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Acinetobacter ursingii peritonitis in a patient on peritoneal dialysis (PD): case report and literature review

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Abstract

Background Peritonitis is a frequent complication of PD that can lead to technique discontinuation and increase morbidity and mortality. It is caused mainly by gram-positive bacteria (up to 70%); however, gram-negative organisms usually have relatively poor outcomes. Among gram-negative bacteria, *Acinetobacter* is rare, especially *Acinetobacter ursingii*.

Case report We report the third case of PD peritonitis caused by *Acinetobacter ursingii*, treated with directed intraperitoneal antibiotics with good clinical response and favorable outcome.

Conclusion Although *Acinetobacter ursingii* is rare, it is potentially harmful because of its challenging identification and antibiotic resistance with therapeutic consequences, requiring at least two antibiotics and careful follow up. Keeping in mind that it is ubiquitous, careful technique, training/retraining seems highly recommended.

Keywords Peritoneal dialysis, Peritonitis, Gram-negative, Acinetobacter, Case report

Introduction

Peritoneal dialysis (PD) as a renal replacement therapy for end-stage renal disease has spread worldwide, as it is less expensive than hemodialysis and has comparable outcomes. Peritonitis is a common complication of PD and increases morbidity and mortality in this subset of patients, being one of the main reasons for PD catheter removal and hemodialysis switch. It is caused mainly by gram-positive bacteria, specifically staphylococci and streptococci (approximately 60–70%) [1], but gram-negative organisms usually have relatively poor outcomes.

The name "*Acinetobacter*" comes from the Greek word "*akinetos*", which means "unable to move", as these bacteria are not motile [2]. Acinetobacter is a group of gram-negative anaerobic coccobacilli, usually opportunistic and nonfermentative. This genus is broadly distributed in nature as a saprophytic bacteria in soil, sewage, water, skin, the gastrointestinal tract and the hospital environment [3]. They are recognized as nosocomial pathogens [4]. *Acinotebacter ursingii*, a species recently described in 2001, affects mainly immunocompromised and severely ill patients [5, 6]. The first cases of *A. ursingii* peritonitis in patients on PD were described in 2014 [7].

Acinetobacter is a type of bacteria that can cause peritonitis in patients undergoing peritoneal dialysis (PD). This bacterium is known for its high level of resistance

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to antibiotics, making it difficult to treat [7]. It can lead to various infections such as pneumonia, urinary tract infections, surgical wound site infections, endocarditis, meningitis, and bacteremia. The susceptibility of Acinetobacter to antibiotics varies widely depending on the geographic region.

These bacteria are able to survive in extreme conditions, including high temperatures, different pH levels, and exposure to high-alcohol detergents and other antiseptics that would normally inhibit bacterial growth [8].

The most common underlying conditions in patients who develop Acinetobacter peritonitis are diabetes mellitus (31%), chronic glomerulonephritis (31%), lupus (4%), and hypertension (4%). The symptoms of Acinetobacter peritonitis are similar to those caused by other microorganisms, including abdominal pain (85%), nausea/vomiting (31%), fever (27%), and diarrhea (15%) [7].

Acinetobacter, which is a member of the SPICE family (Serratia, Providencia, indole-positive Proteus/Acinetobacter/Morganella, Citrobacter, Enterobacter, or Hafnia), has an inherent ability to develop antibiotic resistance. Specifically, Acinetobacter strains possess genetically inducible beta-lactamases, which increases the risk of treatment failure and negative outcomes [7, 9].

The most commonly isolated Acinetobacter species from peritoneal fluid effluent are A. baumannii (54%), A. iwoffii (35%), A. ursingii, and A. junii (4%) [7].

Acinetobacter ursingii can cause peritonitis when wounds are contaminated or when a foreign body, such as a peritoneal dialysis catheter, is used. It is more common in immunocompromised patients or those on broad-spectrum antibiotics for long periods [10]. However, the development of Acinetobacter infection may not only be related to the host's immune system dysregulation but also to peritoneal dialysis exchange sterility failure, similar to staphylococcal infection. According to Chao et al., the most common identifiable causes of PD-related peritonitis due to Acinetobacter were a break in exchange sterility (19%) and translocation of the gastrointestinal microflora (19%), followed by exit site/tunnel infection (8%) [7].

We present the case of a 64-year-old woman with a history of chronic kidney disease of unknown etiology with 2 previous failed renal transplants who was receiving continuous ambulatory peritoneal dialysis (CAPD) and who attended the emergency department with abdominal pain and cloudy effluent, suggestive of peritonitis. During her admission, *Acinetobacter ursingii* was isolated from the PD fluid.

Case report

A 64-year-old woman with a history of end-stage chronic kidney disease of unknown etiology had her first deceased donor kidney transplant in 1989. She lost the first graft due to chronic allograft dysfunction and started CAPD in 1996, receiving a second graft in 2000. Her immunosuppression at that time consisted in OKT3 induction and triple therapy with tacrolimus, steroids and mycofenolate. Her second graft biopsy showed chronic inactive rejection and sclerosis, so she restarted CAPD in 2022 on incremental therapy (2 dwells) but required 4 exchanges a year and a half afterwards as she lost most of her residual renal function. Other relevant past medical history included hypertension with good control on one medication and dyslipemia, and her body mass index was 23.9. She was kept on low dose prednisolone (5 mg) as per our centre protocol to avoid rejection and graft intolerance syndrome. She experienced her first peritonitis episode sixteen months after the start of PD treatment due to *Staphylococcus salivarius*, which was treated on an outpatient basis and completely resolved after two weeks of intraperitoneal (IP) treatment with vancomycin. Moreover, she also had a *Staphylococcus aureus* catheter exit site infection treated with topical vancomycin + tobramycin and oral cloxacilline for 21 days, with an adequate response and complete negative cultures.

She was admitted to our unit with severe abdominal pain, fever (37.8 °C) and cloudy dialysate effluent. On physical examination, vital signs were within the normal range and her PD catheter exit site was perfect with no signs of infection. She presented with abdominal tenderness and cloudy peritoneal drainage. Blood analysis revealed a C-reactive protein (CRP) level of 159.5 mg/L, a procalcitonin level of 3.23 ng/mL, leukocytosis at 11,300/µl with 9,980/µl neutrophils, a platelet count of 456,000/µl, a hemoglobin level of 10 g/dL, a creatinine level of 8.48 mg/dL, a urea level of 95 mg/dL, and hyponatremia at 129 mmol/L without other relevant alterations. Sterile technique blood cultures, catheter exit site and PD fluid cultures were taken. The white cell count of the dialysate was 11,700/µL, with 88.6% polymorphonuclear cells (PMNs) and negative gram staining. Empirical treatment with IP ceftazidime and vancomycin was started, which was one day later changed to IP vancomycin and meropenem to extend the coverage of gram-negative microorganisms. Number of dwells did not require any changes and daily peritoneal fluid samples were collected during admission. Peritoneal fluid cultures were used to isolate highly susceptible *Acinetobacter ursingii* (antibiotic sensitivity testing is shown in Table 1) by mass spectrometry (MALDI-TOF), but blood cultures and exit site sample were negative. In accordance with the sensitivity of the culture, vancomycin was stopped, and tobramycin was added to meropenem. A 10-day course of IP tobramycin was completed, and the mixture was then switched to oral trimethoprim/sulfamethoxazole to prevent ototoxicity. The total duration of treatment was 21 days with double-directed antimicrobial therapy (meropenem plus

Table 1 Antibiotic sensitivity for *Acinetobacter ursingii*

Antibiotics	Susceptibility
Ampicillin/Sulbactam	S
Ceftazidime	S
Cefepime	S
Meropenem	S
Amikacin	S
Tobramycin	S
Ciprofloxacin	S
Levofloxacin	R
Colistin	S
Trimetoprim/Sulfamethoxazole	S

S: sensitive, R: resistant

trimethoprim/sulfamethoxazole). Additionally, a taurolidine citrate-urokinase catheter lock (Taurolock®, 5-mL vials, composed of taurolidine (1.35%), 4% citrate, and 25.000 IU urokinase) was initiated to avoid peritonitis relapse according to our protocol [10].

The patient presented clinical and biochemical improvement over the next few days, with a peritoneal fluid leukocyte count of 40 (<100 leu/μL) on the fourth day. Only in the first culture was Acinetobacter isolated, and the following cultures were sterile. She had a 5-day admission and was discharged as clinically fully recovered, with subsequent clinical and analytical outpatient follow-up presenting an adequate response.

Discussion

Acinetobacter species are widespread in nature, found in various environments such as soil, water, and dry surfaces due to their ability to survive on inanimate objects. As a result, Acinetobacter species are frequently found in hospital settings and are linked to the colonization of patient skin and healthcare workers [6, 11]. Despite their ubiquitous presence, infections caused by this pathogen are rare, likely due to the challenges in identifying it [12].

In a retrospective analysis of 456 cultures with Acinetobacter spp., it was found that A. ursingii infection occurred in 3.28% of cases, mainly in patients with prolonged ICU stay, immunocompromised individuals, those who underwent multiple procedures, or had invasive devices such as central venous catheters [12]. Acinetobacter has been shown to have the ability to colonize skin and intravascular devices. A. ursingii has been isolated in both blood and urine [2] but peritoneal infection by A. ursingii rarely occurs in human patients [6]. Chao CT et al., out of a total of 26 cases of peritoneal dialysis catheter-related peritonitis due to Acinetobacter recorded between 2000 and 2012, identified 1 case (4%) in which A. ursingii was isolated [7]. The treatment regime and follow-up were not described for A. ursingii. Two years later, Atas BD et al. reported another successfully treated patient with community-acquired A. ursingii peritonitis who received a 21-day course of two antimicrobial agents according to the susceptibility pattern with a complete resolution of peritonitis, similar to our case [9]. The schedule reported by Atas et al. was intravenous daptomycin and intraperitoneal ceftazidime. The sensitivity of the A. ursingii grown on culture was as follows: ceftazidime, with a minimum inhibitory concentration (MIC) of 26 μg/mL; piperacillin/tazobactam (24 μg/mL); amikacin (26 μg/mL); and ciprofloxacin (20 μg/mL). Daptomycin was discontinued, and amikacin was added when the antibiogram was received.

The identification of antibiotic susceptibility in A. ursingii reveals resistance to the most common cephalosporins (cephalothin, cefotaxime, cefixime, and moxalactam) and furans. However, they are susceptible to amoxicillin-clavulanate, ticarcillin-clavulanate, piperacillin-tazobactam, imipenem, aminoglycosides, ciprofloxacin, tetracycline, sulfamide, and colistin [6]. Compared with A. baumannii, which is the most common Acinetobacter species, A. ursingii may present lower rates of antimicrobial resistance with better outcomes. Furthermore, in a case-control study, patients infected with A. ursingii had much lower 28-day mortality than those infected with A. baumannii did (6% vs. 37%), even though multidrug resistance and inadequate initial treatment were equally likely in patients infected with either species, indicating lower virulence and, consequently, lower mortality rates [13].

To our knowledge, this is the third reported case of peritonitis due to A. ursingii. Our patient had a previous history of cumulative immunosuppression due to transplantation and was on low-dose steroids and both, combined with an immune system dysfunction due to renal disease, could have been a risk factor in our case. As she had a regular bowel habit without previous gastrointestinal symptoms, we believe the bacteria was saprophytic on her skin/environment. Keeping in mind that patients with advanced chronic kidney disease tend to have some degree of immunocompromise, frequent previous immunosuppression, and the presence of an external device (PD catheter) they have several known risk factors for infections by A. ursingii. Given its identification may be challenging and resistance to common antibiotics, we should be aware of its existence and alert if an Acinetobacter is found in PD fluid, carefully following up on peritonitis treatment response. Furthermore, periodic re-training of PD patients has proven beneficial in reducing peritonitis rates [14] and could reduce Acinetobacter infections.

In our experience, A. ursingii had good sensitivity to antibiotics, and the patient had a smooth and quick response to treatment needing a renal replacement therapy switch or PD catheter removal, neither PD therapy switch, and continued on CAPD.

Conclusion

Peritonitis associated with Acinetobacter species is infrequent but potentially harmful because of antibiotic resistance. Among them, A. ursingii is rare, although it is usually sensitive and has good outcomes. Given its challenging identification and resistance to common antibiotics, we should be aware and alert if an Acinetobacter is found in PD fluid, treating it with two antibiotics and carefully following up peritonitis treatment response. Since this bacteria is widespread, proper training, retraining, and hand washing are crucial to prevent this infection.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-024-03881-8.

Supplementary Material 1

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RHSB: patient management, manuscript writing and review. RSVA and MLQ: recorded data and wrote initial manuscript. CCT: nurse in charge of patient. MFL: reviewed manuscript.MERG: patient management, informed consent, reviewed manuscript.

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Data availability
Data of this case report is available from the corresponding author upon request. (R. Haridian Sosa Barrios, haridian.sosa@salud.madrid.org).

Declarations

Ethics approval and consent to participate
Not applicable.

Consent to publish
Informed consent was obtained from the patient for publication.

Competing interests
The authors declare no competing interests.

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