CORRESPONDENCE

A Case of Peritonitis Due to Rothia Mucilaginosa

Editor:

A 49-year-old African-American male with end-stage renal disease (ESRD) presented with cloudy effluent from his peritoneal dialysis. He had ESRD secondary to hypertensive nephrosclerosis and had been treated with continuous ambulatory peritoneal dialysis (CAPD) for about 3½ years. He had no peritonitis for the first 2½ years on dialysis. One year prior to the current episode, he had peritonitis caused by Klebsiella oxytoca (methicillin-sensitive Staphylococcus aureus was also cultured but was felt to be a contaminant). In his second episode, Micrococcus sp was cultured. The third episode was culture negative.

In the current episode, the first fluid cell count was 2500 WBC/μL, with 86% segmented cells. He was treated empirically with intraperitoneal vancomycin and ceftazidime while cultures were pending. He had suboptimal clearing on this regimen. Initial cultures grew Acinetobacter lwoffii, sensitive to levofloxacin. Vancomycin and ceftazidime were discontinued and oral levofloxacin was started with the intention of 4 weeks of treatment. The patient discontinued therapy during the third week of treatment because of his improvement but did not report back to the dialysis center for follow-up for another 3 weeks. At that point, the peritoneal effluent had become cloudy again over the previous 2 days and the patient had abdominal pain and cramps. Peritoneal fluid cell count was 1583 WBC/μL, with 91% segmented cells. While new cultures were pending, treatment was resumed with oral levofloxacin and intraperitoneal vancomycin and ceftazidime. Culture results were delayed for about 1 week because of specialized growing requirements of the culprit organism, during which time the patient’s condition worsened. By the time the organism was identified, the peritoneal fluid cell count was 4850/μL. The organism was identified as Rothia mucilaginosa, sensitive to amoxicillin. The other antibiotics were discontinued and the patient was started on amoxicillin and rifampin. Within 48 hours he was asymptomatic, peritoneal fluid was clear, and cell count was 2 WBC/μL.

Rothia mucilaginosa (formerly known as Stomatococcus mucilaginosus) is an encapsulated gram-positive catalase-positive coccus found in pairs, clusters, and tetrads and may be normal flora of the mouth and respiratory tract (1,2). Rothia mucilaginosa is considered to have low virulence and is usually associated only with the formations of dental plaque and tooth cavities. There are sporadic reports of the organism causing endocarditis in patients with heart valve abnormalities, as well as meningitis, septicemia, and pneumonia associated with intravenous drug abuse. It appears that the presence of foreign bodies, such as indwelling vascular catheters, and previous treatment with ciprofloxacin increase the risk of invasive infection and bacteremia with Rothia, particularly in immunocompromised patients (3).

Rothia mucilaginosa is an even rarer cause of CAPD-associated peritonitis. The only previous case (4) was reported in 1988. That patient was not immunocompromised, apart from having ESRD. Our patient had a combination of risk factors for this infection. The peritoneal catheter was the obvious portal of entry for the pathogen and he had significant antibiotic exposure during the weeks prior to the infection, including an unfinished course of a fluoroquinolone. These factors could have enabled the opportunistic growth of this organism. The delay in bacteriologic diagnosis was unfortunate and probably contributed to the opportunistic superimposition of yeast infection. It was also unfortunate because Rothia mucilaginosa is readily treatable with antibiotics: our patient responded rapidly to therapy with amoxicillin and rifampin.

Despite its low virulence, Rothia may be emerging as a pathogen of greater importance due to concurrent use of multiple antibiotics in patients with varying levels of immunocompromise. Infections due to this organism are also likely to be underreported since it is not routinely included in the databases of automated microbiologic identification systems.

DISCLOSURES

I, Emin Hodzic, declare that no financial conflict of interest exists.

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Predialytic Period and Baseline Peritoneal Membrane Status: Any Connection?

Editor:

We read the study by Hasegawa et al. (1) with great interest. The authors retrospectively analyzed 37 incident peritoneal dialysis (PD) patients on a prescribed very low protein diet (0.39 g/kg) during the predialysis period at a single center in Japan. Patients were divided into two groups according to their daily protein intake, as calculated from urea nitrogen appearance in 24-hour urine collections. The authors reported that in the group with the lowest protein intake, dialysate-to-plasma ratio (D/P) of creatinine measured with the peritoneal equilibration test at the start of PD therapy was lower than in the group with higher protein intake. Furthermore, a positive correlation was seen between D/P creatinine and protein intake. The authors finally suggested that a “strict low protein diet during the predialysis period may suppress peritoneal permeability at induction of PD.”

We retrospectively analyzed 39 standard peritoneal permeability analyses (SPAs) from 67 consecutive patients that started PD in our hospital during the past 5 years. Patients with a history of previous transplantation (n = 4), hemodialysis (n = 5), or peritonitis episode (n = 1) were excluded. Insufficient data was the reason for excluding 18 other patients. All chronic kidney disease patients with glomerular filtration rate < 20 mL/minute in our center are advised to follow a low protein diet of 0.7 g/kg/day, according to international guidelines. We collected demographic records and laboratory data on residual glomerular filtration rate decline, lipids, calcium, phosphate, and C-reactive protein during the predialytic period. Moreover, we calculated patients’ protein intake from 24-hour urine collections. Means of three measured values during 1 year before starting PD were calculated. Comorbidity during the predialytic period was scored using Davies comorbidity score (2). The SPAs were performed within 6 months after initiation of PD. Mass transfer area coefficient (MTAC) for creatinine was calculated. Table 1 shows the basic characteristics of our patients compared to the Japanese patients. Contrary to the Japanese study, we found no correlation between baseline MTAC creatinine and protein intake. Even in the subgroup of patients in the lowest quartile of protein intake (0.42 – 0.6 g/kg/day), no relationship was found. Furthermore, no correlation was present between baseline MTAC creatinine and decline in residual renal function, Davies index, or any other biochemical parameter.

It would be exciting if we could define factors that influence baseline membrane status. In that ideal case, we could suggest strategies that would protect or alter membrane characteristics before starting PD. So far, published data concerning factors that characterize inherent peritoneal membrane status are inexplicit. In some studies, inherent fast transport status in incident PD patients was associated with higher age, high body mass index, and race (3,4); for example, it seems that Chinese patients have lower D/P ratios than Caucasian and Africans. In other studies, however, only male gender and comorbidity (e.g., diabetes mellitus) seemed to be associated with a fast transport status (5).

The high percentage of slow transporters in the Japanese patients (more than 50%) is remarkable. Could this high percentage of slow and low-average transporters in the Japanese patients be attributed to the low protein diet? MTAC and D/P ratios are dependent mainly on the vascular peritoneal surface area. Moreover, this area seems to be dependent not only on the number of microvessels (anatomic surface area) but also on the number of perfused microvessels (effective surface area). Apparently, genetic factors such as race determine the anatomic surface area, while a variety of known and unknown parameters could affect the effective surface area. Moreover, genetic factors such as interleukin-6 polymorphisms are likely to contribute (6), as well as various substances that are locally produced in peritoneal tissue. In this point of view, the plausibility of a link
between protein intake and vascular peritoneal surface area is difficult to appreciate. Nevertheless, identification during the predialytic period of parameters that affect peritoneal membrane structure is a challenging research field for well-designed studies.

DISCLOSURES

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Bacteremia, Sepsis, and Peritonitis with Pasteurella multocida in a Peritoneal Dialysis Patient

Editor:

Pasteurella multocida, a gram-negative coccobacillus that is part of the normal oral flora of cats and dogs, may be transmitted to humans by bites, scratches, or licks. Eighteen cases of P. multocida peritonitis in chronic peritoneal dialysis (PD) patients have been previously reported. We present the first report of P. multocida peritonitis accompanied by bacteremia with the same organism and the sepsis syndrome in a chronic PD patient.

A 36-year-old white female patient with a medical history that includes end-stage renal disease from systemic lupus erythematosus, diabetes mellitus, and hypertension was maintained on continuous cycler PD. Three days
prior to admission, she developed a fever to 105°F and cloudiness of her PD fluid. She presented to the hospital with nausea, emesis, abdominal pain, fever, and confusion. On arrival she was notably lethargic, with blood pressure 80/40 mmHg and heart rate 110 beats per minute. Signs of PD catheter exit-site infection were not observed but small scratch marks were noted on her upper extremities. Predismission medications included prednisone and hydroxychloroquine, which she took intermittently.

Laboratory studies revealed peripheral white blood cell count 7400/mm³ with 28% band forms, hemoglobin 12.4 g/dL, blood urea nitrogen 64 mg/dL, creatinine 10.2 mg/dL, potassium 2.9 mEq/L, albumin 1.8 g/dL, and lactate 5.5 mmol/L. The PD fluid was cloudy and contained 22 red blood cells/mm³ and 1142 nucleated cells/mm³, with 17% band forms and 83% neutrophils. Gram stain of PD fluid was unrevealing.

After PD fluid and blood cultures were obtained, intravenous and intraperitoneal vancomycin and gentamicin were administered. Because of persistent hypotension and confusion, the patient was placed on high-dose vasopressors, given a tracheal intubation, ventilated, and transferred to the intensive care unit. She was given drotrecogin alfa, which was discontinued because of gastrointestinal bleeding. Her PD catheter was removed to eliminate the presumed source of infection, and continuous venovenous hemodiafiltration via a temporary internal jugular dialysis catheter was used for renal replacement therapy. Ischemia of her toes developed over the next few days.

Blood and PD fluid cultures grew gram-negative cocccobacilli, later identified as Pasteurella multocida sensitive to levofloxacin, ampicillin–sulbactam, penicillin, ceftriaxone, and tetracycline but resistant to erythromycin. Antibiotic therapy was changed to intravenous ciprofloxacin for a total course of 14 days. Within 7 days, she was successfully weaned off ventilatory and vasopressor support. The internal jugular catheter was tunneled and she remained on chronic intermittent hemodialysis. Detailed questioning revealed that the patient had recently adopted two stray cats that regularly licked her skin excoriations. She had not observed that the cats had come into contact with her dialysis supplies or punctured her dialysis tubing. The cats had not been present in the room when she performed PD exchanges.

Pasteurella multocida was named after Louis Pasteur, who first described this organism in 1880 (1). It is an aerobic gram-negative coccobacillus found in the oral cavity of 70%–90% of cats and 66% of dogs (2). It is known to cause a wide spectrum of infections in humans, including cellulitis, pulmonary infections, meningitis, septic arthritis, and osteomyelitis, usually after animal bites or scratches.

*Pasteurella multocida* is an uncommon cause of peritonitis in chronic PD patients. Only 18 cases have been reported to date (3). The suspected source of infection was the domestic cat in 17 of 18 cases, which is similar to the present case in which a potential portal of infection was licking of skin abrasions by pet cats. There was no evidence of disruption of the PD tubing by the cats, as has been observed in about half the prior cases of *P. multocida* peritonitis in chronic PD patients. Because all 18 patients did not appear septic and were clinically stable for outpatient treatment, blood cultures were not obtained in most cases and they did not reveal *P. multocida* in the 4 cases in which blood cultures were obtained. No case of *P. multocida* peritonitis with *P. multocida* bacteremia and sepsis syndrome in a chronic PD patient has been published prior to this report. Our patient was different from prior patients with *P. multocida* peritonitis without bacteremia in that bacteremia caused a sepsis syndrome with vascular collapse, distal ischemia, and respiratory failure. It is unclear if the peritonitis was followed by bacteremia and sepsis or if bacteremia seeded the peritoneal space. It is possible that immunosuppression from systemic lupus erythematosus and/or corticosteroids allowed our patient to develop bacteremia and sepsis and become so much more ill than the 18 chronic PD patients with *P. multocida* peritonitis previously reported.

Spontaneous bacterial peritonitis due to *P. multocida* has been reported in 8 liver cirrhosis patients (4–10). In 2 cases (4,5), blood cultures grew *P. multocida* but PD fluid did not and in a third case (6), PD fluid grew *P. multocida* but blood cultures did not. The other 5 reports (6–10) were similar to our patient, in whom *P. multocida* was cultured from the blood and the PD fluid. Gram stain of the PD fluid was unrevealing in 4 of these 5 cases. Compared to the cat exposures in our patient and the chronic PD patients with *P. multocida* peritonitis (3), the pet exposures in cirrhotic patients with *P. multocida* bacteremia and peritonitis were more varied: no pets in 2 (6,8), a cat in 1 (10), a dog in 1 (9), and a gamecock and pet pig in the other (7). Only 1 cirrhotic patient with *P. multocida* bacteremia and peritonitis (6) had a severe septic picture like our patient, and this patient died. Two others died of hepatic failure (9,10) and the other 2 recovered (7,8). There was no evidence of alcohol abuse or chronic liver disease in our patient.

Similar to the rate in the general population, 31% of chronic PD patients own domestic pets. We now know that *P. multocida* peritonitis and *P. multocida* bacteremia with sepsis may coexist in chronic PD patients. Chronic illness,
such as cirrhosis of the liver or systemic lupus erythematosus, may be predisposing factors.

DISCLOSURES

The authors declare there are no business relationships or conflicts of interest.

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