Spontaneous Bacterial Peritonitis due to *Ochrobactrum anthropi* : A Case Report

We report a case of spontaneous bacterial peritonitis from *Ochrobactrum anthropi*. *O. anthropi* is recognized as an emerging pathogen in immunocompromised patients. In contrast to most previously described cases, the patient reported here had no indwelling catheter. To our knowledge, no case of *O. anthropi* spontaneous bacterial peritonitis has been reported in the medical literature until now.

**INTRODUCTION**

*Ochrobactrum anthropi*, formerly classified as *Achromobacter* species or Centers for Disease Control groups Vd, is an aerobic, oxidase-producing, non-fermenting, Gram-negative bacillus (1). *O. anthropi* is a ubiquitous organism, which is widely distributed in the environment and water sources including normal saline, antiseptic solutions, dialysis liquids, and swimming pools (2). The organism may be part of the normal flora of the large intestine and has been isolated from various clinical specimens (3). *O. anthropi* is currently considered to be an opportunistic pathogen, and most human diseases reported in the literature are consequences of central venous catheter line infections in severely ill or immunocompromised patients (3, 4). The pathogen usually displays resistance to commonly used broad-spectrum antimicrobial agents, including penicillins and cephalosporins (3).

We report a case of *O. anthropi* spontaneous bacterial peritonitis in a patient without evident impairment of immune function other than liver cirrhosis. A review of the literature, found no case of *O. anthropi* spontaneous bacterial peritonitis up to now.

**CASE REPORT**

A 62-yr-old man was admitted to our hospital with a 1-day history of abdominal pain and fever. He was known to have liver cirrhosis. On physical examination, his blood pressure was 105/68 mmHg, pulse rate was 105 beats per min, and body temperature was 38.2°C. Abdominal examination revealed diffuse tenderness on the whole abdomen, diminished bowel sound, and splenomegaly. Laboratory tests showed hemoglobin concentration of 12.2 g/dL, leukocyte count of 7,580/L, platelet count of 233,000/L, serum creatinine level of 1.36 mg/dL, serum bilirubin level of 5.0 mg/dL, and serum albumin level of 2.5 g/dL. The analysis of peritoneal fluid demonstrated an albumin level of 284 mg/dL and leukocyte count of 350/μL (poly 89%, other 5%). The Child-Pugh score was 13. Empirical treatment with ceftriaxone (2 g every 24 hr) was started with the presumptive diagnosis of spontaneous bacterial peritonitis. Several days later, gram negative bacilli were growing in the cultures of blood and ascites processed by the BACTEC 9240 unit (Becton Dickinson, Sparks, MD, U.S.A.). The patient’s subsequent hospital course was uneventful except for low-grade fever (≤38°C). On the 8th day of admission, his blood pressure was 66/40 mmHg, pulse rate was 94 beats per min, and body temperature was 35.2°C. Laboratory tests showed a hemoglobin concentration of 11.0 g/dL, leukocyte count of 7,580/μL, platelet count of 233,000/μL, serum creatinine level of 1.36 mg/dL, serum bilirubin level of 5.0 mg/dL, and serum albumin level of 2.5 g/dL. The analysis of peritoneal fluid demonstrated an albumin level of 284 mg/dL and leukocyte count of 350/μL (poly 89%, other 5%). The Child-Pugh score was 13. Empirical treatment with ceftriaxone (2 g every 24 hr) was started with the presumptive diagnosis of spontaneous bacterial peritonitis. Several days later, gram negative bacilli were growing in the cultures of blood and ascites processed by the BACTEC 9240 unit (Becton Dickinson, Sparks, MD, U.S.A.). The patient’s subsequent hospital course was uneventful except for low-grade fever (≤38°C). On the 8th day of admission, his blood pressure was 66/40 mmHg, pulse rate was 94 beats per min, and body temperature was 35.2°C. Laboratory tests showed a hemoglobin concentration of 11.0 g/dL, leukocyte count of 7,580/μL, platelet count of 233,000/μL, creatinine level of 2.26 mg/dL, aspartate aminotransferase level of 470 mg/dL, and aspartic leukocyte count of 1,190/μL (poly 46%, other 50%). We tried to perform an esophagogastroduodenoscopy (EGD) because of his black
and tarry stool, but the patient and his family refused to give his consent to EGD. At that time, the gram-negative bacillus was identified as *O. anthropi* by the Gram negative Combo 32 kit (Microscan Workaway-96, Dade Behring, West Sacramento, CA, U.S.A.), and the biochemical profile determined by the API 20 NE system (BioMerieux, Marcy l’Etoile, France) also gave unequivocal identification of *O. anthropi*. The organism yielded positive results in tests for urea, ornithine, and esculin hydrolysis and failed to produce hydrogen sulfide in triple-sugar-iron agar, which is collectively consistent with *O. anthropi*. The isolate was in vitro susceptible to amikacin, gentamicin, tobramycin, imipenem, meropenem, ciprofloxacin, levofloxacin, and trimethoprim but resistant to all other tested (β-lactam antibiotics including ceftriaxone). The antibiotic was changed to imipenem (250 mg every 6 hr, Estimated CrCl=40 mL/min). On the 10th day of admission, the patient died in spite of transfusion and inotropic therapy.

**DISCUSSION**

*O. anthropi* is currently considered as an emerging pathogen in immunocompromised patients, especially in the presence of indwelling medical devices. Cytotoxic chemotherapy, recent transplantation, hematologic and non-hematologic malignancies are common underlying conditions (5, 6). The first case of human infection with *O. anthropi*, a pancreatic abscess, was reported in 1980 (7). Since then, most human diseases reported in the literature were a consequence of indwelling medical devices, such as central venous catheter, drainage tubes, and intraperitoneal catheters (3, 4). The ability of the pathogen to adhere to silicone, similar to that of *S. aureus* and *S. epidermidis*, has been shown by in vitro experiment and may play a role in catheter-associated infection (6, 8, 9). In addition to central catheter-related infections, *O. anthropi* may cause localized pyogenic infections (10), meningitis (11), and osteomyelitis of bone flap following the implantation of contaminated allograft tissue (12), endophthalmitis after vitrectomy and cataract surgery (13, 14), and endocarditis (15, 16). Contaminated fluids were the likely source of infection (2). Five organ transplant recipients developed *O. anthropi* bacteremia after infusion of contaminated antilymphocyte globulin. The pathogen has also been identified as a causative agent of peritonitis in patients undergoing continuous ambulatory peritoneal dialysis (17). The organism is not unusual among infants and toddlers without chronic diseases or other known predisposing factors (3). It has also been recovered from bile, urine, wounds, stool, throat, and vagina.

Non-fermenting, Gram-negative bacilli that are uncommonly isolated may be quite difficult to identify to the species level. *O. anthropi* grows readily on MacConkey agar and is differentiated from *Pseudomonas* spp. and *Flavobacterium* by its absence of pigment production and its peritrichous flagella. A positive oxidase reaction differentiates *O. anthropi* from Acinetobacter and Flavomonas. Further segregation of *O. anthropi* from other oxidase-positive organisms such as *Achromobacter*, *Alcaligenes* and *Agrobacterium* is based on the production of 3-ketolactose and hydrogen sulfide, growth on cetrimide and hydrolysis of esculin and urea. Accurate identification may require comparison of results obtained by using several identification systems and may sometimes also require comparison of those results of conventional tests such as growth on triple-sugar-iron agar (18).

Our patient in this report, who had underlying disease of liver cirrhosis as the underlying disease, was suffering from spontaneous bacterial peritonitis caused by *O. anthropi*. The patient had not received intravenous injections for more than 6 months, and we could not find any other predisposing conditions. We speculate that the organism has originated from the intestinal flora. Enteric bacteria could enter the systemic circulation from the portal vein by passage through the liver or by portosystemic shunts in patients with portal hypertension. Enteric bacteria also may gain access to the peritoneal cavity by directly traversing the intact intestinal wall. This observation highlights that *O. anthropi* should also be considered a significant pathogen in liver cirrhosis patients.

Typically, *O. anthropi* isolates are susceptible to commonly used aminoglycosides, carbapenems, cotrimoxazole, quinolones, and sulfonamides; variably susceptible to rifampicin and tetracyclines; and resistant to beta-lactams (other than carbapenem), chloramphenicol, macrolides, and trimethoprim (1). The strain of the present case showed a matched susceptibility pattern. In *O. anthropi* infection, there is a poor correlation, however, of in vitro susceptibility data with clinical efficacy of antimicrobial therapy. Treatment failure with imipenem has been reported despite in vitro susceptibility of the strains (19).

To our knowledge, this case is the first report of spontaneous bacterial peritonitis due to *O. anthropi*. Despite its high level of resistance to antibiotics, considering that the majority of cases have been described in debilitated host, *O. anthropi* has been thought to have low intrinsic pathogenic power and virulence. However, this case emphasizes that the awareness of the potential role of *O. anthropi* in causing spontaneous bacterial peritonitis is important, given the serious morbidity associated with disseminated *O. anthropi* infections.

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