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Case Report

Continuous Ambulatory Peritoneal Dialysis (CAPD) associated peritonitis in a child: a rare case of peritonitis caused by sphingomonous paucimobilis

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Abstract

First identified in 1977, Sphingomonous Paucimobilis has emerged as an opportunistic human pathogen. It is primarily known to cause a range of mostly nosocomial, non-life-threatening infections that typically are easily treated by antibiotic therapy. Sources of its isolation linked to clinical disease include blood, spinal fluid and leg ulcers. It has also been reported as a rare cause of peritonitis in patients on continuous ambulatory peritoneal dialysis. We present a case of a child with peritonitis due to this organism. Clinical features, bacteriology and treatment option and response have been discussed.

Keywords: CAPD, Sphingomonous paucimobilis, Peritonitis.

Introduction

The invention of Continuous Ambulatory Peritoneal Dialysis (CAPD) in 1976 led to a significant increase in the utilization of the peritoneal dialysis (PD).¹ At present, over 130,000 patients are on CAPD worldwide, accounting for 15% of the total dialysis population.² The increasing popularity of CAPD is attributed to the convenience of home administration and similar and even superior survival in the first 2 to 3 years.^{2,3} In Pakistan, CAPD is being performed for the last few years with more than 100 patients in Karachi, Lahore, Peshawar and Dera Ismail Khan³ However, it is not as popular in Pakistan as compared to many other countries, because of the higher cost of dialysis solution and the relatively higher risk of infections, if infection control practices are compromised.⁴

Peritonitis is one of the major complications of PD, including CAPD. It is the main reason why patients switch from PD to haemodialysis (HD).^{5,6} The overwhelming majority of peritonitis cases are caused by pathogenic bacteria, with a small number of cases being caused by fungi, mostly *Candida* species.⁷ Databases from U.S.A and Canada show that gram positive organisms account for 62 and 61 percent of PD associated peritonitis, while gram negative bacteria are responsible for 21 and 24 percent of PD associated peritonitis in these countries respectively. Fungal peritonitis causes slightly less than 4 percent of PD associated peritonitis in both countries.⁸

We report a case of a child who developed CAPD associated peritonitis due to a rare organism, sphingomonous paucimobilis. The child was treated initially with

intraperitoneal amikacin but on developing impaired hearing was switched to IV meropenem to which he responded. To our knowledge this is the first case report from Pakistan and the second reported case of peritonitis due sphingomonous paucimobilis which was treated with IV carbapenem.

Case Report

Patient was a 3 ½ year old boy, known case of end-stage renal disease secondary to Prune Belly syndrome, who was on CAPD since November 2007. He presented on 14/01/09 with one-month history of a cloudy peritoneal effluent and decreased oral intake, oral ulcers and perineal rash for the past 5 days. His last admission was from 03/01/09 to 09/01/09 with complaints of altered peritoneal effluent, abdominal pain, and fever. He was diagnosed as spontaneous bacterial peritonitis in the last admission and treated with IV Ceftriaxone, to which the child responded clinically but the peritoneal effluent did not clear completely. His peritoneal D/R in the previous admission showed Glucose: 742mg/dl; Protein: 169mg/dl and white blood cell count: 500; Neutrophils: 60% and Lymphocytes: 40%. After he was discharged, his peritoneal fluid culture showed growth of sphingomonous paucimobilis. Because of the persistent cloudy dialysate and positive culture, patient was readmitted for treatment of peritonitis.

On examination the child was pale looking, mildly dehydrated and had multiple oral ulcers. His vitals were within normal limits. His abdomen was soft, non-tender, liver and spleen were not palpable and a CAPD catheter was in place. The rest of the examination was unremarkable.

Lab investigations in that admission showed a Haemoglobin(Hb)/Hematocrit: 8.4g/dl/26.4%; White blood cell count: 7.7; neutrophils(N):51% and Lymphocytes:32%; Platelets:644000.

The peritoneal effluent was sent for investigation. The gram stain of peritoneal smear revealed gram negative rods. The peritoneal detail report showed Glucose: 853mg/dl, Protein: 93mg/dl and WBC: 600; neutrophils: 80%; lymphocytes: 20%. The peritoneal fluid was inoculated on 5% sheep blood agar, Chocolate agar and MacConkeys agar. Cultures of the peritoneal dialysate on 5% sheep blood agar and chocolate agar grew small colonies at 24 hours of growth, at 37°C but not at 42°C, aerobically. The colonies were

pigmented an ochre-yellow. The MacConkeys agar showed no growth. The bacteria were gram negative, non-fermenting, and oxidase and catalase positive. The organisms were motile. The isolates were identified as *Sphingomonas paucimobilis* through use of the API 20NE (Bio-Mérieux). Sensitivities were checked by the disc diffusion method. The organism was sensitive to amikacin, meropenem, tetracycline and polymixin B. It was resistant to aztreonam, ceftazidime, gentamicin, ceftriaxone, ofloxacin, ciprofloxacin, cotrimoxazole and cefixime. Cultures were positive at 24 hours.

Hospital Course:

Based on the presentation and the investigations the boy was diagnosed of having moderate peritonitis with a single low virulence organism known as *Sphingomonas paucimobilis*. According to the culture and sensitivity report the boy was started on intraperitoneal amikacin (day/date). On day-1 a loading dose of 2mg/kg/bag of amikacin (250mg in 2L of dialysis solution) was given once. The plan was to keep a maintenance dose of 50mg/2L bag from day 2 onwards for 14 days, aiming for a trough level of 2mg/l.

A repeat peritoneal D/R on 22/01/09 showed few pus cells, Glucose: 144mg/d and Proteins: 90mg/dl. The culture of the same day reported no growth of the organism.

On day 4 of treatment with intraperitoneal amikacin, parents noticed that the boy had decreased hearing. There after amikacin was stopped and a BERA was obtained which showed absence of any reproducible response with a stimulus intensity of up to 105DB. Baseline hearing assessment, prior to the initiation of Amikacin was not available. The child was then placed on intravenous meropenem for 7days. The child recovered with clearing of dialysate fluid and he was discharged on 26/01/09.

Discussion

Sphingomonas paucimobilis is a nonfermentative, gram negative, motile bacterium that produces yellow-pigmented colonies on culture. It was first isolated in a human infection in 1977 and named *Pseudomonas paucimobilis*. It was reclassified in 1990 based upon phylogenetic data and renamed *Sphingomonas paucimobilis*.^{9,10} The natural habitat of this organism has not been totally defined but it is widely distributed in the environment especially in the water and soil. It has also been recovered from hospital environments including tap water, distilled water, nebulizers, respirators, dialysis fluid and other equipment.^{11,12}

Sphingomonas paucimobilis is an unusual pathogen for PD associated peritonitis. The first two cases of PD-associated peritonitis caused by *S. paucimobilis* (as *P. paucimobilis*) were reported by Glupczynski et al¹² in 1984.

Thereafter, 6 more cases have been reported with the latest by Dervisoglu et al in 2008.¹³ Peritonitis due to this organism tends to have a variable course as is evident from the previous case reports. It causes a mild to moderate peritonitis and patients at the time of presentation are clinically stable and often afebrile. Abdominal pain and cloudy dialysate are the usual presenting symptoms. Our patient presented with only a cloudy peritoneal effluent without any systemic signs of illness. It is interesting that the child seemingly responded to IV Ceftriaxone in the previous admission, even though the *sphingomonas* was resistant to ceftriaxone. Perhaps this was because of the indolent nature of organism, or perhaps there was a co-infection at that time with an organism that was treated by ceftriaxone.

The clinical outcomes in the reported cases so far have been variable, with some cases responding to antibiotics only while others eventually requiring catheter removal. Most of the latter patients had clinically improved with antibiotics but continued to have positive cultures. Our patient fortunately responded to antibiotics and became culture negative. The organism also tends to have unpredictable drug sensitivity. None of the case reports have shown any consistent pattern of sensitivity. Hence to date, no definitive guidelines exist for antimicrobial therapy for *Sphingomonas* infections. Potentially active agents include TMP-SMX, chloramphenicol, ciprofloxacin, and aminoglycosides.¹⁴ Imipenem alone and an aminoglycoside plus a third-generation cephalosporin have been suggested as suitable antibiotics for the treatment of susceptible *sphingomonas* infections.¹¹ In previously reported PD-associated peritonitis cases, antibiotic resistance during monotherapy was not noted. We used monotherapy with IV meropenem and were successful in eradicating the infection.

Conclusion

In summary, our report shows that *sphingomonas paucimobilis* can be a possible organism responsible for CAPD associated peritonitis. This organism is mostly resistant to the common antibiotics empirically used to treat peritonitis. If susceptible, IV meropenem can be considered for the treatment of this indolent but hard to treat infection.

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