

CORRESPONDENCE

Neostigmine for the Treatment of Acute Colonic Pseudo-Obstruction (Ogilvie's Syndrome) in a Patient on CAPD

Editor:

A 79-year-old woman, diagnosed with chronic renal insufficiency due to hypertensive nephrosclerosis and who had been treated with continuous ambulatory peritoneal dialysis (CAPD) for 3 months, was admitted to the emergency room having had severe abdominal pain, distension, and vomiting for 2 days. On physical examination, the patient had abdominal tenderness, distension, and hypoactive bowel sounds. Her blood pressure and pulse rate were 90/50 mmHg and 108/minute. Laboratory results were as follows: white blood cells 17 900/ μ L (normal range 3.9 – 9.6/ μ L), hematocrit 30.9% (NR 36.1% – 50.3%), hemoglobin 10.3 g/dL (NR 12.1 – 17.2 g/dL), urea 33 mg/dL (NR 10 – 50 mg/dL), creatinine 6.4 mg/dL (NR 0 – 1.3 mg/dL), sodium 132 mmol/L (NR 135 – 145 mmol/L), potassium 4.0 mmol/L (NR 3.5 – 5.1 mmol/L), chloride 105 mmol/L (NR 98 – 106 mmol/L), calcium 10.2 mg/dL (NR 8.6 – 10.2 mg/dL). Abdominal x ray revealed dilated colonic loops. Intravenous fluid replacement was started initially and nasogastric and endorectal tube decompression were applied. Abdominal computed tomography (CT) with intravenous, oral, and rectal contrast revealed colonic dilatation from the anal canal to the cecum, with no mechanical obstruction. The cecum diameter, which is normally 6 – 7 cm, was measured as 10 cm (Figure 1).

With these clinical and radiological findings, the patient was diagnosed with Ogilvie's syndrome. Pharmacologic decompression with neostigmine perfusion had been planned and consulted with our nephrology, cardiology, and anesthesiology departments for its potential hazards. After cardiac monitoring, the patient was given a single dose of intravenous 2.5 mg/60 minutes neostigmine (Adeka Ilac San, Izmir, Turkey) perfusion. No serious complication was seen other than hypotension and a mild syncope attack. On physical examination 3 hours later, abdominal distention had been resolved, with no abdominal tenderness. White blood cell counts decreased gradually to 12 400/ μ L at 6 hours and 7800/ μ L at 24 hours. Control abdominal CT showed significant regression of

colonic dilatation (cecum diameter 6 cm) (Figure 2). On follow-up, the patient had no complications and was discharged on the third day of admission.



Figure 1 — Distension in the cecum in abdominal CT (diameter 10 cm).



Figure 2 — Significant decrease of distension in cecum 24 hours after neostigmine perfusion in abdominal CT (diameter 6 cm).

Ogilvie's syndrome is seen mainly in the elderly population. It occurs secondary to various conditions such as peritonitis, severe infections, severe electrolyte imbalance (hypokalemia), neurological disorders, spinal and pelvic surgery, and trauma. The use of certain drugs (antidepressants, phenothiazines, anti-Parkinson drugs, opiates, *etc.*) and alcohol abuse may also cause Ogilvie's syndrome (1).

When Ogilvie's syndrome is suspected, the first step in treatment should be conservative (nasogastric and endorectal decompression, correction of electrolyte and fluid imbalance, *etc.*) unless the patient has acute abdomen. Surgery is indicated when conservative follow-up fails or progressive bowel distention may result in cecal wall ischemia and perforation (cecal diameter > 12 cm) (2,3).

Pharmacological and colonoscopic decompression are alternative treatment modalities. Colonoscopy is a successful method in managing Ogilvie's syndrome, but it has potential complications related to the unprepared bowel (morbidity rate 3%, mortality rate 1%) (4). Several pharmacological agents have been used in only a few controlled trials. Neostigmine is a safe and more effective option than colonoscopic decompression (5–7). Intravenous neostigmine, given at 2 – 2.5 mg dose in 1 – 3 minutes to an hour, has been reported as a treatment protocol for colonic pseudo-obstruction. Increased salivation, nausea, vomiting, abdominal pain, bradycardia, hypotension, and bronchospasm are the major side effects of cholinesterase inhibitors. Bradycardia and syncope may be seen after neostigmine administration (1). Neostigmine is contraindicated in patients on beta-blockers, in acidotic patients, and in patients with a history of recent myocardial infarction since they are prone to dysrhythmias (8).

5-HT₄ receptors mediate a number of responses in the gut. MacColl *et al.* reported positive response in patients treated with an initial dose of 10 mg cisapride, given as a slow intravenous bolus per 4 hours for 16 hours, followed by oral cisapride 10 mg 3 times per day. (9). The prokinetic effect of erythromycin is described as it binds to the motilin receptor. Armstrong *et al.* reported that they had treated 2 patients with acute colonic pseudo-obstruction with oral erythromycin (500 mg four times daily) for 10 days (10). No recurrences were observed in either treated case. In acute colonic pseudo-obstruction, cholinesterase inhibitors are usually administered by the intravenous route, although occasional reports use subcutaneous neostigmine. The oral route, the conventional one for pyridostigmine used in some chronic diseases (*e.g.*, myasthenia gravis), seems to be precluded in the acute setting of Ogilvie's syndrome by the erratic absorption of conventional formulations of neostigmine and pyridostigmine (11). Mortality rate

is about 14% of cases treated conservatively, whereas it reaches 30% in patients undergoing surgical treatment. Surgery should not be considered an initial option and should be reserved for patients who do not respond to conservative management. Delayed decompression is responsible for cecal ischemia and/or perforation in 14% – 40% of cases, and is associated with an increased rate of mortality (40% – 50% of patients) (12).

In light of these data, pharmacological therapy should be considered when traditional conservative management fails. Intravenous neostigmine, unless contraindicated, should be considered the current drug of choice.

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Sterile Peritonitis Outbreak Related to Icodextrin Treatment

Editor:

Icodextrin-associated sterile peritonitis has been described recently (1-4). A potential underlying mechanism for icodextrin-related sterile peritonitis could be the introduction of an impurity or allergen during manufacture of icodextrin. In fact, preliminary analysis by a Baxter Healthcare investigation suggests that some batches could contain peptidoglycan, a non-endotoxin weak pyrogen capable of provoking inflammatory responses in the peritoneum (5). We report a cluster of 6 icodextrin-related sterile peritonitis episodes presenting in our automated peritoneal dialysis (APD) patients over a 10-day period. This presentation supports the manufacturing process etiology. Peritoneal membrane transport kinetics and ultrafiltration after icodextrin-related sterile peritonitis were compared to base levels.

From 29 November to 9 December 2002, a total of 6 of our 38 APD patients presented with abdominal discomfort and cloudy effluent. Table 1 shows patient characteristics. No rash, fever, or other hypersensitivity or general symptoms were observed. Cell count

in the effluent ranged from 120 to 1830 white blood cells/ μ L, with a predominance of neutrophils. Patients 1 to 3 were given antibiotics empirically on suspicion of infectious peritonitis. Antibiotic treatment was stopped on the third day because sterile cultures were confirmed. Icodextrin was also discontinued. In Patients 4 to 6, icodextrin was stopped and they received no antibiotic treatment. Dialysates cleared and abdominal symptoms disappeared in all patients. All dialysate cultures were sterile. Patients remained free of symptoms at 2 months' follow-up, and icodextrin treatment was not reintroduced.

Mean icodextrin prescription was 18 months (range 4 - 39 months) and no patient had a previous history of cutaneous hypersensitivity reactions or sterile peritonitis. No peritonitis episode was recorded in these patients during the previous 3 months. Table 2 shows results of peritoneal equilibration tests (PET) performed within the previous year and 1 month after icodextrin-associated sterile peritonitis. No significant changes in ultrafiltration ($p = 0.49$) or peritoneal transport ($p = 0.5$) were observed. Patient 2, however, presented a marked increase in peritoneal transport and a decrease in ultrafiltration capacity following sterile peritonitis.

This description supports the hypothesis of a contaminant in the manufacturing process. First, this cluster of peritonitis appeared as a first sterile peritonitis in 6 patients over a short period of time and, second, all were APD patients and they had shared and used the same icodextrin batch distributed during the month preceding the peritonitis outbreak.

Ten more APD patients shared the icodextrin batch before its withdrawal; none of them presented sterile peritonitis, but we cannot certify if the batch was actually used and, even if it was used, the lack of peritonitis in these patients could be explained by a

TABLE 1
Case Presentations

Patient	Age	Sex	Diabetes mellitus	Delay between Ico & symptoms	Peritonitis date	Cell count (μ L)	Prevalent cells	Dialysate culture	Treatment & outcome
1	70	M	Yes	7 months	29/11/02	1830	78% N	Sterile	Ico withdrawn+ab Resolution
2	34	F	No	39 months	30/11/02	250	85% N	Sterile	Ico withdrawn+ab Resolution
3	60	F	Yes	11 months	30/11/02	320	81% N	Sterile	Ico withdrawn+ab Resolution
4	57	M	No	14 months	05/12/02	120	67% N	Sterile	Ico withdrawn Resolution
5	37	F	No	33 months	05/12/02	180	79% N	Sterile	Ico withdrawn Resolution
6	34	M	No	4 months	09/12/02	1370	53% N	Sterile	Ico withdrawn Resolution

Ico = icodextrin dialysate; N = neutrophils; ab = antibiotics.

TABLE 2
Peritoneal Equilibration Test (PET) Evolution

Patient	D/P creat	Previous PET		PET post icodextrin peritonitis		
		D/D ₀ gluc	UF (mL)	D/P creat	D/D ₀ gluc	UF (mL)
1	0.61	0.42	2200	0.58	0.42	2250
2	0.67	0.38	2800	0.76	0.25	2500
3	0.66	0.47	2200	0.65	0.47	2400
4	0.66	0.30	2150	0.66	0.29	2200
5	0.63	0.33	2400	0.57	0.41	2400
6	0.65	0.28	2500	0.61	0.38	2600

D/P creat = dialysate-to-plasma ratio of creatinine; D/D₀ gluc = dialysate to initial glucose concentration ratio; UF = ultrafiltration.

different amount of contaminant or by an individual susceptibility to icodextrin reactions.

After these peritonitis episodes, attention should be drawn to long-term potential consequences for the peritoneal membrane. In fact, only one case has been published with peritoneal function test and histological data (6). Goffin *et al.* reported characteristic features of acute peritoneal inflammation 7 days after discontinuation of icodextrin. The PET remained virtually unchanged 7 months after the first icodextrin-related sterile peritonitis.

No functional changes were observed in 5 of our patients 1 month after the first icodextrin-associated sterile peritonitis. These results are in agreement with Goffin's results. However, Patient 2 showed a marked increase in dialysate-to-plasma ratio of creatinine and a reduction in ultrafiltration, suggesting more severe peritoneal damage. This result should be confirmed in further follow-up. Interestingly, this patient had the longest duration of peritoneal dialysis (81 months). The patient's record showed only 1 peritonitis episode 25 months after initiation of peritoneal dialysis. We can hypothesize that this late mild peritonitis had consequences different and more aggressive to her peritoneal function compared to the early episodes.

In summary, we describe a cluster of icodextrin-associated sterile peritonitis in 6 APD patients over a 10-day period. This presentation suggests a contamination during the manufacturing process. No changes in peritoneal functional tests were observed, except in a patient with a longer duration of PD.

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Sterile Peritonitis Due to Icodextrin: Experience from a Canadian Center

Editor:

There have been several reports of icodextrin-induced sterile (chemical) peritonitis in patients receiving peritoneal dialysis. Most of these reports come from Europe (1–3); only 1 case has been reported in North America (4). It was initially speculated that the problem was related to some allergen or impurity that might be present in some batches as a result of changes in manufacturing (5). We report here our experience from a community hospital in Canada (see Table 1).

Three of our “early starters” doing a single overnight exchange with an icodextrin bag developed cloudy effluent and mild, if any, abdominal symptoms.

TABLE 1
Patient Characteristics

Patient	Age (years)	Primary disease	Type of PD	Cell count ^a (μL)/type	Culture	Outcome
1	40	PKD	Single exchange	1130/39% macro	Sterile	Cleared; switched to std PD
2	74	IgA nephritis	Single exchange	3640/79% macro	<i>Pseudomonas aeruginosa</i> ^b	Catheter removed
3	83	Nephrosclerosis	Single exchange	872/67% macro	Sterile	Cleared; switched to std PD
4	34	DM	Night cyler; daytime icodextrin	4730/44% macro	Sterile	Cleared; switched to std PD
5	72	DM	Overnight icodextrin; daytime std bag	94/75% macro	Sterile	Ultrafiltration failure

PD = peritoneal dialysis; PKD = polycystic kidney disease; macro = macrophages; std = standard; DM = diabetes mellitus.

^a Highest of repeated counts.

^b Likely contaminant (single isolation in repeated cultures, minimal symptoms, no response to antibiotics).

These patients had started on a single overnight exchange mostly for fluid management. One patient also had uremic symptoms and was thought to be malnourished, a fourth patient was diabetic and on a night cyler plus a daytime icodextrin bag, and a fifth patient was doing icodextrin exchange at night plus a standard solution the next morning. Cell counts in the effluent were between 140 and 4730/μL, with 25% – 80% macrophages. Cultures were negative. On one occasion, *Pseudomonas aeruginosa* was isolated, without concomitant clinical findings. Despite treatment with two antibiotics (ciprofloxacin and ceftazidime), the effluent remained cloudy, with increased cell counts (mostly macrophages) and negative subsequent cultures, prompting catheter removal. That was our first case and, retrospectively, we have no doubts that it was a reaction to icodextrin. Patient 5 noticed ongoing cloudy effluent but did not report it because he had no symptoms and the effluent was clearing with the next standard solution bag. He thought this “might be something normal,” but eventually developed ultrafiltration failure. Patient 4 developed recurrent problems, with asymptomatic, culture-negative cloudy effluent noticed only on draining the icodextrin bag.

In all of our 5 patients, the problem became apparent 2 – 5 months after the start of the icodextrin solution; various periods have been reported by others (6). None of our patients manifested skin reactions at any time during treatment with icodextrin solutions. In all patients, hypercellularity of the peritoneal fluid cleared once the icodextrin was withdrawn. In 1 patient (Patient 3), it recurred with rechallenge.

In our opinion, this is probably a chemical reaction of the solution itself and is not due to “impurities.” In patients on a single exchange for fluid control, the problem becomes easily apparent. In comparison,

patients on other modes of peritoneal dialysis may sustain a prolonged reaction, since subsequent standard fluid washes out the peritoneal cavity, until the cycle is repeated the next day. This could lead to permanent membrane damage and, eventually, ultrafiltration failure.

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Successful Peritoneal Dialysis After Intra-Abdominal Transplantation

Editor:

Adhesions and fibrosis from previous surgeries, infections, and malignancies are relative contraindications to peritoneal dialysis (PD) (1). Extensive abdominal surgical scars may suggest adhesions and/or fibrosis and such patients are often counseled to avoid PD. There are no studies reporting on the success of PD in patients with prior intra-abdominal organ transplants. We report here 2 patients with intra-abdominal transplants and extensive surgical scars, and who have been successfully maintained on PD.

Patient 1 is a 60-year-old Caucasian man who underwent orthotopic liver transplant (OLTx) in 1991 for alcoholic cirrhosis, and then developed end-stage renal disease (ESRD) from cyclosporine nephrotoxicity. Due to transportation difficulty, he wished to attempt PD. At the time of catheter insertion, the patient was noted to have chylous ascites, presumed secondary to thoracic duct ligation at the time of OLTx. He began continuous ambulatory peritoneal dialysis (CAPD) in April 2001. His serial total Kt/V results were as follows: 2.04, 2.11, 2.44, 2.30, and 2.14. He has been on PD for 22 months. His albumin fluctuates between 2.4 and 3.5 g/dL; he continues to have intermittent chylous effluent and has had no episodes of peritonitis.

Patient 2 is a 59-year-old Caucasian woman who underwent a pancreas transplant in 1990 and later developed ESRD due to type I diabetes mellitus, tacrolimus nephrotoxicity, and ischemic cardiomyopathy. After multiple hospitalizations for volume overload, she became refractory to oral diuretics and had progressive kidney disease. CAPD was initiated 14 January 2002. Complications have included hypoalbuminemia (albumin 2.9 – 3.6 g/dL), persistent hypophosphatemia requiring oral supplements, and a peritoneal leak manifested as left-sided abdominal wall and labial swelling. The swelling improved with supine evening exchanges, and she was started on cycler-assisted nocturnal dialysis with a dry day (NPD). The patient also developed small abdominal-wall and umbilical hernias; both have remained stable on NPD. Her total Kt/V measurements were 3.91, 4.08, and 2.70 during her 13 months of PD, all with significant residual function.

In total, 35 months of successful PD have been accomplished by these 2 patients. Significant PD complications, including hernia, peritoneal leak, chyloperitoneum, and hypoalbuminemia, have been experienced by our patients. Despite chronic immunosuppression, they have not had frequent infections. Based upon our experience with these 2 patients, we do not consider prior intra-abdominal transplantation

an absolute contraindication to PD and would recommend this approach to others.

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Early Diagnosis of CAPD Peritonitis Using a New Test Kit for Detection of Matrix Metalloproteinase (MMP)-9

Editor:

Bacterial peritonitis is one of the most important complications in patients on peritoneal dialysis (PD). Some patients with peritonitis on long-term PD may suffer ultrafiltration loss and reduced dialysis efficiency. It has been reported that damage to the peritoneum by peritonitis leads to peritoneal degradation and impairment of peritoneal solute transport. Therefore, early diagnosis and treatment of peritonitis are essential (1,2). MMP-9 may be associated with peritoneal extracellular matrix remodeling during peritonitis, and is expected to become a new marker for early diagnosis in PD patients with peritonitis (3,4). Recently, we developed a new MMP-9 test kit consisting of MMP-9 antigen conjugated with a colloidal dye produced to detect antibody in a nitrocellulose membrane dipstick assay based on immunochromatography.

The objective of the present study was the early diagnosis of peritonitis in patients on PD, using a test kit for detection of MMP-9. The MMP-9 test kit was used to analyze 80 peritoneal effluent samples from 20 PD patients.

Six patients had peritonitis and 14 patients were on maintenance PD without infection. Mean age was 50.7 ± 11.6 years (mean \pm SD; range 26 – 75 years; 15 males, 5 females). The prepared dipstick was

dipped into the sample and left for 10 – 15 minutes at room temperature. If both the detection band and control band were colored purple–blue, the sample was recorded as positive. If the control band was colored purple–blue but the detection band was not colored, it was taken as negative. When neither band was colored, the test reagents were assumed not to be reacting properly. These peritoneal effluents were also used for counting leukocytes and examining micro-organisms. Activities of MMP were measured using enzyme-linked immunosorbent assay (ELISA). The reactivity of the test kit was divided into three groups: positive, trace, and negative.

There was a significant difference in the number of peritoneal leukocytes between the negative and positive groups detected by the MMP-9 test kit ($p < 0.0001$). The reaction of the MMP-9 test kit depended on the number of leukocytes in the peritoneal effluent. MMP-9 concentration was also dependent on the number of leukocytes in the peritoneal effluent ($r = 0.916$, $p < 0.001$). It appeared that more than 100 leukocytes/mm³ indicated a positive result with the MMP-9 test kit. No significant difference in the reactivity of the MMP-9 test kit was found for various micro-organisms at the onset of peritonitis. The critical value for PD peritonitis is generally considered to be > 100 leukocytes/mm³ PD effluent. Since the MMP-9 test kit is adapted to this condition, it is a reliable method for the early diagnosis of CAPD peritonitis. The MMP-9 test kit usually provides results within 10 minutes. Since the kit is based on antigen–antibody reaction, the reactivity of the MMP-9 test kit showed a stable reaction after 30 minutes.

It appears that the MMP-9 test kit is a simple and reliable method for the early diagnosis of CAPD peritonitis, which is reflected in the leukocyte count in the peritoneal effluent.

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Treatment of Subcutaneous Dialysate Leak with Polidocanol: Case Report

Editor:

Dialysate leak represents one of the major noninfectious complications of peritoneal dialysis (PD) and can lead to technique failure unless managed successfully. The incidence of dialysate leak is somewhat more than 5% in PD patients (1), but this percentage probably underestimates the number of early leaks (< 30 days). Polidocanol, a sclerosant used for therapy of esophageal varices by inducing tissue fibrosis, may be effective and safe for treatment of subcutaneous dialysate leak. We report a case of recurrent early dialysate leak successfully managed with polidocanol.

A 60-year-old female end-stage renal disease (ESRD) patient on hemodialysis (HD) was first admitted to our unit to switch her renal replacement therapy from HD to PD because of frequent blood access trouble and hemodynamic instability during HD. She had been suffering from ESRD due to IgA nephropathy since 1987 and had been maintained on PD (1987–1988), cadaverous kidney transplantation (1988–1994), and HD thereafter until this admission. In August 2002, a double-cuff Tenckhoff peritoneal catheter was placed in the left side by paramedian incision. On the third postoperative day of catheter implantation, she started to dwell 0.5 L of glucose dialysate (Midperiq L 135, Terumo, Tokyo, Japan). However, external leak occurred at postoperative day 4 and we stopped instilling dialysate. The next day, she experienced pain and swelling at the left inferior part of her abdomen. Abdominal computed tomography (CT) demonstrated hernia of a surgical scar and she was immediately taken for surgical repair. After 10 days of rest from PD, we started to dwell 0.5 L dialysate, but subcutaneous leak occurred

between the operative scar where the deep-cuff was sutured and the scar of a previous transplantation at the left iliac fossa. Ultrasound revealed low echoic lesion, which disappeared as dialysate drained. We aspirated the low echoic lesion, draining 5 cc of fluid. The glucose concentration of that fluid was 649 mg/dL and peritoneal leak was diagnosed. Again, we stopped dwelling dialysate for more than 10 days. After resting the patient, we started instilling 0.4 L dialysate, but again there was swelling at the same area. As it seemed difficult to repair the leak conservatively, we injected 1 mL polidocanol (Aethoxysklerol 1%; Kaigen, Osaka, Japan) at the same site. After injection, we pressed hard for 15 minutes to allow the hypodermis to conglutinate and prevent polidocanol from being mixed into the abdominal cavity because there may be a potential risk of adhesion of a visceral organ. After 4 days of rest, we again started to instill 0.5 L dialysate, increasing the volume to 1.5 L within 10 days. The patient remained asymptomatic. She started automated PD from postoperative day 39 after catheter implantation and was withdrawn from hemodiafiltration. The patient left our hospital with no abdominal symptoms, and 9 months after discharge she is doing well with a combination of continuous cyclic PD (CCPD) and HD once per week without relapse of the leak.

We have presented an early recurrent subcutaneous leak successfully managed by polidocanol. Dialysate leak is a frequent problem and sometimes results in technique failure; no therapy with high efficacy is known. It is reported that, among PD patients who suffered from dialysate leak, only 60% – 71% can continue CAPD; 37% – 48% need catheter replacement, 11% – 28% switch to CCPD, and 18% switch to HD (1). Early dialysate leak has been related to a weak abdominal wall and early start of dwelling. The causes of weakness include multiple surgeries, multiple pregnancies, obesity, previous use of steroids, hypothyroidism, polycystic kidney disease, the way the catheter is inserted, and chronic lung disease (2). Our patient had previous transplantation surgery and steroid use as risk factors for leak. As her recurrent leak was between the deep-cuff and the area of previous transplantation surgery, it was thought there was communication between them. We thought that conglutination could stop the leak. It is recommended that dialysate leak should be treated by surgical repair, temporary transfer to HD, lower dialysate volumes, and 1 – 2 weeks of rest from PD. Referring to this recommendation, we stopped dwelling for more than 10 days; however, the leak did not resolve. After injection of polidocanol, dialysate leak never recurred. This patient apparently benefited from using polidocanol and was able to continue the PD technique.

Polidocanol is a sclerosant used for therapy of esophageal varices, and is injected into the submucosa around the varices to induce a stricture (3) by inducing mucosal inflammation, resulting in ulcer formation, tissue fibrosis, consolidation, and involution. By using pharmacological effects of this drug, it can also be used for therapies of bronchopleural fistula, oscheocele, and renal cysts (4,5). Major side effects include shock, disseminated intravascular coagulation, thrombocytopenia, and anemia when the drug enters the systemic circulation. Advantages of this sclerosant are safety, low cost, effectiveness, and simplicity. Talc, OK432, tetracycline, minocycline, and mitomycin C could be other candidates for sclerosants. However, these drugs are expensive and have a high frequency of side effects such as fever, pain, and hepatopathy (6,7).

In conclusion, as far as we know, this is the first report that demonstrates successful treatment of early dialysate leak using polidocanol. Polidocanol for treatment of subcutaneous leak may be effective and safe, so it may be considered in cases of refractory and troublesome leak.

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***Aspergillus Terreus* Peritonitis in a CAPD Patient: Report of a Case**

Editor:

Infectious peritonitis is a frequent complication of chronic ambulatory peritoneal dialysis (CAPD). The majority of cases involve bacterial pathogens, although an increased number of fungal isolates have been reported. To our knowledge, the invasive mold *Aspergillus terreus*, a rare human pathogen, has never been identified as a pathogen in an adult CAPD patient.

A 52-year-old woman with chronic renal failure due to systemic lupus erythematosus, treated in the past with steroids and immunosuppressive agents, was admitted with signs and symptoms of peritonitis to our hospital in November 2001. Her end-stage renal failure had been managed for 22 years with hemodialysis before switching to CAPD 5 years ago because of vascular access failure.

On presentation she complained of abdominal pain and was noted to be febrile, at 39°C. Leukocytosis of $21 \times 10^3/\text{mm}^3$ with shift to the left on differential was noted, along with cloudy peritoneal fluid (WBC $5.0 \times 10^3/\text{mm}^3$, 93.7% neutrophils). Cultures were obtained and empiric antibiotic treatment was initiated without therapeutic response. Multiple bacterial cultures returned negative. Ultrasound and computed tomographic examinations of the abdomen revealed a few inaccessible pockets of intraperitoneal fluid.

On the eighth hospital day, peritoneal fluid culture sent on the day of admission became positive for unspecified molds. Antibiotics were discontinued and the Tenckhoff catheter was removed. Treatment was initiated with amphotericin B, which was subsequently switched to itraconazole, 100 mg twice daily, after receipt of sensitivities. Final identification of *Aspergillus terreus* was reported, by which time the patient had improved significantly. On the 38th hospital day,

the patient was discharged in stable condition to continue hemodialysis as an outpatient. Despite clinical resolution of her peritonitis, the patient was readmitted 3 weeks following discharge and succumbed to overwhelming sepsis without evidence of fungemia.

Peritoneal infection is an important complication of CAPD. Due to improvements in technology, the incidence of peritonitis has decreased in the past decade, but it remains as frequent as 0.5 episodes per patient per year (1). Fungal pathogens constitute only about 10% of all cases of CAPD peritonitis, with *Candida* species the most common fungal isolates.

The invasive mold *A. terreus*, although widely found in the environment, is a rare human pathogen, with only a few individual cases reported. Although several cases of CAPD-associated peritonitis due to *Aspergillus* have been reported, only one prior case of *A. terreus* infection (a child undergoing continuous cycling peritoneal dialysis) has been published (2).

As with most cases of fungal peritonitis, our case illustrates that, because of nonspecific symptoms and a delay in organism growth in culture, diagnosis can be difficult. Treatment with an appropriate antifungal agent and removal of the dialysis catheter should be undertaken as soon as possible. Despite appropriate treatment, reported mortality is high, most likely, as in our patient, due to individuals' decreased immunologic status, which is a general prerequisite for developing these infections.

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