Citrobacter youngae and *Pantoea agglomerans* Peritonitis in a Peritoneal Dialysis Patient

Editor:

A 58-year-old man with diabetic nephropathy on continuous ambulatory peritoneal dialysis (PD) for 8 years was admitted because of diarrhea, diffuse abdominal pain, and turbid dialysate for 1 day. He had no fever, chills, nausea, or vomiting.

Physical examination showed a blood pressure of 96/62 mmHg, pulse of 76 bpm, rebounding tenderness of the abdomen, and a clean exit site. The white blood cell counts of peripheral venous blood and effluent were 5100/μL and 2190/μL respectively. History showed that, 3 months earlier, this man had had chronic methicillin-resistant *Staphylococcus aureus* osteomyelitis of the right toe, for which he had received sodium fusidate 250 mg three times daily and sulfamethoxazole–trimethoprim 800 mg and 160 mg twice daily thereafter.

At the current presentation, the patient was admitted and received intraperitoneal ceftazidime and vancomycin empirically. On the 4th day after admission, the effluent culture revealed *Citrobacter youngae* and *Pantoea agglomerans*. A sensitivity test showed that both pathogens were sensitive to levofloxacin and aminoglycosides, but resistant to sulfamethoxazole–trimethoprim and all cephalosporins except cefepime. We changed the patient's antibiotics to intravenous levofloxacin, and his effluent white blood cell count improved 2 days later (to 97/μL). Because of the mixed infection with enteric pathogens, we arranged abdominal computed tomography imaging, which showed no active intra-abdominal

lesions. The patient completed treatment after 2 weeks of intravenous levofloxacin, followed by 1 more week of oral levofloxacin.

DISCUSSION

C. youngae and *P. agglomerans* belong to the gram-negative Enterobacteriaceae and are identified in human feces. *Citrobacter* was initially classified as genomospecies 5 of the 11 genomospecies in the *C. freundii* complex, later regrouped in 1993 on the basis of DNA relatedness (1). *C. freundii* and *C. koseri* are the two most common clinical isolates in *Citrobacter* infections (2,3). The other 9 *Citrobacter* species, including *C. youngae,* are rarely a cause of infections; overall, they accounted for only 5% of all *Citrobacter* infections in a 12-year survey after the regrouping (2). These non-*koseri* and non-*freundii Citrobacter* species can cause intra-abdominal infections in immunosuppressed individuals (4).

Gursu *et al.* recently reviewed the literature concerning peritonitis caused by *Citrobacter* species in PD patients, but the results were very limited because the microbiology findings and clinical outcomes were not detailed in the publications reviewed (5). Only a few cases of non-*koseri* and non-*freundii Citrobacter* peritonitis were reported, and they were successfully treated without catheter removal (6,7). To the best of our knowledge, our case is the first reported of *C. youngae* peritonitis.

Consensus on the treatment of *Citrobacter* peritonitis in PD patients is still lacking because of a limited literature. According to the recommendations by the International Society for Peritoneal Dialysis for PD-related infections, organisms such as *Serratia, Pseudomonas, Providencia, Citrobacter,* and *Enterobacter* are reported to be important causes of relapse and are more often associated with catheter loss and death (8). Moreover, Gursu *et al.* suggested treating *Citrobacter* peritonitis individually by observing the patient's condition and not hesitating to remove the catheter if the patient's clinical condition deteriorates (5). In our case of a patient with diabetes, both pathogens were sensitive to levofloxacin, but resistant to all cephalosporins except cefepime. Our patient had an excellent response to levofloxacin without catheter removal, and he had no further peritonitis episodes during 1 year of follow-up.

CONCLUSIONS

C. youngae and *P. agglomerans* are both rare enteric pathogens that can cause PD peritonitis. Our case is the first reported *C. youngae* peritonitis, and it was successfully treated with levofloxacin without catheter removal. *C. youngae* might cause PD peritonitis in diabetic patients and should be considered in the differential diagnosis.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

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REFERENCES

1. Janda JM, Abbott SL, Cheung WK, Hanson DF. Biochemical identification of citrobacteria in the clinical laboratory. *J Clin Microbiol* 1994; 32:1850–4.
2. Samonis G, Karageorgopoulos DE, Kofteridis DP, Matthaiou DK, Sidiropoulou V, Maraki S, *et al. Citrobacter* infections in a general hospital: characteristics and outcomes. *Eur J Clin Microbiol Infect Dis* 2009; 28:61–8.
3. Mohanty S, Singhal R, Sood S, Dhawan B, Kapil A, Das BK. *Citrobacter* infections in a tertiary care hospital in Northern India. *J Infect* 2007; 54:58–64.
4. Lai CC, Tan CK, Lin SH, Liu WL, Liao CH, Huang YT, *et al.* Bacteraemia caused by non-*freundii,* non-*koseri Citrobacter* species in Taiwan. *J Hosp Infect* 2010; 76:332–5.
5. Gursu M, Aydin Z, Pehlivanoglu F, Ozturk S, Karadag S, Uzun S, *et al. Citrobacter* peritonitis: two cases and review of the literature. *Perit Dial Int* 2011; 31:409–11.
6. Carlini A, Mattei R, Mazzotta L, Lucarotti I, Pioli R, Bartelloni A, *et al. Citrobacter braakii,* an unusual organism as cause of acute peritonitis in PD patients. *Perit Dial Int* 2005; 25:405–6.
7. Wong MY, Lau SK, Tang SC, Curreem SO, Woo PC, Yuen KY. First report of peritoneal dialysis–related peritonitis caused by *Citrobacter amalonaticus*. *Perit Dial Int* 2012; 32:224–5.
8. Li PK, Szeto CC, Piraino B, Bernardini J, Figueiredo AE, Gupta A, *et al.* Peritoneal dialysis–related infections recommendations: 2010 update. *Perit Dial Int* 2010; 30:393–423. [Erratum in: *Perit Dial Int* 2011; 31:512]

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