Neisseria mucosa Peritonitis in the Setting of a Migrated Intrauterine Device

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Peritonitis is a major complication in peritoneal dialysis (PD) patients, often requiring a switch to hemodialysis (HD). Common sources of bacterial peritonitis are touch contamination and PD catheter–related infection. Intra-abdominal pathology is a less common cause of peritonitis in PD patients, and rarely is Neisseria mucosa *the causative organism.*

We present an uncommon case of N. mucosa *peritonitis in a 30-year-old African American female patient treated with nocturnal intermittent PD. The infection occurred in the setting of a translocated intrauterine contraceptive device (IUCD) in the infrahepatic region because of transmural migration. Our patient underwent laparoscopic removal of the IUCD and received empiric intraperitoneal (IP) vancomycin and intravenous ceftriaxone. After the isolate was identified as* N. mucosa*, her regimen was changed to IP ceftriaxone for a total of 21 days. Cell count after completion of antibiotics showed resolution of the peritonitis. The PD catheter was salvaged and transition to HD was avoided.*

Key words
Peritonitis, *Neisseria mucosa,* intrauterine devices

Introduction

Since the end of the 1990s, the overall rate of peritonitis has been seen to decline in peritoneal dialysis (PD) patients. That decline is attributed to multiple factors, such as the use of prophylactic antibiotics, aseptic PD catheter handling techniques, and innovations in PD technology (1). But despite the reduction in overall rates, peritonitis remains a major complication of PD and the main cause of PD technique failure (1,2). Common sources of bacterial peritonitis are touch contamination and PD catheter–related infection. Intra-abdominal pathology, such as migration of an intrauterine contraceptive device (IUCD) through the uterine wall, is a less-common cause of peritonitis in PD patients. However, it should be considered as a root cause in cases of complicated or atypical presentation (1,2).

Most peritonitis in PD patients is caused by common skin or nasal flora—for example, *Staphylococcus epidermidis,* other coagulase-negative staphylococci, and *S. aureus* (1–7). Peritonitis caused by *Neisseria mucosa* is rare in PD patients; only a few cases have been described, with none being in the setting of a migrated IUCD (2–6). *N. mucosa* are typically respiratory flora that have been considered nonpathogenic because the organisms lack virulence factors (2–6). However, with cases emerging (such as the case reported here), *N. mucosa* might be an important causative organism to consider in PD patients with peritonitis.

Case description

A 30-year-old African American female with a history of end-stage renal disease secondary to sickle cell disease and focal segmental glomerulosclerosis receiving PD was admitted with 3-day symptoms of fever, abdominal pain, and cloudy peritoneal fluid. She had been on PD for nearly 18 months with no previous episodes of peritonitis. Her surgical history was remarkable for an IUCD placed 3 months earlier. Her PD prescription was 4 nighttime exchanges with 2 L of 1.5% dextrose dialysate over a dwell time of 130 minutes each, and a last fill with 1.5 L of 1.5% dextrose dialysate over a dwell time of 2 – 3 hours. Her urine output was about 700 mL daily.

On the day of presentation, she had taken 1 dose of oral ciprofloxacin. On admission, she was afebrile and normotensive. Physical examination was significant for right upper quadrant abdominal pain with rigidity and guarding. The PD catheter site appeared perfect, with no surrounding erythema, warmth, or discharge. An initial peritoneal fluid sample showed nucleated

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cells (2566/mm^3), with 90% polymorphonuclear cells, consistent with a diagnosis of peritonitis. Computed tomography imaging of abdomen and pelvis with oral contrast showed trace right upper quadrant pneumoperitoneum and malposition of an IUCD in an extra-uterine location free within the peritoneal cavity inferior to the right lobe of the liver (Figure 1).

The patient was given 1 midday exchange with intraperitoneal (IP) vancomycin 1.2 g and cefepime 1 g and was taken to the operating room. The PD fluid was drained in the operating room, and laparoscopic removal of the IUCD was performed. Nighttime PD was held on postoperatively and resumed on postoperative day 1, initially with low-volume exchanges that were gradually increased.

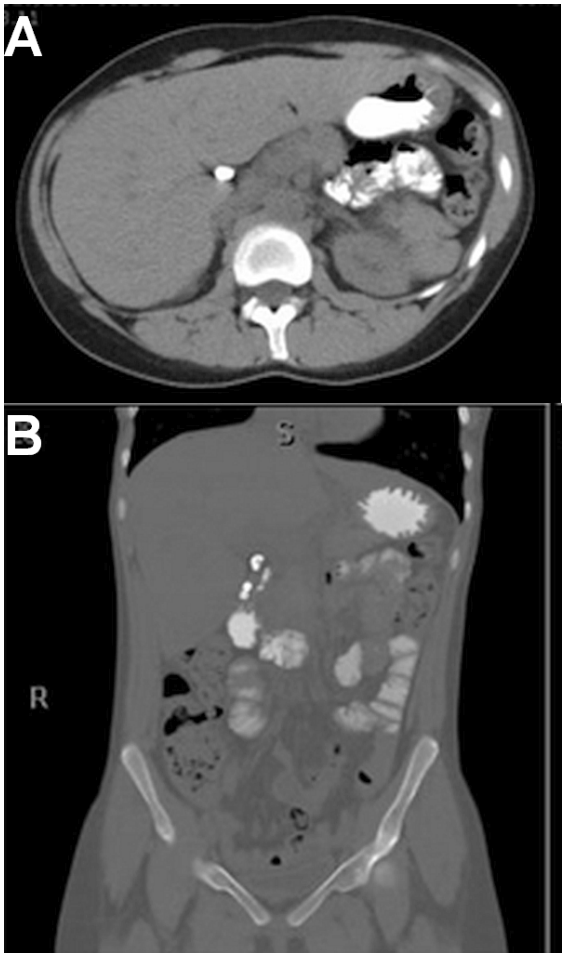

FIGURE 1 Computed tomography imaging of the abdomen with oral contrast, but without intravenous contrast. (A) The transverse and (B) coronal sections show an intrauterine contraceptive device just inferior to the liver and outside the uterus.

Culture of the PD fluid revealed *N. mucosa* as the causative organism, with sensitivity to cefazolin, clindamycin, levofloxacin, and penicillin. The patient's antibiotic regimen was switched from empiric vancomycin and cefepime to intravenous ceftriaxone 2 g every 24 hours. The infectious disease service was consulted, and in addition to the intravenous ceftriaxone, the patient was started on oral levofloxacin with a loading dose of 500 mg in the first 24 hours, followed by 250 mg every 48 hours and oral metronidazole 500 mg every 8 hours. After 3 days of admission, repeat culture of the PD fluid showed that the nucleated cell count had improved to 302/mm^3 with 50% polymorphonuclear cells. After 6 days of admission, the count had improved to 101/mm^3, with 21% polymorphonuclear cells, consistent with resolution of peritonitis. Mild leukocytosis that had been present on the day of admission (white blood cell count: 11.34×10^9/L) had resolved by the following day.

The patient was discharged in stable condition on day 7 after PD fluid cultures showed no growth. The initial discharge plan was to continue oral levofloxacin and metronidazole for a total treatment duration of 14 days. However, upon discharge, the regimen was changed to IP ceftriaxone for 21 days. The PD catheter did not have to be removed. To date, the patient has had no further signs or symptoms of peritonitis, and she is continuing with her home PD regimen.

Discussion

Notwithstanding improvements in aseptic technique and advancements in PD technology, peritonitis remains the leading complication of this dialysis modality (1–6). Peritonitis in PD patients can be devastating to their lifestyle on account of having to switch to hemodialysis. Although peritonitis is most commonly caused by non-sterile technique or PD catheter–related infection, less common causes such as intra-abdominal pathology should also be kept in mind (1,2). We present a rare case of *N. mucosa* peritonitis in the setting of a migrated IUCD.

N. mucosa, a gram-negative diplococcus, is typically a benign oropharynx or nasopharynx commensal organism that has rarely been considered a cause of infection, especially in immunocompetent individuals (2–7). However, *N. mucosa* is now increasingly

being found as a source of peritonitis in PD patients (1–6). *N. mucosa* lacks the typical virulence factors of pathogenic *Neisseria* species (7). It is nonmotile and lives in warm, moist, and humid conditions, such as those that can be found in the genitourinary tract (7). The case report by Osses *et al.* (7) describes *N. mucosa* as the causative agent of a urinary tract infection. Pathogenesis by *N. mucosa* has been described to include an ability to evade antibody opsonization, to replicate inside neutrophils, and to make use of porins that assist with transport into host cells with avoidance of complement activation (7). *N. mucosa* can be transmitted in nasopharyngeal droplets (7).

In our case, we believe that the migrated IUCD was the likely source of the *N. mucosa* peritonitis. In our patient, laparoscopic removal of the IUCD was the mainstay of treatment, together with antibiotics. Our patient was successfully treated with IP ceftriaxone. Guidelines for the treatment of *N. mucosa* peritonitis are not established, and case reports have described success with the use of antibiotics such as vancomycin, cefazolin, ceftriaxone, and ciprofloxacin, both intravenous and IP (2–6). No indication of a need for PD catheter removal was noted in our patient. We were able to salvage the PD catheter and avoid a transition to hemodialysis.

Summary

We highlight a unique case of *N. mucosa* peritonitis in a PD patient in the setting of a migrated IUCD. We highlight the importance of recognizing intra-abdominal pathology as a potential cause of peritonitis in PD patients and of making an effort to salvage the PD catheter when indicated. We also highlight the importance of considering *N. mucosa* as a causative organism of gram-negative peritonitis, and we add to the calls for treatment guidelines to be established, given the increasing episodes of this infection in PD patients.

Disclosures

We understand that *Advances in Peritoneal Dialysis* requires disclosure of any conflicts of interest, and we declare that we have no conflicts to disclose.

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