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From the Clinic

Peritonitis from *Rothia mucilaginosa* in a chronic peritoneal dialysis patient

Introduction

Rothia mucilaginosa is a Gram-positive, coagulase-negative, encapsulated, non-spore-forming coccus considered part of the normal flora of the upper airway in humans. It is considered to have low virulence, but has been reported to cause infections in various organs in immunocompromised patients. Only two cases of *R. mucilaginosa* peritonitis in chronic peritoneal dialysis (PD) patients have been reported previously, and we report the third case here in a patient with human immunodeficiency virus, who was successfully treated with intraperitoneal vancomycin.

Case report

Hemodialysis was started in a 60-year-old Haitian man with end-stage renal disease from human immunodeficiency virus-associated nephropathy in July 2000 and was converted to PD in June 2001. He had four episodes of peritonitis since 2004: culture-negative episodes in 2004 and 2006 treated with intraperitoneal cefazolin and gentamicin, coagulase-negative staphylococcal peritonitis in 2007 treated again with intraperitoneal cefazolin and gentamicin and another episode of culture-negative peritonitis in 2010 treated with intraperitoneal gentamicin and vancomycin. Each episode resolved without the need for catheter removal; in fact, he has retained his original PD catheter for 11 years. The rest of his past medical history includes anemia, bilateral renal cysts, severe gastroesophageal reflux requiring fundoplication, hiatal hernia, transient cerebrovascular ischemic attack, neurogenic bladder, benign prostatic hypertrophy requiring transurethral resection of the prostate and colon polyp.

In August 2012, he experienced a 2-week history of diffuse abdominal pain, which he attributed to acid reflux. He instituted antacid therapy at home, without response. He was maintained on an automated, intermittent PD program: three 2-L exchanges of 1.5% dextrose-containing PD fluid over 9 h, with a last dwell of 2 L for 4 h, followed by a dry remainder of the day. Medications at that time included lamivudine 100 mg daily, ritonavir 100 mg daily, tenofovir 300 mg once weekly, atazanavir 300 mg daily, calcium acetate 667 mg with meals, cinacalcet 30 mg daily, calcitriol 0.5 mcg daily, cyclobenzaprine 10 mg daily, folic acid 1 mg daily, 1 renal vitamin daily, vitamin B daily, pyridoxine 500 mg daily, metoprolol 50 mg twice daily, amlodipine 10 mg daily and Lansoprazole 30 mg twice daily.

When the patient presented to the home dialysis clinic, PD effluent was noted to be cloudy and contained fibrin. He had low-grade fever of 100°F, blood pressure was 140/88 and heart rate was 78 beats per minute. The exit site was clean, and the abdomen was diffusely tender to palpation. The rest of his physical examination was unremarkable. Laboratory evaluation of the PD fluid revealed 3589 nucleated cells per mm^3 with 89% neutrophils. Other laboratory investigation revealed: peripheral white blood cell count 6×10^{9}/L, hemoglobin 11 g/dL, blood urea nitrogen concentration 98 mg/dL, serum creatinine concentration 14.8 mg/dL and serum albumin concentration 2.7 g/dL. His CD4 count was 406 and human immunodeficiency viral load was not detected.

He was empirically treated with gentamicin and vancomycin intraperitoneally while culture and sensitivities were pending. Culture results later identified *R. mucilaginosa* as the sole pathogen with sensitivity to penicillin and levaquin. Intraperitoneal vancomycin alone was used to complete a 2-week course of treatment with resolution of cloudy PD fluid and abdominal pain. The patient denied recent dental procedures and reported adherence with his PD prescription and sterile technique. Since then, he has had no further peritonitis or new medical problems.

Discussion

Rothia mucilaginosa (formerly known as *Stomatococcus mucilaginosus*) is a Gram-positive, coagulase-negative, encapsulated, non-spore-forming coccus considered part of the commensal flora of the oral cavity and upper respiratory tract in humans. Infection in humans was first reported as endocarditis in 1978 [1] and later in other infectious processes in immunocompromised patients: meningitis, catheter-related infections, endophthalmitis, vertebral osteomyelitis, prosthetic hip joint infection and granulomatous dermatitis. These patients were neutropenic or possessed risk factors for infection including profound immunocompromised states, repeated exposure to broad-spectrum antibiotics, intravenous drug use, chronic indwelling catheters or abnormal cardiac valves. Hematogenous spread of *R. mucilaginosa* from the sites of dental caries and dental manipulation to cardiac valves has been suspected. The human immunodeficiency virus is the obvious predisposition in our PD patient. Transmission presumably resulted from hematogenous spread from the oral cavity, although our patient did not have unusually poor dentition or a prior dental procedure.

Two additional species of the genus *Rothia* have been described as human pathogens. *Rothia dentocariosa* is the most frequently isolated species, and *R. aeria*, first isolated from the Russian space laboratory Mir in 1997, has been implicated in cases of infective endocarditis, neck abscess, sepsis, native joint infection, cavitary lung lesion and acute bronchitis. There have been four case reports in the literature of *R. dentocariosa* as the peritoneal fluid pathogen in PD patients, the first reported in 1999 [2]. Three of these cases had recurring and relapsing courses. Infections due to *Rothia* species are likely to be underreported, since they are not routinely included in the databases of automated microbiologic identification systems. Nevertheless, they are generally susceptible to penicillin, ampicillin, cefotaxime, imipenem, rifampin and glycopeptides.

Infection by *R. mucilaginosa* in PD fluid has only been described previously in two case reports. One patient, who had recently lost a renal allograft and was still on immunosuppressive therapy, developed coagulase-negative

staphylococcus bacteremia and culture-negative peritonitis treated with ceftriaxone and ofloxacin with removal of PD catheter. Two months later, she was back on PD and again had another episode of peritonitis, which grew *R. mucilaginosa*. It responded well to intraperitoneal vancomycin with resolution of symptoms in 2 days [3]. The second patient had peritonitis from *Acinetobacter lwoffii* treated with a fluoroquinolone a few weeks prior to peritonitis from *R. mucilaginosa* [4].

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