Skin and Soft-Tissue Infections Caused by Methicillin-Resistant *Staphylococcus aureus*

Robert S. Daum, M.D., C.M.

This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author’s clinical recommendations.

A 37-year-old man presents for the evaluation of localized swelling and tenderness of the left leg just below the knee. He suspects this lesion developed after a spider bite, although he did not see a spider. Examination of the leg reveals an area of erythema and warmth measuring approximately 5 by 7 cm. At the center of the lesion is a fluctuant area measuring approximately 2 by 2 cm, overlaid by a small area of necrotic skin. The man’s temperature is 38.3°C. The pulse rate is 115 beats per minute. The blood pressure is 116/78 mm Hg. How should this patient be evaluated and treated?

**The Clinical Problem**

Methicillin-resistant *Staphylococcus aureus* (MRSA) refers to isolates that are resistant to all currently available β-lactam antibiotics, including penicillins and cephalosporins. MRSA isolates were first recognized shortly after the introduction of methicillin into clinical practice in the early 1960s. Their prevalence slowly increased during the next three decades, although they remained confined almost exclusively to patients who frequented health care facilities; other persons at risk for MRSA colonization or infection included those in contact with a person who had an MRSA infection or with a history of illicit drug use.

In the mid-1990s, MRSA infections began to be detected in the community in persons who did not have contact with the health care system. Molecular typing of isolates from these community-associated cases of MRSA infection has shown that they are largely caused by new MRSA strains.

As compared with health care–associated MRSA isolates, community-associated MRSA isolates are usually susceptible to clindamycin, and they are less often multiply resistant to other non–β-lactam antibiotics. Other distinguishing features of community-associated MRSA isolates include a high prevalence of genes encoding the two-component Panton–Valentine leukocidin; this exotoxin is associated with necrosis of the skin, severe necrotizing pneumonia, and abscess formation, although its role in the pathogenesis of community-associated MRSA infections remains controversial. In addition, small DNA cassettes mediating methicillin resistance have been detected in community-associated MRSA isolates of multiple genetic backgrounds, suggesting easy transfer. These cassettes differ from those in hospital-associated MRSA strains, which are larger and presumably less mobile. The classification of circulating community-associated MRSA strains according to pulsed-field electrophoretic patterns has revealed global, geographic variations. In most areas of the United States, a community-associated MRSA genotype called USA300 has emerged as the major circulating strain and has even emerged as a nosocomial strain in many areas.
Numerous reports have suggested the easy transmission of these new community-associated MRSA isolates in settings where people are in close contact. These settings include households, day-care centers, and military installations. These isolates also may be spread among prison and jail detainees and athletes. Before the 1990s, such evidence of contagion among otherwise healthy members of the community was documented infrequently. Other groups reported to be at increased risk for community-associated MRSA infection include Native Americans and Pacific Islanders and men who have sex with men.

There has been a dramatic increase in the occurrence of S. aureus infections in general and community-associated MRSA infections in particular. At Driscoll Children’s Hospital in Corpus Christi, Texas, the number of community-associated MRSA infections increased from 9 in 1999 to 459 in 2003; in 2003, these infections constituted 98% of S. aureus infections overall in that institution. In most, but not all, U.S. cities, community-associated MRSA is now the most common pathogen cultured from patients with skin and soft-tissue infections in emergency departments. Epidemic community-associated MRSA disease has also been reported from some rural areas, although epidemic disease has not yet spread to all regions of the United States.

Consistent with the occurrence of epidemic, symptomatic, community-associated MRSA disease in the United States are observations of the increasing prevalence of asymptomatic colonization of MRSA among children and adults in the community. Recent data indicate that 9.2% of healthy children in Nashville have asymptomatic colonization of MRSA (Creech CB: personal communication), as compared with 0.8% in 2001, and 7.3% of adolescents and adults in Atlanta have asymptomatic colonization, including both hospital- and community-acquired MRSA isolates.

Skin and soft-tissue infections represent the majority of the community-associated MRSA disease burden and are the focus of this article. Examples of such infections are shown in Figures 1 and 2. (Other examples are in the Supplementary Appendix, available with the full text of this article at www.nejm.org.) Necrotic skin lesions are a common presentation and are often incorrectly attributed to bites by brown recluse spiders (even in areas where these spiders do not live) or insect bites. In addition, necrotizing pneumonia, pleural empyema, necrotizing fasciitis, septic thrombophlebitis with pulmonary embolization, myositis, and severe sepsis with purpura fulminans and the Waterhouse–Friderichsen syndrome have been described in association with community-associated MRSA.

Figure 1. Anterior Abdominal-Wall Abscess in a 15-Year-Old Boy.
There was a 3-day history of drainage from this abscess, which had increased in size to 1 cm in length and become more painful. It was fluctuant and tender on examination. Incision and drainage were performed; about 2 ml of purulent material was obtained. A culture yielded MRSA that was susceptible to clindamycin. The results of the D-zone test were negative.

Figure 2. Swelling and a Small Amount of Drainage Involving the Left Naris in a 10-Year-Old Girl.
There was a 5-day history of drainage from the lesion. The child appeared well and did not have a fever. Incision and drainage yielded 0.5 ml of purulent material. A culture yielded MRSA that was susceptible to clindamycin. The results of the D-zone test were negative.
Strategies and Evidence

Evaluation

Suspicion that community-associated MRSA may be the cause of a skin and soft-tissue infection should be heightened by a history of previous MRSA infection in the patient or a household contact. Table 1 lists other groups likely to be at risk for community-associated MRSA transmission. However, many patients with community-associated MRSA infection have none of these risk factors. Furthermore, no clinical features distinguish with certainty skin and soft-tissue infections caused by MRSA from those caused by methicillin-susceptible S. aureus.

Information on local antibiotic-resistance patterns (e.g., from local hospitals) can help clinicians to assess the likelihood of community-associated MRSA infection and guide decisions regarding empirical treatment. Some have suggested that management strategies should be tailored to the possibility of community-associated MRSA infection on the basis of an arbitrary threshold of 10% or more methicillin resistance among S. aureus isolates.

Obtaining a specimen for culture and susceptibility testing, which was considered to be unnecessary when the prevalence of MRSA was low, is useful in guiding therapy. Specimens are most commonly obtained at the time of incision and drainage of purulent skin and soft-tissue lesions. In nonpurulent cellulitis that is not amenable to incision and drainage, a possible approach is a biopsy with culture of the material obtained. In practice, this procedure is infrequently performed. Moreover, although many patients with MRSA bacteremia also have nasal colonization with the organism, it is not known whether screening for such colonization in a patient with a skin and soft-tissue infection has useful predictive value. Such screening is not currently recommended.

Treatment

The recommended treatment of community-associated MRSA infection depends on an assessment of the severity of the clinical presentation and the type of skin and soft-tissue infection. Purulent skin and soft-tissue infections without associated systemic signs, such as fever, tachycardia, or hemodynamic instability, are generally managed with incision and drainage, with or without oral antimicrobial therapy; incision and drainage alone may suffice, particularly for abscesses that are small. Lee et al. have defined small abscesses as those that are less than 5 cm in length, but this definition may not be appropriate for skin and soft-tissue infections in infants and in certain areas of the body (e.g., the head and neck). In patients with larger abscesses, systemic signs of infection, or both, antimicrobial therapy is generally recommended in addition to incision and drainage (for purulent lesions). The type and route of therapy should be guided by the severity of the clinical syndrome.

Outpatient Therapies

Topical antimicrobial therapy is sometimes used to treat superficial MRSA skin infections such as impetigo, although comparative outcome data are lacking. Bacitracin, alone or in combination with polymyxin and neomycin, mupirocin (Bactroban), and retapamulin (Altabax) are commercially available for this purpose. For bacitracin, in vitro susceptibility factors that predict the clinical outcome have not been defined. For mupirocin, isolates with low-level resistance and those with high-level resistance have been identified; the latter do predict clinical failure and may be increasing in prevalence among MRSA isolates. Retapamulin is newly licensed for children 9 months of age or older. It has good in vitro activity against MRSA infection, but mutants with decreased susceptibility can be selected in vitro.

For oral systemic treatment, β-lactam antibiotics can no longer be considered to be reliable as empirical therapy for community-acquired skin and soft-tissue infections. The optimal antibiotic

<table>
<thead>
<tr>
<th>Table 1. Persons at Risk for Skin and Soft-Tissue Infections Caused by Community-Associated MRSA.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Household contacts of a patient with proven community-associated MRSA infection</td>
</tr>
<tr>
<td>Children</td>
</tr>
<tr>
<td>Day-care center contacts of hospitalized patients with MRSA infections</td>
</tr>
<tr>
<td>Men who have sex with men</td>
</tr>
<tr>
<td>Soldiers</td>
</tr>
<tr>
<td>Incarcerated persons</td>
</tr>
<tr>
<td>Athletes, particularly those involved in contact sports</td>
</tr>
<tr>
<td>Native Americans</td>
</tr>
<tr>
<td>Pacific Islanders</td>
</tr>
<tr>
<td>Persons with a previous community-associated MRSA infection</td>
</tr>
<tr>
<td>Intravenous drug users</td>
</tr>
</tbody>
</table>

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therapy when community-associated MRSA infection is suspected is not clear. Results of susceptibility testing and clinical experience provide support for a primary role of older antibiotics such as clindamycin, trimethoprim–sulfamethoxazole, and tetracyclines, although their effectiveness for skin and soft-tissue infections due to community-associated MRSA has not been rigorously evaluated or compared in clinical trials.

Table 2 lists oral agents that are useful in the outpatient management of community-associated MRSA infections. An observational study showed that clindamycin, a lincosamide antibiotic, was uniformly effective in 39 patients with clindamycin-susceptible community-associated MRSA infection who were mildly to moderately ill.46 The disadvantages of this medication include its association with diarrhea caused by Clostridium difficile and increasing rates of clindamycin resistance in some regions of the world.11,14,48 Clindamycin resistance among community-associated MRSA isolates should be monitored locally, and some experts recommend avoiding empirical therapy with clindamycin when local rates of clindamycin resistance exceed 10 to 15% among MRSA isolates causing skin and soft-tissue infections.

Moreover, the results of testing for clindamycin susceptibility may be misleading; occasional treatment failures have been documented when the results of tests showed that an MRSA isolate was susceptible to clindamycin but resistant to erythromycin.46,49 In such cases, use of the D-zone test (Fig. 3) is warranted to detect inducible clindamycin resistance; positive results in 10 to 20% of tested isolates (with one notable outlier) have been reported, but these rates may be increasing. The Clinical and Laboratory Standards Institute suggests that isolates that are positive on the D-zone test should be reported as being resistant to clindamycin despite a positive result of single-agent susceptibility testing.50 The institute suggests permissive language to accompany the result of the susceptibility testing: “The isolate is presumed to be resistant based on detection of inducible clindamycin resistance. Clindamycin might still be effective in some patients.” In practice, when the results of the D-zone test become known, the use of clindamycin should be reconsidered on the basis of the clinical response.

Neither trimethoprim–sulfamethoxazole nor tetracyclines are generally recommended as sole empirical therapy for a nonpurulent cellulitis of unknown cause because of concerns regarding the resistance of group A streptococci to these agents. Such resistance is well documented for tetracyclines, although it is less clear for trimethoprim–sulfamethoxazole.59 However, these agents are reasonable choices in cases in which community-associated MRSA infection is confirmed or strongly suggested by the presence of purulent material. Some clinicians suggest the addition of a β-lactam antibiotic, that is active against streptococci if trimethoprim–sulfamethoxazole or a tetracycline is used for a nonpurulent cellulitis of uncertain cause.

Testing of nearly all community-associated MRSA isolates shows susceptibility to trimethoprim–sulfamethoxazole, but data on the outcomes of treatment are limited. In a study at an outpatient clinic in Boston where almost half of community-associated MRSA isolates were clindamycin-resistant and where trimethoprim–sulfamethoxazole became the most frequently used antimicrobial agent for skin and soft-tissue infections caused by community-associated MRSA, the percentage of patients with clinical resolution of the MRSA infection increased in parallel with trimethoprim–sulfamethoxazole use during the study period (1998 to 2005). In another study, however, treatment failure occurred in 6 of 12 adults who received double-strength trimethoprim–sulfamethoxazole.51 Few data are available to provide support for the efficacy of doxycycline or minocycline. In one retrospective review of skin and soft-tissue infections caused by community-associated MRSA, the cure rate was 83%.62

Linezolid, a newer antimicrobial agent in the oxazolidinone family, is active against almost all community-associated MRSA isolates and group A streptococci. The disadvantages of this agent include its high cost, the lack of routine availability, hematologic side effects, and the potential for resistance among S. aureus strains, possibly by multiple mechanisms. Prolonged linezolid administration increases the likelihood of resistance, probably through the accumulation of mutations in multiple copies of the 23S ribosomal RNA S. aureus gene.53

Rifampin is highly active against susceptible community-associated MRSA isolates, but a high frequency of mutations to rifampin resistance is a contraindication for the use of rifampin alone.54 Thus, a combination of trimethoprim–sulfamethoxazole or doxycycline with rifampin is sometimes used for the treatment of skin and soft-tissue infections caused by community-asso-
### Table 2. Oral Agents for the Outpatient Treatment of Putative Community-Associated MRSA Infections.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Usual Dose*</th>
<th>Formulations</th>
<th>Main Side Effects and Contraindications</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clindamycin (Cleocin)</td>
<td>300 mg thrice daily</td>
<td>Tablet, suspension</td>
<td>Diarrhea caused by <em>Clostridium difficile</em></td>
<td>Many patients dislike the taste of the suspension</td>
</tr>
<tr>
<td>Trimethoprim–sulfamethoxazole (Bactrim, Septra)</td>
<td>1 to 2 double-strength tablets twice daily (each tablet containing trimethoprim, 160 mg, and sulfamethoxazole, 800 mg)</td>
<td>Trimethoprim, 8–12 mg/kg/day, and sulfamethoxazole, 40–60 mg/kg/day, in two divided doses</td>
<td>Table, suspension</td>
<td>Nausea, vomiting, rash, photosensitivity, hematologic suppression (especially thrombocytopenia), the Stevens–Johnson syndrome</td>
</tr>
<tr>
<td>Tetracyclines</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doxycycline (Doryx, Adoxa, Doxy-100, Monodox, Vibramycin, Vibra-Tabs)</td>
<td>100–200 mg/day, in one dose or two divided doses</td>
<td>2–4 mg/kg/day, in one dose or two divided doses</td>
<td>Capsule, tablet, suspension</td>
<td>Nausea, photosensitivity, deposition in teeth and bones</td>
</tr>
<tr>
<td>Minocycline (Dynacin, Minocin)</td>
<td>200 mg/day, in two divided doses</td>
<td>4 mg/kg/day, in two divided doses</td>
<td>Capsule, tablet, suspension</td>
<td>Nausea, photosensitivity, deposition in teeth and bones, vestibular toxicity</td>
</tr>
<tr>
<td>Linezolid (Zyvox)</td>
<td>600 mg twice daily</td>
<td>30 mg/kg/day, in three divided doses</td>
<td>Tablet, suspension</td>
<td>Myelosuppression (usually thrombocytopenia), but can cause anemia or neutropenia, mostly with prolonged use</td>
</tr>
<tr>
<td>Rifampin (Rifadin, Rimactane)</td>
<td>20 mg/kg/day, in one dose or two divided doses; maximum dose, 600 mg/day</td>
<td>20 mg/kg/day, in one dose or two divided doses; maximum dose, 600 mg/day</td>
<td>Capsule</td>
<td>Discoloration of body fluids, abnormalities in liver function, drug–drug interactions</td>
</tr>
</tbody>
</table>

*Optimal doses have not been established for all drugs listed.*
associated MRSA, although data are lacking to provide support for this approach.

Fluoroquinolones should not be used to treat skin and soft-tissue infections caused by community-associated MRSA. Resistance to them develops readily in *S. aureus* and is already widely prevalent.24

**Inpatient Therapies**

Some patients with community-associated MRSA infection will require more aggressive treatment than incision and drainage with or without oral antimicrobial therapy on an outpatient basis. A decision to hospitalize a patient for parenteral therapy (Table 3) depends on several factors, including clinical judgment regarding the severity of the illness. The presence of a large abscess, fever, other signs of systemic infection, or high-risk characteristics such as an age younger than 6 months, diabetes, or immunodeficiency should prompt consideration of hospitalization. The detailed management of invasive disease due to community-associated MRSA is beyond the scope of this review.

Vancomycin is still considered the first-line treatment for hospitalized patients with invasive *S. aureus* infection. However, this drug should be switched if susceptibility testing indicates that a more rapidly bactericidal β-lactam agent such as oxacillin would be appropriate. Microbiologic treatment failure may occur with vancomycin even if there is no increase in the minimal inhibitory concentration (MIC) on susceptibility testing.55,56 *S. aureus* isolates with low-level (so-called intermediate) resistance to vancomycin (MIC, >2 μg per milliliter) as well as those with high-level resistance (MIC, >16 μg per milliliter) have been described, and they may not be identified by means of routine techniques for susceptibility testing.57 Although resistant isolates are believed to be infrequent, global decreased susceptibility (so-called MIC creep) among *S. aureus* isolates has been documented in several locations in the United States,58-60 and this decreased susceptibility may limit the continued effectiveness of vancomycin. Some experts have proposed that the use of a higher dose and maintenance of high serum levels of vancomycin may be beneficial, but the efficacy of these strategies has not been proved.

Parenteral clindamycin may be useful in regions where the likelihood of a resistant organism is low. It should not be used as sole therapy when the patient is moderately to severely ill.

Intravenous trimethoprim–sulfamethoxazole has undergone minimal evaluation for invasive *S. aureus* infection. A study of intravenous drug abusers with serious *S. aureus* infections antedated the epidemic of community-associated MRSA infection, and it indicated that intravenous trimethoprim–sulfamethoxazole was significantly less effective than vancomycin.61 Parenteral linezolid lacks bactericidal activity, which some experts believe is important in treating intravascular infection, a common feature of invasive disease. Moreover, reports of a case of endocarditis caused by a susceptible organism during linezolid therapy and of clinical failure in patients treated with linezolid for endocarditis have raised concerns about its use alone for severe, invasive *S. aureus* infections62,63 (an exception is health care–associated MRSA pneumonia, for which linezolid has proved efficacious64).

Tigecycline, a parenteral glycyclline–minocycline derivative, was also recently approved by the Food and Drug Administration (FDA) for the...
treatment of skin and soft-tissue infections caused by MRSA. This approval was granted on the basis of data showing microbiologic eradication in 25 of 32 adults (78%) with complicated skin and soft-tissue infections.

A fixed combination of the streptogramins quinupristin and dalfopristin (Synercid) was licensed by the FDA for the treatment of skin and soft-tissue infections caused by methicillin-susceptible S. aureus. Its use has been limited by the potential for drug–drug interactions and by side effects (including arthralgias, myalgias, and gastrointestinal toxic effects).

Daptomycin, a cyclic lipodepsipeptide, has been approved by the FDA for use in patients with skin and soft-tissue infections. The success rate with the use of daptomycin for these infections is 75% — similar to that of vancomycin. It is also approved for MRSA bacteremia, including that associated with right-sided endocarditis, but it should not be used for pneumonia, for which its efficacy has been limited by its propensity for binding surfactant.

### Table 3. Parenteral Agents for the Treatment of Putative Community-Associated MRSA Infections.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Usual Dose*</th>
<th>Main Side Effects and Contraindications</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vancomycin (Vancocin)</td>
<td>2–4 g/day, in two to four divided doses</td>
<td>The red-man syndrome (a histamine-release syndrome usually manifested as flushing)</td>
<td>Slowing the rate of administration is usually sufficient management for the red-man syndrome, but accompanying hypotension may require discontinuation of the drug or additional intervention in rare cases. Excretion is slowed in patients with renal failure, and serum levels should be monitored in such patients to avoid drug accumulation; whether such monitoring is routinely necessary in patients with normal renal function is not clear, but it should be performed when multiple nephrotoxic drugs are administered simultaneously.</td>
</tr>
<tr>
<td>Clindamycin (Cleocin)</td>
<td>300 mg thrice daily</td>
<td>Diarrhea caused by <em>C. difficile</em></td>
<td>Resistance was documented in 6 of 120 patients receiving this therapy†. Excretion is slowed in patients with renal failure, and dosage adjustment is recommended.</td>
</tr>
<tr>
<td>Daptomycin (Cubicin)</td>
<td>4–6 mg/kg, once daily</td>
<td>Unknown</td>
<td>Potential muscle toxicity</td>
</tr>
<tr>
<td>Tigecycline (Tygacil)</td>
<td>100 mg loading dose, then 50 mg every 12 hr</td>
<td>Unknown</td>
<td>Nausea, vomiting, photosensitivity, deposition in teeth and bones. Contraindicated in children younger than 9 years of age because of potential deposition in teeth and bones.</td>
</tr>
<tr>
<td>Linezolid (Zyvox)</td>
<td>600 mg, every 12 hr</td>
<td>30 mg/kg/day, in two to three divided doses</td>
<td>Myelosuppression (usually thrombocytopenia, but also anemia or neutropenia), mostly with prolonged use. The cost is relatively high.</td>
</tr>
<tr>
<td>Quinupristin and dalfopristin (Synercid)</td>
<td>7.5 mg/kg, every 8–12 hr</td>
<td>Hyperbilirubinemia, arthralgias and myalgias, phlebitis, drug–drug interactions (especially with cytochrome P450 3A4 substrates)</td>
<td>Dosage adjustment may be necessary in patients with hepatic impairment.</td>
</tr>
</tbody>
</table>

* Optimal doses have not been established for all drugs listed.
† Data are from Fowler et al.55

**A R E A S  O F  U N C E R T A I N T Y**

The optimal oral antimicrobial regimen for the treatment of skin and soft-tissue infections is not...
known. A trial addressing this question, sponsored by the National Institutes of Health, is expected to be initiated this year.

The optimal management of recurrent community-associated MRSA disease is also uncertain. Although not well studied, the recurrence rate is believed to be 10% or higher. It is not clear whether recurrences represent autoinoculation or a new MRSA infection. At present, recurrent episodes are generally treated in the same way as the initial episode. In addition, “decolonization” strategies are frequently recommended in such cases, although neither the indications for their use nor their effectiveness in reducing the risk of recurrences is clear. One such strategy is the use of intranasal mupirocin to reduce nasal carriage of MRSA; however, eradication of nasal colonization appears to be transient, and the use of this agent remains controversial. Moreover, the recent identification of a mupirocin-resistance gene in USA300 isolates (which accounted for 97% of isolates in a recent study24) and of mupirocin resistance among 11 community-associated MRSA isolates in Boston raises serious concern about exposing populations of staphylococci to this agent.67 Some experts have also proposed adjunctive attempts at skin decolonization. Topical chlorhexidine gluconate or 1 tsp (3.4 g) of bleach diluted in 1 gallon (3.8 liters) of bath water is commonly suggested, although these approaches have not been rigorously evaluated. The optimal strength of the chlorhexidine solution is not known, nor is it clear whether it is more effective if the solution is permitted to remain on the skin before rinsing.

Contagion among the close household contacts of patients, as well as correctional facility, school, and sports-team contacts, is well recognized. Although the risk of transmission has not been well quantified, anecdotal evidence suggests that more than 60% of households of children hospitalized with community-associated MRSA infections have one or more members with a history of a putative MRSA infection in the previous 6 months. If this estimate proves to be correct, it will lend support to the empirical treatment of an entire household (perhaps even including pets) if an effort to eradicate community-associated MRSA colonization in a patient is undertaken. The efficacy of such an approach has not been studied.

The role of fomites needs to be clarified. Hospital-acquired MRSA isolates can survive on a variety of inanimate surfaces, sometimes for weeks. It is unclear whether this is also true for community-associated MRSA isolates; if it is, their presence on such items as clothing, towels, and athletic equipment might contribute to outbreaks. Pets (including dogs and cats), livestock, and birds have been identified as MRSA carriers; their role in MRSA transmission to humans requires further evaluation.68 Local hygiene measures recommended by an expert panel from the Centers for Disease Control and Prevention (CDC) are shown in Table 4.

No vaccine is currently available for S. aureus. Many experts believe that it is unlikely that a single-antigen approach will prove to be effective.

### Table 4. Recommended Measures to Limit the Spread of Community-Associated MRSA Isolates.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cover draining wounds with clean bandages.</td>
<td></td>
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<tr>
<td>Wash hands, especially after contact with a contaminated wound.</td>
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</tr>
<tr>
<td>Launder clothing after contact with a contaminated area on the skin.</td>
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<tr>
<td>Bathe regularly with use of soap.</td>
<td></td>
</tr>
<tr>
<td>Avoid sharing items (e.g., towels, bedding, clothing, razors, or athletic equipment) that may become contaminated by contact with wounds or skin flora.</td>
<td></td>
</tr>
<tr>
<td>Clean sports equipment with agents that are effective against staphylococci (e.g., a detergent or disinfectant registered by the Environmental Protection Agency, such as quaternary ammonium compounds or a solution of dilute bleach).</td>
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</tr>
</tbody>
</table>

*Information is modified from Gorwitz et al.69*
causing skin and soft-tissue infections acquired in the community and recommend inclusion of antimicrobial therapy against community-associated MRSA when managing a putative S. aureus infection.

In a patient such as the man described in the vignette, presenting with an abscess or a purulent and necrotic skin lesion, incision and drainage are the cornerstones of therapy; purulent material should be cultured. In many patients, particularly those with small lesions (<5 cm in length), incision and drainage alone will be adequate therapy. If the skin lesions are large or accompanied by systemic signs of infection or if there is evidence of an increased risk of complicated community-associated MRSA disease, antimicrobial therapy that is active against community-associated MRSA is also recommended. Therapy ultimately should be guided by the results of susceptibility testing of cultures obtained before the initiation of therapy.

Although data directly comparing antimicrobial agents for the treatment of community-associated MRSA infection are lacking, clindamycin, trimethoprim–sulfamethoxazole, or a long-acting tetracycline such as doxycycline is a reasonable initial choice; linezolid is another possibility. Follow-up is essential, since relapse or recurrence may occur.

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REFERENCES


CORRECTION

Skin and Soft-Tissue Infections Caused by Methicillin-Resistant *Staphylococcus aureus*

Skin and Soft-Tissue Infections Caused by Methicillin-Resistant *Staphylococcus aureus*. The last sentence of the third paragraph (page 380) should have read “In most areas of the United States, a community-associated MRSA genotype called USA300 has emerged as the major circulating strain and has even emerged as a nosocomial strain in many areas,” rather than “USA300 has emerged as the major circulating and nosocomial strain.” In Table 2 (page 384) the usual dose of doxycycline for adults should have read “100–200 mg/day, in one dose or two divided doses” rather than “in two or four divided doses.” The usual dose of doxycycline for children should have read “2–4 mg/kg/day, in one dose or two divided doses” rather than “2–4 mg/kg/day, in two or four divided doses.” Also in Table 2, the usual dose of rifampin for adults should have read “20 mg/kg/day, in one dose or two divided doses” rather than “in two or four divided doses.” The usual dose of rifampin for children should have read “20 mg/kg/day, in one dose or two divided doses” rather than “in two or four divided doses.” In Table 3 (page 386) the usual dose of daptomycin for adults should have read “4–6 mg/kg, once daily” rather than “4–6 mg/kg/day, in four divided doses.” The usual dose of linezolid (Zyvox) for adults should have read “600 mg, every 12 hr” rather than “600 mg/day, in two divided doses.” The text and tables have been corrected on the Journal’s Web site at www.nejm.org. We regret the errors.