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Bacterial Skin and Soft-Tissue Infections: A Systematic Approach to Diagnosis and Treatment

Part I: Cellulitis, Furunculosis, and Distal Finger Infections

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This two-part series discusses bacterial skin and soft-tissue infections (SSTIs). Part 1 will discuss bacterial skin infections that quite frequently are encountered in the general practice of medicine and that predominantly are benign in nature. With proper diagnosis and treatment of these entities, no major complication or long-term sequellae should be expected. Part 2 will cover bacterial SSTI diseases that are less benign and more complicated, and/or occur less frequently and that, even with proper therapy, have the potential for severe complications and sequellae.

– The Editor

Introduction

Bacterial infections of the skin and underlying soft tissue encompass a wide variety of clinical entities and are common reasons for presentation to the emergency department (ED) or a primary care provider's office. They range from simple pyodermas characterized by local induration, swelling erythema, warmth, and pain or tenderness that readily can be treated with local care and oral antibiotics, to more severe, life-threatening, necrotizing infections that are accompanied by systemic symptoms such as fever, chills, and, at times, hemodynamic instability, and require hospitalization and surgical intervention. SSTIs sometimes may become disseminated via the circulatory and lymphatic system, particularly in patients who are immune compromised. While many of these infections can be treated in the outpatient setting, moderate or severe cases may require hospitalization and parenteral antibiotic therapy. In 1995, it was estimated that more than 330,000 patients required

hospitalization for SSTIs, with many more treated in the outpatient setting.[1]

The Skin

A healthy normal stratum corneum layer of skin is a natural barrier to bacterial penetration. The exact mechanism of this resistant barrier is not fully understood, however many factors that are directly related to skin play a significant role in the provision of this first line of defense. These factors include the keratinized nature of the layer, skin's natural acidity (pH: 5.5) and relative dryness, skin's normal microflora, skin's continuous desquamation and sloughing, and presence of antibacterial substances and intracellular lipids.[2] Any derangement in any one of these factors can predispose the skin to a variety of infections. Damaged skin is susceptible to bacterial colonization, proliferation, and invasion. This phenomenon occurs even with a minor break in the stratum corneum layer. Once this layer is breached, rapid penetration of bacteria will result in dermal echthymatous lesions or deeper cellulitis. In a more extensive wound, the resultant build-up of serum will permit the growth of the pathogenic bacteria and the rapid replacement of the resident flora. Furthermore, an overall decline in the individual's cellular or humeral immune system also will make the skin susceptible to infection.

Skin and Soft-Tissue Infections

Bacteriology. Bacteriology of the SSTIs differs significantly based upon the depth and level of the infection (epidermal vs dermal vs soft-tissue) and the actual infected tissue (skin or soft tissue vs skin appendices).

Gram-positive bacteria cause the majority of the SSTIs. However, gram-negative organisms (primarily *Pseudomonas aeruginosa*) are occasionally the causative pathogens, particularly in the groin, ear, foot ulcers, and macerated interspace infections. Anaerobic organisms may play a significant role in the deep soft-tissue and necrotizing infections.

Staphylococcus aureus and Group A beta-hemolytic Streptococcus (GAS) are the primary pathogens involved in the primary and secondary SSTIs in outpatient settings.[3] *S. aureus* commonly is found in the environment and on the skin; however, the normal bacterial flora act as the first line of defense. While *S. aureus* often invades the skin via hair follicles, Streptococci invade the skin through the breaks in the stratum corneum layer.

Methicillin-resistant *Staphylococcus aureus* (MRSA) has emerged as an important and common pathogen among hospitalized patients since the 1960s.[4] According to estimates from the National Nosocomial Surveillance System of the Centers for Disease Control, the incidence of MRSA causing nosocomial infections in intensive care unit patients has approached 50%.[5] Recent surveillance data suggest that almost 30% of *S. aureus* isolated from hospitalized patients with SSTIs is methicillin–resistant.[6] Established risk factors for MRSA infections include: prolonged hospitalization or surgery, care in an intensive care unit, prolonged antimicrobial therapy, residence in a long-term care facility, dialysis, close proximity to a patient in the hospital who is infected or colonized with MRSA, and intravenous drug use.[7] Recently, cases of MRSA infection have been reported in both rural and urban communities and in patients without traditional risk factors. Many cases of community-acquired MRSA (CAMRSA) have occurred in children and young adults who typically do not have the associated health care related risk factors. While some of these strains may have origins in the hospital, others appear to be novel and unrelated to known hospital strains.[8] MRSA colonization can persist for months to years and the majority of patients remain asymptomatic. Therefore, the acquisition of MRSA, whether it occurs in the hospital or the

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Table 1. Risk Factors for Specific Pathogens Causing SSTI

RISK FACTOR	CHARACTERISTIC PATHOGEN
Diabetes	*S. aureus*, group B streptococcus, Gram-negative bacilli
Cirrhosis	*Camphylobacter fetus*, *Klebsiella pneumoniae*, *E. coli*, *Capnocytophaga canimorsus*, other Gram-negative bacilli, *Vibrio vulnificus*
Neutropenia	*P. aeruginosa*
Human bite	Oral anaerobic flora, *S.aureus*, *Eikenella corrodens*
Cat bite	*S. aureus*, *Pasteurella multocida*
Dog bite	*S. aureus*, *P. multocida*, *Capnocytophaga canimorsus*
Rat bite	*Streptobacillus moniliformis*
Animal contact	Camphylobacter spp.
Reptile contact	Salmonella spp.
Hot tub exposure	*Pseudomonas aeruginosa*
Fresh water exposure	*Aeromonas hydrophila*
Salt water exposure	*V. vulnificus*, *Mycobacterium marinum*
Intravenous drug use	Anaerobes, MRSA, *P. aeruginosa*
Soil contamination	Clostridium spp. Pseudomonas spp.
Puncture wound of foot	*P. aeruginosa*
Skin trauma in butchers and fishermen	*Erysipelothrix rhusiopathiae*

community, often will go unrecognized unless clinical infection develops.[9] In one study 84% of CAMRSA infections involved the skin and soft tissues (especially abscess and cellulitis), with invasive infections occurring only rarely and usually secondary to a primary SSTI.[10,11] Patients with CAMRSA skin infections were more likely to have received a prescription for an antimicrobial agent in the 180 days preceding the infection than were the group of patients with methicillin-susceptible *S. aureus* skin infections.[11]

The CDC has reported several clusters of CAMRSA SSTIs involving participants in competitive sports in a variety of settings, including high school and college athletic teams, and even in participants in minimal contact sports such as fencing. Physical contact, skin damage, and sharing of equipment, clothing and other personal items that are not laundered between uses were identified as possible risk factors for infection. Sharing of balms and lubricants also may spread infection. Encouraging good hygiene among players and implementing a system to ensure adequate wound management, including covering skin lesions before play and routine cleansing of shared equipment, may assist in controlling the spread if infection.[12] Physicians should be aware of the potential for CAMRSA infections in sports participants when evaluating patients and making treatment decisions. Recurrence of infection potentially may be avoided if cultures are obtained more routinely when athletes have infected wounds. CAMRSA infections also have been reported in both male and female correctional facilities and in men who have sex with men.[10]

Most CAMRSA isolates are susceptible to antibiotics other than beta-lactams, whereas most nosocomial isolates are resistant to antibiotics other than beta-lactams.[4] The emergence of CAMRSA in otherwise healthy young individuals has significant implications in the management of SSTIs, as most SSTIs initially are managed with beta-lactam antibiotics. Inadequate empiric antimicrobial therapy may allow CAMRSA infections to progress, resulting in more serious infections and clinical complications. Obtaining cultures of infected sites is highly recommended when staphylococcal infection is suspected. Optimal treatment of CAMRSA disease should be based on the infecting organisms' antimicrobial susceptibility when available. To prevent clinical complications, health care providers should consider modification of empiric antibiotic regimens in areas where CAMRSA infections are prevalent. Most of these isolates still are susceptible to multiple antimicrobial classes (including trimethoprim-sulfamethoxazole, tetracyclines, and clindamycin), however many CAMRSA isolates are beginning to demonstrate resistance to fluoroquinolones.[10] Treatment of CAMRSA infections should not routinely require the use of vancomycin.[4]

There has been considerable debate regarding the benefits, timing, and methods used to culture SSTIs. Culturing the skin in cases of cellulitis rarely yields a pathogen; however, obtaining a specimen for culture from other wounds for gram stain and culture frequently is useful, especially if the prevalence of MRSA in the community is high. Local antibiotic resistance patterns should be considered when deciding whether to obtain specimens for culture and sensitivity; the lower the prevalence of resistant stains, the less the need to culture.

In specific and clinical or epidemiological circumstances, a number of other infectious pathogens also may cause infections. *(See Table 1.)*

Diagnosis, Management and Classification. Bacterial SSTI must be differentiated from a wide range of non-bacterial or non-infectious skin and soft-tissue diseases and disorders. These diseases may include, but are not limited to, localized and systemic allergic reactions, viral exanthematous rashes, skin manifestation of systemic diseases, insect and marine animal bites and envenomations, parasitic infestations, and drug eruptions.

A successful diagnosis of SSTI depends on the appearance of the lesion; chronology of the process; patient's age; medical history and the integrity of the immune system; evidence of systemic manifestation of infection; history of trauma or surgery; unusual travel; activity; and insect, animal, or human bite(s).

Optimal management of SSTIs begins with a physical examination and obtaining the complete medical and social history of the patient. SSTIs, however, can pose considerable diagnostic and therapeutic challenges. These infections can affect a wide variety of tissues and are caused by a variety of microorganisms. The clinical manifestations are wide ranging and host

factors, which may contribute to the development of these infections, may not be readily apparent. Culturing the causative organism often is difficult and takes several days; hence, initial treatment regimens usually are empiric and targeted at the most common organisms.

Classification. For classification purposes, bacterial skin infections are divided into infections of skin appendices and infections of skin.

Infections of the Skin Appendices

The hair follicles and their supporting and surrounding structures frequently are infected skin appendices. *S. aureus* is the most common causative pathogen. These infections can be classified into the types that follow.

Folliculitis. Folliculitis is an inflammation of the hair follicle. It commonly occurs in areas of skin that are covered by coarse, short hair. Obstruction of the adjacent sebaceous gland is the primary cause of the infection. Friction is a common predisposing factor. Application of mineral-based oils is another predisposing factor. The majority of folliculitis cases are caused by *S. aureus*.[13]

Folliculitis is manifested by patches of 1-5 mm erythematous papules or pustules at the base of the hair follicle. It usually is non-tender; occasionally it can be pruritic or painful. Pustules have a dirty yellow or gray color. Manipulation of folliculitis may result in transient bacteremia. Gram-negative folliculitis, caused by organisms such as *Klebsiella* or *Proteus*, may occur in patients who have been on chronic antibiotic therapy.

Hot tub or spa pool folliculitis is caused by *Pseudomonas aerugenosa*. The organism proliferates in warm and moist environment, especially if the water has high pH (</ = 7/8) or is poorly chlorinated. It also may grow on exfoliative beauty aids (loofah or synthetic sponges). Even normal hosts with intact skin are affected. The resultant folliculitis occurs predominantly in the confined areas of the skin that are covered by the bathing suit and/or immersed in the water. Folliculitis usually is manifested within 6-120 hours post exposure. Papular lesions progress rapidly to pustules or vesicles and may be pruritic. Lesions also may occur in the auditory canal. Patients may experience low-grade fever, headache, dizziness, malaise, and sore throat. Most cases are mild and self-limiting.[13-16]

Treatment. Isolated Folliculitis. Use of warm compresses and gentle cleaning of the lesion(s) with topical benzol peroxide or an antibacterial soap should be a sufficient therapy. Use of topical antibiotics, such as mupirocin, erythromycin, or clindamycin also should be considered and may hasten recovery. Use of systemic antibiotic therapy is not required. With proper approach, the lesions usually resolve without any residual scarring.

Extensive or Refractory Folliculitis. Systemic anti-staphylococci antibiotic therapy should be added to the above-mentioned therapeutic modalities. First-generation cephalosporins (cephalexin 500 mg PO bid x 10 days) or azythromycin (500 mg, day 1 and 250 mg days 2-5, PO qd) are first-line antibiotic agents. Alternative antibiotics include moxifloxacin, ampicillin-clavulanate, dicloxacillin, erythromycin, or minocycline. In refractory cases, the presence of MRSA must be considered and ruled out.

Hot Tub Folliculitis. In most healthy patients, lesions resolve spontaneously, and scarring is rare. If treatment is necessary, it should include topical 0.1% polymyxin B. In adults with severe or refractory cases, oral ciprofloxacin (500 mg PO bid x 7-10 days) should be considered as the drug of choice. In children with severe infection, an anti-pseudomonas semi-synthetic penicillin should be utilized.

Furunculosis (Boil). Furunculosis is due to the extension of folliculitis into the surrounding subcutaneous tissue. Having one episode is a reliable predictor of future similar events. Recurrent furunculosis is a challenging problem in patients suffering from diabetes mellitus, atopic dermatitis, immune deficiency (congenital or acquired), alcoholism, obesity, or malnourishment and use of corticosteroids or cytotoxic agents. These patients may be colonized by *S. aureus* in their nares, or on their skin.

Furunculosis is manifested by an erythematous, deep-seated, painful nodule adjacent to a hair follicle. It can grow rapidly and become fluctuant. The lesion may rupture spontaneously into an ulcer. At times, it is complicated by bacteremia and possible hematogenous secondary seeding of heart valves, long bones and spine, joints, meninges, and viscera (especially kidneys).

Outbreaks of persistent furunculosis on the lower extremities have been reported in patrons of nail salons. The causative organism has been identified as *Mycobacterium fortuitum*. There usually is more than one lesion associated with risk factors such as use of whirlpool footbaths and shaving of the legs with razor before pedicure.

Several conditions can be mistaken as furunculosis. Other lesions and diseases such as hydradenitis suppurativa, herpes simplex infections, fire ant bites, kerions, and molluscum contagiosum must be considered as an alternative diagnosis and excluded. Obtaining a travel history also is very important. In a patient with recent travel to Mexico or Central or South America, the diagnosis of larva of botfly (*Dermatobia hominis*) must be excluded.[17]

Treatment. Simple Furunculosis. In most cases treatment consists of application of local heat. Incision and drainage is the treatment of choice and commonly is required. In most cases, systemic antibiotic therapy is not necessary.

Furunculosis with Surrounding Cellulitis or Systemic Signs. Incision and drainage is the treatment of choice. Use of a systemic antibiotic, such as first-generation cephalosporins (cephalexin 500 mg PO bid x 10 days) or azythromycin (500 mg, day 1 and 250 mg days 2-5, PO qd) usually is required. Other acceptable antibiotics are dicloxacillin, clindamycin, or erythromycin.

Recurrent Furunculosis. In this patient population, colonization of nares or skin by *S. aureus* should be evaluated and, if positive, treated. Therapeutic regimens include use of topical mupirocin ointment or cream every six hours for seven days. Furthermore, diabetes mellitus must be ruled out. Addition of systemic oral antibiotics such as clindamycin (150 mg PO qd x 3-months) (best results), rifampin (600 mg PO qd x 7-10 days), or cloxacillin (500 mg q 6 hours x 7-10 days), also should be considered.[18,19]

Carbuncle. Carbuncle is the result of confluence of several furuncles and further lateral extension of the infectious process

Table 2. Bacteriology and Epidemiology of Cellulitis

ORGANISM	SPECIFIC CONSIDERATION
S. aureus	Centripetally spreading Recurrent cellulitis
Group A, C & G Strep. spp.	Lower extremities cellulites Associated with chronic venous stasis
Group B Strep.	Cellulitis in newborns
H. influenzae	Cellulitis of head and neck in children
Pseudomonas aerugenosa	Associated with penetrating injuries
Aeromonas hydrophilia	Associated with lacerations sustained in fresh water
Erysipelothrix rhusiopathiae	Associated with bone renderers and fishermen
Pasteurella multocida	Associated with cat and dog bites
Eikenella corrodens	Associated with human bites
Vibrio vulnificus	Associated with injuries sustained in salt water
Mycobacterium marinum	Associated with wounds sustained in aquariums or swimming pools

into the surrounding soft tissue. It usually is secondary to manipulation or neglect of the initial furunculosis. It will result in the destruction of the normal fibrous tissue walls and formation of tunnel-like, interconnected sinus tracts and abscesses.[13]

Carbuncle is manifested as a large, erythematous, deep-seated, and very painful lesion. Systemic signs such as fever, chills, malaise, and lymphadenopathy usually are present. Its complications may include cellulitis, septicemia, thrombophlebitis, and secondary bacterial seeding(s).

Treatment. The main therapeutic approach is through incision and drainage of the abscesses. Oral or parenteral systemic anti-staphylococci antibiotics also should be administered. Choices of antibiotic therapy are the same as for furunculosis. Careful exploration of the lesion with a blunt object (i.e., a hemostat) may be necessary to break up any loculated abscess.

Infections of the Skin

Impetigo Contagiosa. Impetigo is a superficial indolent infection of the skin.[20] It is the most common skin infection in children. The causative pathogens include group A beta-hemolytic Streptococcus (GAS) and *S. aureus*. Co-infection by both bacteria also is common. Impetigo easily can be spread from person to person. Due to predilection for a warm, moist environment, late summer and early fall are the peak seasons for the occurrence of this disease.

Impetigo initially is manifested as scattered, discrete, small papules. These lesions will transform into pustules and vesicles. Rupture of these pustules or vesicles will result in the impetigo's 1.0-3.0 cm characteristic golden-yellow, stuck-on crusts. These lesions have an erythematous and inflammatory base. Lesions can be pruritic and occur predominantly on the face and near the mouth and nose. Lesions also may occur at the site of minor trauma. Satellite lesions are fairly common. Regional lymphadenopathy may be present. Rare complications include glomerulonephritis.

Bullous impetigo is a variant of this disease. It presents as a large flaccid bullae filled with a yellow liquid. Following the rupture of the bullae, lesions with a scalded nature appear. The surrounding skin has a superficial erosive pattern. The causative Phage II *S. aureus* produces the extracellular exfoliating exotoxin.[21]

Treatment. In mild cases, topical antibiotic creams and ointments, such mupirocin or bacitracin (topical tid), can be used safely.

In refractory or more severe and widespread cases, systemic oral antibiotics such as amoxicillin-clavulanate or cephalosporins for *S. aureus* should be prescribed. Advanced-generation cephalsporins (e.g., cefdinir) are resistant to beta-lactamase and maybe a better therapeutic choice. Alternative agents include dicloxacillin or erythromycin.

Ecthyma (Ulcerative Impetigo). Ecthyma is an ulcerative infection of the skin, caused most commonly by group A beta-hemolytic Streptococcus or *S. aureus*. It can affect patients of all ages and usually is seen in crowded living conditions. It is associated with poor hygiene, heat, humidity, and poor nutritional status. Occasionally *Pseudomonas aeruginosa* also can be the causative bacteria. Skin lesions caused by *P. aeruginosa* usually are metastatic seeding of a septicemia and are called ecthyma gangerenosum.

Ecthyma is manifested as superficial, punched-out, ulcerative lesions associated with adherent necrotic crust and raised inflammatory borders. Occasionally it may present as a vesicle or pustule. Lesions may be painful or pruritic. Lower extremities mostly are affected. Lesions often occur at the site of minor trauma. The lesions are slow to heal and often result in scarring.

Treatment. Debridement of the necrotic tissue and incision and drainage of the fluctuant vesicles and pustules must be performed. Systemic oral anti-staphylococcus antibiotic also must be administered. Choices of antibiotic agents are the same as the treatment of impetigo contagiosa.

Erythrasma. Erythrasma is a chronic bacterial infection of the intertriginous areas of toes, groins, sub-mammary, gluteal and axilla. It predominantly affects overweight adults. Diabetes mellitus is a predisposing factor. *Corynebacterium minutissimum* is the causative pathogen.

Erythrasma is manifested as erythematous macular lesions covered with fine scaling on a sharply marginated background. The lesions can be scattered or confluent patches. It can be misdiagnosed as Tinea cruris.

Treatment. Econazole cream, which has both antifungal and antibacterial activity, can be used topically. Topical antibiotics are not effective. In extensive cases, systemic oral antibiotics such as macrolides (e.g., erythromycin 250 mg every 6 hours for 14 days) can be used.

Cellulitis. Cellulitis is an acute deep infection of the skin involving the dermis and subcutaneous tissue. Bacteria usually gain access to the epidermis through skin cracks or following minor trauma or burns; however, an entry portal may not be evi-

dent. Risk factors include diabetes mellitus, hematological malignancies, IV drug abuse, chronic lymphedema, and immunocompromise.

Bacteriology and Epidemiology. Cellulitis caused by *S. aureus* spreads centripetally from a localized source of infection such as furunculosis. In contrast, cellulitis due to *Streptococcus pyogenes* is a more rapidly spreading, diffuse process and usually is associated with fever and lymphangitis. *(See Table 2.)*

Recurrent Streptococcal cellulitis of the lower extremities may be caused by group A, C, or G Streptococci in association with chronic venous stasis, saphenous venectomy for cardiac bypass surgery (CABG), or chronic lymphedema secondary to lymph node dissection, elephantiasis, or Milroy's disease. Recurrent *S. aureus* cellulitis is more common in individuals who have eosinophilia and elevated serum levels of IgE (Job's syndrome) and among nasal carriers of the bacteria.

Group B streptococcus (GBS) is the causative pathogen for cellulitis in newborns.

Cellulitis caused by *Streptococcus agalactiae* is seen frequently in patients with diabetes mellitus or peripheral vascular disease.

Haemophilus influenzae is the causative pathogen for periorbital, head, and neck cellulitis in children who are suffering from sinusitis, otitis media, or epiglottitis. Most patients are younger than 3 years of age.

Pseudomonas aeruginosa cellulitis frequently follows penetrating injuries, i.e., sweaty tennis shoe syndrome. Bacteria are introduced into the deep tissues by stepping on a nail. In a neutropenic patient, *P. aeruginosa* infections also can present as ecthyma gangrenosum. These lesions usually occur on the extremities and rapidly become necrotic and ulcerative.

Aeromonas hydrophilia causes very aggressive cellulitis in tissues surrounding lacerations sustained in freshwater lakes, rivers, and streams.

Erysipelothrix rhusiopathiae cellulitis occurs predominantly in bone renderers and fishermen.

Pasturella multocida infections are prevalent in cat and dog bites. *Eikenella corrodens* infections are frequently secondary to human bites. (Both of these topics will be discussed in more detail).

Vibrio vulnificus cellulitis occurs predominantly on the extremities following exposure to sea water.

Mycobacterium marinum may cause cellulitis in patients who are exposed to aquariums or are injured in a swimming pool.

Presentation. Systemic signs, fever, chills, anorexia, or malaise may develop rapidly. High fever usually is associated with streptococcal infections. Lesions present as edematous, erythematous, hot plaques of severe tenderness of various sizes. The borders usually are irregular and elevated but sharply defined. Lesions may proceed to vesicles, bullae, erosions, abscesses, hemorrhage, and necrosis. Among other bacteria, *H. influenzae* and pneumococcal cellulitis may cause a bluish-purple discoloration of the involved skin.

Complications of cellulitis may occur either through local spread (osteomyelitis or arthritis), metastatic (arthritis, infectious endocarditis, and meningitis), or systemic (sepsis).

Diagnosis. Diagnosis primarily is made clinically. Isolation of the etiologic pathogen often is difficult. In most cases the causative pathogen can be suspected based upon the epidemiologic data. Unless there is drainage or an open wound, even with the aspiration of the leading edge or punch biopsy, cultures are positive in fewer than 25% of cases. Nevertheless, cultures should be obtained in severely ill or immunocompromised patients.[22]

Treatment. Uncomplicated Cellulitis. Systemic oral anti-staphylococcal antibiotics should be administered. In adults, acceptable first-line antibiotics may include first-generation or advanced-generation cephalosporins, amoxicillin-calvulanate, clindamycin, advanced flouroquinolones, erythromycin, and dicloxacillin. Considering the ease and frequency of administration and the resultant higher level of patient compliance with therapy, azythromycin (500 mg day 1 and 250 mg days 2-5 PO qd) or moxifloxacin (400 mg PO qd x 7 days) may be considered better therapeutic agents. Similarly, in children, azythromycin, amoxicillin-clavulanate, first- and second-generation cephalosporins, clindamycin, and dicloxacillin are drugs of choice.

Extensive, Complicated or Refractory Cellulitis. Systemic parenteral anti-staphylococcal antibiotics should be used. In refractory cases, MRSA infection should be excluded.

Cellulitis Associated with Diabetes Mellitus or Pressure Ulcers. Broad spectrum systemic parentral antibiotics to provide coverage for gram-positive, gram-negative, and anaerobic bacteria must be started. Antibiotics of choice include fluoroquinolones (ciprofloxacin 500 mg PO bid x 14 days), advanced-generation cephalosporins, ampicillin-sulbactam, ticarcillin-clavulanate, or aminoglycosides plus clindamycin.

Pathogen-specific Antibiotic Therapy. *Pseudomonas aeruginosa*. Drugs of choice include anti-pseudomonas third-generation cephalosporins (ceftazidime, cefoperazone, or cefotaxime), anti-pseudomonas semi-synthetic penicillins (ticarcillin, mezlocillin, or pipracillin), or fluoroquinolones. In cellulitis associated with penetrating injuries involving deep tissue, bones, or joints, surgical inspection and drainage must accompany antibiotic therapy.

Pasteurella Multocida. This bacteria is resistant to dicloxacillin and naficillin, but sensitive to other beta-lactam antibiotics. Other options include quinolones, tetracycline, and erythromycin.

Eikenella Corrodens. Ampicillin-clavulanate, ampicillin-sulbactam, or cefoxitin are drugs of choice. Quinolones are an acceptable alternative.

Aeromonas Hydrophilia. This bacteria is resistant to ampicillin. Drugs of choice include aminoglycosides, advanced fluoroquinolones, chloramphenicol, trimethoprim-sulfamethoxazole, or advanced-generation cephalosporins.

Erysipelothrix Rhusiopathiae. This bacteria remains sensitive to penicillin, erythromycin, clindamycin, tetracycline, and cephalosporins. It is resistant to sulfonamides and chloramphenicol.

Mycobacterium Marinum. Although no comprehensive studies have been conducted, drugs of choice may include rifampin plus ethambutol, tetracycline, or trimethoprim-sulfamethoxazole.

Cellulitis of Specific Areas

Erysipelas. Erysipelas is a superficial skin infection that is associated with lymphatic involvement. It is most exclusively caused by *Streptococcus pyogenes*.[23] On occasions, other pathogens such as non-group A Streptococci (B, C, or G), *H. influenza*, *S. aureus,* and *Streptococcus pneumoniae* (rare) also may be the culprit.[24] It is more common in children and the elderly. The predisposing factors include diabetes mellitus, venous stasis, alcoholism, and chronic lymphatic obstruction and lymphedema. It is fairly common in patients with radical mastectomy and post-operation chest wall radiation therapy. One-third of cases have a recurrent pattern. Patients with either venous or lymphatic stasis are more prone to recurrence. Repeated infections also may cause impairment of lymphatic drainage. In neonates, infection may occur in the umbilical cord stump and spread rapidly over the abdomen.

Presentation. Erysipelas presents with a rapidly expanding, intensely erythematous, edematous, firm, tender, and painful plaque. The borders are well defined and there is no central clearing. The size of the plaque is variable. The common sites for lesions are the face (close to the nares or ears) and the lower extremities. The infections of lower extremities now are more common than the facial infections. Fever, chills, diaphoresis, anorexia, vomiting and other systemic signs are fairly common. Regional lymphadenopathies usually are present. Flaccid bullae may appear during the second or third days of presentation. Lesions rapidly may progress to lymphangitis, cellulitis, abscess, or faciitis.[25] Less acute cases may occur at the leg stasis ulcer sites.

Diagnosis. In most cases the diagnosis is made based on clinical appearance. Cultures rarely are useful.

Treatment. Treatment with a parenteral systemic antibiotic must be initiated immediately. After 4-5 days, antibiotic therapy can be switched to an oral form. The treatment of choice is benzyl penicillin followed by penicillin VK for a total of 10-14 days. Other antibiotic agents include dicloxacillin, nafcillin, first-generation cephalosporins, cefuraxime, and erythromycin. In diabetic patients or in those with facial involvement, the clinician should consider the possibility of infection with beta-lactamase producing organisms. In these cases, use of amoxicillin-clavulanate or advanced-generation cephalosporins is a necessity.

Pyoderma. Pyoderma is due to the secondary infection of a pre-existing skin lesion. Primary lesions include stasis ulcers of lower extremities, eczema, or exfoliative skin disease. The causative pathogen is predominantly *P. aeruginosa*. The infectious process can be aggressive, rapidly progressing, and invasive or may follow a more indolent course. The acute cases present in a moth-eaten pattern surrounded by a raised erythematous border. The chronic cases are manifested as a slowly growing and burrow-forming lesions. Subsequently, these lesions become papulovesicular crops and are covered with a malodorous crust.

Diagnosis. Causative bacteria readily may be cultured from both the acute and chronic lesions.

Treatment. Systemic oral anti-pseudomonas antibiotics should be administered. The drug of choice is ciprofloxacin.

Cellulitis Around the Eye

Cellulitis around the eye is a potentially dangerous process. An intact orbital septum prevents the progression of the inflammatory processes of the eyelids to expand posteriorly. Periorbital cellulitis must be differentiated from orbital cellilitis. CT scans can be a very useful tool in the differentiation process.

Periorbital Cellulitis. Periorbital cellulitis is an acute inflammatory process that is limited to the eyelids. It is more common in children and usually is subsequent to upper respiratory tract infections. Eye or orbit trauma may act as a predisposing factor. Systemic manifestations, such as fever, also may be present. Patients may develop conjunctivitis, too. Cellulitis predominantly is caused by *S. aureus* and *Streptococcus pyogenes* in adults and *H. influenzae* in children. The clinician should consider the significant overall reduction of *H. influenza* infections in the United States and other advanced countries secondary to the widespread use of the HIB vaccine. Periorbital cellulitis rarely is associated with central nervous system (CNS) complication.

Treatment. Adults without any systemic manifestation may be treated with warm soaks and systemic oral anti-staphylococci antibiotics. Young children should be treated more aggressively with a systemic parenteral antibiotic. Advanced-generation cephalosporins, i.e., ceftriaxone, are the antibiotic of choice.

Orbital Cellulitis. Orbital cellulitis is a true emergency. The infection predominantly is secondary to acute sinusitis. The infection spreads either by direct extension or in a retrograde pattern through the facial veins. *S. aureus* is the main pathogen in adults and *H. influenzae* infections are most common in children. Blood cultures are positive in up to 60% of pediatric cases. The cellulitis is associated with proptosis, chemosis, orbital pain, and restriction of eye movements. Visual disturbances occur in more than half of patients. Abscess formation is a common complication and should be ruled out by a CT scan of the orbit. Other complications include permanent blindness, ophthalmoplegia, and diplopia. Meningitis, cavernous sinus thrombosis, and brain abscesses are other rare complications.

Treatment. Systemic intravenous antibiotic therapy should be initiated immediately. Ceftriaxone is the drug of choice. It penetrates the CNS very well and is very effective against *H. influenzae.*

Cellulitis Around the Anus

Perianal Cellulitis. Perianal cellulitis occurs more frequently in children than adults, and boys are affected more often than girls. The children are not appear to suffer from systemic illness. It may be associated with pharyngitis. *Streptococcus pyogenes* is the most common pathogen. Cellulitis presents as bright erythematous perianal patches extending from the anal verge. It often is associated with painful defecation, anal pruritis, blood streaking of the stool, and anal tenderness. This bacterial infection commonly is misdiagnosed as Candidiasis. Correct diagnosis may require obtaining a culture. Recurrence is frequent.

Treatment. Topical antibiotics are useful; however, addition of a systemic oral antibiotic therapy is indicated in most cases. Drugs of choice include penicillin or erythromycin. Therapy should be continued for 10-14 days.

Infections of Distal Fingers and Toes

Based upon the location and the depth of infection, the bacterial infections of distal fingers and toes are divided into paronychia and felon.

Paronychia. Paronychia is a superficial cellulitis or abscess of the paronychial (nail fold) tissues of the hands and feet. Any disruption of the area between the nail bed and nail fold can act as a port of entry for the pathogenic organism. This disruption may be secondary to a minor trauma following nail bed manipulation or spontaneous. Paronychia is one of the most common infections of the hand. The infectious process may proceed either in an acute or chronic mode. Paronychia also may be secondary to non-infectious causes, i.e., chemical irritants and chronic excessive moisture.

Acute Paronychia. Acute paronychia presents with a rapid onset of localized erythematous, tender, and edematous lesions of the lateral and proximal nail folds. A pustular lesion may develop within few days. Pus may penetrate into the nail bed and cause either discoloration and/or elevation of the nail from the underlying matrix. If the infection remains untreated, an abscess also may develop. Drainage of the abscess should result in the abrupt relief of pain. Persistence of pain should suggest a deeper infectious process. If the abscess is left unattended, the pus may spread to the opposite side and cause a run-around abscess.

Acute paronychia commonly are encountered in patients who suck their fingers, bite their nails, have hang nails, or have penetrating trauma. Aggressive manicuring and artificial sculptured nails also may predispose the patient to this infection.

The most common pathogenic organisms are *S. aureus*, in majority of cases, and Streptococci species. In rare occasions, specifically in nail-biting or finger-sucking children, infection may be due to the bacteria that are considered as normal mouth flora. These specific pathogens are the same organisms that cause wound infections following human bites. (This will be discussed in detail in Part 2 of this article).

On occasion, *P. aeruginosa* also may cause a form of acute paronychia. These causes usually are a secondary bacterial infection and follow a primary dermatophytes infection of the nails or a minor trauma to the distal phalanx. The involved skin appears as a pinkish-white in color, associated with moderate to severe pain and edema. Lesions have a tendency to become ulcerative and macerated. *P. aeruginosa* also may cause the green nail syndrome. Subsequent to the development of paronychia, and due to pyocyanin pigment, the adjacent nail becomes develops a greenish discoloration. Even with proper treatment, this benign discoloration may last for months.

Treatment. Conservative therapy, such as warm soaks and possibly topical antiobiotics, may be effective in the early phase of acute paronychia. A more advanced infection or the development of an abscess will necessitate the use of oral anti-staphylococcal antibiotic agents. The drugs of choice include amoxicillin-clavulanate or clindamycin. The frequently encountered resistance patterns associated with *S. aureus* should negate the use of antibiotics such as penicillin, ampicillin, or first-generation cephalosporins.

With the development of an abscess, an incision and drainage procedure will be required. In more advanced stages, the removal of the involved nail may be necessary.

Paronychia caused by *P. aeruginosa* should be treated with topical dilute acetic acid (0.25-1.0%) or polymyxin B (0.1%).

Chronic Paronychia. Chronic paronychia is rare following the acute disease. It is more common in individuals whose hands are chronically moist. Manipulation of the nail cuticles also can predispose the individual to this disease. Most commonly, all fingernails are affected simultaneously. The infection is insidious and presents with separation of the cuticle, tenderness, and edema of the proximal and lateral nail folds. Occasionally a small amount of pus can be expressed from under the nail fold. Although a brownish discoloration of the nail may occur, the nail plate is not affected and remains intact. Lack of subungual thickening will differentiate chronic paronychia from fungal infections. This infection can be due to gram-positive or gram-negative organisms or Candida.

Treatment. Avoidance of the predisposing factors plays a significant role in treatment of chronic paronychia. Systemic oral antibiotics or antifungal medications do not effectively penetrate the infectious site and generally are ineffective. Treatment with a combination of topical steroids and antifungal agents have proven to be successful. Other treatment modalities may include application of topical 3% tymol in 70% ethanol drops. Addition of fluconazole to this regimen also may improve the therapeutic outcome and control the recurrence. Other therapeutic options may include use of topical antibiotics or acetic acid soaks (1:1 ratio of vinegar to water). Excellent responses have been reported with eponychial marsupialization technique or complete removal of the infected nails, followed by the application of the above-mentioned topical treatments.

Felon. Felon is a serious infection that involves the pulp of the distal phalanx of a finger. It usually is secondary to a minor injury, a localized wound, or extension of a localized superficial infection. Infection is manifested as a severely painful, erythematous, hot, and edematous area involving the palmar aspect of the distal phalanx. *S. aureus* and *Streptococcus pyogenes* are the most common pathogens. Gram-negative organisms also occasionally have been implemented. If neglected, undiagnosed, or untreated, it commonly is complicated with abscess formation and loss of tissue. In these situations, secondary loss of function is a very common occurrence.

Treatment. Surgical intervention remains the first line of therapy. Systemic intravenous anti-staphylococci antibiotics also should be administered promptly.

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Physician CME Questions

121. Which one of the following factors does *not* play a significant role in the function of skin as a natural barrier to bacterial penetration?
    A. Skin's relative high moisture
    B. Continuous desquamation of skin
    C. Keratinized nature of skin's superficial layer
    D. Skin's normal pH

122. In outpatients, most of the skin and soft-tissue infections are caused by:
    A. group A beta hemolytic streptococcus.
    B. *Staphylococcus aureus.*
    C. A and B.
    D. Gram-negative rods.

123. Community acquired methicillin-resistant *Staphylococcus aureus* is usually susceptible to which of the following antibiotics?
    A. Trimethoprim-sulfamethoxazole
    B. Tetracyclines
    C. Clindamycin
    D. All of the above

124. In acute cellulitis culture of the aspiration of the leading edge or punched biopsies is positive in what percentage of cases?
    A. > 15%
    B. 25%
    C. 50%
    D. 75%

125. If antibiotic therapy is required for the treatment of hot tub folliculitis, which one of the following is the drug of choice?
    A. Amoxicillin-clavulanate
    B. Ciprofloxacin
    C. Tetracycline
    D. First-generation cephalosporin

126. In a patient with travel history to Central and South America, infestation of skin with larva of botfly (*Dermatobia hominis*) must be included in the differential diagnosis of which of the following skin infections?
    A. Cellulitis
    B. Folliculitis
    C. Impetigo contagiosa
    D. Furunculosis

127. What is the causative pathogen in erythrasma?
    A. *Staphylococcus aureus*
    B. *Streptococcus pyogenes*
    C. *Corynobacterium minutissimum*
    D. *Erysipelothrix rhusiopathiae*

128. Recurrent streptococcal cellulitis is associated with which of the following disease entities?
    A. Saphenous venectomy
    B. Chronic venous stasis
    C. Chronic lymphedema
    D. All of the above

Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

129. Which one of the following bacteria is *not* usually associated with the pathogenesis of periorbital cellulitis?
    A. *Streptococcus pneumoniae*
    B. *Streptococcus pyogenes*
    C. *Staphylococcus aureus*
    D. *H. influenzae*

130. The treatment of choice for felon includes which of the following modalities?
    A. Warm soaks and compresses
    B. Surgical intervention and parenteral antibiotics
    C. Conservative management with oral antibiotics only
    D. Surgical intervention

Answer Key:

121. A
122. C
123. D
124. B
125. B
126. D
127. C
128. D
129. A
130. B

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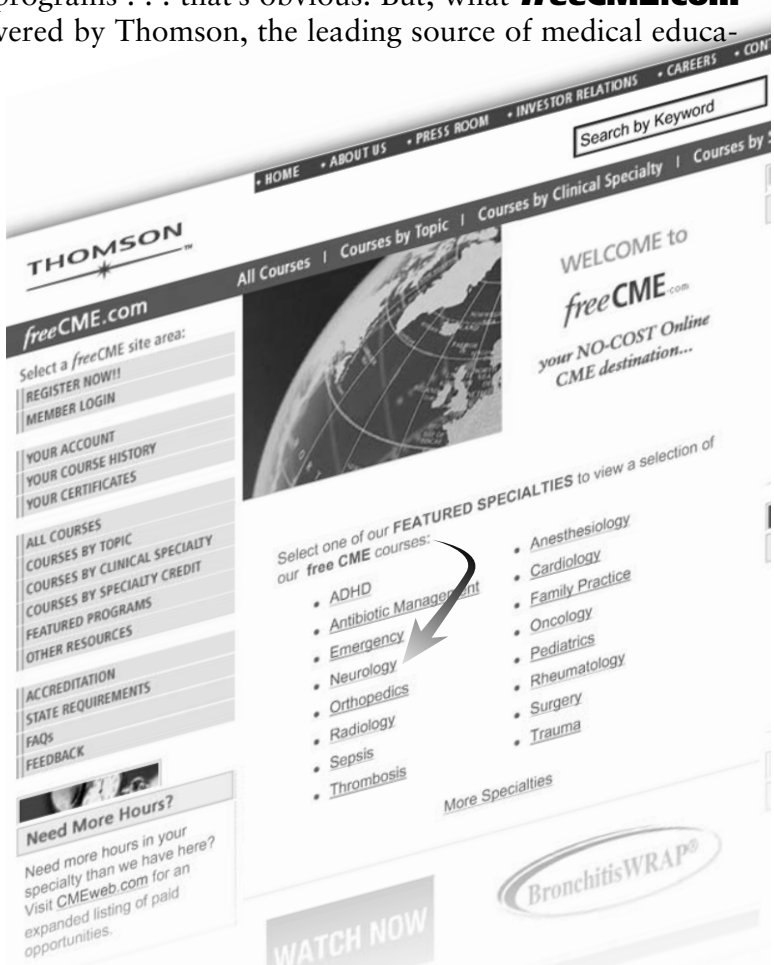

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Skin and Soft-Tissue Infections Part I

Risk Factors for Specific Pathogens Causing SSTI

RISK FACTOR	CHARACTERISTIC PATHOGEN
Diabetes	*S. aureus*, group B streptococcus, Gram-negative bacilli
Cirrhosis	*Camphylobacter fetus*, *Klebsiella pneumoniae*, *E. coli*, *Capnocytophaga canimorsus*, other Gram-negative bacilli, *Vibrio vulnificus*
Neutropenia	*P. aeruginosa*
Human bite	Oral anaerobic flora, *S.aureus*, *Eikenella corrodens*
Cat bite	*S. aureus*, *Pasteurella multocida*
Dog bite	*S. aureus*, *P. multocida*, *Capnocytophaga canimorsus*
Rat bite	*Streptobacillus moniliformis*
Animal contact	Camphylobacter spp.
Reptile contact	Salmonella spp.
Hot tub exposure	*Pseudomonas aeruginosa*
Fresh water exposure	*Aeromonas hydrophila*
Salt water exposure	*V. vulnificus*, *Mycobacterium marinum*
Intravenous drug use	Anaerobes, MRSA, *P. aeruginosa*
Soil contamination	Clostridium spp. Pseudomonas spp.
Puncture wound of foot	*P. aeruginosa*
Skin trauma in butchers and fishermen	*Erysipelothrix rhusiopathiae*

Bacteriology and Epidemiology of Cellulitis

ORGANISM	SPECIFIC CONSIDERATION
S. aureus	Centripetally spreading Recurrent cellulitis
Group A, C & G Strep. spp.	Lower extremities cellulites Associated with chronic venous stasis
Group B Strep.	Cellulitis in newborns
H. influenzae	Cellulitis of head and neck in children
Pseudomonas aerugenosa	Associated with penetrating injuries
Aeromonas hydrophilia	Associated with lacerations sustained in fresh water
Erysipelothrix rhusiopathiae	Associated with bone renderers and fishermen
Pasteurella multocida	Associated with cat and dog bites
Eikenella corrodens	Associated with human bites
Vibrio vulnificus	Associated with injuries sustained in salt water
Mycobacterium marinum	Associated with wounds sustained in aquariums or swimming pools

Supplement to *Emergency Medicine Reports*, June 14, 2004: "Bacterial Skin and Soft-Tissue Infections, Part I: Uncomplicated and Benign Bacterial Skin Infections." *Author:* **Ramin R. Samadi, MD, FACEP, FACP,** Chief Medical Officer, Tarrant Acute Care Physicians, PA, Fort Worth, TX; Medical Director, Trinity XpressMed Medical Center, Fort Worth, TX; and Assistant Clinical Professor, University of Texas Southwestern Medical School, Dallas; and Gregory A. Volturo, MD, FACEP, Vice Chairman and Associate Professor, Department of Emergency Medicine, University of Massachusetts Medical School, Worcester.