**Streptococcus bovis** Discitis and Possible Endocarditis

A 71-year-old woman had a six-month history of fever and low back pain. Her past medical history was unremarkable. Physical examination revealed a temperature of 38.8°C, diffuse petechiae at the lower extremities, retinal hemorrhage which was not a typical Roth’s spot, and a grade 3 holosystolic murmur over the precordium. Laboratory parameters included a leukocyte count of 5,900/µl (81% neutrophils), with a hemoglobin level of 9.4 g/dl and a platelet count of 144,000/µl. Rheumatoid factor was negative. The level of circulating immune complex Clq was high at 6.2 µg/ml (normal range < 3.0 µg/ml). Transesophageal echocardiography showed no vegetations, but a small, perimembranous ventricular septal defect was found. Computed tomography showed erosive lesions in the L4 endplate. T2-weighted magnetic resonance imaging also revealed a L4 endplate erosion and a L4–L5 disc space narrowing. Technetium bone scan showed increased activity in L4 to L5. Two sets of blood cultures yielded *Streptococcus bovis*. Gastroscopy and colonoscopy showed no adenomas or carcinomas. The presumptive diagnoses were discitis and possible endocarditis (1) caused by *S. bovis*. Despite one week cefazolin (2 g intravenously every 12 hours) followed by one week of penicillin (18 million units intravenously per day) plus gentamicin (30 mg intravenously every 8 hours) administration, the fever persisted. Susceptibility testing of *S. bovis* disclosed a minimal inhibitory concentration (MIC) of cefazolin of 2 µg/ml, gentamicin greater than 16 µg/ml, and penicillin, 4 µg/ml. *S. bovis* was susceptible to imipenem (MIC, 0.25 µg/ml) and vancomycin (MIC, 2 µg/ml). Defervescence and improvement of low back pain followed one week of imipenem (0.5 g intravenously every 8 hours) and streptomycin (0.5 g intramuscularly twice a week) administration. One week after the fever disappeared, the patient developed diarrhea. Vancomycin (0.5 g intravenously every 12 hours) and streptomycin were given subsequently to the subside of the diarrhea. Two weeks later, diarrhea and fever again appeared with clostridium difficile toxin positive in stool. Cessation of vancomycin and streptomycin therapy and oral vancomycin administration ameliorated the symptoms. The patient recovered and the vertebral radiographic abnormalities improved.

According to the Duke criteria (1), the present case included one major criterion, positive *S. bovis* in blood cultures, and two minor criteria, presence of ventricular septal defect and fever (≥38.0°C). Moreover, the patient had petechiae, retinal hemorrhage, and a high level of circulating immune complex Clq. We concluded to treat the patient as having endocarditis.

Two cases of endocarditis caused by penicillin-resistant *S. bovis* were reported 21 years ago (2). Recently, Poyart et al (3) showed a vancomycin-resistant *S. bovis* strain from the stool of a patient. Appropriate antimicrobial therapy based on the susceptibility analysis of the isolated organisms is needed in the treatment of gram-positive cocci infections.

Musculoskeletal manifestations were common and initial symptoms in patients with endocarditis. Churchill et al (4) demonstrated musculoskeletal manifestations in 84 of 192 patients with endocarditis. Moreover, twenty-four patients including five complicated with discitis had low back pain. Radiographic examinations should be considered in patients with bacteremia or endocarditis manifesting musculoskeletal symptoms.

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References