Management of Skin and Soft-Tissue Infections in the Emergency Department

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Skin and soft-tissue infections (SSTIs) are among the most common infections encountered by emergency physicians. The spectrum of disease severity as seen by emergency physicians is wide, and can range from mild, uncomplicated cellulitis to cutaneous abscesses and necrotizing SSTIs. Infections can include acute, recurrent, and chronic wounds, and community-associated and health care-associated infections in immunocompetent or immunocompromised hosts.

Because of the nature of their practice, emergency physicians encounter patients on a daily basis with a wide variety of mechanisms that lead to SSTIs. These can include animal and human bites, gunshot wounds, illicit drug injection, work-related injuries, pressure sores, and iatrogenic injuries caused by procedures (eg, intravenous lines). Similarly, infectious disease (ID) specialists see a variety of SSTIs in their practice; however, for the ID specialist the spectrum of disease is more skewed toward patients with a complicated course, those who had multiple treatment failures, and recurrent, severe, or rare and unusual infections. Unlike ID specialists, emergency physicians more frequently have to initiate empiric antibiotics because of the absence of culture and susceptibility results, more often have to consider life
and limb-threatening SSTIs, and commonly perform minor surgical care of abscesses and infected wounds.

This article is written from the perspective of the evaluation and initial management of SSTIs in the emergency department (ED). It highlights the management pitfalls and clinical dilemmas pertinent to emergency physicians that are not often encountered by ID specialists. Emphasis is placed on the utility of wound and blood cultures, disposition, methicillin-resistant *Staphylococcus aureus* (MRSA) infections, animal and human bites, and necrotizing SSTIs.

### Classification

Numerous classification schemes have been proposed for SSTIs. Each classification divides SSTIs based on specific variables, such as infection of normal skin (primary skin infection) versus infection complicating a chronic skin disorder (secondary infection), acute versus chronic, localized versus diffuse, and nonnecrotizing versus necrotizing infections. Another proposed classification system divides SSTIs based on the severity of local and systemic signs, and symptoms of infection and the presence of comorbidities [1].

The most commonly used classification system for SSTIs is based on the presence or absence of complicating factors (ie, uncomplicated versus complicated infections)[2,3]. This classification system is used by the pharmaceutical industry in the design of registration studies for new antimicrobials.

The Center for Drug Evaluation and Research (CDER) at the United States Food and Drug Administration (FDA) has proposed a series of guidance documents for the pharmaceutical industry in development of antimicrobial drugs for the treatment of SSTIs [3]. According to CDER, the uncomplicated category of SSTI includes simple abscesses, impetiginous lesions, furuncles, and cellulitis. The complicated category includes infections involving deeper skin structures, those requiring formal surgical interventions (eg, infected ulcers, burns, major abscesses), and the presence of a significant underlying medical condition that complicates the response to treatment. Infections involving anaerobic or gram-negative organisms (eg, rectal abscesses) are considered complicated [3]. Table 1 depicts examples of skin infections based on the uncomplicated versus complicated classification scheme [2,3].

The most recent practice guidelines developed by the Infectious Disease Society of America (IDSA) for the diagnosis and management of SSTIs do not categorize skin infections based on the uncomplicated versus complicated terminology [4]. The guidelines are written in reference to specific disease entities (eg, cellulitis, abscess, necrotizing infection), mechanism of injury (eg, animal bites, human bites, infection associated with animal contact, surgical site infection), or host factors (eg, infections in the immunocompromised patient) [4]. Unfortunately, these guidelines were written just as community-associated MRSA (CA-MRSA) was emerging as an
important cause of SSTI. The management of SSTIs has changed considerably in the last few years and some of these changes are not reflected in the IDSA guidelines.

**Microbiology**

The microbiology of SSTIs is dependent on numerous factors, such as the host, the environment (eg, community- versus health care-associated), the mechanism of injury, and the duration and severity of illness. Knowledge of these variables is imperative in the selection of an initial empiric antimicrobial regimen.

In general, *S. aureus*, and to a lesser extent streptococci, are by far the most common causes of both uncomplicated and complicated SSTIs, with a few notable exceptions, such as animal and human bite-wound infections [5,6]. Polymicrobial infections with gram-negative and anaerobic organisms are typically seen in complicated infections [2,5–8].

The most important new development in the era of SSTI is the emergence of CA-MRSA, a phenomenon largely recognized and studied in ED populations. CA-MRSA infections have become a global emerging health problem. Most infections are noninvasive [9], involve skin and soft-tissue structures, and present as purulent skin infections [10–12]. The spectrum of skin infections caused by CA-MRSA is wide and can range from simple cutaneous abscesses to fulminant necrotizing fasciitis [12,13].

In the United States, the most common strain of MRSA associated with community infections is USA300, and less commonly USA400 and USA1000 [12,14]. Compared with health care-associated strains, CA-MRSA strains more frequently produce toxins and appear to be more virulent. The Panton-Valentine leukocidin genes, which produce cytotoxins that cause tissue necrosis, damage host cell membranes, and leukocyte
destruction, are commonly seen with CA-MRSA and rarely with health care-associated MRSA (HA-MRSA) strains [15–19]. Novel peptides have been recently identified that are cytotoxic to leukocytes and may be important virulence factors of CA-MRSA [20].

In a recent multicenter, prospective, prevalence study involving adult subjects with purulent SSTIs, MRSA was isolated from 59% of subjects (n = 422; range: 15% to 74%). In this study, MRSA was isolated from 61% of abscesses, 53% of infected wounds, and 47% of cellulitis associated with purulent exudate. Methicillin-susceptible S. aureus (MSSA) was isolated from 14% of abscesses, 21% of infected wounds, and 34% of cellulitis associated with purulent exudate. Streptococcal species was isolated from 7% of abscesses, 9% of infected wounds, and 13% of cellulitis associated with purulent exudate [12]. Although it is clear that MRSA is now a common cause of SSTI overall, it is not clear to what extent it is now responsible for all possible subtypes of SSTI, such as diabetic foot infections or bite-wound infections.

In one investigation of ED patients with SSTI, historical and clinical features associated with MRSA compared with other bacteria include a history of close contact with similar infection (odds ratio [OR], 3.4; 95% confidence index or CI, 1.5–8.1; \( P < .05 \)), prior history of MRSA infection (OR, 3.3; 95% CI, 1.2–10.1; \( P < .05 \)), reported “spider bite” (OR, 2.8; 95% CI, 1.5–5.3; \( P < .05 \)), received antibiotics in the past 1 month (OR, 2.4; 95% CI, 1.4–4.1; \( P < .05 \)), and abscess (OR, 1.8; 95% CI, 1.0–3.1; \( P < .05 \)) [12]. A common pitfall is to assume one can reliably exclude MRSA based on the absence of risk factors. However, even in the absence of risk factors, CA-MRSA is still prevalent, and was present in 48% of ED SSTI patients in one investigation [12]. There are no clinical or epidemiologic risk factors that can exclude MRSA etiology and reliably distinguish between patients infected with CA-MRSA and other organisms [11,12,21,22].

Another common mistake is to assume one can also reliably differentiate CA-MRSA and HA-MRSA based on epidemiologic factors. The epidemiologic definition of HA-MRSA infection includes MRSA identified after 48 hours of hospital admission, or identified in patients with a history of surgery, hospitalization, dialysis, or residence in a long-term care facility within the last year, an indwelling catheter or other invasive medical devices, and previous history of MRSA infection or colonization [9,23,24]. The exclusive use of this definition can underestimate the true prevalence of CA-MRSA infection. In the ED-based study by Moran and colleagues [12], almost all MRSA isolated from SSTIs had molecular characteristics of CA-MRSA, even though more than 25% of subjects met the epidemiologic definition of HA-MRSA.

Another pitfall is to assume that pure cellulitis is exclusively caused by streptococci, as suggested by the recent IDSA guidelines [4]. Investigations into the etiology of cellulitis without drainage are limited by the lack of specimen availability. Studies using tissue biopsies and aspirate specimens, and
relying on conventional and nonconventional identification methods, such as immunofluorescence and serologic testing, suggest that *S. aureus*—and to much lesser extent streptococci—are the most common pathogens implicated in the pathogenesis of cellulitis [25–32]. With the emergence of CA-MRSA, the role of this pathogen in these difficult-to-study infections is uncertain. In the ED-based study by Moran and colleagues, among cellulitis cases accompanied by purulent drainage, MRSA was found in 47% [12]. Thus, it is reasonable to assume CA-MRSA has a role in these infections. Also, it has become evident that many occult cutaneous abscesses, which are now predominantly caused by CA-MRSA, are misdiagnosed as cellulitis on clinical evaluation, potentially leading to treatment failure, and their detection is greatly enhanced with the use of bedside soft-tissue ultrasonography [33,34].

Necrotizing fasciitis and myonecrosis are typically caused by infection with Group A streptococcus, *Clostridium perfringens*, or, most commonly, aerobic and anaerobic organisms as part of a polymicrobial infection that may include *S. aureus*. In case series, CA-MRSA has recently been described as a predominantly monomicrobial cause of necrotizing fasciitis [35,36]. A retrospective review of patients presenting with necrotizing fasciitis between 2000 and 2006 indicated that MRSA was the most common pathogen, accounting for one-third of the organisms isolated [37].

Other important causes of SSTI, including dog, cat, and human bite infections have been investigated among ED patients. Bite-wound infections are often mixed aerobic and anaerobic infections, with some unique pathogens transmitted by the biter’s oral flora. In a study of 107 subjects with dog and cat bite infections, mixed aerobic and anaerobic infections were present in 56% of all wounds (dogs: 48%; cats: 63%) [5]. *Pasteurella* species was the most common pathogen isolated from both dog (50%) and cat (75%) bites and was commonly isolated from abscesses and puncture wounds. The most common pasteurella strain isolated from infected dog bites was *P. canis* (26%), while in infected cat bites it was *P. multocida* subspecies *multocida* (54%). Streptococci were seen in 46% of both types of bite infections. *S. aureus* (all MSSA) was only isolated in 20% of dog bite infections and 4% of cat bite infections, suggesting animal oral flora dominates human skin flora, especially for deep puncture wounds. Anaerobes (eg, *Fusobacterium nucleatum*, *Bacteroides tectum*, *Porphyromonas* species, *Prevotella heparinolytica*) were usually present as mixed infections with aerobic organisms.

Similar to animal bites, human bites are also frequently mixed aerobic and anaerobic infections. In a study of 50 subjects with infected human bites, 54% of wounds were mixed aerobic and anaerobic infection with organisms [6]. The most common organisms recovered from human bite wounds included *Streptococcus* species (84%; *S. anginosus* was the most common pathogen, isolated in 52% of cases), *S. aureus* (30%), *Eikenella corrodens* (30%), *Prevotella* species (36%), *Fusobacterium* species (34%), and *Veillonella* species (24%) [6].
Mixed aerobic and anaerobic bacteria are also commonly observed in SSTIs among injecting-drug users (IDUs). A comparative microbiologic study of cutaneous abscesses among IDUs and non-IDUs found a greater frequency of mixed aerobic and anaerobic infections in IDUs. The prevalence of oral anaerobic organisms was higher among IDUs. In addition to \textit{S. aureus}, which was found in 50\% of IDU abscess cases, \textit{Streptococcus milleri, F. nucleatum, Prevotella species, Peptostreptococcus micros, Actinomyces odontolyticus,} and \textit{Veillonella species} were found [38].

**Clinical presentation**

The clinical manifestations of SSTIs are variable and depend on factors such as the host, the infecting organism, and the inciting event. CA-MRSA SSTIs commonly present as a spontaneous abscess [10–12]. The patient often attributes the infection to a “spider bite.” Misclassification of a deep abscess as cellulitis is a common pitfall. Physicians often attribute treatment failure of “cellulitis” to antimicrobial resistance and change to a different, or often multiple, antibiotic regimen. The presence of an underlying deep abscess should be considered in patients with cellulitis who “fail” initial antimicrobial therapy. Treatment failure may be caused by an undrained abscess that was missed on the initial presentation, rather than inadequate antimicrobial therapy. Bedside ultrasound is more frequently available in the ED and is recommended for evaluation of all suspicious SSTIs.

Some wounds may be associated with injuries to deeper structures, such as bones, joints, tendons, and neurovascular structures. Animal and human bite injuries that appear to be minor may have underlying joint and other structural injuries. Clenched fist injuries (“fight bites”) are particularly prone to underlying bone or metacarpal joint involvement that can be missed on initial presentation. Cats, with their sharp thin teeth, often produce small deep puncture wounds that seem to be trivial, but further evaluation may reveal joint or cortical injuries. Pain out of proportion to the wound and physical findings should raise suspicion for joint or bony injuries.

Different terms and classifications have been used to describe various types of necrotizing SSTIs (eg, synergistic necrotizing cellulitis, necrotizing fasciitis, streptococcal myonecrosis, gas gangrene). Although various factors may distinguish each type of infection, it is currently recommended to describe these generally as “necrotizing soft-tissue infections” or necrotizing SSTI, because their initial treatment, which emphasizes early surgical intervention and broad-spectrum antimicrobials, is the same [39].

Patients with necrotizing SSTI most often present with severe pain (often out of proportion to physical findings) and rapidly advancing induration and tenderness that extends beyond the area of erythema. These patients appear ill and most often have abnormal vital signs on presentation. However, the recognition of a necrotizing SSTI, especially in the early stages, is
extremely difficult. Patients present in variable stages in the spectrum of the illness. In a retrospective review of 89 patients with necrotizing fasciitis, only 13 (15%) patients had the diagnosis of necrotizing fasciitis at the time of admission [35]. At the time of the presentation, patients can be afebrile [36,40] and have minimal pain at the site of infection [36]. The cutaneous findings early in the course of disease may be lacking or are nonspecific. In a retrospective review of patients with necrotizing SSTIs, the most specific signs, crepitus and blistering, were absent in 63% (n = 189) and 76% (n = 190) of patients, respectively [40]. In 15% of patients (n = 170), none of the specific findings, (ie, crepitus, blistering, or radiographic evidence of soft-tissue gas) were present [40]. Lymphangitis and lymphadenopathy are typically absent [41].

Recent reports (mostly case reports and case series) of necrotizing SSTIs caused by CA-MRSA have revealed that this type of necrotizing SSTI presents more subacutely and is a more benign syndrome than necrotizing infections caused by other etiologies [13,42–44]. In a retrospective study of 14 patients with necrotizing SSTIs, 57% of patients had a preoperative diagnosis of abscess, and necrotizing infection was not suspected [13]. In this series, the onset of disease was often subacute (average 6 days; range, 3–21), and even in the presence of serious complications (eg, need for reconstructive surgery and prolonged stay in the intensive care unit), none of the patients died [13]. The 100% survival rate in this series, compared with the approximately 70% survival in most necrotizing SSTI series [36,40], suggests that perhaps some of these may be a different type of necrotizing infection. Large abscesses that can be caused by CA-MRSA are sometimes found to have necrotic tissue, even though they are not associated with the high mortality of what has been traditionally considered to be a necrotizing infection.

**Laboratory evaluation and imaging**

Microbiologic studies, such as wound cultures and blood cultures, can be of value in selected patients with SSTIs; however, their routine use in all SSTI is debated [45]. Historically, routine wound cultures were rarely performed because of the predictable infection etiology and the associated antimicrobial susceptibility patterns. In the current era of increased prevalence of SSTIs caused by CA-MRSA, and varying antimicrobial susceptibility patterns—even among CA-MRSA strains—more physicians are performing wound cultures to assess the etiology of the infection and guide further antimicrobial therapy.

The decision to do any diagnostic test, including wound or blood cultures, depends on its clinical utility and the likelihood that the result will change management. The majority of patients with CA-MRSA infection will present with simple, uncomplicated abscess [12]. Although the additional benefit of CA-MRSA active antibiotics requires further study, based on the current
available evidence, the management of uncomplicated abscesses, even those caused by CA-MRSA, is solely incision and drainage, which is associated with a cure rate of 85% to 90% [12,46–48]. Therefore, it is unlikely that culture and susceptibility testing would be helpful in the patient’s care.

Wound cultures should be reserved for patients that have a greater chance of treatment failure, such as patients with complicated infections, immunocompromising conditions, moderate-to-severe illness, refractory or recurrent infections, and those that have failed prior surgical therapy. Wound cultures are also appropriate for patients who will be treated with an antibiotic with variable activity against the presumed pathogen (eg, CA-MRSA and clindamycin). Cultures should be obtained for all patients admitted to the hospital, both to ensure that the empiric regimen has activity against the pathogen, and to allow appropriate narrowing of the antimicrobial spectrum (eg, switch vancomycin to oxacillin if MSSA is identified).

Although pathogen prevalence rates and susceptibility patterns can be surmised from the selected group of patients who require cultures, broader local surveillance can help to determine optimal empiric therapy for future patients. However, patients should be spared the expense of tests for which they receive no direct benefit, and costs of surveillance testing should be borne by public health departments or health care systems. At this point in time, CA-MRSA appears to be a frequent and endemic cause of SSTI; however, its antimicrobial susceptibility pattern appears to be changing.

As with wound cultures, the utility of blood cultures in all types of SSTIs is also debated. A typical case of a cellulitis deemed appropriate for outpatient antimicrobial therapy is unlikely to be associated with bacteremia. Even for hospitalized patients with community-acquired cellulitis, bacteremia is an uncommon finding [49], and discordant antibiotic therapy is rare. In a study of 757 hospitalized subjects with community-acquired cellulitis [49], blood cultures were performed on 553 subjects. A “true” pathogen (ie, not a contaminant) was isolated from the blood cultures in only 11 cases (2%) [49]. In order of decreasing frequency, isolated organisms included: Group G streptococcus (5), Group A streptococcus (3), *S. aureus* (1), *Vibrio vulnificus* (1), and *Morganella morganii* (1). Univariate regression analysis revealed that an age greater than 45 years, short duration of symptoms before presentation, temperature higher or equal to 38.5°C, and white blood cell (WBC) count greater than 13,300/mm³ at admission were predictive of bacteremia. Initial empiric antimicrobial therapy was concordant in all patients with bacteremia. For the two patients with gram-negative bacteremia, there was sufficient historical information to suspect an infection with an unusual organism. One patient with *V. vulnificus* bacteremia was a 69-year-old male, with a history of fish bone injury to his right hand second digit, who developed cellulitis and chills 6 hours after the injury. Another patient with *M. morganii* bacteremia was a 50-year-old female with past medical history of noninsulin-dependent diabetes mellitus and end-stage
renal disease, receiving hemodialysis that was temporality administered through an indwelling central catheter. Contaminated samples were almost twice as common as true-positive blood cultures. False-positive blood culture results have the potential for introducing inappropriate treatment and can increase length of hospital stay, resource use, and costs [50].

The latest IDSA SSTI guidelines [4] mention the low yield of blood cultures; however, they fail to clearly recommend which patients would benefit from them. Other sources recommend reserving blood cultures for patients with signs and symptoms of systemic toxicity, severe infections, those with chills and high-grade fever, elderly patients with acute onset of illness, significant leukocytosis, and immunocompromised patients [1,4,49,51]. However, most patients that are considered for hospitalization have some to all of these loosely defined criteria.

Blood cultures are only helpful if the results would identify the need for a change in a patient’s therapy such that it would improve clinical outcomes. ID specialists should reconsider the need for routine blood cultures in ED patients who are to be admitted to the hospital with SSTI, especially if the pathogen can be isolated from the infected wound or abscess. Instances in which blood cultures could be of clinical utility can include patients with SSTI who present with severe sepsis or septic shock [52], are at risk for an unusual infection requiring unusual therapy, have immunocompromising conditions, and for whom the finding of bacteremia would have important implications, such as patients at risk for endovascular infection (eg, those with prosthetic heart valves).

Other diagnostic tests to investigate the microbiologic etiology of SSTIs have included cultures from needle aspirations of inflamed skin (either at the point of maximal inflammation or the leading edge) and tissue punch biopsies. The positive yields of these techniques are variable and, overall, low [25–28,53], and as a result are not recommended.

Tissue biopsy and frozen section examination has been advocated for patients with suspected necrotizing SSTI [54,55]. However, correct interpretation of this test requires an experienced pathologist (not often available at all times), and the process can result in a delay in surgical intervention. Most surgeons prefer to explore the site of infection directly in the operating room. Rapid streptococcal antigen testing of the wound (which is only approved for pharyngeal infections) in the ED has been reported to provide early identification of streptococcal toxic shock syndrome [56]. Serologic tests for streptococcal infection are not helpful in the ED because meaningful interpretation of these tests requires paired acute and convalescent titers.

Basic hematologic studies and serum chemistries are commonly performed on patients requiring hospital admission. These tests can aid in assessing the severity of disease, reveal organ dysfunction, and may expose underlying medical conditions (eg, anemia, renal disease). The most recent IDSA SSTI guidelines recommend obtaining these tests in patients with
signs and symptoms of systemic toxicity (eg, fever, hypothermia, tachycardia, and hypotension) [4].

A diagnostic scoring system based on laboratory tests has been proposed as a tool for distinguishing necrotizing SSTI from other SSTIs [57]. The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score is depicted in Table 2. The score assigns points based on the level of various laboratory variables that include C-reactive protein, total WBC count, serum sodium, creatinine, glucose, and hemoglobin. The maximum cumulative score is 13. A score greater than or equal to 6 had a positive predictive value of 92% (95% CI, 84.3–96.0) and negative predictive value of 96% (95% CI, 92.6–97.9) for necrotizing SSTI. The probability of necrotizing SSTI increased to more than 75% when the LRINEC score was greater than or equal to 8 (Table 3). The authors advocate using this tool as an adjunct in the management of SSTI. However, the ability of the LRINEC score to detect early cases of necrotizing SSTI is unproven.

The derivation of the LRINEC score was based on a retrospective observational study of subjects divided into developmental or derivation (n = 314) and validation (n = 140) cohorts [57]. Because of the retrospective nature of the study, for both groups (developmental and validation cohorts) the diagnosis of necrotizing SSTI and nonnecrotizing SSTI was already made. Unfortunately, the utility of the LRINEC score is potentially limited

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<th>Laboratory parameter (units)</th>
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<td>&lt;150</td>
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<td>≥150</td>
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<td>11–13.5</td>
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<td>Creatinine (mg/dL)</td>
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in that it needs to be prospectively validated in patients for whom the diagnosis of necrotizing SSTI is not apparent on initial history and physical examination, thus testing the ability of the score to detect early and atypical presentations that would benefit from earlier surgical intervention.

It is also unclear if the LRINEC score can be applied to all age groups. The youngest subjects enrolled in the developmental and validation cohorts were 27 and 13 years of age, respectively [57]. C-reactive protein, a test component of LRINEC score, is also not a universally available test and has a variable turn-around time; hence, its utility is somewhat limited. However, finding abnormalities that make up the LRINEC score in patients with SSTI should increase suspicion of a necrotizing infection such that further observation and evaluation should be considered. Although not part of the LRINEC score, an elevated creatine kinase should also increase suspicion of a necrotizing infection. Urinalysis can provide a clue to the presence of rhabdomyolysis by demonstrating blood in urine in the absence of red blood cells.

Other diagnostic tests used in the evaluation of SSTIs include soft-tissue radiographs, CT scans, ultrasonography, and magnetic resonance imaging. Utilization of these adjunctive diagnostic tests is based upon the physician’s index of suspicion for osteomyelitis, subcutaneous emphysema, or deep abscess. However, it is also important to understand the limitations of these tests. In a study of 148 subjects with necrotizing SSTI, 27% of subjects did not have evidence of soft-tissue gas in plain radiographs or CT scans [40]. In another study, only 29% (n = 65) of subjects had evidence of soft-tissue gas in plain radiographs or CT scans [36].

Ultrasonography can be useful in the identification of deep abscesses, especially in cases in which the physical examination is equivocal or there is a broad area of what appears to be cellulitis. In this situation, ultrasound has been shown to have greater sensitivity and specificity in detecting deep abscesses than clinical examination alone (sensitivity: 98% versus 86%; specificity: 88% versus 70%) [33,34].

### Drainage and debridement

Incision and drainage is a commonly performed procedure to treat cutaneous abscesses and emergency physicians are very familiar with performing
this procedure. A step-by-step, descriptive video clip of how to perform incision and drainage can be viewed at http://content.nejm.org/cgi/content/short/357/19/e20 [58].

The initial step in performing an incision and drainage is to prepare the site for incision and remove any dirt or debris. The infected area is further cleaned with a skin-cleansing agent (eg, chlorhexidine, povidone iodine, or isopropyl alcohol). Although this procedure is not considered sterile, the physician should attempt to keep the area as clean as possible and devoid of unnecessary contamination.

The area is anesthetized with preservative-free 1% to 2% lidocaine. Injection of lidocaine within the abscess cavity has not been shown to alter microbiologic data [59]. After 2 to 5 minutes, when the onset of anesthesia has occurred, using a number 11 scalpel, a straight incision is made over the area of maximal fluctuance. The length of the incision will depend on the size of the abscess; however, in general, the incision ought to be made large enough to promote adequate drainage. After obtaining culture material (if indicated) and the initial decompression of purulent material, the abscess cavity is probed thoroughly using a curved or straight hemostat. This action will further release any pockets of purulent material and break any remaining loculations and adhesions. All necrotic and devitalized tissue is also debrided.

To remove all loosened purulent and necrotic material, the abscess cavity is irrigated with sterile saline, using an 18-gauge angiocatheter attached to a 10-mL syringe. Although it is recommended [58], the additional clinical utility of irrigating the abscess cavity is unknown.

After thorough drainage and removal of purulent material and any devitalized tissue, the abscess cavity is loosely packed using ¼ - or ½ -inch plain packing strips. In order to ensure that the incision site will remain open and allow for continued drainage, 1 cm to 2 cm of the packing material is left extending outside of the wound. The last step involves covering the wound with absorbent 4 × 4 gauze dressing. Patients are instructed to return within a few days for removal of the packing material, or can be instructed to change the packing themselves at home. Some patients will need a prescription of narcotic analgesic for dressing changes.

**Antimicrobial therapy**

The choice of initial empiric antimicrobial therapy is dependent upon prediction of the most likely microbiologic etiology and local antimicrobial susceptibility patterns. Table 4 depicts recommended initial empiric antimicrobial therapy options for commonly encountered SSTIs in the ED.

The mechanism of injury is crucial to predicting the bacterial etiology, which is especially important if organisms are involved that require specific treatment. Examples of acute infections in which mechanism of injury is key include dog and cat bite infections (*Pasteurella* species), human bite...
infections (*Eikenella* species), salt-water (*Vibrio* species) and fresh-water (*Aeromonas* species) exposure.

CA-MRSA is the most common cause of purulent SSTIs in most areas of the United States [12]. It demonstrates variable susceptibility patterns to commonly used agents, such as clindamycin and tetracyclines. In vitro susceptibility patterns of CA-MRSA to a variety of antimicrobial agents are depicted in Table 5 [12,21,23,60]. Antibiotics that have adequate activity against CA-MRSA include trimethoprim/sulfamethoxazole (TMP/SMX), rifampin, vancomycin, linezolid (Zyvox), daptomycin (Cubicin), tigecycline (Tygacil), and quinupristin-dalfopristin (Synercid) [12,21,23,60–68].

Currently, the clinical trials for off-patent antibiotics for uncomplicated SSTIs are being planned with support of the National Institutes of Health. Other potential future MRSA antimicrobials that are currently in various phases of clinical trials for SSTI include novel glycopeptides, such as dalbavancin (administered once weekly), telavancin, oritavancin (LY333328), cefotobiprole (a cephalosporin with anti-MRSA activity), and Iclaprim (formerly AR-100, Ro 48-2622; a specific and selective microbial dihydrofolate reductase inhibitor) [69–73].

The most common type of SSTI associated with CA-MRSA is an abscess [10–12,47,60]. Although further study is needed to determine if antibiotics lead to additional benefit for CA-MRSA abscesses, it appears that 85% to 90% of patients do well with a treatment of incision and drainage alone [12,46–48]. In a recent observational study of 492 adult patients with CA-MRSA SSTIs, the use of an inactive antimicrobial agent was found to be an independent predictor of treatment failure (adjusted OR, 2.80; 95% CI, 1.26–6.22; \( P = .01 \)) [60]. However, of the 45 patients who experienced treatment failure, 38 (84%) were attributed to the need for additional incision and drainage.

In an observational study of 69 children with culture-proven CA-MRSA abscesses, a patient with an abscess greater than or equal to 5 cm in diameter was more likely to require subsequent hospitalization with incision and drainage alone [46]. Based on this study, The Sanford Guide recommends instituting antibiotics for patients with abscesses greater than or equal to 5 cm in diameter [74]. Unfortunately, these findings derive from observational data in which the incision and drainage procedure was not standardized. In the authors’ experience, the most common reason abscesses fail initial management is inadequate incision and drainage, irrespective of the activity of the antibiotic against CA-MRSA, and inadequate drainage is more likely with larger abscesses if there is not careful attention to proper technique.

In the absence of large randomized trials conducted in the area of CA-MRSA, current recommendations on antimicrobial therapy of CA-MRSA SSTIs are based on expert opinion [24]. In conjunction with surgical drainage, the IDSA SSTI guidelines recommend the addition of systemic antimicrobial agents in patients with cutaneous abscesses in the following
Table 4
Recommended initial empiric antimicrobial therapy options for commonly encountered skin and soft-tissue infections in the emergency department

<table>
<thead>
<tr>
<th>Infection</th>
<th>Therapeutic setting</th>
<th>Initial empiric antimicrobial therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mild cellulitis</strong></td>
<td>Outpatient therapy</td>
<td>• TMP/SMX DS (160/800 mg) 1–2 tablets bid plus Cephalexin 500 mg qid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Clindamycin 300 mg qid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Minocycline 100 mg bid (first dose, 200 mg)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Doxycycline 100 mg bid</td>
</tr>
<tr>
<td><strong>Abscess with mild cellulitis</strong></td>
<td>Outpatient therapy</td>
<td>• Incision and drainage (no need for antimicrobial therapy).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Use of outpatient antimicrobial therapy is recommended in patients with multiple lesions, immunocompromised state, those with evidence of mild systemic toxicity (eg, fever), recurrent infections, those who have failed initial surgical therapy (after exclusion of inadequate drainage or deeper abscess), and abscesses that cannot be completely drained.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• TMP/SMX DS (160/800 mg) 1–2 tablets bid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Clindamycin 300 mg qid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Minocycline 100 mg bid (first dose, 200 mg)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Doxycycline 100 mg bid</td>
</tr>
<tr>
<td><strong>Moderate-to-severe cellulitis</strong></td>
<td>Inpatient parenteral therapy</td>
<td>• Clindamycin 600 mg–900 mg IV q 8 h</td>
</tr>
<tr>
<td>or abscess</td>
<td></td>
<td>Monotherapy with clindamycin is preferred only for moderate infections.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Vancomycin 1 g IV q 12 h ± cefazolin 1 g IV q 6 h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May replace cefazolin with nafcillin or oxacillin 1 g–2 g every 4 hours.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Addition of β-lactam agent may provide enhanced activity against MSSA or streptococci (preferred for patients suspected to be bacteremic with S. aureus).</td>
</tr>
<tr>
<td><strong>Diabetic foot infection</strong></td>
<td>Outpatient therapy</td>
<td>• Clindamycin 300 mg qid plus Ciprofloxacin 500 mg bid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Amoxicillin/clavulanate 875/125 mg bid ± TMP/SMX DS (160/800 mg) 1–2 tablets bid</td>
</tr>
</tbody>
</table>

(continued on next page)
situations: multiple lesions, cutaneous gangrene, immunocompromised state, extensive surrounding cellulitis, and those with evidence of systemic toxicity (eg, high fever) [4]. The initiation of antibiotics is also reasonable in patients requiring hospitalization (a reflection of the severity of the disease), recurrent infections, those who have failed initial surgical therapy (after exclusion of inadequate drainage or deeper abscess), and in abscesses associated with unusual pathogens (eg, human bite).

TMP/SMX is a commonly used drug in the United States for outpatient management of CA-MRSA SSTIs. The Sanford Guide recommends two double-strength TMP/SMX tablets twice a day, with the goal of ensuring

### Table 4 (continued)

<table>
<thead>
<tr>
<th>Infection</th>
<th>Therapeutic setting</th>
<th>Initial empiric antimicrobial therapy</th>
</tr>
</thead>
</table>
| Diabetic foot infection       | Inpatient parenteral therapy      | - Ceftriaxone 1 g IV q 24 h plus metronidazole 500 mg IV q 6-8 h ± Vancomycin 1 g IV q 12 h  
|                               |                                   | - Ertapenem 1 g IV q 24 h ± vancomycin 1 g IV q 12 h                                                  |
|                               |                                   | - Tigecycline 50 mg IV q 12 h (first dose, 100 mg IV)                                                 |
| Dog, cat, and human bites     | Outpatient therapy                | - Amoxicillin/clavulanate 875/125 mg bid                                                             |
|                               |                                   | - Moxifloxacin 400 mg daily                                                                         |
|                               |                                   | - Clindamycin 300 mg qid plus Ciprofloxacine 500 mg bid                                                |
| Dog, cat, and human bites     | Inpatient parenteral therapy      | - Ampicillin/sulbactam 1.5 g–3 g IV q 6 h                                                             |
|                               |                                   | - Moxifloxacin 400 mg IV qd                                                                         |
| Necrotizing soft-tissue infections |                                   | - Vancomycin 1 g IV q 12 h plus Clindamycin 900 mg IV q 8 h plus Piperacillin/tazobactam 3.375 g IV q 6 h |
|                               |                                   | May replace vancomycin with daptomycin 4 mg/kg–6 mg/kg IV qd.                                        |
|                               |                                   | Clindamycin (or alternatively linezolid, see below) is recommended because of its ability to inhibit toxin production. |
|                               |                                   | May substitute piperacillin/tazobactam with imipenem or meropenem.                                   |
|                               |                                   | - Linezolid 600 mg IV q 12 h plus Piperacillin/tazobactam 3.375 g IV q 6 h                           |

Recommended dosages are for a non-pregnant 70-kg adult with a normal renal and hepatic function. Antimicrobial therapy should be initiated based on knowledge of local susceptibility patterns and adjusted once culture and susceptibility data are known.

**Abbreviations:** DS, double-strength; IV, intravenous; TMP/SMX, trimethoprim-sulfamethoxazole.
adequate serum levels with respect to the minimum inhibitory concentration (MIC) and maximizing concentration-dependent killing [74]. Although this recommendation seems logical and is made to prevent under-dosing, it should be noted that there are no prospective human trials demonstrating the superiority of a two double-strength, compared with a one double-strength, regimen. TMP/SMX has been shown to have adequate penetration into experimentally made human skin blisters [75,76]; however, the same may not apply to abscesses even with an increased dosage regimen. Most importantly, the issue of penetration into the abscess cavity may be a moot point if they are treated with adequate incision and drainage. Dosage increases may also lead to increased side-effects and potentially lower patient compliance with the advocated regimen.

Rifampin, a highly active agent against CA-MRSA, is commonly used in combination with TMP/SMX or doxycycline. It should not be used alone because of its rapid tendency to select resistant strains [77]. The Sanford Guide recommends the addition of rifampin to TMP/SMX for patients who have an abscess associated with fever, those with large or multiple abscesses, and in severe infections [74]. The only supporting data are from a retrospective study of CA-MRSA SSTIs, in which clinical resolution was achieved in all of six patients treated with a combination of TMP/SMX and rifampin, but in only 6 of 12 patients treated with double-strength TMP/SMX [78]. Rifampin has numerous drug-drug interactions and an

<table>
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<tbody>
<tr>
<td>TMP/SMX</td>
<td>100% (n = 217)</td>
<td>100% (n = 120)</td>
<td>95% (n = 106)</td>
<td>99% (n = 322)</td>
</tr>
<tr>
<td>Rifampin</td>
<td>100% (n = 186)</td>
<td>100% (n = 120)</td>
<td>96% (n = 106)</td>
<td>99% (n = 318)</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>95% (n = 226)</td>
<td>95% (n = 102)</td>
<td>83% (n = 106)</td>
<td>98% (n = 482)</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>92% (n = 226)</td>
<td>81% (n = 120)</td>
<td>92% (n = 106)</td>
<td>93% (n = 455)</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>NT</td>
<td>100% (n = 120)</td>
<td>94% (n = 106)</td>
<td>100% (n = 320)</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>60% (n = 176)</td>
<td>15% (n = 101)</td>
<td>79% (n = 106)</td>
<td>73% (n = 354)</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>6% (n = 226)</td>
<td>7% (n = 120)</td>
<td>44% (n = 106)</td>
<td>5% (n = 23)</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>NT</td>
<td>100% (n = 120)</td>
<td>100% (n = 106)</td>
<td>100% (n = 492)</td>
</tr>
<tr>
<td>Linezolid</td>
<td>NT</td>
<td>100% (n = 19)</td>
<td>NT</td>
<td>NT</td>
</tr>
</tbody>
</table>

Susceptibility patterns are dynamic and may vary markedly by geographic regions. Physicians’ familiarity with the prevalence and susceptibility patterns of CA-MRSA in their community is a crucial element in the management of CA-MRSA infections.

Abbreviations: NT, Not tested; TMP/SMX, Trimethoprim/sulfamethoxazole.

a Four (approximately 2%; n = 226) MRSA isolates had inducible clindamycin resistance detected by an antimicrobial susceptibility D-zone disk diffusion test.

b Two (3%; n = 59) MRSA isolates had inducible clindamycin resistance detected by an antimicrobial susceptibility D-zone disk diffusion test.

unpleasant side-effect profile (eg, discoloration of bodily fluids). In combination with another agent, it introduces complexity and confusion regarding dosing regimens, which in turn may translate into noncompliance. A potential role of rifampin, because of its greater penetration into mucosal tissue, may lie with its ability to eradicate nasal MRSA colonization and potentially reduce the rate of recurrence, but this has not been demonstrated in the setting of CA-MRSA skin infections [79,80].

The activity of clindamycin against CA-MRSA is geographically variable, with a higher prevalence of resistance compared with TMP/SMX. However, compared with TMP/SMX, it has superior activity against S. pyogenes and some anaerobes (eg, Peptostreptococcus), and has the capability of inhibiting toxin production [81]. Some strains of MRSA display inducible clindamycin resistance. Pretherapy, these strains demonstrate in vitro erythromycin-resistant and clindamycin-sensitive susceptibility patterns. However, when exposed to clindamycin, they develop in vitro resistance to clindamycin. This trait can be detected by the double-disk diffusion assay (D test). The prevalence of such strains is geographically variable and the clinical significance is not well understood, as both clinical cure and treatment failures have been reported [82–87]. In a small retrospective study of invasive CA-MRSA infections in children, clindamycin demonstrated clinical efficacy in patients infected with clindamycin-susceptible CA-MRSA isolates [88]. However, because of lack of published experience, clindamycin is not advocated for use as a sole agent in severe infections [89].

Extended-spectrum tetracyclines, such as doxycycline and minocycline, are also reasonable choices for oral agents against CA-MRSA if local isolates display a high susceptibility rate [90,91]. Minocycline may have a slight advantage over doxycycline in its staphylococcal (MSSA) and streptococcal activity [74]. These drugs should be avoided in children under 9 years old, pregnant patients, and nursing mothers.

Vancomycin is the most commonly used intravenous agent for the treatment of MRSA infections. Although vancomycin has been used for many decades without many alternatives, its poor tissue penetration, increasing staphylococcal MICs associated with clinical failure, and inferior clinical efficacy, compared with antistaphylococcal β-lactams in the treatment of MSSA infections, has raised concerns regarding its efficacy relative to newer agents [92–94]. Alternate intravenous agents with FDA approval for the treatment of MRSA SSTIs include linezolid (Zyvox), daptomycin (Cubicin), and tigecycline (Tygacil). Quinupristin-dalfopristin (Synercid) has in vitro activity against MRSA and an indication for treatment of complicated SSTI; however, it does not currently have specific approval for MRSA infections [66–68].

Linezolid has excellent in vitro activity against MRSA [62], and unlike vancomycin has the ability to suppress toxin production [81]. In a randomized, open-label, multicenter study of complicated SSTIs, based on subgroup analysis, linezolid outcomes were statistically superior to vancomycin at the
test-of-cure visit for patients with MRSA infections [63]. However, a reanalysis of this study challenged the differential treatment effect based on the microorganism [95]. Although both daptomycin and tigecycline are FDA-approved for the treatment of complicated SSTIs caused by MRSA, the associated clinical studies are limited by the small number of subjects with documented MRSA SSTIs [96,97].

If indicated, empiric antimicrobial therapy for SSTIs associated with purulence should include agents that have been shown to have adequate in vitro activity against CA-MRSA [12]. The role of CA-MRSA in nonpurulent SSTIs (eg, nonpurulent cellulitis) is unclear. However, in light of the emergence of CA-MRSA in purulent SSTI and the prominent role of MSSA in previous studies of cellulitis, empiric CA-MRSA coverage is recommended. Because streptococci have been shown to be another common cause of cellulitis [25–28,53], it is reasonable to include an agent or agents with in vitro activity against CA-MRSA and \textit{S. pyogenes}.

It should be noted that the clinical efficacy of TMP/SMX for SSTIs caused by Group A streptococci is unclear. In addition, the activity of TMP/SMX is not routinely tested against \textit{S. pyogenes}, and as a result, current resistance rates of \textit{S. pyogenes} to TMP/SMX are unknown. Although there is evidence that TMP/SMX has in vitro activity [98,99] and some clinical efficacy in infections caused by \textit{S. pyogenes} [100,101], the most recent Centers for Disease Control and Prevention guidelines do not advocate use of this agent as monotherapy for patients presenting with cellulitis [24].

Empiric antimicrobial therapy for infected dog and cat bite wounds should include coverage against \textit{Pasteurella}, streptococci, staphylococci, and anaerobic species [5]. For infected human bites, empiric antimicrobial therapy should include coverage against \textit{Eikenella}, streptococci, staphylococci, and anaerobic species [6]. \textit{S. aureus} has been found in 4%, 20%, and 30% of cat, dog, and human bite infections, respectively, and none were MRSA [5,6]. Although these studies were done before the emergency of CA-MRSA, at the present time colonization rates in human beings remain low, so empiric CA-MRSA coverage is not recommended in these types of infections [102].

Because of inadequate activity against \textit{Pasteurella} or \textit{Eikenella} species, monotherapy with first-generation cephalosporins, dicloxacillin, erythromycin, clindamycin, and metronidazole should be avoided [103]. The combination therapy of penicillin plus a cephalosporin has been advocated for the initial treatment of human bites [51]. However, this combination, especially if a first-generation cephalosporin is used, does not cover β-lactamase producing anaerobes [6]. Empirical regimens for marine- and freshwater-acquired infection, should cover \textit{Vibrio} and \textit{Aeromonas} species, respectively, with agents such as third-generation cephalosporins (eg, cefotaxime) and fluoroquinolones.

The management of necrotizing SSTI in the ED involves aggressive resuscitation, hemodynamic stabilization, initiation of antibiotics, and surgical
consultation. Broad-spectrum empiric antimicrobial therapy should be initiated as soon as possible once the diagnosis is suspected. The spectrum of antibiotic coverage should include gram-positive, gram-negative, and anaerobic organisms, with special attention to resistant organisms (eg, CA-MRSA) [13]. Suitable multidrug regimens include vancomycin plus piperacillin or tazobactam plus clindamycin. Clindamycin have been shown to inhibit toxin production with streptococcal and clostridial infections [104–106], which in turn may be an important intervention in controlling the inflammatory response. One observational study of children with invasive streptococcal infections found superior clinical outcomes in patients receiving antibiotics inhibiting protein synthesis, compared with agents active at the cell wall [107]. In a more recent study, clindamycin and linezolid, both protein synthesis inhibitors at the ribosomal level, were shown to have the capability to suppress toxin production in CA-MRSA strains [81].

The optimal duration of antimicrobial therapy of SSTI is unknown and is ultimately dependent on how the infection responds. Most SSTIs are treated with a 7 to 10 day course of antimicrobial therapy. A shorter course of 3 to 5 days may be appropriate for infections associated with a drainable abscess, though this recommendation is not based on any comparative studies. Complicated cases, such as infections associated with osteomyelitis, an immunocompromising condition, or diminished vascular supply may require a longer duration of therapy. In a randomized, double-blind, placebo-controlled trial of 87 subjects with uncomplicated cellulitis, there was no significant difference in clinical outcome at 14 and 28 days of therapy among subjects who were treated with 5 days, compared with 10 days, of antimicrobial therapy [108]. In a recent retrospective cohort study of 492 subjects with CA-MRSA SSTIs, subjects who had a successful outcome received antibiotics for a median duration of 10 days (interquartile range, 7–14 days) [60].

**Disposition**

The majority of patients with SSTI can be treated as outpatients. Hospitalization is indicated for patients with hemodynamic instability, altered mental status, severe infection (including those requiring formal operative intervention), intractable nausea and vomiting, the presence of immunocompromising infection, failure of outpatient therapy, and poor social support. The latest IDSA SSTI guidelines indicate hospitalization should be considered in patients with “hypotension and/or an elevated creatinine level, low serum bicarbonate level, elevated creatine phosphokinase level (2–3 times the upper limit of normal), marked left shift, or a C-reactive protein level >13 mg/L” [4].

Studies of risk factors for mortality, complications, and treatment failures in patients with SSTIs exist [109–111]; however, only a few small studies have attempted to determine a set of variables that may predict the need for
(and benefit from) hospitalization [112]. In a study of patients with extremity cellulitis, independent predictors of “need” for hospital admission were a history of diabetes, temperature, hand infections, induration, area greater than 70 cm$^2$, and the absence of fluctuance. The need for hospitalization was defined as hospital stay greater than 24 hours, operative incision and drainage, or failed outpatient management [112].

In an expert panel recommendation on the management of SSTIs [1], the panel classified patients with SSTIs into four classes: Class I was afebrile and healthy, other than cellulitis; Class 2 was febrile and ill-appearing, but with no unstable comorbidies; Class III was toxic appearing, or with at least one unstable comorbidity or a limb-threatening infection; and Class IV was with a sepsis syndrome or life-threatening infection (eg, necrotizing fasciitis).

For Class I patients, outpatient care on oral antimicrobials was recommended. For the Class IV patients, hospital admission was recommended. For both the Class II and Class III patients, a period of observation, and depending on the outcome, either outpatient care on oral or intravenous (home or infusion center delivered) antimicrobials or hospital admission was recommended. The proposed classification system does not address the most important question as to which patients actually achieve benefit from hospitalization. Most EDs do not have observation units. In addition, for most patients and settings, outpatient parenteral antimicrobial therapy is not feasible or practical.

Some EDs may have observation units that are either run by emergency physicians or general internists. SSTIs are among the most common reason for admission to an observation unit [113]. For ED physicians, it is not unusual to encounter patients who initially present with worrisome clinical findings and equivocal criteria for hospital admission, but after a few simple interventions (eg, intravenous fluids, antipyretics) and a period of observation, they become suitable for outpatient therapy. In some instances, if feasible, patients are brought back to the ED for a few additional doses of intravenous antibiotics, or are treated at home with intravenous antimicrobial therapy [114,115].

For the appropriate indication, dalbavancin (administered in two doses, 1 week apart [69]) may provide a potentially attractive alternative and avert hospitalization for otherwise stable patients who are thought to require parenteral antibiotic therapy or for whom noncompliance is a concern.

**Infection control**

Since the majority of SSTIs presenting to the ED are likely to be caused by CA-MRSA [12], it would be ideal for all these patients to be placed in private rooms with MRSA contact precautions until culture results are available. However, many EDs are currently dealing with severe overcrowding issues, and isolation capacity is extremely limited. Similarly, because of
a limited number of isolation beds in the hospitals, such patients often endure long waits for admission, which in turn results in delays in ED patient flow and disposition. Rapid real-time polymerase chain reaction assay for MRSA may allow rapid identification of these patients and in turn better facilitate infection control measures [116].

MRSA isolation precautions that are used in most hospitals were developed in the era of hospital-associated MRSA. Their utility in the era in which CA-MRSA is prevalent in the community at large is unclear. However, it is known that CA-MRSA strains can spread within hospitals [117]. A common scenario in many facilities is that SSTI patients are admitted without MRSA precautions, then placed in isolation 2 days later when MRSA is confirmed. Although this scenario is clearly suboptimal, it may be impractical to admit every SSTI patient to a private room with MRSA precautions. Because it is known that MRSA is now a likely cause of SSTI, a cohorting strategy for admitted SSTI patients may help reduce nosocomial spread [118]. Whether the infection is caused by MRSA or MSSA, standard precautions should be used for any patient with a purulent wound to prevent exposing other patients or personnel to infected material. Gloves should always be used when handling purulent material, such as when performing incision and drainage or changing dressings of infected wounds. Gowns and eye protection should be used for procedures that are likely to generate splashes or sprays of fluids [24]. Frequent handwashing should always be encouraged in the ED.

MRSA decolonization should be considered in patients with recurrent, active MRSA infections not responding to appropriate therapy, or MRSA infection occurring in closely-associated cohorts (eg, MRSA infection in a family) [24]. Although the efficacy of commonly prescribed decolonization regimens in the prevention of recurrent CA-MRSA skin infections has not been studied, commonly prescribed agents for the purpose of decolonization in the United States includes mupirocin 2% nasal ointment plus chlorhexidine 4% skin cleanser. In a randomized controlled trail, treatment with topical mupirocin, chlorhexidine gluconate washes, oral rifampin, and doxycycline for 7 days was safe and effective in eradicating MRSA colonization in hospitalized patients for at least 3 months [80].

Proper decolonization practices require obtaining cultures from multiple body sites (eg, nares, axilla, groin), performing special susceptibility testing (eg, mupirocin) [119], and educating and possibly treating the patient’s family or other close contacts [24]. EDs may not be the optimal place to initiate these interventions. However, many patients do not have access to primary care physicians and EDs are the only site for their medical care. Initiating a decolonization regimen, in selected cases, without obtaining cultures or special susceptibility testing and not involving the family is not uncommon. Although this practice may not be the optimal or preferred way, it is often the only opportunity to initiate a potential therapy for patients who have limited amount of resources and poor follow-up capability.
Summary

The most important new development in the area of SSTI is the increased prevalence of CA-MRSA, a phenomenon largely recognized and studied in ED populations. There are no clinical or epidemiologic risk factors that can reliably exclude MRSA. The emergence of CA-MRSA has made a significant impact in the empiric treatment of SSTIs.

The misclassification of a deep abscess as cellulitis is a common pitfall. The presence of an underlying deep abscess should be considered in patients with cellulitis who fail initial antimicrobial therapy. Treatment failure may be caused by an undrained abscess that was missed on the initial presentation, rather than inadequate antimicrobial therapy. Ultrasonography can be useful in the identification of deep abscesses, especially in cases in which the physical examination is equivocal or there is a broad area of what appears to be cellulitis. Wound and blood cultures can be of value in selected patients with SSTIs; however, their routine performance, clinical utility, and cost-effectiveness in all types of SSTIs are debatable.

The recognition of a necrotizing SSTI, especially in the early stages, is extremely difficult. The utility of the LRINEC score is limited in that it needs to be prospectively validated in patients for whom the diagnosis of necrotizing SSTI is not apparent on initial history and physical examination. Protein synthesis inhibitors (eg, clindamycin, linezolid) have a potential role in the management of necrotizing SSTIs. The majority of patients with SSTI can be treated as outpatients. Prospectively derived sets of variables that may predict the need for (and benefit from) hospitalization in patients with SSTIs are insufficient at this time, and further studies in this area are indicated.

References


[48] Rajendran PM, Young D, Maurer T, et al. Randomized, double-blind, placebo-controlled trial of cephalexin for treatment of uncomplicated skin abscesses in a population at risk for


