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CASE REPORT

Treating *Acinetobacter lwoffi* Peritonitis in a patient undergoing peritoneal dialysis

Julian Delicata, Paula Grech, Roberta Callus

Acinetobacter spp are increasingly recognized as an important cause of nosocomial infections, especially in relation with indwelling catheters. They are ubiquitous, gram negative bacilli, being normally found on the skin and oropharynx and are notorious for their broad antimicrobial resistance pattern.

Only a few cases of peritoneal dialysis-associated *Acinetobacter lwoffi* peritonitis have been reported with most of the affected patients being diabetic and/or immunosuppressed. Literature concerning the management of non-pseudomonas gram negative peritonitis is scarce.

We describe a case of a sixty-six year old gentleman with end stage kidney disease due to autosomal dominant polycystic kidney disease on Automated Peritoneal Dialysis who was successfully treated for *Acinetobacter lwoffi* peritonitis.

The patient did very well, did not require hospital admission and the peritoneal catheter remained in-situ.

Julian Delicata* MD
Department of Medicine
Nephrology Division
Mater Dei Hospital,
Msida, Malta
julian.delicata@gov.mt

Paula Grech MD
Department of Medicine
Mater Dei Hospital,
Msida, Malta

Roberta Callus MD
Department of Medicine
Division of Nephrology
Mater Dei Hospital,
Msida, Malta

*Corresponding author

Introduction

Peritonitis is accepted as being one of the major complications of peritoneal dialysis (PD), contributing, directly or indirectly, to the death of 16% of PD patients.[1-2] Moreover, it may lead to catheter removal, compromising dialysis access and significantly impacting quality of life.

Skin and nasopharynx gram negative commensals such as *Acinetobacter*, *Stenotrophomonas* and *Pseudomonas* contribute up to 5% of PD-associated peritonitis.[2-3] This is significant as gram-negative peritonitis episodes have been shown in various studies to have a worse mortality and a higher risk of catheter removal when compared to gram-positive ones.[2,4]

A literature review showed that data on *Acinetobacter spp* PD-associated peritonitis, and its subsequent management, is scarce. We know that these gram-negative bacillli are prone to cause multidrug resistant infections.[2,4] This makes their early detection and prompt eradication critical, especially since a foreign body, the peritoneal catheter, is involved.

It has been reported that almost half of Acinetobacter PD-associated peritonitis require hospitalization.[5] Acinetobacter PD-associated peritonitis seems to be more prevalent in immunosuppressed and/or diabetic patients.[5-6]

Case Presentation

Our case describes a sixty-six year old gentleman who had been on automated peritoneal dialysis (APD) for 2.5 years and presented to the Dialysis Unit with vague abdominal pains and turbid peritoneal fluid. He was a smoker of 40-pack years and his past medical history included hypertension (on three anti-hypertensive medications), end stage kidney disease (ESKD) secondary to autosomal dominant polycystic disease (ADPKD), and peripheral vascular disease. He did not have a history of previous peritonitis.

On clinical examination he was afebrile and had mild central abdominal tenderness with no rebound or guarding. There was no evidence of tunnel or exit site infection.

The white cell count (WCC) from his peritoneal fluid was 380/mm^3 with 68% neutrophils. An initial gram stain showed no bacteria. Serum white blood cell count was within range while the inflammatory marker C-reactive protein (CRP) was only mildly elevated at 10mg/L (normal range: 0-5mg/L).

Since the patient was not systemically unwell, it was decided to manage the patient in the community with daily visits to the dialysis unit for the administration of intraperitoneal antibiotics.

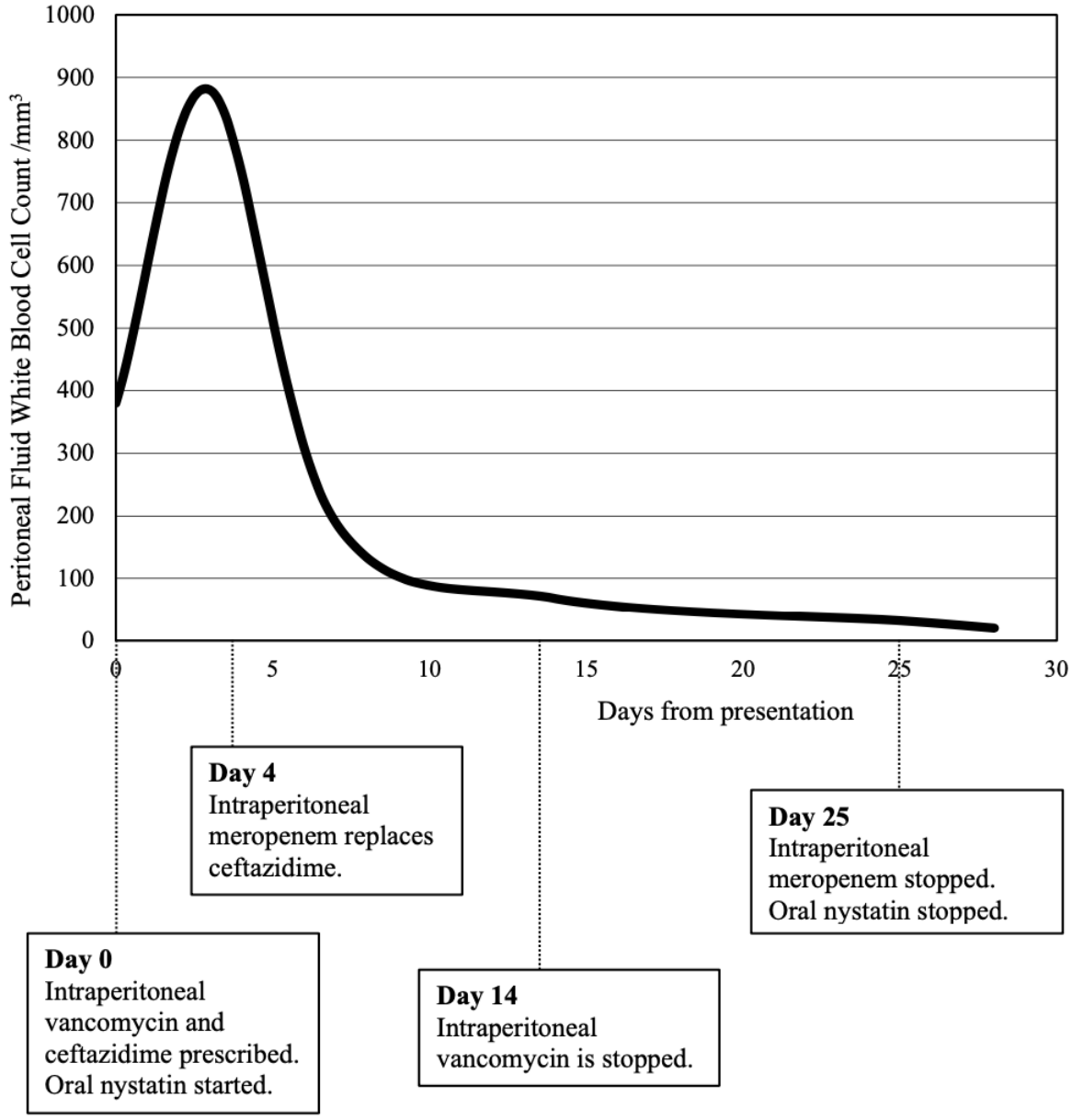
Once the diagnosis of peritonitis was confirmed, an intraperitoneal antibiotics regime including daily ceftazidime and vancomycin according to serum levels was started as per local protocol. Antifungal prophylaxis in the form of daily oral nystatin was also prescribed.

Despite antibiotics, the patient's symptoms however persisted. On day 3 of treatment, the peritoneal fluid WCC went up to 880/mm^3. At this stage, *Acinetobacter lwoffi* was grown in the culture medium obtained from the peritoneal fluid. This was resistant to 3rd generation cephalosporins. On the other hand, it was shown to be sensitive to gentamicin, ciprofloxacin, trimethroprim/sulfamethoxazole and the carbapenems meropenem and imipenem.

The intraperitoneal antibiotic regime was changed on day 4. Intraperitoneal meropenem at 1 gram daily replaced ceftazidime. No intravenous or oral antibiotics were given while the intraperitoneal vancomycin was continued.

The peritoneal white cell count dropped to less than 200/mm^3 after three days of this new regime and less than 100/mm^3 after a further week (Figure 1). Intraperitoneal meropenem was given for a total of 3 weeks, while vancomycin was stopped after 2 weeks.

Figure 1 The graph above illustrates the change in peritoneal fluid white cell count (WCC) from the first day of intraperitoneal antibiotics until the last. The intraperitoneal antibiotics regimes are illustrated to emphasize the affect intraperitoneal meropenem had on the peritoneal fluid WCC when introduced.



Outcome and follow-up

The patient's symptoms completely resolved within three days of starting treatment with intraperitoneal meropenem. The peritoneal catheter remained in situ and at no stage was dialysis efficiency compromised.

During a Renal Unit clinic visit 6 weeks after this event, the patient was symptomatically well and peritoneal fluid was clear with a normal WCC.

Discussion

Acinetobacter lwoffi is a nonfermentative aerobic gram-negative bacillus that normally inhabits the skin and oropharynx. Being so ubiquitous makes multi-drug resistance a problem. This may be attributed to various mechanisms, including beta-lactamases, permeability defects and aminoglycoside-modifying enzymes.[7]

Acinetobacter baumanni is the most common Acinetobacter spp cultured in PD-associated peritonitis as outlined in the literature.[5] Reports of the actual management of Acinetobacter spp peritonitis, and even more so *Acinetobacter lwoffi* are scarce. Furthermore *Acintetobacter spp* are not mentioned in the most recent 'ISPD Peritonitis Recommendations' (2016).[1]

It has been demonstrated from previous studies that a high proportion of patients diagnosed with acinetobacter spp peritonitis are diabetic and/or on immunosuppressive therapy. This was not so in this case report, however our patient did have a history of heavy smoking.

Based on the sensitivities obtained in our case, one may argue that an intraperitoneal aminoglycoside or fluoroquinolone may have sufficed, rather than a carbapenem. Literature on the management of non-pseudomonas gram negative peritonitis is scarce with the ISPD recommending (Grade 2c) at least a three-week course of effective antibiotics. In view of the risk of ototoxicity associated with aminoglycosides, such a prolonged course is controversial. Moreover, a previous computed tomography (CT) angiography had shown evidence of atherosclerosis throughout the iliac and femoral arteries and abdominal aorta. This, along with the significant history of hypertension, makes the use of fluoroquinolones contentious. Epidemiological studies have shown that the use of fluoroquinolones is linked to a higher risk of aortic aneurysm and dissection in populations with these clinical features.[8-9]

Our experience highlights the role of intraperitoneal carbapenems as an alternative to third generation cephalosporins when managing *Acinetobacter lwoffi* peritonitis. As referenced above, the use of alternative antibiotics such as aminoglycosides and fluoroquinolones presents a challenge in patients with ESKD due to possible complications.

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