A Case of Suppurative Peritonitis by a Commensal Oral Organism, *Kingella denitrificans*, in an Adult Peritoneal Dialysis Patient

CASE REPORT

A 57-year-old female with end-stage kidney disease secondary to autosomal dominant polycystic kidney disease is admitted with a 3-day history of worsening abdominal pain. She has been on peritoneal dialysis (PD) therapy with the same catheter for the last 7 years with no previous episodes of peritonitis. She relates no prior history of similar abdominal pain. Past medical history is negative for diverticulitis or previous abdominal surgery and there was no indication for previous antibiotic prophylaxis. She uses a cycler for PD with 6 L of 2.5 % delfrex solution as 3 exchanges through the night and 2, 2-L manual exchanges in the afternoon with 2.5% delfrex solution. The PD exchanges were clear at the time of onset of her abdominal pain with good flow and no fibrin noted. She developed worsening abdominal pain despite therapy with tramadol causing her to present to the emergency room (ER). At the time of arrival in the ER, the PD bag from home was noted to be slightly cloudy but without fibrin or blood. A CAT scan of her abdomen was read as demonstrating a partial small bowel obstruction. There was no evidence of diverticulitis. Surgery consult felt that the patient had an ileus and recommended keeping her "nothing by mouth" (NPO) and with continuous nasogastric (NG) tube suction. The admitting white blood cell count (WBC) was 4.3 thousand/cmm and no shift. The chemistries were appropriate for a PD patient with normal potassium, acid base status, and liver function tests. After pan cultures were obtained, the ER physician initiated therapy with intravenous vancomycin and cefepime. Nephrology was then consulted. She was continued on continuous ambulatory PD (CAPD) while in the hospital with 5 manual exchanges performed utilizing 1.5% delfrex solution, 2.5 L per exchange. No intraperitoneal therapy was initiated at this time because she received parenteral therapy already in the ER. Peritoneal fluid analysis obtained from her first exchange after admission demonstrated 333 white cells, with 89% neutrophils and negative Gram stain. It was noted subsequently that the PD fluid became progressively cloudier with fibrin over the first 24 hours. Heparin could not be added into the PD fluid because of the history of heparin-induced thrombocytopenia (HIT). The PD exchange frequency was increased to 6 times per day. She remained afebrile with diffuse abdominal pain. The broth culture was subsequently positive for gram-negative *diplococci* on the Gram stain and was inoculated on chocolate agar media. Biochemical analysis of the chocolate agar colony revealed the colony to be positive for glucose, nitrate, and proline p-nitroanilide. It was negative for urea and the organism was identified as *Kingella dentifrices*. Infectious disease had been consulted when the broth culture was noted to be positive. The ileus improved with conservative therapy. Antibiotics

were narrowed to Ceftriaxone 2 grams intravenously daily as per infectious disease. Repeat PD fluid analysis after 4 days of antibiotics showed an increase in white cells to 1,380, with 47% neutrophils and on inspection the fluid was noted to be cloudier. The patient's abdominal pain continued to worsen progressively. After discussion with the infectious disease team, it was felt that the catheter should be removed due to worsening symptoms despite appropriate antibiotic therapy. She subsequently had her PD catheter removed and was transitioned to intermittent hemodialysis with a tunneled right IJ catheter. Peritoneal dialysis catheter cultures were negative with no evidence of any biofilm. On further questioning, after the peritonitis was identified to be secondary to *Kingella*, the patient did note a dry hacking cough for a month prior to admission. She felt this was related to valsartan as it improved after discontinuation of the medication. She also mentioned there were several occasions over last month that she performed her PD exchanges without a facemask while she had the cough. She also denied any recent dental procedures. Peritoneal dialysis catheter cultures subsequently came back negative. After completing 2 weeks of intravenous ceftriaxone, she was changed to oral ciprofloxacin 500 mg daily as per infectious disease and recommended to complete 4 more weeks of antibiotics for a total duration of 6 weeks of therapy.

CASE REVIEW

We are reporting the first documented case of PD peritonitis due to *Kingella denitrificans* in adults. *Kingella* is a slow-growing, fastidious, aerobic coccoid to medium-sized gram-negative rod. It belongs to the family *Neisseriaceae*, which also includes the genera *Neisseria, Moraxella, Acinetobacter*, and *Oligella*. There are 4 known species: *Kingella kingae, Kingella indologenes, Kingella denitrificans*, and *Kingella oralis*. Of these, *Kingella kingae* has been found to be the most common human pathogen. *Kingella kingae* and *Kingella denitrificans* are oxidase-positive non-motile organisms that are hemolytic when grown on blood agar. Type 4 pili are adherence factors, which are important as virulence determinants in bacteria related to *Kingella spp*. These pili are composed of pilin subunits with N-methylphenylalanine as the first residue of the mature protein, a highly conserved amino terminus, and a disulfide bridge near the carboxy terminus (1).

Kingella denitrificans is one of several species which are colistin-resistant and may be routinely isolated on selective medium for *N. gonorrhoeae*. Colonies of *K. denitrificans* are usually glistening and smooth in consistency (2). Although cells of *K. denitrificans* are coccobacilli, the cell morphology of some isolates may appear to be diplococci and very similar in appearance to *N. gonorrhoeae*. In contrast to *Neisseria spp., K. denitrificans* isolates are catalase- and superoxol-negative. *Neisseria gonorrhoeae* and *K. denitrificans* may, however, be confused because isolates of both species produce acid from glucose in acid production tests and hydroxylprolylaminopeptidase in enzyme substrate tests. Cross-reactions have also been noted with isolates of *K. denitrificans* in coagglutination serologic tests for the identification of *N. gonorrhoeae* (3). The nitrate test permits differentiation between *N. gonorrhoeae* (nitrate-negative) and *K. denitrificans* (nitrate-positive) (4). *Kingella* is a very slow-growing, fastidious organism.

Kingella is a component of the so-called HACEK group (*Haemophilus aphrophilus* and *H. parainfluenzae, Actinobacillus, Cardiobacterium, Eikenella*, and *Kingella*). *Kingella kingae* (previously known as *Moraxella kingae*) was the first named species of a new genus proposed in 1976 by Henriksen and Bøvre (5). In the same year Snell and Lapage (6) proposed the name *Kingella denitrificans* for a group of organisms described by Hollis *et al.* under the designation TM- 1 (7). *Kingella denitrificans* are commensals of the human respiratory tract (7), and they occasionally cause serious infections. The organism may enter the circulation with minor oral trauma such as tooth brushing. *Kingella denitrificans* has been implicated in cases of endocarditis (8–11), bacteremia (8), empyema (11), and corneal ulcers (12), whereas *Kingella kingae* causes bacteremia, septic arthritis, spondylitis and endocarditis (13). They are susceptible to penicillin, ampicillin, and erythromycin.

A review of the literature suggests that there is 1 documented case of *Kingella denitrificans* infection causing peritonitis in a child (14). It was 1 of the microbes isolated from the peritoneal fluid of a 10-year-old child diagnosed with polymicrobial peritonitis following a puncture of dialysis tubing by a pet dog. Canine oral microfloras isolated from this patient on PD were *Kingella denitrificans*, *Actinomycetes* species, *Capnocytophaga cynodegmi* and *Mycoplasma edwardii*. A reported case in the literature of endocarditis from this organism suggests a systemic bacterial origin of the infection. Our patient did not have any other oropharyngeal complaints or lesions. Sub occlusion was not present as with conservative therapy and her ileus resolved with no clinical evidence of a partial obstruction. A review of the literature has not specially mentioned the virulence of the organism, as there are only a few case reports with the organism primarily causing endocarditis. A few of the endocarditis patients required valve replacement surgery, while others were treated medically with intravenous antibiotics alone. A literature review does mention the necessity for a long duration of antibiotic therapy, between 28 – 52 days, depending on the response (15).

The only report of *in vitro* sensitivity of *Kingella* species to antibiotics is contained in a Swedish report of 13 strains of *Kingella kingae* shown to be sensitive to beta lactams, cephalosporins and aminoglycosides; 1 strain was resistant to erythromycin and all were resistant to clindamycin (16). No *in vitro* sensitivity data exist for *Kingella denitrificans* and treatment protocols are based on extrapolation from the findings of *Kingella kingae*. On the basis of the experience of Kahn *et al.* (9), and the recommendations of Geraci and Wilson (17), intravenous ampicillin in a dose of 12 g initial treatment is adequate for endocarditis even though by convention a combination with an aminoglycoside is often used. Cefotaxime may be used for people who are allergic to ampicillin, while those who have cross allergy may benefit from the quinolones (ciprofloxacin). Since there is no documented case of PD peritonitis

due to *Kingella* species, nothing has been mentioned about using intraperitoneal antibiotics with PD fluid.

Although our patient attempted to maintain strict sterile precautions and took appropriate care of her PD catheter site, 1 possibility may be that she contaminated the PD set with her oral secretions while coughing. She did admit that maybe 1 – 2 times she failed to use a facemask. This is a hypothesis only, as there is no definitive evidence of causality in our case. There is also no evidence in our case of the organism traversing the gastrointestinal tract to gain entrance into the peritoneal cavity. The possibility of causation via blood borne entry from the oral cavity or aerosol contamination seems to be the most plausible. Figueiredo *et al.* (18) suggested that use of a face mask during CAPD bag exchange gives no extra protection against peritoneal contamination. More importantly, patients must be educated to perform the connect/disconnect operation carefully, and to strictly observe accidental contamination of the hands. But the description was limited to a single renal unit with a seemingly small number of patients. Also, the patients were healthy and not sick like our patient who was coughing intermittently. This stresses the importance of strict hand hygiene and using a mask, especially if sick, before doing PD exchanges. She will be continued on long-term antibiotics for 6 weeks, her PD catheter was removed, and she will undergo intermittent hemodialysis with a tunneled catheter. In retrospect, she may have had a better outcome with intraperitoneal antibiotics. After completing her 42 days of antibiotics and close follow-up with regard to the clinical response, she will be re-evaluated regarding timing for the placement of a new PD catheter.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

Nelson Kopyt*
Anshul Kumar
Vibha Agrawal

Lehigh Valley Health Network
Allentown, PA

*email: nkopyt@gmail.com

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