Renal Replacement Therapy

CASE REPORT

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Fatal peritoneal dialysis-associated peritonitis caused by *Mycobacterium mageritense*: a case report with review

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Abstract

Background Peritonitis is a serious and potentially fatal complication of peritoneal dialysis. We report a case of fatal peritonitis caused by *Mycobacterium mageritense* that was detected for the first time in peritonitis.

Case presentation A male patient in his 60 s undergoing peritoneal dialysis was admitted for catheter diversion with exit-site renewal. The patient had a refractory exit-site infection. Mycobacterial culture was not performed at the exit site prior to admission. After the surgery, the patient developed a fever, and a cloudy effluent was observed. Various antibiotics, including anti-tuberculosis drugs, were administered; however, his symptoms did not improve. The catheter was removed on the thirty-seventh day of admission. Bacteria positive for Ziehl–Neelsen staining were found in the peritoneal sample collected during the surgery. Since nontuberculous mycobacteria were considered the cause of peritonitis, the patient was administered imipenem/cilastatin, amikacin, and clarithromycin. However, he died of septic shock on the fifty-first day after admission. *Mycobacterium mageritense* was detected in the ascites culture after death.

Conclusion This is, to our knowledge, the first report of peritonitis caused by *Mycobacterium mageritense*. In patients undergoing peritoneal dialysis, when a refractory exit-site infection is observed, mycobacterial culture is necessary to prevent the development of peritonitis.

Keywords Nontuberculous mycobacteria (NTM), *Mycobacterium mageritense*, Peritonitis, Peritoneal dialysis

Background

Peritoneal dialysis (PD)-associated peritonitis is a serious complication of PD that causes withdrawal from dialysis and even death. Nontuberculous mycobacteria (NTM) account for 3% of all culture-positive exit-site infections and cases of peritonitis [1], and previous studies have reported that the mortality rate of NTM peritonitis was 14–30% [2, 3]. Treatment regimens for NTM peritonitis are not completely understood [4]. In this report, we describe a fatal case of PD-associated peritonitis caused by *Mycobacterium mageritense* (*M. mageritense*), which was detected in peritonitis for the first time.

Case presentation

A male patient in his 60 s undergoing PD for 7 years was admitted to our hospital for a catheter diversion procedure with exit-site renewal. The patient had an end-stage kidney disease associated with primary IgA nephropathy. The patient had no history of diabetes mellitus and had not received immunosuppressants. He had granulation at the exit site one year prior to admission.

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Methicillin-susceptible *Staphylococcus aureus* was detected in the culture of the exit-site sample. The patient was treated with topical gentamicin, oral first-generation cephems, and quinolones. However, the granulation increased, and methicillin-resistant *Staphylococcus aureus* was detected at the site 6 months later. Moreover, the external cuff had moved closer to the exit site. No mycobacterial culture test was performed at the exit site before admission. The patient had received vancomycin 5 days prior to admission. On admission, the patient's body temperature was 38 °C. The white blood cell count and C-reactive protein (CRP) levels were 3100/μL and 0.3 mg/dL, respectively. Laboratory data on admission are summarized in Table 1.

The patient's fever was believed to be due to viral infection, and on the second day of admission, the catheter diversion surgery was performed as planned (Fig. 1). Culture tests of blood collected on admission were negative. Methicillin-resistant coagulase-negative *Staphylococci* were detected in the pus cultures from the exit site and the resected catheter during catheter diversion surgery. On the tenth day of admission, the patient received 1 g of additional vancomycin. The next day, his body temperature reached 39 °C, and a rash appeared, which was considered a side effect of vancomycin. However, since the patient's CRP level had increased to 3.7 mg/dL and bacterial infection could not be ruled out, 4.5 g tazobactam/piperacillin twice daily was initiated on the thirteenth day of admission. On the twenty-second day of admission, the patient's effluent was observed to be cloudy, without symptoms of peritoneal irritation. The nucleated cell count of the effluent was 200 cells/μL (62% neutrophils and 7% lymphocytes). The patient's antibiotics were changed to cefazolin and cefepime, both at a dose of 1 g once daily, which were intraperitoneally administered. Bacterial culture, mycobacterial culture, and tuberculosis PCR tests of the effluent were all negative. On the twenty-eighth day after admission, the turbidity of the effluent persisted, and the nucleated cell count of the effluent increased to 2300 cells/μL (57% neutrophils and 12% lymphocytes). Cefazolin and cefepime were replaced with 0.25 g doripenem twice daily (a carbapenem antibiotic) and 0.3 g (6 mg/kg) daptomycin every 48 h. However, peritoneal irritation symptoms appeared on the thirty-first day after admission. Computed tomography revealed an increase in the density of the mesenteric fat tissue and thickening of the visceral peritoneum, indicating peritonitis (Fig. 2). We believed that the patient had developed tuberculous peritonitis because the bacterial culture of the effluent was negative, and antibiotics had no effect. Anti-tuberculosis drugs (0.3 g isoniazid daily, 0.45 g rifampicin daily, 1 g ethambutol every other day, and 1.5 g pyrazinamide every other day) were administered on the thirty-second day after admission. Doripenem and daptomycin were continued at the same volume. However, the patient's abdominal symptoms worsened, and the CRP level increased to 20 mg/dL. On the thirty-seventh day after admission, the PD catheter was removed laparoscopically (Fig. 1). Laparoscopy revealed fibrotic adhesions in the peritoneum, which indicated peritonitis. Bacteria positive for Ziehl–Neelsen staining were found in the peritoneal sample collected during the surgery. As the anti-tuberculosis drugs were ineffective, we believed that NTM caused the peritonitis. On the forty-sixth day after admission, doripenem and daptomycin were replaced with 0.25 g imipenem/cilastatin twice daily, 0.3 g amikacin on dialysis days only, and 0.5 g clarithromycin daily, which were believed to be effective against NTM. However, on the fifty-first day of admission, the patient died of septic shock. The postmortem ascites culture collected at the time of catheter removal surgery was positive for *M. mageritense*. The susceptibility results are shown in Table 2.

Table 1 Laboratory data of the patient at admission

Blood cell count		
Red blood cell	3.63×10^6	/μL
Hemoglobin	11.4	g/dL
Hematocrit	34.5	%
White blood cell	3100	/μL
Platelet count	14.3×10^4	/μL
Blood chemistry		
Total protein	6.0	g/dL
Albumin	3.2	g/dL
Total bilirubin	0.3	mg/dL
AST	17	IU/L
ALT	11	IU/L
LDH	221	IU/L
CK	98	IU/L
Blood urea nitrogen	47	mg/dL
Creatinine	12.5	mg/dL
Sodium	138	mEq/L
Potassium	3.0	mEq/L
Chloride	100	mEq/L
Calcium	9.3	mEq/L
Phosphorus	4.5	mEq/L
C-reactive protein	0.3	mg/dL
Iron	44	μg/dL
TIBC	245	μg/dL
Ferritin	120	ng/mL
Intact PTH	267	pg/mL
BNP	57	pg/mL

AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; LDH, Lactate dehydrogenase; CK, Creatine kinase; TIBC, Total iron binding capacity; Intact PTH, Intact parathyroid hormone; BNP, Brain natriuretic peptide

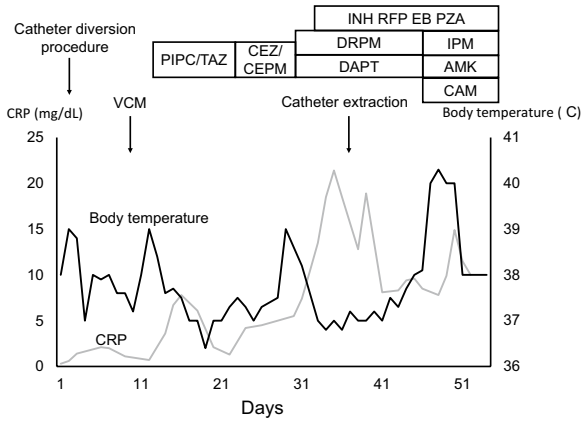

Fig. 1 Therapeutic process in this case. CRP, C-reactive protein; VCM, Vancomycin; PIPC/TAZ, Tazobactam/piperacillin; CEZ, Cefazolin; CEPM, Cefepime; DRPM, Doripenem; DAPT, Daptomycin; INH, Isoniazid; RFP, Rifampicin; EB, Ethambutol; PZA, Pyrazinamide; IPM, Imipenem; AMK, Amikacin; CAM, Clarithromycin

Discussion

We encountered a fatal case of PD-associated peritonitis caused by *M. mageritense*, a type of NTM. The vast majority of NTM peritonitis cases are caused by *Mycobacterium abscessus, Mycobacterium chelonae, and Mycobacterium fortuitum* [1, 2, 11–13]; however, in this case, the peritonitis was caused by *M. mageritense*, which has not been reported until now.

Diagnosing NTM peritonitis is difficult because the associated symptoms are fever, abdominal pain, and cloudy effluent, which are indistinguishable from those of bacterial and tuberculous peritonitis [2, 13]. In this case, anti-NTM drugs were initiated 25 days after the appearance of the cloudy effluent; according to previous reports, this duration is not long enough to affect the efficacy of the drugs [2, 12]. Here, we used imipenem/cilastatin, amikacin, and clarithromycin as anti-NTM drugs, to which the causative bacterium, *M. mageritense,* is susceptible. However, the patient still died.

ISPD Guideline 2022 recommends prompt catheter removal in cases of refractory peritonitis [4]. The guideline also notes that NTM peritonitis requires both effective antibiotics and catheter removal, although no specific antibiotic regimen has been presented. In this case, the patient could not be saved despite catheter removal and the administration of effective antibiotics. The mortality rate of NTM peritonitis has been reported to be as high as 14–30% [2]. Therefore, we believe that it is important to prevent the development of NTM peritonitis to avoid fatal outcomes. NTM peritonitis is caused mainly by the progression of exit-site or tunnel infections [14]. Mycobacterial cultures at the exit site, early surgical intervention, and antibiotic administration prevent the development of NTM peritonitis [14, 15]. Mycobacterial cultures should be performed to check for an NTM infection if the patient has a history of refractory exit-site infections. Early identification of the species and susceptibility allows the selection of an appropriate antibiotic. In this case, early diagnosis of the NTM infection would have allowed for earlier administration of antibiotics and possibly saved the patient.

The incidence of infections caused by *M. mageritense*, a nontuberculous mycobacterium first discovered in 1997, is growing rapidly [16]. Similar to this case, *M. mageritense* primarily infects the skin and soft tissues [5, 8, 9, 17–19]. In a previous report that investigated *M. mageritense antibiotic susceptibility*, all 23 specimens were susceptible to imipenem, amikacin, linezolid, ciprofloxacin, and trimethoprim-sulfamethoxazole [20]. However, our review of papers revealed that the susceptibility of *M. mageritense* was inconsistent with those findings (Table 2). In addition, most of the cases, such as this case, required some type of surgical intervention.

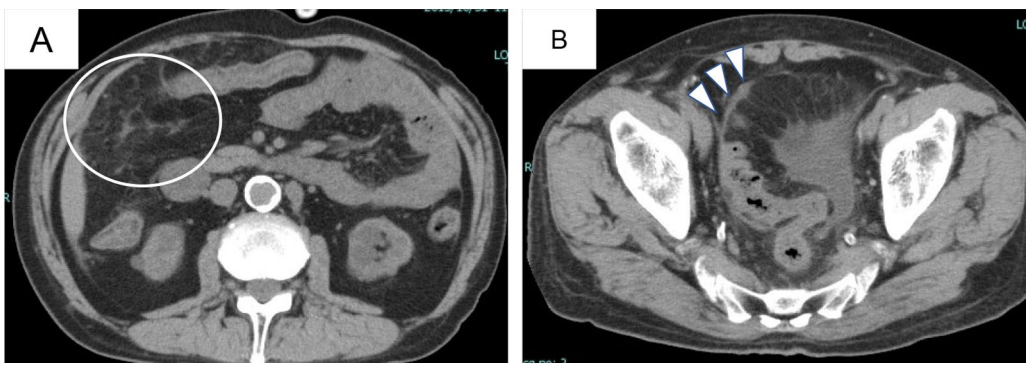

Fig. 2 Abdominal computed tomography images on the thirty-first day after admission. **A** White circle indicates increased mesenteric fat tissue concentration. **B** White arrowheads indicate thickening of the visceral peritoneum

Table 2 A review of past cases and our case on drug susceptibility and clinical features of *M. mageritense*-infected cases

	Present case	Case 1	Case 2	Case 3	Case 4	Case 5	
Age	60's	40's	66	77	5	44	
Sex	Male	Male	Male	Male	Girl	Female	
Underlying condition	ESKD on peritoneal dialysis	None specified	None specified	COPD, CAD	Acute encephalopathy, adrenal insufficiency	Breast cancer	
Site of infection	Skin and peritoneum	Parotid gland	Prosthetic joint	Pleura	Soft tissue	Soft tissue and CVP	
MIC (μg/mL)							Breakpoint of resistance (μg/mL)
IPM	< 0.5	1	4	< 0.5	0.5	< 2	≥ 32
AMK	2	> 64	8	4	32	< 8	≥ 64
CAM	0.25	N/A	> 32	16	> 512	> 8	≥ 8
TOB	4	N/A	N/A	> 16	> 16	N/A	≥ 8
LZD	1	2	< 1	< 4	8	< 4	≥ 32
CPFX	< 0.12	0.5	32	< 0.5	0.25	1	≥ 4
MFLX	< 0.06	N/A	< 0.25	< 0.25	0.19	< 1	≥ 4
MINO	0.5	N/A	< 1	2	4	N/A	≥ 8
ST	< 0.25/4.3	2	1/19	< 2	> 80	> 4	≥ 4/76
Antibiotics	IPM	LVFX	CPFX	IPM	Tosufloxacin	AMK	
	AMK	ST	ST	LVFX	LZD	CPFX	
	CAM		AMK				
			IPM				
Surgical intervention	Catheter removal	Parotid gland excision	Prosthetic joint explantation	Pleural fluid drainage	Abscess drainage	CVP removal and debridement	
Outcome	Death due to peritonitis	Cured	Cured	Death due to aspiration pneumonia	Cured	Cured	
Reference number	Present case	[5]	[6]	[7]	[8]	[9]	

Breakpoint of resistance values was based on those provided by Woods et al. [10]

MIC, Minimal inhibitory concentration; ESKD, End-stage kidney disease; IPM, Imipenem; AMK, Amikacin; CAM, Clarithromycin; TOB, Tobramycin; LZD, Linezolid; CPFX, Ciprofloxacin; MFLX, Moxifloxacin; MINO, Minomycin; ST, Trimethoprim-sulfamethoxazole; LVFX, Levofloxacin; COPD, Chronic obstructive pulmonary disease; CAD, Coronary artery disease; CVP, Central venous port

As mentioned above, in such cases, it is important to prevent the development of peritonitis to avoid fatal outcomes. Furthermore, since treatment regimens for peritonitis and catheter-related infection due to NTM have not been established [4, 21], it is necessary to accumulate many cases and establish a standard treatment regimen for infections caused by NTM.

Conclusion

Here, we report, to the best of our knowledge, the first case of peritonitis caused by *M. mageritense*. Since peritonitis can be fatal, physicians should perform mycobacterial culture in cases of refractory exit-site infections. Furthermore, if NTM infection is detected, appropriate treatment, including the use of antibiotics with high bacterial sensitivity, should be administered to prevent the development of peritonitis.

Abbreviations

NTM	Nontuberculous mycobacteria
CRP	C-reactive protein
M. mageritense	*Mycobacterium mageritense*

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Not applicable.

Author contributions

NH was primarily involved in patient treatment and wrote the manuscript. NK, SM, TN, HM, KO, RU, SB, RI, YU, and TH contributed to patient treatment and related discussions and reviewed the manuscript. All authors have read and approved the final manuscript.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
This case report was written in compliance with the Declaration of Helsinki.

Consent for publication
Consent for the publication of this case report was obtained from a family member of the patient.

Competing interests
The authors declare that they have no competing interests.

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