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.
Laboratory findings
Leukocytosis, left shift of the nuclei in leukocytes, elevated levels of CRP, liver failure and coagulation abnormality (when DIC is caused) are present. Prior to administration of antibiotics, bacteria are detected from the puncture fluid, necrotizing tissue at débridement, and blood. MRI, CT, and X-ray images are helpful in testing for the depth and size of lesion and for any retention of gas.

Diagnosis
Prompt diagnosis and initiation of treatment are important. Necrotizing fasciitis is diagnosed by its sudden onset, rapid progression, intense systemic symptoms, purpura, blisters, bloody blisters and the depth of necrosis measured by débridement. Bacterial culture and skin biopsy are conducted for differential diagnosis.

Differential diagnosis
Some cases are difficult to differentiate from ordinary cellulitis; however, necrotizing fasciitis is characterized by rapid progression of skin lesion, purpura and bloody blisters, and intense systemic symptoms. The spread of inflammation and involvement of fascia can be determined by MRI. Gas gangrene is found in the muscle layer of the lesion. Marked retention of gas is observed by X-ray. The causative bacteria are Clostridium.

Treatment, Prognosis
Large doses of antibiotics that are effective against the pathogen (e.g., drugs containing penicillin, clindamycin) and surgical débridement in the early stages are essential. Unless treated in the early stages, the prognosis is extremely poor.

5. Gas gangrene

Outline
● Most cases are caused by anaerobic bacteria such as those of the genus Clostridium. Mortality is high.
● Intense systemic symptoms, muscular necrosis and aerogenesis occur. There is crepitation from gas in the tissues.
● Rapid incision and lavage of the lesion, large doses of antibiotics, and hyperbaric oxygen therapy are the main treatments.

Clinical features
Six to 72 hours after injury, gas gangrene begins with a localized sharp pain. Systemic symptoms such as chills and tachycardia occur. The skin becomes dark purple or blackish. Hematoid serous blisters form. Liquefactive necrosis occurs in muscle tissue. The lesion swells with the gas. The affected site releases foul
odor. When the site is pressed, the gas moves, causing crepitation. Bubbles are observed by X-ray. If left untreated, exotoxin circulates through the bloodstream to the entire body, leading to jaundice, DIC or shock, and resulting in death.

**Pathogenesis**

Gas gangrene is most frequently caused by *Clostridium perfringens* (formerly *Clostridium welchii*), *Clostridium oedematiens*, *Clostridium septicum* and *Clostridium histolyticum*, but in some cases by non-*Clostridium* bacteria. The causal bacteria exist in soil and sometimes in the feces of humans and animals, invading the body through a severely crushed and contaminated wound. These bacteria grow in the anaerobic environment and produce an exotoxin containing proteolytic enzymes, which induce hemolysis and shock.

**Treatment, Prognosis**

Quick incision, lavage and surgical débridement are important. At the same time, penicillin G or cefem antibiotics are administered in large doses. Proliferation of anaerobic bacteria can be prevented by opening the lesion. When the bacteria are anaerobic, hyperbaric oxygen therapy is useful. Systemic care is performed for shock, kidney failure and DIC. Amputation of extremities may be necessary in severe cases.

**6. Sepsis**

Sepsis occurs when a localized cutaneous infection such as in abscess, cellulitis or erysipelas aggravates and disseminates in the blood flow. The bacteria themselves or thrombosis caused by bacteria in the blood induces septic vasculitis, resulting in septicemic formation including erythema, purpura, bloody blisters and pustules.

**7. Osler’s node**

This is transient, painful, nodular erythema that often accompanies subacute bacterial endocarditis. Elevated erythema of 5 mm in diameter occurs on the finger pads, thenar and hypothenar eminences (Fig. 24.18). Sharp pain occurs as a precursor, and a brown patch appears and disappears in 1 or 2 days. Osler’s node accompanied by painless erythema or infiltrative purpura is called Janeway lesion. Allergic reaction against the bacteria and vascular blockage are thought to be the causes in all cases. Osler’s node is known to appear in 15% of infectious endocarditis cases.