Peritoneal dialysis-related peritonitis with encapsulated ascites due to *Mycobacterium abscessus* subsp. *massilience* and subsp. *bolletii*: a case series and literature review

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Abstract

Background As there is no established standard of care for non-tuberculous mycobacterium (NTM) peritoneal dialysis (PD)-related peritonitis, its treatments have to be case-dependent, which is often difficult. Additionally, several reported cases were accompanied by encapsulated ascites, adhesive ileus, and encapsulating peritoneal sclerosis, suggesting treatment difficulties. We report two cases of PD-related peritonitis with encapsulated ascites due to *Mycobacterium abscessus* subsp. *massilience* and subsp. *bolletii*. To the best of our knowledge, this is the first case series to report PD-related peritonitis caused by *Mycobacterium abscessus* subsp. *bolletii*.

Case presentation The first case is that of a 74-year-old male patient who started PD six years ago for end-stage renal failure due to diabetic nephropathy. In February 2021, he presented with signs of infection at the exit-site and swelling of the tunnel. *Mycobacterium abscessus* subsp. *massilience* was detected in the culture of the exit-site exudate; thus, he was diagnosed with tunnel infection (caused by NTM). Subsequently, fever, abdominal pain, and increased cell counts in the PD drainage fluid were observed, and he was judged to have NTM peritonitis. His general condition improved after PD catheter removal in addition to antimicrobial treatment and encapsulated ascites drainage. The second case is that of a 52-year-old man who commenced PD for end-stage renal failure due to nephrosclerosis 12 years ago. In May 2022, he was diagnosed with PD-related peritonitis based on signs of infection at the exit-site, encapsulated ascites on computed tomography, and a cloudy PD drainage fluid. *Mycobacterium abscessus* subsp. *bolletii* was detected in the culture of the exit-site exudate, which led to the diagnosis of NTM peritonitis. In addition to antimicrobial treatment, PD catheter removal and encapsulated ascites drainage were performed. The patient also had adhesive bowel obstruction due to peritonitis and required decompression therapy with the insertion of a gastric tube.

Conclusions PD catheter removal and encapsulated ascites drainage might have improved inflammation and treatment outcomes. Additionally, *Mycobacterium abscessus* might be prone to forming encapsulated cavities and/or intestinal adhesions; however, further accumulation of cases clarifying "subspecies" of *Mycobacterium abscessus* is necessary to confirm this hypothesis.

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Background

PD-associated peritonitis due to NTM accounts for 0.3%–1.3% of peritonitis cases [1, 2]. Risk factors for PD-related peritonitis have been reported to include diabetes, obesity, race, climate and depression [3]. NTM peritonitis usually occurs in patients with a history of recurrent peritonitis, local trauma, catheter exit-site leaks, and poor aseptic manipulation techniques [4, 5]. Risk factors for NTM peritonitis also include immunodepression, inefficient dialysis, low residual renal function, exposure to environmental sources of the pathogen (such as soil and contaminated water), and a history of long-term broad-spectrum antibiotherapy for recurrent peritonitis [4, 5]. Treatment with clarithromycin and amikacin has been reported; however, no standard of care has been established [6, 7]. The duration of treatment is also unclear and previous reports vary [7]. Therefore, treatment for NTM peritonitis needs to be case-dependent, which is often difficult. Additionally, several reported cases were accompanied by encapsulated ascites, adhesive ileus, and encapsulating peritoneal sclerosis (EPS), all of which suggest treatment difficulties [8–13]. In this paper, we report two rare cases of PD-related peritonitis with encapsulated ascites due to *Mycobacterium abscessus.*

Case presentation

The first case is that of a 74-year-old male patient who had been undergoing PD for diabetic nephropathy-induced end-stage renal failure since 2015. In February 2021, he was diagnosed with exit-site and tunnel infection caused by NTM after signs of exit-site infection and swelling of the tunnel were observed and *Mycobacterium abscessus* subsp. *massilience* was detected in the exit-site exudate culture. In July 2021, he underwent simultaneous PD catheter removal and replacement and subcutaneous abscess drainage at another hospital (day 0). However, twelve days after, he developed fever, abdominal pain, and increased cell counts in the PD drainage fluid (total: 210/μL, polynuclear cells: 117/μL); thus, he was diagnosed with PD-related peritonitis due to *Mycobacterium abscessus*. Antimicrobial treatment (lascufloxacin 75 mg oral + clarithromycin (CAM) 200 mg oral + meropenem (MEPM) 0.5 g intravenous injection (IV) + isepamicin 400 mg IV) was started. *Mycobacterium abscessus* detected in the PD fluid showed high sensitivity to CAM and amikacin (AMK), intermediate sensitivity to imipenem (IPM),

and resistance to MEPM, tobramycin (TOB), moxifloxacin (MFLX), sulfamethoxazole-trimethoprim (ST), doxycycline (DOXY), and linezolid (LZD). Seventeen days after the surgery, he was transferred to our hospital; then, based on the antibiotic sensitivity profile, he was switched to CAM 800 mg oral, IPM/cilastatin (CS) 1 g IV, and IV AMK dosed with an adjusted trough concentration < 10 μg/mg (Fig. 1A). Additionally, from day 18, he was switched to hemodialysis (HD) with a temporary dialysis catheter. HD was performed every day because of his worsening congestive heart failure due to poor ultrafiltration from PD. On day 22, the PD catheter was removed. Due to the lack of improvement in inflammation based on blood test results, a CT scan was performed on day 40, which showed encapsulated ascites (Fig. 1B, C), and the drainage tube was inserted on day 45. The drainage fluid was a cloudy yellow–brown liquid. As inflammation improved with drainage, the drainage tube was removed on day 51, and IPM/CS was discontinued on day 64. He was switched to intramuscular (IM) injections of AMK and discharged on day 70. A few days after his discharge, AMK was discontinued due to new-onset hearing impairment that was probably drug-related. CAM was continued until six months after PD catheter removal, and after the discontinuation, no signs of relapse have been detected.

The second case is that of a 52-year-old man who had been undergoing PD for nephrosclerosis-induced end-stage renal failure since 2010. He had PD-related peritonitis and underwent simultaneous PD catheter removal and replacement in April 2022. In May 2022, he developed an exit-site infection and encapsulated ascites on CT (Fig. 2A, B). Additionally, as he also had severe abdominal pain and nausea that made him ineligible for PD, he was admitted to our hospital for close examination and treatment (day 0). He was diagnosed with PD-related peritonitis because of increased cell counts in the dialysate (total: 100/μL, polynuclear cells: 50/μL), and *Mycobacterium abscessus* subsp. *bolletii* was detected in both exit-site exudates and dialysate cultures (Fig. 2C). Based on CT findings, abdominal pain and nausea were considered to be due to adhesive bowel obstruction; therefore, decompression was performed by inserting a gastric tube while avoiding oral feeding. The PD catheter was removed on day 1 and he was switched to HD. On days 4 and 9, encapsulated ascites drainage was performed. Initially, antimicrobial treatment was started with 400 mg of oral CAM, IV
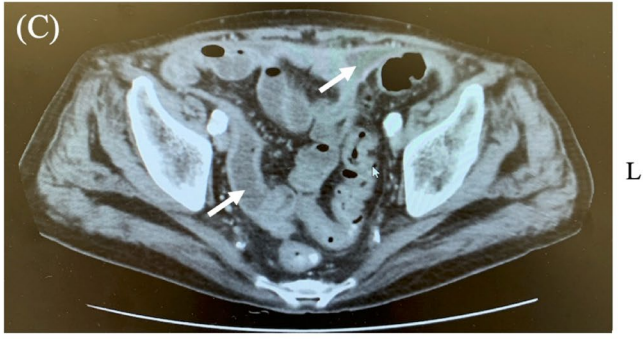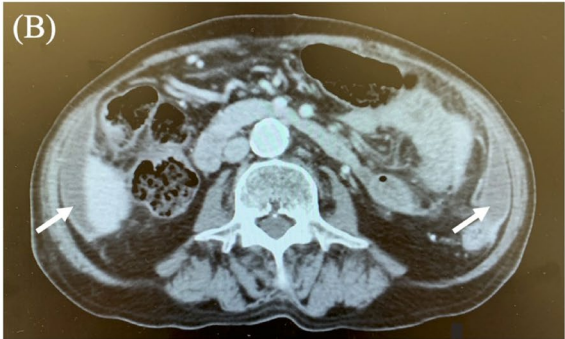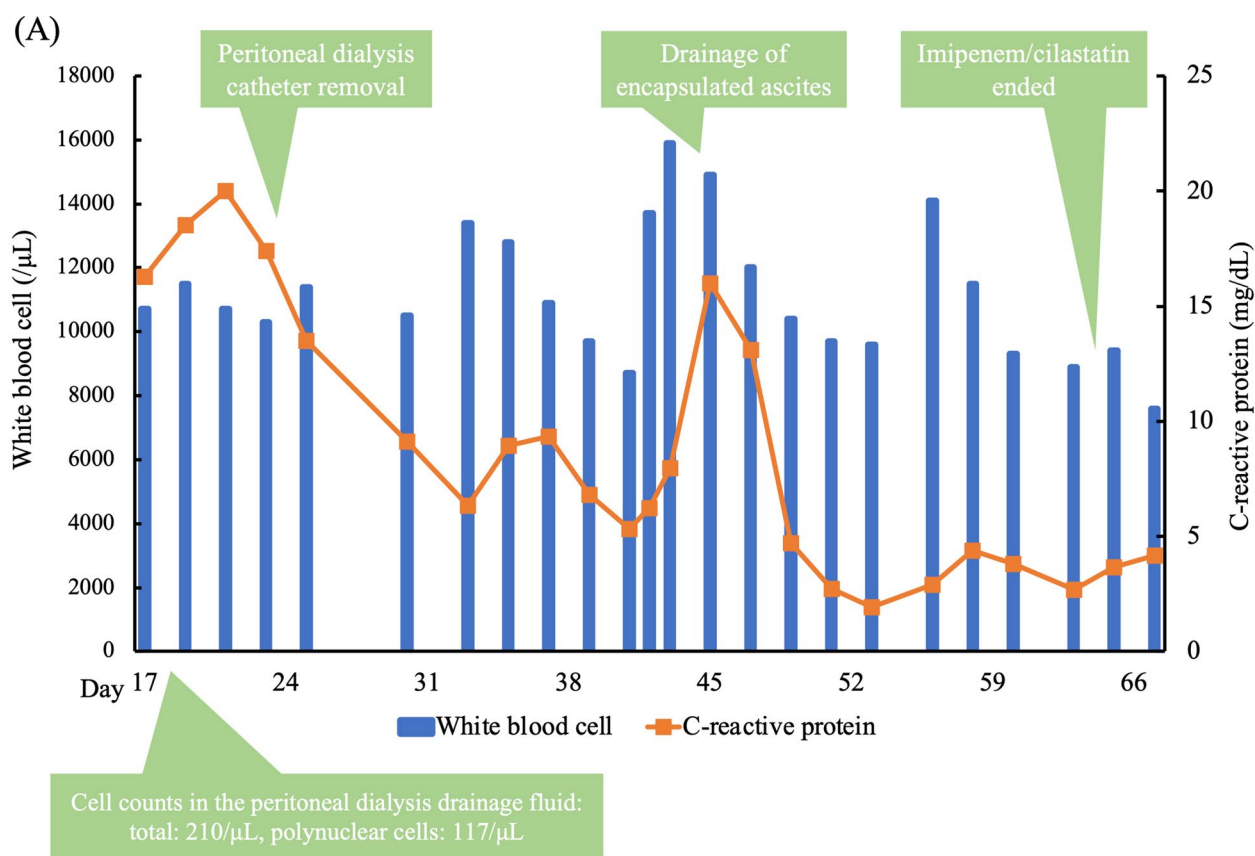
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Fig. 1 Clinical course (**A**) and radiological findings (**B** and **C**) of the first patient. Abdominal computed tomography revealed encapsulated ascites (arrows in **B** and **C**)

IPM/CS (1 g), and IV AMK, which was dosed with an adjusted trough concentration of < 10 μg/mg; however, CAM was changed to azithromycin (AZM) 250 mg IV on day 7 because of adhesive bowel obstruction, which made it difficult for him to take oral medication. Following these treatments, his blood test results and general condition improved, and he resumed oral feeding. As it was possible that he could have developed EPS due to persistent infection, he was temporarily transferred to another hospital where surgical dissection of the adhesions was possible on day 16. Although it was possible for him to develop EPS due to persistent infection, his abdominal symptoms diminished with gradual diet progression, and AZM 250 mg IV was switched to oral CAM (800 mg) on day 31. At this time, the culture results of the PD fluid collected in the early stages of hospitalization showed that *Mycobacterium abscessus* demonstrated high sensitivity to AMK, intermediate sensitivity to IPM and TOB, and resistance to MEPM, MFLX, ST, DOXY, and LZD. On day 55, antimicrobials

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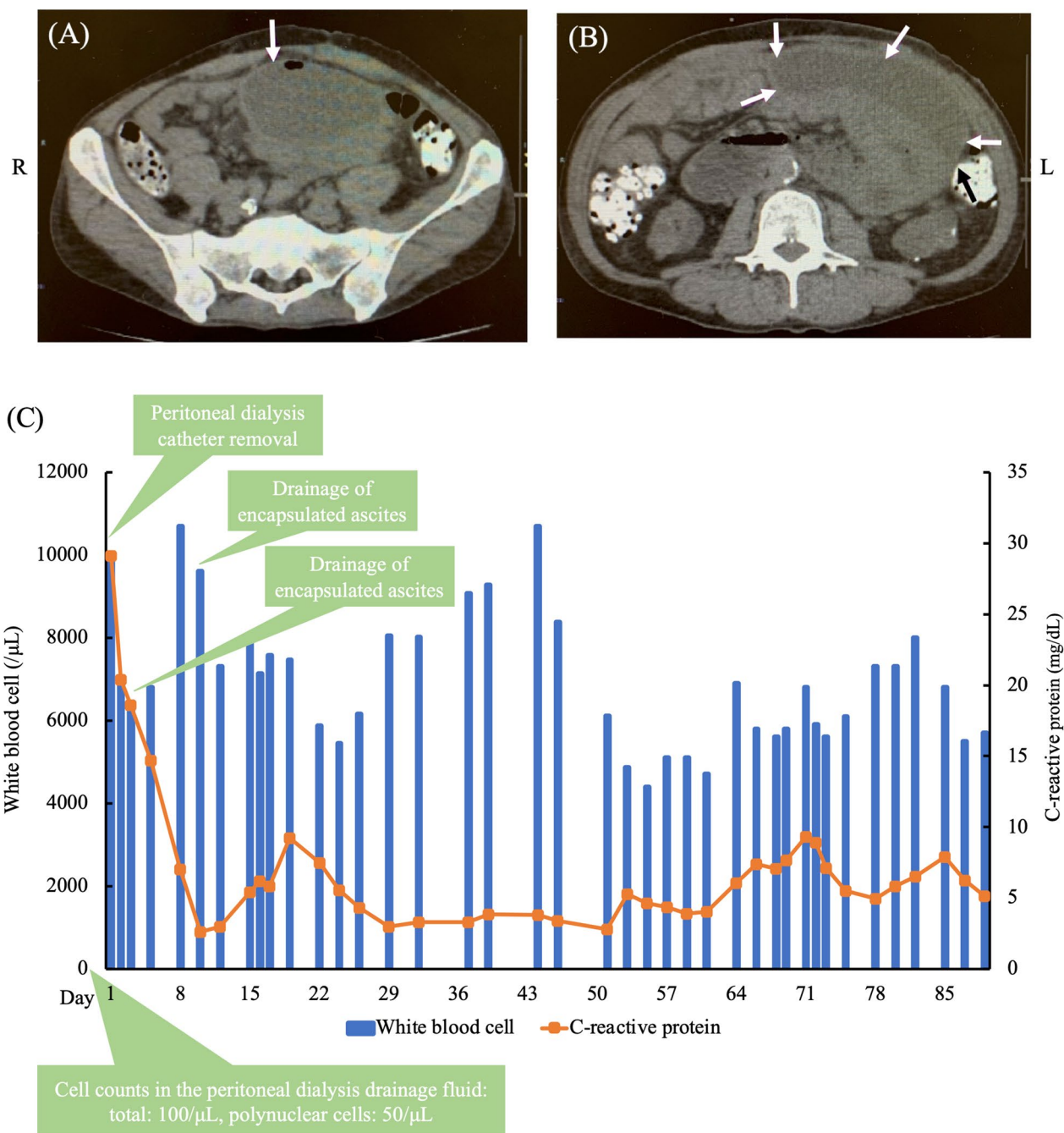
Fig. 2 Radiological findings (**A** and **B**) and clinical course (**C**) of the second patient. Abdominal computed tomography revealed encapsulated ascites (arrows in **A** and **B**) in addition to intestinal fluid-induced intestinal dilatation

were switched to oral sitafloxacin (STFX, 50 mg) every other day, oral clofazimine (CLF, 100 mg) every other day, with IV IPM/CS (1 g) and IV AMK. Subcutaneous debridement of the tunnel area was performed to decrease the bacterial population on day 72. As the patient's general condition had stabilized, IV IPM/CS was rounded up on day 84 and he was discharged on day 93. Six months after starting antimicrobial treatment, the patient is currently continuing antimicrobial treatment with IV AMK (500–600 mg) three times a week, oral STFX (50 mg), and oral CLF (100 mg).

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Discussion and conclusions

Herein, we present two cases of peritonitis caused by *Mycobacterium abscessus*, one by the *massilience* subsp and the other by the *bolletii* subsp. In both cases, the patient had encapsulated ascites, resulting in catheter removal and ascites drainage. The removal of the PD catheter is important in the treatment of NTM peritonitis, and treatment response is poor unless the catheter is removed. In three case reports, treatment failure, recurrent peritonitis, and death were observed in cases in which PD catheters were not removed [14–16]. Conversely, rapid improvement was observed after PD catheter removal [17]. In the current two cases, especially in case 1, inflammation also improved with PD catheter removal.

We conducted a systematic review of the literature focusing on PD-related peritonitis caused by NTM since May 2011 because there has already been a systematic review of the cases reported in PubMed up to April 2011 [17]. The number of cases with NTM peritonitis reported in PubMed from May 2011 to December 22, 2022, was 61, including our two cases (Tables 1 and 2) [6, 8–13, 18–44]. Therefore, combined with the 57 cases reported up to April 2011 [17], the number of cases with NTM peritonitis reported in PubMed to date is 118. In our review, 24 patients were females and 37 males, and their ages ranged from 2.7 years to 78 years; the geographical spread included reports from Asia (37 cases), Oceania (15 cases), North America (6 cases), and Europe (3 cases). *Mycobacterium abscessus* was the most commonly reported cause with 25 cases (41.0%), followed by *Mycobacterium fortuitum* with 12 cases (19.7%), in contrast to the previous review in which *Mycobacterium fortuitum* was the most common cause (38.6%) [17]. In our review, the catheter was completely removed in 41 cases but it was not removed in 20 cases, including 8 cases with simultaneous or bi-phasic catheter removal and re-insertion. As expected, death was observed in five (12.2%) of the cases with catheter removal, a lower mortality rate than that recorded among cases without catheter removal (4 cases, 20.0%). Additionally, all catheters were removed among cases who underwent simultaneous or bi-phasic catheter removal and re-insertion, indicating that these procedures were not recommended in NTM peritonitis. Therefore, we believe that the catheter should be removed once the causative organism of PD-related peritonitis is identified as NTM.

Next, we compared the characteristics between the deceased ($n=9$, 6 men) and non-deceased ($n=52$, 31 men) patients. The median age was comparable between the two groups [56 (25th–75th percentile, 50.5–71.5) and 54 (40–64.8) years, respectively]. PD vintage was recorded in 8 cases in the deceased and

Table 1 Demography, medical history, and clinical features of 61 cases of peritoneal dialysis-related peritonitis due to non-tuberculous mycobacterium reported from May 2011

Feature	No. of patients
Age, year	56 (45–64.5)
Sex	
Male/female	37/24 (60.7%/39.3%)
Country of origin of case report	
Asian countries	37 (60.7%)
Oceanian countries	15 (24.6%)
North American countries	6 (9.8%)
European countries	3 (4.9%)
Kidney disease	
Diabetic nephropathy	10 (16.4%)
Chronic glomerulonephritis	5 (8.2%)
IgA nephropathy	3 (4.9%)
Bilateral hypo/dysplastic kidneys	2 (3.3%)
Bilateral multicystic dysplastic kidneys	1 (1.6%)
Focal segmental glomerulosclerosis	1 (1.6%)
Crescentic mesangioproliferative glomerulonephritis	1 (1.6%)
Lupus nephritis	1 (1.6%)
Factor-H deficient hemolytic uremic syndrome	1 (1.6%)
Finnish-type congenital nephrotic syndrome	1 (1.6%)
Allograft failure after kidney transplant	1 (1.6%)
Graft versus host disease	1 (1.6%)
Unknown etiology	33 (54.1%)
Catheter removed	
Yes	41 (67.2%)
Simultaneous catheter removal and re-insertion	5 (8.2%)
Bi-phasic catheter removal and re-insertion	3 (4.9%)
No	12 (19.7%)
Outcome	
Recovery	52 (85.2%)
Death	9 (14.8%)

38 cases in the non-deceased group and was similar between the two groups [2 (1.5–3) and 1.5 (0.7–4) years, respectively]. Among the patients who died, the timing of PD catheter removal was only described in one case and the catheter was removed on day 7. Conversely, among the patients who survived, the timing of PD catheter removal was described in 26 cases; the catheters were removed at 11.5 (4.8–24.5) days. Furthermore, the antimicrobials used in each group are summarized in Table 3. Although the use of aminoglycosides and macrolides is comparable between the deceased and non-deceased groups, the rate of using new quinolones and beta-lactams seems higher in the non-deceased group than in the deceased group. Therefore, besides the most frequently used aminoglycosides

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Table 2 Isolates from 61 cases of peritoneal dialysis-associated peritonitis due to non-tuberculous mycobacterium reported from May 2011

Isolates	Frequency	Percent (%)
Mycobacterium abscessus	21	34.4
Mycobacterium abscessus subsp. *massiliense*	3	4.9
Mycobacterium abscessus subsp. *abscessus*	1	1.6
Mycobacterium abscessus subsp. *bolletii*	1	1.6
Mycobacterium fortuitum	11	18
Mycobacterium fortuitum subsp. *porcinum*	1	1.6
Mycobacterium chelonae	5	8.2
Mycobacterium avium complex	3	4.9
Mycobacterium wolinskyi	2	3.3
Mycobacterium smegmatis	2	3.3
Mycobacterium heckeshornense	1	1.6
Mycobacterium iranicum	1	1.6
Mycobacterium chlorophenolicum	1	1.6
Microbacterium paraoxydans	1	1.6
Mycobacterium chelonei	1	1.6
Mycobacterium hassiacum	1	1.6
Mycobacterium neoaurum	1	1.6
Unspecified	5	8.2
Total	61	100

Table 3 Antimicrobial use among the 9 deceased and 52 non-deceased patients with peritoneal dialysis-related peritonitis caused by a non-tuberculous mycobacterial infection reported since May 2011

	Deceased (*n* = 9)	Non-deceased (*n* = 52)
Aminoglycosides	8 (88.9%)	42 (82.7%)
Amikacin	6 (66.7%)	30 (57.7%)
Gentamicin	1 (11.1%)	9 (17.3%)
Tobramycin	1 (11.1%)	2 (3.8%)
Isepamicin	0 (0%)	2 (3.8%)
Macrolides	6 (66.7%)	36 (69.2%)
Clarithromycin	4 (44.4%)	32 (61.5%)
Azithromycin	2 (22.2%)	3 (5.8%)
Erythromycin	0 (0%)	1 (1.9%)
New quinolones	1 (11.1%)	25 (48.1%)
Ciprofloxacin	1 (11.1%)	12 (23.1%)
Moxifloxacin	0 (0%)	7 (13.5%)
Levofloxacin	0 (0%)	4 (7.7%)
Lascufloxacin	0 (0%)	1 (1.9%)
Sitafloxacin	0 (0%)	1 (1.9%)
Tetracyclines	1 (11.1%)	10 (19.2%)
Tigecycline	1 (11.1%)	4 (7.7%)
Minocycline	0 (0%)	3 (5.8%)
Doxycycline	0 (0%)	3 (5.8%)
Beta-lactams	2 (22.2%)	29 (55.8%)
Carbapenems		
Imipenem	1 (11.1%)	9 (17.3%)
Meropenem	0 (0%)	7 (13.5%)
Cephems		
Cephazolin	1 (11.1%)	3 (5.8%)
Cefoxitin	0 (0%)	4 (7.7%)
Cefmetazole	0 (0%)	1 (1.9%)
Penicillins		
Ticarcillin/clavulanic acid	0 (0%)	3 (5.8%)
Piperacillin/tazobactam	0 (0%)	1 (1.9%)
Flucloxacillin	0 (0%)	1 (1.9%)
Others		
Ethambutol	1 (11.1%)	2 (3.8%)
Rifampicin	1 (11.1%)	1 (1.9%)
Linezolid	0 (0%)	7 (13.5%)
Tedizolid	0 (0%)	1 (1.9%)
Vancomycin	0 (0%)	9 (17.3%)
Sulfamethoxazole–trimethoprim	0 (0%)	4 (7.7%)
Clofazimine	0 (0%)	3 (5.8%)
Metronidazole	0 (0%)	1 (1.9%)

and macrolides, the addition of new quinolones and/or beta-lactams based on the sensitivity of the organisms can yield successful treatment of patients with NTM peritonitis.

Including the current two cases, only five cases of NTM peritonitis with encapsulated ascites were reported [8–10], one of which was accompanied by EPS [9]. Interestingly, the organisms in all these cases were *Mycobacterium abscessus*. Additionally, three cases of EPS [9, 11, 12] and two cases of adhesive ileus [13] (including our second case) were reported. The culprit pathogens were *Mycobacterium fortuitum* in one case [11] and *Mycobacterium abscessus* in four cases [9, 12, 13]. These findings suggested that *Mycobacterium abscessus* tends to form encapsulated cavities and/or intestinal adhesions compared to other NTMs. Regarding the treatment, both of the current two cases underwent encapsulated ascites drainage, and rapid improvement of the persistent inflammation was observed after drainage. Additionally, among the three cases with encapsulated ascites that were previously reported, one recovered after drainage [10] and one case died without drainage [8]. Therefore, it is suggested that encapsulated ascites drainage was effective in improving inflammation; however, it is difficult to determine whether there was a direct causal relationship between drainage and inflammation abatement.

We hereby raise the issue that most reports did not describe the subtypes of *Mycobacterium abscessus*. *Mycobacterium abscessus* was first isolated from a knee

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abscess in 1952 [45]. Later, subspecies such as *Mycobacterium massiliense* and *Mycobacterium bolletii* were discovered. Following the consolidation and isolation of subspecies, genomic comparisons of several studies now show that *Mycobacterium abscessus* is composed of three subspecies: *abscessus*, *massilience*, and *bolletii* [46]. Different subspecies have different expression patterns of the *inducible erythromycin ribosome methyltransferase (erm) (41)* gene, which determines resistance to macrolides, and this results in different treatment outcomes for each subspecies [47]. *Mycobacterium abscessus* subsp. *massiliense* has been proposed to have a non-functional *erm (41)* gene, macrolide sensitivity, and good therapeutic outcome. In contrast, *Mycobacterium abscessus* subsp. *bolletii,* which has a functional *erm (41)* gene, is macrolide resistant [46, 47]. Indeed, in the present cases, *Mycobacterium abscessus* subsp. *massiliense* was sensitive to macrolides while *Mycobacterium abscessus* subsp. *bolletii* was resistant to them, making the choice of antimicrobial agent difficult. Therefore, there is a substantial need to clarify the subspecies of *Mycobacterium abscessus* to predict antimicrobial treatment response and further evaluate the clinical characteristics of each subspecies from a future accumulation of cases. To the best of our knowledge, this is the first case report of PD-related peritonitis caused by *Mycobacterium abscessus* subsp. *bolletii.*

In conclusion, we experienced two cases of PD-related peritonitis with encapsulated ascites due to *Mycobacterium abscessus* subsp. *masillience* and subsp. *bolletii*. In both cases, PD catheter removal and encapsulated ascites drainage might have improved inflammation and treatment outcomes. Additionally, *Mycobacterium abscessus* may tend to form encapsulated cavities and/or intestinal adhesions. However, further accumulation of cases clarifying the "subspecies" of *Mycobacterium abscessus* is necessary to confirm our hypothesis. Although no absolutes can be drawn from this case report, prompt catheter removal and administration of new quinolones and/or beta-lactams in addition to aminoglycosides and macrolides may be the key to achieve successful treatment of patients with NTM peritonitis.

Abbreviations

NTM	Non-tuberculous mycobacterium
PD	Peritoneal dialysis
CAM	Clarithromycin
MEPM	Meropenem
IV	Intravenous injection
AMK	Amikacin
IPM	Imipenem
TOB	Tobramycin
MFLX	Moxifloxacin
ST	Sulfamethoxazole-trimethoprim
DOXY	Doxycycline
LZD	Linezolid
CS	Cilastatin
HD	Hemodialysis
IM	Intramuscular
AZM	Azithromycin
EPS	Encapsulating peritoneal sclerosis
STFX	Sitafloxacin
CLF	Clofazimine
erm	Inducible erythromycin ribosome methyltransferase

Availability of data and materials
The patient results used and/or analyzed during the current case report are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
Not applicable (case report).

Consent for publication
Informed consent was obtained from the patients whose cases are reported in this article for publication of their personal information that appears in this manuscript.

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

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