This is hidradenitis in the opening of a hair follicle that is contained in the apocrine sweat gland. The follicular opening is blocked by a keratin plug, leading to secretion deposition where *Staphylococcus aureus* infects (Fig. 24.10). One or several subcutaneous nodules about 5 mm in diameter occur, most frequently on the axillary fossae of women. The nodules soften, rupture, excrete pus, and heal with scarring. Hidradenitis suppurativa often becomes chronic. Other sites with apocrine sweat glands, such as the genitalia, anus and breasts, may also be involved. Administration of antibiotics and incision and drainage of pus are the main treatments.

2. *Dermatitis papillaris capillitii*  
This is also known as keloidal folliculitis. Folliculitis appears multiply and continuously on the occipital and nuchal region of middle-aged men. Infiltration of the lesion gradually becomes intense, and connective tissue proliferates and forms a keloidal plaque (Fig. 24.11). Abscess formation and secretion of pus may be present in severe cases. Administration of antibacterials is the first-line treatment. Plastic surgery may be necessary.

3. *Pyodermia chronica glutealis*  
Middle-aged men are most frequently affected. Acne-like pustules or papules appear on the lumbar region, genitalia, and thighs, gradually coalescing into a large infiltrative plaque. Abscesses with intricately netted fistulae form, and these excrete pus when pressed (Fig. 24.12). There is hidradenitis suppurativa or acne conglobata as an underlying disease in many cases. Systemic administration of antibiotics, incision and drainage of pus, and removal and skin graft are the main treatments.

### 1. Staphylococcal scalded-skin syndrome (SSSS)

**Synonym:** Staphylococcal toxic epidermal necrolysis (S-TEN)

**Outline**
- It is caused by exfoliative toxins of *Staphylococcus aureus* in the epidermis.
- It occurs most frequently in infants and children up to age 6. A fever and reddening around the mouth or eyes first appear, followed by painful exfoliation, erosion and blistering.
- Nikolsky’s sign is positive.
- Systemic management and care, and administration of antibiotics are the main treatments.
Clinical features

Staphylococcal scalded-skin syndrome (SSSS) occurs most frequently in infants and children up to age 6; it is extremely rare in adults. It begins with reddening and blistering around the mouth, nostrils, and periocular area. This is accompanied by systemic symptoms, such as a fever of about 38°C, irritability, and poor appetite. Wrinkles and fissures around the mouth, eye discharge and crust formation result in characteristic facial features. Erythema occurs on the neck, axillary fossae, and groin, and the whole body skin begins to exfoliate as if burned, which leads to erosion (Figs. 24.13-1 and 24.13-2). Skin at normal sites also exfoliates easily by friction (Nikolsky’s sign is positive). Sharp pain is present. The mucous membranes tend not to be affected. Exfoliation in the scalp can occur in rare cases. SSSS begins to heal after exfoliation is accelerated by systemic administration of antibiotics. The entire course of SSSS is 1 to 2 weeks (Fig. 24.14).

Pathogenesis

SSSS is caused by exfoliative toxin (ET) produced by Staphylococcus aureus. The nasopharynx, conjunctivae, external ears, and navel are most frequently infected. When ET spreads to the whole body by blood circulation, desmoglein 1, a desmosome structural protein, is broken. Pemphigus foliaceus-like acantholytic and intraepidermal blisters form on the upper epidermal layer.

Pathology

Acantholysis, lacunae and infiltration of polymorphonuclear cells are observed under the horny cell layer and in the granular cell layer.

Diagnosis

SSSS can be diagnosed by the characteristic facial features, burn-like systemic epidermal exfoliation, marked Nikolsky’s sign, and normality of oral mucosa.

Differential diagnosis

Most cases of toxic epidermal necrolysis (TEN) are induced by drugs. Histopathologically, there is necrosis in all epidermal layers and severe infiltration to the mucous membranes. Infants are rarely affected by TEN. In widely spread multiple bullous impetigo, the characteristic facial features of SSSS do not occur and Nikolsky’s sign is negative.

Treatment

Hospitalization and systemic care including transfusion and intravenous antibiotics that are effective against Staphylococcus aureus are given. The affected site is sterilized and treated with topical application of ointments that contain antibiotics or petrolatum. SSSS tends to have a good prognosis; however, it may become severe, with sepsis or pneumonia appearing as complica-
tions in infants and in adults with immunodeficiency.

Fig. 24.14 Clinical course of staphylococcal scalded-skin syndrome (SSSS).

2. Toxic shock syndrome (TSS)

Synonym: Staphylococcal toxic shock syndrome (STSS)

TSS is caused by an exotoxin called toxic shock syndrome toxin (TSS toxin-1 or TSST-1), which is produced by infection and proliferation of *Staphylococcus aureus*. It may occur in burn patients and women who use sanitary tampons. TSS is a systemic toxic disease whose main symptoms are acute fever, decreased blood pressure, scarlet-fever-like erythema, and multiple organ dysfunction (Fig. 24.15). Diffuse erythema, chills, headache, and arthralgia on the whole body are present, leading to systemic fatigue, vomiting and diarrhea. Antibiotics, mainly β-lactum drugs, are useful in large doses, in combination with immunoglobulin preparations.

3. Scarlet fever (Streptococcal infection)

- It is erythematous exanthema and enanthema caused by group-A β-hemolytic streptococci (GAS).
- It begins with pharyngeal pain and high fever. It is
characterized by reddening of the tongue (strawberry tongue) and dense erythema on the whole body.

- Eruptions do not appear around the mouth (perioral pallor).
- Increased antistreptolysin O has diagnostic value. Penicillin is administered.

**Clinical features**

Scarlet fever most frequently occurs in late childhood. The incubation period is known to be 2 to 3 days. It begins with a sudden fever and pharyngeal pain, soon followed by strawberry tongue. At the early stages, tongue fur which is often referred to as “white strawberry tongue” is seen in many cases. However, the tongue fur resolves in a day or two, leaving the typical strawberry tongue. Eruptions appear 1 to 2 days after onset of streptococcal infection. Patchy, vivid red papules appear on the neck, axillary fossae, and medial thighs, spreading over the whole body. The eruptions may be accompanied by itching and burning. Diffuse erythema appears on the face, except at the peripheries of the mouth and nasal alae (perioral pallor). Hemorrhagic lesions and systemic lymph node enlargement also occur. As the fever subsides on the third or fourth day after onset, the eruptions begin to disappear. After exfoliation, the eruptions heal without post-inflammatory pigmentation. Clinical features that characterize scarlet fever are shown in Fig. 24.16.

**Pathogenesis**

Eruptions are caused on the whole body by an exotoxin produced by *Streptococcus pyogenes*. This bacterium first infects the palatal tonsil or skin.

**Complications**

Post-infectious complications of *Streptococcus pyogenes* may occur, such as acute glomerulonephritis and rheumatic fever.

**Laboratory findings**

Elevated levels of ASO and ASK, leukocytosis, and left shift of the nuclei in leukocytes are caused by streptococcal infection. Bacteria are detected from the primary infection site, such as the pharynx. The rapid diagnostic test kit is also useful, although the detection sensitivity is relatively low.

**Differential diagnosis**

Rubella, Kawasaki disease and drug eruptions should be differentiated from streptococcal fever (Fig. 23.23).

**Treatment, Prognosis**

Oral penicillin G is the first-line treatment. Although eruptions disappear in 2 to 3 days, administration of penicillin G should be continued for at least 2 weeks because if the medication is stopped early, *Streptococcus* may proliferate again in the pharynx.
Bacterial Infections

ynx, causing complications such as nephritis or rheumatic fever. After termination of medication, periodic examinations such as urine test are necessary to detect bacteria.

4. Necrotizing fasciitis

Outline

- It is an acute bacterial infection in subcutaneous tissue and superficial fascia (Fig. 24.3). The extremities and genitalia of persons middle-aged and older are most frequently affected.
- The main systemic symptoms are reddening and swelling of skin, ulceration, and fever accompanied by intense pain.
- High doses of antibiotics at the early stages and surgical débridement are the main treatments. Multiple organ failure may lead to death.

Clinical features

The extremities (lower legs in particular), genitalia and abdomen of persons over age 40 are most frequently affected. Necrotizing fasciitis begins with localized reddening and swelling that rapidly progress with marked systemic symptoms. In 1 to 3 days, purpura, blisters, bloody blisters, concave necrosis and ulceration occur (Fig. 24.17). The sensation of touch is reduced according to the progression of the fasciitis. Even when the periphery of the lesion appears normal to the naked eye, the subcutaneous tissue is affected. Necrotizing fasciitis is characterized by intense systemic symptoms such as high fever, severe arthralgia, muscle pain, shock and multiple organ failure. Necrotizing fasciitis of the genitalia is called Fournier’s gangrene. Necrotizing fasciitis frequently occurs as a complication of toxic-shock-like syndrome (MEMO).

Pathogenesis

The main causative bacteria are Streptococcus pyogenes and anaerobes such as Bacteroides fragilis and Peptostreptococcus anaerobius. Streptococcus pyogenes may infect healthy persons, leading to a sudden onset of necrotizing fasciitis. Anaerobic bacteria tend to infect individuals with an underlying disease, such as diabetes. In some cases, a micro-injury or tinea pedis induces necrotizing fasciitis; however, details of the pathogenesis are unknown.

Pathology

Edema is marked throughout the dermis. Panniculitis, necrosis, blockage of the blood vessels, and infiltration of polymorphonuclear leukocytes occur from the lower dermal layer to the underlying fat tissue and fascia.