early staphylococcal implant-associated infections treated with a fluoroquinolone plus rifampin is >90%. Rifampin is efficacious against staphylococcal biofilms of ≤3 weeks' duration. Similarly, fluoroquinolones are active against biofilms formed by gram-negative bacilli. In these cases, an initial 2-week course of IV therapy with a β-lactam is suggested in order to minimize the risk of emergence of resistance to the oral drugs. The total duration of treatment is 3 months, and the device can be retained even after antibiotics have been discontinued. In contrast, in cases caused by rifampin-resistant staphylococci or fluoroquinolone-resistant gram-negative bacilli, the hardware should be removed after consolidation of the fracture and before discontinuation of antibiotics. These patients are treated with an oral antibiotic (suppressive therapy) as long as retention of the hardware is necessary.

COMPLICATIONS

The main complication of long-bone osteomyelitis is the persistence of infection with progression to chronic osteomyelitis. This risk is especially high after internal fixation of an open fracture and among patients with implant-associated osteomyelitis that is treated without surgical debridement. In chronic osteomyelitis, recurrent sinus tracts result in severe damage to skin and soft tissue (Fig. 158-2). Patients who have chronic open wounds need a therapeutic approach combining orthopedic repair and plastic reconstructive surgery.

PERIPROSTHETIC JOINT INFECTION

PATHOGENESIS

Implanted foreign material is highly susceptible to local infection due to local immunodeficiency around the device. Infection occurs by either the exogenous or the hematogenous route. More rarely, contiguous spread from adjacent sites of osteomyelitis or deep soft-tissue infection may cause periprosthetic joint infection (PJI). The fact that foreign devices are covered with host proteins such as fibronectin favors the adherence of staphylococci and the formation of a biofilm that resists phagocytosis.

EPIDEMIOLOGY

The risk of infection manifesting during the first 2 postoperative years varies according to the joint. It is lowest after hip and knee arthroplasty (0.3–1.5%) and highest after ankle and elbow replacement (4–10%). The risk of hematogenous PJI is highest in the early postoperative period. However, hematogenous seeding occurs throughout life, and most cases therefore develop >2 years after implantation.

MICROBIOLOGY

About 70% of cases of PJI are caused by staphylococci (*S. aureus* and coagulase-negative staphylococci), 10% by streptococci, 10% by gram-negative bacilli, and the rest by various other microorganisms. All microorganisms can cause PJI, including fungi and mycobacteria. *P. acnes* causes up to one-third of episodes of periprosthetic shoulder infection.

CLASSIFICATION AND CLINICAL MANIFESTATIONS

PJI is traditionally classified as early (<3 months after implantation), delayed (3–24 months after surgery), or late (>2 years after implantation). For therapeutic decision-making (see below), it is more useful to classify PJI as (1) acute hematogenous PJI with <3 weeks of symptoms, (2) early postinterventional PJI manifesting within 1 month after surgery, and (3) chronic PJI with symptom duration of >3 weeks.

Acute exogenous PJI typically presents with local signs of infection (Fig. 158-3). In contrast, acute hematogenous PJI, most often caused by *S. aureus*, is characterized by new-onset pain that initially is not accompanied by prominent local inflammatory signs. In most cases, an ongoing sepsis syndrome dominates the clinical picture. Key findings in chronic PJI are joint effusion, local pain, implant loosening, and occasionally a sinus tract. Chronic PJI is most commonly caused by low-virulence microorganisms such as coagulase-negative staphylococci or *P. acnes*. These infections are characterized by nonspecific symptoms, such as chronic pain caused by low-grade inflammation or early loosening.

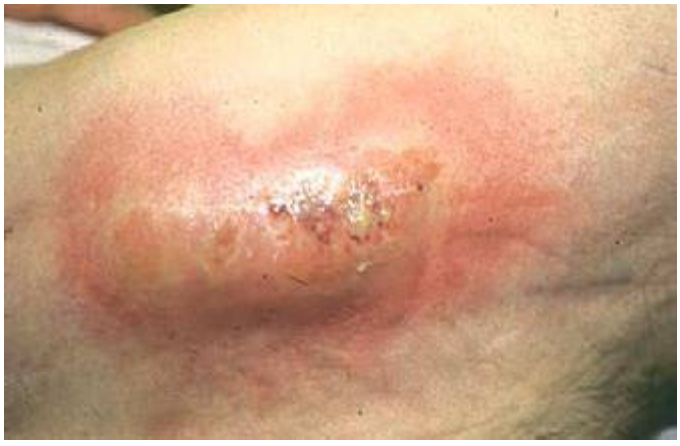

FIGURE 158-3 Early periprosthetic joint infection of the left hip caused by group B streptococci in a 68-year-old woman.

DIAGNOSIS

Blood tests such as the measurement of CRP (elevated levels, ≥10 mg/L) and erythrocyte sedimentation rate (elevated rates, ≥30 mm/h) are sensitive (91–97%) but not specific (70–78%). Synovial fluid cell counts are ~90% sensitive and specific, with threshold values of 1700 leukocytes/μL in periprosthetic knee infection and 4200 leukocytes/μL in periprosthetic hip infection. During debridement surgery, at least three but optimally six tissue samples should be obtained for culture and histopathology. If implant material (modular parts, screws, or the prosthesis) is removed, sonication of this material followed by culture and/or use of molecular methods to examine the sonicate fluid allows the detection of microorganisms in biofilms.

The three-phase bone scan is very sensitive for detecting PJI but is not specific. As mentioned above, this test does not differentiate bone remodeling from infection and therefore is not useful during at least the first year after implantation. CT and MRI detect soft tissue infection, prosthetic loosening, and bone erosion, but imaging artifacts caused by metal implants limit their use. ^{18}F-fluorodeoxyglucose PET is an alternative method with fair sensitivity and specificity for the detection of PJI. However, this technique is not yet an established procedure for this purpose.

TREATMENT PERIPROSTHETIC JOINT INFECTION

Treatment of PJI requires a multidisciplinary approach involving an experienced orthopedic surgeon, an infectious disease specialist, a plastic reconstructive surgeon, and a microbiologist. Therefore, most patients are referred to a specialized center. In general, the goal of treatment is cure—i.e., a pain-free functional joint with complete eradication of the infecting pathogen(s). However, for patients with severe comorbidity, lifelong suppressive antimicrobial therapy may be preferred. As a rule, antimicrobial therapy without surgical intervention is not curative but merely suppressive. There are four curative surgical options: debridement and implant retention, one-stage implant exchange, two-stage implant exchange, and implant removal without replacement. Implant retention offers a good chance of infection-free survival (>80%) only if the following conditions are fulfilled: (1) acute infection, (2) stable implant, (3) pathogen susceptible to a biofilm-active antimicrobial agent (see below), and (4) skin and soft tissue in good condition.

Table 158-2 summarizes pathogen-specific antimicrobial therapy for PJI. Initial IV therapy is followed by long-term oral antibiotics. Efficacious treatment is best defined in staphylococcal implant-associated infections. Rifampin exhibits excellent activity against