biofilms composed of susceptible staphylococci. Because of the risk of rapid emergence of resistance, rifampin must always be combined with another effective antibiotic. If gram-negative infections are treated with implant retention, fluoroquinolones should be used because of their activity against gram-negative biofilms.

PREVENTION OF HEMATOGENOUS INFECTION

As mentioned above, hematogenous seeding may occur throughout life. This risk is highest during *S. aureus* bacteremia from a distant focus. Therefore, documented bacterial infections should be promptly treated in patients with prosthetic joints. However, according to a large, prospective, case-control study, the risk of prosthetic hip or knee infection is not increased following dental procedures. Therefore, antibiotic prophylaxis is not needed during dental work.

STERNAL OSTEOMYELITIS

PATHOGENESIS

Sternal osteomyelitis occurs primarily after sternal surgery (with the entry of exogenous organisms) and more rarely by hematogenous seeding or contiguous extension from adjacent sites of sternocostal arthritis. Exogenous sternal osteomyelitis after open sternal surgery is also called *deep sternal wound infection*. Exogenous infection may also follow minor sternal trauma, sternal fracture, and manubriosternal septic arthritis. Tuberculous sternal osteomyelitis typically manifests during hematogenous seeding in children or as reactivated infection in adults. Reactivation is sometimes preceded by blunt trauma. In rare cases, tuberculous sternal osteomyelitis is caused by continuous infection from an infected internal mammary lymph node.

EPIDEMIOLOGY

The incidence of poststernotomy wound infection varies from 0.5% to 5%, but figures are even higher among patients with risk factors such as diabetes, obesity, chronic renal failure, emergency surgery, use of bilateral internal mammary arteries, and reexploration for bleeding. Rapid diagnosis and correct management of superficial sternal wound infection prevent its progression to sternal osteomyelitis. Primary (hematogenous) sternal osteomyelitis accounts for only 0.3% of all cases of osteomyelitis. Risk factors are IV drug use, HIV infection, radiotherapy, blunt trauma, cardiopulmonary resuscitation, alcohol abuse, liver cirrhosis, and hemoglobinopathy.

MICROBIOLOGY

Poststernotomy osteomyelitis is generally caused by *S. aureus* (40–50% of cases), coagulase-negative staphylococci (15–30%), enterococci (5–12%), or gram-negative bacilli (15–25%). Fungal infections caused by *Candida* species also play a role. The fact that ~20% of cases are polymicrobial is indicative of exogenous superinfection during therapy. Hematogenous sternal osteomyelitis is caused most commonly by *S. aureus*. Other microorganisms play a role in special populations—e.g., *P. aeruginosa* in IV drug users, *Salmonella* species in individuals with sickle cell anemia, and *M. tuberculosis* in patients from endemic areas who have previously had tuberculosis.

CLINICAL MANIFESTATIONS

Exogenous sternal osteomyelitis manifests as fever, increased local pain, erythema, wound discharge, and sternal instability (Fig. 158-4). Contiguous mediastinitis is a feared complication, occurring in ~10–30% of patients with sternal osteomyelitis. Hematogenous sternal osteomyelitis is characterized by sternal pain, swelling, and erythema. In addition, most patients have systemic signs and symptoms of sepsis.

The differential diagnosis of hematogenous sternal osteomyelitis includes immunologic processes typically presenting as systemic or multifocal inflammation of the sternum or the sternoclavicular or sternocostal joints (e.g., SAPHO [synovitis, acne, pustulosis, hyperostosis, osteitis], vasculitis, and chronic multifocal relapsing osteomyelitis).

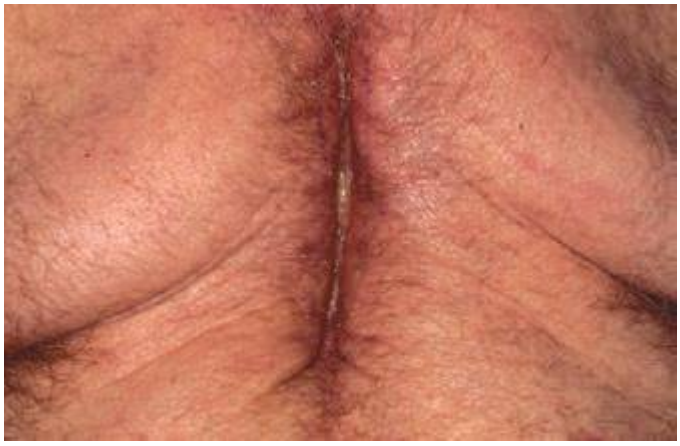

FIGURE 158-4 **Sternal osteomyelitis caused by *Staphylococcus epidermidis* 5 weeks after sternotomy for aortocoronary bypass in a 72-year-old man.**

DIAGNOSIS

In primary sternal osteomyelitis, the diagnostic workup does not differ from that in other types of hematogenous osteomyelitis (see above). When a patient has grown up in regions where tuberculosis is endemic, a specific workup for mycobacterial infection should be performed, especially if osteomyelitis had its onset after a blunt sternal trauma. In secondary sternal osteomyelitis, leukocyte counts may be normal, but the CRP level is >100 mg/L in most cases. Tissue sampling for microbiologic studies is crucial. In osteomyelitis associated with sternal wires, low-virulence microorganisms, such as coagulase-negative staphylococci, play an important role. In order to differentiate between colonization and infection, samples from at least three deep biopsies should be subjected to microbiologic examination. Superficial swab cultures are not diagnostic and may be misleading. No studies have compared the value of the various imaging modalities in suspected primary sternal osteomyelitis. However, MRI is the current gold standard for detection of each type of osteomyelitis.

TREATMENT STERNAL OSTEOMYELITIS

In cases of deep sternal wound infection, antibiotic therapy should be started immediately after samples have been obtained for microbiologic analyses in order to control clinical sepsis. To protect a newly inserted heart valve, initial treatment should be directed against staphylococci, with consideration of the local susceptibility pattern. In centers with a high prevalence of methicillin-resistant *S. aureus*, vancomycin or daptomycin should be added to a broad-spectrum β-lactam drug. As soon as cultures of blood and/or deep wound biopsies have confirmed the pathogen's identity and susceptibility pattern, treatment should be optimized and narrowed accordingly. Tables 158-1 and 158-2 show appropriate therapeutic choices for the most frequently identified microorganisms causing sternal osteomyelitis in the absence and presence, respectively, of an implanted device. In a recent observational study of patients with staphylococcal deep sternal wound infection, the use of a rifampin-containing regimen was predictive of success. The optimal duration of antibiotic therapy has not been established. In acute sternal osteomyelitis without hardware, a 6-week course is the rule. In patients with remaining sternal wires, treatment duration is generally prolonged to 3 months (Table 158-2). Like other types of tuberculous bone infection, tuberculous sternal osteomyelitis is treated for 6–12 months.

Primary sternal osteomyelitis can generally be treated without surgery. In contrast, in secondary sternal osteomyelitis, debridement is always required. This procedure should be performed by a team of experienced surgeons, since mediastinitis, bone infection,