Serratia odorifera Biogroup 1 Causing an Invasive Human Infection

HERMAN CHMEL

Division of Infectious and Tropical Diseases, Department of Internal Medicine, College of Medicine, University of South Florida, Tampa, Florida 33612

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Serratia odorifera biogroup 1 was isolated from the blood and urine of an alcoholic male with cirrhosis and signs of septic shock. The organism is rarely reported to occur in clinical specimens. This is the first case in which the organism was found to be responsible for invasive human infection.

In 1978, Serratia odorifera was described as a new species of Serratia (4). The habitats of S. odorifera in nature are relatively unknown, although occasionally an isolate has been recovered from plants (3). Rarely has S. odorifera been isolated from clinical specimens, yet alone identified as the cause of disease. This report describes a patient with an invasive infection due to S. odorifera biogroup 1.

A 67-year-old male was admitted to a hospital affiliated with the University of South Florida College of Medicine because of lethargy and fever. His past history was significant, with several hospital admissions because of cirrhosis, hepatorenal syndrome, hepatic encephalopathy, and upper gastrointestinal tract bleeding related to chronic alcohol dependence. On admission, physical examination revealed a thin, chronically ill-appearing, lethargic male with a temperature of 103°F (ca. 39°C), a pulse of 120/min, a respiratory rate of 22/min, and a systolic blood pressure of less than 90 mm Hg. The remainder of the exam was remarkable for spider angiomata, collateral venous circulation over the abdominal wall, and ankle clonus. Laboratory evaluation revealed a leukocyte count of 21,900/mm³, with 15% band forms on peripheral smear. Blood urea nitrogen was 35 mg/dl, creatinine was 2.6 mg/dl, total bilirubin was 5 mg/dl, and alkaline phosphatase was 250 IU/liter. Serum electrolytes were normal. On microscopic examination, the urine showed leukocytes too numerous to count and a large amount of bacteria. A Gram stain of the urine showed gram-negative rods and leukocytes. Chest X-ray and electrocardiogram results were unchanged from prior admissions.

The patient was admitted to the intensive care unit with a presumptive diagnosis of septic shock. Blood and urine cultures were done. The patient was treated with intravenous fluids and a dopamine drip to maintain a systolic blood pressure greater than 90 mm Hg. Treatment with amikacin (300 mg every 8 h) and cefotaxime (1 g intravenously every 8 h) was begun. All blood cultures (four of four sets) and urine cultures grew a gram-negative rod within 24 h. Subsequently, the organism was identified as S. odorifera biogroup 1. By disk diffusion and microdilution susceptibility testing, the isolates were susceptible to amikacin (MIC, 2 µg/ml), gentamicin (MIC, 0.50 µg/ml), cefoxitin (MIC, 4 µg/ml), and cefotaxime (MIC, 2 µg/ml) and were resistant to mezlocillin, piperacillin, ampicillin, carbencillin, tetracycline, chloramphenicol, and cephalothin. The patient improved within 48 h and was transferred from the intensive care unit to the medical floor. Amikacin was discontinued, and cefotaxime (1 g intravenously every 8 h) was continued for a total of 12 days. The patient did well and was transferred to a nursing home.

Six isolates of S. odorifera were recovered on 5% sheep blood agar and produced lactose-fermenting colonies on MacConkey agar. On opening the incubator, a strong poto- tolike odor was evident. The urine and blood isolates proved to be the same. The organism was catalase positive, oxidase negative, and gelatinase positive, and it produced DNase. The organism produced an acid (slant) and gas (but) and no H₂S on a triple sugar iron slant. Biochemical testing was done with the API 20E System (Analytab Products, Plainview, N.Y.). The organism gave positive reactions with beta-galactosidase, L-lysine and L-ornithine decarboxylase, citrate, indole, and Voges-Proskauer. Negative results were seen with L-arginine dihydrolase, H₂S, urea, and tryptophane deaminase. Acid was produced from D-glucose (no gas), D-mannitol, inositol, D-sorbitol, L-rhamnose, sucrose, melibiose, amylidalin, and arabinose.

Serratia marcescens is a well-recognized human pathogen and is the most commonly isolated Serratia species recovered from clinical specimens (2, 3). Other Serratia species rarely cause human infection (2, 3, 5). One such species, S. odorifera, has rarely been isolated from clinical specimens. In a recent review, 52 clinical isolates were studied (2). Two biogroups exist. S. odorifera biogroup 1 is ornithine decarboxylase positive and ferments raffinose and sucrose; biogroup 2 lacks these biochemical reactions (2, 3). To date, 21 isolates of biogroup 1 have been studied, with most strains recovered from the respiratory tract (2, 3). None of the isolates were associated with clinical disease, prompting the researchers to cast doubt on the ability of S. odorifera biogroup 1 to cause human illness. In contrast, 31 isolates of S. odorifera biogroup 2 have been studied, with most isolates recovered from the respiratory tract (2). However, six strains were cultured from spinal fluid and blood, suggesting a more invasive role in human infections for biogroup 2.

The case described in this report represents the first documented human infection due to S. odorifera biogroup 1. The patient was a debilitated host with underlying chronic liver disease secondary to alcohol abuse. Patients with chronic liver disease, particularly when it is related to alcohol abuse, are known to be at an increased risk for a variety of infections, especially those due to gram-negative organisms (1, 6). The patient described herein presented with signs and symptoms compatible with a diagnosis of urosepsis due to gram-negative organisms. S. odorifera biogroup 1 was isolated from both urine and blood cultures. Both isolates had the same antibiotic susceptibility patterns and gave identical results in biochemical testing. The environmental source of S. odorifera in the case described was unknown. Further studies, including reports of other human...
infections, are needed to define the epidemiology of this unusual *Serratia* species.

**LITERATURE CITED**


