Isolated Tricuspid Valve Endocarditis Due to *Streptococcus bovis*

Involvement of the tricuspid valve occurs in 10%-15% of patients with infective endocarditis. *Staphylococcus aureus* is the most common cause of tricuspid valve endocarditis (TVE), particularly in drug addicts and patients with central venous catheters. Various species of streptococci cause TVE, which most often occurs in association with left-sided endocarditis. *Streptococcus bovis* is a rare cause of TVE. We report what we believe to be the second documented case of isolated TVE secondary to *S. bovis* infection [1] and note the virulence of this organism when it infects the tricuspid valve.

A 34-year-old male with a history of mild asthma and hepatitis B, but no high-risk behavior, was admitted to the hospital because of dyspnea, persistent dry cough, and high fevers. These symptoms had gradually worsened during the 2 weeks before admission. The patient did not have a history of heart murmur and engaged in daily vigorous aerobic exercise. On physical examination, he appeared restless; his oral temperature was 104°F, and his respiratory rate was 40. He appeared to have mild jaundice. A systolic murmur could be heard over the tricuspid area. Auscultation of the lungs revealed bilaterally decreased respiratory sounds, with 87% segmented neutrophils, 6% band forms, 5% lymphocytes, and 1% monocytes. Laboratory studies revealed the following values: hemoglobin, 74 g/L; hematocrit, 21.4%; and platelet count, 124 x 10^9/L. Liver function tests revealed the following abnormal values: alkaline phosphatase, 194 U/L; alanine aminotransferase, 152 U/L; aspartate aminotransferase, 117 U/L; γ-glutamyl transpeptidase, 103 U/L; and lactate dehydrogenase, 843 U/L. The prothrombin time was 13.4 seconds, and the partial thromboplastin time was 25.5 seconds. Chest radiographs obtained on admission revealed diffuse patchy infiltrates in both lung fields. An electrocardiogram did not reveal any abnormalities. A transesophageal echocardiogram obtained on the second hospital day showed a 1.4 x 1.1-cm vegetation on the septal leaflet of the tricuspid valve, as well as significant tricuspid regurgitation.

Two cultures of blood drawn on admission were positive for a *Streptococcus* species. The organism was identified by the Mini ID Screen (Carr-Scarborough Microbiological, Decatur, GA) as *S. bovis*; it was susceptible to ampicillin, cephalothin, clindamycin, erythromycin, tetracycline, and vancomycin. Susceptibility to gentamicin was not determined, as *S. bovis* does not fit the screen for testing with this method.

The patient was initially treated with ampicillin and gentamicin, followed by vancomycin and gentamicin. Despite aggressive antibiotic treatment, he continued to have high spiking fevers and respiratory compromise, and bilateral infiltrates persisted on the chest radiographs, indicating pulmonary septic embolization from a tricuspid valve vegetation. Repeated blood cultures remained negative. Two weeks after admission, transesophageal echocardiography was repeated and showed a new 2 x 1-cm vegetation on the anterior leaflet of the tricuspid valve, in addition to the septal leaflet vegetation present on the initial echocardiogram. He therefore underwent excision of the tricuspid valve, which was replaced with a porcine valve (#29). Pathological examination of the excised valve tissue revealed two tricuspid valve vegetations with multiple ruptured chordae. Cultures of the tissues subsequently yielded *S. bovis*. The postoperative medical regimen included anti-coagulative therapy with intravenous heparin and antibacterial therapy with vancomycin and gentamicin.

In the postoperative period, the patient developed several complications including acute renal failure secondary to gentamicin toxicity, metabolic encephalopathy, and mechanical ventilation–associated pneumonia and bacteroides empyema that was treated by adding clindamycin to his regimen. Because he had difficulty being weaned off mechanical ventilation, he required tracheostomy. Two weeks after surgery, when he had been successfully weaned off the ventilator and the complications had resolved, he underwent pancolonoscopy for a possible source of *S. bovis* because there is a recognized association between colon cancer or polyps and *S. bovis* endocarditis. This procedure did not reveal any abnormalities. The patient's liver function abnormalities gradually abated, and 8 weeks after admission he was discharged to his home.
Fluconazole-Resistant Candida parapsilosis Fungemia in a Patient with AIDS

Candida species are frequently responsible for opportunistic infections in HIV-seropositive patients. Typical infections include oral thrush, esophagitis, and—less frequently, especially in patients with advanced AIDS—fungemia, which is often associated with the presence of a central venous catheter and administration of parenteral nutrition [1, 2]. As already reported in the medical literature [3–5], the fluconazole-resistant Candida strains account for 9%–11% of the isolates from HIV-seropositive patients with candidiasis. A recent report has also documented the isolation of fluconazole-resistant Candida species from patients with cancer, either after the use of fluconazole as prophylaxis or after its use as primary therapy for superficial candidiasis [6].

In this report we describe what we believe to be the first case of (1) fungemia due to fluconazole-resistant Candida parapsilosis in a patient with AIDS who had repeatedly received fluconazole for Candida albicans oral infections and (2) C. parapsilosis fungemia in a patient with HIV infection.

A 31-year-old female iv drug abuser, seropositive for antibodies to HIV since 1985, was admitted to our ward for the first time in March 1994. She had been receiving antiretroviral therapy since June 1991 and primary prophylaxis with pentamidine for Pneumocystis carinii pneumonia since March 1992. The patient had had toxoplasmosis encephalitis in 1993, for which she was receiving chronic suppressive therapy with pyrimethamine (50 mg daily po) and sulfadiazine (2 g daily po). In the previous 3 years the patient had been treated with repeated (more than five times a year) courses of fluconazole (50–300 mg daily po for 7–14 days) for recurrent episodes of oral thrush due to C. albicans.

The chest roentgenographic findings were normal, and no clinical or echocardiographic signs of valvular lesions were noted. Three blood cultures were positive for a fungus, and a Candida antigen test was positive (titer, 1:2). On the basis of this data, the catheter was changed and fluconazole (400 mg iv daily) was administered for a few days but produced no improvement.

Subsequently, the fungus was morphologically and biochemically (Automicrobic Vitek System, bioMérieux, Rome) identified as C. parapsilosis. Its in vitro susceptibility to antifungal drugs was determined with use of a microdilution broth [7]. The inoculum of the isolate was adjusted to a final inoculum of 10^5 cells mL⁻¹, and the results (obtained after 48 hours of incubation) were expressed as MICs. The MICs of amphotericin B, flucytosine, ketoconazole, itraconazole, and fluconazole were 2.5 μg/mL, 0.19 μg/mL, 25 μg/mL, > 100 μg/mL, and >100 μg/mL, respectively. On the

References