SECTION J: Skin and Soft-Tissue Infections

Superficial Bacterial Skin Infections and Cellulitis

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Providing a permeability barrier, which prevents both the loss of water and electrolytes and the invasion of pathogens, the skin has a primary protective function. Intact skin is resistant to colonization and invasion of bacteria via a number of mechanisms.  

The outermost layer of the epidermis, the stratum corneum, constitutes the principal barrier against infection. The stratum corneum consists of corneocytes (anucleated keratinocytes or skin cells), and an outer lipid matrix. Corneocytes fit together in an overlapping fashion, making penetration by organisms difficult; they are shed from the skin after approximately 14 days, thus pathogenic organisms have limited time to invade further into the epidermis. The lipid matrix surrounding corneocytes is acidic, providing antimicrobial activity against pathogenic organisms, including *Staphylococcus aureus* and *Streptococcus* species.  

Keratinocytes also participate in innate immunity via secretion of antimicrobial peptides (AMPs) such as human β-defensins, cathelicidin, psoriasin, and dermicidin. AMPs are small peptides with broad-spectrum antimicrobial activity against bacteria, viruses and yeast.  

Production of AMPs by keratinocytes is stimulated by inflammatory cytokines produced in the skin as a result of injury or inflammation and by invasive organisms via pathogen-associated molecular patterns (PAMPs).  

Resident bacteria on the skin provide additional protection against infection by preventing colonization with pathogenic organisms via competitive binding to cell surface receptors, and by the production of toxic substances called bacteriocins that inhibit the growth of similar bacteria.  

Skin microflora generally can be categorized into two groups: resident flora and transient flora. Resident flora establish secure attachments to the skin, are present in stable numbers, and are able to tolerate an acidic environment. Transient flora are introduced from the environment and only attach if the skin is disordered.  

Group A streptococcus (GAS, *Streptococcus pyogenes*) and *S. aureus* are the most common transient bacteria on the skin that cause infection.  

Many different bacteria are considered to be normal, resident flora, and each organism has a predilection for specific anatomic locations and for hosts of a particular age. The skin becomes colonized with microorganisms during the birth process and through contact with the environment. Infants born vaginally acquire *Staphylococcus epidermidis* during passage through the vaginal canal; and within hours, coryneform bacteria also are found on neonatal skin. The dry surface of the stratum corneum is colonized by micrococci and coagulase-negative staphylococci (CoNS).  

While coryneform organisms and gram-negative bacilli prefer moist, intertriginous areas, *Propionibacterium* spp. grow in hair follicles and sebaceous glands, and are mainly found after puberty, when sebaceous activity increases.  

Hair follicles are colonized by micrococci and CoNS superficially; *Corynebacterium* and *Propionibacterium* spp. are found deep in follicular canals.  

Transient colonization of the skin by pathogens is facilitated by factors that harm the resident flora, including elevated temperature, humidity, and antibiotic therapy. When a pathogen achieves successful colonization of the skin, the other cutaneous defense mechanisms must also be overcome before infection commences. Therefore, the main determinant of cutaneous infection is the balance between the virulence of the organism and the defense mechanisms of the host.  

Compromised cutaneous barrier function occurs in patients with chronic dermatitis and premature infants, making their skin more susceptible to pathogenic colonization and cutaneous infection.  

Primary bacterial infection of the skin can involve the epidermis, dermis, or subcutaneous tissue, whereas soft-tissue infections extend deeper, to the fascia or muscle. Superficial skin infections are mainly limited to the epidermis and dermis; although secondary inflammation can involve the subcutis. Several types of lesions can form in the skin as the result of a primary infectious process (Table 70-1); however, a pathogen usually produces a characteristic primary lesion with a characteristic pattern of spread. When cutaneous bacterial infection occurs, recognition of the type and depth of lesion produced are helpful in determining the likely causative agent. Primary, superficial bacterial infections of the skin and cellulitis are the focus of this chapter (Table 70-2). Other infectious agents (viruses, fungi) that sometimes resemble these bacterial infections are discussed briefly.

**SUPERFICIAL INFECTIONS**

**Impetigo**

Impetigo is a common skin infection caused by *S. aureus* and GAS. Impetigo occurs in two forms: bullous and nonbullous, and is highly contagious. Children aged 2 to 5 years are affected most often, and infection rates peak in the summer and late fall. Impetigo can be a primary infection or a secondary infection involving skin compromised by dermatitis or trauma. Factors that predispose to infection include poor hygiene, crowded living conditions, humidity, pre-existing dermatitis, and minor skin trauma.

**TABLE 70-1. Primary Skin Lesions**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Macule</td>
<td>Flat lesion, &lt;1 cm</td>
</tr>
<tr>
<td>Patch</td>
<td>Flat lesion, &gt;1 cm</td>
</tr>
<tr>
<td>Papule</td>
<td>Elevated lesion, &lt;1 cm</td>
</tr>
<tr>
<td>Plaque</td>
<td>Elevated broad flat lesion, &gt;1 cm</td>
</tr>
<tr>
<td>Nodule</td>
<td>Dome-shaped or rounded lesions, &gt;1 cm. Arising from the dermis or subcutis</td>
</tr>
<tr>
<td>Pustule</td>
<td>Pus-filled lesion</td>
</tr>
<tr>
<td>Vesicle</td>
<td>Elevated lesion, &lt;1 cm. Filled with serous fluid</td>
</tr>
<tr>
<td>Bula</td>
<td>Elevated lesion, &gt;1 cm. Filled with serous fluid</td>
</tr>
</tbody>
</table>

All references are available online at www.expertconsult.com
Nonbullous impetigo is the most common form of the infection, accounting for more than 70% of cases of impetigo. Lesions of nonbullous impetigo typically form on traumatized skin and are most often located on the exposed skin of the face and extremities. Lesions initially begin as small vesicles or pustules that rupture, forming an adherent, honey-colored crust. Impetigo is associated with minimal pain or erythema, and constitutional symptoms generally are absent. Pruritus, regional adenopathy, and leukocytosis are commonly associated with nonbullous impetigo. The differential diagnosis of nonbullous impetigo includes contact dermatitis, as well as viral, fungal, and parasitic (scabies, pediculosis) infections, all of which can be complicated by secondary infection with impetigo. If left untreated, most cases of nonbullous impetigo resolve in approximately 2 weeks without scarring. The bacterial etiology of lesions of nonbullous impetigo cannot be predicted clinically. The predominant cause of nonbullous impetigo in the United States is S. aureus, though infection can also be attributed to GAS or mixed pathogens. Anaerobic bacteria also have been isolated from lesions of nonbullous impetigo, although a pathogenic role is unclear. S. aureus causes impetigo in children of all ages, and the bacteria usually are present in the nose, perineum, axillae, or underneath the fingernails prior to causing cutaneous infection. In contrast, infections due to GAS are unusual before 2 years of age, except in highly endemic areas; and GAS colonizes the skin an average of 10 days before development of impetigo via inoculation of organisms into a break in the skin. Several types of GAS can cause nonbullous impetigo and they are different from the strains implicated in streptococcal pharyngitis.

Bullous impetigo occurs mainly in infants and young children. It is caused by strains of S. aureus, usually from phage group 2, capable of producing an exfoliative toxin that disrupts cell-to-cell adhesion in the superficial epidermis, leading to superficial blister formation. Since the lesions of bullous impetigo are a manifestation of localized toxin production, they develop on intact skin. The flaccid bullae and pustules of this form of impetigo occur beneath the stratum corneum and are easily ruptured, leaving shallow erosions with a collarette of scale (Figure 70-1). Bullae can be single or clustered; regional adenopathy and systemic symptoms are unusual. The differential diagnosis of bullous impetigo in the neonate includes transient neonatal pustular melanosis, epidermolysis bullosa, bullous mastocytosis, and herpetic infection. Insect bites, contact dermatitis, burns, erythema multiforme, and autoimmune bullous dermatoses must be considered in older children, particularly if the lesions are unresponsive to therapy.

<table>
<thead>
<tr>
<th>Disease Entity</th>
<th>Skin Lesions</th>
<th>Infectious Agent(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthrax</td>
<td>Papule, vesicle, bulla</td>
<td>Bacillus anthracis</td>
</tr>
<tr>
<td>Blistering distal dactylytis</td>
<td>Vesicle</td>
<td>Streptococcus pyogenes, group B streptococcus, Staphylococcus aureus</td>
</tr>
<tr>
<td>Cellulitis</td>
<td>Plaque</td>
<td>Streptococcus pyogenes, Staphylococcus aureus</td>
</tr>
<tr>
<td>Diphtheria</td>
<td>Papule, pustule, ulcer</td>
<td>Corynebacterium diphtheriae</td>
</tr>
<tr>
<td>Ecthyma</td>
<td>Pustule, ulcer</td>
<td>Streptococcus pyogenes</td>
</tr>
<tr>
<td>Ecthyma gangrenosum</td>
<td>Papule, vesicle, ulcer</td>
<td>Pseudomonas aeruginosa</td>
</tr>
<tr>
<td>Erysipelas</td>
<td>Plaque, sometimes vesicles/bullae</td>
<td>Streptococcus pyogenes, groups B, C, G streptococci</td>
</tr>
<tr>
<td>Erysipeloid</td>
<td>Patch, plaque</td>
<td>Erysipelothrix rhusiopathiae</td>
</tr>
<tr>
<td>Folliculitis</td>
<td>Pustule, papule</td>
<td>Staphylococcus aureus, Malassezia spp.</td>
</tr>
<tr>
<td>Sycosis barbae</td>
<td>Pustule, papule</td>
<td>Staphylococcus aureus</td>
</tr>
<tr>
<td>Gram-negative folliculitis</td>
<td>Pustule, papule</td>
<td>Klebsiella spp., Enterobacter spp., Escherichia coli, Pseudomonas aeruginosa</td>
</tr>
<tr>
<td>Hot-tub folliculitis</td>
<td>Pustule, papule</td>
<td>Pseudomonas aeruginosa</td>
</tr>
<tr>
<td>Erythrasma</td>
<td>Patch</td>
<td>Corynebacterium minutissimum</td>
</tr>
<tr>
<td>Furuncles, carbuncles</td>
<td>Nodule</td>
<td>Staphylococcus aureus</td>
</tr>
<tr>
<td>Impetigo</td>
<td>Vesicle, bulla, pustule, plaque, erosion</td>
<td>Staphylococcus aureus</td>
</tr>
<tr>
<td>Intertrigo</td>
<td>Patch, plaque, erosion</td>
<td>Streptococcus spp., Staphylococcus aureus, Candida albicans, Streptococcus pyogenes</td>
</tr>
<tr>
<td>Paronychia</td>
<td>Patch</td>
<td>Mixed flora (see text)</td>
</tr>
<tr>
<td>Perianal dermatitis</td>
<td>Patch</td>
<td>Staphylococcus aureus, Streptococcus pyogenes</td>
</tr>
<tr>
<td>Pitted keratolysis</td>
<td>Erosion</td>
<td>Kytococcus sedentarius, Dermophilus congolensis, Corynebacterium spp.</td>
</tr>
</tbody>
</table>
Complications of impetigo are rare, and include invasive infections such as pneumonia, osteomyelitis, pyogenic arthritis, and septicemia. Cellulitis can complicate nonbullous impetigo but rarely is associated with the bullous form. Streptococcal impetigo can be followed by lymphangitis, suppurative lymphadenitis, scarlet fever, and acute glomerulonephritis. Acute poststreptococcal glomerulonephritis occurs after skin and pharyngeal infections with nephritogenic strains of *S. pyogenes* (M groups 2, 49, 53, 55, 56, 57, and 60 for epidemics associated with impetigo). Symptoms develop an average of 18 to 21 days following skin infection, and children aged 3 to 7 years old are most commonly affected.\(^{10}\) The anti-DNAase B titer is the best serologic test for detecting preceding streptococcal impetigo as the etiology of acute glomerulonephritis.\(^{10,12}\) Strains of *S. pyogenes* associated with endemic impetigo in the U.S. have little or no nephritogenic potential and acute rheumatic fever does not occur as a result of impetigo.\(^{10}\)

Localized impetigo can be treated with topical antimicrobial agents. Topical mupirocin has been demonstrated to be as effective as oral erythromycin for the treatment of impetigo, and may be associated with fewer side effects.\(^{19,20}\) Retapamulin is a newer topical antibiotic, which like mupirocin, is approved by the U.S. Food and Drug Administration for treatment of impetigo caused by methicillin-susceptible *S. aureus* (MSSA) and *S. pyogenes*.\(^{11,21}\) Systemic therapy is recommended for patients with widespread lesions and for lesions associated with fever or evidence of deeper involvement (cellulitis, furunculosis, abscess formation, suppurative lymphadenitis).\(^{10}\) An agent effective against both staphylococcus and streptococcus, such as a first-generation cephalosporin, penicillinase-resistant penicillin, macrolide, or clindamycin, usually is utilized when systemic antimicrobial therapy is indicated.\(^{10,12,14,17}\) Antibiotic selection should be based on local resistance patterns to ensure coverage for methicillin-resistant *S. aureus* (MRSA) when appropriate. Antibiotic therapy does not prevent acute glomerulonephritis resulting from streptococcal impetigo, but will help prevent the spread of nephritogenic strains of the bacteria.\(^{10,11}\) Patients with recurrent impetigo sometimes are evaluated for carriage of *S. aureus*; decolonization can be attempted.\(^{10}\) Frequent handwashing and attention to personal hygiene should be emphasized to patients to prevent spread of infection.\(^{14}\)

### Perianal Bacterial Dermatitis

Perianal bacterial dermatitis usually occurs in children between the ages of 6 months and 10 years,\(^{1,2}\) and peaks between the ages of 3 and 5 years.\(^{2}\) The infection is most commonly due to GAS, but *S. aureus* also can cause infection.\(^{24}\) Physical examination characteristically reveals superficial, well-demarcated, circumferential erythema extending around the anus and surrounding skin (Figure 70-2).\(^{22,25,26}\) Vulvovaginitis, vaginal discharge, and vulvar redness can be seen in girls and penile involvement in boys.\(^{22,25}\) *S. aureus* is the likely pathogen when small papules and pustules on the buttocks and inguinal area are seen in addition to the characteristic findings, or when there is extension of erythema onto the buttocks.\(^{25}\) Additional manifestations include irritability,\(^{26}\) pruritus, painful defecation (at times associated with refusal to defecate), blood-streaked stools, fissures, mucoid discharge, and yellow crust.\(^{22,23,27}\) Familial spread of perianal dermatitis is common.\(^{22,23}\) Perianal streptococcal dermatitis is reported to be an infectious trigger for guttate psoriasis, and patients presenting with guttate psoriasis should be examined for asymptomatic GAS infection.\(^{27}\)

The differential diagnosis of perianal bacterial dermatitis includes psoriasis, irritant contact dermatitis, candidiasis, and pinworm infestation. Diagnosis of perianal bacterial dermatitis often is delayed due to misdiagnosis and lack of awareness of the condition,\(^{23}\) with patients frequently undergoing treatment with topical antifungal agents or topical corticosteroids before the correct diagnosis is made.\(^{22,23}\) Isolation of *S. pyogenes* or *S. aureus* from culture of perianal swab specimens confirms the diagnosis.\(^{22}\) A 10–14-day course of systemic antibiotics, often penicillin or erythromycin, is recommended.\(^{25}\) Clindamycin, a β-lactamase resistant penicillin, or a cephalosporin may be useful in cases due to *S. aureus*;\(^{24}\) therapy is guided by culture results and susceptibility test results. Topical erythromycin\(^{26}\) and mupirocin\(^{24}\) are reportedly as effective as solitary treatment; however, topical antibiotics generally are used as an adjunct to systemic therapy.\(^{27}\)

#### Intertrigo

Intertrigo is a disorder of the skin folds resulting from the friction created by opposing skin surfaces, combined with a moist environment. Because infants tend to have deep skin folds as a result of their short necks, generally flexed posture, and chubbiness, and due to their tendency to drool, infants are particularly susceptible to intertrigo.\(^{27}\) Secondary infections with *Candida albicans*, GAS, *S. aureus*, and mixed organisms can occur. Bright red, well-demarcated weeping patches and plaques are seen in the folds of the neck, axillae, antecubital fossa, inguinal area, or popliteal fossae (Figure 70-3). The presence of satellite lesions is suggestive of *Candida*.
infection, while streptococcal intertrigo commonly is associated with a foul odor. Affected infants usually appear well, but can have associated fever, fussiness, or malaise.\textsuperscript{31,32}

In addition to intertrigo, the differential diagnosis of intertriginous dermatitis includes seborrheic dermatitis, atopic dermatitis, irritant or allergic contact dermatitis, erythrasma, “inverse” psoriasis, scabies, and Langerhans cell histiocytosis. Diagnosis is confirmed by culture of a lesion. Treatment of candidal intertrigo consists of topical antifungal agents such as nystatin, econazole, or ketoconazole, while streptococcal intertrigo can be treated with a 10-day course of penicillin or cephalaxin, in combination with topical mupirocin. Anti-inflammatory agents, like topical hydrocortisone 1\%, can be used for associated erythema. Use of barrier ointments and ensuring that skin folds are completely dry will reduce the friction and moisture of intertriginous areas, helping to prevent intertrigo.\textsuperscript{29}

**Erythrasma**

* Corynebacterium minutissimum* is the causative agent of erythrasma. This cutaneous infection is manifest as well-demarcated reddish-brown patches and plaques located in moist intertriginous zones. The condition may be asymptomatic or associated with pruritus and involved skin may be thin with a “cigarette paper” quality.\textsuperscript{31} Commonly affected sites include the groin, axillae, intergluteal folds, submammary region, and interdigital spaces of the toes. Heat, humidity, obesity, diabetes mellitus, hyperhidrosis, and poor hygiene are predisposing factors.\textsuperscript{31,32}

Erythrasma can be confirmed with Wood lamp examination. *C. minutissimum* produces porphyrins that fluoresce a brilliant coral-red color under ultraviolet light; however, bathing within 20 hours before the Wood lamp examination can remove the water-soluble porphyrins. When Wood lamp examination is negative, a potassium hydroxide examination is useful to exclude dermatophyte infection and skin scrapings can be stained with periodic acid-Schiff, methenamine silver, or Gram stain to evaluate for coccobacilli.\textsuperscript{31}

The treatment of choice is erythromycin 250 mg four times daily for 2 weeks. Topical clindamycin twice daily also can be used. For severe cases, a combination of oral erythromycin and topical antibiotics may be needed. Recurrence can be minimized by the use of an antibacterial soap.\textsuperscript{32}

**Pitted Keratolysis**

Pitted keratolysis is a skin infection affecting the thick stratum cornue of the plantar surface of the feet and less commonly the palms. Characteristic findings include white, hyperkeratotic areas studded with multiple 1 to 7 mm pitted or erose lesions on the soles, particularly over pressure-bearing sites (Figure 70-4).\textsuperscript{31,33}

Ringed erythematous lesions that coalesce can be seen on non-hyperkeratotic areas.\textsuperscript{31} The condition can be asymptomatic or associated with hyperhidrosis, malodor, pain, and sliminess of the skin. People whose feet are moist for prolonged periods due to hyperhidrosis, immersion in water, or use of occlusive shoes are most frequently affected. Males are affected more often than females.\textsuperscript{31,33}

*Kytococcus sedentarius* (formerly *Micrococcus* spp.), the suspected causative agent of pitted keratolysis, produces serum proteases capable of degrading keratin in calloused skin.\textsuperscript{31} *Dermophilus golenolis* and *Corynebacterium* spp. also have been implicated as pathogens.\textsuperscript{31,33} Diagnosis is based on characteristic clinical findings. Effective therapeutic regimens include topical or systemic erythromycin, miconazole, fusidic acid, and control of hyperhidrosis with topical aluminum chloride or botulinum toxin. Improved hygiene and use of adequately fitting shoes also may help.\textsuperscript{31}

**Trichobacteriosis**

Trichobacteriosis (formerly trichomycosis) is an asymptomatic infection of the axillary and less commonly the pubic hair shafts caused by *Corynebacterium tenuis* and other coryneform species.\textsuperscript{31,33} A bacterial biofilm encases the hair, creating yellow or white concretions distributed along the length of the hair shaft. Hyperhidrosis and a foul odor can be associated with the condition. The diagnosis is clinical, though a pale yellow fluorescence may be seen with Wood light examination. Treatment consists of shaving the affected area, applying topical antibiotics like clindamycin or erythromycin, and use of antiperspirants to control hyperhidrosis.\textsuperscript{31}

**ADNEXAL AND FOLLICULAR INFECTIONS**

**Folliculitis**

Bacterial folliculitis is a superficial infection of the hair follicle manifest by discrete 2 to 5 mm papules and pustules on an erythematous base. The papules and pustules can be single or grouped, and often a hair shaft is seen in the center of the lesion (Figure 70-5).\textsuperscript{17} Lesions usually are located on the scalp, buttocks, or extremities,\textsuperscript{15,17} but can occur on any hair-bearing area.\textsuperscript{15} Folliculitis can be asymptomatic or accompanied by pruritus; systemic symptoms usually are absent.\textsuperscript{17} Gram stain and culture of purulent material from the follicular orifice can identify the causative organism of folliculitis. *S. aureus* is the predominant pathogen,\textsuperscript{17,18} and affected patients often are chronic carriers of *S. aureus* in the nares, perineum, or axillae,\textsuperscript{16} however, gram-negative bacteria also can cause folliculitis (see below). The differential diagnosis of bacterial folliculitis includes inflammation of the hair follicle due to physical injury or chemical irritation, eosinophilic folliculitis, insect bites, scabies, pseudofolliculitis barbae, and infection due to *Malassezia* species. Simple bacterial folliculitis often resolves spontaneously without scarring.\textsuperscript{17} When treatment is desired, topical antibiotic cleansers, such as chlorhexidine, topical antibacterial agents (mupirocin, erythromycin, clindamycin), or benzoyl peroxide usually are effective for mild infections.\textsuperscript{17,18} Systemic antibiotic therapy with a first-generation cephalosporin, penicillinase-resistant penicillin, clindamycin, macrolide, or fluoroquinolone (depending on local resistance patterns) is used in severe or refractory cases.\textsuperscript{17}

Less common forms of bacterial folliculitis include sycoec barbae, gram-negative folliculitis, and hot tub folliculitis. Sycoec barbae is a severe, recurrent form of facial folliculitis due to *S. aureus*. Painful erythematous, follicular papules and pustules involving the entire depth of the follicle develop on the chin, upper lip, and angle of the jaw, primarily in young black males. Papules can coalesce into plaques, and healing may occur with scarring. Treatment includes warm saline compresses and a topical antibiotic such as mupirocin. Extensive or recalcitrant cases may require therapy with a systemic antistaphylococcal antibiotic with attempted eradication of *S. aureus* carriage.\textsuperscript{16}

![Figure 70-4. White plaques with numerous shallow pits on the plantar surface of the foot of a patient with pitted keratolysis.](image-url)
Folliculitis is an eruption that manifests as pruritic, 2 to 3 mm, monomorphic, erythematous, perifollicular papules and papulopustules on the back, chest, and upper arms.\textsuperscript{43,44} Predisposing factors include diabetes mellitus, malignancy, HIV/AIDS, organ transplantation, or other causes of immunosuppression and prolonged oral antibiotic therapy.\textsuperscript{43,44,46} Malassezia also has been implicated in the form of eosinophilic folliculitis associated with advanced HIV infection.\textsuperscript{43,46} Diagnosis is made by microscopic examination of a potassium hydroxide-treated scraping from a lesion, which reveals budding yeast and spores.\textsuperscript{43,44} Skin biopsies of Malassezia folliculitis show dilated follicular ostia with budding yeast and spores and a mixed inflammatory infiltrate; however, a biopsy rarely is necessary for diagnosis.\textsuperscript{43,44} Isolation of the organism in culture requires use of a special lipid-containing medium.\textsuperscript{1,12} While individuals with furuncles usually have no constitutional symptoms, fever, leukocytosis, and bacteremia can accompany carbuncles. Both lesions tend to heal with scarring. The causative agent of furuncles and carbuncles is almost always \textit{S. aureus}. The staphylococcal isolates (both MSSA and MRSA) associated with furunculosis often possess the virulence factor factor Panton–Valentine leukocidin, a pore-forming toxin that targets neutrophils.\textsuperscript{1,3,6,47} Conditions that predispose to furuncle formation include obesity, immunosuppression, diabetes mellitus, hyperhidrosis, maceration, friction, and pre-existing dermatitis.\textsuperscript{56} Outbreaks of furunculosis have been reported in sports teams, families, and other populations with close contact.\textsuperscript{1,43,48} Recurrent furunculosis frequently is associated with carriage of \textit{S. aureus} at multiple sites (nares, axillae, perineum) or with sustained close contact with someone who is a carrier. Rarely, children with recurrent furunculosis may have an underlying immunodeficiency.\textsuperscript{1} Other bacteria or fungi occasionally cause furuncles or carbuncles; therefore Gram stain and culture of the purulent exudate are indicated. The differential diagnosis of furunculosis includes epidermal cysts, cystic acne, and hidradenitis suppurativa.

Treatment consists of frequent application of a hot, moist compress to lesions to promote drainage. Large furuncles and most
carbuncles require surgical drainage, with disruption of any existing loculations and wound packing as appropriate. When lesions are large, multiple, there is extensive surrounding cellulitis, or fever is present, treatment with an oral antistaphylococcal agent is indicated. For recurrent furunculosis, attempts to eradicate staphylococcal carriage can be undertaken. Attention to personal hygiene, bleach baths, or use of chlorhexidine soap may be beneficial.

HAND AND NAIL INFECTIONS

Paronychia

Acute paronychia is an infection of the soft-tissue folds surrounding a fingernail or toenail. The infection occurs when minor trauma allows bacteria to enter the cuticle or nail fold. Paronychia is seen most commonly in children who suck their fingers, bite their nails or cuticles, or have poor hygiene; and also is associated with dishwashing, manicures, and use of artificial nails. The lateral nail fold becomes warm, erythematous, edematous, and painful; purulent fluid can accumulate underneath the nail plate. Usually only one nail is affected. In most cases, the infection is caused by S. aureus or mixed aerobic and anaerobic flora. The most common aerobic organisms are S. aureus, S. pyogenes, and Eikenella corrodens; anaerobic pathogens include Bacteroides spp., Fusobacterium spp., and gram-positive cocci. Diagnosis of acute paronychia is based on clinical examination. Both aerobic and anaerobic cultures of purulent material are recommended to identify the causative pathogen(s) and perform susceptibility testing of S. aureus. Warm compresses generally are curative for superficial lesions. Antibiotic therapy with an oral antistaphylococcal agent, in addition to incision and drainage, is needed for treatment of deeper lesions with abscesses. When exposure to oral flora is suspected (nail biting), a broad-spectrum oral antibiotic effective against anaerobes (clindamycin or amoxicillin-clavulanate) is indicated.

The differential diagnosis of acute paronychia includes chronic paronychia, psoriasis, and herpetic whitlow. Chronic paronychia can be distinguished from acute infection by the duration of symptoms. Chronic paronychia often is associated with C. albicans, and usually is seen in people whose hands are frequently exposed to water (finger sucking, dishwashers, house cleaners, bartenders, food handlers, nurses). Like bacterial paronychia, herpes simplex virus infection of the fingers can occur after sucking or parental nail-trimming by biting. Herpetic whitlow can resemble staphylococcal infection. Multiple coalescing vesicles of the digit associated with edema and a dusky appearance are typical of whitlow. Direct fluorescent antibody testing and viral culture can confirm the diagnosis. Herpetic lesions should not be incised or debrided; instead, oral antiviral therapy should be given.

Blistering Distal Dactylitis

Blistering distal dactylitis is an acral infection caused by S. pyogenes, and less commonly S. aureus or CoNS. It usually is seen in children aged 2 to 16 years, but has been reported in infants and adults. This infection manifests as a tense non tender bulla with an erythematous base involving the distal volar fat pad of the phalanges. Dark discoloration of the surrounding skin may be associated. One or more digits can be affected, as can the nail fold, the volar surfaces of the proximal phalanges, the toes, and the palm. Systemic symptoms generally are absent. The blisters are filled with a thin purulent fluid containing neutrophils and the infecting organisms. Infections caused by S. aureus may be more likely to be associated with pain and involvement of more than one digit. The diagnosis is based on examination and culture. Treatment consists of incision and drainage or a 10-day course of systemic therapy or both using an agent with staphylococcal and streptococcal coverage.

ULCERATIVE INFECTIONS

Anthrax

Bacillus anthracis causes inhalational, gastrointestinal, meningeal, and cutaneous infections. Cutaneous anthrax in the U.S. is limited mainly to individuals who work with contaminated animals or animal products, including carcasses, hair, and wool; however, cases due to acts of bioterrorism have been reported. Cutaneous infection occurs 1 to 7 days after exposure/inoculation of an endospore into the skin, usually at the site of a cut or abrasion. The primary lesion is a small, painless, purulent papule that transforms over a few days into a 1- to 2-cm bulla, which can be associated with satellite vesicles. Characteristic brawny, nonpitting edema surrounds the lesion. The bulla ruptures, forming an ulcer with a central black eschar. (Figure 70–7) Malaise, low-grade fever, and regional lymphadenopathy frequently are noted. Differential diagnosis includes ecthyma, ecthyma gangrenosum, a furuncle, and necrotic arachnidism. Diagnosis is based on Gram-stained smear and culture of vesicular fluid, eschar, or tissue. Incision and drainage of the lesion can precipitate bacteremia; however, skin biopsy of a lesion while administering antibiotic therapy likely confers minimal risk. Treatment of mild naturally occurring cutaneous anthrax consists of penicillin for 7 to 10 days. Cutaneous anthrax associated with bioterrorism in children is treated with ciprofloxacin or doxycycline for 60 days due to the risk of inhalational exposure to the spores; therapy can be modified based on results of antimicrobial susceptibility test results.

Diphtheria

Cutaneous infection with Corynebacterium diphtheriae is rare in developed countries, and is seen mainly in travelers to endemic tropical areas, the immunosuppressed, and those living in crowded, unsanitary conditions. Infection can occur despite adequate immunization for the organism. Cutaneous diphtheria occurs in three forms: primary infection, which consists of a tender pustule that evolves into a punched-out ulcer with an adherent membrane and erythematous, edematous rim;
secondary infection of a pre-existing ulcer or wound; and super-
infection of eczematous skin lesions. Lesional skin sheds bac-
teria for 2 to 6 weeks without treatment, and thus is an important
reservoir for person-to-person transmission and environmental
contamination of pathogenic organisms, which can cause both
cutaneous and respiratory disease. Systemic complications,
including neurologic symptoms and myocarditis, are rare in
immunized patients and more often are associated with the
respiratory tract than cutaneous diphtheria. Culture of a lesion or its overlying membrane confirms the
diagnosis, and laboratory personnel must be notified when
*C. diphtheriae* is suspected. Treatment consists of cleansing the
affected skin and systemic antibiotic therapy with penicillin or
erythromycin for 10 days. The use of antitoxin in addition to
systemic antibiotics is controversial, since cutaneous lesions
are either nontoxigenic or produce small amounts of toxin. To
document eradication of the organism, two negative cultures
should be obtained after treatment concludes.

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**Figure 70-7.** Cutaneous anthrax. Note ulcer with
vesicular ring, induration and erythema (A). As eschar forms, induration lessens; surrounding
desquamation occurs, but erythema persists (B).
(Source: http://www.bt.cdc.gov/agent/anthrax/
anthrax-images.)

**Figure 70-8.** *Pseudomonas aeruginosa* septicemia and ecthyma gangrenosum in a young male with severe neutropenia (A). Microscopic appearance of
ecthyma gangrenosum in fatal *P. aeruginosa* septicemia in an infant with leukemia (B). Haematoxylin–eosin stain shows superficial necrosis and elevation as well
as bland ischemic necrosis beneath. Gram stain of fluid from bullous lesion (B) shows dense gram-negative bacilli with rare inflammatory cells (C). (Courtesy of
J.H. Brien©.)
**Ecthyma**

Ecthyma is a deep ulcerative infection of the skin that penetrates down to the dermis and is most commonly caused by *S. pyogenes*. Initially a vesicle or vesicopustule with surrounding erythema is noted; over time the lesion evolves into a crusted ulcer with an elevated rim. Lesions usually are located on the legs, and frequently are seen in association with pruritic conditions such as insect bites or scabies. Lymphangitis, cellulitis, and poststreptococcal glomerulonephritis are potential complications of ecthyma. Systemic antibiotic therapy with an agent effective against streptococci is recommended.11

**Ecthyma Gangrenosum**

Ecthyma gangrenosum is the characteristic skin lesion found in association with *Pseudomonas aeruginosa*. *Pseudomonas* septicaemia usually is seen in the setting of neutropenia or congenital neutrophil dysfunction, but also has been described in healthy patients; often less than one year of age.64,65 Cutaneous findings are the result of a necrotizing bacterial vasculitis affecting small veins in the skin. Lesions begin as erythematous, indurated papules, vesicles, and nodules that progress, over hours to days, into a necrotic ulcer with a black eschar and an erythematous rim (Figure 70-9). Lesions are commonly observed on the gluteal and perineal areas, but may be found all over the body.64,65 Ecthyma gangrenosum also can manifest in a localized form, usually on the buttocks and legs, after inoculation of the organism into the skin. This localized form of ecthyma gangrenosum usually is not associated with bacteremia11,66,67 and can occur in healthy children or those with occult immunodeficiency.37 Culture of the lesions, and blood cultures, confirm the diagnosis. Effective treatment requires prompt initiation of an antibiotic effective against *P. aeruginosa*. Ecthyma gangrenosum-like lesions can develop as a result of infection with other agents, usually in the setting of immuno-suppression. Etiologic agents include gram-negative bacteria other than *P. aeruginosa*,12,36 MSSA, MRSA,66 Streptococcus species,12 *Candida* species, fungi (*Aspergillus, Mucor, and Fusarium* species),12 and herpes viruses.66

**SOFT-TISSUE INFECTIONS**

**Erysipelas**

Erysipelas is a superficial skin infection affecting the upper dermis and the lymphatic system. In most cases, GAS is the cause of erysipelas.12,13,17,68 however, group B, C, and G streptococci occasionally can cause the infection,12,68 and rarely, *S. aureus, Strep- tococcus pneumoniae, Klebsiella pneumoniae, Versinia enterococitica,* and *Haeinophilus influenzae* are implicated as pathogens.68 Erysipelas has a bimodal distribution, being seen most often in young children and older adults.12,68 Infection occurs when disruption of the skin barrier allows entry of organisms into the skin, and often is associated with abrasions, leg ulcers, intertriginous or pedal fungal infections, insect bites, venous or lymphatic obstruction, and chronic edema.12,13,68 In neonates, infection can originate at the umbilical stump and spread to the abdominal wall.12

The onset of erysipelas is abrupt and is characterized by a painful, bright red, shiny, edematous plaque with well-demarcated and slightly raised borders. In severe cases, bullae and necrosis can occur. Infection usually occurs on a lower extremity (most often), or the face, and can be associated with regional lymphadenitis.12,13,17,68 Fever, chills, and malaise can precede the onset of cutaneous findings. Potential systemic complications of erysipelas include septicemia, streptococcal toxic shock syndrome, endocarditis, and meningitis; however, complications are rare with prompt diagnosis and appropriate treatment. The differential diagnosis includes contact dermatitis, burns, cellulitis, ecthyma gangrenosum, and urticaria.68 Diagnosis of erysipelas is made primarily on clinical grounds.12,68 Blood cultures, skin biopsies, and needle aspirations are of low yield.12

Treatment for erysipelas in immunocompetent patients consists of oral penicillin for 10 to 14 days, with follow-up after 48 to 72 hours to ensure the infection is improving. Patients with severe infections, young infants, and the immunosuppressed initially may require hospitalization for parenteral therapy.68 For penicillin allergic patients, macrolide therapy usually is effective;12,68 and if the presence of staphylococci is a concern, an antistaphylococcal antibiotic should be used.12 Local wound care and attention to predisposing factors (treating tinea pedis, elevating edematous legs) also are important aspects of treatment. Prophylactic therapy is considered infrequently for patients with recurrent disease.12,68

**Cellulitis**

Cellulitis is an acute infection of the skin involving the dermis and subcutaneous tissues. Cellulitis is manifested by edema, warmth, erythema, and tenderness of the skin. The lateral margins of cellulitis tend to be indistinct, unlike the well-demarcated borders of erysipelas (Figure 70-10). Vesicles, bullae, and petechiae can occur on involved skin. The lower legs are affected most commonly, but infection can occur at any site. Associated findings include lymphangitis, regional lymphadenopathy, fever, chills, and malaise. When cellulitis occurs in a periorbital distribution, and especially in the absence of a break in the skin, orbital cellulitis should be considered. Predisposing factors for cellulitis include pre-existing skin infections (ecthyma, impetigo), breaks in the skin due to trauma or insect bites, lymphatic obstruction or other causes of edema, leg ulcers, and obesity.12,13

The most common etiologic agents of cellulitis are *S. pyogenes* and *S. aureus*.12,36 When facial cellulitis in children is associated with a portal of entry such as a tooth abscess or cutaneous trauma, the etiology may also be due to oral anaerobic bacteria.67 *H. influenzae* capsular type b (Hib) was an important cause of peri orbital11 and facial cellulitis in children aged 3 months to 3 years of age prior to the introduction of the Hib conjugate vaccine. This form of cellulitis has a characteristic bluish discoloration resembling a bruise and often is associated with bacteremia.67 Bacteremic *S. pneumoniae* also can cause facial cellulitis in children resembling that due to *H. influenzae*.72 In patients who are immunocompromised or have been exposed to animals or special conditions, a
number of other bacterial or fungal agents have been implicated in causing cellulitis (Table 70-3).

Diagnosis of cellulitis is based on physical examination and a detailed history to determine if there are any factors predisposing to cellulitis caused by one of the less common pathogens. Attempts to determine the specific etiology of the infection may be of low yield. Blood culture should be performed in the setting of young age or systemic illness; otherwise yield is low. Culture of an aspirate from the site of inflammation yields a pathogen in 10% of patients, with an aspirate taken from the point of maximum inflammation yielding the causal organism more often than does a leading-edge aspirate. Tissue culture is positive in 18% to 20% of patients and the density of organisms present is low, although a higher density of organisms has been noted in specimens taken from a site near an ulcer. Uncomplicated cellulitis in immunocompetent patients should resolve with antimicrobial therapy targeting streptococci and staphylococci. If fever and lymphadenopathy are absent, outpatient treatment using a penicillinase-resistant penicillin, first-generation cephalosporin, or macrolide is appropriate. The need for an agent providing coverage for CA-MRSA should be guided by local prevalence and antibiotic susceptibilities. Parenteral treatment is instituted if fever, rapid progression, lymphangitis, or lymphadenitis is present. When erythema, warmth, edema, and fever have decreased substantially in uncomplicated cases, a 10-day course of treatment can be completed with oral therapy. Adjunctive therapies include elevation of the affected extremity, analgesics for pain, and tetanus immunization when appropriate.

Erysipeloid

Erysipeloid is a rare acute cutaneous infection resulting from traumatic inoculation of Erysipelothrix rhusiopathiae into the skin. Infection usually occurs in patients with occupational exposure to raw fish, poultry, and meat products or to contaminated animals, especially swine. Localized cutaneous infection manifests as a well-demarcated erythematosus-purple inflammatory plaque with raised borders on the dorsal aspect of one hand and/or fingers; and typically occurs 2 to 7 days after inoculation. The lesion spreads peripherally and can display central clearing. Untreated limited cutaneous infection can resolve spontaneously after 2 to 3 weeks, but can recur weeks to months later. Rarely a diffuse cutaneous form of infection occurs, manifesting as lesions over several areas of the body, and may be associated with fever, lymphadenopathy, myalgia, and arthralgia. Rare systemic complications of erysipeloid include encephalitis, endocarditis, pyogenic arthritis, and sepsis. E. rhusiopathiae is difficult to isolate, although tissue culture of the advancing edge of the lesion might identify the organism. Diagnosis is based mainly on the patient’s occupation or exposure history, clinical findings, and rapid improvement with antibiotic therapy. The recommended treatment for localized cutaneous infection is penicillin or a cephalosporin for 7 days, with improvement generally seen after 2 to 3 days.

<table>
<thead>
<tr>
<th>TABLE 70-3. Special Causes of Cellulitis</th>
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<tr>
<td><strong>Exposure</strong></td>
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<td>Penetrating trauma</td>
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<td>Freshwater immersion</td>
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Figure 70-10. Ill-defined erythema and edema on the cheek of a boy with cellulitis. (Courtesy of Brandon Newell, MD.)
REFERENCES