Peritoneal Dialysis-Associated Peritonitis Caused by Dermabacter hominis

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Dermabacter hominis was the cause of a peritoneal dialysis-associated peritonitis. D. hominis was identified by phenotypic criteria and by sequencing the 16S rRNA gene. Clinical cure was achieved with cefuroxime treatment despite the isolate’s reduced susceptibility to this drug (MIC, 12 mg/liter) on in vitro testing. The successful treatment was probably due to the high concentrations attained by intraperitoneal administration of the drug.

CASE REPORT

Our patient was a 67-year-old woman with end-stage renal disease due to hypertensive kidney disease. Since 1994 she had been receiving renal replacement therapy with peritoneal dialysis (PD), which was exclusively performed as continuous ambulatory peritoneal dialysis (CAPD). She was admitted to the hospital at the end of March 2000 with symptoms of peritonitis. Cultures of the dialysate yielded coagulase-negative staphylococci. The infection was successfully treated with cloxacillin given intraperitoneally. In the middle of April, the cuff of the dialysis catheter (single-cuff Quinton-Tenckhoff catheter, 41 cm long) slid out. The catheter was therefore exchanged. Resumption of dialysis after this was uncomplicated. Almost 3 weeks later, while still in the hospital awaiting transfer to a nursing home, she again became ill with abdominal pain and a hazy dialysate. The PD effluent contained \(0.54 \times 10^9\) leukocytes per liter. We routinely culture peritoneal fluid from PD patients with signs of peritonitis in Vital aerobic and anaerobic blood culture bottles (bioMerieux SA, La Balmes-des-Grottes, France). Two sets of samples were obtained on day 1, and one set of samples was obtained on each of the following 2 days. A gram-positive coryneform bacterium grew in all bottles from days 1 and 2 and in the aerobic bottle from day 3. Growth occurred after 18 to 24 h in the aerobic bottles and after approximately 4 days in the anaerobic bottles. A swab from the catheter exit site yielded no growth.

From day 1 the patient was empirically treated with a combination of intraperitoneal cloxacillin (125 mg/liter of dialysis fluid) and netilmicin (6.25 mg/liter) in 2-liter bags in each of the four daily instillations. New samples taken on day 6 yielded growth of the same coryneform bacterium, although the dialysate had cleared and the patient was improving. Because of the results of disk diffusion susceptibility testing, treatment was changed to cefuroxime (125 mg/liter of dialysis fluid) and continued for a further 14 days, achieving clinical cure. Cultures from days 8 and 13 were sterile.

Microbiology. The peritoneal fluid inoculated into blood culture bottles yielded bacteria that grew on blood agar and chocolate agar (but best on blood agar) as 0.5- to 1-mm-diameter white colonies after 18 h. The colonies were white, convex, slightly viscous, and catalase positive and had a sweet, pungent odor. As judged from the blood culture bottles, the organism was nonmotile and gram positive and had coryneform morphology without branching. The isolate hydrolyzed esculin and was nitrate and xylose negative. The bacterium was identified by the API Coryne system (bioMerieux SA) as Dermabacter hominis (code 4570365). Antibiotic susceptibility testing was initially performed with Neo-Sensitabs tablets (A/S Rosco, Taastrup, Denmark) on Mueller-Hinton agar with 5% defibrinated horse blood. The isolate was resistant to chloramphenicol, clindamycin, erythromycin, netilmicin, and penicillin and was intermediately resistant to ampicillin. The isolate was susceptible to cefalothin, cefuroxime, doxycycline, trimethoprim-sulfamethoxazole, and vancomycin by disk diffusion testing. The strain did not produce \(\beta\)-lactamase (nitrocefin test). MICs were determined with E-test strips (AB Biodisk, Solna, Sweden) on Mueller-Hinton agar with 5% defibrinated horse blood (Table 1). A discrepant result was noted for cefuroxime by E-test (MIC, 12 mg/liter; intermediately susceptible), compared to the result obtained by the disk diffusion method (fully susceptible). The 16S rRNA gene was amplified by PCR, and the amplicon (approximately 1,500 bp) was purified from agarose gel by using a QIAquick Gel Extraction Kit (Qiagen, Hilden, Germany). Analysis of the nucleotide sequence was performed using a MicroSeq Full Gene 16S rDNA Bacterial Sequencing Kit (Applied Biosystems, Foster City, Calif.) and an automated DNA sequencer (ABI PRISM 310 Sequencer; Applied Biosystems). The resulting 1,468-bp sequence was compared to sequences available in databases using BLAST (1). More than 99% similarity was found to two D. hominis and two Dermabacter spp. published sequences.

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PD has become the dialysis modality of choice for an increasing number of patients. However, peritonitis is the major source of morbidity and treatment dropout in this form of renal replacement therapy, resulting in only a small number of long-term PD patients. A vast number of different species of microorganisms have been identified as causes of PD-associated peritonitis, and most of the isolates are originally part of the normal skin flora.

*Dermabacter hominis* was first described in 1988 as a cutaneous coryneform bacterium (9). It was later found to be similar to strains that have been present in the special reference laboratory collection of the Centers for Disease Control and Prevention since the 1970s and provisionally grouped as fermentative coryneform group 3 and 5 organisms. These two groups of isolates are identical apart from the fact that members of group 3 are fermenters of xylose while members of group 5 are not (3, 6, 8).

Chromatographically, *D. hominis* resembles *Brevibacterium* spp. in its lack of mycolic acid and in the fact that it has a cell wall based on *meso*-diaminopimelic acid, but the phylogenetic distance is large (4, 6). *D. hominis* is a recently established species, and its recognition as an opportunistic human pathogen is even newer. Very few case reports of documented infections caused by *D. hominis* have been published (2, 7). A search in the database of 16S rRNA gene sequences of the National Center for Biotechnology Information for *Dermabacter* sp. and *D. hominis* revealed six entries. In our CAPD patient, *D. hominis* caused peritonitis as a nosocomial infection. PD-associated peritonitis is defined by three criteria: abdominal pain, leukocyte counts above 0.1 × 10⁹/liter (with 50% or more of the leukocytes being neutrophils), and demonstration of bacteria in the peritoneal effluent by Gram staining or culture. Two of these three criteria are sufficient for the diagnosis (10). In the present case of peritonitis, clinical cure with ceftoxime was achieved despite the relatively high MIC of this antibiotic. This was probably due to the very high local antibiotic concentrations employed by intraperitoneal instillation. The present *D. hominis* isolate was resistant or showed reduced susceptibility to several antimicrobial agents, including penicillin, ampicillin, and cephalosporins. Apart from cloxacillin and netilmicin, the patient had received no other antibiotic treatment prior to the episode of peritonitis in question. The MICs of β-lactam antibiotics tested are higher for this isolate than has been reported previously by others (5). Chloramphenicol, ciprofloxacin, clindamycin, erythromycin, and aminoglycosides other than amikacin have shown reduced activity against most *D. hominis* isolates (2, 5, 6). Susceptibility testing of clinically important *D. hominis* isolates is therefore highly warranted.

**Nucleotide sequence accession number.** The 16S rRNA gene nucleotide sequence of the current isolate has been given GenBank accession number AF343728.

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**REFERENCES**


