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CORRESPONDENCE

Pericatheter Leak with Air Bubbles in the Effluent of a Patient on Chronic Peritoneal Dialysis Without Peritonitis: Your Diagnosis?

Editor:

A 67-year-old woman had been on chronic peritoneal dialysis (PD) for almost 9 years following bilateral nephrectomy for renal cell carcinoma. Over the past 3 – 5 years, she has developed extensive metastases in lungs, abdomen, and possibly brain and has been treated with sunitinib, a tyrosine kinase inhibitor.

The patient went for a 3-week vacation and was stable. On her return, she developed a profuse leak of clear fluid around the exit site of the PD catheter. She did not complain of any pain or fever. On examination while she was lying flat and draining her effluent, a leak of clear fluid could be seen around the catheter at the exit site and there were large volumes of air bubbles in the effluent. On further questioning, she said that the leak and the presence of air in effluent appeared simultaneously. The patient had no signs of exit-site infection, no trauma, peritonitis, or any change in the effluent to suggest a perforation of any intra-abdominal viscera (bowel or bladder). There was no distension to indicate abnormal collection of air in the abdominal cavity.

The PD catheter had been in place for almost 9 years and was cloudy and slightly deformed (Figure 1). We suspected that the explanation for the air seen bubbling in the catheter with the effluent was a leak through a hole in the catheter itself. Careful examination of the external part of the PD catheter did not reveal any tear or leak. In the absence of any other diagnosis, the patient was referred to the surgeon with a request to de-roof the subcutaneous part of the catheter and determine whether there was a leak from the part of the catheter below the skin. If this were the case, the surgeon could salvage the catheter by cutting out and replacing the damaged part with an extension.

One gram of vancomycin was administered intraperitoneally a day before the procedure. During the operation, the surgeon indeed observed that, when the catheter was clamped proximal to the leak, fluid infused under pressure (Figure 2) leaked from the catheter. That particular part was dissected out, an extension was inserted (Peri-Patch Repair Kit; Quinton Instrument, Bothwell, Washington, USA), and the two ends were attached with a titanium connector held with 2-0 polypropylene sutures. Replacing the external portion of a damaged catheter makes a full catheter replacement unnecessary. After this procedure, dialysis was discontinued and then resumed after 24 hours without any technical problem.

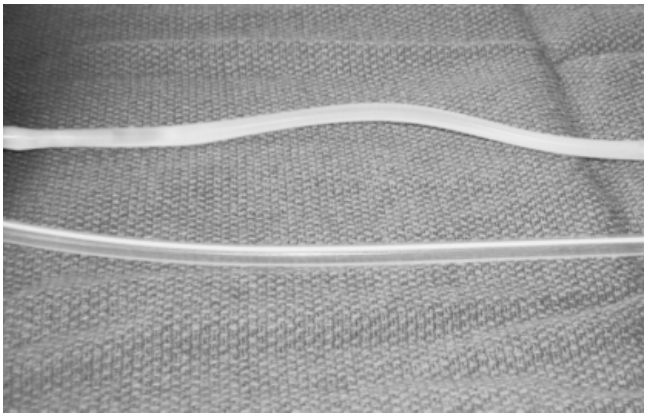

Figure 1 — Opaque peritoneal dialysis (PD) catheter (upper) from the patient compared with a new PD catheter (lower).

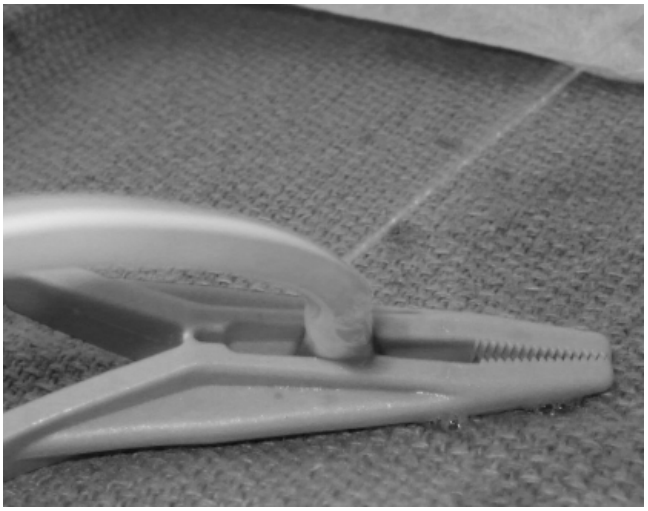

Figure 2 — Leak from a micropuncture when fluid was infused under pressure against a clamped distal end.

Damage to PD catheters may occur due to long use, exposure to antibacterial agents (strong oxidants), and accidental injury from sharp objects. We previously described evidence of damage to the peritoneal catheter in the form of cloudiness, thinning, and leaks after long-term use (1). This complication may be associated with long-term use of mupirocin and we suggest continuous observation with the intention that, if the catheter becomes cloudy, one can administer mupirocin by the intra-nasal route before there is any further serious damage to the catheter.

The Twardowski group described 7 patients that had 11 catheter repairs without any subsequent complications and no change in peritonitis rates (2). Seven of the 11 splicing procedures repaired damage to the original catheter; 4 splicing procedures were done in previously repaired catheters. The paper recommended that catheter repair should be attempted if the break is near the exit site. This would extend catheter life and reduce costs and patient inconvenience (2).

In our patient, we restarted dialysis through the newly attached catheter after 24 hours. The patient continued dialysis without any further technical problems: initially 1.5 L every 2 hours, returning to her regular regime of 5 × 2-L exchanges 3 days after the repair.

In conclusion, in the absence of peritonitis, a leak around the peritoneal catheter with large volumes of air in the effluent should raise the suspicion of a hole in the catheter, even if it is not present in the external part of the catheter.

S.N. Tapiawala[1]
T.C. Penner[2]
J.M. Bargman[1]
S. Izatt[1]
D.G. Oreopoulos[1]*

Division of Nephrology[1]
Division of Surgery[2]
University Health Network and University of Toronto
Toronto, Ontario, Canada

*e-mail: dgo@teleglobal.ca

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Outcomes and Risk Factors for Mortality After Transfer from Hemodialysis to Peritoneal Dialysis in Uremic Patients

Editor:

Transfer to peritoneal dialysis (PD) is a choice for hemodialysis (HD) patients in cases of such complications as access problems, hemorrhage risks, and cardiovascular problems. This study was performed to identify outcomes and risk factors for mortality in uremic patients transferred from HD to PD.

The transfer group included 28 HD patients that transferred to PD from 1 January 2002 to 1 March 2007 in our center. These patients had been maintained with 4-hour HD 3 times weekly for at least 3 months before being transferred. The no-transfer group included 40 patients that had chosen PD as their first dialysis modality from 1 January 2002 to 1 March 2007. The causes for transfer consisted of cardiovascular problems (16/28), vascular access problems (6/28), patient choice (3/28), and hemorrhage (3/28). There were no significant differences in age, gender, or cause of end-stage renal disease between the transfer group and no-transfer group. The initial parameters of residual renal function (RRF), adequacy of dialysis, nutritional status, and peritoneal transport are shown in Table 1. The transfer group had a higher proportion of patients with malnutrition (50% vs 22.5%, $p = 0.018$) and lower levels of serum albumin (34.2 ± 4.8 vs 37.1 ± 4.1 g/L, $p = 0.012$), normalized total protein equivalent of nitrogen appearance (0.82 ± 0.22 vs 0.98 ± 0.27 g/kg body weight/day, $p = 0.015$), and RRF (4.1 ± 3.4 vs 8.0 ± 2.8 mL/minute, $p < 0.0001$) compared to the no-transfer group.

The mean Davies comorbidity score (1,2) was remarkably higher in the transfer group (1.29 ± 0.10) than in the no-transfer group (0.43 ± 1.01, $p = 0.006$). As for disease details, there were no significant differences in rates of malignancy, ischemic heart disease, left ventricular dysfunction, diabetes mellitus, or systemic collagen vascular disease between the transfer group and the no-transfer group, except that the frequency of peripheral vascular disease was much higher in the transfer group (36% vs 8%, $p = 0.004$) due to a higher number of cerebrovascular accidents in these patients.

At the end point of follow-up, there were 12 patients being maintained on PD, 3 patients had transferred back to HD, 2 patients had received kidney transplantation, and 11 patients in the transfer group had died. Mean duration on PD was 16.2 ± 16.1 months. There were no significant differences in outcomes or duration on PD between the transfer group and the no-transfer group,

TABLE 1
Initial Parameters of Residual Renal Function (RRF), Adequacy of Dialysis, Nutritional Status, and Peritoneal Transport in Patients That Transferred from Hemodialysis (HD) to Peritoneal Dialysis (PD) (Transfer Group) and PD Patients That Did Not Transfer from HD (No-Transfer Group)

	Transfer group	No-transfer group	*p* Value
Patients (*n*)	28	40	
Serum albumin (g/L)	34.2±4.8	37.1±4.1	0.012
Blood hemoglobin (g/L)	86±12	80±14	0.07
nPNA (g/kg body weight/day)	0.82±0.22	0.98±0.27	0.015
Malnutrition by SGA score (%)	50	22.5	0.018
RRF (mL/min)	4.1±3.4	8.0±2.8	<0.0001
D/P creatinine	0.64±0.10	0.61±0.09	NS
Total Kt/V urea	1.79±0.36	1.97±0.61	NS
Total CCr (L/week/1.73 m^2)	64.5±15.7	73.0±23.5	NS
Total fluid removal (mL/day)	1132±286	1265±402	NS

nPNA = normalized total protein equivalent of nitrogen appearance; SGA = Subjective Global Assessment; D/P = dialysate-to-plasma ratio; CCr = creatinine clearance; NS = not significant.

except that the death rate was remarkably higher in transfer group (39% vs 15%, $p = 0.03$). Cardiac disease was the main cause of death in each group. The survival rate in the transfer group was 71.1% at 6 months, 65.6% at 1 year, and 52.5% at 2 years, which was significantly lower than in the no-transfer group (Wilcoxon $p = 0.007$, log rank $p = 0.017$). Only comorbidity score (risk ratio 5.88, 95% confidence interval 0.80 – 6.00; $p = 0.026$) and malnutrition (risk ratio 2.19, 95% confidence interval 0.25 – 10.0; $p = 0.002$) were found to be independent predictors of mortality in Cox proportional hazards multivariate analysis.

Our study found that patients that transferred from HD to PD had a worse outcome than patients that had initiated dialysis with PD, which has been confirmed in non-Chinese patients (3,4). Van Biesen *et al.* discussed that HD patients that transferred to PD usually had no RRF left and sufficient PD adequacy was more difficult to obtain in those patients (3,5). Numerous comorbid diseases at the beginning of PD in transfer patients could also explain the poor outcome (4). In our study, lower levels of RRF and higher prevalence of malnutrition and comorbid diseases were observed in the transfer group compared with the no-transfer group. There were no differences in total fluid removal or small solute clearance between the two groups. Therefore, we have demonstrated that comorbidities and malnutrition, but not level of RRF, are important predictors of mortality in patients transferring from HD to PD.

X. Zhang
F. Han
Q. He
H. Huang
X. Yin
J. Ge
J. Chen*

Kidney Disease Center
The First Affiliated Hospital
College of Medicine, Zhejiang University
Hangzhou, Zhejiang Province, P.R. China

*e-mail: chenjianghua@zju.edu.cn

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Peritonitis Due to *Streptococcus anginosus* in Patients Treated with CAPD: a Report of Two Cases

Editor:

Streptococcus anginosus is a member of the *Streptococcus anginosus* group (SAG), which includes *S. anginosus*, *S. intermedius*, and *S. constellatus*. The SAG pathogens, which are found in the oral cavity, gastrointestinal tract, and urogenital tract (1,2), are the cause of significant infections such as various abdominal or hepatobiliary diseases, abscess formation, and endocarditis (3,4). However, to our knowledge, there is no case report of a *S. anginosus* peritonitis that developed during continuous ambulatory peritoneal dialysis (CAPD) therapy. We present 2 patients with peritonitis caused by *S. anginosus*.

The first patient was a 74-year-old man with chronic renal failure of 15 years' duration. He was admitted to our unit with a 2-day history of cloudy dialysate effluent, mild abdominal pain, and fever. His medical history was significant for a marked swelling in the left cervical region that developed 5 days before his admission. This patient had no history of invasive dental procedures or pharyngotonsillitis but he had poor dental health, with six tooth caries.

On physical examination, a non-elastic non-tender lymphadenitis in the left cervical region was noted. Ultrasonographic examination of the left cervical region showed lymphadenitis in an area approximately 1.3 cm in diameter. The results of the examinations of other organ systems and the peritoneal catheter were within normal limits. The white blood cell (WBC) count in the peritoneal fluid was 17 900/μL (95% neutrophils). Gram stain of the peritoneal fluid revealed no bacteria. Peritoneal fluid samples that had been inoculated into BACTEC bottles for culture (Becton Dickinson, Franklin Lakes, New Jersey, USA) grew *S. anginosus*, which was identified by conventional methods and automated BBL Crystal (Becton Dickinson) identification kits.

The second patient was a 58-year-old woman with chronic renal failure of 11 years' duration. She had been receiving CAPD therapy for 4 years when she was admitted to our hospital with moderate abdominal pain, vomiting, nausea, and cloudy dialysate effluent. Her symptoms had developed about 24 hours before her admission. On physical examination, she had poor dental health; examinations of other organ systems and the peritoneal catheter were within normal limits. The WBC count in the peritoneal fluid was 6000/μL (85% neutrophils). Gram stain of the peritoneal fluid revealed no bacteria. Peritoneal fluid samples that had been inoculated into BACTEC bottles for culture grew *S. anginosus*.

After dialysate aspirate specimens were taken for culture, both patients received ampicillin–sulbactam 1.5 g twice daily and ciprofloxacin 200 mg twice daily intravenously. This therapy was continued for 14 days. In both cases, peritoneal fluid WBC count decreased to normal limits during the following week. Both patients are well and still receiving CAPD at time of writing.

The SAG group is a member of the *Streptococcus viridans* group, which was previously termed the "*Streptococcus milleri* group." These gram-positive cocci are distinguished by their microaerophilic growth requirements, their formation of colonies <0.5 mm in diameter, and the presence of a distinct caramel-like odor that they release when cultured (1). These organisms are commensals of the oral cavity, gastrointestinal tract, and urogenital tract. SAG organisms cause pyogenic invasive infections and have been found in dental, neck, liver, brain, pelvic, and subcutaneous tissue abscesses, in cases of bacteremia with endocarditis, and in patients with thoracic empyema (1,3,5). People with underlying medical conditions such as cirrhosis, diabetes mellitus, or a malignancy, are predisposed to invasive infections with SAG (3).

Pathogens in the *S. anginosus* group are often associated with abscesses (3); however, ultrasonographic examination of the abdomen in Patients 1 and 2 and of the neck in Patient 1 revealed no abscess. These patients had no history of invasive dental procedures or pharyngotonsillitis, but they had poor dental health.

On the basis of our findings, we concluded that the infection in both patients was caused by the direct inoculation of bacteria through the peritoneal catheter and into the peritoneal fluid. Because we could not isolate the organism in blood samples, we assumed that transmission had resulted from direct inoculation rather than from hematogenous spread. That hypothesis was supported by findings such as the patients' poor dental health and lower socioeconomic status. They may have infected themselves during peritoneal exchange.

We suggest that *Streptococcus anginosus* be kept in mind as a cause of CAPD peritonitis, particularly in patients with cervical lymphadenitis or poor dental health. Microbiologic procedures should be performed with care to isolate this fragile micro-organism. Also, we think that CAPD patients should be informed of and under regular control of a dentist for dental hygiene.

A. Ibis[1]*
S. Sezer[1]
E. Tutal[1]

O.K. Azap[2]
F.N. Ozdemir[1]

Department of Nephrology[1]
Department of Infectious Disease and
Clinical Microbiology[2]
Baskent University Hospital
Ankara, Turkey

*e-mail: avsinibis@yahoo.com

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Aeromonas salmonicida Peritonitis After Eating Fish in a Patient Undergoing CAPD

Editor:

Aeromonas salmonicida, a gram-negative bacterial pathogen that is the causal agent of furunculosis in salmonid fish, Atlantic cod (*Gadus morhua*), grayling (*Thymallus*), salmon, turbot, shark, carp, *etc.*, and can cause a debilitating and often lethal disease in fish. The virulence and pathogenicity of *Aeromonas salmonicida* (1,2) has been attributed to extracellularly secreted proteins and bacterial surface factors. It has been reported that *A. salmonicida* may seriously damage the fish's intestinal lining. Exposure to such a pathogen damages the intestinal epithelial cells, sheds cell debris into the intestinal lumen, and disorganizes the microvilli (3). The likely route of infection for *A. salmonicida* infections in Atlantic salmon is passage of bacteria through the foregut (rather than the hindgut) (4). Of the clinical isolates of *Aeromonas*, 29.4% have been shown to be enterotoxic, 43.1% were hemolytic, and 89% were cytotoxic (5). *Aeromonas spp* may cause gastroenteritis in children, most often during the warmer months (6).

The genus *Aeromonas* is usually grouped into two subdivisions: The psychrophilic group, in which the only species is *A. salmonicida*, is nonmotile and does not grow at 37°C. The mesophilic group, members of which grow at 37°C and are motile, contains the species *A. hydrophila*, *A. caviae*, and *A. sobria*. Although peritonitis episodes due to *A. hydrophila* and *A. caviae* have been reported in patients on continuous ambulatory peritoneal dialysis (CAPD) (7,8), there have been no reports of *A. salmonicida* peritonitis in patients on CAPD. We describe here an episode of *A. salmonicida* peritonitis that developed in a CAPD patient after she ate fish.

A 68-year-old diabetic woman who had been on CAPD for 11 months was admitted with abdominal pain and cloudy peritoneal fluid. She had no history of peritonitis. She had a history of chronic gastritis and duodenal ulcer for more than 20 years; she was receiving erythropoietin, iron, and insulin. One week before admission, she ate cooked freshwater fish at a restaurant on two occasions and ate cooked fish at home once. During the week before admission, she suffered from abdominal pain and diarrhea (4 – 5 times per day) and, after taking a traditional Chinese medicine, the diarrhea subsided. Three days before admission she noted that the peritoneal fluid was slightly cloudy and she had occasional abdominal pain. Meanwhile, she had flu-like symptoms with headache and cough, accompanied by nausea, intermittent abdominal pain, and constipation. Two days before admission, the peritoneal fluid became cloudier but she had no fever or vomiting. On admission, her temperature was 36.8°C, heart rate was 80 bpm, respiration 20/minute, and blood pressure 130/65 mmHg. She was tender in the lower abdomen but the exit site of the peritoneal catheter was clean. Peripheral WBC count was 4.66×10^9, with 70% polymorphonuclear leukocytes. Hemoglobin level was 87 g/L, serum iron 6.0 µmol/L, transferrin 1.9 g/L, and ferritin 160 µg/L. Liver function tests were normal and viral markers for hepatitis B and C were negative. Stool appearance was normal. Peritoneal effluent contained $>10 \times 10^6$ WBC/L and a few red blood cells were also present. The patient was treated empirically with intraperitoneal cephradine 1.0 g and ceftazidime 1.0 g per day for 14 days. Soon after the initiation of therapy, abdominal pain improved and the peritoneal effluent gradually cleared. Peritoneal effluent culture showed *A. salmonicida* sensitive to ciprofloxacin, ceftriaxone, cefoxitin, imipenem, and moxifloxacin, and resistant to erythromycin, oxacillin, teicoplanin, and vancomycin. After 2 weeks of treatment, the patient recovered completely, without complications.

Aeromonas bacteremia or peritonitis usually appears in patients with an underlying disease, including chronic hepatic disease, malignancy, and intestinal perforation (8). *Aeromonas salmonicida* is an opportunistic and ubiquitous fish pathogen that causes furunculosis in a variety of fish. CAPD patients, especially those with previous chronic gastrointestinal tract illness, may be prone to invasion of the bacteria from the gut and infection of the peritoneal cavity, causing peritonitis. Also, the organism may cause diarrhea, which may lead to peritoneal infection. Since we did not do a stool culture, we can only speculate on the route of infection. This case suggests that CAPD patients should be cautioned against eating raw or incompletely cooked fish, which may predispose them to such infections.

X. Yang
Q.Q. Yang
Q.Y. Guo
C.Y. Yi
H.P. Mao
J.X. Lin
Z.P. Jiang
X.Q. Yu*

Department of Nephrology
The First Affiliated Hospital
Sun Yat-sen University
Guangzhou, China

*e-mail: yuxq@mail.sysu.edu.cn

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