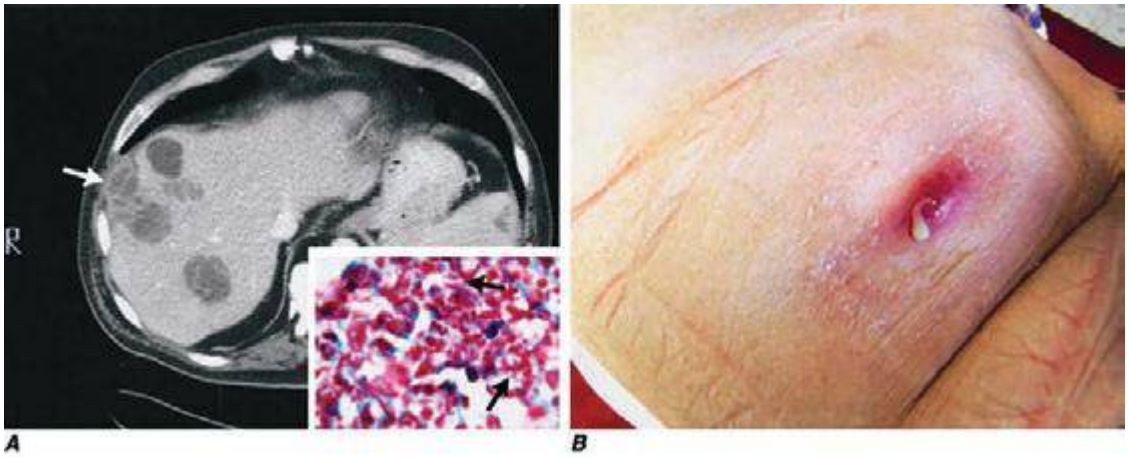

FIGURE 200-3 Hepatic-splenic actinomycosis. *A.* Computed tomogram showing multiple hepatic abscesses and a small splenic lesion due to *A. israelii.* Arrow indicates extension outside the liver. *Inset:* Gram's stain of abscess fluid demonstrating beaded filamentous gram-positive rods. ***B.*** Subsequent formation of a sinus tract. *(Reprinted with permission from Saad M: Actinomyces hepatic abscess with cutaneous fistula. N Engl J Med 353:e16, 2005. © 2005 Massachusetts Medical Society. All rights reserved.)*

surgery, osteoradionecrosis and bisphosphonate osteonecrosis (limited to mandibular and maxillary bones), or hematogenous spread. Because of slow disease progression, new bone formation and bone destruction are seen concomitantly. Infection of an extremity is uncommon and is usually a result of trauma. Skin, subcutaneous tissue, muscle, and bone (with periostitis or acute or chronic osteomyelitis) are involved alone or in various combinations. Cutaneous sinus tracts frequently develop.

Disseminated Disease Hematogenous dissemination of disease from any location rarely results in multiple-organ involvement. *A. meyeri* is most commonly involved. The lungs and liver are most commonly affected, with the presentation of multiple nodules mimicking disseminated malignancy. The clinical presentation may be surprisingly indolent given the extent of disease.

DIAGNOSIS

The diagnosis of actinomycosis is rarely considered. All too often, actinomycosis is first mentioned by the pathologist after extensive surgery. Since medical therapy alone is frequently sufficient for cure, the challenge for the clinician is to consider the possibility of actinomycosis, to diagnose it in the least invasive fashion, and to avoid unnecessary surgery. The clinical and radiographic presentations that suggest actinomycosis are discussed above. Of note, hypermetabolism has been demonstrated by ^{18}F-fluorodeoxyglucose positron emission tomography (FDG-PET) in actinomycotic disease. Aspirations and biopsies (with or without CT or ultrasound guidance) are being used successfully to obtain clinical material for diagnosis, although surgery may be required. The diagnosis is most commonly made by microscopic identification of sulfur granules (an in vivo matrix of bacteria, calcium phosphate, and host material) in pus or tissues. Occasionally, these granules are identified grossly from draining sinus tracts or pus. Although sulfur granules are a defining characteristic of actinomycosis, granules also are found in mycetoma **(Chaps. 199 and 243)** and botryomycosis (a chronic suppurative bacterial infection of soft tissue or, in rare cases, visceral tissue that produces clumps of bacteria resembling granules). These entities can easily be differentiated from actinomycosis with appropriate histopathologic and microbiologic studies. Microbiologic identification of actinomycetes is often precluded by prior antimicrobial therapy or failure to perform appropriate microbiologic cultures. For optimal yield, the avoidance of even a single dose of antibiotics is mandatory. Primary isolation usually requires 5–7 days under anaerobic conditions but may take as long as 2–4 weeks. Although not routinely used, 16S rRNA gene amplification and sequencing have been successfully applied to increase diagnostic sensitivity and specificity. Because actinomycetes are components of the normal oral and genital-tract flora, their identification in the absence of sulfur granules in sputum, bronchial washings, and cervicovaginal secretions is of little significance.

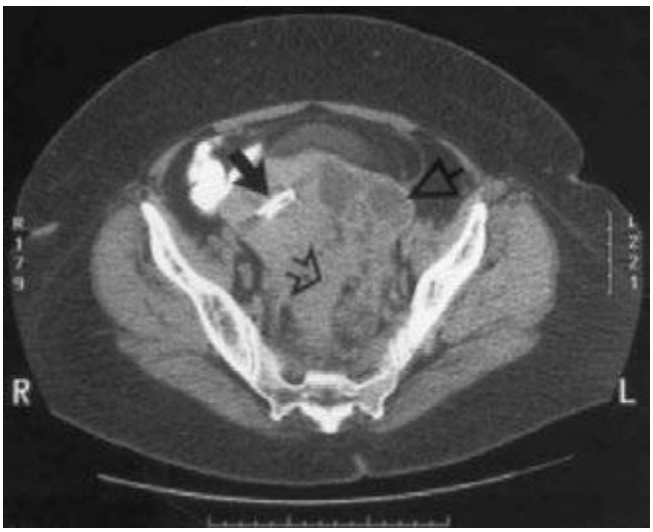

FIGURE 200-4 Computed tomogram showing pelvic actinomycosis associated with an intrauterine contraceptive device. The device is encased by endometrial fibrosis (*solid arrow*); also visible are paraendometrial fibrosis (*open triangular arrowhead*) and an area of suppuration (*open arrow*).

TREATMENT ACTINOMYCOSIS

Decisions about treatment are based on the collective clinical experience of the past 65 years. Actinomycosis requires prolonged treatment with high doses of antimicrobial agents; suitable antimicrobial agents and those deemed unreliable are listed in **Table 200-1**. The need for intensive treatment is presumably due to the drugs' poor penetration of the thick-walled masses common in this infection and/or the sulfur granules themselves, which may represent a biofilm. Although therapy must be individualized, the IV administration of 18–24 million units of penicillin daily for 2–6 weeks, followed by oral therapy with penicillin or amoxicillin (total duration, 6–12 months), is a reasonable guideline for serious infections and bulky disease. Less extensive disease, particularly that involving the oral-cervicofacial region, may be cured with a shorter course. If therapy is extended beyond the resolution of measurable disease, the risk of relapse—a clinical hallmark of this infection—will be minimized; CT and MRI are generally the most sensitive and objective techniques