Shewanella algae Peritonitis in Patients on Peritoneal Dialysis

Patients with peritonitis present with abdominal pain, diarrhea, fever, and turbid peritoneal dialysis (PD) fluid. *Shewanella algae* peritonitis has not yet been reported in PD patients in the literature. We present the first 2 cases of *Shewanella algae* peritonitis in PD patients. Mupirocin cream is applied on the exit site as prophylactic antibiotic therapy.

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CASE REPORT 1

A 64-year-old man on weekly cycler peritoneal dialysis (PD) presented with abdominal pain and diarrhea preceded by 2 days of fever and lethargy. Empirical intraperitoneal (IP) cloxacillin 250 mg *quater in die* (qid) and ceftazidime 250 mg qid was initiated. Turbid PD fluid showed numerous gram-negative rods, white blood cell (WBC) of 450 cells/mm^3 (majority polymorphs). Culture revealed *Shewanella algae* in aerobic BACTEC blood culture bottle (BD, Franklin Lakes, NJ, USA) and in PD fluid, sensitive to ceftazidime, amikacin, gentamicin, and piperacillin but resistant to imipenem and ciprofloxacin. Intraperitoneal medication was switched to IV ceftazidime, and IV gentamicin was initiated in view of septicemia and elevated blood WBC. Infection resolved completely after 14 days of treatment, without removal of the catheter, and there was no recurrence of peritonitis.

CASE REPORT 2

A 54-year-old man on continuous ambulatory PD (CAPD) for 2 years presented with cloudy PD fluid, abdominal pain preceded by 3 days of reduced appetite, fever, and lethargy. Intraperitoneal cloxacillin 250 mg qid and IP ceftazidime 250 mg qid was initiated. Dialysate WBC was 250 cells/mm^3 (majority polymorphs). Following rapid deterioration, he succumbed within 48 hours of admission. His peritoneal catheter exit site grew *Staphylococcus aureus* and *Corynebacterium sp*. Peritoneal dialysis fluid culture revealed *Shewanella algae*, sensitive to imipenem, meropenem, gentamicin, amikacin, and piperacillin/tazobactam. Blood WBC was 10.5×10^9/L and blood culture revealed no growth.

DISCUSSION

Shewanella spp are hydrogen sulphide-producing motile gram-negative bacilli, first described in 1931(1). Isolated from putrefied butter and water supplies in dairies, this species was initially named *Achromobacter putrefaciens* (1). James Shewan then went on to reclassify this genus as Group IV *Pseudomonas* (2). Eventually in 1985, phylogenetic studies by Mac Donell and Colwell led to the reclassification of this genus under *Vibrionaecea* and a new genus for these organisms, *Shewanella* spp, in honour of James Shewan. Following the discovery of a tetrodotoxin producing *Shewanell*a isolate from red algae, *Shewanella algae* was initially proposed as a new species in 1990 (3). Out of more than 30 known species of *Shewanella*, *S. putrefaciens and S. algae* are the only *Shewanella* spp isolated in humans (4,5). Nitrite-reducing ability and production of mucoid colonies with beta-hemolysis on sheep blood agar distinguishes these 2 species from each other. Unlike *S. putrefaciens*, *S. algae* is unable to produce acid from maltose, sucrose, or L-arabinose (6). Its ability to produce beta-hemolytic substance is the reason *S. algae* has been proposed to be more pathogenic than *S. putrefaciens*.

To the best of our knowledge, there have been 12 reported cases of *S. Algae* infections, mostly involving soft tissue infection. There have been 6 reported cases of CAPD peritonitis caused by *S. putrefaciens* (7–10). *Shewanella* bacteraemia in hemodialysis was reported in 5 cases, 2 caused by *S. algae*, 3 caused by *S. putrefaciens*.

In our hospital, identification of the bacteria involved putting the medium into selective growth in an aerobic environment, and then using an automated microbial identification system to identify the microorganism. Our first patient appeared to have resistance to imepenem according to the antibiotic sensitivity report from our laboratory. Resistance to carbapenem has also been described in 2 other cases elsewhere. This resistance is thought to be due to a carbepenem-hydrolyzing ability mediated by chromosome encoded beta-lactamase gene OXA-55 in a known carbapenem-resistant strain of *S. algae* KB-1. Resistance to the quinolone group of antibiotics appears to be predominantly plasmid-mediated by QnrA determinants in *Shewanella algae* found in nature (11,12). Our second patient lived in one of the largest ex-mining communities, raising suspicion that exposure to mining water and minerals could have been the route of infection. He succumbed within 48 hours of presentation. His strain of *S. algae* may have been more virulent. Furthermore, concomitant infections on board could have compounded the severity of peritonitis that led to his untimely demise. The estimated route of infection in the first case remains unclear as the details were not documented.

CONCLUSION

We encountered 2 extreme outcomes with our 2 cases, 1 person surviving and the other succumbing to infection. Although *Shewanella* peritonitis is rare, its pathogenic potential must not be underestimated. Our first case demonstrated that *Shewanella algae* peritonitis can be treated with antibiotic without removal of the catheter despite the propensity of this organism to form biofilms. The organism may be naturally carbapenem- and quinolone-resistant and either antibiotics should not be the first choice when treating this organism. Our second case highlights the need to raise suspicion of *Shewanella* infection in patients living in environments surrounded by any form of water source.

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