CAPD Peritonitis Due to *Acinetobacter baumannii* Bacteremia after Coronary By-Pass Surgery

The genus *Acinetobacter* consists of strictly aerobic, pleomorphic gram-negative coccobacillary rods, including *Acinetobacter baumannii*, a water-borne organism that preferentially colonizes in aquatic environments. This bacterium is commonly isolated from irrigating solutions and intravenous solutions in hospital settings. *Acinetobacter* isolates that are recovered from respiratory secretions and urine samples of hospitalized patients represent colonization rather than infection. When infection does occur in relation to *Acinetobacter* isolates, it usually involves organ systems that have a high fluid content (e.g., the respiratory tract, the urinary tract, cerebrospinal fluid, and peritoneal fluid) or it manifests as either nosocomial pneumonia associated with continuous ambulatory peritoneal dialysis (CAPD) or catheter-associated bacteriuria (1). Infections, including peritonitis and catheter-related and tunnel infections, are some of the most important causes of peritoneal dialysis discontinuation (2). Several reports related to CAPD peritonitis caused by the *Acinetobacter* species exist in the literature (3,4), and these reports attribute the infections to bacterial colonization of the CAPD catheter. In this case study, we present an instance where CAPD peritonitis was suggested to be related to *Acinetobacter* bacteremia that occurred following a coronary artery bypass graft operation.

CASE REPORT

The patient, a 68-year-old man with diabetes mellitus type 2, hypertension, and coronary artery disease, and who had been on a CAPD program for 3 months with 4 × 2 L (Physioneal (Baxter Healthcare Corporation, Deerfield, IL, USA): 2× 2.27% glucose; 2× 1.36% glucose), was hospitalized for coronary artery bypass graft surgery. On the first postoperative day, 7.5% Icodextrin (Baxter Healthcare Corporation, Deerfield, IL, USA) (8 hours of dwell time) was added to the treatment. Two days after the operation, the patient, who was in the intensive care unit (ICU), developed a disturbance of consciousness. The patient's vital signs were as follows: pulse rate: 96/minute; blood pressure: 100/60 mmHg; respiration rate: 26/bpm; body temperature: 36.3°C; oxygen saturation: 95% (while 4 L/min of oxygen was applied). The general physical examination of the patient revealed bibasilar crackles in the lungs, trace/+1 bilaterally pretibial edema. The neurological examination was negative and diffusion magnetic resonance imaging (MRI) of the brain revealed increases of multiple intensity and multiple acute infarct areas in the bilateral frontal and parietal lobes. However, this observation could not explain the neurologic disturbance of the present case. Laboratory examinations of blood were as follows: white blood cells (WBC): 11,300/μL; platelets: 124,000/mL; hemoglobin: 10.7 g/dL; creatinine: 5.6 mg/dL; blood urea nitrogen: 87 mg/dL; C-reactive protein: 97 mg/L (reference range: 0 – 5 mg/L). On the first day of admission to the ICU, in the evening, a subfebrile fever (37.6°C) developed, and then 2 subsequent peripheral blood cultures were taken. Empirical moxifloxacin treatment was started. In addition, the peritoneal drain fluid was examined; the color of the fluid was clear and the cell count was 70/μL. On the second day in the ICU, because O_2 saturation began to drop to 65 – 70%, the patient was intubated and ventilated. On the third day in the ICU (postoperative day 5), cloudy effluent occurred in the peritoneal drain bags of the patient. The peritoneal drain fluid cell count was 23,000 WBC/μL and 70% of the WBCs were composed of neutrophils. The peritoneal drain fluid sample was sent for microbiological examination. Gram stain of the sample revealed the presence of gram-negative coccobacilli. Intraperitoneal cefazolin sodium (1 g) and ceftazidime (1 g) were started, empirically. On the fifth day in the ICU, it was reported that multi-drug resistant (MDR) *Acinetobacter baumannii* had been found in both of the blood cultures analyzed by the microbiology laboratory. Due to the

antibiogram report, the antibiotic treatment was shifted to colistin (intravenous) and the treatment plan included a 160-mg dose of that medication every 36 hours. On the sixth day in the ICU, a bacterial culture of the peritoneal drain fluid found that *Acinetobacter baumannii* had been isolated. The bacteria isolated from both the blood culture and the peritoneal drain fluid culture were similarly MDR but only sensitive to colistin. Other bacterial cultures for samples from areas such as the urinary tract and a wound (located on the patient's back) yielded results of "no growth." Upon deterioration of the patient's general condition, his CAPD treatment was ceased and hemodialysis was applied once, for 3 hours. The patient, who also experienced cardiac arrest, died on the seventh day of admission to the ICU although all necessary interventions had been undertaken. To conclude, we thought that the development of CAPD peritonitis in our patient could have resulted from bacteremia.

DISCUSSION

Acinetobacter baumannii have rarely been seen among the causative agents of CAPD-associated peritonitis (5). In general, when this agent is detected in the peritoneal fluid, it results in drop-out or mortality for patients undergoing CAPD (6).

Acinetobacter baumannii frequently causes infections along a spectrum that varies from skin infections and pneumonia to bacteremia and sepsis among immunosuppressed patients, particularly those who are hospitalized in the ICU. Few reports exist in the literature of cases where MDR-*Acinetobacter baumannii* is a causative agent of CAPD peritonitis. Zhang and colleagues (3) reported a series of 7 cases in the most recent (to our knowledge) of these reports. The authors indicated that 3 of these cases dropped out of the study and were changed to hemodialysis; 4 cases continued with the initial treatment. Our case varied from other case reports with respect to the order in which peritonitis resulted from *A. baumannii* bacteremia. Given this, hematogenous spread was suggested. Our report is similar to that of Zhang and colleagues (3); with the exception of colistin, *A. baumannii* was resistant to all antibiotics used according to the standard protocol. Another characteristic of our case is that it is among the first set of cases (to our knowledge) reported as CAPD peritonitis with hematogenous spread in the published literature.

In conclusion, *A. baumannii* peritonitis can hematogenously develop in CAPD patients shown to have *Acinetobacter* bacteremia.

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