


CASE REPORT

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Pantoea peritonitis in peritoneal dialysis: a report of two cases and literature review

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Abstract

Background *Pantoea* spp., a non-encapsulated, non-spore-forming Gram-negative rod bacterium that belongs to the *Erwiniaceae* family, can be found as a colonizer in humans, plants, and the environment, such as water and soil. Although it has the pathogenic potential to cause disease in humans, patients infected with this pathogen generally experience favorable outcomes. In this article, we present two cases of peritoneal dialysis (PD)-associated peritonitis caused by *Pantoea* spp. along with literature review.

Case presentation The first case is a 66-year-old male patient with end-stage kidney disease (ESKD) on PD, admitted for *P. dispersa* peritonitis. He presented with abdominal pain and cloudy dialysis effluent, responding well to intraperitoneal vancomycin and cefepime. Antibiotics were deescalated to ceftazidime monotherapy on the basis of antibiotic susceptibility testing. Despite initial recovery with a 3-week course of antibiotics, he developed recurrent peritonitis with *P. dispersa*, necessitating PD catheter removal and transition to hemodialysis. The second case is a 42-year-old male patient with ESKD on PD who was admitted after 6 days of bloody PD fluid without trauma or associated symptoms. With elevated PD fluid cell counts and positive PD fluid culture showing *Streptococcus mitis* and *P. agglomerans*, he was empirically treated for PD-associated peritonitis with intraperitoneal vancomycin and cefepime. Due to a sub-optimal response in repeat PD fluid cell counts at day 5, the PD catheter was removed, and he was switched to hemodialysis, followed by a 3-week course of intravenous ceftriaxone.

Conclusions We described two unique cases of *Pantoea* peritonitis in PD, recurrent *P. dispersa* peritonitis and refractory *P. agglomerans* peritonitis, both of which resulted in PD catheter removal. Our cases indicate the formation of bacterial biofilm as a potential reason for recurrence of infection and underscores the importance of vigilant monitoring and need for PD catheter removal in *Pantoea* peritonitis.

Keywords *Pantoea dispersa*, End-stage kidney disease, Peritoneal dialysis, Peritonitis

Introduction

Pantoea spp. are Gram-negative bacillus that are non-encapsulated and non-spore-forming. *Pantoea* became its own species in 1989, after being separated from *Enterobacteriaceae* [1], and is now placed within the *Erwiniaceae* family. Currently, at least 20 different subspecies have been identified. *Pantoea* spp. can be found as a colonizer in humans, plants, and the environment, such as water and soil. Its adaptability has led to promising applications in commercial and agricultural sectors, including biotechnology, environmental protection, soil

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bioremediation, and plant growth stimulation [2]. However, while infrequent, *Pantoea* species can be pathogenic to humans.

Previous case reports involving *Pantoea* spp. have included both immunocompetent and immunocompromised patients. Though described as a species with low virulence, fatal incidents have been seen in infants, postoperative patients, or those who are immunocompromised [3, 4]. Of the cases being reported, infections typically occur via traumatic, sinopulmonary, hepatobiliary, or iatrogenic routes and can lead to bacteremia. In this report, we present two cases of *Pantoea* peritonitis, recurrent peritonitis due to *P. dispersa*, and refractory peritonitis due to *P. agglomerans*, each of which developed in a different patient with end-stage kidney disease (ESKD) receiving peritoneal dialysis (PD).

Case presentation

Case 1

A 66-year-old male patient with a past medical history of ESKD, hypertension, type 2 diabetes, and gout was admitted with a 2-day history of worsening diffuse abdominal pain and cloudy PD effluent. Additional symptoms included chills, nausea, emesis, diarrhea, and worsening pedal edema. He had been treated with PD for 4 years and was on continuous cycling PD with two daytime exchanges at the time of presentation. He was a low-average transporter. Vital signs were within normal limits. Physical exam was notable for a tender abdomen to all quadrants. There was no rebound or guarding. His PD catheter exit site did not have erythema, warmth, drainage, or tenderness. Laboratory tests showed mild peripheral leukocytosis ($11.9 \times 10^3/\text{mm}^3$: reference, $4.0\text{--}10.0 \times 10^3/\text{mm}^3$), but otherwise compatible with

the history of ESKD. Analysis of PD effluent revealed an elevated white blood cell (WBC) count ($9749 \text{ cells}/\text{mm}^3$) with neutrophil predominance (98%). Vancomycin and cefepime were empirically initiated due to the high local prevalence of methicillin-resistant *Staphylococcus aureus* and the susceptibility patterns of Gram-negative bacteria in the region. The loading doses of vancomycin 15 mg/kg and cefepime 1 g were given intravenously, followed by continuous intraperitoneal (IP) doses (i.e., 25 mg/L and 125 mg/L, respectively). The patient reported significant improvement in abdominal pain by day 3 when PD fluid culture grew *P. dispersa*, identified by Vitek® 2 MS (bioMérieux Co., Nürtingen, Germany), an automated mass spectrometry microbial identification system that uses matrix assisted laser desorption ionization time-of-flight (MALDI-TOF). Susceptibility testing showed low minimum inhibitory concentrations (MIC) to all tested antibiotics including aminoglycosides, trimethoprim/sulfamethoxazole, fluoroquinolones, and cephalosporins except for ceftazidime, which was resistant (Table 1). The antibiotic regimen was changed to continuous IP ceftazidime monotherapy using a concentration of 125 mg/L. A repeat PD fluid analysis on day 4 confirmed a good response to antibiotic therapy (WBC $113 \text{ cells}/\text{mm}^3$). The patient was discharged and completed a 3-week outpatient antibiotic course of intermittent IP ceftriaxone 1 g/day as outpatient. Another PD fluid analysis was done on the last day of the 3-week antibiotic course and showed a WBC count of $38 \text{ cells}/\text{mm}^3$. Blood culture remained negative throughout the course (Fig. 1).

However, 12 days after he completed the antibiotics, he required re-hospitalization due to severe, acute-onset abdominal pain associated with nausea and vomiting. He was febrile to $38.0 \text{ }^\circ\text{C}$ and had diffuse,

Table 1 Susceptibilities of isolated *Pantoea* species in the two cases

Antibiotics	<i>Pantoea dispersa</i> in case 1		<i>Pantoea agglomerans</i> in case 2	
	Minimum inhibitory concentration	Interpretation	Minimum inhibitory concentration	Interpretation
Amikacin	< =2	Susceptible	< =2	Susceptible
Cefepime	< =1	Susceptible	< =1	Susceptible
Cefoxitin	32	Resistant	16	Intermediate
Ceftazidime	< =1	Susceptible	< =1	Susceptible
Ceftriaxone	< =1	Susceptible	< =1	Susceptible
Ciprofloxacin	< =0.25	Susceptible	< =0.25	Susceptible
Gentamicin	< =1	Susceptible	< =1	Susceptible
Levofloxacin	< =0.12	Susceptible	< =0.12	Susceptible
Meropenem	< =0.25	Susceptible	N/A	N/A
Tobramycin	< =1	Susceptible	< =1	Susceptible
Trimethoprim + sulfamethoxazole	< =20	Susceptible	< =20	Susceptible

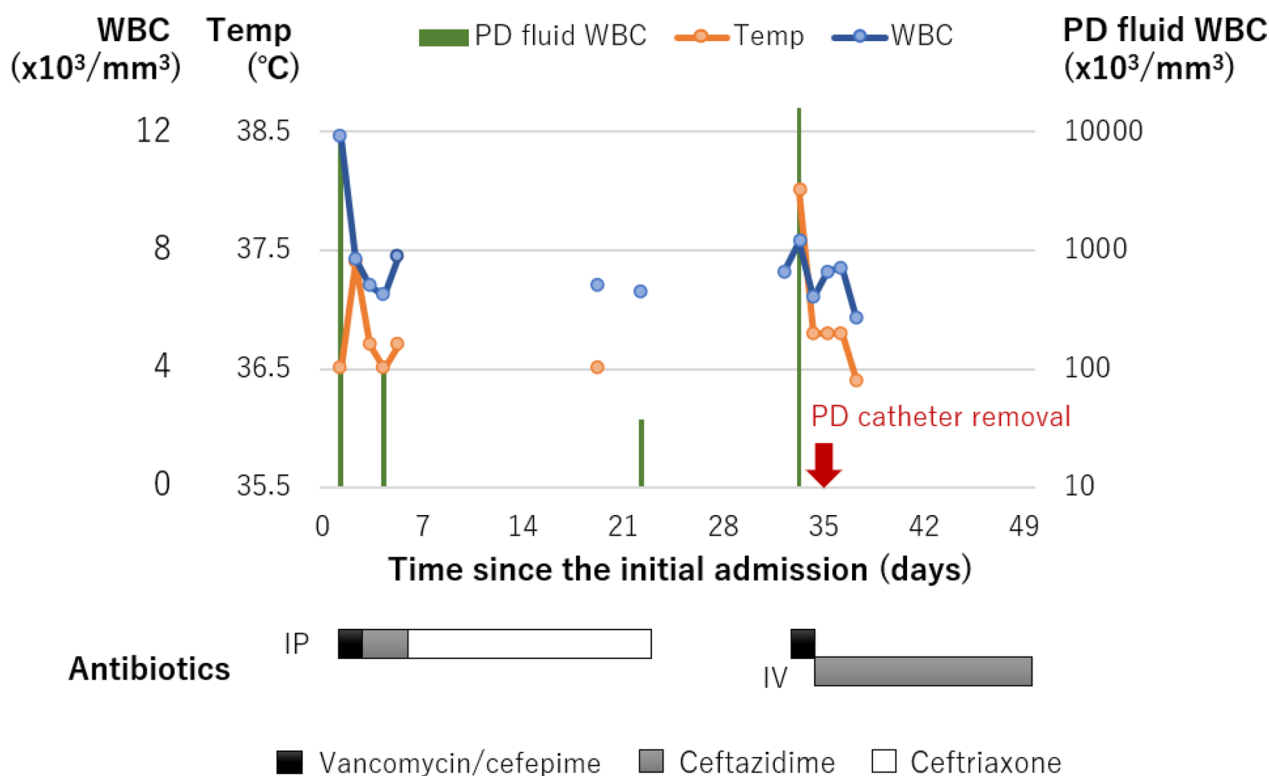


Fig. 1 Timeline of clinical events from the initial admission for *Pantoea dispersa* PD-associated peritonitis. PD, peritoneal dialysis; temp, body temperature; IP, intraperitoneal; IV, intravenous

moderate abdominal tenderness. He did not have leukocytosis (WBC 7.8×10^3 cells/mm³). PD effluent showed an elevated WBC count ($15,967$ cells/mm³) with neutrophil predominance (97%), and the patient was started on the same empirical IP antibiotic regimen with the initial episode. PD fluid culture grew *P. dispersa* again, confirming recurrent peritonitis. Susceptibility testing showed the same levels of MIC to all the tested antibiotics. The patient again showed good clinical improvement, but the decision was made to remove the patient’s PD catheter given the recurrent nature of peritonitis. The patient underwent PD catheter removal and started intermittent hemodialysis via a tunneled dialysis catheter. He was discharged home and completed a 2-week course of IV ceftazidime 2 g three times a week at his dialysis clinic. He remained stable for 2 weeks after the completion and was cleared for PD catheter placement. However, he made the decision to continue receiving in-center hemodialysis and declined the offer to resume PD.

Case 2

A 42-year-old male patient with a past medical history of ESKD and hypertension was admitted after 6 days of bloody PD fluids without any recent trauma or systemic illness. He had been treated with PD for 2.5 years

and was on continuous cycling PD without a last fill at the time of presentation. He was a high-average transporter. He reported no symptoms of abdominal pain, fever, chills, nausea, vomiting, or diarrhea and confirmed he was not on any anticoagulants. At admission, his vitals were largely normal except for a mildly elevated blood pressure at 174/88 mmHg. Computed tomography (CT) imaging showed no significant abnormalities. There was no peripheral leukocytosis (WBC 9.0×10^3 cells/mm³; 67% neutrophils), and the whole blood RBC count was 3.22×10^6 cells/mm³. However, a repeat PD fluid analysis showed worsening cell counts with a WBC count of 2235 cells/mm³ (43% neutrophils) and an RBC count of $473,922$ cells/mm³, compared with the results from 2 days prior, which showed a WBC count of 11 cells/mm³ (74% neutrophils) and an RBC count of 2556 cells/mm³. Although small hemoperitoneum might have influenced the PD effluent WBC count to some degree, the patient was started on the empirical vancomycin and cefepime for presumed PD-associated peritonitis using the same regimen with case 1. On day 2, PD fluid cultures identified *Streptococcus mitis* and *Pantoea agglomerans*, which showed good susceptibility to all tested antibiotics except for penicillin G (intermediate) and cefoxitin (intermediate), respectively, and intraperitoneal cefepime

Table 2 Case summaries of *Pantoea dispersa* infection

Author (year)	Sex	Age	Site of Infection	Underlying disease/ risk factors	Treatment	Antibiotic resistance	Outcome
Asai, et al. (2019) [7]	F	38	Bacteremia from cholangitis	Choledocholithiasis	Meropenem	Pan-sensitive	Improved
Schmid, et al. (2003) [30]	F	71	Respiratory infection	Acute myeloid leukemia	Clarithromycin amoxicillin/clavulanate	Pan-sensitive	Improved
Hagiya and Otsuka (2014) [31]	M	64	Bacteremia from pacemaker infection	Dilated cardiomyopathy, sick sinus syndrome, and diabetes	Cefepime	N/A	Improved
Mehar et al. (2013) [6]	M	Neonate	Bacteremia of unknown source	N/A	Piperacillin-tazobactam + amikacin followed by meropenem + amikacin	Cefazolin	Improved
	M	Neonate	Bacteremia of unknown source	Preterm (31 weeks)	Ampicillin-sulbactam + amikacin	Cefazolin	Deceased
Preis et al. (2022) [9]	M	33	Folliculitis	N/A	Trimethoprim-sulfamethoxazole	N/A	Improved
Panditaro et al. (2018) [5]	F	23	Hospital-acquired pneumonia	Intrauterine operation following intrauterine death	Colistimethate	MDR	Deceased
Yang et al. (2022) [8]	M	51	Bacteremia	Cirrhosis and hepatocellular carcinoma	Ceftazidime followed by cefepime	Ceftazidime	Improved
Ruan and Qin (2022) [4]	F	72	Bacteremia after thoracentesis	Status post-thoracentesis, diabetes	Cefoperazone-sulbactam + daptomycin + imipenem	MDR	Deceased
NI, L et al. (2023) [26]	M	84	Peritonitis	ESKD on PD	Tobramycin	MDR	Improved
Our case 1	M	66	Peritonitis	ESKD on PD	Ceftriaxone followed by ceftazidime	Cefoxitin	Improved with PD catheter removal

Bold letters indicate clinically significant findings

ESKD, end-stage kidney disease; PD, peritoneal dialysis; MDR, multidrug resistance

was continued. However, repeat peritoneal dialysis fluid analysis on day 5 indicated a suboptimal response to the antibiotic regimen, showing a WBC count of 473 cells/mm³ with 17% neutrophils. Although a longer observation for antibiotic effect was an option, patient decided to remove the PD catheter and switch his dialysis modality to hemodialysis after discussing potential advantages and disadvantages of each approach. Post-catheter removal, repeat PD fluid culture obtained on day 5 turned out to be negative, and the patient was treated with intravenous ceftriaxone 2 g three times a week at his dialysis clinic for a total of 3 weeks.

Literature research

The literature search was conducted using PubMed, covering the period from January 1989 to January 2024. We used a combination of keywords “*Pantoea*” and “peritonitis” to identify relevant articles. Additionally, another combination of “*Pantoea dispersa*” and “infection” was used in a separate search to ensure comprehensive coverage of the topic.

Discussion

We described two unique cases of *Pantoea* peritonitis in PD, recurrent *P. dispersa* peritonitis and refractory *P. agglomerans* peritonitis, both of which resulted in PD catheter removal despite the standard duration of susceptible antibiotic treatment. The route of *Pantoea* spp. peritonitis remained unclear in both cases despite our best efforts. Previous reports of *P. agglomerans* soft tissue and blood stream infections suggested ingestion of infected plants or thorn pricks as potential causes. However, both patients denied such events, engaging in gardening activities, or any touch contamination, but reported strict adherence to hygiene techniques. These cases underscore the importance of vigilant monitoring and the potential need for PD catheter removal in *Pantoea* peritonitis.

The most common isolated *Pantoea* species in the healthcare setting is *P. agglomerans* [4], but there is an increasing number of reports on infections attributable to *P. dispersa*, especially among infants, elderly, and immunocompromised patients [4–9]. Interestingly, *P. dispersa* infection may be more common in certain geographic areas or in rhinosinusitis. A multicenter

Table 3 Case summaries of peritoneal dialysis-associated peritonitis with *Pantoea species* infection

Author (year)	Sex	Age	Pathogen	Antibiotics used/route	Outcome
Lau et al. (2005) [13]	F	2	<i>P. agglomerans</i>	Cefotaxime, gentamycin/IP	Improved, PD catheter replaced
Lim et al. (2006) [14]	F	49	<i>P. agglomerans</i>	Ceftazidime, amikacin/IP	Improved
Magnette et al. (2008) [15]	M	65	<i>P. agglomerans</i>	Ciprofloxacin/IP	Improved
Ferrantino et al. (2008) [16]	M	51	<i>P. agglomerans</i>	Cefepime/IP	Improved
Habhab et al. (2008) [17]	F	52	<i>P. agglomerans</i>	Ciprofloxacin/oral	Improved
Moreiras-Plaza et al. (2009) [18]	M	45	<i>P. agglomerans</i>	Ciprofloxacin/IP	Improved
Borras et al. (2009) [19]	M	56	<i>P. agglomerans</i>	Tobramycin/NA	Improved
Kahveci et al. (2011) [20]	F	89	<i>P. agglomerans</i>	Imipenem/IV	Deceased after refusing PD catheter removal
Choi et al. (2012) [21]	M	52	<i>P. agglomerans</i>	Cefazolin, gentamycin/IP	Improved
Chen et al. (2013) [22]	F	58	<i>P. agglomerans</i>	Levofloxacin/IV	Improved
Sastre et al. (2017) [23]	M	83	<i>P. agglomerans</i>	Cefazolin, tobramycin/IP	Improved
Essam et al. (2018) [24]	M	19	<i>Pantoea species</i>	Ceftriaxone/IP	Improved
Mateus et al. (2022) [25]	F	33	<i>P. agglomerans</i>	Meropenem, gentamycin/NA	Improved, PD catheter removed
	M	63	<i>P. agglomerans</i>	Ceftazidime, ciprofloxacin/IP	Improved
	F	64	<i>P. agglomerans</i>	Vancomycin, gentamycin, and ciprofloxacin/IV	Improved
NI et al. (2023) [26]	F	45	<i>P. agglomerans</i>	Cefazoline and ceftazidime/IV	Improved
	M	84	<i>P. dispersa</i>	Tobramycin/IP	Improved
	M	66	<i>P. dispersa</i>	Ceftriaxone followed by ceftazidime/IP	Improved, PD catheter removed
Our case 2	M	42	<i>P. agglomerans</i>	IP cefepime followed by IV ceftriaxone	Improved, PD catheter removed

IP, intraperitoneal; IV, intravenous; NA, not available; PD, peritoneal dialysis

study in Ethiopia evaluated 426 positive blood culture results confirmed by MALDI-TOF between 2019 and 2020 and reported that 21 samples (5%) were positive for *P. dispersa* [10]. Of those, 20 (95%, 18 from the neonatal ICU and 2 from the adult emergency department) were isolated at one specific hospital in the northern part of the country where *P. dispersa* was the second most frequent pathogenic bacteria (15%). Additionally, *P. dispersa* showed an overall multidrug resistance frequency of 81%. In Italy, however, the prevalence of *Pantoea species* accounted for only 19 of 4996 G-negative isolates from blood culture, of which only one isolate was identified as ampicillin-resistant *P. dispersa* [11]. Another case series report from a hospital in Taiwan evaluated 274 rhinosinusitis patients with positive sinus culture and found that *P. dispersa* was identified in a total of 36 patients (13%) [12]. Patients with *P. dispersa*, compared with those with other pathogens, received a significantly shorter duration of antibiotic treatment and had lower surgery rate without significant difference in clinical outcomes. No data were available regarding the prevalence of *P. dispersa* infection among other infections at their institution or in other geographic areas of Taiwan.

Otherwise, there have been only ten reported cases of *P. dispersa* infection, as summarized in Table 2. Of those, two cases were neonates and two adults were immunocompromised due to acute myeloid leukemia and

cirrrosis, respectively, while the remaining were immunocompetent adults. Of eight cases reporting antibiotic sensitivities, five noted single or multidrug resistance. Commonly used antibiotic classes included beta-lactams, carbapenems, macrolides, aminoglycosides, and glycopeptides. Patients with *P. dispersa* infection were reported to have favorable outcomes [6], but three of the ten patients died [3–5].

The management strategy for *Pantoea spp.* peritonitis is yet to be established. Literature review identified a total of 17 cases of PD-associated peritonitis caused by *Pantoea spp.* (Table 3) [13–26], with only one case being *P. dispersa* PD-associated peritonitis published as a conference abstract [26]. Of those, three had unfavorable outcomes despite the use of antibiotics on the basis of susceptibility; two cases required PD catheter removal because of the lack of clinical improvement [13, 24], and the other case with *P. agglomerans* died due to septic shock after refusing PD catheter removal [20]. The previous case of *P. dispersa* PD-associated peritonitis showed multidrug resistance but was successfully treated with IP tobramycin without relapse or complication [26]. Our case responded well to the targeted antibiotic therapy, including ceftazidime and ceftriaxone, and susceptibility testing revealed fairly susceptible organism. Therefore, resistance to antibiotics is unlikely the underlying cause of peritonitis recurrence. Instead, the formation of

bacterial biofilm, as seen in the case of *P. agglomerans* [27, 28], could be a potential reason for the recurrence of PD-associated peritonitis by *P. dispersa* in our case. Microbiological evaluation of a removed PD catheter should be considered in future cases to examine this hypothesis.

One of the pitfalls in identifying *Pantoea* species is the multiple types of analysis that this bacterium needs to undergo to be correctly identified. As done in our case, MALDI-TOF-based identification methods are commonly used in clinical laboratories as a rapid means of identification, but a previous study using *cpn60*-based molecular typing demonstrated that 24% of such isolates were misidentified and were actually strains of *Citrobacter*, *Enterobacter*, *Kosakonia*, *Klebsiella*, or *Pseudocitrobacter*, members of the newly described *Erwinia gerundensis*, and even several unclassified members of *Enterobacteriaceae* [29]. However, none of the centers at which 24% isolates were misidentified used the same MALDI-TOF system as our laboratory.

In conclusion, we presented two rare cases of PD-associated peritonitis caused by *Pantoea* spp. along with literature review. It is imperative to correctly identify this species along its susceptibility, as there have been documented cases of this organism being multidrug resistant. Additionally, further investigation is necessary to evaluate the risk of recurrence and to develop the best treatment strategy including the duration of antibiotic treatment and PD catheter salvage versus removal in the setting of *Pantoea* peritonitis.

Abbreviations

PD	Peritoneal dialysis
ESKD	End-stage kidney disease
MALDI-TOF	Matrix assisted laser desorption ionization time-of-flight
MIC	Minimum inhibitory concentrations

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Author contributions

Y.O., P.V., B.C.M., M.H., and Z.A.K. participated in patient care. Y.O. and B.C.M. drafted the manuscript. All authors contributed to interpretation of data, reviewed the manuscript critically for important intellectual content, and approved the final version of the manuscript to be published. All authors are accountable for the manuscript ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Declarations

Ethics approval and consent to participate

A single case report does not meet the definition of "research" and does not require IRB review per the policy of the University of Mississippi Medical Center.

Consent for publication

The authors declare that they have obtained consent from the patient reported in this article for publication of the information about him that appears within this case report.

Competing interests

The authors declare that they have no competing interests.

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