settings, 2006." The document contains abundant information on the prevention and control of MRSA that encompasses administrative support, education, antimicrobial stewardship, surveillance, infection control precautions, environmental measures, and decolonization.

As community and hospital strains of MRSA continue to cross paths in hospital environments, the distinction between them is beginning to blur. It remains to be seen how the presence of different strains in the same environment will affect resistance and virulence in the future.

Prevention strategies

Prevention and control strategies in the community include several key measures.[1] (See "Patient education.") The first is awareness by healthcare providers of the possibility of CA-MRSA in patients with SSTIs and with other more severe illnesses typical of *S. aureus* infection.

The second measure—early detection and appropriate treatment in settings where outbreaks have occurred—is critical. Screening methods for early signs and symptoms of SSTIs should be implemented in correctional facilities, among contact sports teams and in community living settings (homeless shelters, camps, boarding schools, and day cares). Household members and other close contacts of patients infected with MRSA need to be closely monitored.

The third measure for prevention is promoting good hygiene and maintaining a clean environment. Education is necessary to help control transmission of MRSA, especially in crowded conditions, household settings, and

Patient education

Because the spread of CA-MRSA among household members is common, and recurrent infection occurs with some frequency, counseling patients and their families on measures to prevent the spread of infection is prudent. Instruct patients to:

- Follow instructions given to treat the infection, which may include taking antibiotics as prescribed, daily showers with antibacterial soap, and applying antibiotic ointment in the nose for several days.
- Keep wounds and lesions (especially draining wounds) covered with clean, dry bandages. Don't pick or pop lesions or attempt self-lancing of boils.
- Patients should wash their hands after touching infected skin and bandages. Disposable items such as bandages and wipes should be placed in a separate bag and closed tightly before they're thrown out with the regular trash.
- Household members should wash their hands after coming in contact with infected wounds, bodily fluids, or soiled bandages. Consider wearing nonsterile vinyl gloves when changing patient's clothes.
- Change towels, washcloths, clothing, and sleepwear daily. Don't share these personal items with other household members.
- Wash soiled linens and clothes in hot water and laundry detergent. Use a dryer on a "hot" setting.
- Disinfect non-clothing items that come in contact with the wounds, or drainage from the wounds, using a store-bought household disinfectant.
- Avoid participation in contact sports, or other skin-to-skin contact, until the infection has healed.
- Inform other healthcare providers who treat you that you have a "resistant staph infection."

Source: Siegel JD, Rhinehart E, Jackson M, Chiarello L, Healthcare Infection Control Practices Advisory Committee. Management of Multidrug-Resistant Organisms in Healthcare Settings, 2006. Available at: http://www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf. Accessed December 4, 2007.

places where sharing of personal items and equipment is routine.

There are many unanswered questions about the epidemiology and pathophysiology of CA-MRSA that warrant further study.[14] It is clear, however, that MRSA is no longer strictly a hospital- or healthcare-acquired microorganism. M

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