Skin and Soft Tissue Infections
Robert L. Rogers, MD, FAAEM, FACEP, FACP*, Jack Perkins, MD

Department of Emergency Medicine, The University of Maryland School of Medicine, 110 South Paca Street, Suite 200, Emergency Medicine, Sixth Floor, Baltimore, MD 21201, USA

Skin and soft tissue infections represent a continuum of symptoms that range from uncomplicated cellulitis to the potentially lethal entity necrotizing fasciitis (NF). The primary care physician will see a myriad of infectious skin disorders in the outpatient setting, and must be capable of discerning which presentations warrant emergency department evaluation and inpatient admission. This article will highlight three entities under the broad umbrella of skin and soft tissue infections. Cellulitis, cutaneous abscess, and NF present not only to the emergency department but also to the outpatient setting. This article aims to help primary physicians recognize patterns of disease that herald significant illness and the need for more extensive evaluation than an outpatient setting can offer.

This article discusses the etiology, presentation, evaluation, and management of cellulitis, cutaneous abscess, and NF. Particular attention will be spent addressing the emerging problem with community-acquired methicillin-resistant Staphylococcus aureus (ca-MRSA). Community-acquired MRSA has significantly changed the emergency department approach to cellulitis, and has altered the spectrum of antimicrobials that are used for outpatient treatment of cellulitis. Familiarity with ca-MRSA is essential for the primary care physician, because misdiagnosis and improper antibiotic selection can lead to significant morbidity and mortality [1].

Cellulitis

In 1996, cellulitis was estimated as the 28th most common discharge diagnosis in the United States. The actual incidence and prevalence are
difficult to estimate due to the number of outpatient presentations; however, one study reported that cellulitis was responsible for 2.2% of outpatient office visits [2]. Cellulitis is defined as an infection of the dermis with variable extension into the subcutaneous tissues [2]. It usually develops on extremities, but has been documented on every area of the body. Cellulitis generally occurs after the protective barrier of the skin, the epidermis, has been compromised (eg, trauma, ulcers, eczema) allowing bacterial access to the subepidermal tissues. Often, the portal of bacterial entry is unknown. The predilection of cellulitis to occur on areas exposed to the environment (eg, hands, feet) supports the theory that disruption of the epidermis is essential in the development of cellulitis. Factors that predispose to cellulitis include *tinea pedis*, diabetes mellitus, peripheral vascular disease, peripheral edema, and prior history of cellulitis [3].

**Bacterial etiology**

The majority of cases of cellulitis are caused by *beta-hemolytic streptococci*, mostly from subtypes A and B. *S aureus*, and more recently ca-MRSA, have become increasingly prominent pathogens throughout the United States. Other causes of cellulitis are associated with specific clinical scenarios and are summarized in Table 1 [5–7].

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Cellulitis key clinical features organism</th>
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<tbody>
<tr>
<td>Periorbital cellulitis</td>
<td>Must be distinguished from Strep and staph in adults, H. influenza in children</td>
</tr>
<tr>
<td>Orbital cellulitis</td>
<td>Orbital cellulitis Strep, staph</td>
</tr>
<tr>
<td>Perianal cellulitis</td>
<td>Must evaluate for underlying Group A strep (GAS)</td>
</tr>
<tr>
<td>Crepitant cellulitis</td>
<td>May represent necrotizing fasciitis GAS, Anaerobes, Clostridia</td>
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<tr>
<td>Puncture wound</td>
<td>Often in plantar aspect of foot Pseudomonas</td>
</tr>
<tr>
<td>Salt water exposure</td>
<td>History of exposure is key element Vibrio vulnificus</td>
</tr>
<tr>
<td>Fresh water exposure</td>
<td>History of exposure is key element Aeromonas hydrophilia</td>
</tr>
<tr>
<td>Dog bite/Cat bite</td>
<td>Look for puncture wound Pasteurella multocida</td>
</tr>
<tr>
<td>Cat Scratch</td>
<td>Look for proximal lymphadenopathy Bartonella henselae</td>
</tr>
<tr>
<td>Human bite</td>
<td>Do NOT suture closed Anaerobes, Strep pyogenes, Eikenella corrodens</td>
</tr>
<tr>
<td>Calf cellulitis</td>
<td>Suspect underlying DVT Multiple Organisms</td>
</tr>
<tr>
<td>Foot cellulitis</td>
<td>Suspect origin as <em>Tinea Pedis</em> Multiple organisms</td>
</tr>
<tr>
<td>Cellulitis in infants</td>
<td>Often hematogenous spread; consider Predominantly Group B</td>
</tr>
<tr>
<td>Immunocompromised</td>
<td>HIV, Cancer patients Strep</td>
</tr>
<tr>
<td>Cell mediated immunity dysfunction</td>
<td>HIV, Leukemia patients, may be disseminated Cryptococcus neoformans</td>
</tr>
<tr>
<td>Hot tub exposure</td>
<td>Cellulitis in bathing suit distribution Pseudomonas aeruginosa</td>
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**Presentation**

Cellulitis can develop on any area of the body, but is most likely to occur on extremities that are susceptible to microtrauma. The hallmarks of cellulitis include erythema, warmth, swelling, and tenderness of the affected area. The absence of all of these signs would make cellulitis a highly improbable diagnosis [2]. The erythema in cellulitis is generally confluent, and leading edges tend to be poorly demarcated. Sharply demarcated borders are more indicative of *erysipelas*, which is a distinct form of cellulitis due almost exclusively to *beta-hemolytic streptococci* [4]. Other clinical features of cellulitis include tender lymphadenopathy and occasional abscess formation, especially if *S aureus* is the causative organism. Although systemic features such as fever, chills, or rigors may be associated with cellulitis, evidence of systemic toxicity should raise suspicion for a more serious pathology such as NF or hematogenous dissemination of the cellulitic organism.

**Laboratory and radiographic evaluation**

Laboratory investigation of cellulitis is of limited value because it is unusual to definitively establish a causative organism [2]. Blood culture results are positive in less than 5% of patients [5]. Blood cultures are frequently obtained when patients are admitted to the hospital for cellulitis; however, they just as often represent contamination as a true pathogen. Sending a complete blood count or any other laboratory studies are not recommended in the outpatient setting, and have limited value in the inpatient setting. Occasionally, consideration is given to a punch biopsy of the cellulitic area; however, the results can be quite variable, with culture identifying a likely organism 5% to 40% of the time, depending on the patient population [5].

Radiographic evaluation is recommended in the diagnostic workup of cellulitis only when there is a question of whether another process may be involved. Occasionally, the clinical examination may suggest an occult abscess or perhaps NF. In such cases, it is reasonable to consider plain films to evaluate for gas in the soft tissue or even a CT scan of the area in question with intravenous contrast to evaluate for abscess or muscle involvement. Plain films are also indicated if a retained foreign body is considered as the source of the overlying cellulitis (see Fig. 1).

**Community acquired MRSA**

MRSA infections began in the early 1960s, a few years after methicillin was introduced to combat the problem of resistance to penicillin [8]. By the 1980s, MRSA had become a common problem throughout US hospitals. In the 1990s, patients who had no risk factors for hospital-acquired MRSA infection (eg, recent hospitalization, intravenous drug use, hemodialysis, or resident in a chronic care facility) were developing cellulitis from MRSA [9]. Interestingly, this strain of MRSA was susceptible to a broader range of antibiotics.
than hospital-associated or -acquired MRSA. This new strain of MRSA was given the name ca-MRSA or community-associated MRSA. The incidence and prevalence of ca-MRSA has risen dramatically over the past decade, and its emergence has changed clinical practice in outpatient settings, urgent care facilities, emergency departments, and inpatient hospital settings.

Although anyone may become colonized or present with an illness due to ca-MRSA, there appears to be certain populations that are more susceptible to outbreaks. Prisoners, children (especially in daycare centers), intravenous drug users, sports participants, soldiers, homosexual men, and homeless individuals are at particular risk of ca-MRSA colonization and infection [1]. All of these at-risk populations share the similarities of large numbers of people who convene or habitate in close proximity to others where hygiene may be difficult to maintain. There also seems to be a preponderance of ca-MRSA among children. A few studies have reported ca-MRSA isolates in up to 50% of children in whom \( S \text{ aureus} \) is found on routine colonization cultures [10].

Clinical manifestations of ca-MRSA infection are varied, and include skin and soft tissue infections, osteomyelitis, pneumonia, joint infection, and sepsis. The spectrum of disease presentations can be diverse. Cellulitis is a common presentation of ca-MRSA although it appears less prevalent than abscess formation with or without associated cellulitis. Any patient who presents with cellulitis and an abscess should be considered to have ca-MRSA until proven otherwise. Patients who have cutaneous abscesses due to ca-MRSA tend to have frequent recurrences, and family members are prone to developing similar symptoms. ca-MRSA has recently been reported in association with NF [11]. These case reports are interesting in that ca-MRSA was the only organism isolated in what is generally a polymicrobial infection. Osteomyelitis, septic joints, and severe community-acquired pneumonias have all been documented [1,9].

Mortality in ca-MRSA pneumonia was close to 25% in one study, although most of the patients were previously healthy hosts [11]. Morbidity and mortality are dependent upon proper antibiotic selection, which makes
it essential for the primary care provider to consider ca-MRSA as a possible pathogen in all skin or soft tissue infections presenting to the outpatient setting.

Treatment

The treatment of cellulitis has changed significantly in the past several years. Cephalexin (Keflex), amoxicillin-clavulanate (Augmentin), and dicloxacillin (Dynapen) have been staples of treatment for outpatient cellulitis in emergency departments, urgent care, and primary care clinics for many years. The emergence of ca-MRSA has led to a significant increase in the number of treatment failures for skin and soft tissue infections seen in the emergency department. The most important aspect of selecting an appropriate antibiotic involves being familiar with the patient population (eg, intravenous drug users, prisoners) and the susceptibility patterns in the area. Current infectious disease guidelines still recommend beta-lactam (eg, cephalexin, dicloxacillin) antibiotics as first-line treatment for cellulitis that will not require admission [1,12]. Those patients who are more likely to have ca-MRSA as a cause of their cellulitis (eg, high risk populations or cellulitis associated with abscess) should receive trimethoprim–sulfamethoxazole (TMP-SMZ), clindamycin, doxycycline, minocycline, or third- or fourth-generation fluoroquinolones. Patients who have significant comorbid illnesses such as diabetes mellitus, congestive heart failure, or who are on dialysis should be given agents that will cover ca-MRSA as they may not tolerate 24 to 48 hours of improper antibiotic selection. Recently, clindamycin-inducible resistance has become a problem in some areas, prompting some clinicians to select TMP-SMZ as their initial choice when they suspect ca-MRSA is the pathogen. Clindamycin resistance should be suspected when a ca-MRSA susceptibility pattern reveals sensitivity to clindamycin but resistance to erythromycin. This indicates a high likelihood of “inducible clindamycin resistance” [8]. TMP-SMZ failures are rare when treating cellulitis, usually only occurring with isolates of methicillin-sensitive S. aureus (MSSA) [1,5]. No definitive data supports the concept that administration of a single dose of an intravenous antibiotics before discharge from the emergency department or from outpatient clinic has any benefit over oral therapy alone [13]. If patients are well enough to be sent home for treatment, they are likely well enough for oral antibiotic therapy.

Admission to the hospital and administration of parenteral antibiotics are recommended for patients with systemic symptoms (eg, fever, rigor, emesis), unstable vital signs (eg, hypotension, tachycardia), or those who have failed an appropriate course of oral antibiotic therapy that should have covered the suspected pathogen [1,5,12]. Consideration should also be given to patients who have serious comorbid conditions (diabetes, congestive heart failure, end-stage renal disease) or those who are immunocompromised from cancer, organ transplantation, or HIV infection.
Patients admitted to the hospital should receive parenteral antibiotics. Many authorities conclude that vancomycin is the medication of choice for skin and soft tissue infections when resistant *S. aureus* is considered as a possible pathogen. Cultures from the blood and any wounds should be obtained in these circumstances to try to isolate a pathogen. Other appropriate inpatient medications include quinupristin–dalfopristin (Synercid), linezolid (Zyvox), or daptomycin (Cubicin). All of these antibiotics have strong activity against both hospital-acquired MRSA and ca-MRSA. These medications are prohibitively expensive for routine use, but are appropriate when vancomycin-resistant enterococcus is involved or when the patient has a vancomycin allergy.

**Cutaneous abscess**

The incidence and prevalence of skin abscesses has risen in parallel with the emergence of ca-MRSA [5,14]. The diagnosis of a cutaneous abscess is usually not difficult, and often the patient may complain of spontaneous drainage. The treatment of these abscesses frequently involves incision and drainage. Because this is considered a minor surgical procedure, some primary care physicians will choose to refer for definitive management. In addition, procedures such as these may not be possible in the outpatient setting due to time constraints. The primary care office setting is, however, an ideal location for subsequent follow-up evaluation after a patient has undergone incision and drainage. Patients with these disorders need close follow-up, and primary care physicians are in a perfect position to play a pivotal role in the patient’s recovery.

**Microbiology and clinical features**

*S. aureus* is by far the most common pathogen involved in cutaneous abscess formation [15]. Roughly 50% of cutaneous abscesses are due to *S. aureus*; however, most abscesses are not cultured. Treatment is empirically directed against this organism [14]. Most skin abscesses result from minor trauma, although in innercity populations, injection drug use is an extremely common portal of entry, especially for ca-MRSA. Occasionally, skin abscesses will be the result of bacteremia. Risk factors for recurrent abscess formation include obesity, diabetes mellitus, corticosteroid use, neutrophil dysfunction, and injection drug use [16].

Presentation of a skin abscess almost always involves pain, swelling, warmth, and erythema [14]. Local lymphadenopathy and signs of systemic involvement (eg, fever, chills) are unusual unless there is an associated cellulitis. Furuncles are a specific type of cutaneous abscess that occur around hair follicles, and are more common in African American populations. Carbuncles are defined as a series of connected furuncles, and are most commonly found in the posterior scalp area. Hidradenitis suppurativa is an
inflammatory condition where recurrent abscess formation occurs in the buttocks or axillary area largely in connection with excessive perspiration and obesity. Abscesses often drain spontaneously before presentation to the clinic or emergency department, but occasionally may involve deeper tissues requiring CT imaging or ultrasound for diagnosis.

**Treatment**

The most important aspect of abscess treatment is drainage of the fluid collection [17,18]. Spontaneous drainage makes the decision for further incision rather straightforward; however, many abscesses will not spontaneously drain, nor will they be pointing and fluctuant. A recommended tactical maneuver to evaluate for a fluid collection is the insertion of a small bore needle (eg, 22 or 25 gauge) into the area of maximum induration and apparent fluctuance. Return of pus indicates that incision and drainage is indicated [14]. The initial incision is best made by use of a #11 blade scalpel. The tip of the scalpel is aimed at the center of fluctuance and directed perpendicular to the skin surface. The incision should be deep enough to fully evacuate the fluid collection. In addition, loculations should be probed and broken up with the use of a hemostat if possible [17,18]. If there is any question about whether the patient is to be admitted, or if they have failed previous antibiotic therapy, a culture of the fluid should be obtained to help identify a causative organism. The cavity should be irrigated after drainage and loosely packed with iodoform gauze. Often, pain is a rate-limiting factor of debridement, especially in intravenous drug users who may have increased tolerance to narcotic analgesics. As a rule of thumb, lidocaine does not work efficiently in acidic environments such as in areas of pus; thus, lidocaine (with epinephrine if in areas other than ears, fingers, toes, nose, or genitalia) should be injected generously throughout the surrounding tissue and in the subcutaneous area overlying the abscess [19]. Patients will sometimes present with an abscess that is not ready for incision and drainage. Induration may be present, but no discrete area of fluctuance may be appreciated. In these cases, antibiotics and warm compresses to the area are indicated to try to bring the abscess to a head for drainage. Although antibiotic therapy has never been shown to be helpful for an uncomplicated abscess, the induration that often precedes discreet abscess formation can be confused for uncomplicated cellulitis. Follow-up should be arranged in 24 to 48 hours, regardless of whether the abscess is drained, so that healing can be evaluated, or to see if drainage is now indicated. Risks of incision and drainage include bleeding, pain at the incision site, scar formation, and bacteremia [18].

The incidence of bacteremia following surgical incision of an abscess is unclear, as data is conflicting. The American Heart Association recommends one intravenous dose of antibiotics to those who are at moderate to high risk for bacterial endocarditis [20,21]. These patients would include
those with any type of implanted cardiovascular device, such as stents, grafts, porcine, or mechanical valves. A dose of an antistaphylococcal antibiotic such as cefazolin or nafcillin would be appropriate [20,21].

There has always been some controversy regarding whether antibiotics need to be administered following debridement of an abscess. In those patients who have no evidence of systemic infection and no surrounding cellulitis, antibiotics are not indicated. Available evidence supports withholding antibiotics in these clinical circumstances. Patients who have systemic symptoms, have surrounding cellulitis, are immunocompromised (e.g., diabetes mellitus, HIV), or whose abscess is not ready for drainage should receive an antistaphylococcal antibiotic such as cephalexin (Keflex) or dicloxacillin (Dynapen). However, strong consideration should be given to the presence of ca-MRSA in at risk populations. If ca-MRSA is a possible cause of the abscess and surrounding soft tissue infection, TMP-SMZ, doxycycline, clindamycin, or a third- or fourth-generation fluoroquinolone should be considered [22]. In general, a 7-day course of antibiotics is considered sufficient [17,18]. Abscesses in the oral, perirectal, or genital areas should be considered multibacterial in etiology, and consideration should be given to using agents with Gram-positive, Gram-negative, and anaerobic activity [23]. Suitable medications would include amoxicillin–clavulanate (Augmentin) or third- or fourth-generation fluoroquinolones. The importance of follow-up for any patient who has been treated for skin and soft tissue infections cannot be overstated. The presence of ca-MRSA has significantly increased the number of treatment failures. As many as 61% of ca-MRSA infections are initially treated inappropriately with beta-lactam antibiotics. This may lead to an increased morbidity and mortality rate [14,24].

Necrotizing fasciitis

NF is a rare clinical entity accounting for only 500 to 1500 cases of severe soft tissue infections annually in the United States [25]. Although it is a relatively rare clinical entity, its enigmatic presentation and overlap with other more benign skin and soft tissue infections make this a frequently missed diagnosis. The clinical significance of recognizing this disease process early in its course is crucial because mortality is estimated at 24% to 34%. In addition, the associated morbidity (eg, amputations, need for dialysis, multiple surgical procedures, prolonged hospital courses) in those who survive is quite high [25,26]. Primary care physicians may never encounter NF in their clinical careers; however, it is a diagnosis that cannot be missed, as prompt surgical intervention can significantly affect patient outcome. Any case of suspected NF should be referred immediately to the nearest appropriate center that has the capability of caring for the patient. In many cases, the patient will be transferred to the nearest emergency department for surgical evaluation.
Microbiology and clinical features

NF is defined as a rapidly progressing soft tissue infection with fulminant tissue destruction, rapid bacterial spread along tissue planes, thrombosis of blood vessels, systemic signs of toxicity, and high rates of morbidity and mortality [27]. NF is typically divided into two separate categories based on involved organisms. Type I NF is the less common variety, and is due to infection with aerobic and anaerobic bacteria. Frequently, Type I NF is a mixed bacterial infection. Nearly 66% of cases in one study were attributed to a combination of aerobic and anaerobic bacteria [25]. Common isolates from Type I NF include *streptococcus* (other than Group A, which causes Type II NF), *Staphylococcus aureus*, *enterococcus*, *peptostreptococcus*, *Escherichia coli*, *Bacteroides fragilis*, and *Clostridium* species. Type I NF includes Fournier’s gangrene, which involves the perineal area and can expand rapidly to involve the genitalia and anterior abdominal wall. Patients at risk for Type I NF include diabetics, those with surgical wounds, peripheral vascular disease, alcohol abuse, obesity, hypoalbuminemia (used as a clinical marker of malnutrition) and illicit drug users who “skin pop” or inject into the muscle [25,26].

Type II NF is a monomicrobial infection caused almost exclusively by *group A streptococcus* (GAS or streptococcus pyogenes) [27]. In the past few years, cases of NF caused by MRSA (mostly ca-MRSA) have been reported [8,28]. The incidence of Type II NF infections increased in the 1990s for unknown reasons. It differs from Type I NF in that any age group is a susceptible host for infection. Some predisposing risk factors include injection drug use, surgical procedures, burns, surgical wounds, blunt trauma, varicella, and even perhaps nonsteroidal anti-inflammatory medications [26,27]. It is hypothesized that hematogenous spread of GAS from a pharyngeal source to the site of muscle or soft tissue injury is one possible explanation for the development of NF. Type II NF is often associated with streptococcal toxic shock syndrome, which is heralded by multiorgan failure and severe shock.

The most important clinical symptom of NF is that patients often report pain that is out of proportion to findings during the clinical examination [29]. Patients may complain of extreme pain in an area with few or no cutaneous manifestations. The presence of warmth, erythema, tenderness, or skin discoloration is non-specific, and their presence or absence does not help significantly in securing a diagnosis of NF. However, the development of bullae, especially hemorrhagic bullae, should be taken as an ominous clinical finding, and should prompt consideration of an aggressive soft tissue infection. Crepitus has often been heralded as a strong clinical sign of NF, but this is only found in 10% of all cases of NF [27]. Fever, malaise, myalgias, rigors, tachycardia, and hypotension may all be seen at presentation or develop in the first 24 hours of the clinical course. Because NF spreads rapidly along tissue planes, one may see rapid progression of erythema or significant
change in other clinical symptoms or signs (e.g., tachycardia, pain) only hours after presentation (see Fig. 2, 3, and 4 for examples of NF). The key take-home point is to remember to be hypervigilant about skin and soft tissue infections, and to always consider NF in the differential diagnosis. Failure to do so may prove to be disastrous for the patient.

**Laboratory and radiographic evaluation**

NF, as opposed to uncomplicated cellulitis, necessitates casting a wider diagnostic net. Laboratory analysis as well as prompt radiographic evaluation, is indicated if NF is a possibility. A peripheral white blood count is frequently elevated, and is typically accompanied by a marked left shift. Hyponatremia, elevated BUN and creatinine, hypocalcemia, hypoalbuminemia, and hyperglycemia are found quite often in cases of NF. One study reported that the combination of a white blood cell count $> 14,000$ cells/mL and serum sodium $< 135$ mEq/L resulted in a 90% sensitivity and specificity of 76% for distinguishing between necrotizing and nonnecrotizing soft tissue infections [25]. Blood cultures are positive in 60% of cases of Type II NF due to the rapid ability of group A strep (GAS) to grow in available culture medium [27]. Type I NF has a lower return of positive cultures due to more fastidious organisms being involved. An elevated lactic acid level reflects the presence of anaerobic metabolism in the involved tissue and muscle. Elevated creatine phosphokinase levels are also a common finding that indicates muscle tissue death. An arterial blood gas will likely reveal a metabolic acidosis (elevated lactic acid level) and perhaps a compensatory respiratory alkalosis. Disseminated intravascular coagulation (elevated prothrombin time [PT], partial thromboplastin time [PTT], international normalized ratio [INR], D-dimer, fibrin split products) can accompany NF, especially if caused by GAS or streptococcal toxic shock syndrome.

Radiographic imaging is indicated as soon as NF is suspected to look for evidence of subcutaneous gas. This finding, however, only has a sensitivity of

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Fig. 2. Lower extremity necrotizing fasciitis. (Copyright © Challenger Corporation (Memphis, TN). 2005. All rights reserved.)
39%, specificity of 95%, and negative predictive value of 88% [25]. CT is superior to plain film for detection of gas. Additionally, CT can reveal fluid collections, muscle necrosis, fat stranding, and fascial thickening suggestive of NF. The potential problem with CT is that it requires the patient to leave the resuscitation area. Also, evaluating for an abscess or fluid collection requires the administration of intravenous dye. This could lead to the development of contrast nephropathy, especially if the patient is already hypotensive. MRI has even higher sensitivity and specificity than CT; however, it is time consuming and will delay definitive treatment.

Treatment

The treatment of NF can be considered similar to the philosophy that underlies the treatment of an acute ST elevation myocardial infarction, "time equals tissue." No antibiotic or supportive measure will be an adequate substitute for definitive operative debridement. The role of the primary care physician is to identify that NF is a possible diagnosis and refer for rapid,
definitive treatment. The key mistake made in outpatient clinical practice is assigning a benign diagnosis, such as cellulitis. Again, evidence of systemic toxicity, pain out of proportion, or presence of bullae or crepitus, should be considered NF until proven otherwise.

Antibiotics play a key supportive role in treatment but do not provide the same definitive therapy as they do in cellulitis. Intravenous broad-spectrum antibiotics are indicated for either type of NF, and early administration of antibiotics is recommended. An extended spectrum beta-lactam penicillin (eg, piperacillin/tazobactam, ampicillin/sulbactam), combined with clindamycin, is noted as the regimen of choice from most trials [27,30]. Clindamycin is important, as it has been established as effective in suppression of bacterial toxin production (especially GAS toxin). It also enhances synthesis of penicillin-binding protein [29]. Other supportive measures such as aggressive fluid resuscitation and invasive monitoring are important adjunctive measures [30,31].

Summary

The spectrum of skin and soft tissue infections seen by the primary care physician can be as benign as folliculitis to as life-threatening as NF. Cellulitis remains the most common skin and soft tissue infection seen in primary care. The ever present danger of ca-MRSA, however, has changed the way primary care physicians approach the common problem of cellulitis. The presence of risk factors for colonization with ca-MRSA and a history or examination finding of skin abscess should raise the suspicion of ca-MRSA, and antibiotic therapy should include TMP-SMZ, clindamycin, doxycycline, or minocycline. Skin abscess may occur independently of cellulitis, and often may safely be incised and drained in the primary care setting as long as timely follow-up is assured to assess for wound healing. Available evidence suggests that abscess formation without accompanying cellulitis does not require oral antibiotic therapy. Finally, although NF is rare as an outpatient clinical presentation, it is a diagnosis that the primary care physician should be familiar with. Failure to consider the diagnosis and refer may lead to significant morbidity and even mortality.

References

SKIN AND SOFT TISSUE INFECTIONS


