Research Paper

**Citrobacter** Peritoneal Dialysis Peritonitis: Rare Occurrence with Poor Outcomes

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**Abstract**

**Introduction:** Non-Pseudomonas gram-negative bacteria are responsible for an increasing proportion of cases of peritoneal dialysis (PD)-related peritonitis. The role of *Citrobacter* species in the etiology of PD-related peritonitis is often underestimated. In the present study, we aimed to describe the clinical features, laboratory findings, and short- and long-term outcomes in PD-related peritonitis caused by *Citrobacter*.

**Methods:** A retrospective review of all episodes of PD-related peritonitis caused by *Citrobacter* from a single center between 1990 and 2010 was performed. Clinical features, microbiological data, and outcomes of these episodes were analyzed.

**Results:** *Citrobacter* species was responsible for 11 PD-related episodes (1.8% of all peritonitis episodes) in 8 patients. *Citrobacter freundii* was the most common etiologic species (73%), and mixed growth was found in the other 3 episodes (27%). Approximately half (46%) of the episodes were associated with constipation and/or diarrhea. Of the *Citrobacter* isolates from all episodes, 54% were resistant to cefazolin, and only 18% were susceptible to cefmetazole. All isolates were susceptible to ceftazidime, cefepime, carbapenem, and aminoglycosides. More than half of the patients (54%) were hospitalized for index peritonitis, and 27% of the episodes involved a change in antibiotic medication. One patient had relapsing peritonitis caused by *C. koseri* (9%). The mortality rate of PD-related peritonitis caused by *Citrobacter* was 18%, and 89% of surviving patients developed technique failure requiring a modality switch after an average of 12 months of follow-up (range 1.2–31.2 months).

**Conclusion:** PD-related peritonitis caused by *Citrobacter* is associated with poor outcomes, including high rates of antibiotic resistance, a high mortality rate, and a high rate of technique failure among survivors during the follow-up period.

Key words: *Citrobacter*, end-stage renal disease, gram-negative bacteria, peritoneal dialysis, peritonitis.

**Introduction**

Peritonitis is the major infectious complication observed in patients undergoing peritoneal dialysis (PD), accounting for nearly one-fourth of hospitalizations and cases of technique failure and catheter loss [1-3]. Gram-negative bacteria (GNB) are responsible for 15–30% of peritonitis cases. This percentage is
gradually increasing, as the number of cases caused by gram-positive bacteria continues to decline, due to the improvement in exchange systems and the use of topical anti-staphylococcal medications [4-7]. Peritonitis episodes caused by GNB are more often associated with adverse outcomes than those caused by gram-positive bacteria [8, 9].

GNB consist primarily of members of Enterobacteriaceae and other non-fermentative GNB [4, 10]. By virtue of their ability to form biofilms, these GNB are potentially less susceptible to antibiotics effective in vivo [11]. Recently, the term, “SPICE,” has been proposed as an acronym for a group of several GNB—including Serratia, Pseudomonas, Indol-positive organisms (such as Proteus and Acinetobacter), Citrobacter, and Enterobacter—recognized for their increased potential to induce treatment failure and cause relapsing peritonitis [12, 13]. These pathogens possess inducible chromosomally mediated beta-lactamase, which increases their probability of developing in vivo resistance to administered penicillin or cephalosporins [14]. Consequently, physicians should be alert to the potential presence of these organisms when prescribing appropriate antibiotics for PD-related peritonitis.

Among the SPICE pathogens, Citrobacter species have a low incidence of pathogenicity. Data from the ANZDATA registry show that Citrobacter species are responsible for only 1.7% of all non-Pseudomonas PD-related peritonitis episodes, which is significantly fewer than those caused by Enterobacter (8.8%), Serratia (8.2%), and other Indol-positive GNB (8.1%) [7]. Szeto and colleagues also reported that Citrobacter species account for only 4.8% of all Enterobacteriaceae peritonitis cases, which is fewer than those caused by Enterobacter (5.8%) and Serratia (8.6%) [9]. Previous articles describing PD-related peritonitis caused by Citrobacter have all been case reports, and have been limited both in patient number (fewer than 3) and in available laboratory and clinical data, such as antibiograms [15-18]. In the present study, we aimed to report our experience with PD-related peritonitis caused by Citrobacter, and also describe the clinical features and course, microbiological data, and short- and long-term outcomes of these cases.

Materials and Methods

Ethical considerations

The ethics committee of the NTUH approved the current study (NO. 201212165RINC). The local institutional review board did not mandate patient consent, since no interventions were made and patient privacy was not breached.

Study design, setting, and clinical data collection

This was a retrospective analysis of a prospectively assembled cohort of the National Taiwan University Hospital (NTUH) PD program [19]. Only conventional Tenckhoff catheters (double-cuffed and straight) were utilized in our institute, with exit site orientated downward. Also, all the PD catheters were inserted by surgeons with laparoscopic approach, without ultrasound guidance. In our PD registry, all patients with end-stage renal disease (ESRD) initiated on PD or initially on hemodialysis but later switched to PD in our institute were identified and entered into our PD registry. Only patients under maintenance PD for more than 3 months were eligible in the current study. We identified all patients with episodes of culture-confirmed Citrobacter species-associated peritonitis between 1990 and 2010. Peritonitis was diagnosed according to the presence of symptomatology and cloudy effluent with leukocyte count >100/µL since 1985, but the diagnostic criteria were later expanded to include neutrophils >50% of effluent leukocytes to better accommodate international guidelines after 1990 [20].

We reviewed all PD patients’ demographic profiles, which included age and gender; their comorbidities, such as diabetes mellitus (DM), congestive heart failure (CHF), liver cirrhosis, and history of autoimmune diseases; and the causes of their primary renal disease. The patients’ regular medications, including steroids and immunosuppressants, were recorded. Antibiotics use within 1 month of the peritonitis episode was also recorded.

For each episode of PD-related peritonitis caused by Citrobacter, we documented the PD vintage and modality (continuous ambulatory peritoneal dialysis [CAPD] or automated peritoneal dialysis [APD]); the patient’s initial symptoms; pathogens identified; and antibiograms. In addition, laboratory data were obtained. The presumed etiology of peritonitis included break in sterility during the exchange procedure, constipation and/or diarrhea associated (potentially gastrointestinal [GI] flora transmural migration) [21-23], major intra-abdominal events (diverticulitis or perforated viscus), and undetermined. Recommendations for empirical antibiotic regimen for PD peritonitis varied with time. Vancomycin/aminoglycoside were initially favored in 1980s for better coverage of resistant pathogens, but subsequently replaced by first-generation cephalosporin/aminoglycoside or third-generation cephalosporin during late 1990s for fear of vancomycin-resistance emergence and adverse effects of aminoglycoside on residual renal function [20, 24]. In our PD program, empirical antibiotics for patients with peritonitis included intraperitoneal...
cefazolin/aminoglycoside (before 1998) or cefazolin/ceftazidime (after 1998), according to International Society for Peritoneal Dialysis (ISPD) recommendations, unless otherwise indicated [12, 25, 26].

**Outcome variables**

The outcome measures examined included short- and long-term components. Short-term outcomes of index peritonitis consisted of primary response, antibiotic switch (secondary response), relapse peritonitis, repeat peritonitis, Tenckhoff catheter removal, temporary or permanent transfer to hemodialysis, and patient death. The primary response was defined as symptomatic improvement of the patient with effluent leukocyte count <100/µL within 3 days of receiving first-line antibiotic treatment. The secondary response was defined as a response to the second-line antibiotic treatment when a clear effluent was not noted following treatment with first-line antibiotics [12, 25, 26]. Repeat peritonitis was defined as peritonitis recurring after 4 weeks of the previous episode involving the same pathogen, whereas relapse peritonitis was defined as peritonitis occurring within 4 weeks after treatment of the previous episode involving the same pathogen [12, 25, 26]. Peritonitis-related death was considered if the patient’s death occurred within 1 month of the peritonitis episode and was attributable to peritonitis.

In addition, we followed each patient until end of 2012. Long-term outcomes of index peritonitis included technique failure, with a modality switch to hemodialysis.

**Results**

**Clinical features of PD-related peritonitis caused by Citrobacter**

During the study period, a total of 328 patients developed PD-related peritonitis over 35,211 patient-months, with an overall peritonitis incidence of 1 per 56 patient-months. Among the 627 episodes of PD-related peritonitis, 8 patients developed 11 episodes caused by *Citrobacter* species (1.8% of all episodes). One patient developed 3 separate episodes within 3 years, another developed 2 episodes within 3 years, and the remaining patients each developed 1 episode. All *Citrobacter* PD peritonitis episodes occurred when the patients were using Dianeal 1.5% or 2.5% dialysate.

The demographic profiles and clinical characteristics of patients developing PD-related peritonitis caused by *Citrobacter* and other members of Enterobacteriaceae are listed in Table 1. The mean age of patients with *Citrobacter* peritonitis was 53 years, significantly younger than ones with peritonitis from other Enterobacteriaceae; 63% of the patients were male. The mean PD duration of patients with *Citrobacter* peritonitis was 36 months (range, 3–71). Most *Citrobacter* peritonitis patients had no comorbidities, except for CHF (25%). The most common etiology that resulted in the requirement for dialysis in *Citrobacter* peritonitis patients included chronic glomerulonephritis (75%), followed by DM (13%) and hypertension (13%).

**Table 1. Baseline features of patients with *Citrobacter* PD-associated peritonitis.**

| Characteristics | Citrobacter (n=8) | non-Citrobacter Enterobacteriaceae members (n=161) | P |
|------------------|-------------------|-----------------------------------------------|----|
| **Age (years)** | 53 (25 – 74) | 60 (22 – 89) | 0.021 |
| **Men (%)** | 5 (65) | 61 (58) | 0.259 |
| **Modality (CAPD) (%)** | 4 (50) | 100 (62) | 0.978 |
| **Vintage (months)** | 36 (5 – 71) | 37 (3 – 127) | 0.765 |
| **Comorbidities** | | | |
| DM | 1 (13) | 39 (24) | 0.351 |
| CHF | 2 (25) | 14 (9) | 0.115 |
| Liver cirrhosis | 1 (13) | 10 (6) | 0.452 |
| **Cause of primary renal diseases** | | | 0.102 |
| DM | 1 (13) | 31 (19) | |
| CGN | 6 (75) | 74 (46) | |
| Hypertension | 1 (13) | 31 (19) | |
| PCKD | 0 (0) | 8 (5) | |
| Unknown | 0 (0) | 917 (11) | |
| **Laboratory data** | | | |
| Hemoglobin (g/dL) | 10.0 (5.7 – 13.1) | 10.0 (6.2 – 14.9) | 0.074 |
| Albumin (mg/dL) | 3.9 (3.2 – 4.6) | 3.8 (2.6 – 4.9) | 0.696 |
| Creatinine (mg/dL) | 10.7 (5.0 – 13.7) | 10.0 (4.6 – 17.9) | 0.582 |
| Total cholesterol (mg/dL) | 233 (161 – 299) | 200.3 (90 – 329) | 0.518 |

Continuous variables are expressed in mean (ranges), while categorical variables are expressed in number (percentage). Abbreviations: APD, ambulatory peritoneal dialysis; CGN, chronic glomerulonephritis; CHF, congestive heart failure; DM, diabetes mellitus; PCKD, polycystic kidney disease.

None of the patients had undergone intra-abdominal surgery or endoscopy before the development of index peritonitis. In addition, none of the patients had taken antibiotics in the month prior to developing index peritonitis, and none of the patients had been prescribed any steroid or immunosuppressant medications. The presenting symptoms of PD-related peritonitis caused by *Citrobacter* were abdominal pain (100%), followed by fever (27%), nausea/vomiting (18%), and diarrhea (9%). All patients exhibited a turbid dialysate. The mean effluent leukocyte counts were 9200/µL (range, 500–46,500/µL), with 92% neutrophils. The initial blood leukocyte levels were 974/µL (range, 4430–17,560/µL), with 85% neutrophils. The most common species from the effluent was *Citrobacter freundii* (73%), followed by *C. koseri* (27%); the cultures...
from 3 episodes (27%) showed polymicrobial growth (all included C. freundii). Viridans Streptococci, Enterobacter aerogenes, and Klebsiella oxytoca were each responsible for 1 episode of polymicrobial growth. None of the patients had concomitant bacteremia or exit-site infections.

The most common cause of PD-related peritonitis caused by Citrobacter was constipation and/or diarrhea associated (46%), followed by break in exchange sterility (9%). Structural GI lesions were not identified in most of the patients, after upper/lower GI endoscopic examinations. No definite source could be identified in the other episodes of PD-related peritonitis caused by Citrobacter (46%).

The antibiotic susceptibilities of the Citrobacter species are shown in Table 2. Approximately 64% of Citrobacter species were resistant to ampicillin, followed by cefazolin (55%) and ampicillin/sublactam (46%). The rate of resistance to cefmetazole was 27%, with another 27% showing intermediate resistance to cefmetazole. None of the Citrobacter isolates showed resistance to ceftaxime, cefazidime, cefepime, carbapenem, aminoglycosides, piperacillin/tazobactam, or fluoroquinolones. With regard to the first-line antibiotic treatment, intraperitoneal cefazolin/ceftazidime was initially used in 7 episodes (64%) and intraperitoneal cefazolin/aminoglycosides was used in 3 episodes (27%; all before 1998). One patient (9%) was initially treated with intravenous vancomycin/ciprofloxacin for treatment of infection by potential nosocomial pathogens. After culture results and antibiograms available, we discontinued the antibiotic to which the pathogens were resistant or the antibiotic that was of later generation (ceftazidime) if cefazolin/ceftazidime were both effective.

A total of 6 patients (54%) required hospitalization, with a mean hospital duration of 9 days (range, 6–14 days). Table 3 lists the clinical outcomes of patients with PD-related peritonitis caused by Citrobacter. A primary response was achieved in 6 episodes (54%), and after switching antibiotics, a secondary response was noted in another 2 episodes (18%). The average time to antibiotic switch was 6 days (range, 3–7 days). Those responding to antibiotics were maintained on susceptible antibiotics for an average of 14 days (range, 11–21 days). Two patients (18%) with PD-related peritonitis caused by Citrobacter died within 2 weeks of diagnosis, both due to sepsis. One of them had no response to antibiotics at all, while the other had secondary response to antibiotics with decreasing dialysate turbidity and effluent leukocyte counts. The other patients continued PD throughout the disease period, although 1 patient (9%) later developed relapsing peritonitis. The patient with relapsing peritonitis subsequently still had primary response to cefazolin, but later developed technique failure with modality switch to hemodialysis 2.5 months later. The patients who recovered from PD-related peritonitis caused by Citrobacter had poor long-term outcomes. Approximately 89% of these patients had technique failure, with a subsequent change in dialysis modality within 1 year (Table 3). Five of the 8 patients had subsequent technique failure due to refractory peritonitis; the other 2 patients died due to sepsis, and the remaining patient dropped out due to ultrafiltration failure. Of the 5 patients with late refractory peritonitis related technique failure, two patients were caused by E. coli, one was caused by Enterobacter aerogenes, while the other two were caused by refractory Citrobacter peritonitis. The two patients died later were due to healthcare-associated pneumonia (culture negative) and intra-abdominal infection (E. coli bacteremia) respectively.

Table 2. Antibiotics susceptibility of Citrobacter species isolate from effluent.

| Antibiotics             | Susceptible (%) | Intermediate (%) | Resistant (%) | Not tested (%) |
|-------------------------|-----------------|------------------|--------------|---------------|
|                         | Citrobacter     | Other Enterobacteriaceae | Citrobacter | Other Enterobacteriaceae | Citrobacter | Other Enterobacteriaceae |
| Ampicillin              | 0               | 23               | 9            | 5             | 64           | 70          | 27           | 2             |
| Ampicillin/sublactam    | 18              | 71               | 9            | 12            | 46           | 15          | 27           | 2             |
| Piperacillin/tazobactam| 73              | 96               | 0            | 1             | 0            | 0           | 27           | 3             |
| Cefazolin               | 18              | 82               | 0            | 1             | 55           | 17          | 27           | 0             |
| Cefmetazole             | 18              | 89               | 27           | 2             | 27           | 9           | 27           | 0             |
| Cefotaxime              | 73              | 95               | 0            | 2             | 0            | 3           | 27           | 0             |
| Cefazidime              | 73              | 74               | 0            | 2             | 0            | 4           | 27           | 20            |
| Cefepime                | 73              | 100              | 0            | 0             | 0            | 0           | 27           | 0             |
| Imipenem                | 73              | 98               | 0            | 1             | 0            | 1           | 27           | 0             |
| Gentamicin              | 73              | 87               | 0            | 0             | 0            | 13          | 27           | 0             |
| Amikacin                | 73              | 85               | 0            | 0             | 0            | 0           | 27           | 15            |
| Ciprofloxacin           | 73              | 90               | 0            | 0             | 0            | 10          | 27           | 0             |
Discussion

In this study of cases of PD-related peritonitis caused by *Citrobacter*, *C. freundii* was the most common *Citrobacter* species identified from the effluent, and constipation and/or diarrhea associated origin was the most common source of infection. Most *Citrobacter* isolates were resistant to first- and second-generation cephalosporins, as well as to ampicillin/sulbactam. Nearly half required hospitalization, with a high mortality rate (18%). After recovery from *Citrobacter* peritonitis, patients remained at high risk of developing technique failure within the following year.

The most common etiology of ESRD in our *Citrobacter* peritonitis cohort was chronic glomerulonephritis, differing from the conventional belief that DM nephropathy accounts for most ESRD (Table 1). However, ESRD patients from DM nephropathy less often received PD due to concerns of glycemic control. In addition, several PD registries reported similar findings. For example, a Hong Kong PD cohort identified glomerulonephritis to be the most common ESRD cause, followed by DM [9]. Another ANZDATA report also found glomerulonephritis to be more common than DM [7]. We suggested that both GN and DM are common etiologies than other causes, but the order of GN/DM might vary among studies.

*Citrobacter* species, gram-negative bacilli belonging to the Enterobacteriaceae family, are found to colonize human or animal GI tracts [27]. The main pathogenic strains are *C. koseri* and *C. freundii*, accounting for more than 70% of human infections [27-29]. In our study, *C. freundii* was the most frequent species identified from the effluent (73%). The risk factors for *Citrobacter* infections include advanced age, hospitalization, debilitation, and presence of multiple comorbidities [28, 30]. However, our PD patients had none of these risk factors but still developed *Citrobacter* peritonitis (Table 1). GI colonization by antibiotic-resistant gram-negative bacteria, such as *Citrobacter* species, might be more common in dialysis patients than the general population [31, 32]. Among these resistant gram negative bacteria, *Citrobacter* species are reportedly the most common type [33]. Furthermore, in our PD registry, we defined GI flora transmural migration as the origin of PD peritonitis, if the clinical scenarios presented with Gram-negative or polymicrobial effluent isolates in the absence of anatomically-documented bowel lesions, and in the presence of constipation and/or diarrhea. This is compatible with the constipation/diarrhea associated PD peritonitis origin in our current study. In light of these arguments and our finding, PD patients might acquire *Citrobacter* PD peritonitis more frequently through GI microbial transmural migration (45%) than PD peritonitis of other pathogens (usually 5-10%) [21,22]. We postulated that the combination of potentially higher GI colonization rate and more frequent translocation might explain the development of *Citrobacter* PD peritonitis in these patients. The potential explanations for GI microbial transmural migration include alterations of intestinal flora, mucosal

Table 3. Clinical outcomes of *Citrobacter* PD-associated peritonitis episodes.

| Outcomes | *Citrobacter* (n=11) | Other Enterobacteriaceae (n=161) | P value* |
|----------|----------------------|-------------------------------|---------|
| **Short-term (per episode)** | | | |
| **Antibiotics** | | | |
| Primary response (%) | 6 (55) | 97 (60) | 0.664 |
| Antibiotic switch (%) (Secondary response) | 2 (18) | 27 (17) | 0.376 |
| Time to antibiotic switch (days) | 6 (3 - 7) | 7 (3 - 15) | 0.006 |
| Tenckhoff catheter removal (%) | 0 (0) | 26 (16) | 0.143 |
| Relapse peritonitis (%) | 1 (9) | 0 (0) | 0.005 |
| Repeat peritonitis (%) | 0 (0) | 0 (0) | 1.000 |
| Mortality (%) | 2 (18) | 11 (7) | 0.015 |
| Time to mortality (days) | 11 (7 – 14) | 16 (2 - 46) | 0.601 |
| **Long-term (per episode)** | | | |
| Technique failure with modality switch to hemo dialysis (%) | 8 (89) | 88 (59) | 0.023 |
| Time to technique failure (months) | 13 (1 – 31) | 19 (1 – 75) | 0.04 |
| **Causes of Technique failure** | | | |
| Refractory peritonitis (%) | 5 (63) | 75 (85) | |
| Ultrafiltration failure (%) | 1 (13) | 1 (1) | |
| Mortality (%) | 2 (25) | 4 (5) | |
| Renal transplantation (%) | 0 (0) | 8 (9) | |

Continuous variables are expressed in mean (ranges), while categorical variables are expressed in number (percentage). * Compared with independent t-test.
rates (15–55%) [30, 37, 45]. Among patients in the fourth-generation cephalosporins, carbapenems, or other GNB infections; these include high mortality. Consequently, physicians should also be cautious of fluoroquinolones, with or without aminoglycosides. Caused by antibiotics for the treatment of PD-related peritonitis. Citrobacter species are less susceptible to antibiotics, with high resistance to penicillin (70–90%) and cephalosporins (first generation, 40–70%; fourth generation, 1–5%), moderate resistance to aminoglycosides (10–40%), and variable resistance to quinolones [29, 36, 37, 40, 41]. Most Citrobacter species are susceptible to penicillin/beta-lactamase inhibitors and carbapenems (>90%) [29, 36, 37, 40, 41]. Prior use of broad-spectrum antibiotics (especially third-generation cephalosporins), or other antibiotics (such as vancomycin), is potentially associated with resistant strains of Citrobacter [37, 41]. However, none of our patients had been exposed to antibiotics prior to developing current peritonitis. Among all multidrug-resistant Enterobacteriaceae isolates, Citrobacter is the most common genus identified [41, 42]. In our cases, all Citrobacter isolates were resistant to ampicillin and highly resistant to ampicillin/sulbactam as well (Table 2), which is in contrast to the past Citrobacter antibiograms. Piperacillin/tazobactam would appear to be a useful alternative, but its intraperitoneal delivery could be problematic, given its incompatibility with aminoglycosides and its potential neurotoxicity [13, 25, 43]. Antibiotic susceptibility among Citrobacter species also differs; for example, C. koseri has lower antibiotic resistance than C. freundii [28, 44]. We also found that C. koseri demonstrated higher susceptibility to ampicillin/sulbactam, cefazolin and cefmetazole than C. freundii in our study. In addition, ineffective empirical antibiotics are associated with higher mortality in Citrobacter infections [41]. The most reliable first-line antibiotics for the treatment of PD-related peritonitis caused by Citrobacter would be third- or fourth-generation cephalosporins, carbapenems, or fluoroquinolones, with or without aminoglycosides. Consequently, physicians should also be cautious of prescribing penicillin/beta-lactamase inhibitors in cases of PD-related peritonitis caused by Citrobacter.

The clinical outcomes in patients with Citrobacter infections are often worse than those in patients with other GNB infections; these include high mortality rates (15–55%) [30, 37, 45]. Among patients in the present study, the mortality rate was 18%, similar to the rates in the literature but significantly higher than ones in PD-associated peritonitis caused by other pathogens (<3%) [1, 9]. These adverse outcomes could not be attributed to the slightly shorter duration of antibiotics use (11 days in 2 patients; 18%), since both patients were not hospitalized and were successfully cured by first-line antibiotics. PD-related peritonitis caused by Citrobacter is associated with a higher hospital admission rate (54%) and longer hospitalization duration (9 days); these findings are also reported in other non-Pseudomonas GNB (NPGNB) peritonitis episodes [7]. However, the mortality rate is significantly higher in PD-related peritonitis caused by Citrobacter than in other NPGNB peritonitis episodes (18% vs. 4%) [7], suggesting that Citrobacter should be considered separately from the spectrum of NPGNB in terms of peritonitis. The long-term outcomes of patients with PD-related peritonitis caused by Citrobacter are also poor, with most patients developing technique failure within 1 year (Table 3), most often caused by infection-related complications (7 cases; 88%). Indeed, the development of Citrobacter peritonitis in PD patients serves almost as a warning sign for infection-related technique failure within the following year. These patients should be closely monitored after their recovery from the episode of Citrobacter PD peritonitis.

Conclusion

To our knowledge, this study is the first consecutive series of episodes of PD-related peritonitis caused by Citrobacter to date, with focused descriptions of their clinical features. Constipation/diarrheas associated origin (potentially GI flora transmural migration) was the most common route of Citrobacter peritoneal infection. Patients with PD-related peritonitis caused by Citrobacter had high rates of hospitalization and mortality, with high technique failure rates within the upcoming year due to refractory peritonitis.

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Conflict of Interest

The authors have no relevant financial or non-financial competing interests to declare in relation to this paper.
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