These data are summarized in Figure 137-1.

Both is unknown. Intriguingly, whereas CA-MRSA is now found in all populations of patients and in health care and community settings alike, the epidemic of skin infections it has caused is most apparent among ED patients for reasons that remain unclear. 6

The number of ED visits for skin and soft tissue infections increased from 1.2 million in 1993 to more than 3.4 million in 2005; whether this was due to an increase in abscess, cellulitis, or bacterial multiplication, resulting in folliculitis or abscess. Venous blood and lymph drain from the orbits and the skin around them into the cavernous sinuses, and thus bacterial infections in this area can lead to central nervous system infection.

The dermal-epidermal junction is a complex basement membrane whose disruption results in vesicles and bullae. The dermis consists of cells, fibers, and ground substance, which is an acellular material composed of glycoproteins and other macromolecules. 7 The hypodermis, or subcutaneous tissue, is composed largely of adipocytes.

Pathophysiology

Many skin infections arise from breaks in the protective epidermal layer, known as portals of entry. Hematogenous seeding is another common source. Often the source is not clear. When the pilosebaceous follicle becomes obstructed, sebum provides a site for bacterial multiplication, resulting in folliculitis or abscess. Venous blood and lymph drain from the orbits and the skin around them into the cavernous sinuses, and thus bacterial infections in this area can lead to central nervous system infection.

CLINICAL FEATURES

Symptoms and Signs

Most skin infections are present with redness, a symptom, and its corresponding sign, erythema. Erythema is difficult to see in darkly pigmented people. The cause of erythema is microvascular dilation due to the immune response. Confluent erythema is typical of most skin infections; discrete macules and morbilliform (measles-like) eruptions are not typical. Well-demarcated erythema with a raised border, particularly on the face, is typical of...
erysipelas, a streptococcal cellulitis. Linear erythema tracking from distal to proximal along a vascular pathway suggests lym-
phangitis or phlebitis and usually represents the action of cyto-
kines involved in combating the infection, although proximal
spread of the infection itself is a possibility.

Less common color changes associated with infection stem
from small hemorrhages, vasculitis, or septic emboli. Janeway’s
lesions are painless red, purple, or brown spots, usually seen on
the hands or feet, due to septic emboli from infective endocarditis.

Painless discolorations of the palms and soles should also trigger
concern for secondary syphilis or Rocky Mountain spotted fever.
When the diagnosis of infection is not clear, vasculitides such as
Kawasaki syndrome in children and Wegener’s granulomatosis
should be considered. Petechiae and purpura can reveal over-
whelming bacterial infection, as with meningococcemia. Vesicles
suggest contact dermatitis, herpes simplex, varicella-zoster, or
impetigo. Intracutaneous pustules on the palms or soles of the feet
are often due to a form of psoriasis called palmoplantar pustulosis,
which is troublesome but benign.

Pruritic serpiginous (snakelike) lesions that are not particularly
tender suggest an intracutaneous parasite, such as scabies (hands,
intertriginous areas), hookworm larvae (feet or buttocks), or
strongyloidiasis. Parasitic nematodes (Guinea worm) and insects
(botfly) should be considered in the setting of a nodule after expo-
sure to fresh water or insects in developing countries. Induration
simply means hardening and is a common finding with many
inflammatory lesions of the skin. Skin that is indurated because of
cellulitis sometimes becomes engorged with interstitial fluid and
takes on the texture of an orange peel. This classic finding is known
by the French phrase peau d’orange, that is, “orange peel skin.”
Fluctuance describes a fluid collection palpated on examina-
tion. “Pointing” or “coming to a head” conveys a sense of immi-
nent rupture. Crepitance describes skin that feels crackly when it
is palpated and suggests that gas is present in the soft tissues. This
suggests necrotizing infection, discussed later.

Fever is present in half of patients with bacterial skin infections
presenting to the ED.1 Febrile skin infections are more common in
children; in adults, fever may indicate a more serious infection.

Skin infections can suggest underlying systemic illness. For
example, a young man who presents with a first episode of bala-
nitis may have diabetes as the underlying problem. Disseminated
varicella (other than a first episode of chickenpox) suggests immu-
nocompromise. Janeway’s lesions and splinter hemorrhages
suggest endocarditis.

**DIAGNOSTIC STRATEGIES**

For cellulitis and abscess, the only relevant laboratory test is a
wound culture and Gram’s stain when pus is present. However,
there is debate about the necessity of obtaining culture specimens
from skin infections. A culture is prudent for complicated puru-
 lent infections, such as surgical wound infections that may involve
deep structures or abscesses in immunocompromised patients.
Uncomplicated skin abscesses are usually caused by CA-MRSA,
and the need for culture is debatable. Nonpurulent cellulitis is not
routinely cultured because of low yield. A stat Gram’s stain can be
helpful in the setting of diagnostic uncertainty.

Blood cultures are not indicated in patients with skin infection,
with the following exceptions: septic shock, necrotizing infections,
immunocompromise, multifocal infections suggesting hematogenous seeding, and infections complicating lymphedema. Early studies found pediatric facial cellulitis often to be accompanied by Haemophilus influenzae bacteremia. However, the relevant strain is type b, which is now covered by childhood vaccination. Moreover, this organism is typically the target for treatment of facial cellulitis, and there is no evidence that blood culture is of benefit.

When a foreign body is suspected, plain radiographs are traditional, although they will miss foreign bodies that are small or radiolucent. Plain radiographs may reveal soft tissue gas but do not rule out necrotizing infection. Ultrasonography is promising for detection of foreign bodies associated with infection. Ultrasonography can also identify gas, which suggests necrotizing infection. Ultrasound examination is useful for differentiation of abscess from cellulitis, as discussed later.

Plain radiographs are used to seek evidence of osteomyelitis for chronic skin infections, especially in patients with diabetes, peripheral vascular disease, and secondarily infected nonhealing ulcers. Plain films are not definitive, and bone scanning and magnetic resonance imaging (MRI) have higher sensitivity for osteomyelitis. Computed tomography (CT) scanning is often used when a necrotizing infection is considered, although its sensitivity has not been quantified. CT is also helpful when there is concern that a skin infection is actually an extension of a deeper infection, such as after surgery or in the case of recurrent perianal abscesses.

Skin scrapings for microscopy are key to the accurate diagnosis of some infections, including scabies, varicella, herpes simplex, tinea, candidiasis, and leishmaniasis. When necrotizing infection is clinically suspected, operative inspection by a surgeon is considered the definitive “rule-out” procedure.

**CELLULITIS**

**Perspective**

Cellulitis is an inflammatory abnormality of skin and subcutaneous tissue thought to be the result of bacterial infection. Cellulitis may be purulent or nonpurulent and may occur in the setting of wounds, foreign bodies, or impaired perfusion. Purulent cellulitis drains freely, in contrast to abscesses, which are walled off by fibrous tissue and epidermis. CA-MRSA is the leading cause of purulent skin and soft tissue infections in ED patients, but its role in nonpurulent cellulitis is unknown.

**Principles of Disease and Clinical Features**

The cardinal feature of cellulitis is inflammation due to increased local blood flow. In darkly pigmented patients, inflammation may be subtle. Pain is variable. The inflammation of cellulitis is typically confluent, although it may be patchy. The borders are typically well defined but irregular, and linear or circular lesions should prompt a search for other underlying causes (such as contact dermatitis or Lyme disease). In some cases of cellulitis, there are streaks of inflammation extending proximally from the main area of inflammation, along vascular tracts. This finding is known as lymphangitis and is commonly seen with cellulitis due to streptococci and bite wound–associated Pasteurella multocida.

When localized edema becomes severe, separation of epidermal layers can occur, leading to vesicles or bullae. This can make it difficult to distinguish cellulitis from other infectious and noninfectious causes of dermatitis. When the border of an area of cellulitis becomes well demarcated, raised, and palpable, the term erysipelas is used. This form of cellulitis is most often caused by Streptococcus pyogenes. The bacterial causes of cellulitis vary according to body site, comorbidities, and environmental exposures (see Table 137-2).

**Diabetic Foot Infections**

Diabetic foot infections are the most common cause of hospitalization for patients with diabetes, and an infected wound precedes two thirds of lower extremity amputations in patients with diabetes. Neuropathy, vascular insufficiency, and hyperglycemia are important factors in the development of diabetic ulcers and foot infections. Nonetheless, it is important to avoid antibiotic overuse, and uninfected ulcers should not be treated with antibiotics.

The most likely organisms in an acute diabetic foot infection are S. aureus and streptococci. Chronic wounds are more likely to be polymicrobial with both Gram-positive and Gram-negative organisms as well as anaerobes. Chronic wounds that have previously been treated with antimicrobials are more likely to involve multidrug-resistant organisms. Pseudomonas is a traditional concern but is not common. Deep tissue for aerobic and anaerobic culture or bone samples for biopsy should be obtained at the time of débridement if deep tissue infection or osteomyelitis is suspected, but organisms cultured from superficial swabs are not reliable for predicting the pathogens responsible for deeper infection. Osteomyelitis should be considered a potential complication of any deep or extensive ulcer, especially one that is chronic or overlies a bone prominence. In addition to antibiotics, diabetic foot infections require careful wound care and in some cases débridement, revascularization, or amputation.

**Bite Wounds**

A high proportion of cat bites become infected, and presumptive antibiotic treatment is appropriate in the absence of signs of infection. The typical agent is P. multocida. Human bites also become infected frequently, and oral anaerobes are typical. Dog bites become infected infrequently and may be cleansed and observed or treated presumptively; antibiotics are recommended for sutured bites. Amoxicillin–clavulanic acid is an appropriate agent for cat and human bites and for infected or sutured dog bites.

**Water-borne Infections**

Exposure and travel history are important considerations in the evaluation of skin and soft tissue infections. Vibrio species, in particular Vibrio vulnificus, are associated with exposure to seawater and can cause severe soft tissue infections. Patients with liver disease, such as cirrhosis, are at risk. Infection occurs from contamination of open wounds by seawater or shellfish and rarely by hematogenous spread from ingestion of contaminated seafood such as raw oysters. Edwardsiella tarda is a rare cause of wound infection after seawater exposure; it has been implicated in serious soft tissue infections including myonecrosis, particularly in patients with liver disease. Erysipelas rhusiopathiae is usually associated with a localized erysipelas eruption with minor trauma, often on the hands of seafood workers. Aeromonas myonecrosis is associated with exposure to fresh water by either penetrating trauma or exposure to aquatic animals. It causes rapidly progressive supplicative infections that often require surgical drainage. Mycobacterium marinum causes “fish tank granuloma.” It is typically manifested weeks after exposure as a papule or nodule that may ulcerate and drain serosanguineous fluid. Multiple nodular lesions may develop along the lymphatics.

**Differential Considerations and Diagnostic Strategies**

When lymphangitis and fever are present, a bacterial infection is probable. In the absence of these signs, other possibilities include contact dermatitis, ringworm, burns, viral infections, and allergies
including fixed drug eruptions, which can manifest with localized inflammation. Cellulitis must be distinguished from more severe necrotizing infections, discussed later. Consider Lyme cellulitis in endemic areas.

Needle aspiration and even biopsy of cellulitis lesions are unlikely to reveal the etiology and are not recommended. Blood cultures are not recommended except in the presence of a presumed hematogenous source of infection, in septic shock, and in cellulitis complicating lymphedema. Blood cultures are also traditional for facial cellulitis but may be omitted in well-appearing patients who are treated with agents active against H. influenzae. The role of high-sensitivity pathogen identification, such as polymerase chain reaction analysis of surface skin swabs, has yet to be determined.

Venous stasis dermatitis is similar to cellulitis but is not infectious in origin. It is typically located above the ankle and is often (but not always) circumferential. When above-the-ankle dermatitis is accompanied by fever, cellulitis may be present, but other causes of fever should be sought, especially if the inflammation is bilaterally symmetrical. Symmetrical venous stasis dermatitis in the afibrile patient should not be confused with cellulitis. When there is inflammation near a joint, the differential includes gout, pseudogout, septic arthritis, tenosynovitis, Baker’s cyst, traumatic joint effusion, hemorrhrosis, and autoimmune arthritis.

### Management

Emergency physicians have changed their prescribing practices dramatically since CA-MRSA was first described. In 1993, antibiotics targeted to CA-MRSA were almost never prescribed for skin and soft tissue infections. By 2005, 38% of antibiotic regimens included an agent typically active against CA-MRSA. Multiple studies have shown that antibiotics do not make a difference in the treatment of abscess, and the effectiveness of agents targeting CA-MRSA in nonpurulent skin infections (i.e., cellulitis) is unknown.

Table 137-1 summarizes relevant antibiotics. Oral agents with activity against CA-MRSA include trimethoprim-sulfamethoxazole, clindamycin, tetracyclines, and linezolid. The common strains of CA-MRSA (i.e., USA-300) are almost universally susceptible to trimethoprim-sulfamethoxazole in vitro. It is well tolerated and causes severe reactions like Stevens-Johnson syndrome no more commonly than do other agents, such as ampicillin. However, it is not considered effective against streptococci. Thus, when CA-MRSA is suspected but streptococci remain a possibility, the best therapy is most likely trimethoprim-sulfamethoxazole plus a β-lactam such as cephalexin or clindamycin monotherapy.

Clindamycin is commonly suggested as an agent for treatment of cellulitis because of its appropriate spectrum and high potency. However, it must be taken four times daily on an empty stomach—a very challenging task—and causes gastrointestinal upset. Doxycycline is another option for coverage of CA-MRSA. However, this agent must also be taken on an empty stomach and causes gastrointestinal upset. Moreover, it is not thought to cover streptococci unless the etiology of the infection is known. Doxycycline causes photosensitivity, and patients should be warned to avoid sun exposure.

Clindamycin is an important agent because most CA-MRSA isolates are sensitive to it, and it is an excellent agent for streptococci. It can be taken with food. CA-MRSA can become resistant to clindamycin during a single course of treatment. This phenomenon is known as inducible resistance and occurs in about 2% of CA-MRSA isolates. Most microbiology laboratories in the United States now test for inducible resistance among MRSA strains that are susceptible to clindamycin in vitro. The test for inducible resistance is known as the D-test. The name comes from a D-shaped pattern of the area of clindamycin’s inhibition of bacterial growth, which results from resistance induced by a pellet (“disk”) of erythromycin that is placed next to the clindamycin pellet.

Early reports of susceptibility of CA-MRSA to rifampin gave way to later reports of widespread resistance, both inducible and de novo. This agent is not recommended for skin infections.

Vancomycin is the standard parenteral agent for MRSA. Others include linezolid, daptomycin, tigecycline, and telavancin. No penicillin or cephalosporin is active against CA-MRSA, except ceftaroline, a parenteral cephalosporin approved by the U.S. Food
and Drug Administration in 2010 and indicated for complicated skin infections. 21

Nonpurulent cellulitis is treated with coverage for streptococci and methicillin-sensitive S. aureus. When the infection is purulent, a culture and Gram's stain may be performed and coverage for CA-MRSA provided. Otherwise, agents effective against CA-MRSA are not recommended except in cases not initially responsive to first-line therapy and when septic shock is present. 18 The first-line agent is cephalexin, which is safe, well tolerated, and well absorbed and need not be taken on an empty stomach. Inadequate dosing and failure to adhere to recommended adjunctive measures may lead to treatment failure. Relatively severe infections may be managed with one or more initial doses of the intravenous equivalent cefazolin, which, like cephalexin, should be given in maximal doses (e.g., 2 g every 8 hours for adults). Nafcillin is an equivalent option. In patients allergic to cephalaxin, clindamycin is an excellent choice. Table 137-2 provides further treatment recommendations.

Adjunctive measures are important in the treatment of cellulitis. Extremity cellulitis responds dramatically to compression and elevation. The extremity should be elevated above the level of the heart. A splint is useful as an anchor for elevation and can be hung from an intravenous pole. Patients with cellulitis complicating venous stasis or lymphedema should be educated about the importance of compression not only for the acute infection but also to prevent future episodes. Nonsteroidal anti-inflammatory agents (e.g., ibuprofen) are effective and should not be forgotten. 22 As stated before, many cases of cellulitis treatment failure may be due to failure to adhere to these adjunctive measures rather than antibiotic choice.

Figure 137-3 is a universal treatment algorithm for skin and soft tissue infections. The algorithm assumes no prior treatment and no toxic shock syndrome. Previously treated infections require broader spectrum antibiotic coverage and customized management decisions.

Disposition

Immunocompetent cellulitis patients who can be trusted to adhere to recommended medications and adjunctive measures can be managed as outpatients. In severe cases, one or more initial intravenous doses are often given in the setting of an ED observation unit. Hospitalization is generally required for immunosuppressed patients and patients with diabetic foot infections, infected lymphedema, and multifocal cellulitis or suspected necrotizing infection.

ABSCESS

Principles of Disease and Clinical Features

Historically, abscesses were usually caused by methicillin-sensitive S. aureus or mixed flora, but by 2004, CA-MRSA accounted for 61% of abscesses. 2 An abscess begins when bacteria multiply in the lumen of a hair follicle or at other locations beneath the epidermis. Neutrophils are drawn to the site of infection, and various cytokines combine with bacterial toxins to promote development of purulence. The overlying epidermis prevents drainage. A painful red mass is usually seen; it may be tender and often is warm. Skin abscesses are rarely fatal, and most will eventually rupture through the epidermis and drain spontaneously.

Bartholin's cyst abscess is caused by an obstructed Bartholin duct. Bartholin's gland is located at the upper part of the lower third of the labium majus, and its duct opens onto the mucosa in this area, medially, but externally to the labium minus. Bacteria cultured are usually a mixture of aerobic and anaerobic flora from the vagina. Chlamydia trachomatis or Neisseria gonorrhoeae is isolated approximately 10% of the time.

Differential Considerations and Diagnostic Strategies

The differentiation of abscess from cellulitis can be challenging. Bedside ultrasound examination is the best option. 2 A high-frequency linear probe is used. Abscesses are seen as hypoechoic areas with posterior acoustic enhancement. The hypoechoic areas are pus and may be heterogeneous with some bright signals (Fig. 137-4A). Cellulitis is seen either as a uniformly hyperechoic area or as hyperechoic areas separated by curvilinear hypoechoic areas.
Inflamed cutaneous nodules and cystic masses in returning travelers and immigrants from developing countries present special diagnostic challenges. Typical staphylococcal abscesses are most common, but parasitic causes, such as dracunculiasis and myiasis, should be considered.24

**Management**

The treatment of abscess is surgical, and antibiotics are not indicated.25 Exceptions for which antibiotics may be beneficial include the following: severe or extensive disease (e.g., involving multiple sites of infection), severe associated cellulitis, signs and symptoms of systemic illness, associated comorbidities or immunosuppression, extremes of age, abscess in an area difficult to drain (e.g., face, hand, and genitalia), septic phlebitis, and poor response to incision and drainage alone.

Needle aspiration has not been found to be an adequate alternative to incision and blunt dissection.25 Abscess incision and drainage is a nonsterile procedure, but the operator and all environmental fomites should be protected from contamination and transmission of MRSA. The main challenge is to attain adequate analgesia. Injection of local anesthetics into the skin overlying an abscess is difficult because the skin is usually edematous and tense. Also, superficial anesthesia is often inadequate for blunt dissection. An alternative option is to administer procedural sedation. Another excellent option is regional anesthesia (nerve block). Oral analgesia plus a ring block may also provide adequate anesthesia and analgesia. The following medications are safe together and have additive effects: ibuprofen, acetaminophen, oxycodone, and low-dose diazepam. The ring block is performed

(Fig. 137-4B). This appearance is known as cobblestoning and results from interstitial edema. The ultrasound probe and the rest of the machine are fomites and should be cleaned carefully.

Necrotizing fasciitis should be considered, although it is rare. Fistula should be considered when perianal or perivaginal infections are evaluated, and the mucosa should be examined digitally. When perirectal abscess recurs, a deep abscess may be the source, and CT scanning should be considered.

The epidermoid cyst represents another diagnostic challenge. These lesions, formerly known as sebaceous cysts, are benign cystic tumors resulting from pathologic accumulation of keratinaceous material. Patients report a long history of a cutaneous mass, often intermittently painful. These lesions become inflamed periodically and sometimes rupture spontaneously. With rupture, they drain a pearly white or yellowish, glistening, waxy material. Pus, which appears dull and viscous rather than waxy, may indicate infection. Isolated mild inflammation of an epidermoid cyst does not contraindicate primary excision by the practiced emergency physician, although a brief course of antibiotics and anti-inflammatories with delayed excision in the ED or by a dermatologist or surgeon is always an option. Primary excision is more difficult during an episode of inflammation. A minimally invasive method of cyst removal has been described.23

Vascular aneurysms and malignantly or benignly enlarged lymph nodes can be misdiagnosed as abscesses. Ultrasound examination can be helpful in this regard, and color Doppler study should be used to interrogate perivascular abscesses. When there is doubt, needle aspiration should be used to confirm the presence of pus and the absence of blood, or a surgeon should be consulted.

### Table 137-2 Bacteriology of and First-Line Antibiotic Therapy for Skin Infections

<table>
<thead>
<tr>
<th>ANATOMIC VARIANT OR PREDISPOSITION</th>
<th>LIKELY BACTERIAL CAUSE</th>
<th>FIRST-LINE THERAPY (NONTOXIC AND IMMUNOCOMPETENT)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncomplicated cutaneous abscess</td>
<td>CA-MRSA, others</td>
<td>Incision and drainage without antibiotics</td>
</tr>
<tr>
<td>Nonpurulent bacterial skin infections</td>
<td>Various species in the <em>Streptococcus</em> genus, <em>Staphylococcus aureus</em></td>
<td>Cephalexin or clindamycin</td>
</tr>
<tr>
<td>Purulent cellulitis and wound infections</td>
<td>CA-MRSA, others</td>
<td>Cephalexin plus trimethoprim-sulfamethoxazole, or clindamycin monotherapy</td>
</tr>
<tr>
<td>Diabetic foot infection</td>
<td>Mixed Gram-positive, Gram-negative, and anaerobes</td>
<td>Amoxicillin–clavulanic acid plus trimethoprim-sulfamethoxazole Avoid antibiotics for uninfected ulcers</td>
</tr>
<tr>
<td>Any cat bite or infected dog bite</td>
<td>Pasteurella multocida, others</td>
<td>Amoxicillin–clavulanic acid</td>
</tr>
<tr>
<td>Human bite (treat presumptively)</td>
<td>Oral anaerobes, others</td>
<td>Amoxicillin–clavulanic acid</td>
</tr>
<tr>
<td>Erythema migrans</td>
<td><em>Borreli burgdorferi</em> (Lyme)</td>
<td>Doxycycline</td>
</tr>
<tr>
<td>Puncture wound through sole of shoe (treat presumptively)</td>
<td><em>Pseudomonas aeruginosa</em></td>
<td>Ciprofloxacin</td>
</tr>
<tr>
<td>Buccal cellulitis</td>
<td><em>Haemophilus influenzae</em> type b (vaccine serotype)10</td>
<td>Ceftriaxone or ampicillin-sulbactam</td>
</tr>
<tr>
<td>Balanitis</td>
<td><em>Candida albicans</em> or group A streptococcus</td>
<td>Fluconazole plus penicillin or amoxicillin</td>
</tr>
<tr>
<td>Liposuction</td>
<td><em>Peptostreptococcus</em> (anaerobe), group A streptococcus</td>
<td>Amoxicillin–clavulanic acid ± trimethoprim-sulfamethoxazole</td>
</tr>
<tr>
<td>Saltwater exposure</td>
<td><em>Vibrio vulnificus</em></td>
<td>Doxycycline</td>
</tr>
<tr>
<td>Freshwater exposure</td>
<td><em>Aeromonas species</em></td>
<td>Ciprofloxacin</td>
</tr>
<tr>
<td>Butcher, clam handler, veterinarian</td>
<td><em>Erysipelotrix rhusiopathiae</em></td>
<td>Amoxicillin</td>
</tr>
<tr>
<td>Black necrotic eschar with raised border and severe surrounding edema</td>
<td><em>Bacillus anthracis</em> (anthrax)</td>
<td>Ciprofloxacin</td>
</tr>
</tbody>
</table>

*For life- or limb-threatening infections, use intravenous equivalents and add vancomycin.

CA-MRSA, community-associated methicillin-resistant *Staphylococcus aureus*.
to probe the cavity and to disrupt loculations by opening of the clamp through the loculations. Blunt dissection rarely risks injury to vessels and nerves, but the initial incision should be made with such structures in mind. The drained cavity can be irrigated to further break loculations; saline mixed with a small amount of povidone-iodine or a 1:1 dilution of saline and hydrogen peroxide is a reasonable choice for irrigation fluid. The cavity is packed loosely and covered with a loose, absorbent dressing.

Bartholin’s abscesses are drained from the mucosal rather than from the cutaneous surface. The Word catheter is a device used to keep the surgical wound from closing (Fig. 137-5) because the abscess will recur if the wound is allowed to close. A very small incision (about 3 mm) is made, and the cavity is drained. Testing for chlamydia or gonorrhea is recommended. The catheter is inserted and inflated with about 4 mL of water or saline. The catheter should be left in place for 4 to 6 weeks so that a sinus tract will have time to form. Sitz baths may help keep the area clean and dry.

Figure 137-3. Universal algorithm for skin and soft tissue infections (assuming no prior treatment).
Self-sufficient immunocompetent patients can be discharged to home after incision and drainage of an uncomplicated superficial abscess. It is traditional to schedule one or more visits for “wound checks,” but many patients can remove the packing themselves after 2 to 4 days and be instructed to return for reevaluation only in the event of persistent or worsening pain or other symptoms indicative of treatment failure.

IMPETIGO

Perspective

Impetigo is a common superficial skin infection that is most prevalent in children aged 2 to 5 years, but it can occur at any age. It is communicable, spread by both person-to-person transmission and by fomites. It may be manifested as an infection of previously intact skin or may infect skin that has been damaged from minor trauma or atopic dermatitis.

Impetigo rarely progresses to systemic illness. However, most cases of poststreptococcal glomerulonephritis are believed to be caused by impetigo and not pharyngitis. The onset is usually 10 days after the onset of impetigo but may be up to 5 weeks later.

Principles of Disease and Clinical Features

The two main forms of impetigo are nonbullous and bullous. Nonbullous impetigo, or impetigo contagiosa, is the most common form. It was believed for many years that group A streptococcus was the primary cause of this disorder, but studies have subsequently shown that most cases are due to S. aureus. Approximately one third of cases have S. pyogenes isolated, usually in combination with S. aureus. The lesions begin as thin-walled vesicles that progress to pustules; subsequent rupture results in the characteristic “honey crusted” lesions. Lesions are typically found on the face or extremities, and associated lymphadenopathy is common.

Bullous impetigo is caused by S. aureus, including CA-MRSA. The bacteria produce an epidermolytic toxin that causes separation of the dermal-epidermal junction, resulting in bullae. The lesions in bullous impetigo are fewer and larger (0.5-3 cm) but rupture less readily than the vesicles of the nonbullous form. After rupture, the bullae leave a thin brown crust.
Ecthyma or “deep impetigo” is a less common ulcerative form of impetigo that has extension through the epidermis into the dermis. It is manifested as ulcers with a punched-out appearance with raised reddened margins that are covered with thick crust. It has a predilection for the lower extremities. Unlike impetigo, ecthyma leaves scars.

Impetigo can be confused with contact dermatitis, varicella, herpes simplex, bullous pemphigoid, and Stevens-Johnson syndrome. Impetigo does not affect mucous membranes.

Management

Nonbullous impetigo should be treated with topical mupirocin, which is active against most MRSA strains. Systemic antibiotics may be used for nonbullous impetigo when it would be impractical to use topicals because of the extent or location of the infection. Bullous impetigo should be treated with systemic antibiotics active against MRSA and streptococcus. Clindamycin is a good choice. Cephalexin plus trimethoprim-sulfamethoxazole is another option.

Infections of the Pilosebaceous Follicle

Perspective

Folliculitis, furuncle, and carbuncle are purulent infections originating in the hair follicle. They are more likely to occur after damage to the hair follicle, such as from shaving.

Clinical Features and Management

Folliculitis is a superficial inflammation of the hair follicle that is limited to the epidermis. It has many causes, including eosinophilic and drug related, but it is most commonly an infection due to *S. aureus*. The diagnosis is made clinically by its characteristic appearance of a small (2-5 mm), raised, erythematous, painful, tender lesion that is typically pruritic. It can affect any hair-bearing area of the skin. Folliculitis barbae involves the shaved beard area of the face or the shaved scalp.

Hot tub folliculitis is a pruritic condition caused by *Pseudomonas aeruginosa* that develops within 48 hours of bathing in a contaminated hot tub or swimming pool or from use of contaminated sponges. The rash consists of large pustules and may have well-demarcated margins, typically involving the area of skin that was under the bathing suit.

Candidal folliculitis occurs primarily in immunosuppressed patients and in individuals treated with broad-spectrum antibiotic therapy. Eosinophilic folliculitis is a noninfectious recurrent disorder of unknown etiology. It is more likely in immunocompromised patients and is considered an AIDS-defining illness.

Folliculitis usually resolves on its own but can be treated with warm compresses or topical mupirocin. Multiple sites or a large cluster can warrant systemic antibiotics, although no randomized trials have been conducted on the efficacy of this treatment. Shaving of the involved area should be avoided. Hot tub folliculitis usually resolves on its own without specific treatment, but anti-histamines and ciprofloxacin are treatment options. Fungal folliculitis is treated with topical antifungal agents. AIDS-associated folliculitis may be eosinophilic or fungal and may be treated with isotretinoin topically or systemic antifungals, respectively.

Furuncles and Carbuncles

A furuncle or “boil” is an infection of the hair follicle in which suppuration extends through the dermis into the subcutaneous tissue (refer to Fig. 137-2). Furuncles are painful and erythematous and often drain spontaneously. The most common cause is *S. aureus*, both methicillin sensitive and CA-MRSA. Whirlpool baths at nail salons have been implicated in mycobacterial furunculosis. A carbuncle comprises multiple furuncles with loculations and connecting sinuses, often with multiple sites of drainage. Systemic symptoms may occur. Carbuncles are more likely to occur on the back of the neck and are more prevalent in diabetics.

Furuncles and carbuncles are treated in the same manner as skin abscesses, primarily with incision and drainage, although antibiotics with activity against CA-MRSA are treatment options. Small furuncles may be treated initially with a trial of warm compresses to promote drainage.

Acne

Acne results from obstruction of sebaceous glands. It is most common during adolescence because of hormonal stimulation. Acne is treated in the primary care or dermatology clinic, but the emergency physician may choose to initiate therapy with oral doxycycline, topical clindamycin, or topical retinoids.

Hidradenitis Suppurativa (Acne Inversa)

Hidradenitis suppurativa (acne inversa) is an exquisitely painful condition most often seen in the axilla. It may also occur in other apocrine gland–bearing skin, including the perineum, breasts, and inner thighs. It is about three times more common in females than in males. There is some familial predisposition. The typical onset is between puberty and 40 years. It is currently believed to be an acneiform disorder that begins with follicular occlusion, rather than infection of the sweat glands. This has led to suggestions that the term *hidradenitis suppurativa*, which means suppurative inflammation of the sweat glands, be replaced with the term *acne inversa*, which implies an origin of follicular obstruction. The pathophysiologic mechanism remains incompletely understood and is likely to be a complex interaction of hormonal, environmental, and genetic factors.

The clinical course varies from intermittent isolated inflamed nodules to recurrent draining cysts and sinuses that can progress to a chronic and debilitating condition that is difficult to treat. Recurrences can lead to scarring, sinus tract formation, and disfigurement. This is a debilitating disease, and patients suffer not only from pain but also from social stigma due to the odor that may accompany the lesions. They may suffer reactive depression.

The diagnosis of abscess is made on the basis of the characteristic clinical presentation. Perianal and vulvar manifestations of Crohn’s disease may be similar.

Most emergency physicians manage exacerbations with incision and drainage, although it is uncertain whether this accelerates healing. Certainly, incision of a painful, deep, nondraining abscess that is under pressure may bring symptomatic relief. Systemic antibiotics are usually prescribed and should cover CA-MRSA. Perianal lesions should be treated more broadly, with agents active against CA-MRSA, Gram-negative organisms, and anaerobes. Amoxicillin–clavulanic acid plus trimethoprim-sulfamethoxazole is a good choice.

Long-term treatment is complex and remains a subject of debate. Options include immunomodulators (e.g., steroids, cyclosporine), hormones, and en bloc resection. All patients should be instructed to stop smoking and to keep the area clean and dry. Pain control is essential. Patients rarely show signs of systemic illness and thus can be discharged. They should be referred to a plastic surgeon or dermatologist.
Necrotizing and Soft Tissue Infections

Principals of Disease and Clinical Features

Necrotizing infections progress rapidly, cause extensive tissue destruction, and are often fatal despite aggressive treatment. Clinical manifestations that suggest a necrotizing infection are signs of systemic toxicity, including abnormal vital signs, severe pain or pain out of proportion to physical findings, altered mental status, rapidly advancing infection, crepitation, hemorrhage, sloughing, and blistering. Some patients appear well at presentation, and overlying skin may not be involved initially. Extensive tissue destruction occurs eventually, and the mortality rate is 20%.

Risk factors include diabetes, vascular insufficiency, and immunosuppression, although healthy people are vulnerable. Inciting events include penetrating trauma, recent surgery, varicella infection, injection drug use, burns, and childbirth.

Typical bacterial isolates include group A beta-hemolytic streptococcus, S. aureus including CA-MRSA, enterococci, Enterobacteriaceae, and the anaerobes Bacteroides and Clostridium. Most cases are polymicrobial. The classification schemes historically used for necrotizing infections are less important than the general principles discussed earlier.

Necrotizing fasciitis is an aggressive infection of subcutaneous tissues that spreads rapidly along fascial planes. In the operating room, the fasciae are inflamed and tissue layers separate friably. It is caused by direct extension from a skin lesion in 80% of cases. Type I is polymicrobial, with aerobes and anaerobes; it is more common in diabetics and the immunocompromised. Type II is caused by a single organism, in any age group and among patients who are not chronically ill. Group A streptococcus is most common and is the aggressively virulent agent known in the lay press as flesh-eating bacteria. CA-MRSA is also a cause, although it appears to be less virulent. Initial symptoms may be vague (e.g., malaise, fever, body aches, nausea, and diarrhea). There may initially be diffuse or fusiform swelling of an extremity, or it may appear to be a simple cellulitis or wound infection. Physical findings may not be obvious initially, and pain out of proportion to physical findings is a clue. Eventually the skin turns violaceous or ecchymotic. Anesthesia may develop over the involved tissue because of infarction of superficial nerves. Subsequent inflammation may result in the classic sign of “wooden-hard” subcutaneous tissues.

Skin infections in the perineum warrant extra caution. Fournier’s gangrene is the name given to necrotizing polymicrobial infections of the perineum. Fournier’s gangrene progresses rapidly to extend to the entire perineum or abdominal wall.

Myonecrosis, myositis, and pyomyositis refer to infections of muscle, which are rare. They may result from local spread of an adjacent infection, penetrating trauma, vascular insufficiency, or hematogenous spread. Clostridial myonecrosis, also known as gas gangrene, has two forms, a more common traumatic form and a rare spontaneous form. The traumatic form typically occurs from an injury that results in an interruption in the blood supply, and crush injuries are often implicated. The infection is most commonly due to Clostridium perfringens, a Gram-positive spore-forming bacillus that is ubiquitous in nature, including the normal human body. Inoculation of the organism into tissue with low oxygen tension allows proliferation. Exotoxins destroy tissue, contribute to shock, and may cause intravascular hemolysis with anemia and disseminated intravascular coagulation. Patients present with severe pain. The skin may initially be pale, then bronze, and eventually purplish red. Hemorrhagic bullae may develop. Soft tissue gas may not be present initially. Systemic toxicity and shock ensue when aggressive treatment is not initiated early and sometimes despite aggressive treatment. The spontaneous form of clostridial myonecrosis is very rare and occurs without any inciting wound. It is usually due to Clostridium septicum and occurs in patients with bowel disease, such as colon cancer. Synergistic nonclostridial myonecrosis is a related syndrome, most commonly seen in the immunocompromised.

Anaerobic streptococcal myositis usually results from trauma or is a postoperative complication. It resembles clostridial myonecrosis but has a more insidious course. It is caused by anaerobic streptococci, including Peptostreptococcus, but the infection may also include group A streptococcus and S. aureus.

Spontaneous gangrenous myositis (also known as spontaneous streptococcal gangrenous myositis, group A streptococcal necrotizing myositis, or streptococcal myonecrosis) is rare but aggressive and fatal in most cases. It occurs spontaneously, without trauma, in immunocompetent hosts. It is preceded by a prodromal influenza-like phase. Gangrenous necrosis of skeletal muscle then results in severe pain with tense local swelling.

Pyomyositis is a deep abscess within striated muscle resulting from hematogenous spread of bacteria in the setting of muscle injury. It is usually due to S. aureus, including CA-MRSA, and is more common in the immunocompromised. Mortality is less than 10%.

Differential Considerations

The most obvious diagnostic dilemma is distinguishing necrotizing infections from simple cellulitis. A necrotizing infection should be considered when a patient with cellulitis presents with a rapidly progressing course or pain out of proportion to clinical findings or when the patient appears acutely ill or has tachypnea, hypotension, or tachycardia not explained by fever or dehydration. Crepitation or radiographic air is diagnostic of a necrotizing infection unless there is another explanation (such as recent surgery).

Phlegmasia cerulea dolens is iliofemoral vein thrombosis and can be confused with necrotizing fasciitis. Arterial insufficiency causes gangrene, and it may be difficult to determine whether infection is present in severe or chronic cases. Similarly, compartment syndrome can be confused with necrotizing infection or coexist with it.

Diagnostic Strategies

The diagnostic “gold standard” is the characteristic appearance of the tissue by direct visualization in the operating room. Some surgeons may elect to perform a biopsy at the bedside.

A panel of blood tests has been evaluated as a way to differentiate necrotizing infections from other skin infections: the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score. Points are assigned for laboratory abnormalities, and a score of 6 or more is strongly predictive of necrotizing infection, with a positive predictive value of 92% and a negative predictive value of 96%. However, this tool is too preliminary for clinical applicability for several reasons, including methodologic flaws, lack of validation of the findings at other sites, uncertainty about how the score should be implemented in ill-appearing patients, and absence of any data to indicate when the score should be calculated for well-appearing patients. An even simpler decision aid relies on serum sodium concentration and white blood cell count but is similarly lacking in external validation and other criteria needed for clinical application.

Necrotizing infections are very rare and other skin infections are common. Diagnosis of necrotizing infection remains clinical, and universal laboratory screening of well-appearing patients is not recommended.

Plain radiographs may show air in the soft tissues, but absence of this finding does not rule out necrotizing infection. Ultrasound examination can visualize the abscess of pyomyositis. CT and MRI may show compelling evidence of a necrotizing infection, but their negative predictive values have not been quantified.
Management and Disposition

Patients with suspected necrotizing infections should be resuscitated aggressively and have coagulation studies and a type and screen performed because emergent surgery may be needed. Renal function should be assessed and goal-directed therapy used to guide resuscitation.

Empirical broad-spectrum antimicrobials should be administered promptly. A good regimen is clindamycin plus a broad-spectrum β-lactam like ampicillin-sulbactam plus vancomycin to cover MRSA. In patients at risk for hospital-acquired infections, drug-resistant P. aeruginosa and extended-spectrum β-lactamase–producing Gram-negative bacteria should be considered.

When a necrotizing infection is suspected, a surgeon should be consulted. Repeated operative débridement is often needed. Fasciotomies are often necessary as these syndromes are associated with elevated compartment pressures, which contribute to myonecrosis. The efficacy of hyperbaric oxygen in the management of necrotizing infections is unproven, and a dive should not delay surgery.40

TOXIC SHOCK SYNDROMES

Perspective

The principal systemic toxin-mediated bacterial skin syndromes are staphylococcal scalded skin syndrome, streptococcal toxic shock syndrome, and staphylococcal toxic shock syndrome. These syndromes are caused by bacterial exotoxins that are known as superantigens because they cause a severe and pathologic host immune system response by stimulating T-lymphocyte activation and functioning as mitogens in vitro.41 Systemic disease results from the immune system’s response to the toxin, but it may be accompanied by or simply resemble bacteremic septic shock (Table 137-3).

Streptococcal Toxic Shock Syndrome

Streptococcal toxic shock syndrome (TSS) is a severe, toxin-mediated syndrome that rapidly progresses to shock with multiorgan failure and death. Identified in the mid-1980s, this syndrome is caused by group A streptococci, often in the setting of a severe soft tissue infection. Most victims are previously healthy. The syndrome is a rare sequel of disseminated varicella (chickenpox).

Invasive group A streptococcal infections are often due to M-type isolates with potent exotoxins. Signs and symptoms are caused by pyrogenic exotoxins A and B. These act as superantigens and cause overactivation of T cells with massive release of cytokines, including interleukins and tumor necrosis factor.41 The incidence of streptococcal TSS may be increasing, but it remains rare.

Patients may have an influenza-like prodrome with nausea, vomiting, diarrhea, myalgias, and chills. High fever, hypotension, and tachycardia are typical. Altered mental status with confusion is common. A diffuse rash is sometimes present (10% of cases), which may make differentiation from staphylococcal TSS more difficult.

On presentation, the patient has a severe streptococcal infection; necrotizing fasciitis is present in half of cases. Pain is often out of proportion to physical findings. Most patients present with shock or develop it within 4 to 6 hours. Bacteremia is common, with positive blood cultures in about 60%. Serious multisystem complications are common, including disseminated intravascular coagulation, acute renal failure, and acute respiratory distress syndrome. In contrast to staphylococcal TSS, which is infrequently fatal, about 30 to 80% of patients diagnosed with streptococcal TSS die. Epidermolysis, typical of staphylococcal TSS, is not characteristic of the streptococcal variety.

Staphylococcal Toxic Shock Syndrome

While staphylococcal TSS is not as severe as the streptococcal variety, it remains a life-threatening systemic illness. The classic presentation is of fever, rash, and hypotension, often in patients who were previously healthy. It was first described in 1978, and beginning in 1980 there was an epidemic of cases associated with the use of highly absorbent tampons. Menses-associated cases have since declined dramatically when such tampons were eliminated from the market, although tampon use continues to remain a risk factor.

Nonmenstrual cases, which currently account for about half of cases, are associated with a variety of conditions, including surgical procedures (e.g., rhinoplasty, abortion), nasal packing, burns, injection drug use, and the postpartum state. To the clinician, menstrual and nonmenstrual staphylococcal TSS appear similar, and the source infection is often not readily apparent.

S. aureus exotoxins are superantigens that are able to activate large numbers of T lymphocytes, resulting in the massive release of inflammatory mediators including interleukins, tumor necrosis

<table>
<thead>
<tr>
<th>Table 137-3</th>
<th>Comparison of Features of Streptococcal TSS, Staphylococcal TSS, and Staphylococcal Scalded Skin Syndrome</th>
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<tr>
<td><strong>STREPTOCOCCAL TSS</strong></td>
<td><strong>STAPHYLOCOCCAL TSS</strong></td>
</tr>
<tr>
<td>Organism</td>
<td>Streptococcus pyogenes</td>
</tr>
<tr>
<td>Toxin</td>
<td>Pyrogenic exotoxins</td>
</tr>
<tr>
<td>Patient</td>
<td>Previously healthy</td>
</tr>
<tr>
<td>Source</td>
<td>Necrotizing infection</td>
</tr>
<tr>
<td>Rash</td>
<td>Erythematous rash in only 10%</td>
</tr>
<tr>
<td></td>
<td>Stigmata of necrotizing infection are present</td>
</tr>
<tr>
<td></td>
<td>Exfoliation weeks later</td>
</tr>
<tr>
<td>Systemic illness</td>
<td>Hypotension, shock, multiorgan failure likely</td>
</tr>
<tr>
<td>Mortality</td>
<td>30-80%</td>
</tr>
<tr>
<td>Treatment</td>
<td>Critical care resuscitation, operative débridement</td>
</tr>
</tbody>
</table>

SSSS, staphylococcal scalded skin syndrome; TSS, toxic shock syndrome.
Staphylococcal Scalded Skin Syndrome

Staphylococcal scalded skin syndrome (SSSS) is a desquamating skin disorder caused by exfoliating toxins produced by **S. aureus**. SSSS was historically known as fourth disease and in newborns as Ritter’s disease. A disease of infants, it is rare in older children and adults. It can cause outbreaks in nurseries and daycare centers. SSSS is caused by certain strains of **S. aureus**, including CA-MRSA, that produce epidermolytic toxin A or epidermolytic toxin B. These toxins probably act as proteases that target the protein desmoglein 1 on the stratum granulosum layer of the epidermis. Whether they meet the T-lymphocyte mitogenic criterion for a superantigen is a subject of debate, and this may be relevant to the prognosis, which is good relative to TSS. The severity of the disease ranges from a few blisters at the site of infection to exfoliation of most of the body. People with pre-existing toxin antibodies develop the localized form, in which toxin is found in the wound periphery; those without develop the generalized form, in which toxin spreads through the bloodstream. Cultures of the bullae are negative unless they are contaminated or secondarily infected.

Typically, a young child presents with fever, irritability, and a tender red rash. The erythema progresses to bullae formation and subsequent exfoliation of the affected skin. The skin exhibits Nikolsky’s sign, which is separation of the epidermal layer of skin on gentle stroking. Desquamation may be patchy or sheetlike, leaving the skin denuded with a red moist base and predisposing it to secondary infection. Perioral, perianal, and flexural skin may be affected more severely. Mucous membranes are spared. SSSS is not associated with multisystem illness and typically does not lead to shock. Mortality is 1 to 5% and usually a result of complications from comorbid conditions or superimposed infection.

Early SSSS may be difficult to differentiate from bullous impetigo. The lack of mucosal involvement helps differentiate SSSS from toxic epidermal necrolysis and Stevens-Johnson syndrome. Other differential considerations include Kawasaki syndrome, Rocky Mountain spotted fever, meningococcemia, leptospirosis, and heat stroke.

Diagnostic Strategies

Streptococcal TSS should be suspected in any patient presenting with shock, especially if the patient was previously healthy. Diagnostic criteria for streptococcal TSS include the presence of group A streptococcal infection, hypotension, and two of the following:

- Renal impairment, liver abnormalities, acute respiratory distress syndrome, coagulopathy, necrotic soft tissue infection, and rash.
- These criteria were developed for epidemiologic purposes; failure to meet all criteria should not exclude the clinical diagnosis in suspicious cases.

Staphylococcal TSS should be considered in any patient presenting with diffuse rash and hypotension. The diagnosis is made on the basis of the clinical presentation. The characteristic rash often raises clinical suspicion and ultimately aids in establishment of the diagnosis. Isolation of **S. aureus** is not necessary for the diagnosis to be made; in fact, blood cultures are positive in a small minority of cases. In severe cases, laboratory abnormalities are those resulting from shock and organ damage.

Management

Both streptococcal and staphylococcal TSS require critical care resuscitation. In streptococcal TSS, immediate surgical consultation for operative débridement of necrotizing infections is critical. In staphylococcal TSS, any potential source of infection should be removed, such as tampons or wound packing, and all postoperative wounds should be explored for infection.

Clindamycin and vancomycin should be administered. Gram-negative coverage should be added when Gram-negative sepsis is in the differential diagnosis.

Intravenous immune globulin has theoretic benefit, but its efficacy has not been shown in clinical trials. It is a reasonable option in cases of presumed staphylococcal TSS unresponsive to intravenous fluids and vasopressors, but this is not standard in the ED setting. There is conflicting evidence on its efficacy in streptococcal TSS. SSSS is treated with antibiotics active against **S. aureus**, including MRSA. Wound care and hydration are important.

Disposition

Patients with suspected TSS should be admitted, most often to the intensive care unit, for intravenous fluids, antibiotics, and close monitoring. Patients with necrotizing soft tissue infections associated with streptococcal TSS usually require surgical débridement.

Children with mild SSSS may be considered for outpatient management with oral antibiotics and close follow-up. Those with more severe skin involvement often need admission for pain control as well as for temperature regulation and fluid and electrolyte management. Severely affected patients may need intensive care or burn center care. With proper supportive care and antibiotic treatment, the prognosis is excellent, with an overall mortality of less than 5%. Scarring is rarely severe.
other than vesicular and includes the palms and soles; there is usually diffuse lymphadenopathy. Syphilis is rare in the United States, although about 10,000 cases still occur per year (see Chapter 98).

Rocky Mountain spotted fever, caused by Rickettsia rickettsii, is even rarer, diagnosed only about 2000 times each year in the United States. A few days after a bite by a dog tick or wood tick, a nonspecific illness ensues. The characteristic rash begins on the wrists and spreads everywhere, including the palms and soles. It starts macular and becomes petechial and then dusky. The untreated mortality rate approaches 25%, but treated patients do well. Ten percent of those infected never have a rash (see Chapter 134).

Cutaneous anthrax occurs on exposed areas of veterinarians and farmers. A spore of the Gram-positive anaerobe Bacillus anthracis enters a break in the skin, and after an incubation period of about a week, a vesicle forms. This ruptures, leaving a shallow-based ulcer with a raised border. The lesion progresses to painless necrosis and the characteristic eschar. Severe surrounding edema is due to bacterial toxins. Unlike with inhalational anthrax, treated cases do well, and even untreated cases have a mortality below 20%. The lesions may be confused with recluse spider bites.

Tularemia is a rare disease resulting from exposure to animals and is endemic in much of the United States, especially the south central states. The ulceroglandular form is most common and involves an influenza-like illness with a single raised ulcer that has mild central eschar formation. The lesion itself is raised, rather than the border (which might suggest anthrax) (see Chapter 134).

The floor of the mouth is a dangerous location for soft tissue infections. Severe infections may progress to Ludwig's angina, in which the floor of the mouth becomes severely indurated, leading to airway compromise and death. Broad-spectrum antibiotics are indicated. Steroids may reduce swelling. Intubation should be considered and a difficult airway anticipated (see Chapter 75).

Scabies is a skin infestation of the parasitic mite Sarcoptes scabiei. It is endemic worldwide and can cause institutional outbreaks. Lesions are most prominent on the dorsal hands and in intertriginous areas. It is diagnosed by visualization of characteristic burrows and, in ambiguous cases, by microscopy of skin scrapings. It is treated with topical permethrin or a single dose of oral ivermectin (200 µg/kg). Norwegian scabies, also known as crusted scabies, is an aggressive infestation that occurs in the immunocompromised; it is treated with both permethrin and ivermectin (see Chapter 120).

Cat-scratch disease results from Bartonella henselae infection after a cat bite or scratch. Its hallmark is regional lymphadenopathy that appears weeks after a primary lesion at the site of inoculation. Treatment is with a standard 5-day course of azithromycin (with a double dose on day 1).

Strongyloidiasis is caused by infection with the parasitic nematode Strongyloides stercoralis. Skin lesions can appear years after infection and are urticular or serpiginous. A rapidly extending burrow that is pruritic and erythematous is diagnostic; this finding, due to rapid migration of larvae in the skin, is known as larva currens (running larva). Such findings or unexplained eosinophilia in people who have lived in Southeast Asia or tropical Africa should prompt consideration of strongyloidiasis. Diagnosis is by an enzyme-linked immunosorbent assay performed on serum. Detection is important because unlike other nematodes, strongyloides can complete its life cycle in the human host, leading to lifelong infection. When the infected patient becomes immunosuppressed by medications or illness, the strongyloides hyperinfection syndrome can result and is often fatal (see Chapter 133).

Cutaneous larva migrans is another serpiginous skin lesion caused by migrating larvae. In this case, the organism is hookworm, and the site is typically the foot or buttock; it is often seen after a vacation on the beach in Mexico. Treatment is with a single dose of ivermectin 200 µg/kg (see Chapter 133).

Cutaneous leishmaniasis is common in many parts of the world and is found on every continent except Australia and Antarctica. It is caused by protozoans of the genus Leishmania and transmitted by sandflies. Lesions are most common on the face and are painless, ulcerative, and disfiguring. Papules in returning travelers and immigrants should raise suspicion of myiasis (botfly) and, rarely, dracunculiasis (Guinea worm).

**KEY CONCEPTS**

- Skin infections are common and are rarely life-threatening. Deadly necrotizing skin and soft tissue infections are rare, and there is insufficient evidence to motivate screening by laboratory tests.
- Necrotizing infection is suggested by pain out of proportion to physical findings, crepitance, gas seen on imaging studies, or clinical instability. Suspected necrotizing infection is managed aggressively, with broad-spectrum antibiotics, critical care resuscitation, and surgical consultation.
- Emergency physicians should be familiar with toxic shock syndrome and Rocky Mountain spotted fever, which are rare life-threatening skin infection–related syndromes. Lyme disease should be considered in endemic areas.
- For the management of most skin abscesses, antibiotics are not recommended. Adequate analgesia or sedation are essential to good patient care. There is debate about the necessity of wound culture and Gram's stain.
- Current recommendations for the treatment of cellulitis suggest agents effective against streptococci and methicillin-sensitive Staphylococcus aureus (e.g., cephalaxin at maximal doses). Adjunctive measures are essential to a good treatment response (nonsteroidal anti-inflammatory agents, immobilization, elevation, and compression).
- Clindamycin monotherapy is an excellent choice for treatment of skin infections as it covers streptococci and the great majority of staphylococci, including most CA-MRSA isolates.
- Although they are active against CA-MRSA, trimethoprim-sulfamethoxazole and tetracyclines may not be effective for streptococci and are not recommended for cellulitis monotherapy.
- There is insufficient evidence to recommend measurement of the white blood cell count in patients with skin infections.
- Blood cultures are not necessary for the workup of skin infections, except with septic shock, necrotizing infections, immunocompromise, multifocal infections suggesting hematogenous seeding, infections complicating lymphedema, and perhaps facial cellulitis.
- Skin infection mimics include venous stasis dermatitis and other forms of dermatitis.

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The references for this chapter can be found online by accessing the accompanying Expert Consult website.
References


