Fungal spondylitis
A case of Torulopsis glabrata and Candida tropicalis infection

A case of spondylitis due to Torulopsis glabrata and Candida tropicalis is reported. Fungal osteomyelitis should be suspected in the presence of predisposing factors, such as long antibiotic treatment or reduced immune defense.

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Case report
A 63-year-old woman was admitted to our hospital for treatment of osteomyelitis of the spine, located at the 7th and 8th dorsal vertebrae. For a few years, she was known to have nephrolithiasis. Three months previously, she was admitted to another hospital with a fever up to 40°C and renal colics. The intravenous pyelogram showed renal calculi on the left side. For treatment of the urosepsis, she first received mezlin and garamycin because the bacteriologic examination of the urine showed growth of Proteus mirabilis and Klebsiella with sensitivity to these antibiotics. The fever did not respond, and the antibiotics were changed eight times. Four weeks after the beginning of antibiotic therapy, Candida tropicalis was cultured from blood and urine and was sensitive to miconazole. Four weeks after this antifungal therapy, she recovered and could be discharged.

Figure 1. Fungal spondylitis of the 7th and 8th dorsal vertebrae with narrowing of the disc space and a lytic lesion.
Two weeks later the patient was readmitted with fever and pain in both flanks with a strong tenderness of the whole dorsal back and extreme pain at the 7th and 8th dorsal vertebrae. The neurologic examination was normal. Radiographic examination of the dorsal spine showed an osteolytic process of the 7th and 8th dorsal vertebrae with narrowing of the vertebral bodies, destruction of the bone plates, and diminished intervertebral space (Figure 1). A costotransversectomy was performed with removal of the inflamed tissue and stabilization with autologous bone graft from the iliac crest. The microbiologic examination with culture of the bone marrow showed growth of *Torulopsis glabrata* and *Candida tropicalis*, both being sensitive to amphotericin B and 5-fluorocytosine; the patient received respectively 0.6 mg/kg and 150 mg/kg daily of these two drugs. Postoperatively, she was immobilized for 12 weeks in a body cast. After 8 weeks, the antifungal therapy was changed to ketoconazol, 400 mg daily for 12 weeks combined with flucloxacillin, 3 grams daily, as antibacterial prophylaxis. Under this therapy, the clinical and chemical signs showed improvement, and the pain subsided. After 5 months of immobilization, the patient could be mobilized in a plastic brace. Radiographic controls showed improvement of the osteomyelitis and stabilization of the infected area.

At the follow-up examination 1.5 years after surgery, the patient was almost painless and without any clinical or radiographic signs of inflammation (Figure 2). The erythrocyte sedimentation rate was 42 mm; the latex test for *Candida* antigens was negative, and the *Candida* antibody titer had decreased to normal.

**Discussion**

To our knowledge, our patient is the third reported case of osteomyelitis and the second one of spondylitis due to *Torulopsis glabrata*. She is a further example of the predisposing capacity of long-lasting antibiotic therapy to infection with saprophytic agents. *Candida tropicalis* and *Torulopsis glabrata* could be cultured from bone specimens, and we presume that both yeasts were pathogenic agents.

Marks et al. (1970) found that all patients in whom *Torulopsis glabrata* was considered to be the pathogenic agent had major underlying illnesses and/or had received antibiotics, corticoids, or immunosuppressives. Also, in nearly all the cases of *Candida* osteomyelitis some predisposing factors could be found.

The diagnosis of mycotic osteomyelitis requires a high index of suspicion by the clinician in the presence of predisposing factors. Clinical and radiographic signs and blood tests are am-
ambiguous, and an altered erythrocyte sedimentation rate often is the only positive finding (Simpson et al. 1977).

The final diagnosis can only be done by microbiologic examination of specimens obtained from the inflamed tissue by surgery.

The treatment of fungal osteomyelitis consists of three parts: chemotherapy, immobilization, and surgical drainage if necessary. Among the few chemotherapeutic agents the best choice is probably to combine amphotericin B and 5-fluorocytosine (Bennett 1974, Gustke & Wu 1981, Thurston & Gillespie 1981, Webb et al. 1970). If there are contraindications to these drugs, chemotherapy with imidazol derivatives is another possibility. A sufficient immobilization for weeks is necessary until the acute inflammation has subsided.

Surgical treatment is indicated in cases with neurologic complications, abscess formation, destruction of vertebral bodies without expectation of sufficient healing, septicemia, and relapse or lacking in response to nonsurgical treatment. To prevent mycotic osteomyelitis, clinicians should pay attention to the presence of predisposing factors that facilitate the outburst of infection: e.g., a primary fungal nonosteogenic infection should be carefully treated. In cases of long-term antibiotic or immunosuppressive therapy or planned major surgical procedures, a general prophylaxis might even be considered (Commers & Pizzo 1983, Meunier-Carpentier 1983).

References