Acinetobacter Peritonitis in CAPD
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Abstract: A 54 year old lady with chronic kidney disease stage 5 on CAPD presented to us with peritonitis. This was her second episode of peritonitis. The previous episode was a year before and was culture negative. The present culture grew Acinetobacter which was sensitive to aminoglycosides, fluoroquinolones, fourth generation cephalosporins and carbapenems. We treated her with Imipenem for 4 weeks and the peritonitis resolved. A month later she developed peritonitis again which was culture negative and carbapenem resistant requiring CAPD catheter removal for refractory peritonitis. Acinetobacter is an uncommon cause of gram negative peritonitis with a tendency for antibiotic resistance warranting catheter removal as highlighted by this case.

Keywords: Acinetobacter, Peritonitis, CAPD.

Case Report
A 54 year old lady a known case of ischaemic heart disease presented to us in May 2014 with acute breathlessness. She was detected to have chronic kidney disease stage 5 and Cardiorenal syndrome type 2. She was managed conservatively initially with diuretics. However a month later was initiated into hemodialysis initially due to recurrent pulmonary edema followed by CAPD catheter insertion. Dialysis was started with 3 bags 1.5% each and she was maintaining well. Seven months later she developed abdominal pain with a cloudy effluent and was detected to have peritonitis. Cultures were sent and she was empirically started on intraperitoneal antibiotics (I.P) cefazolin and ceftazidime. Cultures came as negative but she responded well to empirical treatment and cell counts decreased. Two weeks of I.P antibiotics were given and cell count normalised and she was asymptomatic.

She was maintaining well for another 12 months when she again developed peritonitis. Her CAPD cell counts were 11000 cells (90% neutrophils)/µL. Acinetobacter was isolated from cultures. The organism was sensitive to aminoglycosides, fluoroquinolones, fourth generation cephalosporins and carbapenems. We treated her with I.P Imipenem 250 mg to each bag. Cell counts reduced by day 3 and fluid cleared. We treated her for 4 weeks with I.P imipenem and cell counts normalised and she was asymptomatic.

2 weeks later she presented to us again with abdominal pain and a cloudy effluent. On evaluation the PD effluent cell count was 2800/µL with 60% neutrophils. A break in sterile precaution while doing the PD exchanges was documented as a new care giver with inadequate training had carried out the procedure. She was started on I.P antibiotics Imipenem 250
mg to each bag after sending cultures. The next day cell counts reduced to 1600 cells. We continued I.P imipenem but the cell counts remained at 800 cells with 70% neutrophils on day 5. PD fluid Culture was negative. We performed CAPD catheter removal in view of refractory peritonitis.

**Discussion**

Peritonitis is a serious infectious complication of CAPD resulting in 8-20% of infection related mortality and 30% of technique failure. Gram negative bacilli account for 30-40% of peritonitis episodes worldwide (1). Among Gram negative peritonitis, the acronym SPICE (Serratia, Providencia, indole-positive Proteus/Acinetobacter/Morganella, Citrobacter and Enterobacter) denotes a spectrum of organisms with an intrinsic tendency for antibiotic resistance. These pathogens have inducible beta-lactamase production during initial therapy, potentially leading to empirical antibiotic treatment failure, prolonged peritoneal damage and worse outcomes (1). Acinetobacter species are responsible for less than 20% peritonitis episodes of this SPICE group.

Acinetobacter species are pleomorphic nonfermenting aerobic bacilli which are ubiquitous and can be isolated from human sites such as the skin and respiratory tract (2). They were initially thought to be colonizers but now are considered as serious pathogens. Acinetobacter baumannii cause nosocomial outbreaks and are prone to antimicrobial resistance. Acinetobacter baumannii is the most common species to be isolated from CAPD effluent among cases of acinetobacter peritonitis followed by Acinetobacter iwoffii (2).

In contrast to non-CAPD-related Acinetobacter infections, which are commonly nosocomial, Acinetobacter peritonitis is usually community-acquired (3). A study by Chao et al from Taiwan who analysed Acinetobacter peritonitis episodes from their centre between 1985 to 2012 found 26 peritonitis episodes from 25 patients, accounting for 3.5% of all peritonitis episodes (1). The average age of the patients was 52 years and the PD vintage was 29 months. One third of their patients were diabetics. Other factors which predispose patients to Acinetobacter peritonitis were age > 60 years, anaemia (Hb < 7 g/dl), hypalbuminemia (< 3.5 g/dl), hypokalemia and diverticulosis (4). In a similar study by Lye et al from Singapore showed an incidence of Acinetobacter peritonitis of 10.7% and this was the most common cause of gram negative peritonitis in their study (5).

In the Chao et al study 62% presented with acinetobacter peritonitis as the first episode of peritonitis and in those who had peritonitis previously there was an average of 2 episodes before developing Acinetobacter infection. In contrast a study by Galvao et al showed Acinetobacter peritonitis to occur shortly after the treatment of another peritonitis episode, during a “vulnerable period” in which the host is immunocompromised (6). Chao et al showed the most common cause of acinetobacter peritonitis to be break in sterile techniques in 19% (as happened in our case), gut translocation in 19% and exit site infection in 8% (1). He proposed that break in sterile technique to be more important in the modern era than a dysregulated host immune system as proposed by Galvao (1).

The most common species were Acinetobacter baumannii (54%), followed by A. iwoffii (35%), A. ursingii (4%), and A. junii (4%). The presenting symptoms were abdominal pain (85%), vomiting (31%), fever (27%) and diarrhea (13%) in their study (1). The mean CAPD cell count was 5296 cells with 95% neutrophils (1).

The antibiotic susceptibility for Acinetobacter peritonitis and its outcomes varies between studies. In the Chao et al study the isolates were susceptible to aminoglycosides, fourth
generation cephalosporins and fluroquinolones while 10% showed resistance to third generation cephalosporins and anti pseudomonals (1). They conclude that fourth generation cephalosporins are the drug of choice followed by Aminoglycosides and that empiric use of ceftazidime may carry a 10-30% risk of treatment failure (1). The duration of treatment was an average of 17 days (1). The outcomes were good in their study with only 12% requiring catheter removal and no mortality. Lye et al study also showed good outcomes with only 1 patient requiring catheter removal. Majority of the patients responded to intraperitoneal gentamicin alone in their study (5,7).

While carbapenems (imipenem and meropenem) are the drugs traditionally thought to be most effective against Acinetobacter, a study by Zhang et al describing a case series of 7 patients with Acinetobacter peritonitis shows certain strains to be carbapenem and multidrug resistant (MDR) (4). MDR was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories. Compared to the Chao study which had good outcomes attributed to the good antibiotic susceptibility, the Zhang study showed a less favourable outcome with 3 of the 7 patients requiring catheter removal. The main reason being carbapenem resistance and MDR strains.

To conclude Acinetobacter though being an uncommon cause of gram negative peritonitis, can pose a serious threat warranting catheter removal especially when carbapenem and MDR strains are present. Patient and care giver education regarding maintenance of sterile technique and prevention of malnutrition seem vital in preventing this complication.

Reference