



Course Objectives

The purpose of this program is to provide nurses with up-to-date information on a virulent new strain of methicillin-resistant *Staphylococcus aureus* (MRSA) appearing in our communities. After studying the information presented here, you will be able to —

- Identify three conditions associated with transmission of community-acquired MRSA.
- Discuss three important differences between community-acquired MRSA and hospital-acquired MRSA.
- Describe three important community-acquired MRSA prevention strategies.

At first, there didn't seem to be any cause for alarm when a young, healthy Royal Marine recruit in England scratched his leg during training. But his leg quickly became infected with a new, virulent strain of methicillin-resistant *Staphylococcus aureus* (MRSA). The 18-year-old died two days later with necrotizing pneumonia caused by the new MRSA strain.¹

During the mid-1990s, a more virulent and potentially lethal strain of MRSA emerged in the community among healthy people with no ties (direct or indirect) to health care. This new strain, named community-acquired MRSA (CA-MRSA), is genetically distinct and unrelated to the MRSA strains found in hospitals.

CA-MRSA carries a particularly aggressive, leukocyte-killing toxin called Panton-Valentine leukocidin (PVL)

www.cdc.gov/ncidod/EID/vol10no1/03-0144.htm that increases its ability to infect skin

http://www.msjensen.gen.umn.edu/webanatomy/wa_cell_chem/wa/_wcb_skin1.htm and soft tissues in otherwise healthy young people. It has caused numerous community outbreaks of aggressive skin and soft-tissue infections, including pustular lesions, furuncles (boils), carbuncles, abscesses, and cellulitis. The skin infections are frequently misdiagnosed as brown recluse spider bites since they seem to appear spontaneously, often without an antecedent skin break.^{2,3} In addition to skin infections, CA-MRSA causes a severe necrotizing pneumonia that can kill within 24 hours of onset, as well as necrotizing fasciitis, osteomyelitis, endocarditis, and a rapidly fatal sepsis syndrome. Nurses in all settings need to know about this new, potentially deadly strain of MRSA so that they can be on the alert for signs and symptoms in their patients.

Notorious *S. aureus*

S. aureus http://www.cdc.gov/ncidod/hip/aresist/ca_mrsa_public.htm is a well-known and notoriously successful human pathogen. For centuries it has been one of the major bacterial causes of human suffering and death throughout the world. In the preantibiotic era, invasive infections caused by *S. aureus* were fatal more than 80% of the time. With the introduction of antibiotics, the mortality rate for invasive disease was reduced by more than half and now ranges from 20% to 40%.⁴

Today, *S. aureus* is the leading overall cause of hospital-acquired infections in the United States, the most frequent cause of nosocomial pneumonia and surgical site infections, and the second most frequent cause of bloodstream, cardiovascular, eye, ear, nose, and throat infections.⁵ The pathogen can be found everywhere and is able to survive after drying on environmental surfaces for long periods (days to months) under adverse environmental conditions.⁶ Humans are a natural reservoir for *S. aureus* (including MRSA), and as many as 30% to 50% of adults are colonized with the bacteria. Colonization occurs in the nasopharynx, perineum, or skin. It can be transient, or it can be persistent, and it can last for years.⁷

MRSA was first identified as a hospital-acquired pathogen in the 1960s shortly after methicillin was introduced to replace penicillin. Widespread resistance to the antibiotic developed slowly over 30 years, and it wasn't until the 1990s that resistance rates accelerated. By 2002, in hospital ICUs over 57% of *S. aureus* infections were methicillin-resistant — up from 29% in 1991. Unfortunately, during the 1990s, at the same time that methicillin resistance was increasing among hospital-acquired *S. aureus* infections, a new more virulent strain of MRSA, CA-MRSA, clinically distinct from hospital-acquired MRSA (HA-MRSA), began emerging in communities. Infections caused by the new strain were being seen among young, previously healthy people with no known health care-associated exposures. In addition, the CA-MRSA infections, unlike the more common MRSA infections in hospitals, could be treated with a number of nonbeta-lactam antibiotics, trimethoprim-sulfamethoxazole (Bactrim), minocycline (Minocin), linezolid (Zyvox), or clindamycin (Cleocin).

A frightening debut

The nation first became aware of CA-MRSA in 1999, when the Centers for Disease Control and Prevention's Morbidity and Mortality Weekly Report . <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4832a2.htm> described four pediatric deaths from the pathogen. All four children had died of MRSA infections that were initially treated with beta-lactam antibiotics. One of the deaths was a 16-month-old girl admitted to a hospital

in shock and with a temperature of 105.2 F (40.6 C), seizures, a diffuse petechial rash, and irritability. Although treated with ceftriaxone (Rocephin), she developed respiratory failure and cardiac arrest and died within two hours. MRSA was cultured from blood and cerebrospinal fluid drawn from the child immediately postmortem. An autopsy revealed multiple small abscesses of the brain, heart, liver, and kidneys, and autopsy cultures of meninges, blood, and lung tissue grew MRSA.⁹

Another of the four fatal cases was a 13-year-old girl who was admitted to the hospital with fever, hemoptysis, and respiratory distress. The day before admission she had a productive cough and a 2-cm papule on her lower lip. A chest radiograph revealed a left lower lobe infiltrate and a pleural effusion. She was treated with ceftriaxone and nafcillin (Nallpen, Unipen), but within five hours of arriving at the hospital, she became hypotensive. She was intubated and treated with vancomycin (Vancocin) and cefotaxime (Claforan). However, despite pulmonary and hemodynamic support, her respiratory status continued to deteriorate, and she died of progressive cerebral edema and multiorgan failure. An autopsy revealed consolidated hemorrhagic necrosis of the left lung. Each of the four deaths in the CDC report was attributed to a new lethal strain of MRSA (PVL-positive CA-MRSA) that had recently emerged in the community.⁹

Easy transmission

Infections caused by CA-MRSA are easily transmitted through simple skin-to-skin contact (even without a skin break) with an infected person or by contact with contaminated objects or surfaces. Outbreaks have been reported among athletes (especially football players and wrestlers), military recruits, correctional facility inmates, injection drug users, students, and children in day care centers. The outbreaks have been linked to sharing personal items (soap, towels, razors) and using shared equipment in gyms, health clubs, and spas in cases in which people have had bare skin contact with items contaminated with the bacteria. The most common conditions associated with spread of infections include the five Cs:^{2,10}

- Close, crowded living quarters (e.g., prisons, homeless shelters, military barracks, and college dorms)
- Skin-to-skin contact with an infected person or carrier
- Compromised skin integrity
- Contaminated surfaces
- Suboptimal cleanliness

What to look for

Furunculosis is the most common clinical syndrome that CA-MRSA causes. Furunculosis is a bacterial infection of hair follicles characterized by the development of necrotic lesions of the skin and soft tissues with subsequent destruction of subcutaneous tissue. These lesions can rapidly progress to cellulitis www.mayoclinic.com/invoke.cfm?id=DS00450 or, more commonly, to abscesses. People with extensive lesions often require hospitalization for debridement and IV antibiotics.⁸ Although most patients with CA-MRSA skin and soft-tissue infections are treated successfully, each infected patient has the potential to develop a rapidly progressive, life-threatening invasive infection, such as necrotizing fasciitis <http://www.nnff.org/>, myonecrosis, necrotizing pneumonia, sepsis, or nonmenstrual toxic shock-like syndrome.^{2,11} Bacteremia, acute hematogenous osteomyelitis, pyomyositis (abscesses in the muscle), and mediastinitis have also been reported.⁸

PVL toxin-carrying strains of CA-MRSA can cause a rapidly progressive, hemorrhagic, necrotizing pneumonia with an extremely high mortality rate. The pneumonia is often preceded by influenza or an influenza-like respiratory illness. Patients develop high fevers, dyspnea, hemoptysis, hypotension, leukopenia, and multilobular alveolar infiltrates that, unlike HA-MRSA pneumonias, often progress into abscesses. Invasive infections usually progress rapidly to septic shock and acute respiratory distress syndrome.^{3,8} Death occurs in as many as 75% of cases. Patient autopsies typically show diffuse bilateral necrotic hemorrhagic pneumonia.^{3,8}

Researchers in Los Angeles have identified cases of necrotizing fasciitis caused by monomicrobial (a single-strain) CA-MRSA. Necrotizing fasciitis is a rapidly progressive infection that involves the skin, subcutaneous fat, deep fascia, and occasionally muscle. Early clinical symptoms include skin blisters, edema that extends beyond the area of erythema, focal skin ecchymoses or ischemia, crepitus, and skin numbness. Patients often complain of severe pain.¹¹ Infections are life-threatening and require immediate medical and surgical therapy. Until the recent emergence of CA-MRSA, necrotizing fasciitis was most commonly caused by group A streptococci, *Clostridium perfringens*, or a mixture of anaerobic and aerobic organisms. For this reason, broad-spectrum empirical antimicrobial treatment did not include antibiotics effective against MRSA. Today, however, even though *S. aureus* by itself is an extremely uncommon cause of necrotizing fasciitis, empirical treatment needs to include nonbeta-lactam antibiotics active against CA-MRSA.¹²

Regardless of the type of infection caused by CA-MRSA — furunculosis, necrotizing pneumonia, necrotizing fasciitis, osteomyelitis, etc. — the presence of the PVL genes in the CA-MRSA strain seems to be associated with increased virulence and severity of disease. PVL genes code for the production of toxins that cause cell deaths by producing pores (punch holes) in the cellular membranes of leukocytes. These genes are rarely found in methicillin-sensitive *S. aureus* or in HA-MRSA.^{2,3}

Community acquired vs. hospital acquired CA-MRSA should be considered in the differential diagnosis of all patients who present with skin and soft-tissue infections, as well as more severe illnesses, compatible with *S. aureus*. Infections with CA-MRSA can generally be distinguished from HA-MRSA in a variety of ways.

1. The infections are diagnosed in the outpatient setting or by a culture positive for MRSA within 48 hours of admission to a hospital. Patients diagnosed with CA-MRSA must have:²

- No prior medical history of MRSA infection or colonization
- No history of hospitalization or admission to a nursing home, skilled nursing facility, or hospice during the past year
- No dialysis or surgery (including minor procedures) or indwelling catheters or other percutaneous medical devices in the preceding 12 months

2. CA-MRSA infections can be treated by a number of nonbeta-lactam antibiotics, including clindamycin (Cleocin), trimethoprim-sulfamethoxazole (Bactrim), linezolid (Zyvox), and minocycline (Minocin) while HA-MRSA isolates are often resistant to most other antibiotic classes as well as to the beta-lactam antibiotics.²

3. The differences in antibiogram resistance patterns in CA-MRSA and HA-MRSA can be better understood by looking at bacterial genetics. *S. aureus* was able to develop resistance to all beta-lactam antibiotics (penicillins, monobactams, cephalosporins, and carbapenems) when it acquired the *mecA* gene that encodes for an enzyme called beta-lactamase, which is a penicillin-binding protein. The *mecA* gene allows the organism to grow and divide in the presence of methicillin and other beta-lactam antibiotics. This gene, together with two regulatory genes, is located on a mobile genetic element (a set of genes that can move from one location to another) called a staphylococcal chromosomal cassette (SCCmec).

So far, five distinct types of MRSA chromosomal cassettes, designated as I, II, III, IVa, and IVb, have been described, and the differences between each type help explain the recent emergence and behavior of CA-MRSA.⁶ The first three chromosomal genetic elements (SCCmecA I, II, III) are found in HA-MRSA strains and are larger than SCCmecA IV found in CA-MRSA strains.

The larger chromosomal cassettes are not only able to code for methicillin resistance but also have room to code for resistance to multiple other nonbeta-lactam antibiotics. In contrast, CA-MRSA strains have a Type IV SCCmec. The Type IV SCCmec is one-third smaller than types I, II, and III. This may explain why CA-MRSA strains are resistant only to beta-lactam antibiotics. There may not be enough room on the Type IV chromosomal cassette to code for resistance to other antibiotics.^{2,7}

4. CA-MRSA is further distinguished from HA-MRSA by a number of genes encoding for toxins, such as PVL and 18 other toxin genes not found in other *S. aureus* genomes. PVL causes tissue necrosis and leukocyte destruction. PVL genes are associated with skin and soft-tissue infections and with necrotizing pneumonia and necrotizing fasciitis.^{2,6}

5. CA-MRSA strains grow significantly faster than HA-MRSA strains. This allows CA-MRSA to overgrow other bacterial species that are part of the normal flora. This factor, along with the combination of PVL genes and the Type IV SCCmec, has created a superadaptable *S. aureus* strain that can spread rapidly through the community.²

Fighting CA-MRSA

Patients with mild, uncomplicated skin and soft-tissue infections can be treated with local wound care, including incision and drainage, without the use of antibiotics.¹ Wound specimens should be sent for culture and susceptibility testing. In areas where 10% to 15% or more of skin infections are caused by CA-MRSA, empirical antistaphylococcal beta-lactam treatment, such as dicloxacillin (Dynapen) or cephalexin, is not recommended for skin and soft-tissue infections. In these areas, if antibiotics are used, infections are usually susceptible to trimethoprim-sulfamethoxazole, minocycline, linezolid, or clindamycin.^{2,8} (Note that a significant proportion of CA-MRSA strains have recently been found to be clindamycin resistant.)

For patients seriously ill with infections that may be due to CA-MRSA, empirical antibiotic treatment should include vancomycin. For patients who cannot tolerate vancomycin, other options are iplinezolid, daptomycin (Cubicin), and quinupristin-dalfopristin (Synercid).^{2,7,8}

At present, treatments to eradicate MRSA colonization are not routinely recommended for most people infected or colonized with CA-MRSA.⁸

How to prevent CA-MRSA

CA-MRSA is becoming the predominant pathogen associated with severe invasive skin and soft-tissue infections in many parts of the United States. These infections are easily spread among family members and other close contacts. Consequently, nurses should inform patients of the following special precautions to take to prevent spreading CA-MRSA infections:¹³

- Wash hands and dry with disposable paper towels or air blowers.
- Keep skin lesions (e.g., boils, open sores, wounds, and cuts) covered with clean, dry bandages.
- Limit sharing of personal items, such as towels, washcloths, clothing (including athletic uniforms and protective gear), razors, antiperspirants, and soap.
- Wash soiled clothing and linens in hot water (over 160 F) and laundry detergent. Dry clothing in a hot dryer rather than air-drying it.
- Avoid participating in contact sports or other skin-to-skin contact until skin lesions have healed.
- Use a towel or layer of clothing as a barrier between bare skin and surfaces of shared equipment (e.g., benches, exercise machines, and massage tables).
- Wipe surfaces of shared equipment with disinfectant before and after use, especially if the surface has become wet with sweat. A dilute bleach solution can be an economical disinfectant outside the hospital environment.

- Inform all health care providers who treat you if you have MRSA.

Infections with CA-MRSA are becoming increasingly prevalent among young, otherwise healthy adults and children in the United States. Nurses can minimize the risk of becoming infected with CA-MRSA or of spreading the infection to others by practicing frequent hand hygiene and by following contact precautions when caring for patients with draining wounds. Check your facility's policy on the care of these patients.

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