Short Communication

Peritoneal Dialysis-Related Peritonitis Caused by Microbacterium paraoxydans

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SUMMARY: A 54-year-old woman receiving continuous ambulatory peritoneal dialysis was admitted, complaining of diffuse abdominal pain. Peritoneal fluid cell analysis showed that the white blood cell count was 2,990 cells/mm³, with a neutrophil count of 2,510 cells/mm³. The patient was treated empirically with intraperitoneal cefazolin and ceftazidime. After 6 days, Microbacterium species grew on a peritoneal dialysate culture that had been collected on the day of admission. We analyzed the 16S rRNA gene nucleotide sequence and identified the organism as Microbacterium paraoxydans. Based on the results of the antibiotic susceptibility test, the patient was treated with intraperitoneal vancomycin and oral clarithromycin. She recovered uneventfully without interruption of peritoneal dialysis. This is a unique case of peritoneal dialysis-related peritonitis due to M. paraoxydans.

Microbacterium species are Gram-positive rods, and are universally present in animals and environments such as soil and water. The genus Microbacterium, one of the coryneform bacteria, was redefined from the genera Microbacterium and Aureobacterium (1), and comprises 96 species at present (2). Microbacterium occasionally cause opportunistic infection in immunocompromised humans, and Microbacterium paraoxydans, one of the Microbacterium spp., rarely causes human infection. Here, we present a case of peritoneal dialysis (PD)-related peritonitis caused by M. paraoxydans, which was identified as the underlying pathogen using 16S rRNA gene sequencing.

A 54-year-old woman receiving continuous ambulatory peritoneal dialysis (CAPD) for end-stage renal disease (ESRD) was admitted to the emergency department at Chonnam National University Hospital, complaining of diffuse abdominal pain that persisted over 5 h. She had a history of hypertension and type 2 diabetes mellitus (for which she received medication) for over 15 years. She started hemodialysis due to ESRD was admitted to the emergency department at Chonnam National University Hospital, complaining of diffuse abdominal pain that persisted over 5 h. She had a history of hypertension and type 2 diabetes mellitus (for which she received medication) for over 15 years. She started hemodialysis due to ESRD in January 2015. She experienced left middle cerebral artery territory infarction 14 years ago, and 1 episode of PD-related peritonitis 5 months before, which was treated successfully with empirical antibiotics (intraperitoneal cefazolin and ceftazidime). However, the causative organism was not isolated.

The patient’s blood pressure was 110/70 mmHg, her heart rate was 84 beats/min, respiratory rate was 20 breaths/min, and her body temperature was 36.1°C. Initial laboratory tests showed white blood cell (WBC) count of 9,000 cells/mm³ (absolute neutrophil count, 7,300/mm³). C-reactive protein, blood urea nitrogen, and a creatinine levels were 1.92 mg/dL, 39.8 mg/dL, and 5.6 mg/dL, respectively. A 50 mL sample of peritoneal fluid was obtained. We performed a cell count and differential count of the peritoneal fluid. The sample was then centrifuged at 3,000 × g for 15 min, followed by resuspension of the sediment in 5 mL of sterile saline and inoculation on solid culture medium. The results of the peritoneal fluid cell count showed that the WBC count was 2,990 cells/mm³, with a neutrophil count of 2,510 cells/mm³ (84% of total WBC). Consequently, immediately after sampling, the patient was treated empirically with intraperitoneal cefazolin (15 mg/kg/day) and ceftazidime (1 g/day) for the first 11 days. After 6 days of aerobic incubation on blood agar, Microbacterium species (formerly the Centers for Disease Control and Prevention (CDC) coryneform group A-4, A-5 in part) grew on the peritoneal dialysate, which had been collected on the day of admission.

We analyzed 16S rRNA gene sequence to confirm the exact species of the causative organism. Genomic DNA was extracted from the cultivated microorganisms using proteinase K, and PCR for 16S rRNA was performed (3). The 16S rRNA gene was amplified with universal primers (forward, 5'-AGTTTGATCCTGCTGCTG-3'; reverse, 5'-GTATTGCGCGGTCTTCA-3') using Biometra T3000 Thermocycler (Analytik Jena, Germany). It was sequenced using Applied Biosystems 3130xl Genetic analyzer (Applied Biosystems, Foster City, CA, USA). Microbacterium paraoxydans strain W7B_4.2 (GenBank accession no.: KT720177.1) was verified with 99% similarity, which was confirmed using a nucleotide basic local alignment search tool analysis (nucleotide BLAST, <http://blast.ncbi.nlm.nih.gov/>). In vitro susceptibility testing for selected antibiotics (erythromycin, gentamicin, penicillin, and vancomycin) was performed using the E-test method (4). The cultured strain showed susceptibility to all of the antibiotics tested. Minimal inhibitory concentrations (MICs) for each antibiotic were 0.064 μg/mL for erythromycin, 3.0 μg/mL for gentamicin, 1.0 μg/mL for penicillin, and 2.0 μg/mL for vancomycin. Based on the results of the antibiotic susceptibility test, we...
stopped empirical antibiotic administration and started intraperitoneal administration of vancomycin (2 g loading, followed by 1 g every 5 days) and oral administration of clarithromycin (500 mg every 12 h) on day 12. The patient’s abdominal pain improved, and the WBC count of the peritoneal fluid decreased to less than 100 cells/mm³ within 5 days of the start of vancomycin and clarithromycin administration. Both antibiotics were administered for a total of 2 weeks, and the patient recovered uneventfully.

Peritonitis is one of the major complications of PD and is the leading cause of hospitalization, catheter loss, switching to hemodialysis, and considerable morbidity in these patients (5). The most common pathogens of PD-related peritonitis are Gram-positive organisms, in particular coagulase-negative Staphylococcus species. Only one case of PD-related peritonitis caused by *M. paraoxydans* has been reported so far (6). The patient in the previous report (6) worked in agriculture, whereas this patient’s husband was a farmer. Therefore, the 2 patients might have been exposed to similar environmental factors. The patient in the previous report was treated with a combination of intravenous erythromycin and oral trimethoprim/sulfamethoxazole, and PD was suspended for 3 weeks (6). Unlike the previous case, we selected intraperitoneal administration of vancomycin and oral administration of clarithromycin, and PD was continued during the treatment. Furthermore, Miyamoto et al. (6) performed 16S rRNA gene sequencing after completion of treatment, whereas we performed it immediately after the causative agent’s identification.

In our case, *M. paraoxydans* was identified using 16S rRNA gene sequence analysis. Analyzing 16S rRNA gene sequences enables us to identify rarely isolated *Microbacterium* strains and to describe novel species of bacteria. The patient was treated successfully with the correct antibiotics based on the pathogen’s identification. There is no established consensus for the optimal treatment of *Microbacterium* peritonitis. Most (98%) *Microbacterium* spp. are susceptible to vancomycin (7), but susceptibility to antibiotics is different depending on the species. Therefore, susceptibility of each species to antibiotics should be tested. To the best of our knowledge, this is a unique case of PD-related peritonitis due to *M. paraoxydans* that was treated with intraperitoneal administration of vancomycin without interruption of CAPD.

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Conflict of interest None to declare.

REFERENCES