Peritoneal Dialysis Peritonitis Caused by *Enterococcus avium*

Editor:

Enterococcus species are normal human bowel flora, but they can also be major pathogens causing community-acquired urinary tract infections, bacteremia, and antibiotic-resistant nosocomial infections (1). Among its kindred, *E. faecalis* and *E. faecium* account for more than 90% of infections; other species (*E. casseliflavus*, *E. gallinarum*, *E. flavescens*) constitute fewer than 5% of cases (2). *E. avium*, a low-virulence pathogen in humans, has previously been reported in bacteremia (3), endocarditis (4), meningitis (5), and intra-abdominal infections (6), but never in peritoneal dialysis (PD) peritonitis. Here, we reported a case of a PD patient developing *E. avium*-related peritonitis.

CASE DESCRIPTION

A 79-year-old woman with hypertension, duodenal ulcer, Parkinsonism, and hepatitis B had end-stage renal disease caused by chronic glomerulonephritis and had been on maintenance PD for more than 2 years. She visited the emergency department, reporting 1 day of fever up to 38.1°C. During the preceding 2 years, she had experienced 2 episodes of polymicrobial PD peritonitis caused by *Escherichia coli*, *Klebsiella pneumoniae*, and *Enterococcus* species (with an episode of bacteremia during the second peritonitis). Repeated gastrointestinal investigations yielded no anatomic lesions except for healed duodenal ulcers. At the time of presentation, she was receiving two daily 2-L bags with 2.5% dextrose and two daily 2-L bags with 1.5% dextrose, with fair clearance.

Besides the fever for 1 day, the patient also reported general weakness and diffuse abdominal pain, accompanied by turbid effluent. At the emergency department, a physical examination disclosed diffuse abdominal rebounding with tenderness. A hemogram revealed leukocytosis (15 510/μL, with 87% neutrophils), and an effluent analysis showed a leukocyte count of 1900/μL with 88% neutrophils. Empiric antibiotics (intraperitoneal cefazolin and ceftazidime) were administered.

One day later, a preliminary effluent culture reported gram-positive cocci, favoring *Streptococcus viridans*. Ceftazidime was discontinued, and cefazolin was replaced with ampicillin. The fever and abdominal pain soon subsided, with the effluent turning clear. The final effluent culture reported *E. avium* susceptible

to penicillin, vancomycin, teicoplanin, gentamicin, and ampicillin; intermediately susceptible to ciprofloxacin; and resistant to tetracycline. Blood cultures were negative. A follow-up effluent analysis 3 days later showed minimal leukocyte counts. The patient completed 2 weeks of intraperitoneal antibiotics without recurrence.

DISCUSSION

E. avium, formerly called group Q *Streptococcus* because of the presence of group Q antigen, was first identified in 1955 from human feces (3). It was initially placed in the *Streptococcus* genus as *S. avium* (so named because most isolations came from chicken feces). Subsequent studies revealed its prevalence in feces of mammals such as dogs, pigs, and humans at about 20% (7). Most of the biochemical features of *S. avium* are akin to those of *Enterococcus* species, and so it was relocated to the genus *Enterococcus* during the 1980s (8).

Presumptively with low virulence, *E. avium* has been implicated in human infections, most commonly intra-abdominal infections and endocarditis (3–6). However, few clinically important infections had been reported to date, and only two case series have described the clinical features of *E. avium* infection. Patel *et al.* (3) first reported 9 cases of *E. avium* bacteremia in a single center. More than half the patients had significant gastrointestinal diseases (liver abscess or cholangitis, among others), and most cases were polymicrobial. A larger series of *E. avium* bacteremia reported by Na and colleagues (9) yielded similar findings, with 50.9% patients having biliary tract infection and 24.5% having intra-abdominal infection; 67.9% of the episodes were polymicrobial.

Our patient had a gastrointestinal disease (duodenal ulcer, though healed), and in the preceding 2 years, she had developed 2 episodes of polymicrobial peritonitis (*E. coli*, *K. pneumoniae*, *Enterococcus* species) and 1 episode of polymicrobial bacteremia during the second PD peritonitis. Review of her history revealed that she did not keep pets and that she had not been recently exposed to animal feces. Those findings are compatible with the existing literature, and we believe that her *E. avium* might have originated from pre-existing bowel flora rather than from contamination because of a break in sterility. That clinical scenario suggests that her previously unidentified *Enterococcus* species might also have been *E. avium*.

The antibiogram for *E. avium* is typified by susceptibility to penicillin, ampicillin, gentamicin, clindamycin, and vancomycin (3,9,10). This feature is useful for

differentiating between *E. avium* and other common enterococci (*E. faecalis*, *E. faecium*), because the latter organisms are commonly resistant to clindamycin and gentamicin (10). In our patient, the *E. avium* was found to be susceptible to gentamicin and penicillin, compatible with previous reports. Administering intraperitoneal ampicillin is sufficient in this scenario to eliminate *E. avium* peritonitis. In addition, *E. avium* resistance to vancomycin has not been reported to date, but vancomycin resistance in *E. faecalis* and *E. faecium* has been reported worldwide (9,10). These hints could assist in separating the various enterococcal species, because prompt institution of the proper antibiotics in PD peritonitis can not only reduce damage to the peritoneum, but also potentially the rate of antibiotic resistance.

DISCLOSURES

The authors declare that they have no competing interests.

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