

EMERGENCY MEDICINE PRACTICE

AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

Skin And Soft-Tissue Infections: The Common, The Rare, And The Deadly

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CME Objectives

Upon completing this article, you should be able to:

1. describe common etiologies and common presenting signs and symptoms of skin and soft-tissue infections;
2. identify appropriate therapeutic measures for the management of skin and soft-tissue infections;
3. discuss empiric treatment options in patients with skin and soft-tissue infections; and
4. identify and review pitfalls in the management of patients with skin and soft-tissue infections.

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See "Physician CME Information" on back page.

July 24, 2000 3:27 p.m.: A 23-year-old medical student presents to the ED with a small cut on his hand. He was swimming in a local canal on the previous day and scraped it on the pier while climbing out of the water. The wound appears superficial and only slightly red, with a small amount of serosanguinous drainage. The emergency physician reflects that the student is making quite a fuss about such a little wound; in fact, the young man is so anxious that his heart rate is 125. The emergency physician orders a dressing, ruefully thinks that only a medical student would swim in that canal, and prescribes cephalexin.

July 25, 2000 5:12 a.m.: A 20-something male is brought to the ED in a moribund state. His blood pressure is 80 by palpation, and he is unable to provide any history. While searching for clues, the emergency physician notices a dressing on the right hand. The arm above the dressing is swollen, tense, and crepitant. The chastened physician quickly reaches for the phone.

SKIN and soft-tissue infections are among the most common problems seen in the emergency department. They range from the utterly benign to true "lights and sirens" emergencies. Most recommendations for the diagnosis and treatment of skin and soft-tissue infections are based on tradition, consensus, or (too often) medical mythology. The literature on this subject is crippled by a paucity of randomized, controlled trials.

This issue of *Emergency Medicine Practice* focuses on the infections that involve the skin, subcutaneous fat, fascia, and muscle. From impetigo to necrotizing fasciitis, we review the most common etiologies and treatment choices using the best available evidence. (Post-operative wound infections are beyond the scope of this article and will not be discussed in this issue.)

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Epidemiology And Etiology

Skin and soft-tissue infections generally result from a violation of the skin or its defenses. Less frequently, hematogenous or lymphangitic spread may arise from a distant source.

Diverse clinical presentations can complicate the diagnosis. Classification often depends upon the depth of infection. (See Figure 1.) Impetigo is localized to the stratum corneum of the epidermis, while ecthyma is confined to the superficial epidermis. Erysipelas and cellulitis involve deeper structures to the level of the dermis. Carbuncles and furuncles can extend to the fat, necrotizing fasciitis to the fascia, and myositis to the muscle. Some pathogens are slow-growing, such as *Mycobacterium* species, while others, such as virulent strains of group A streptococcus, may progress over hours.

Most superficial skin and soft-tissue infections are caused by aerobic gram-positive bacteria, predominantly *Staphylococcus aureus* and group A streptococcus. Gram-negative, anaerobic, or a mixture of organisms can cause deep, complicated infections, usually in the immunocompromised host. (See Table 1.)

History

The history can distinguish the depth and gravity of the contagion. A history of the present illness should begin with the circumstances of any wound the patient suf-

fered. Determine the specifics of the wound. If the patient was bitten, what bit them and when? Some patients will misrepresent the mechanism of injury to the hand. They do not realize the serious nature of a "fight bite" (which occurs when a clenched fist meets an opponent's tooth)

Table 1. Skin And Soft-Tissue Infection Bacteriology.

Gram-positive cocci

Staphylococcus aureus
Group A streptococcus
Streptococcus pneumoniae
Peptostreptococcus sp.
Peptococcus sp.

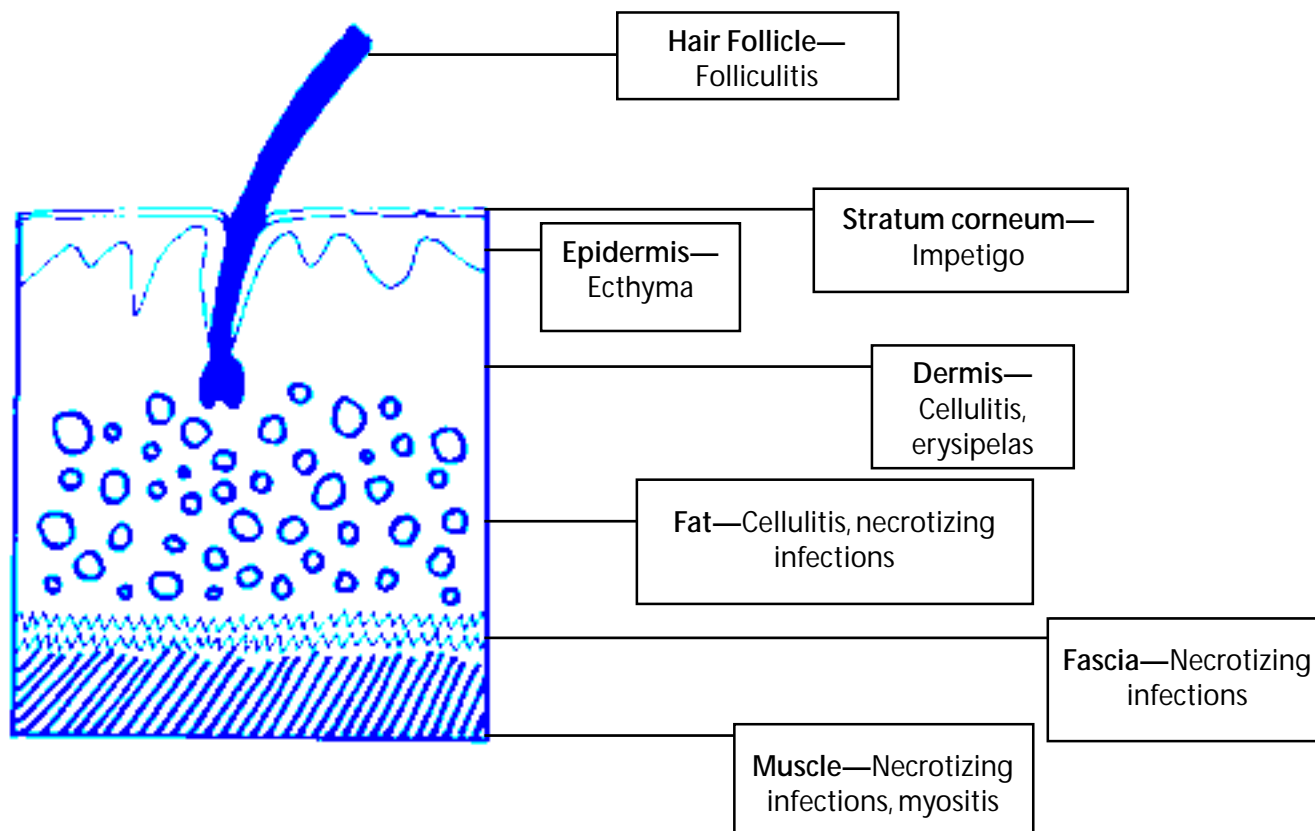
Gram-positive bacilli

Clostridium sp.
Propionibacterium acnes

Gram-negative bacilli

Vibrio sp.
Haemophilus influenzae type B
Pseudomonas aeruginosa
Escherichia coli
Klebsiella sp.
Proteus sp.
Enterobacter sp.
Aeromonas hydrophila
Bacteroides sp.
Fusobacterium sp.

Figure 1. Skin And Soft Tissue Anatomy And Infection Types.



and may manufacture an innocuous story (something like, “I cut it on a card while playing bridge”). Ask whether the wound occurred in water, and, if so, was it seawater or fresh? The emergency physician should be scrupulous in determining the likelihood of a foreign body in the wound. Unless the foreign body is suspected and removed, even gallons of antibiotics will have no effect. The time course of the disease is also crucial. A rapidly advancing infection will require a more aggressive approach. Pain out of proportion to the appearance of the wound is an important characteristic of necrotizing infections.¹

The emergency physician should also ask about systemic symptoms such as fever, chills, nausea, and vomiting. Systemic symptoms suggest a more invasive process and possibly bacteremia. Polyuria and polydipsia may hint at underlying diabetes.

The patient’s past medical history and especially co-morbid conditions will play a dramatic role in the outcome. Elicit any history of diabetes, splenectomy, liver disease, renal failure, alcoholism, HIV, and other causes of immune suppression.

To differentiate complicated from uncomplicated infections, consider the “How, What, When, and Where” approach. (See Table 2.)

Physical Examination

The physical examination provides the best clues to the extent of disease. While a careful inspection of the involved area is important, other aspects of the clinical examination may be as telling. An experienced clinician can diagnose “toxicity” from the hallway. A “doorway” inspection of the patient may reveal cachexia, parietal scalp hair loss, temporal wasting, or other signs of systemic disease. On closer inspection, look for additional clues to co-morbid disease or underlying immunosuppression. Oral thrush is always a worrisome sign in the infected adult. It is usually indicative of an immunocompromised host but is sometimes present with out-of-control diabetes. “Track marks” from intravenous drug use suggest a variety of possibilities, including immune suppression, endocarditis, deep infections, or

unusual organisms. While heart murmurs may accompany the high-flow state associated with severe infection, they may also be heard with endocarditis.

The patient’s vital signs provide rapid clues to the severity of infection. Tachycardia, hypotension, and temperature alterations may be the harbingers of sepsis syndromes.

Examine the involved body part. Completely undressing the patient may yield important findings. While pulling up the pants exposes the infected foot, the associated lymphangitis is missed unless the pants are removed.

Classic findings of infection include erythema, warmth, edema, and pain. However, these represent nonspecific signs of inflammation and can occur in many non-infectious conditions. Erythema may be deceptively minimal despite complicated infections in the immunocompromised. In necrotizing infections, the erythema may darken to a blue-gray patch after the first day, followed by bullae several days later. Occasionally, the overlying skin may appear normal despite necrotizing disease. “Shiny” skin is also frequent with deep infections.¹

Note the extent and location of erythema. It is helpful to mark the advancing border of erythema with ink. Comparing the initial marked area to the size measured at a later time documents the speed of progression or response to therapy.

During the examination, look for lymphangitis. These linear erythematous streaks extend proximally from an infected wound. Lymphangitis is not always contiguous with the infected skin, and “skip” areas are frequent. Swollen nodes may litter the extremity and are distributed in a predictable anatomic fashion. Inflamed and tender epitrochlear nodes (found on the medial side of the proximal elbow) and axillary nodes are common in upper-extremity infections, while inguinal adenopathy is frequent with lower-extremity involvement. Acute lymphangitis is often associated with infection due to group A streptococcus.²

Palpate the involved area for pain, crepitus, or foreign body. Necrotizing infections range from mildly to exquisitely tender early in the disease. As blood vessels

Table 2. Practical Historical Questions: The How, What, When, And Where Of Skin And Soft-Tissue Infections.

How did the initial injury occur?

- Was there an inciting event or trauma?
- Is there *any* possibility of a foreign body?
- Could this be a bite?

What are the complicating factors?

- What is the patient’s immune status?
- Is there a history of liver disease, alcoholism, renal failure, HIV, diabetes, splenectomy, or chemotherapy?
- Is there a past history of poor wound healing, tobacco smoking, or vascular disease?
- Has the patient recently been on antibiotics?
- What is the patient’s tetanus status?

- Does the patient have a heart murmur?
- Does the patient have an indwelling prosthetic device?
- Is there a history or likelihood of poor compliance?
- Is the patient allergic to specific antibiotics, predominantly penicillin?

When did the infection occur?

- Has there been a rapid progression?
- Is the patient worsening by hours or days?

Where did this injury occur?

- Is there a possibility of soil, fecal, or water contamination?
- Has the patient recently been abroad?

thromboses and superficial nerves are destroyed, the skin may become insensate. Pain remote from the erythema or soft-tissue air may signify advanced infection. Assess the infected limb for vascular integrity. Check for and document distal pulses, capillary refill, and skin temperature. Document neurological function as well. This may include screening for sharp-dull discrimination and sensation to light touch. Impaired motor function, particularly of the distal extremities, suggests involvement of deep tissues and compartments.

Diagnostic Testing

The diagnosis of skin and soft-tissue infections is usually based on clinical presentation. Laboratory tests play no role in the routine evaluation of such patients, nor do they predict culture results.³ Likewise, the presence or absence of leukocytosis and/or anion gap are not helpful in distinguishing between cellulitis and necrotizing infections.⁴

Some authorities feel that patients who are immunocompromised, appear toxic, or have a complicated history may benefit from additional studies. This may be based on sporadic reports of immunocompromised patients with cellulitis who have blood and tissue cultures yielding less common pathogens.^{5,6} However, there are no randomized, controlled trials to support this practice.

Laboratory Studies

Very few laboratory studies are helpful in the patient with cellulitis; therefore, they should not routinely be ordered. Studies should be based on clinical indicators such as jaundice, toxicity, or co-morbid disease. Measurement of glucose is appropriate in patients with known or suspected diabetes. Infection may impair homeostatic mechanisms and lead to significant hyperglycemia.

Cultures

Gram's stains and surface cultures of skin and soft-tissue infections are not recommended due to contamination by colonizing organisms. However, intraoperative cultures of necrotizing infections are appropriate.

Aspiration Of The Skin

Many investigators have attempted to aspirate pathogens from patients with cellulitis, with varying results.^{3,6-8} The technique is not well-standardized. It is performed using a needle ranging in size from 18 to 22 gauge. The needle is placed into the infected skin, usually at the leading erythema. The physician then attempts to aspirate material for culture. If the initial aspirate is nonproductive, non-bacteriostatic saline can be injected intradermally and immediately withdrawn for culture. While cultures were positive in 100% of the cases (7 out of 7) in one series, most authors report rates *much* lower—some as low as 10%.^{5,8} A negative aspiration does *not* rule out cellulitis.

In general, most authorities believe that aspiration of the leading edge is nearly useless in the initial evaluation

of uncomplicated cellulitis.⁹ It may be reasonable to aspirate the leading margin of the infected skin when an unusual pathogen is suspected—for example, in an immunocompromised patient, or when a patient is failing initial empiric antibiotic therapy.

Blood Cultures

Blood cultures are rarely necessary in patients with skin or soft-tissue infection. They are seldom positive, and the frequent false-positives lead to additional testing, expense, and therapeutic torture. In a retrospective study of more than 750 patients with cellulitis, a specific (non-contaminant) pathogen was isolated only 2% of the time.¹⁰ In 73% of these cases, the pathogen was beta-hemolytic streptococci, and management changes were limited to switching eight patients from cefazolin to penicillin. In regards to the pediatric patient, blood cultures are not cost-effective in the child admitted to the hospital with cellulitis.¹¹

Blood cultures are often drawn in patients with fever plus one of the following: systemic signs of infection, prosthetic devices, suspected endocarditis, or history of failed outpatient antibiotics. However, empiric evidence for this practice remains slim. In patients with necrotizing cellulitis, blood cultures are often drawn, but most papers regarding the microbiology of this disease rely on cultures of the infected tissue.

Diagnostic Imaging

Radiographs of the involved area may be warranted if a foreign body, osteomyelitis, or tissue gas is a possibility. Diabetics and others with peripheral neuropathy, as well as intoxicated persons, may suffer a penetrating injury without realizing it. Glass is a frequent offender. The size of the piece of glass determines whether it can be seen on x-ray; whether the glass is leaded or unleaded is immaterial.¹²

Soft-tissue air is another important radiographic finding. In general, plain films are insensitive to small amounts of soft-tissue air. Ultrasound, if it can be obtained expediently, is more sensitive for this condition, and it is particularly useful in cases of suspected Fournier's gangrene (necrotizing infection of the perineum).^{13,14} Ultrasound can also aid in the detection of deep abscesses and pyomyositis.^{15,16} Computed tomography is also useful in detecting deep-tissue pathology, including subcutaneous emphysema, deep space abscess, or foreign body.¹⁷

The exact role of imaging techniques such as ultrasound, CT, or MRI for suspected deep soft-tissue infections remains unclear. These modalities cannot always clearly distinguish between inflammation, hematoma, or abscess.

Differential Diagnosis

The emergency physician must consider other causes of localized redness and warmth. Because of the central imperative of our specialty, we must rule out the most

lethal diagnoses first. Excluding other serious diseases is based on clinical examination, the law of probabilities, diagnostic testing, or some combination of the three.

Patients with lymphedema or a ruptured Baker's cyst may present with an inflamed lower extremity. Alternatively, they may have a soft-tissue infection. However, the emergency physician must obsess on a potentially lethal clot desperate to fly to the lungs. Deep venous thrombosis (DVT) is often clinically indistinguishable from cellulitis. Both conditions may present with fever and an elevated D-dimer level. Duplex scanning is the best method to differentiate cellulitis from DVT.

An important consideration in the swollen, red joint is the distinction between superficial cellulitis and septic arthritis. The most important clinical factor relates to the finding of pain on range of motion, which is the sine qua non of joint infection.¹⁸

One of the most common causes of a red, warm patch of skin is an insect sting. These rarely, if ever, become infected. A MEDLINE search revealed no articles relating to infection after Hymenoptera stings. Fire ants cause multiple pustules on the skin when they attack with their acid urine. Despite their cloudy fluid and erythematous base, these pustules are sterile and do not require antibiotics.^{19,20}

At other times the patient cannot recall a bite, and the patient or physician only assumes this history. In

cases of diagnostic confusion, such patients may be treated with both an antibiotic and antihistamine.

Because the signs of infection are nonspecific, the differential diagnosis of soft-tissue infections includes other inflammatory states. Such diseases include collagen vascular disorders such as systemic lupus erythematosus or dermatomyositis. Other mimics of soft-tissue infection include dermatitis, toxic epidermal necrolysis, peripheral vascular disease, trauma, Lyme disease, and cancer. (See Table 3.)

Superficial Skin Infections

Impetigo

Impetigo, or pyoderma, is a localized purulent skin infection usually caused by group A streptococcus (*Streptococcus pyogenes*). While *Staphylococcus aureus* usually causes bullous impetigo (see subsequent section), recent reports have identified this organism as a cause of nonbullous impetigo as well.²¹

Impetigo is most common in children, especially in warm and humid climates. The lesion begins as a vesicle, becomes pustular, and then ruptures to form the classic honey-colored crust. Impetigo is not painful, but it can be pruritic, and scratching often leads to the spread of infection. Regional lymphadenopathy is common, but fever is rare.²² While these infections do not lead to acute rheumatic fever, poststreptococcal acute

Table 3. Cutaneous Manifestations Of Infectious Diseases.

Viral

Varicella-zoster	Chickenpox/Herpes zoster
Rubeola	Measles
Rubella	German measles
Enterovirus	Rash
Coxsackievirus	Hand-foot-mouth disease
Human herpes virus-6	Roseola
Parvovirus B19	Erythema infectiosum (fifth disease)
Epstein-Barr	Infectious mononucleosis
Human papillomavirus	Warts
Dengue/Yellow fever	Hemorrhagic fever

Protozoan

<i>Trypanosoma cruzii</i>	Chagas's disease with chagoma
<i>Leishmania</i> species	Leishmaniasis
Hookworms (Ancylostoma duodenale)	"Ground itch"
<i>Dracunculus medinensis</i>	Guinea worm disease
Filariasis (<i>Wucheria bancrofti</i>)	Elephantiasis
Loiasis (<i>Loa loa</i>)	Calabar swellings
<i>Ancylostoma braziliense</i>	Cutaneous larva migrans
Schistosomes	"Swimmer's itch"

Spirochetes

<i>Treponema pallidum</i>	Syphilis
<i>Borrelia burgdorferi</i>	Lyme disease with erythema migrans

Fungal

<i>Blastomyces dermatitidis</i>	Blastomycoses
<i>Coccidioides immitis</i>	Coccidioidomycoses
<i>Cryptococcus neoformans</i>	Cryptococcosis
<i>Histoplasma capsulatum</i>	Histoplasmosis
<i>Pseudallescheria boydii</i>	Mycetoma

Bacteria

<i>Staphylococcus aureus</i>	Toxic shock syndrome
<i>Neisseria meningitidis</i>	Meningococemia
<i>Streptococcus viridans</i>	Endocarditis
<i>Neisseria gonorrhoeae</i>	Disseminated gonococcal infection
<i>Salmonella typhi</i>	Enteric fever with rose spots
<i>Pseudomonas aeruginosa</i>	Sepsis with ecthyma gangrenosum

Rickettsial

<i>Rickettsia rickettsii</i>	Rocky Mountain spotted fever
<i>R. prowazekii</i>	Endemic typhus
<i>R. typhi</i>	Epidemic (murine) typhus
<i>Bartonella henselae</i>	Bacillary angiomatosis
<i>Ehrlichia chaffeensis</i>	Ehrlichiosis

Mycobacterial

<i>Mycobacterium tuberculosis</i>	Scrofula
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glomerulonephritis may result from infection with nephritogenic strains of group A streptococci. Typically, impetigo heals without scarring.

Treatment Of Impetigo

Current recommendations for treatment include either systemic or topical therapies.

Topical mupirocin (Bactroban) is effective for impetigo. Two advantages of this topical agent are that patients tend to be compliant with it²⁶ and it is not systemically absorbed.²⁷ Mupirocin is also active against MRSA.²⁸ Mupirocin is not recommended for patients with extensive lesions, systemic signs of infection, or perioral lesions (where the medication may be licked off).

Systemic therapy is best achieved with an oral first-generation cephalosporin for seven days.^{23,24} Erythromycin is an effective alternative in the penicillin-allergic patient. Once found exclusively in hospitals, methicillin-resistant *Staphylococcus aureus* (MRSA) is a growing concern in community-acquired infections. Reports from Japan indicate that MRSA accounts for more than 40% of skin infection isolates.²⁵ The implications of this phenomenon in the United States remains unknown.

Bullous Impetigo

Bullous impetigo accounts for about 10% of all cases of impetigo and is generally caused by *S. aureus*. It is most frequently seen in newborns and young children, but it may be found in school-age children as well. Like the lowly impetigo, bullous impetigo begins as a vesicle, but then forms a flaccid bulla. The bulla soon ruptures, to leave a thin, varnish-like, light-brown crust. *S. aureus* produces penicillinase, rendering treatment with penicillin ineffective.²⁴ Penicillinase-resistant penicillin, or an oral first-generation cephalosporin such as cephalexin, are the drugs of choice. (Note that compliance can be an issue; children [and some adults] may resist ingestion of penicillinase-resistant penicillins, which are reputed to be the foulest tasting substances in the pharmaceutical world.) In the penicillin-allergic patient, a third-generation cephalosporin or doxycycline may be effective. (See the sidebar, "Penicillin Allergy And Cephalosporin Use.") Note that doxycycline should be used only in non-

pregnant adults and children older than 8 years, as it may discolor the enamel of growing teeth.

Erythrasma

Corynebacterium minutissimum causes a superficial infection of the skin, usually in the genitocrural area. The rash is scaly, finely wrinkled, and reddish-brown in color. It is intensely pruritic, and without treatment, the rash slowly spreads. Erythrasma is more common in men and diabetics. When the rash is viewed under a Wood's lamp, it fluoresces a bright coral red. Because the differential diagnosis includes tinea cruris, the Wood's lamp test allows for proper therapy. Erythromycin (250 mg po QID x 14 days) is the treatment of choice.

Fungal Genital Infections

Trichophyton rubrum, a dermatophyte, is a common pathogen responsible for tinea cruris (or "jock itch"). It is most common in young adult men during the summer months and in tropical climates. Tinea cruris *does not* fluoresce under a Wood's lamp. The rash is usually bilateral, does not involve the scrotum or penis, and is scaly with central areas that are reddish-brown.²⁹

Topical antifungal cream or ointment twice a day for two weeks provides effective therapy. Tolnaftate is available without a prescription.

Candidiasis is also in the differential diagnosis of the superficial irritating inguinal rash. Clues to the presence of *Candida* include involvement of the penis or scrotum and the presence of small satellite lesions beyond the margin of the rash. It is treated with topical antifungal cream or ointment for two weeks.

Intradermal Infections

Ecthyma

Group A streptococcus causes ecthyma. This skin infection is similar to impetigo, but in this case, the infection penetrates through the epidermis. The lesions appear as "punched out" ulcers with greenish-yellow crusts. Infection may arise spontaneously, or present secondarily in preexisting lesions. They are commonly seen on the lower extremities of children and the elderly. The treatment is the same as for impetigo.

Penicillin Allergy And Cephalosporin Use

Cephalosporin use is contraindicated in penicillin-allergic patients only if an IgE-mediated reaction such as urticaria, angioedema, or anaphylaxis occurs. Estimates of cross-sensitivity of cephalosporins and penicillins vary widely, ranging between 2% and 16%.¹¹² However, even in patients with a stated penicillin allergy, true

anaphylaxis to cephalosporins is extremely rare (< 0.02%).¹¹³ In fact, cross-reactions appear limited to patients given first-generation cephalosporins. *Studies of second- and third-generation cephalosporins show no increase in allergic reactions in patients who have a history of penicillin allergy.*¹¹³ ▲

Adapted from: O'Brien J, Howell JM. Allergic Emergencies And Anaphylaxis: How To Avoid Getting Stung. *Emerg Med Pract* 2000;2(4):1-20.

Erysipelas

Erysipelas, previously named St. Anthony's fire, is a distinctive skin infection that involves lymphatic drainage. It is caused primarily by group A streptococcus; however, group C and group G streptococcus have been implicated as well.³⁰

Erysipelas is common in infants, young children, and older adults. It is found on the lower extremities in 70% of patients and on the face in 20%.³⁰ The rash is painful, bright red in color, and indurated, with a clearly demarcated border. It may present as a "butterfly" rash over the malar area. Erysipelas tends to occur in areas of impaired lymphatic drainage, such as the tissue surrounding a radical mastectomy. Because the infection itself impairs lymphatic drainage, it tends to recur at a rate of 30%.³⁰

Treatment of mild cases is with oral penicillin or, in penicillin-allergic patients, erythromycin. The treatment should include hospitalization and intravenous antibiotics if the infection is extensive, if the patient has systemic signs of infection, or if the patient suffers co-morbidities such as diabetes or immunosuppression. In these high-risk hosts, penicillinase-resistant penicillin (oxacillin or nafcillin) or a parenteral first-generation cephalosporin (cefazolin) are often suggested.

Folliculitis

Folliculitis is a small pustular infection of the hair follicle, usually caused by *S. aureus*. The pustules range from 2-5 mm in diameter, are erythematous, and are typically pruritic. Local measures like saline compresses and topical antibiotics provide sufficient therapy.

Folliculitis barbae (sycosis barbae) is an inflammation of hair follicles in the beard distribution, although it can occur on the shaved scalp.³¹ It is caused by shaving and is most often seen in African-American males. Patients should be advised to temporarily stop shaving, and topical mupirocin or oral antibiotics such as cephalexin or dicloxacillin may be required. Resistant cases may be due to fungi.

A variant of folliculitis is "hot-tub folliculitis," caused by *Pseudomonas aeruginosa*.³² This intensely pruritic condition develops within 48 hours after bathing in a less-than-pristine hot tub or swimming pool, but it also can result from the use of contaminated synthetic or natural sponges.^{33,34} The lesions are classically found on areas of the body covered by a bathing suit. Healing occurs spontaneously after five days,³⁵ but a variety of antibiotics, including fluoroquinolones or macrolides, are sometimes prescribed.

Gram-negative pustular dermatosis can be acquired from mud wrestling. It generally occurs in college students.³⁶ (Advise them to wrestle in Jell-O instead.)

Herpetic Whitlow

Herpetic whitlow is a superficial infection with the herpes simplex virus. It is commonly encountered on the hands of dental workers, anesthesiologists, and others who come into contact with oral mucosa contaminated

with herpes simplex 1. (Among wrestlers, the disease carries the heroic appellation of "herpes gladiatorum.") While the thumb and index finger are most commonly involved, any digit can be affected. The initial lesions are painful, contagious, and are usually located on the distal finger—either lateral to the nail plate or on the volar tip. The lesion may resemble a paronychia, with erythema surrounding a vesiculopustular lesion. The vesicular fluid is initially clear but often becomes turbid. In a felon, the pulp space is tense and painful.

Incision and drainage is contraindicated and may complicate and spread the viral infection.³⁷ The infection is self-limited. Healing occurs within 2-3 weeks, but treatment with acyclovir (400 mg po TID for 10 days) may shorten the course.

Sporotrichosis

Sporothrix schenckii is a soil-derived fungus that infects human skin following direct inoculation. It is commonly seen in farmers and gardeners. Sporotrichosis occurs in warm areas, and the infection typically begins as a lesion on the extremities. Weeks to months after inoculation, a painless red papule develops at the site, and over time the lesion enlarges, ulcerates, and may suppurate. In most cases, the involved skin becomes erythematous and indurated, and patients develop painless red nodules along the course of the lymphatics. These nodules may ulcerate. The differential diagnosis of such lymphocutaneous diseases includes *Mycobacterium marinum*, *Nocardia brasiliensis*, and *Leishmania* species.³⁸ The diagnosis of sporotrichosis is made by scraping and then culturing the skin. The condition is treated with itraconazole (100-200 mg/d po for 6 months).³⁹

Abscesses

*"Thou art a boil, a plague-sore,
an embossed carbuncle, in my corrupted blood."
—King Lear, Act II, Scene IV*

Approximately 2% of all adult patient visits to the emergency department are for cutaneous abscesses.⁴⁰ Abscesses are localized pyogenic infections occurring anywhere on the body and are caused by the bacteria that normally colonize the skin. Exceptions include abscesses that develop following trauma that directly inoculate bacteria. These include wounds contaminated with soil and bite wounds.

A *furuncle* is a deep-seated subcutaneous nodule, or boil. Risk factors include obesity, steroid therapy, absolute or relative neutropenia, and diabetes. Job's syndrome (named after a long-suffering biblical character) is characterized by widespread furuncles. Furuncles are commonly located on the face, neck, axillae, or buttocks and begin as a tender, firm nodule that becomes fluctuant. A *carbuncle* is a cluster of furuncles with multiple draining sinuses. Patients may experience systemic manifestations such as fever and malaise.

Microbiology

S. aureus is the most common pathogen isolated in pure culture, and aerobic Gram-negative bacilli are infrequently recovered.⁴¹ One group of researchers cultured 135 trunk and extremity abscesses and found predominantly mixed aerobic bacteria, such as *S. aureus* and beta-hemolytic streptococci.⁴¹ While mixed infections (aerobes and anaerobes) can occur in any body location, most mixed infections are confined to the perineal areas. One-third of the perineal abscesses contained only anaerobic bacteria, such as *Bacteroides*, *Peptostreptococcus*, *Clostridium*, *Lactobacillus*, and *Fusobacterium*.

Diagnosis

The diagnosis of abscess is made clinically by identifying a fluctuant, tender, soft-tissue mass with erythema and surrounding induration. If the area is tender, but not fluctuant, needle aspiration may be helpful to confirm the presence of pus. Ultrasound may be valuable to detect deep infections.

Laboratory data is not helpful in making the diagnosis. However, blood glucose measurements may be useful in the known or suspected diabetic patient. Gram's staining and culture of purulent material is not indicated in the uncomplicated cutaneous abscess. Microbiological testing

may be useful in the immunocompromised, acutely ill, or those with complicated abscesses. Some authors recommend aspirating purulent material through the skin after sterile preparation and prior to incision and drainage.⁴²

Treatment

The treatment of an abscess is incision and drainage (I&D). If the abscess is not "ripe" (either not fluctuant, or no pus is found on needle aspiration), the patient may be reassessed in 24-48 hours to determine need for I&D. In the interim, the application of heat (hot soaks) may promote the localization of pus and ease pain.

Most small and uncomplicated abscesses should be drained in the ED, but certain abscesses are better managed in the operating room. These may include abscesses located near major neurovascular structures and those that may tract deep into the body, such as a large perirectal (as opposed to perianal) abscess. Consider the use of parenteral opiates and anxiolytics in all but small, superficial boils.

Following adequate local anesthesia with lidocaine, a single incision is made the full length of the abscess cavity. A tiny stab wound is generally insufficient for proper drainage. (However, to avoid excessive scarring, a modest incision in the dependent aspect of a facial

Cost-Effective Strategies For Patients With Skin Or Soft-Tissue Infections

1. Do not take surface cultures of a wound infection.

Culturing material from an infected wound or pressure ulcer is not indicated because colonizing bacteria contaminate all open wounds.

*Risk-management caveat: Intraoperative cultures **are** useful in patients with necrotizing fasciitis or osteomyelitis.*

2. Do not prescribe antibiotics for simple abscesses.

The treatment of an abscess is incision and drainage. A true emergency physician must love pus. Nothing is as satisfying as a fountain of pus as it erupts from a ripe boil. (Well, not many things.) Antibiotics are unnecessary and can lead to complications such as allergic reactions, toxic epidermal necrolysis, and *C. difficile* infections, not to mention antibiotic resistance.

*Risk-management caveat: Antibiotics **might** be valuable in those with surrounding cellulitis, systemic symptoms, or immune suppression. They should be given before incision and drainage to patients at high risk for endocarditis.*

3. Do not obtain blood (or tissue) cultures in a normal host who has cellulitis or lymphangitis.

Routine blood cultures and tissue aspiration are not cost-

effective. They are rarely positive and almost never change management.

*Risk-management caveat: Cultures **might** be valuable in patients who are immunosuppressed or suffer from a resistant or recurring infection. Patients suspected of endocarditis should have several sets of cultures drawn from various sites.*

4. Laboratory studies such as CBCs or electrolytes are unnecessary in most patients with soft-tissue infections.

A CBC adds no further information to the clinical examination of the patient with cellulitis or lymphangitis. It cannot differentiate between cellulitis and septic arthritis and does not provide prognostic information. Likewise, a routine metabolic workup is "money down the drain" in most cases of soft-tissue infection.

Risk-management caveat: Patients with a history of diabetes or those with symptoms of diabetes may benefit from a serum glucose level (and, if indicated, measurement of ketones and bicarbonate). Patients with prolonged vomiting, toxic appearance, hypotension, or petechiae may require a full laboratory panel to evaluate for a metabolic catastrophe or disseminated intravascular coagulation. ▲

abscess may suffice.) A blunt instrument is used to explore the cavity and to break up loculations. Beware of probing abscesses with a gloved finger, because sharp foreign bodies may be retained within the cavity.⁴³ Some authorities suggest irrigating the cavity with normal saline to aid in removing purulent material; however, there is no hard evidence to support this recommendation. Loosely pack the wound with thin strip gauze to prevent premature closure of the wound edges and to assist in drainage. While frequently used, iodoform gauze (gauze impregnated with antibiotics) has no proven value over plain gauze.

The abscess must be re-evaluated in 24-72 hours. High-risk abscesses, such as central facial abscesses, should be re-evaluated in 24 hours.

Antibiotics

Antibiotic therapy has *not* been demonstrated to shorten the clinical course of cutaneous abscesses.^{44,45} In immunocompetent patients who are not acutely ill, I&D is the only treatment required. Antibiotics are generally given to those who are immunocompromised, although no scientific data support this practice. Antibiotics are also indicated in patients with surrounding cellulitis, lymphangitis, fever, or an abscess located on the face below the eyebrows and above the upper lip (due to potential spread of bacteria into the cavernous sinus via facial emissary veins).⁴¹

If antibiotics are indicated because of immune compromise, sepsis syndrome, or cellulitis, the next question becomes, "When should they be given?" While no study has documented timing of antibiotic therapy in relation to I&D, it seems logical to administer antibiotics prior to, and within one hour of, the procedure. This is because bacteremia may occur following manipulation of infected tissue, especially in those with perineal infections.⁴⁶ However, in another study, bacteremia was absent in 50 afebrile patients following I&D of a localized cutaneous abscess having no mucosal involvement.⁴⁷ The duration of antibiotic therapy has not been well investigated. Some authors recommend 5-7 days for immunocompromised patients and 3-5 days for immunocompetent patients.⁴²

The selection of antibiotics depends upon the location and type of abscess. *S. aureus* is a major pathogen and is capable of bacteremic spread resulting in remote infections (such as endocarditis or septic arthritis) or sepsis. If antibiotics are indicated, use an anti-staphylococcal drug (i.e., penicillinase-resistant penicillin [dicloxacillin, oxacillin, or nafcillin] or a first-generation cephalosporin [cephalexin or cefazolin]). Antibiotics for perineal abscesses must provide coverage for *S. aureus*, *Enterobacteriaceae*, and anaerobic bacteria such as *Bacteroides* species.

Endocarditis Prophylaxis

The American Heart Association has published recommendations for the prevention of bacterial endocarditis in

patients with cardiac disease. Those at risk include patients with prosthetic cardiac valves, a prior history of endocarditis, complex cyanotic congenital heart disease, and mitral valve prolapse with valvular regurgitation.⁴⁸ *These patients should receive antibiotics prior to I&D of an abscess where bacteremia is expected.* Give an oral antistaphylococcal penicillin, first-generation cephalosporin, or clindamycin for penicillin-allergic patients one hour prior to I&D.

Perianal And Perirectal Abscesses

Perianal abscesses are superficial infections that do not track deep into the perineum or ischioanal space. The patient with a perianal abscess should not demonstrate tenderness or fluctuance inside the rectal wall, and in general will not be febrile. Patients with perirectal disease exhibit rectal wall tenderness, fluctuance, and may appear toxic. Perirectal abscesses are more serious, with a mortality rate up to 6.5%.⁵¹ Risk factors for morbidity and mortality include associated systemic disease, inadequate initial examination, and delay in treatment.⁵¹

While perianal abscesses may be incised in the ED, those with perirectal infections should undergo I&D in the operating room to ensure exposure, wide drainage, and adequate analgesia.

Cellulitis

Cellulitis is a progressive bacterial infection of the dermis and subcutaneous fat associated with leukocyte infiltration and capillary dilation. In primary cellulitis, bacteria gain entry into the dermis from small breaks in the skin. The offending agents in primary cellulitis are likely to be *S. aureus* or *Streptococcus pyogenes* (group A streptococcus). Other streptococci (such as groups C or G), *Haemophilus influenzae*, *Pseudomonas aeruginosa*, many other gram-negative bacilli, and anaerobes have also been implicated.⁵²

In secondary cellulitis, pathogens invade through larger wounds, or preexisting dermatitis. These bacteria may be either normal flora or transient microorganisms of the skin. Additionally, infection may be acquired through contact with soil or water, or may arise from nosocomial pathogens.

In cellulitis, certain clinical features may suggest the offending agent. For example, staphylococci species are capable of rapid necrosis and early suppuration with significant amounts of purulent drainage. They may form abscesses without evidence of skin disruption.

Group A streptococci is a more common cause of cellulitis and tends to produce a rapidly advancing infection. Such patients often have an obvious portal of entry, such as cracked skin between the toes from tinea pedis.

Cellulitis is characterized by an erythematous skin lesion that is tender, swollen, and warm, and is usually associated with regional lymphadenopathy. *Ancillary testing, such as complete blood count, erythrocyte sedimentation rate, blood culture, or imaging studies are neither required*

nor helpful in making the diagnosis of cellulitis. Sometimes, however, a competing diagnosis such as deep venous thrombosis must be ruled out by objective means.

Therapy

While the decision to treat with an oral vs. parenteral antibiotic regimen hinges upon the severity of infection, this criterion is often subjective. The severity of infection varies based on host factors such as age and underlying immune status.⁵² It also involves the extent of infection, including the amount and depth of skin involved and the rapidity of spread. Areas with proximity to vital organs, such as the face and perineum, are also a consideration. Some therapeutic decisions may depend on the patient's ability to care for him- or herself and tolerate oral medications. In those patients treated as outpatients, the emergency physician should ask whether the patient has the necessary funds to purchase the medication and make some determination regarding the general reliability of the patient and/or caregiver.

The emergency physician should select an antimicrobial agent based on clinical criteria, determining the most likely pathogen in any given scenario.

In general, treat cellulitis with an agent active against penicillinase-resistant *S. aureus*. If the infection is mild and the patient is reliable, then an oral antibiotic such as dicloxacillin or cephalexin for 7-10 days is appropriate. Macrolides such as erythromycin or advanced-generation fluoroquinolones such as levofloxacin may be used in the penicillin-allergic patient. Older fluoroquinolones such as ciprofloxacin are inappropriate for patients with uncomplicated soft-tissue infections due to their poor activity against many important gram-positive organisms.⁵³

If the patient has a moderate to severe infection, or one complicated by chronic illness, then parenteral antibiotics may be warranted. Options here include oxacillin, nafcillin, or cefazolin. Again, a macrolide, clindamycin, or third-generation fluoroquinolone may be appropriate in the penicillin-allergic patient.

Parenteral therapy may be employed on an outpatient basis. In one study, 194 ED patients with cellulitis or soft-tissue infections were randomized to receive either 2 g of ceftriaxone or 2 g of cefazolin, each with 1 g of probenecid, on a daily basis. The subjects were also given a prescription for oral penicillin and cloxacillin. Outcomes were the same in each group, but costs were significantly lower in those randomized to cefazolin.⁵⁴

The need for admission in a patient with extremity cellulitis may be estimated by a clinical decision rule. Important factors that influence the necessity for inpatient care include a history of diabetes, fever greater than 101.5°F, cellulitis of the hand, induration, an area of cellulitis larger than 70 cm², and absence of fluctuance. Of these, diabetes, fever, and hand location are the most significant predictors for admission.⁵⁵ This data, however, has only been published in abstract form. If the patient appears toxic or has had unusual environmental or nosocomial exposure, then antibiotic therapy should

cover a wider variety of bacteria, including resistant organisms. Evidence-based medicine provides little guidance regarding antibiotics in this setting, and many options are available. Broad-spectrum agents such as the beta-lactam/beta-lactamase inhibitors, or third- or fourth-generation cephalosporins are reasonable choices. Combination therapy may include aminoglycosides (for gram-negative pathogens), and either clindamycin or metronidazole for anaerobes. Patients who have recently been hospitalized may harbor MRSA and may benefit from the use of vancomycin (15 mg/kg). Patient response and information obtained from culture will dictate subsequent changes in antibiotic therapy.

Water-Borne Skin Infections

Patients whose wounds are contaminated with water may become infected with unusual organisms. Cuts exposed to seawater may yield *Vibrio vulnificus*. Patients with freshwater exposures are more likely to develop *Aeromonas* infections.

Vibrio vulnificus is a free-living, gram-negative, curved bacillus found in warm marine waters. It is capable of causing terrifying life- and limb-threatening infections, especially in those with underlying liver disease (usually due to alcoholism or viral hepatitis).⁵⁶ Skin infection may follow exposure of open wounds to contaminated seawater or shellfish or may spread hematogenously following ingestion of contaminated seafood, usually raw oysters. Skin involvement may range from mild cellulitis to rapidly progressing necrotizing fasciitis and myositis. Patients may develop hemorrhagic bullae. Treatment requires prompt recognition, surgical debridement, and early institution of antimicrobial therapy. Clinical trials are lacking, but some authors recommend third-generation cephalosporins (e.g., ceftazidime) with an aminoglycoside.⁵⁷ Others suggest adding doxycycline to the empiric antibiotic regimen based on animal studies and in vitro susceptibility testing.⁵⁸

Aeromonas hydrophila is a gram-negative bacillus found in freshwater lakes and streams. Serious soft-tissue infection may follow wound exposure to such water.⁵⁹ These suppurative infections progress rapidly, often requiring surgical drainage. Cephalosporins (second-, third-, or fourth-generation), trimethoprim/sulfamethoxazole, and fluoroquinolones are active against *Aeromonas*.

Skin infection caused by *Mycobacterium marinum* develops following exposure to contaminated water and is generally seen in swimmers and fishermen. A small papule, or nodule, develops after 2-6 weeks of incubation. This "fish tank" or "swimming pool" granuloma will then ulcerate and drain serosanguinous fluid. In approximately 20%-40% of patients, nodular lesions develop along the lymphatics (known as the "sporotrichoid" distribution).⁶⁰ Bacterial culture may require 2-4 weeks of incubation.

M. marinum is resistant to isoniazid and pyrazina-

mide. Treatment options include clarithromycin, minocycline, or trimethoprim/sulfamethoxazole as single agents, or a combination of ethambutol and rifampin. The duration of antimicrobial therapy is approximately 12-24 weeks, and surgical debridement is often required.

Necrotizing Soft-Tissue Infections

*"Flesh, sinews, and bones fell away in large quantities....
There were many deaths. The course of the disease was
the same to whatever part of the body it spread.
But the most dangerous cases...were when the pubes
and genital organs were attacked."
—Hippocrates⁶¹*

Necrotizing infections have been recognized since the time of Hippocrates. In more modern times, Joseph Jones, a Confederate Army surgeon in the Civil War, described it as hospital gangrene in 1871.⁶²

The emergency physician has an essential task when evaluating any soft-tissue infection: to determine the presence or absence of a necrotizing component. If the physician suspects a necrotizing infection, he or she should obtain an immediate surgical consult.

While a variety of bacteria cause these infections, all produce progressive inflammation and necrosis of the tissues, including skin, fat, fascia, or muscle. The classification schemes reported in the literature are complex and confusing. Necrotizing soft-tissue infections have been variably named: gas gangrene, anaerobic cellulitis, Clostridial cellulitis, crepitant cellulitis, synergistic necrotizing cellulitis, non-Clostridial gas gangrene, necrotizing fasciitis, anaerobic myonecrosis, gangrenous erysipelas, hemolytic streptococcal gangrene, Fournier's gangrene, Meleney's synergistic gangrene, and more!⁶³ Most classification schemes are based on gross surgical findings, histopathology, or microbiology. However, none of this information is initially available to the emergency physician.

Risk factors for necrotizing infection include diabetes, obesity, peripheral vascular disease, malnutrition, and injection drug use.¹ In a compromised host, surgery, traumatic wounds (especially open fractures), insect bites, minor trauma, and injection drug use may all lead to infection. However, in some cases, no identifiable injury or skin disruption is identified.⁶³

Epidemiology

Necrotizing fasciitis generally, but not always, occurs in particular clinical circumstances. It follows from trauma (abrasion, bite, laceration, or burn), surgical procedures, decubitus ulcers, or intestinal perforation. Underlying diseases such as diabetes mellitus, alcoholism and cirrhosis, injection drug use, peripheral vascular disease, or immunosuppression predispose to this deadly condition.

Most necrotizing soft-tissue infections are caused by a mixture of aerobic and anaerobic bacteria that act synergistically.⁶⁴ Commonly isolated bacteria include *S.*

aureus, beta-hemolytic streptococci, enterococci, enterobacteriaceae, and anaerobes such as *Bacteroides* species and *Clostridium* species.⁶⁴

Clostridium perfringens is the classic organism responsible for "gas gangrene" or clostridial myonecrosis, although any Clostridial species can produce such infections. *Clostridium perfringens* is especially likely in wounds contaminated with soil. Clinically, *Clostridium* infections begin within hours of an inciting trauma, or surgery, with the sudden onset of pain that rapidly extends beyond the wound. A thin, watery discharge may develop, and large hemorrhagic bullae appear. A Gram's stain of the discharge often reveals gram-positive bacilli with a paucity of white blood cells.⁶⁴

Clostridium septicum can cause spontaneous, nontraumatic necrotizing infections. A colonic lesion, such as carcinoma, will predispose to this highly lethal disease.⁶⁵

Group A streptococcus, known as the "flesh-eating bacteria" in the lay press, causes a wide spectrum of soft-tissue infections. They range from the mild and superficial, such as impetigo, to a rapidly progressive and deadly necrotizing contagion. One study reported an almost five-fold increase in the incidence of necrotizing fasciitis due to group A streptococcus in the past 10 years.⁶⁶ The Centers for Disease Control and Prevention (CDC) estimates 10,000 to 15,000 cases of invasive infection due to group A streptococcus occur annually, with 5%-10% of those representing necrotizing fasciitis.⁶⁷

Many of these patients develop hypotension, renal dysfunction, and coagulopathies resembling staphylococcal toxic shock syndrome. The mortality rate remains higher than 30% despite appropriate antibiotics and supportive care.⁶⁶

Clinical Presentation

While necrotizing infections threaten both life and limb, early illness may appear deceptively benign. Those with advanced infections appear toxic and demonstrate tachycardia, tachypnea, and fever. Patients may be confused and in septic shock. While necrotizing soft-tissue infections can occur anywhere on the body, they are most common on the extremities. Characteristic clinical findings include bronze or ecchymotic discolored skin, pain out of proportion to the physical exam, and anesthesia of the overlying skin.

Presenting symptoms include pain and fever. Rarely, the overlying skin is intact, without signs of infection, and fever and tenderness offer the only clues. More commonly, however, necrotizing infections demonstrate erythema without clear margins; the area is warm, swollen, and very tender. As the infection progresses over hours to days, the skin becomes ecchymotic and bullae form. These bullae often contain hemorrhagic fluid. In advanced disease, cutaneous nerves may become damaged and the overlying skin anesthetic. If a gas-producing organism, such as *Clostridium* species (*not*

Continued on page 14

Clinical Pathway: Management Of Patients With Skin Or Soft-Tissue Infections

History of Present Illness

1. History of trauma or neuropathy?
2. Water contamination?
3. Systemic symptoms (vomiting, fever, rigors)?
4. Bite wound?

Yes

1. Consider foreign body (Class IIa)
2. Consider *Vibrio*, *Aeromonas* infection (Class IIb)
3. Consider need for early antibiotics (Class IIb)
4. Scrupulous wound care (Class I)
 - Avoid suturing human bites to hand (Class IIa)
 - Antibiotics (amoxicillin/clavulanate) (Class IIb)

No

Host Factors

1. Asplenia
2. Alcoholism
3. Diabetes
4. HIV
5. Cancer
6. Other immune suppression

Yes

1. Consider unusual infections
 - *Vibrio vulnificus* (Class IIb)
 - *Capnocytophaga canimorsus* (Class IIb)
 - *Clostridium septicum* (Class IIb)
 - Pyomyositis (Class IIb)
2. Recognize increased susceptibility to necrotizing fasciitis (Class IIb)

No

Physical Examination

1. Assess for toxicity, dehydration
2. Evidence of immune suppression (oral thrush, splenectomy scar, stigmata of liver disease)?
3. Evidence of IVDA?

Yes

1. Resuscitate as needed (Class I)
2. Consider atypical or severe infections (Class IIa)
3. Evaluate for endocarditis (Class I)
4. Consider blood cultures and early broad spectrum antibiotics (Class IIa)

No

Go to top of next page

The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II a:** Acceptable and useful. Very good evidence provides support. **Class II b:** Acceptable and useful. Fair-to-good evidence provides support. **Class III:** Not acceptable, not useful, may be harmful. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

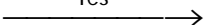
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Clinical Pathway: Management Of Patients With Skin Or Soft-Tissue Infections (continued)

Physical Examination (continued)

- Pain out of proportion to appearance?
- Unexplained tachycardia?
- Bronze discoloration of skin?
- Subcutaneous air?
- Hemorrhagic bullae?
- Anesthetic skin?

Yes



Consider Necrotizing Infection (Class IIa)

- Surgical consult (Class I)
- X-ray or stat US to detect subcutaneous air (Class IIb)
- Intravenous antibiotics (Class I):
 - beta-lactam/beta-lactamase inhibitor or second-generation cephalosporin, plus clindamycin

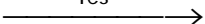
No



Physical Examination (continued)

1. Proximity to joint?
2. Swollen extremity?
3. Wound on hand?
4. Perineal involvement?

Yes



1. Assess for septic joint; pain on range of motion, joint effusion. Arthrocentesis as indicated. (Class I)
2. Determine risk factors for DVT. Liberal use of color flow Doppler if not clearly localized cellulitis. (Class IIa)
3. Assess for fight bite. (Class IIa)
4. Assess for Fournier's gangrene (surgical consult, US for subcutaneous air). (Class IIa)

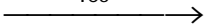
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Abscess

1. Cellulitis or lymphangitis?
2. Systemic signs or immune suppression?
3. Risk of endocarditis (artificial valve, etc.)?
4. Deep space abscess (neck, perirectal)?

Yes



Incise and Drain Abscess

Antibiotics *not* routine

- 1, 2. Give antibiotics for cellulitis, lymphangitis, toxicity, immune suppression. (Class IIb)
3. Antibiotics prior to I&D if risk of endocarditis (Class IIa)
4. Surgical consult (Class I)

No

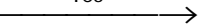


Cellulitis

High-risk criteria?

- Rapid spread
- Fever (>101.5°F)/toxicity
- Diabetes/immune suppression
- Hand infection/central face infection
- Large area
- Probable non-compliance or inability to purchase medications
- Vomiting

Yes



- Antibiotics (oral or parenteral depending upon risk) (Class I)
- Draw circle around erythema (Class indeterminate)
- Admit or discharge based on risk category

Outpatients:

- Recheck in 24-48 hours (Class IIa)
- Return sooner if any worsening (Class I)

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group A streptococcus), is present, crepitus may be noted either by palpation or radiography. Anaerobes may generate an unforgettable putrid odor.

Diagnosis

While many methods have been proposed to aid in rapid identification, the diagnosis of necrotizing soft-tissue infection is difficult to make with certainty outside of the surgical suite. Radiography may reveal gas in the subcutaneous tissues, but the lack of this finding does not exclude the diagnosis. Ultrasound, CT, and MRI are more accurate in detecting subcutaneous emphysema, but these tests should never delay surgical consultation when there is strong clinical suspicion of a necrotizing infection.

In 1970, Wilson recommended passing a probe, or finger, through a skin incision into the fascial planes. The ability of the probe to pass without resistance indicated subcutaneous and fascial necrosis.⁶⁸ Stamenkovik and Lew demonstrated that frozen-section tissue biopsy early in the course of necrotizing soft-tissue infections aided in reducing mortality.⁶⁹ The drawback of this method

includes the requirement for emergency histopathologic evaluation 24 hours a day. Both of these procedures are invasive, and most authorities maintain that the only way to confirm or exclude the diagnosis is by direct visualization of the fascia and muscles in the operating room.⁷⁰

Treatment

Immediate resuscitation, including intravenous fluid, respiratory and inotropic support, and the early institution of antibiotics, is required for patients with septic shock. However, without emergent surgical debridement, most patients will succumb.

Antibiotic Therapy

Although prompt and aggressive surgical debridement is the mainstay of therapy, antibiotics should be given as soon as possible once the diagnosis is suspected. Despite the lack of evidence demonstrating any substantial improvements in mortality, all authors agree that antibiotics must be administered early.⁷¹ Empiric antibiotic coverage must be sufficiently broad to include *S. aureus*, group A streptococcus, gram-negative bacilli including *Pseudomonas aeruginosa*,

Ten Excuses That Don't Work In Court

1. "How was I supposed to know the patient used IV drugs?"

Missing this historical tidbit could be costly. In addition to the history, multiple skin and soft-tissue findings will identify intravenous drug users. Track marks are the most obvious. When all accessible veins are sclerosed, addicts turn to "skin popping," which causes local necrosis or infection. Most authorities believe that intravenous drug abusers with fever should be admitted due to concerns over compliance, risk of endocarditis, and accurate follow-up.¹¹

2. "What's the big deal about his alcohol consumption? He only has a small laceration on his foot from stepping on an oyster shell."

When an emergency physician hears the words "alcohol" and "shellfish" in the same sentence, he or she should be thinking about *Vibrio vulnificus* and not an oyster shooter. This fulminant disease is most common in patients with pre-existing liver disease (usually alcoholics and those with viral hepatitis). Ideal antibiotic coverage includes third-generation cephalosporins plus an aminoglycoside.⁵⁷ Surgical consult will be necessary in most cases.

3. "The patient didn't tell me that he stepped on anything!"

The plaintiff's attorney made quite a point about diabetic neuropathy and how diabetics can step on a needle and

never even know it. Missed foreign bodies consistently reap high awards for litigating attorneys. Diabetics and patients with altered sensorium (drug and alcohol abuse) are at greatest risk for an occult foreign body. Consider ordering x-rays, ultrasound, or even CT in the appropriate clinical circumstances.

4. "Why would I do a serum glucose? The patient doesn't have a history of diabetes."

The nursing note read, "Blurred vision and abscess." Coincidence? Consider diabetes and other immunocompromising conditions in patients with non-healing wounds, unusual infections, or frequent recurrences. A quick screen (even simply asking about new-onset polyuria or nocturia) may reduce future complications and alter follow-up.

5. "I was confident that her persistent tachycardia and shortness of breath were due to her leg pain. After all, she had bad cellulitis."

Actually, the pathologist plans to testify that the saddle embolism and iliac thromboses were the final cause of death. Consider deep venous thrombosis in the patient with a painful, swollen leg (or even a painful,

Continued on page 15

and anaerobes.

Antimicrobial agents for necrotizing fasciitis must cover gram-positive cocci (including *S. aureus*), gram-negative organisms, and anaerobes. Therefore, most necrotizing fasciitis antibiotic regimens include several drugs, such as clindamycin with ceftriaxone alone or both penicillin and gentamicin. The penicillin-allergic patient could receive vancomycin plus metronidazole or clindamycin plus aztreonam.

Single agents providing appropriate coverage include: any of the beta-lactam/beta-lactamase inhibitors (ampicillin/sulbactam, ticarcillin/clavulanate, and piperacillin/tazobactam), the carbapenems (imipenem/cilastatin or meropenem), or second-generation cephalosporins (cefotaxime, cefotetan, or cefmetazole).⁶⁴

Clindamycin, tetracycline, and chloramphenicol all inhibit protein synthesis and may therefore lessen toxin production.^{65,72} Clindamycin is particularly effective in animal models of necrotizing streptococcal infection and demonstrates a number of beneficial effects. In addition to inhibiting toxin production, clindamycin facilitates phagocytosis of *Streptococcus pyogenes*, may destabilize bacterial cell walls, and improves host immune re-

sponse.²² Clindamycin appears to be a useful drug in the patient with necrotizing infections.

Hyperbaric oxygen therapy is often used as an adjunct in necrotizing soft-tissue infections. However, its benefit was proven in only one retrospective study.⁷³ Some authorities hold that until the utility of hyperbaric oxygen is demonstrated in a prospective, randomized trial, it does not represent a standard of care.⁷⁰

Pyomyositis

Until recently, pyomyositis was rare in the United States, but relatively common in tropical areas.⁷⁴ There, it may occur in healthy individuals—especially in those who have suffered local trauma or medical instrumentation. Pyomyositis is now seen in patients with AIDS who have never traveled abroad as well as in patients with other underlying immunosuppressive conditions.^{75,76}

S. aureus is the offending organism in approximately 98% of tropical myositis. In temperate zones, *S. aureus* represents 70%-80% of cases; streptococci, about 12%; and gram-negative organisms, about 10%.⁷⁷

Abscesses develop deep within large striated muscle, and the fascia limits the spread of infection. Subcutane-

Ten Excuses That Don't Work In Court (continued)

swollen arm if the patient has risk factors for an upper-extremity DVT). Be liberal with color-flow Doppler studies in such circumstances.

6. "I routinely place all of my abscess patients on oral antibiotics. What's the big deal?"

It certainly was bad luck that the patient developed *C. difficile* colitis (although she did survive her three-week hospitalization). While your attorney will argue that many physicians use antibiotics after incision and drainage of an abscess, this suit could have been avoided. There is no role for routine antibiotic therapy for patients with uncomplicated soft-tissue abscesses. Antibiotics are not without risk and side effects.

7. "I made the diagnosis of necrotizing fasciitis, gave antibiotics, and even got a hyperbarics consult. I did everything right."

Not everything. The single most important intervention in necrotizing fasciitis is a telephone call—to a surgeon.

8. "It was a simple cellulitis of the knee. I gave him some Keflex and told him to come back if there were any problems."

There certainly was a problem. The patient had septic

arthritis and ultimately suffered weeks of hospitalization and three surgeries. Document the presence or absence of an effusion when evaluating a presumed cellulitis near a joint. If the patient has pain on range of motion, he or she needs an arthrocentesis—not a prescription.

9. "But all I did was I&D an abscess. I didn't know the patient had a mechanical heart valve."

Fortunately, this gentleman's valve didn't have to be replaced, or the settlement would have been greater. Perform an adequate history and physical examination, even for a "little boil." The physician should have realized that the three-foot scar on this patient's chest was not a "stick-on" tattoo. Current AHA endocarditis prophylaxis indications include prosthetic heart valves, prior history of endocarditis, mitral valve prolapse with regurgitation, and cyanotic congenital heart disease.

10. "What do you mean, incision and drainage of that felon was the wrong thing to do?"

Incision and drainage of herpetic whitlow can spread the viral infection and delay resolution. History of exposure is the key element in these cases and will prevent complications in this self-limited disease. ▲

ous and cutaneous tissues are only involved when the illness has progressed. Multiple abscesses occur in one-third of patients.

Pyomyositis is characterized by a subacute onset of muscle pain followed by fever in the ensuing days to weeks. The involved areas are “hard” or “woody” upon palpation. Blood cultures are positive in fewer than 5% of patients. Leukocytosis and bacteremia are even less frequent in those with HIV-related infection.⁷⁷

Treatment is drainage. Patients may undergo surgery or have an ultrasound-guided aspiration.⁷⁸

Chronic Skin Infections

Diabetic Foot Ulcers

“So went Satan forth from the presence of the Lord, and smote Job with sore boils from the sole of his foot unto his crown.”

Diabetic patients are at risk for chronic infection of the feet due to vascular insufficiency, neuropathy, and impaired immune function. These infections account for one-half of all nontraumatic lower-extremity amputations in the United States.⁷⁹ In uncomplicated diabetic foot infections, gram-positive cocci (staphylococci, streptococci, or enterococci) are isolated 94% of the time if the patient has not previously received antimicrobial therapy.⁸⁰ Anaerobes are rare in these uncomplicated infections. Chronic ulcers are more likely to display multiple organisms, including gram-positive cocci, aerobic bacilli, and anaerobic bacteria.

Swab cultures of diabetic foot ulcers are not useful due to multiple bacteria colonizing and contaminating the open wound. The most reliable culture techniques include surgical debridement and sampling of previously unexposed tissue and aspiration through uninvolved skin that is prepared in a sterile fashion.

While advanced osteomyelitis can be diagnosed by x-ray, early disease may be radiographically indistinct. Instead, a simple physical examination maneuver can provide a definitive diagnosis. Gently probe the foot ulcer with a sterile metal probe. Hold the probe or hemostat like a pencil and rub the ulcer base to detect the gritty surface beneath. In osteomyelitis, the probe feels as if there is no intervening soft tissue as it grinds against the bone. In a study of 76 diabetic foot ulcers, this test had a sensitivity of 66%, a specificity of 85%, a positive predictive value of 89%, and a negative predictive value of 56% for osteomyelitis.⁸¹ While this approach has a reductionist appeal, it should not dissuade the consultant from a follow-up bone scan or MRI when osteomyelitis is suspected.

If neither radiography nor the probe test suggests osteomyelitis, these patients may be safely treated at home with any of a number of antibiotics. Choices include clindamycin, cephalexin, ampicillin/clavulanate, or ofloxacin.^{80,82}

Patients with chronic or recurrent ulcers resistant to outpatient therapy, as well as those with signs of severe

cellulitis, osteomyelitis, necrotizing infections, or deep-space infections, should be admitted for parenteral antibiotic therapy and surgical consultation. If the infection is mild, a single agent such as cefoxitin may be used. A combination of agents, such as ciprofloxacin and either metronidazole or clindamycin, is also appropriate. If the infection is severe or the patient appears septic, administer a carbapenem (imipenem or meropenem), or a beta-lactam/beta-lactamase inhibitor (ampicillin/sulbactam, ticarcillin/clavulanate, or piperacillin/tazobactam). Alternatively, employ a combination of penicillin-resistant penicillin (oxacillin or nafcillin) plus an aminoglycoside and clindamycin. Surgical consultation is essential in such cases.

Pressure Ulcers (Infected Decubiti)

Pressure ulcers are a common and serious affliction among the elderly and nursing-home inhabitants. They are associated with a fourfold increase in the risk of death, and they are among the most common sources of infection in this population.⁸³ Immobility, the most important risk factor, is required for the development of pressure ulcers.⁸⁴ Bacteremia may develop with a pressure ulcer source, and up to one-third of patients will have polymicrobial sepsis.⁸⁴

Gram-negative bacilli, such as *Escherichia coli*, *Proteus mirabilis*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*, are the most common pathogens. Gram-positive cocci, such as *S. aureus*, group A streptococcus, and *Enterococcus* are also frequent. Anaerobes occur in up to 50% of patients.⁸⁵

An infected ulcer is suggested by surrounding erythema, purulent drainage, foul odor, and fever. Culturing material from a swab of a pressure ulcer is not indicated, because colonizing bacteria contaminate all open wounds. Tissue cultures taken during surgery are required to obtain an adequate specimen.

Systemic antibiotics are required for patients with cellulitis, sepsis, or osteomyelitis. First-generation cephalosporins may not penetrate tissue involved in pressure ulcers, but both clindamycin and gentamycin do.⁸⁶ If necrotic tissue is present, surgical debridement will be necessary.

Bite Wounds

Animal bites alone result in approximately 300,000 ED visits each year.⁸⁷ Dog, cat, and human bites make up the majority of all bites, but other animals, such as horses, pigs, rodents, and camels, will also sink their teeth into human flesh. Although not all bites become infected, these wounds are considered high-risk. The incidence of infection following animal bites ranges from 3% to 18% for dog bites and 28% to 80% for cat bites.⁸⁸

Bite infections are frequently polymicrobial. In addition to *S. aureus* and *Streptococcus* species, bite wounds frequently become infected with bacteria that are natural inhabitants of the oral cavity (i.e., gram-positive and gram-negative bacilli and anaerobes). *Pasteurella*

canis and *Pasteurella multocida* are the most commonly isolated pathogens isolated from infected dog and cat bites, respectively.⁸⁸ Human bites are frequently infected with *Streptococcus viridans*, *S. epidermidis*, *S. aureus*, *Corynebacterium*, *Eikenella*, and *Bacteroides* species.

Part of medical legend holds that human bites are more likely to become infected than other mammalian bites. This is probably not true.⁸⁹ Human bites to the face, lips, and ears are at very low risk for infection (< 3%) if treated early. It is the clenched fist injury (CFI) that gives human bites their bad name. These devastating injuries can lead to amputation of the fingers and require meticulous wound management and antibiotics.⁹⁰ Amoxicillin-clavulanate is frequently used.

Prophylaxis of bite wounds is controversial, and there is no data to support its routine use. In five of eight randomized trials, prophylactic antibiotics reduced the

rate of infection, but the difference was clinically significant in only one of these studies (using amoxicillin-clavulanate).⁹¹ Wounds at risk for infection—that is, deep wounds, puncture wounds, wounds located on the hands, wounds caused by cat bites, and those requiring surgical repair—may benefit from prophylaxis. Antibiotics that cover the most likely pathogens should be given within 12 hours after injury. A five-day course of amoxicillin/clavulanate is recommended, although the exact duration of prophylactic therapy is unknown.⁸⁸ First-generation cephalosporins such as cephalexin are inadequate due to their lack of activity against *Pasteurella*, *Eikenella*, and anaerobes.^{92,93} Tetracycline or doxycycline is sometimes preferred in penicillin-allergic patients who suffer animal bites, since erythromycin has limited activity against *Pasteurella*.⁹⁴ Azithromycin and levofloxacin are other alternatives in the penicillin-

Table 4. Selected Antimicrobials For Cutaneous Infections.

Disease (duration of treatment)	First Choice	Alternative	Penicillin-allergic
Impetigo (10 days)	mupirocin topically TID	cephalexin 40-50 mg/kg/d QID	erythromycin 40 mg/kg/d QID
Bullous impetigo (10 days)	cephalexin	dicloxacillin 12-25 mg/kg/d QID	erythromycin
Erythrasma (14 days)	erythromycin 250 mg QID	N/A	N/A
Herpetic whitlow (10 days)	acyclovir 400 mg	N/A	N/A
Erysipelas—mild (7-10 days)	penicillin 500 mg po QID	dicloxacillin 500 mg po q6h	a macrolide po or IV
Erysipelas—severe (7-10 days)	nafcillin/oxacillin 2 g IV q4h	cefazolin 1 g IV q8h	a macrolide
Sporotrichosis (6 months)	itraconazole 100-200 mg po	N/A	N/A
Cellulitis—mild (7-10 days)	dicloxacillin 500 mg po QID	cephalexin 500 mg po QID	a macrolide
Cellulitis—severe (7-10 days)	beta-lactam/beta-lactamase inhibitor*	first-generation cephalosporin**	a macrolide vancomycin
Necrotizing fasciitis†	beta-lactam/beta-lactamase inhibitor* ± clindamycin	second-generation cephalosporin** ± clindamycin	clindamycin plus vancomycin

*Ampicillin/sulbactam 1.5-3.0 g IV q6h

Ticarcillin/clavulanate 3.1 g IV q4-6h

Piperacillin/tazobactam 3.375 g IV q6h

** Cefazolin 1-2 g IV q8h (first-generation cephalosporin)

** Cefoxitin 1-2 g IV q8h or cefotetan 1-2 g q12h (second-generation cephalosporin)

† The most important consideration for necrotizing disease is early surgical consultation.

allergic patient.¹¹⁴ Proper wound care is essential and may be more important than antibiotics. Debridement of devitalized tissue, wound irrigation, and restraining the urge to close high-risk wounds (such as clenched-fist injuries) are more important than dousing the patient with antimicrobials.^{95,96}

For established infections, parenteral therapy with a beta-lactam/beta-lactamase inhibitor combination (ampicillin/sulbactam, ticarcillin/clavulanate, or piperacillin/tazobactam), a second-generation cephalosporin with anaerobic activity (cefoxitin or cefotetan), or combination therapy with penicillin and a first-generation cephalosporin or clindamycin and a fluoroquinolone is indicated.⁸⁸ However, antibiotics merely nip at the heels of some infections. Infected fight bites require intravenous antibiotics and surgical consultation.

One life-threatening bite infection that deserves special mention is due to *Capnocytophaga canimorsus* (formerly termed Dysgonic fermenter 2 [DF-2]). This organism is transmitted through the bite of a cat or dog and can cause rapid septicemia, especially in the immunosuppressed. Most victims have been those with a prior history of splenectomy or alcoholism.⁹⁷ In addition to the sepsis syndrome, it may cause meningitis, endocarditis, arthritis, and pleural and eye infections. Rash or even gangrene is frequent.

The organism is susceptible to a wide variety of antibiotics, including penicillin, ampicillin, cefaclor, cefuroxime, erythromycin, clindamycin, and tetracycline.⁹⁸

Cat Scratch Disease

Cats are reservoirs for *Bartonella henselae*, and humans can become infected following bites, scratches, or licks from an infected cat. Most victims are children and young adults. The disease is self-limited and generally benign. A papular, or pustular, lesion develops 3-10 days after inoculation, usually on the hand. Two weeks later, enlarged nodes develop in the axillary, cervical, or submandibular areas. Only 10% of nodes develop abscesses, and enlarged nodes resolve spontaneously after several months. Atypical disease, including pulmonary, hepatic, and central nervous system involvement, is rare.

B. henselae is difficult to culture, and serologic testing may make the diagnosis (although a classic history and physical examination alone is usually adequate). Polymerase chain reaction (PCR) technology is promising, and biopsy is rarely necessary. A wide variety of antibiotics have been used but only a few have proved efficacious, including rifampin (87% effective), ciprofloxacin (84%), trimethoprim-sulfamethoxazole (58%), and intramuscular gentamicin sulfate (73%).⁹⁹ In one placebo-controlled trial, a five-day course of azithromycin significantly decreased lymph node swelling.¹⁰⁰ On the other hand, some authorities believe that antibiotic treatment should be reserved for severe disease.¹⁰¹

Pediatric Considerations

Most pediatric soft-tissue infections differ little from their adult counterparts, but there are some exceptions.

Scalp Infections

Some children present to the ED with a "soupy" infection of the scalp. These boggy areas involve hair loss, while the surrounding hairs pluck easily from the scalp. Swollen occipital nodes can be palpated where the back of the head meets the neck.

Before writing a prescription for cephalexin in such cases, reconsider. Bacterial scalp infections are extremely rare, yet fungal infections are overwhelmingly common. You have just diagnosed a kerion, a complication of tinea capitis.¹⁰² Treat with 6-12 weeks of griseofulvin 10 mg/kg/day. Itraconazole, terbinafine, and fluconazole are alternatives, but none have the excellent track record and safety profile of griseofulvin.¹⁰³

Facial Cellulitis

Buccal and periorbital cellulitis were once feared infections of childhood. However, since the advent of the HiB (*Haemophilus influenzae* type B) vaccine, these infections have become nearly extinct.¹⁰⁴ Buccal cellulitis involves swelling and redness of the cheek, while periorbital cellulitis manifests as a cellulitis around the eye without proptosis or other evidence of orbital cellulitis. The vast majority of orbital or periorbital cellulitis in children is now related to an underlying sinusitis.¹⁰⁵ Other etiologies include local trauma and odontogenic sources.¹⁰⁶ With the near disappearance of invasive *H. influenzae* disease, buccal cellulitis in children is a fading entity. If a child in the ED displays a red, swollen cheek, obtain some history before drawing blood cultures and administering ceftriaxone. The child may be a victim of "Popsicle panniculitis," which results from assiduous sucking on a frozen treat.¹⁰⁷

In the *H. influenzae* era, lumbar puncture was routine for young children with facial cellulitis. Now a spinal tap may be restricted to the high-risk child, as defined by age under 2 months, meningeal or focal neurologic signs, vision loss, limitation of eye movement, eye malformation or operation in the vicinity, and the clinically toxic child.¹⁰⁸

Children with buccal or periorbital cellulitis may be treated with a third-generation cephalosporin or ampicillin/sulbactam.

Staphylococcal Scalded Skin Syndrome

S. aureus can produce exfoliative toxins that result in widespread bullae and peeling of the skin. This condition begins with fever, skin tenderness, and a diffuse scarlatiniform rash. Large bullae form, then rupture, exposing large areas of bright red skin. Nikolsky's sign is the hallmark of this disease. The sign is elicited by gently rubbing the involved skin, which will easily

peel off.

Treatment includes intravenous fluid therapy and intravenous penicillinase-resistant penicillin (such as nafcillin or oxacillin). Cool saline compresses may offer some symptomatic relief. Recovery is remarkably rapid, with full re-epithelialization occurring within 1-2 weeks. Mortality is less than 5%.¹⁰⁹

Toxic epidermal necrosis (TEN), the most severe manifestation of a drug reaction, may appear clinically indistinguishable from staphylococcal scalded skin syndrome (SSSS). While TEN may occur in all age groups, it is more common in adults, particularly the elderly. Fever is common, and patients present with a burning erythematous rash with bullae. The bullae subsequently rupture, denuding the skin. Although

Nikolsky's sign is present, the cleavage plane of the skin in TEN is deeper, occurring at the epidermal-dermal junction. This pathology finding is best determined by biopsy. Because of the potential for huge fluid losses, supportive care is best delivered in an intensive care or burn unit. The use of steroids, plasmapheresis, and intravenous immunoglobulin remains controversial. The mortality rate is approximately 30%, and most die from sepsis.¹¹⁰

Summary

Skin and soft-tissue infections can range from the benign to the life-threatening. While severe manifestations are relatively rare, ED physicians must remain cautious in all

Tool 1. Sample Discharge Instructions For The Patient With Skin And Soft-Tissue Infections.

You have been diagnosed with _____, an infection of your skin and surrounding tissue. Please return to the emergency department or see your doctor right away if you (or your child) gets any of the following:

- Worsening pain, numbness, or discoloration of the skin (blue or black)
- Increased swelling, new blisters, or increasing redness
- Red streaks up the arm or leg
- High fever (>102°F) or shaking chills
- Vomiting or inability to swallow your medicines
- Failure to get better after two days of antibiotics
- Confusion or strange behavior
- Any worsening at all

Follow-up Instructions

____ Return to the emergency department in _____ hours for recheck.
____ See your doctor if not improving in _____ days.
____ See your doctor in _____ days.

Medications

Take the following medications:

Possible side effects of the medications you have been prescribed include:

Additional Instructions

- No alcohol
- No tobacco

Please return at any time; we are open 24 hours a day and happy to care for you.

cases. Because serious illness may masquerade as a run-of-the-mill infection, effective management and follow-up are a must. ▲

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. **In addition, the most informative references cited in the paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.**

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Physician CME Questions

1. A 45-year-old male presents with a painful, sharply demarcated, raised, erythematous rash covering both cheeks. He is non-toxic in appearance. The most likely diagnosis is:
 - a. ecthyma.
 - b. erysipelas.
 - c. St. Elmo's fire.
 - d. erythrasma.
 - e. pyoderma gangrenosum.

2. A five-year-old child presents with multiple sites of pustular vesicles on the face, elbows, and knees. The lesions have mild erythema and “honey-crusted” scabs. Of the following, the most likely pathogen associated with this condition is:
 - a. *Streptococcus pyogenes*.
 - b. *Listeria monocytogenes*.
 - c. *Streptococcus pneumoniae*.
 - d. *Haemophilus influenzae*.
 - e. *Staphylococcus epidermidis*.
3. A previously healthy 28-year-old male presents with a localized, tender, palpable, fluctuant nodule in his right axilla. He is not febrile and has no surrounding cellulitis. The most appropriate management would include:
 - a. incision and drainage with IV antibiotics.
 - b. incision and drainage with oral antibiotics.
 - c. incision and drainage.
 - d. warm compresses.
 - e. incision and drainage with TIG.
4. Group A streptococcus is most commonly associated with:
 - a. toxic shock syndrome.
 - b. scalded skin syndrome.
 - c. bullous impetigo.
 - d. folliculitis.
 - e. ecthyma.
5. A 28-year-old dentist presents with a swollen right index finger with a pustule at the base of the fingernail. The most appropriate management in this case would include:
 - a. incision and drainage.
 - b. incision and drainage with oral antibiotics.
 - c. nail removal.
 - d. nail removal and oral antibiotics.
 - e. local wound care and acyclovir.
6. A 54-year-old male presents complaining of “jock itch” that has not resolved despite repeated dosing of topical and even oral antifungals. A Wood’s lamp directed to the area reveals a red fluorescence. The best treatment is:
 - a. erythromycin.
 - b. diflucan.
 - c. povidone-iodine scrub.
 - d. penicillin.
 - e. trimethoprim/sulfamethoxazole.
7. Which of the following distinguishes cat bites from dog bites?
 - a. The need for tetanus prophylaxis
 - b. The need for rabies prophylaxis
 - c. The need for local wound care
 - d. The bacteria isolated
 - e. Infection rates
8. A 38-year-old male presents with rapidly progressive left leg cellulitis. He has a history of cirrhosis and a recent ingestion of oysters. Antibiotic selection should include:
 - a. penicillin.
 - b. clindamycin.
 - c. a third-generation cephalosporin plus an aminoglycoside.
 - d. a first-generation cephalosporin.
 - e. metronidazole.
9. A 38-year-old male presents following a puncture wound to his right calf 12 hours prior to presentation. Findings include severe pain, crepitus, watery discharge, and hemorrhagic bullae. Which is the most important intervention?
 - a. Intravenous antibiotics
 - b. I&D in the ED
 - c. CT scan of the limb
 - d. Surgical consult
 - e. Hyperbaric oxygen
10. Which of the following types of human bites presents the most risk?
 - a. Bites to the ear
 - b. Clenched-fist injuries
 - c. Bites to the lip
 - d. Bites to the face
11. Facial cellulitis:
 - a. has become more frequent since the advent of the HiB vaccine.
 - b. occurs more often in adults than children.
 - c. is characterized by rash and fever.
 - d. is often related to an underlying sinusitis.
12. A 45-year-old male is bitten on the hand during an altercation. He has no signs of infection. Of the following, which is the best management approach?
 - a. Wound suture
 - b. Oral trimethoprim/sulfamethoxazole
 - c. Wound irrigation, ampicillin/sulbactam, and a recheck in 24 hours
 - d. Amputation
13. Risk factors for furuncles include:
 - a. obesity.
 - b. steroid therapy.
 - c. diabetes.
 - d. neutropenia.
 - e. all of the above.
14. Pyomyositis:
 - a. is rarely caused by *S. aureus*.
 - b. can be treated topically.
 - c. occurs in tropical areas and in the immunosuppressed.
 - d. heals spontaneously within two weeks.

15. Erysipelas:

- is a systemic infection that requires hospitalization.
- tends to occur in areas of impaired lymphatic drainage.
- is characterized by lesions with indistinct borders.
- rarely recurs.

16. Herpetic whitlow:

- is caused by the herpes simplex virus.
- requires incision and drainage.
- commonly occurs on the feet.
- is not contagious.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives an alpha-numerical score based on the following definitions.

Class I

- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness
- Must be used in the intended manner for proper clinical indications

Level of Evidence:

- One or more large prospective studies are present (with rare exceptions)
- Study results consistently positive and compelling

Class IIa

- Safe, acceptable
- Clinically useful
- Considered treatments of choice

Level of Evidence:

- Generally higher levels of evidence
- Results are consistently positive

Class IIb

- Safe, acceptable
- Clinically useful
- Considered optional or alternative treatments

Level of Evidence:

- Generally lower or intermediate levels of evidence
- Generally, but not consistently, positive results

Class III:

- Unacceptable
- Not useful clinically
- May be harmful

Level of Evidence:

- No positive high-level data
- Some studies suggest or confirm harm

Indeterminate

- Continuing area of research
- No recommendations until further research

Level of Evidence:

- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

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