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Community-Acquired Methicillin-Resistant *Staphylococcus aureus*

An Emerging Problem in the Athletic Population

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Participants of contact sports are at risk for outbreaks of skin and soft tissue infection. Causes of reported outbreaks include *Staphylococcus aureus*, herpes simplex virus, *Streptococcus pyogenes*, and several fungi. Although once thought of solely as a nosocomial pathogen, methicillin-resistant *Staphylococcus aureus* has been identified as an emerging problem in the community, particularly in the athletic population. Despite a recent increase in reported outbreaks of community-acquired methicillin-resistant *Staphylococcus aureus* soft tissue infection in athletic teams, many sports medicine physicians are unfamiliar with the epidemiology of this pathogen. It is spread via person-to-person contact and is harbored within the anterior nares and on the skin of carriers. Outbreaks of community-acquired methicillin-resistant *Staphylococcus aureus* soft tissue infection are not treated by traditional β -lactam antibiotics, and they can be difficult to eradicate. Such infections have been associated with significant morbidity, with up to 70% of involved team members requiring hospitalization and intravenous antibiotics. A thorough understanding of community-acquired methicillin-resistant *Staphylococcus aureus* is essential for the sports medicine physician to properly identify, treat, and control infectious outbreaks.

Keywords: community; methicillin resistant; *Staphylococcus aureus*; abscess; cellulitis

Outbreaks of skin and soft tissue infection among athletes are well documented in the literature, with reported causes including *Staphylococcus aureus*, herpes simplex virus, *Streptococcus pyogenes*, and several fungi.^{1,4,6,12,19,33,43} The majority of these reports involve contact sports, such as wrestling, football, and rugby. Repeated close physical contact and skin injury (ie, turf burns, lacerations) place contact athletes at risk for these infections. *S aureus* is a well-known human pathogen and is one of the most common causes of soft tissue and skin infection. Methicillin-resistant *S aureus* (MRSA) are strains of staphylococcus that are resistant to an array of antibiotics, including the commonly used β -lactam antibiotics and macrolides. Although traditionally viewed as a hospital pathogen, recent reports identify MRSA as an emerging problem in the community.^{8-10,14,17,22} Similar to sensitive strains of

S aureus, the most common manifestations of community-acquired MRSA (CA-MRSA) are skin and soft tissue infections. In contrast to most hospital-acquired strains of MRSA (HA-MRSA), strains of CA-MRSA are often susceptible to non- β -lactam antistaphylococcal antibiotics such as clindamycin and sulfamethoxazole-trimethoprim (SMX-TMP).^{14,26,35}

Several reports of CA-MRSA outbreaks among athletic teams have recently been reported.^{9,26,31,45} These outbreaks have been difficult to eradicate and have been associated with significant morbidity, with up to 70% of involved team members requiring hospitalization and intravenous antibiotics.⁹ Despite a recent increase in awareness of this problem in both the medical literature and the public media, many sports medicine physicians are unfamiliar with this disease. The purpose of this article is to review the epidemiology of CA-MRSA soft tissue infection and to provide the sports medicine physician with an understanding of the diagnosis, treatment, and control of CA-MRSA infection in the athletic population.

S AUREUS

S aureus is a Gram-positive coccus that grows in clusters. It is a well-known human pathogen and is one of the most

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common causes of soft tissue and skin infection. Approximately 30% of healthy, asymptomatic people and as many as 65% of patients with staphylococcal skin infections carry *S aureus* in their anterior nares.²⁸ Transmission is thought to occur through direct person-to-person contact, when open skin wounds are exposed to bacteria from an infected lesion or from the secretions of people who are carriers. Airborne droplets and fomites (ie, inanimate objects that can transmit infectious organisms from one person to another) are not thought to play a significant role in the transmission of *S aureus* infection.^{3,44}

Antibiotics have long been used to treat staphylococcal infections. Shortly after the introduction of penicillin, *S aureus* developed resistance because of the plasma-mediated production of β -lactamase, an enzyme that breaks the β -lactam bond of penicillin. Synthetic penicillins (eg, methicillin, oxacillin, nafcillin) are resistant to the effects of β -lactamase. Strains of *S aureus* that remain sensitive to these synthetic penicillins are commonly referred to as methicillin-sensitive *S aureus* (MSSA).

MSSA outbreaks in high school athletic teams have previously been reported. In 1982, Bartlett et al³ reported an outbreak of *S aureus* furuncles (boils) among a high school football team, which involved 26 players with a total of 55 lesions. The majority of the lesions were located on the extremities (89%) and in areas not usually covered by the football uniform (80%). Sixty-one percent of the affected players reported the development of a furuncle at the site of a previously open wound, and 27% reported the development of a furuncle at the site of a previous bruise. In this series, cultures obtained from the lesions of 2 players grew *S aureus* that was sensitive to nafcillin, clindamycin, erythromycin, cephalosporin, tetracycline, and sulfa and resistant to penicillin and ampicillin. Sharing of equipment and towels and frequency of showering were not found to be risk factors for infection when comparing the affected and unaffected players.

In 1989, Sosin et al⁴⁴ reported an outbreak of furuncles that involved 31 members of the football and basketball teams of a Kentucky high school. A total of 62 lesions were reported among these affected athletes. The basketball season overlapped with the end of the football season, and the 2 teams shared a locker room. Six players participated on both teams. The majority of affected players (71%) were treated with oral antibiotics; 3 players required hospitalization for intravenous antibiotic therapy for infections that did not respond to oral therapy. One of the hospitalized players developed a disseminated *S aureus* infection and a lung abscess. Eighty-one percent of the lesions occurred on the extremities. Players who sustained skin injury (abrasions, cuts) were 3 times more likely to develop infection than were those who did not report skin injury. Use of the school showers and locker room and sharing of clothing and towels were not found to be risk factors for infection. *S aureus* was isolated from 14 of 52 (27%) nasal cultures taken from the athletes, which was not higher in proportion to *S aureus*-positive nasal cultures in a group of student controls. None of the infectious lesions were cultured during the outbreak.

These studies support person-to-person contact as the method of transmission of the disease, with injury to the skin

serving as an entry point for infectious organisms. Nasal carriage of *S aureus* did not appear to be a risk factor for developing infection. Fomites (ie, clothing, towels, equipment) did not contribute to disease transmission in these reports.

METHICILLIN-RESISTANT *S AUREUS*

The MRSA are strains of *S aureus* that have acquired the methicillin-resistant gene *mecA*, which encodes a penicillin-binding protein (PBP2a) with a very low affinity for β -lactam antibiotics.^{21,46} The *mecA* gene is found on mobile genetic units called staphylococcal chromosomal cassettes (SCC*mec*).²⁴ Within the SCC*mec*, the *mecA* gene is positioned next to genetic elements that confer additional antibiotic resistance, yielding the multidrug-resistant strains that inhabit health care settings.²⁵ The MRSA was first recognized in the early 1960s in England.² It then gradually disseminated throughout Europe and reached the United States in the 1970s.^{2,20} It has become prevalent in hospitals worldwide, currently accounting for more than 50% of all nosocomial *S aureus* infections in some areas.^{5,38} Risk factors for nosocomial MRSA infection, including extended hospitalization, hospitalization in the intensive care setting, severe underlying illness, recurrent exposure to antibiotics, and the presence of indwelling devices, are well established in the literature.^{27,32}

Since the 1990s, strains of MRSA different from those found in health care settings have been identified as community pathogens. Although still debated, the majority of CA-MRSA appears to be epidemiologically distinct from HA-MRSA.^{11,23,26,37} In general, these community isolates are resistant to fewer antibiotics than are the nosocomial strains. This decrease in multidrug resistance has been attributed to a novel *mecA* gene that is found in strains of CA-MRSA.^{13,23,34} This *mecA* gene is located on a smaller SCC that is devoid of additional genetic elements that confer multidrug resistance (SCC*mec* type IV).^{13,23,34} Unlike strains of HA-MRSA, some strains of CA-MRSA have been found to carry a gene that encodes Panton-Valentine leukocidin, a leukocyte-killing toxin that has been associated with severe skin and soft tissue infections as well as necrotizing pneumonia.^{17,26,39}

The majority of those affected by CA-MRSA are otherwise healthy people who lack the known risk factors for MRSA acquisition. Similar to MSSA, skin and soft tissue infections are the most common manifestations. Skin and soft tissue involvement can range from a localized cellulitis to a soft tissue abscess (Figure 1). Often, patients present to the treating physician complaining of a skin lesion that they describe as a spider bite.^{10,15} Abscesses can be large, measuring in one series from 5 to 7 cm in diameter.²⁶ Multiple abscesses can occur simultaneously on different areas of the body. More serious CA-MRSA infections, such as septicemia and necrotizing pneumonia, have been reported.^{8,10,17} Although most cases of CA-MRSA infections occur sporadically, the majority of reported cases affecting athletes involve clusters of infections among teammates.

The treatment of CA-MRSA soft tissue infections usually involves drainage of the lesion as well as systemic antibiotic therapy. Cultures of the infectious lesion at the time of



Figure 1. Community-acquired methicillin-resistant *Staphylococcus aureus* abscess on the elbow of a high school football player. The infection occurred at the site of a recent skin abrasion.

drainage are imperative to guide antibiotic therapy. In contrast to most strains of HA-MRSA, CA-MRSA is usually susceptible to clindamycin, SMX-TMP, and fluoroquinolones. Antibiotic susceptibility patterns vary with geographic distribution and are always evolving. Recently, a subset of CA-MRSA that demonstrates inducible clindamycin resistance has been identified.^{18,40,41} These strains of CA-MRSA possess the genetic potential to become resistant to clindamycin during the course of therapy. For this reason, strains that are initially found to be resistant to erythromycin and sensitive to clindamycin should undergo an additional laboratory test (ie, the erythromycin-clindamycin double-disk diffusion test) to differentiate between strains that are fully susceptible to clindamycin and those that have the potential to develop inducible clindamycin resistance.^{29,30} The SMX-TMP may be a better treatment option for those strains of CA-MRSA that demonstrate inducible clindamycin resistance. Because of the evolving patterns of antibiotic resistance, culturing infectious lesions that are suspicious for CA-MRSA and performing antibiotic susceptibility testing are essential to the diagnosis, treatment, and control of CA-MRSA infections.

REPORTS OF CA-MRSA AMONG ATHLETIC TEAMS

Our current understanding of CA-MRSA in athletes is based on several reported outbreaks of CA-MRSA soft tissue infection among athletic teams of various sports, including football, wrestling, rugby, and fencing. The number of team members involved in each outbreak ranges from 2 to 13 and includes athletes who range from the high school to the professional level. Despite the small number of affected subjects in each series, these reports have

shed light on an emerging problem that is increasingly recognized among athletes participating in various sporting activities.

The 2 initial reports of CA-MRSA outbreaks among athletic teams were published in 1998.^{31,45} Stacey et al⁴⁵ reported an outbreak involving 5 rugby players who developed cutaneous lesions that did not respond to β -lactam antibiotics prescribed by the team physician. Over a period of 10 days, the 5 players developed progression of the infection into large soft tissue abscesses in various locations. Cultures from the abscesses of each player grew MRSA, which was resistant to penicillin and methicillin but sensitive to erythromycin, ciprofloxacin, and tetracycline. Only 1 of the 5 had a nasal culture that was positive for MRSA. Additional testing confirmed that all 5 infections were caused by an identical strain of MRSA, which was different from those strains endemic to the local hospitals. The infected players were treated with effective antibiotics and were initially excluded from play. This study did not evaluate risk factors for infection. All 5 players were, however, forward players who were involved in repeated close physical contact during play. The authors recommended, in addition to antibiotic treatment and exclusion from participation of involved players, routine inspection of players for suspicious skin lesions.

Lindenmayer et al³¹ reported an outbreak of CA-MRSA involving a high school wrestling team and the surrounding community. Seven wrestlers from the same team were identified as having soft tissue infections that were positive for MRSA on culturing. Six of 7 were identified by culturing the skin lesions. The remaining wrestler reported having a skin lesion earlier in the season and had an MRSA-positive nasal culture. Five of the 6 available MRSA isolates were found to be of an identical strain on further testing. All isolates were sensitive to ciprofloxacin, clindamycin, and vancomycin. Six of 11 nonwrestlers in the community who were diagnosed with CA-MRSA infection had some connection with the involved high school (ie, students, relatives). Efforts were made to eradicate the outbreak. Wrestlers with known infections were banned from participation and advised to seek treatment by their physicians. Wrestling mats were disinfected twice daily, and wrestlers were advised to shower with antibacterial soap after practices. Routine inspection of wrestlers for suspicious lesions was performed. No CA-MRSA was detected among the team the next season. No variables that were investigated via questionnaire (eg, demographics, wrestling history, use of training room Jacuzzi, sharing of towels or clothing, contact with health care facilities) were identified as risk factors for CA-MRSA infection.

The Centers for Disease Control and Prevention (CDC) has been increasingly involved in the investigation of CA-MRSA outbreaks.⁷⁻¹⁰ In 2003, the CDC reported several clusters of CA-MRSA infection that occurred among athletic teams throughout the United States.⁹ Five cases of CA-MRSA were identified among a fencing team in Colorado (4 team members and 1 household contact). Three of these cases were confirmed by culture, whereas 2 were suspected cases based on clinical presentation during the time of the outbreak. Three patients (60%) required hospitalization for intravenous antibiotics, 1 of whom developed paraspinal myositis and bacteremia and required an 11-day hospital stay. Two

patients reported recurrent cases of infection despite empiric antibiotic treatment. These patients required multiple health care visits before their lesions were cultured and treated with appropriate antibiotics. The CDC also identified several clusters of CA-MRSA infections among high school and college football and wrestling teams. One such outbreak among a Pennsylvania college football team involved 10 players, 70% of whom required hospitalization for intravenous antibiotic therapy. These CA-MRSA infections were identified by culturing the infectious lesions. All isolates obtained from the affected players had identical pulsed-field gel electrophoresis band patterns, indicating that the outbreak was caused by a single strain of CA-MRSA. Two members of a Los Angeles college football team were diagnosed with CA-MRSA soft tissue infections. Both players required hospitalization; 1 player required surgical debridement and skin grafting. The CDC reported that frequent skin injury and inconsistent covering of areas of skin trauma seemed to place athletes at risk for infection.

Recently, Kazakova et al²⁶ reported an outbreak of CA-MRSA soft tissue infections that involved 5 of 58 members of the St Louis Rams professional football team during the 2003 season. All identified infections developed at the site of previous skin injury, and all players had abscesses that required drainage. Risk of infection was associated with a lineman or linebacker position and a higher body mass index. As part of the study, these authors performed surveillance nasal swabs of the players and staff members. Of 84 nasal cultures obtained, none grew MRSA, and 42% grew MSSA. Cultures taken from shared environmental sources, such as surfaces in the weight room, towels, and water from the whirlpools, did not grow MRSA. However, MSSA cultured from the environmental samples were identical to the strains of MSSA found in the nasal cultures, suggesting a potential environmental role in transmission. All isolates of CA-MRSA cultured from infectious lesions had identical antibiotic susceptibility patterns including susceptibility to ciprofloxacin, clindamycin, tetracycline, SMX-TMP, and vancomycin. All isolates contained the gene that encodes Pantone-Valentine leukocidin as well as the *SCCmec* type VI genetic unit. All isolates had identical band patterns on testing with pulsed-field gel electrophoresis, suggesting the presence of a single clone of CA-MRSA among the team. This pattern was also identical to several isolates of CA-MRSA obtained during various cases and outbreaks of CA-MRSA infection throughout the United States, including an infection in a member of a professional football team that occurred after a game with the St Louis Rams. The strain of CA-MRSA responsible for this outbreak was different from MRSA isolates known to inhabit health care settings.

In 2005, we retrospectively reviewed our experience with the largest reported outbreak of CA-MRSA among an athletic team to date in the English literature.⁴² This outbreak involved 13 members of a high school football team who had 20 episodes of infection during the 2003 season. The majority of infections occurred on the extremities (ie, arm, elbow, knee, and leg), areas of the body that are uncovered during play and most susceptible to skin injury. Eighteen of 20 affected players had abscesses that required drainage.

Playing a lineman position carried a 4-fold likelihood of infection, suggesting that the repetitive, close contact inherent to this position predisposes to infection with CA-MRSA. Sharing of equipment and towels, hygiene behavior (ie, washing equipment, showering habits, use of antibacterial soap), and exposure to health care facilities were not associated with infection. Nasal cultures were performed on all players and staff members. Three of 102 (2.9%) nasal cultures had a positive result for MRSA, and 32 of 102 (31.4%) had a positive result for MSSA. Of the players who developed CA-MRSA infection, 8 had negative surveillance nasal culture results, 3 had nasal cultures that were positive for MSSA, and 2 had nasal cultures that were positive for MRSA. Therefore, nasal cultures were not thought to be helpful in predicting infection. Eleven isolates available for antibiotic susceptibility testing had identical patterns, including susceptibility to clindamycin, SMX-TMP, ciprofloxacin, rifampin, and vancomycin and resistance to erythromycin, ampicillin, oxacillin, and cefazolin. None of the isolates demonstrated inducible clindamycin resistance on double-disk diffusion testing. The CA-MRSA isolates from 2 nasal cultures and 4 wound cultures were available for field inversion gel electrophoresis testing and showed identical band patterns, suggesting that a single clone of CA-MRSA was responsible for this outbreak.

During the outbreak that we reviewed, treatment of infected players varied throughout the season depending on the treating physician. Eight of 20 infectious episodes were treated with drainage and empiric β -lactam antibiotics. Seven of 8 of these infections demonstrated an initial healing response to this treatment. These players were, however, much more likely (odds ratio, 33.0; 95% confidence interval, 2.5-443.6) to develop a recurrent infection compared with those either treated with antibiotics guided by cultures taken from infectious lesions or treated empirically with antibiotics known to be effective against CA-MRSA (ie, clindamycin or SMX-TMP). Four players required hospitalization for intravenous antibiotics with a mean length of hospitalization of 4 days (range, 3-5 days).

Mupirocin has been reported to be effective in clearing nasal carriage of HA-MRSA.^{16,36} The strain of CA-MRSA responsible for this outbreak was found to be sensitive to mupirocin. Therefore, mupirocin nasal ointment was used in an attempt to control the outbreak. Initially, this treatment was prescribed only for those who had nasal cultures positive for MRSA. After the realization that players with negative nasal culture results were developing infection, mupirocin nasal ointment was prescribed for the entire team. Only 39% of players, however, reported using the mupirocin nasal ointment as directed. Those players who were compliant did not use the ointment simultaneously. Reasons for noncompliance included cost, inability or unwillingness to fill the prescription, and the discomfort associated with administration of the ointment. Six of the 13 (46.2%) affected players had reported appropriate use of mupirocin nasal ointment before developing the infection. The continued occurrence of CA-MRSA infections after this intervention suggested that the administration of mupirocin in this fashion was ineffective in controlling such outbreaks. It is possible that mupirocin would be

more effective if it were used by all players simultaneously in conjunction with the treatment of infected players with systemic antibiotics. Additional studies are needed to determine the efficacy of mupirocin nasal ointment in treating CA-MRSA infectious outbreaks.

On the basis of these reports, several conclusions can be drawn and recommendations made. It is clear that new strains of MRSA are emerging in the community. These strains of CA-MRSA are different from HA-MRSA in that they exhibit less multidrug resistance, they affect previously healthy people with no history of exposure to health care settings, and they primarily cause skin and soft tissue infection. It is clear that there is significant potential for CA-MRSA to cause outbreaks of soft tissue infection among athletic teams. These outbreaks can lead to significant morbidity and can be difficult to control. Given this understanding, those involved with the care of athletes (ie, athletic trainers and physicians) should be aware of CA-MRSA.

Although *S aureus* is known to inhabit the anterior nares of carriers of the bacteria, the nasal carriage of CA-MRSA during an outbreak is variable, with most reports finding few to no positive nasal culture results in the involved population. It is well documented that infected players can have nasal cultures that show no CA-MRSA growth. Therefore, nasal cultures of athletes during an outbreak cannot be endorsed as a means of predicting infection.

The most commonly reported risk factor for the development of CA-MRSA infection is injury to the skin. The majority of infections occur in areas of the body most susceptible to skin trauma. Furthermore, the above reports suggest that players involved in frequent, repetitive contact (ie, linemen and linebackers in football, forward players in rugby) are most susceptible to developing infection. These findings support the belief that transmission occurs from direct person-to-person contact; bacteria from the skin lesions of infected players is transmitted to the skin of other players during contact. Areas of skin injury facilitate entry of the pathogen. To minimize this person-to-person spread, those areas of the body most susceptible to skin injury, particularly in players involved in frequent, close contact, should be adequately protected. When injuries to the skin are recognized, they should be kept clean and should be covered. Furthermore, areas of skin infection should be completely covered with clean dressings during all forms of sports participation until the lesions are healed.

The majority of reports did not find that environmental sources (ie, towels, equipment, shared personal items, whirlpools) or hygiene behavior (ie, showering habits, use of antimicrobial soap) played a significant role in the transmission of disease. Nonetheless, the potential for spread of CA-MRSA through these sources exists, and guidelines for preventing the transmission of skin infections among athletes should be followed. Good hygiene among team members, including routine hand washing and showering, should be encouraged by trainers, team physicians, and coaches. Clean facilities and showers, as well as an adequate supply of antibacterial soap and towels, should be available to players. Players should be instructed to avoid sharing equipment, towels, clothes, and other personal items and to report any suspicious skin lesions to the athletic trainer. Additional

studies are needed to further characterize the role of environmental sources in the transmission of CA-MRSA.

When skin lesions are identified, they should be drained and cultured by the treating physician. It is evident from the above reports that players with CA-MRSA infection who are treated empirically with β -lactam antibiotics are susceptible to severe and recurrent infections. Therefore, antibiotic susceptibilities should be performed on all cultures. This will help identify cases of CA-MRSA infection and will allow for appropriate, culture-guided antibiotic treatment. Most strains of CA-MRSA reported in the literature are sensitive to clindamycin and SMX-TMP. Because of changing patterns of antibiotic resistance, however, it is recommended that cultures with antibiotic susceptibility testing be performed and used to guide treatment when possible. Laboratories should test for inducible resistance to clindamycin (ie, the erythromycin-clindamycin double-disk diffusion test) if antibiotic susceptibility testing reveals the microbe to be resistant to erythromycin, as this has been increasingly found with CA-MRSA. The role of mupirocin nasal ointment in the control of CA-MRSA outbreaks remains unclear. Although mupirocin has been reported to be effective in clearing nasal carriage of HA-MRSA, information regarding the use of mupirocin during outbreaks of CA-MRSA infection is lacking. Studies that address the use of mupirocin in this setting are needed.

Among athletes, CA-MRSA infection is emerging as a challenging and potentially devastating problem that sports medicine physicians will increasingly be confronted with. All of those involved in the care of athletes should (1) be aware of the potential for CA-MRSA infection among athletes; (2) be able to recognize skin lesions that are suspicious for CA-MRSA infection; (3) understand the importance of culturing the infectious lesions in making an accurate diagnosis and guiding antibiotic treatment; (4) fully cover all infectious lesions with a clean, dry dressing until the lesion is healed; (5) encourage athletes to protect areas of the body that are most susceptible to skin injury and to fully cover areas of skin injury with a clean, dry dressing; (6) make efforts to educate athletes on CA-MRSA infection and good hygiene practices; and (7) report the occurrence of CA-MRSA infection to a local infectious disease specialist and/or the CDC.

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