Unusual Case of Aeromonas hydrophila Endocarditis

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We describe a case of Aeromonas hydrophila endocarditis in a 66-year-old man with myelodysplastic syndrome and non-A, non-B hepatitis. The infection resolved with antibiotic therapy, but the patient succumbed to complications of his underlying illness. This is the second case of Aeromonas endocarditis reported in the world literature.

Aeromonas hydrophila is a waterborne, facultatively anaerobic, gram-negative bacillus associated with gastroenteritis (2, 9) and soft tissue infections (8) in normal individuals and disseminated infections in immunocompromised individuals (3, 11). A case of endocarditis caused by an Aeromonas sp. has previously been described in a patient with cirrhosis and renal failure (4). We report an additional case of Aeromonas endocarditis in a patient with multiple underlying diseases.

A 66-year-old man was admitted to St. Luke’s-Roosevelt Hospital Center in October 1989 with 3 days of chest pain, fever, and right knee swelling. During the preceding 6 months, he had been diagnosed with myelodysplastic syndrome, transfusion-related non-A, non-B hepatitis with cirrhosis, colon carcinoma in situ, and subacute thryoiditis. His other illnesses included non-insulin-dependent diabetes, essential hypertension, angina, and congestive heart failure. He had no history of smoking or alcohol or other substance abuse. He had no pets, and there was no history of recent travel. At the time of admission, his medications included prednisone, 20 mg daily; hydrochlorothiazide, 50 mg daily; nitroglycerin, 6.5 mg twice daily (BID); propranolol, 40 mg BID; and oxymetholone, 50 mg BID.

Physical examination disclosed a temperature of 39.3°C (102.8°F), a i/vi systolic ejection murmur at the left sternal border without radiation, and a swollen, warm, tender right knee. There were no other localizing findings, Roth spots on fundoscopic examination, or additional peripheral stigmata of endocarditis. The chest roentgenogram and electrocardiogram were unremarkable. The initial laboratory evaluation revealed a peripheral leukocyte count of 4,700/mm³, with 84% granulocytes, hematocrit of 28.1%, and platelets of 10,000/mm³. Liver function tests were also markedly abnormal, with serum glutamic oxalacetic transaminase-aspartate aminotransferase levels of 988 U/liter, lactate dehydrogenase level of 656 U/liter, γ-glutamyl transpeptidase of 1232 U/liter, total bilirubin level of 11.2 mg/dl, alkaline phosphatase level of 139 U/liter, and serum ammonia level of 195 μmol/liter. Treatment with intravenous piperacillin, 3 g every 6 h, and gentamicin adjusted for creatinine clearance was begun. Cultures of urine and joint fluid from the right knee were without growth. However, two sets of blood cultures drawn on admission and inoculated into BACTEC 6A aerobic and 7A anaerobic bottles (BACTEC; Johnston Laboratories, Towson, Md.) were detected as positive by a BACTEC 600 analyzer after 72 h of incubation; Gram stain indicated gram-negative rods. Because of the blood culture results and the murmur noted on auscultation of the chest, echocardiography was performed. Aortic valve vegetations and decreased left ventricular compliance were observed; these were indicative of left-sided endocarditis. No vegetations were observed on the patient’s right knee.

The isolate from the patient’s blood was identified from subcultures to chocolate and MacConkey agars as A. hydrophila by using MicroScan Negative Combo 5 Dry Panels (Baxter Healthcare Corp., West Sacramento, Calif.) and API 20E (Analytab Products, Sherwood Medical, Plainview, N.Y.). The organism was oxidase positive, fermented glucose and sucrose, was indole and o-nitrophenyl-β-D-galactopyranoside positive, and metabolized lysine and arginine but not ornithine.

Breakpoint susceptibilities obtained by using the Microscan Negative Combo 5 Dry Panels read on the AutoScan 4 analyzer (Baxter Healthcare Corp.) and verified by visual inspection indicated that the organism was susceptible to amikacin, cefazolin, cefotetan, cefoxitin, ceftriaxone, ceftazidime, cefoperazone, ciprofloxacin, gentamicin, imipenem, mezlocillin, piperacillin, ticarcillin, tetracycline, tobramycin, and trimethoprim-sulfamethoxazole but was resistant to ampicillin and ampicillin-sulbactam. Testing with Microscan Negative Combo Type 8 Frozen Panels (Baxter Healthcare Corp.) confirmed the resistance of the organism to ampicillin and ampicillin-sulbactam, with MICs of >16 and >16/8 μg/ml, respectively. MICs of the other antibiotics were as follows: amikacin, 4 μg/ml; cefazolin, 4 μg/ml; cefotetan, ≤16 μg/ml; cefoxitin, ≤2 μg/ml; ceftazidime, ≤2 μg/ml; ceftriaxone, ≤2 μg/ml; cephalothin, ≤8 μg/ml; ciprofloxacin, ≤1 μg/ml gentamicin, 2 μg/ml; mezlocillin, ≤8 μg/ml; pipercillin, ≤8 μg/ml; tetracycline, ≤4 μg/ml; tobramycin, 2 μg/ml; trimethoprim-sulfamethoxazole ≤0.5/9.5 μg/ml.

Antibiotic therapy was changed to intravenous cefazolin, 2 g every 8 h, plus gentamicin on the fifth hospital day. The patient defervesced the next day and remained afebrile until day 28. Cultures drawn at that time yielded Pseudomonas aeruginosa susceptible only to ceftazidime, imipenem, and ciprofloxacin. Antibiotic therapy was changed to parenteral ceftazidime and gentamicin, and the patient again defervesced. However, his hepatic function continued to deteriorate, and he died on the 42nd hospital day.

As with other gram-negative organisms, the risk of disseminated Aeromonas infections is increased in immuno-

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compromised individuals, particularly those with malignancies or impaired hepatobiliary function (3). In some cases, the source of infection has been exotic, such as alligator (7) or piranha (10) bites, or following the use of medicinal leeches (1). In the United States, Aeromonas spp. have been isolated from the feces of 2 to 3% of individuals studied (12), and the gastrointestinal tract may serve as a source of infection. Indeed, an environmental source was initially postulated by Davis et al. (4), who described the first case of Aeromonas endocarditis in a 63-year-old woman with alcoholic cirrhosis and renal failure on ambulatory peritoneal dialysis who had been in contact with flood water in her basement. However, cultures from the patient’s basement and dialysate were negative for Aeromonas spp., and those authors concluded that the source was probably gastrointestinal.

Our patient suffered from multiple illnesses that increased his susceptibility to infection with Aeromonas spp. His hepatic function was severely impaired as a result of non-A, non-B hepatitis with cirrhosis, and hepatic failure was the ultimate cause of death. Although the precise reason for the increased susceptibility to gram-negative infections in patients with liver failure is unknown, decreased clearance of microorganisms by the hepatic reticuloendothelial system has been postulated. Additionally, acquired complement deficiency as a result of impaired synthetic ability and defective opsonization have been demonstrated in serum from patients with fulminant liver failure (13). An impaired hematopoietic response caused by myelodysplasia with decreased production of granulocytes and other phagocytic cells may have been a factor, as in the case of an A. hydrophila infection in a patient with cyclic neutropenia described by Revord et al. (10). Treatment with prednisone, immune dysfunction as represented by the development of subacute thyroiditis, and underlying colon carcinoma may also have been contributory.

Our patient had no pets and no history of recent marine injury or other environmental exposure. No Aeromonas spp. were isolated from cultures of local or hospital water samples inoculated onto chocolate agar or cooked meat broth (BBL Products, Becton Dickinson Microbiology Systems, Cockeysville, Md.). Thus, the source of infection in our patient was also most likely gastrointestinal.

Although the isolate from our patient was susceptible to piperacillin in vitro, resistance of A. hydrophila to the ureidopenicillins has been demonstrated previously (5, 6). The rapid lysis of fever in our patient after the change in therapy to cefazolin and gentamicin suggests that cephalosporin therapy may be preferable.

In summary, we reported an additional case of Aeromonas endocarditis in a man with myelodysplastic syndrome and cirrhosis caused by non-A, non-B hepatitis. Despite successful antibiotic therapy, the patient succumbed to his underlying hepatic disorder.

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REFERENCES