Chapter 24  
Bacterial Infections

Cutaneous bacterial infections are caused by resident or transient bacteria in the epidermis and mucosa. These bacteria invade the skin where its barrier function is weaker, such as at hair follicles, sweat glands or sites of minor trauma. The severity of infection tends to depend on the relative balance between the amount and virulence of the bacteria and the defenses of the host. When a cutaneous bacterial infection is suspected, the causative bacteria must be identified by culture and microbial sensitivity test in order to choose the appropriate antibacterial drugs. This chapter introduces four main subtypes of bacterial infections, classified by the clinical features, and the representative diseases of each subtype: 1) acute cutaneous infections (acute pyoderma), 2) chronic cutaneous infections (chronic pyoderma), 3) systemic infections caused by toxins that are produced by bacteria, and 4) diseases with specific clinical features that are caused by specific bacteria.

A. Acute pyodermas

1. Impetigo

   Synonym: Impetigo contagiosa

   Outline
   • Bacterial infection occurs under the horny cell layer, producing toxins that cause blisters and crusts. The infection spreads by autoinoculation.
   • Infants are most frequently affected. Impetigo is divided into bullous impetigo, in which blistering is caused by Staphylococcus aureus, and nonbullous impetigo, in which crusts form from group-A β-hemolytic Streptococcus infection.
   • Antibiotics and keeping skin clean are the main treatments.

1) Bullous impetigo

Clinical images are available in hardcopy only.

Fig. 24.1-1 Impetigo.
Erosions, blisters, pustules and crusts are present.
scalded-skin syndrome (SSSS, described later).

**Pathogenesis**

*Staphylococcus aureus* proliferate in the horny cell layer, producing exfoliative toxin (ET), which leads to intraepidermal blisters.

**Differential diagnosis**

Insect bites, in which blisters are severely inflammatory and contain sterile components, can be distinguished from bullous impetigo. In staphylococcal scalded-skin syndrome, there are the characteristic features of lesions around the eyes and mouth, and positive Nikolsky’s sign; it should be differentiated from bullous impetigo. Cases whose onset is in adulthood should be differentiated from pemphigus foliaceus.

**Treatment**

The skin should be kept clean. To prevent transmission, patients should not share towels until crusts form. Topical application of antibiotic ointments and oral cefem antibiotics are useful.

2) Nonbullous impetigo

*Synonym: Streptococcal impetigo*

**Clinical features, Epidemiology**

A few blisters form. Nonbullous impetigo begins as small erythema, followed by multiple pustules and formation of yellowish-brown crusts. The crusts are thick and firmly adherent; they discharge pus when pressured. Pain and swelling occur in the regional lymph node, often accompanied by pharyngeal pain and fever. Unlike in bullous impetigo, the onset of nonbullous impetigo is acute and is independent of age and season. The prevalence has been increasing among patients with atopic dermatitis.

**Pathogenesis**

It is mainly caused by subcorneal infection of group-A β-hemolytic *Streptococcus* (*Streptococcus pyogenes*).

**Differential diagnosis**

It is difficult to distinguish nonbullous impetigo from Kaposi’s varicelliform eruption, particularly in children with atopic dermatitis. The two conditions may occur at the same time.

**Treatment**

Oral antibiotics are the first-line treatment. Urine analysis is conducted in cases with streptococcal nonbullous impetigo, because glomerulonephritis may occur as a complication. To prevent nephritis, administration of oral antibiotics is continued 10 days after remission of the eruptions.
It is most often caused by group-A β-hemolytic streptococcal infection (*Streptococcus pyogenes*). It occurs with sudden fever. The face is most frequently affected. Sharply demarcated edematous erythema rapidly spreads. Intense tenderness and heat sensation are present. Because *Streptococcus pyogenes* is not easily detected by culture, ASO and ASK values are also measured. Penicillin antibiotics are the first-line treatment.

Clinical features

Sharply demarcated edematous erythema accompanied by chills and fever occurs suddenly, frequently on the face and legs. The erythema surface is tense and glossy. There is intense tenderness. The eruptions spread rapidly and centrifugally. Blistering may occur on the edematous erythema (*erysipelas bullosa*). When the face is involved, first one side is affected and soon the other side is affected (Fig. 24.2). Systemic symptoms such as fever, nausea and vomiting are present. In about 1 week, the eruptions and fever disappear. However, the eruptions may recur repeatedly on previously affected sites; this is called recurrent erysipelas.

Pathogenesis

Erysipelas is a purulent inflammatory disease that affects primarily the dermis. It is most frequently caused by group-A β-hemolytic streptococcus (*Streptococcus pyogenes*). *Streptococcus pyogenes* of other groups (group B in newborns), *Staphylococcus aureus*, and pneumococcus may cause symptoms similar to those of erysipelas. The pathogenesis of recurrent erysipelas is thought to be local lymphatic blockage or inadequate treatment of erysipelas; the details are unknown.

Laboratory findings

Antistreptolysin O (ASO) and antistreptokinase (ASK) increase as a result of streptococcal infection. Elevated erythrocyte sedimentation rate, leukocytosis (left shift of the nuclei in leukocytes), and CRP positive are observed. The rate of bacterial detection from tissue fragment or aspirated tissue fluid is low. *Streptococcal Laboratory findings*

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Erysipeloid

Erysipeloid is caused by the gram-positive bacillus *Erysipelothrix rhusiopathiae*. It occurs most frequently in those who handle animals, meat or seafood. The bacilli invade a minor trauma in a hand or finger. After 1- to 4-day incubation, sharply circumscribed, painful, edematous erythema appears. The lesion enlarges centrifugally, and the center tends to heal. Oral penicillin and tetracycline drugs are extremely effective.
bacteria can be detected by PCR.

**Differential diagnosis**

Cellulitis is more deeply seated than erysipelas, (Fig. 24.3), and its erythema edges are less clearly defined. Necrotizing fasciitis can be distinguished from erysipelas by the rapidly progressing necrotic lesions and intense systemic symptoms. Insect bites, thrombophlebitis, Sweet’s disease, herpes zoster, and carcinoma erysipelatodes also must be differentiated from erysipelas.

**Treatment**

Antibiotics such as penicillin drugs and next-generation oral cefem are administered. Treatment is continued for 10 days after remission to avoid recurrence and to prevent the complication of nephritis.

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**3. Cellulitis**

**Outline**

- This acute purulent inflammation occurs extensively in the deep dermal layer and subcutaneous tissue (Fig. 24.3).
- Vaguely demarcated erythema, swelling, localized heat sensation, and sharp pain occur suddenly in the face and extremities.
- It may progress to necrotizing fasciitis or septicemia.
- The main treatments are bed rest and parenteral antibiotics.

**Clinical features**

The face and extremities, particularly the lower legs, are most frequently involved. Cellulitis begins with ill-demarcated erythema, swelling and localized heat sensation, quickly becoming intense infiltration that is accompanied by tenderness and spontaneous pain (Figs. 24.4-1 and 24.4-2). Although the infiltration is usually absorbed in the skin over time and heals, a pustule may form at the soft center of the lesion. Systemic symptoms such as fever, headache, chills and arthralgia are present. Cellulitis may progress to necrotizing fasciitis or septicemia.

**Pathogenesis**

Most cases of cellulitis are caused by *Staphylococcus aureus*. Group-A β-hemolytic *Streptococcus* and *Hemophilus influenzae* are among the causative species. Bacteria usually invade the skin through a minor trauma, cutaneous ulcer, folliculitis or tinea pedis, causing cellulitis secondarily; however, the entry route may not be identifiable. Localized impairment in venous circulation and lymphatic edema may induce cellulitis.

**Laboratory findings**

Elevated erythrocyte sedimentation rate, leukocytosis (left
shift of the nuclei in leukocytes), and CRP positive are observed. Hepatic enzyme levels increase in some cases. Bacteria are easily detected from the pus in the lesion. Bacterial culture is more difficult to perform in cases without pus discharge.

**Differential diagnosis**

Lesions caused by erysipelas are superficial and the progressive lesions are sharply circumscribed; however, differentiation from cellulitis is difficult. Necrotizing fasciitis is accompanied by purpura, blisters, bloody blisters and severe systemic symptoms. Thrombophlebitis, erythema nodosum, insect bites and herpes zoster should also be differentiated from cellulitis.

**Treatment**

Systemic administration or intravenous cefem antibiotics and bed rest are the main treatments. Necrotizing fasciitis is suspected when non-localized symptoms present, including high fever, abnormally high leukocyte and CRP levels, and marked systemic symptoms.

4. Folliculitis

**Synonym:** Acne vulgaris

**Outline**

- It is a localized bacterial infection in a single hair follicle.
- It is a pustule accompanied by erythema.
- Folliculitis that occurs on the face in puberty is called acne vulgaris.
- It may progress to furuncle or carbuncle.
- The main treatments are skin care and topical or oral antibiotics.

**Clinical features**

Erythema and pustule occur at the hair follicle (Fig. 24.5). The skin lesion forms crust in several days and heals without scarring in most cases. Superficial folliculitis that causes multiple eruptions on the face especially in puberty is called acne vulgaris (Chapter 19). Deep-seated folliculitis is accompanied by intense inflammatory symptoms and may progress to furuncle or carbuncle in some cases. The deep-seated folliculitis in the barba areas is called sycosis vulgaris.

**Pathogenesis**

A hair follicle is infected by *Staphylococcus aureus* or *Staphylococcus epidermidis*. A minor trauma, obstruction and scratch around a hair follicle, or topical application of steroids may induce the infection. The hair follicle becomes inflamed.

**Treatment**

When there are only a few eruptions, folliculitis heals