Renal Replacement Therapy

CASE REPORT

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Peritoneal dialysis-associated peritonitis caused by *Tsukamurella inchonensis*: a case report and literature review

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Abstract

Background Peritoneal dialysis (PD)-associated peritonitis is a serious complication that can lead to PD discontinuation and mortality. *Tsukamurella* species are uncommon opportunistic pathogens that can cause peritonitis, often necessitating catheter removal.

Case presentation A 46-year-old woman with a 1.5-year history of PD presented with fever and lower abdominal pain, along with cloudy peritoneal effluent showing an elevated cell count. Empiric antibiotic therapy with ciprofloxacin, cefazolin, and ceftazidime was started for PD-associated peritonitis. The peritoneal effluent culture yielded a gram-positive rod with weak acid-fast staining. A rebound in the cell count necessitated a switch of antibiotics to meropenem and vancomycin. This led to improvement, and the treatment was switched to oral levofloxacin on day 30 and the patient was discharged on day 35. Subsequently, 16S rRNA gene sequencing confirmed the isolate as *Tsukamurella inchonensis*.

Conclusion This case highlights the challenges in identifying and treating *Tsukamurella* peritonitis. Successful treatment without catheter removal in this case suggests that early detection and appropriate antibiotics may enable catheter salvage in this rare infection. However, further research is needed to establish optimal treatment strategies.

Keywords *Tsukamurella inchonensis*, Opportunistic pathogen, Peritonitis, Peritoneal dialysis

Introduction

Tsukamurella species are aerobic, gram-positive, weakly acid-fast bacilli found in various environmental sources and are increasingly recognized as opportunistic pathogens in humans [1, 2]. They can cause a wide range of infections, including pulmonary [3, 4], cutaneous [5], ocular [6], bloodstream [7, 8], central nervous system [9], and peritoneal infections [10]. However, accurate identification of these species remains challenging in many clinical microbiology laboratories due to phenotypic and biochemical similarities to other actinomycetes, such as *Corynebacterium*, *Rhodococcus*, *Nocardia*, and non-tuberculous mycobacteria [1].

One of the most significant complications of peritoneal dialysis (PD) is PD-associated peritonitis, which is a leading cause of PD discontinuation and mortality [11]. Although prompt empiric antibiotic treatment with broad-spectrum coverage of both gram-positive and gram-negative organisms is crucial, the emergence of less common and drug-resistant pathogens presents an obstacle to successful treatment. Reports of PD-associated peritonitis caused by *Tsukamurella* spp. are limited, and catheter removal is frequently required for resolution [10, 12]. Recent advances in molecular diagnostic

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methods, particularly matrix-assisted desorption ionization time-of-flight mass spectrometry (MALDI–TOF MS) and 16S rRNA gene sequencing, have significantly improved the identification of *Tsukamurella* infections.

We report a rare case of PD-associated peritonitis caused by *Tsukamurella inchonensis* that was successfully treated without catheter removal, highlighting the potential for catheter salvage treatment strategies.

Case presentation

A 46-year-old Japanese woman with a 1.5-year history of automated PD due to autosomal dominant polycystic kidney disease presented with a 3-day history of fever and lower abdominal pain. She had no prior history of PD-associated peritonitis. She had body temperature of 38.9℃, blood pressure of 124/94 mmHg, and heart rate of 131 beats per minute. The PD effluent was cloudy with a cell count of 1000 cells/μL. There was no sign of exit-site infection, history of PD tube troubles, or touch contamination. She was a homemaker and did PD exchange manually without use of a PD connection assist device. She had no pets or hobbies such as gardening. She was admitted with the diagnosis of PD-associated peritonitis. Laboratory findings on admission are presented in Table 1. After collection of blood, urine, and peritoneal effluent culture samples, she was started on oral ciprofloxacin (CPFX), intraperitoneal (IP) cefazolin (CEZ), and IP ceftazidime (CAZ) daily. The IP antibiotics were administered continuously (every 6 h). The blood and urine cultures were all negative. The aerobic culture bottle of the peritoneal effluent was positive, and gram-positive rod bacteria with weak acid-fast staining were isolated (Fig. 1a, b). The bacterial isolate could not be identified by MALDI–TOF MS (Bruker MALDI Biotyper®). Our facility was not able to identify the species, but we suspected *Tsukamurella* spp. due to its yellowish cream-colored colonies and its gram-staining and other staining characteristics (Fig. 1c–e). We sent the specimen to the Medical Mycology Research Center of Chiba University for molecular analysis. The peritoneal effluent cell count initially decreased after treatment initiation. However, on day 11, a rebound in the cell count to 2770/μL prompted discontinuation of oral CPFX and IP CEZ, and addition of intravenous (IV) meropenem and IP vancomycin. This treatment modification resulted in a decrease in cell count to 70/μL by day 21. By day 30, the count remained consistently below 10/μL, allowing for a switch to oral levofloxacin (LVFX). On day 34, 16S rRNA gene sequencing identified the bacterial isolate as *Tsukamurella inchonensis* (antibiogram in Table 2) [10, 13, 14]. The following day, the patient was discharged with a 1-week prescription of LVFX.

Table 1 Laboratory data on admission

Blood cell count	
White blood cells (/μL)	10,300
Red blood cells (/μL)	478
Hemoglobin (g/dL)	13.0
Hematocrit (%)	42.9
Platelet count (/μL)	219
Blood chemistry	
Total protein (g/dL)	7.5
Albumin (g/dL)	3.6
Total bilirubin (mg/dL)	0.36
AST	13
ALT	11
LDH	240
Blood urea nitrogen (mg/dL)	50.1
Creatinine (mg/dL)	11.54
Sodium (mmol/L)	141
Potassium (mmol/L)	4.2
Chloride (mmol/L)	104
Calcium (mg/dL)	8.4
Phosphorus (mg/dL)	3.8
C-reactive protein (mg/dL)	9.49
Peritoneal effluent	
Cell count (/μL)	1,000
Neutrophils (%)	96.0

AST aspartate aminotransferase, *ALT* alanine aminotransferase, *LDH* lactate dehydrogenase

Two months later, the patient developed peritonitis caused by *Micrococcus luteus* and *Moraxella osloensis*, which resolved with a 3-week course of antibiotics. Although this episode was unrelated to the prior *Tsukamurella* infection, its occurrence within 2 months of discharge, coupled with the involvement of environmental bacteria, highlighted the need for improved hygiene practices, particularly in areas prone to bacterial growth, such as the kitchen and bathroom. Therefore, although we did not perform environmental culturing, we recommended improving the patient's home environment, including the bathroom and shower, and thoroughly wiping off any water droplets that may have adhered to the catheter after bathing. There have been no signs of peritonitis for 6 months since then.

Discussion

Here, we presented a rare case of PD-associated peritonitis caused by *Tsukamurella inchonensis*, an emerging opportunistic pathogen. To our knowledge, only a few cases of PD-associated peritonitis due to *Tsukamurella* have been reported. Although our MALDI–TOF MS analysis of the bacterial isolate failed to provide definitive

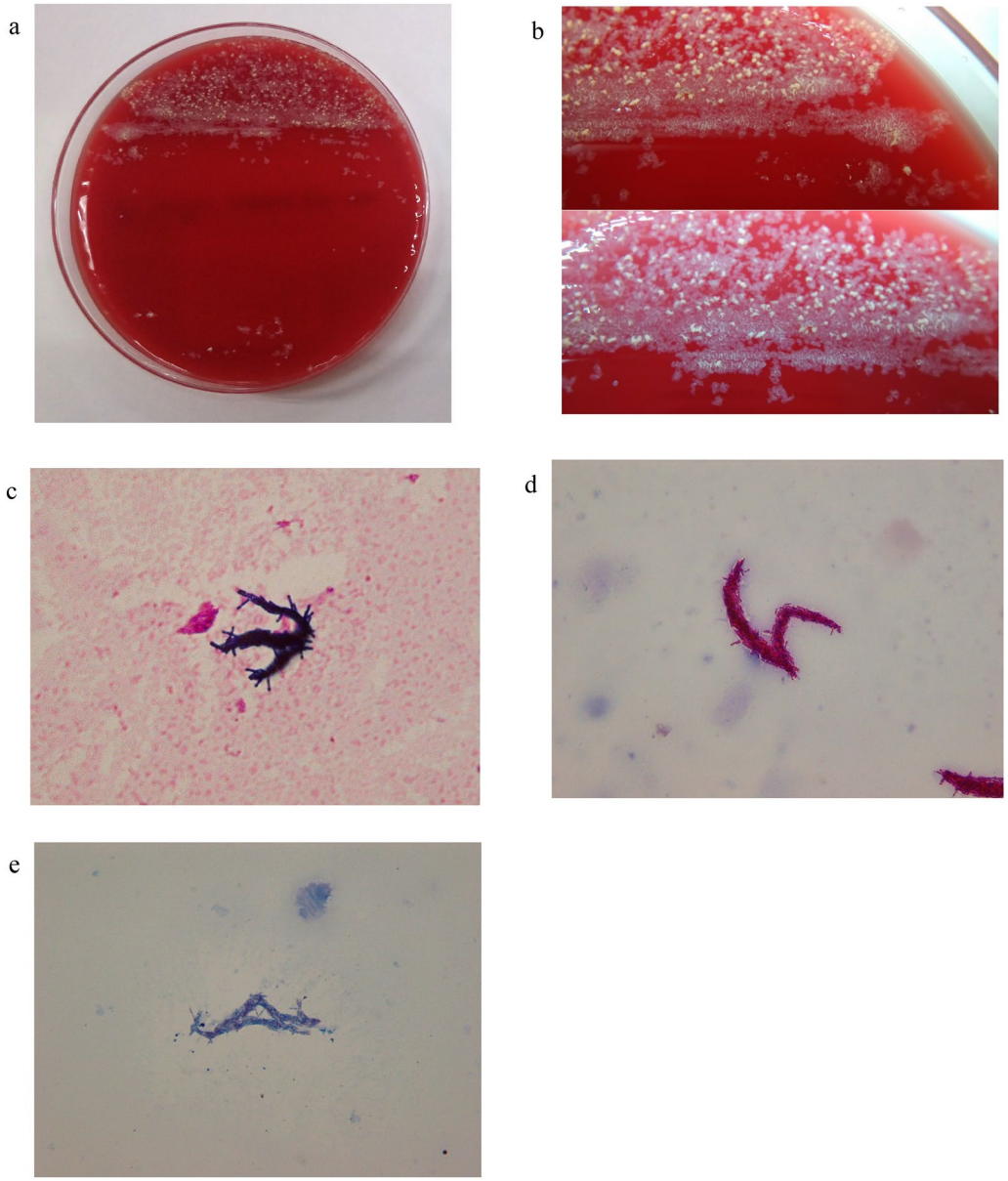

Fig. 1 Colony appearance and cell morphology for *Tsukamurella Inchonensis*. **a** Isolates were grown on TSA II 5% sheep blood agar M® (BD, Tokyo, Japan) aerobic culture for 2 days. **b** Close-up image. Small, dry, round, yellowish cream-colored were observed. **c** Gram staining. **d** Kinyoun staining. **e** Ziehl–Neelsen staining

identification, the isolate's colony morphology and Gram-staining characteristics suggested the possibility of *Tsukamurella* infection. This prompted further analyses such as 16S rRNA gene sequencing at another institution because of the lack of specialized equipment for *Tsukamurella* identification at our facility. *Tsukamurella* colonies are typically small and dry, with convex elevation, and appear white, cream-colored, or orange [1]. Only peritoneal effluent culture collected in an aerobic bottle yielded positive results, and the bacterial isolate from this case displayed slow growth, forming small, dry, round, yellowish cream-colored colonies.

According to an in vitro study, *Tsukamurella* spp. are susceptible to quinolones, trimethoprim/sulfamethoxazole, amikacin, minocycline, linezolid, and tigecycline [15]. In that study, *Tsukamurella* isolates were also susceptible to the third- and fourth-generation cephalosporins. Cases of PD-associated peritonitis caused by *Tsukamurella* spp. are limited, and the optimal management strategy has not yet been determined (Table 3)

Table 2 Antibiograms for peritoneal dialysis-associated peritonitis caused by *Tsukamurella*

Antibiotics	MIC of *T. inchonensis* (present case)	Susceptibility	MIC of *T.* spp. [10]	MIC of *T. paurometabola* [13]	MIC of *T. inchonensis* [14]
Amikacin	4	S	–	–	–
Ampicillin/clavulanate	> 32/16	R	–	> 4/2	–
Ceftriaxone	8	S	–	–	–
Ciprofloxacin	2	I	–	–	1
Imipenem	< 0.5	S	–	0.125	≤ 1
Linezolid	2	S	–	–	≤ 1
Minocycline	1	S	–	–	–
Sulfamethoxazole-trimethoprim	19/1	S	–	> 76/4	> 38/2
Tobramycin	> 16	R	–	–	–
Cefotaxime	4	S	–	> 2	–
Cefepime	2	S	–	> 2	–
Doxycycline	4	I	–	–	–
Gentamicin	2	S	8	–	≤ 0.5
Ampicillin	> 8	NA	–	> 8	8
Clarithromycin	4	I	–	0.25	–
Erythromycin	> 2	NA	2	> 2	–
Vancomycin	–	–	4	> 1	–

I intermediate, *MIC* minimum inhibitory concentration, *NA* not available, *R* resistant, *S* susceptible

[10, 12–14]. Caution may be needed regarding the use of quinolone since the mechanism conferring quinolone resistance in *Tsukamurella tyrosinosolvens* has been reported [15], which might be one of the reasons for the difficulty we encountered using CPFX to treat *Tsukamurella inchonensis*. Another reason was the discrepancy between the isolate's specific antibiogram, which showed "intermediate" susceptibility against CPFX, and our facility's standard antibiogram, which indicated it was "susceptible." This discrepancy could be due to the lack of species-specific data for *Tsukamurella* in our standard antibiogram.

For treatment of catheter-related bloodstream infections caused by *Tsukamurella* spp., the combination of catheter removal and appropriate antibiotic treatment is considered essential [8, 15]. In some previous reports, successful treatment of *Tsukamurella* PD-associated peritonitis required catheter removal [10, 12, 13]. However, a case from China [14] and our case demonstrated successful treatment without catheter removal. These reports are summarized in Table 3. Although we were unable to identify the route of infection, PD connection troubles [13] and diarrhea [14] prior to peritonitis have been reported as infection routes. The necessary treatment duration for *Tsukamurella* PD-associated peritonitis remains unclear. The case report from China described an 18-day treatment course [14]. Our case required 42 days of treatment including the duration of oral antibiotics administered after discharge. Also, repeat peritonitis caused by *Tsukamurella* has not been reported. Treatment recommendations for *Gordonia* peritonitis, a genus phenotypically similar to *Tsukamurella*, suggest at least 3 weeks of carbapenem–aminoglycoside combination therapy [16]. While evidence supporting successful treatment of *Tsukamurella* peritonitis without catheter removal is limited, our case demonstrates the potential for successful treatment with an antimicrobial therapy course exceeding 3 weeks, with subsequent close follow-up. Accurate identification of *Tsukamurella* can be achieved through newer molecular biological techniques, thus contributing to the appropriate selection of definitive therapy for infections caused by *Tsukamurella* [17]. Prompt therapy and understanding of the clinical, microbiological, and molecular characteristics of *Tsukamurella* spp. might prevent catheter removal in PD-associated peritonitis caused by these organisms.

Conclusion

Our experience with PD-associated peritonitis caused by *Tsukamurella inchonensis* suggests the potential for successful treatment without catheter removal. Accurate identification of *Tsukamurella* might contribute to the selection of appropriate therapy and infectious resolution.

Table 3 A review of published cases on PD-associated peritonitis caused by *Tsukamurella*

Author	Species	Age (years)	Sex	Cause of ESKD	Duration of PD	Infection route	Cell counts in PD effluent (/μL)	Antibiotic therapy	Duration of antibiotics	Catheter removal
Present case	*T. inchonensis*	47	F	ADPKD	1.5 years	UK	1000	Initial: CEZ IP., CAZ IP., CPFX p.o. Secondary: VCM IP, MEPM IV, LVFX p.o.	42 days	No
Shaer et al. [10]	*T.* spp. (not identified)	23	F	Alport syndrome	1 year	UK	833	Initial: VCM IP, GM IP, CPFX p.o. Secondary: CAZ IP, RFP p.o	2 months	Yes, after 2 months
Ismayilov et al. [12]	T. paurometabola	67	M	DM	8 years	UK	4,400	Initial: SBT/ABPC IV. Secondary: GM IV	51 days	Yes, on day 35
Nagamatsu et al. [13]	*T. pulmonis*	67	M	AGN	4 years	PD tube cap lost in bathroom	376	Initial: VCM IV, CAZ IP. Secondary: ST p.o., AMK IP, LVFX p.o., RFP p.o	49 days	Yes, on day 17
Tang et al. [14]	*T. inchonensis*	34	M	Type 1 DM	3 years	Diarrhea 1 month prior	5,450	VCM IP, MEPM IP, FCZ p.o	18 days	No

ADPKD autosomal dominant polycystic kidney disease, *AGN* acute glomerulonephritis, *AMK* amikacin, *DM* diabetes mellitus, *CAZ* ceftazidime, *CEZ* cefazolin, *CPFX* cefpodoxime, *FCZ* fluconazole, *GM* gentamicin, *IP* intraperitoneal, *IV* intravenous, *LVFX* levofloxacin, *MEPM* meropenem, *p.o.* per os, *RFP* rifampin, *ST* sulfamethoxazole-trimethoprim; *UK* unknown, *VCM* vancomycin

Abbreviations

CAZ	Ceftazidime
CEZ	Cefazolin
CPFX	Ciprofloxacin
IP	Intraperitoneal
LVFX	Levofloxacin
MALDI-TOF MS	Matrix-assisted desorption ionization-time of flight mass spectrometry
MEPM	Meropenem
PD	Peritoneal dialysis

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Author contributions
R.Y. wrote the manuscript. R.Y., M.K., K.I., Y.K., T.M., S.H., and M.A. contributed of management for the case. M.A. revised the manuscript. R.Y., H.T., T.M., and M.A. discussed the results and contributed to the final manuscript. All authors have read and approved the final manuscript.

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Availability of data and materials
The data used in this study are available from the corresponding author.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing interests
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