Complications of Peritoneal Dialysis

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INTRODUCTION

Peritoneal dialysis (PD) is a form of renal replacement therapy that is primarily limited to patients with end-stage renal disease (ESRD) who have had previous minimal abdominal surgery. Multiple abdominal surgeries can produce adhesions that reduce the surface area of the peritoneal membrane, which is an important determinant of the clearance of solutes from the blood. PD takes advantage of the permeability characteristics of the peritoneal membrane, particularly its ability to hinder the movement of glucose from the peritoneal space into the circulation. The osmotic gradient created by the instillation of hypertonic solutions (which are principally dextrose-based) is responsible for the removal of urea and creatinine from the blood (solute clearance) and translocation of fluid (ultrafiltration).

After a PD catheter has been surgically placed into a patient’s lower abdomen, dialysate exchanges ideally are not initiated for 2 to 3 weeks to allow for adequate healing of the incision and to prevent leakage of dialysate fluid. A typical continuous ambulatory PD (CAPD) prescription consists of 4 exchanges of a dextrose solution per day, with each dwell containing 2 L of fluid. Generally, a final daily exchange is performed, and the solution is allowed to dwell overnight.

Continuous cycling PD (CCPD) is an automated exchange system that allows patients to connect their PD catheter to a computerized cycler during the night while they are sleeping. The cycler regulates the frequency and volume of the exchanges. A typical CCPD regimen consists of 3 to 5 infusions overnight, with a final infusion of fluid that may dwell for 12 hours during the daytime.

Initially, patients’ lifestyles and preferences are used to determine whether CAPD or CCPD is the most appropriate treatment modality. However, complications of PD may require modification of the modality and regimen of exchanges employed.

Complications of Peritoneal Dialysis

This review describes 3 complications that are commonly associated with peritoneal dialysis: PD-related peritonitis, ultrafiltration failure (UFF), and decreased effective solute clearance.

CASE PATIENT 1: PERITONITIS

INITIAL PRESENTATION

A 45-year-old man who is on CCPD presents to the emergency department (ED) with diffuse morning abdominal pain and cramping as well as a cloudy dialysate effluent.

History

The patient was started on CCPD 1 month ago after developing ESRD secondary to polycystic kidney disease. His treatment regimen consists of 5 2-L exchanges of 2.5% dextrose solution. After the last exchange for the day, the patient infuses 2 L of 1.5% dextrose solution to dwell for 6 hours. After some questioning, the patient reveals that he dropped the bag connector on the floor and connected it to the catheter without first sterilizing it.

Physical Examination

In the ED, the patient has an oral temperature of 38°C and a standing blood pressure of 142/82 mm Hg. Abdominal examination reveals diffuse tenderness and rebound pain. No expressible drainage is observed from the patient’s PD catheter site.

Initial Laboratory Testing

Laboratory studies reveal a peripheral leukocyte count of 15,000/mm³, 92% of which are neutrophils. The effluent contains 22,300 leukocytes, 90% of which are neutrophils. A Gram stain of the effluent reveals only neutrophils; no organisms are found. Liver function
Complications of Peritoneal Dialysis

tests are normal. An abdominal radiograph shows the catheter to be directed toward the left lower quadrant without free air.

DIFFERENTIAL DIAGNOSIS

Given this patient’s history of polycystic kidney disease, his abdominal pain could reasonably be explained by diverticulosis with perforation. Alternatively, his peritoneal signs also could result from other perforated viscera (eg, gallbladder, appendix, peptic ulcer). However, the abrupt onset of symptoms and lack of antecedent symptomatology (eg, postprandial upper abdominal discomfort) in this patient are suggestive of PD-related peritonitis.

- What clinical information is helpful for confirming a diagnosis of peritonitis?

Definition and Prevalence of Peritonitis

Peritonitis is a relatively common complication in PD patients. Most patients with peritonitis present with cloudy dialysate fluid, and abdominal pain or cramping is seen in 95% of patients. Occasionally, nausea and vomiting may be present in addition to fever. Peritonitis is diagnosed by the presence of more than 100 leukocytes/mm³ of dialysate, more than 50% of which should be neutrophils. If a patient presents early in the course of peritonitis, the initial fluid leukocyte count may be less than 100/mm³. If the index of suspicion is high for peritonitis, a subsequent cell count often confirms the diagnosis and should be obtained after several hours.

Patients on CCPD incur peritonitis at a rate of 0.51 episodes per patient-year, compared with 0.94 episodes per patient-year for patients on CAPD. The difference in incidence rates most likely is due to the fewer catheter connections that are performed by CCPD patients. In CAPD patients, the incidence of peritonitis also is related to the type of mechanism used. Use of newer dialysate tubing systems (eg, V-set) that allow for catheter flushing prior to installation of the dialysate into the abdomen has decreased the risk for developing peritonitis by 40%. In addition, use of “flush before fill” tubing systems has decreased the incidence rate for recurring peritonitis in CAPD patients from 1 case per 9 months to 1 case per 5 months.

Secondary versus PD-Related Peritonitis

A first step in determining the etiology of a patient’s peritonitis is to review the medical history to determine whether the current episode of peritonitis is PD-related or secondary to a perforation. Patients with secondary peritonitis present with symptoms similar to those common to PD-related peritonitis; in both situations, patients may appear to be toxic. The only early evidence that would suggest the presence of an intra-abdominal perforation is a Gram stain of the effluent that demonstrates both gram-negative and gram-positive organisms. It is incumbent upon the physician to consider the possibility of secondary peritonitis and the potential need for an abdominal computed tomography (CT) scan.

Most episodes of PD-related peritonitis develop when the bag-tubing or tubing-catheter connection becomes contaminated and the dialysate washes bacteria into the abdomen. Therefore, the physician should determine whether the patient had difficulty connecting the tubing with the bag or catheter. Nonsterile connections may lead to the introduction of Staphylococcus species into the PD fluid. Alternatively, patients may report drainage or discomfort around the catheter exit site during the preceding several days. These symptoms may indicate the presence of exit site or tunnel infections.

- What diagnostic tests are used to confirm the suspicion of PD-related peritonitis?

Diagnostic Testing

To determine whether the peritonitis is PD-related or secondary to a perforation, additional testing is necessary. A culture of the dialysate should be obtained; however, because culture results are not available for at least 24 hours, they are not useful early in a patient’s clinical course.

An initial Gram stain of the dialysate may be useful in identifying the infecting organism. However, a Gram stain of the dialysate is positive in less than 50% of culture-positive episodes of peritonitis. The most common gram-positive bacterium associated with PD-related peritonitis is Staphylococcus epidermidis, with Staphylococcus aureus the next most common microbe. (S. aureus is the most common organism in exit site infections.) Approximately 5% to 10% of peritonitis episodes are caused by gram-negative organisms, such as Escherichia coli and Pseudomonas species.

The finding of gram-negative organisms or a polymicrobial infection may indicate the presence of an intra-abdominal process such as diverticulitis. Imaging studies such as CT scans of the abdomen are useful to confirm a clinical suspicion of secondary peritonitis. Intra-abdominal air may be seen as a result of the catheter and does not necessarily indicate perforation. In less than 10% of cases, peritonitis also may result from infection with Candida organisms or other fungi.
What therapeutic interventions are used to treat peritonitis?

TREATMENT OPTIONS

If an infecting organism is identified by Gram stain of the peritoneal fluid, a directed antimicrobial regimen should be initiated (Table 1). However, the Gram stain usually is inconclusive, and initial antibiotic therapy should target both typical gram-positive and gram-negative organisms. Choice of antibiotic therapy can be narrowed following results from culture studies. Because of the emergence of vancomycin-resistant Enterococcus species, clinicians are discouraged from empirically using vancomycin unless the patient is known to be a carrier of methicillin-resistant S. aureus or is allergic to cephalosporins. If the patient responds rapidly to the antibiotic regimen, with alleviation of symptoms and decrease of the effluent leukocyte count to normal, therapy should be continued for 2 weeks for gram-positive organisms. If a gram-negative microbe is the cause of infection, therapy should be individualized based on the difficulty of eradicating these infections. Catheter removal also may be necessary if the gram-positive infection does not respond within 72 hours after the initiation of therapy or if a relapse of peritonitis occurs. The latter suggests that a tunnel infection is present or that the catheter cannot be sterilized. Fungal peritonitis may be treated without catheter removal but requires prolonged antimicrobial therapy and vigilance to contain and eradicate the infection.

CASE 1 RESOLUTION

The patient initially is treated with cefazolin and gentamicin; after his PD fluid cultures yield S. aureus sensitive to cefazolin, his treatment is limited to cefazolin. The patient’s symptoms abate, and his effluent leukocyte count rapidly decreases. He is treated for 2 weeks with oral cephalexin and undergoes additional training in the PD clinic regarding sterile techniques.

CASE 2: ULTRAFILTRATION FAILURE

INITIAL PRESENTATION

A 50-year-old woman on CAPD presents to her nephrologist with increasing bilateral ankle edema and dyspnea on exertion. In addition, the volume of her CAPD drains has decreased, and she develops mild pain when she drains her abdomen dry.

History

The patient was started on CAPD 5 years ago after developing renal failure as a complication of chronic glomerulonephritis. During the 3 months prior to the current presentation, her weight increased 10 lb. The patient’s medical history is otherwise notable for a negative cardiac exercise stress test performed 2 months ago by her primary care physician for the new onset of dyspnea. Her medications include estrogen replacement therapy; calcium carbonate (500 mg orally 3 times daily after meals), and erythropoietin (3500 U subcutaneously twice weekly). Her PD regimen consists of 5 exchanges per day using alternating 1.5% and 2.5% dextrose dialysate mixtures in 2-L volumes with no additives.

Physical Examination

Physical examination reveals a supine blood pressure of 176/100 mm Hg, pulsus paradoxus of 4 mm Hg, respiratory rate of 18 breaths/minute, and pulse that is 86 bpm and regular. The patient weighs 132.3 lb (60 kg)—8.8 lb (4 kg) above her estimated dry weight—with no dialysate. Jugular venous pulsations are seen at 8 cm at a 30° angle. Cardiac examination reveals an S3 and an early systolic ejection murmur at the right upper sternal border without radiation. Pulmonary examination reveals dullness at the lung bases bilaterally. Abdominal examination reveals a PD catheter in the left lower quadrant and no evidence of organomegaly. There is 1+ pitting edema to her midcalves bilaterally without tenderness.

Initial Laboratory Testing

Initial laboratory studies reveal the following: sodium, 134 mEq/L; potassium, 3.5 mEq/L; blood urea nitrogen (BUN), 60 mg/dL; creatinine, 6.2 mg/dL; hemoglobin, 11 mg/dL; and white blood cell count, 7800 cells/mm³. An electrocardiogram (ECG) reveals a normal sinus rhythm, normal axes, and intervals with
nonspecific ST-T wave changes that are unchanged from the baseline ECG performed at her stress test. Chest radiography reveals bilateral effusions and no cardiomegaly or infiltrates.

• What are possible causes of this patient’s dyspnea?

DIFFERENTIAL DIAGNOSIS

Inadequate ultrafiltration is a common cause of pulmonary edema and dyspnea in patients on CAPD, but other causes of dyspnea must first be considered (Table 2). Ruling out underlying cardiac disease that produces a decreased ejection fraction should be a primary concern given its prevalence in patients on PD. However, this patient’s recent cardiac stress test and unchanged ECG provide significant evidence against this diagnosis. Likewise, obstructive cardiac outflow from aortic or mitral valve disease is not suggested by this patient’s cardiac examination. Although shortness of breath in a dialysis patient also may be the presenting symptom of a pericardial effusion or tamponade, normal cardiac size on chest radiograph, normal voltage, and the absence of electrical alternans argue against this etiology. However, if the pretest probability for this etiology is high, a transthoracic echocardiogram may be used to confirm the diagnosis.

For unclear reasons, some patients on PD develop a hydrothorax resulting from leakage of the dialysate into the pleural cavity (usually on the right side) via diaphragmatic channels. This diagnosis is confirmed by a high glucose concentration in the pleural fluid.

Given this patient’s lack of cardiac or other conditions, the most likely cause of her dyspnea is inadequate ultrafiltration leading to pulmonary edema.

Table 2. Causes of Dyspnea in Peritoneal Dialysis Patients

<table>
<thead>
<tr>
<th>Cause</th>
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<tbody>
<tr>
<td>Aortic stenosis</td>
</tr>
<tr>
<td>Cardiac ischemia</td>
</tr>
<tr>
<td>Hydrothorax</td>
</tr>
<tr>
<td>Inadequate ultrafiltration</td>
</tr>
<tr>
<td>Mitral regurgitation</td>
</tr>
<tr>
<td>Pericardial effusion or tamponade</td>
</tr>
<tr>
<td>Pneumonia</td>
</tr>
<tr>
<td>Pulmonary edema</td>
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<tr>
<td>Pulmonary embolism</td>
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</tbody>
</table>

Note: This list does not include all possible causes of dyspnea in peritoneal dialysis patients.

Table 3. Peritoneal Dialysis–Related Causes of Abdominal Pain

<table>
<thead>
<tr>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catheter-related problems</td>
</tr>
<tr>
<td>Dialysate hypertonicity</td>
</tr>
<tr>
<td>Hernia</td>
</tr>
<tr>
<td>Intolerable volume</td>
</tr>
<tr>
<td>Peritonitis</td>
</tr>
</tbody>
</table>

• What is the most likely cause of this patient’s abdominal pain?

As with the situation of case patient 1, peritonitis may result in abdominal pain in patients on PD. However, in the absence of infection, patients may experience pain during any phase of a PD cycle (Table 3). Infusion of the acidic, hypertonic dialysate may cause discomfort, especially in patients who have recently begun PD. The volume of the fluid also may lead to overdistention and pelvic floor pressure. Temporarily reducing the fluid volume or infusing it at a slower rate may help to alleviate these symptoms. Pain at the end of the drain may be due to the rapid loss of volume and intraperitoneal irritation by the catheter. Allowing a small amount of fluid to remain may alleviate this symptom.

Increased intra-abdominal pressure from the dialysate may lead to hernias around the catheter or incision sites. Hernias are more likely to occur in patients on CAPD than in those on CCPD, most likely because of the position of the patient during dialysate dwelling. Intra-abdominal pressure is greater when patients stand while fluid is in the abdomen compared with when they are lying down. Incarcerated hernias may develop infrequently. Hernias that become enlarged or continue to produce discomfort may require repair, and patients should be converted to hemodialysis for several weeks to allow for healing. If hemodialysis is not an option, the patient should be placed on a cycler at night (ie, CCPD) and remain free of dialysate during daytime hours when the patient is ambulatory.

FURTHER LABORATORY TESTING OF CASE PATIENT

The patient’s dialysate effluent is analyzed and found to contain 50 leukocytes, 90% of which are lymphocytes. An abdominal radiograph reveals the catheter to be directed toward the left lower quadrant. A rapid exchange of 2 L of 2.5% dextrose solution produces 2010 mL of effluent. Based on these findings, a peritoneal equilibration test (PET) is performed. The patient’s dialysate to plasma (D/P) urea ratio is 0.90.
and her D/P creatinine ratio is 0.72 at 2 hours. Based on this finding, the patient is classified as having type I UFF.

• What are potential reasons for decreased drain volumes in this patient?

DECREASED DRAIN VOLUME

Decreased drain volume in patients on PD may result from catheter-related problems, changes to the peritoneal membrane, or other factors (Table 4).

Catheter-Related Problems

Periodically, patients may notice that the volume of fluid drained is less than the volume instilled. This problem can be confirmed by a rapid 2-L exchange of a 2.5% dextrose dialysate solution that is instilled for 30 minutes and allowed to drain over 10 to 15 minutes. A drain volume less than 2 L is indicative of a catheter-related problem. The most common problems responsible for decreased drain volumes are altered bowel motility, fibrin production, and catheter migration.

Altered bowel motility. Constipation is a frequent cause of outflow problems in patients on PD. Distended bowel lumina tend to compress the catheter in an intraabdominal location that obstructs both inflow and outflow of dialysate. Use of cathartic and laxative agents can rectify this problem, although phosphate-based medications should be avoided due to the diminished clearance of phosphate in patients with renal disease.

Fibrin production. The hypertonicity of the dialysate may stimulate production of new fibrin strands that may occlude the catheter. The appearance of fibrin also may indicate the beginning of an infection, and a cell count of the effluent should be obtained. When a patient notices fibrin in his or her drained dialysate, heparin should be added to the new dialysate bags. If poor flows (ie, slow drainage rates) are obtained, urokinase may need to be instilled into the catheter to attempt dissolution of occluding fibrin strands.

Catheter migration. Catheter movement may be responsible for obstructive outflow, which results from the attachment of omentum or adhesions (ie, scar tissue) to the catheter. The catheter should be directed toward the pelvis, as confirmed by abdominal radiography. If the catheter is directed cephalad, it should be repositioned.

Changes to the Peritoneal Membrane

Approximately one third of all patients on PD cannot be maintained on PD after 6 years secondary to changes in the characteristics of the peritoneal membrane. Patients with UFF typically are unable to control their fluid status, which leads to the development of edema in their extremities and potentially to congestive heart failure.

Although the etiology of UFF is uncertain, some researchers postulate that prolonged exposure to acidic and hypertonic substances stimulates connective tissue cells to produce fibrin that decreases the permeability of the peritoneal membrane. In contrast, recurrent episodes of peritonitis can cause increased permeability of the membrane to dextrose. Peritonitis will not allow the required osmotic gradient to be maintained, and effective clearance of solutes or fluid removal cannot occur. A PET can be performed to aid in determining the characteristics of a patient’s UFF.

To perform a PET, 2 L of 2.5% dextrose dialysate are infused for a dwell time of 4 hours. The abdomen is drained and the recovered volume measured. At specific intervals (eg, 2 or 4 hours), blood and dialysate are sampled for urea and creatinine content. The concentration of glucose also is measured and compared with the initial dialysate glucose concentration. These measured values are then plotted against a nomogram; based on the D/P ratio for urea or creatinine, patients can be categorized as high, high average, low average, or low transporters (Figure 1). For example, a patient who is a high transporter—defined by the rapid diffusion of creatinine and urea across the peritoneum—will have a D/P urea ratio greater than 0.83.

There are 4 types of UFF (Table 5), and the results of a PET are useful for identifying patients with type 1 and type 2. Type 1 UFF is more common than type 2 UFF and results from the high permeability of the peritoneal membrane to glucose. Prolonged exposure of the dialysate to the overly permeable membrane causes the solute gradient to dissipate. Without an osmotic gradient between the capillaries and dialysate, ultrafiltration cannot occur and PD ceases to be an

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**Table 4. Causes of Decreased Drain Volumes and Volume Overload in Peritoneal Dialysis Patients**

<table>
<thead>
<tr>
<th>Cause</th>
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<tbody>
<tr>
<td>Catheter migration or kinking</td>
</tr>
<tr>
<td>Constipation</td>
</tr>
<tr>
<td>Noncompliance with treatment regimen</td>
</tr>
<tr>
<td>Production of new fibrin strands</td>
</tr>
<tr>
<td>Ultrafiltration failure</td>
</tr>
</tbody>
</table>

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**Table 5. Types of UFF**

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>High permeability to glucose</td>
</tr>
<tr>
<td>2</td>
<td>Lower permeability to glucose</td>
</tr>
<tr>
<td>3</td>
<td>High permeability to urea</td>
</tr>
<tr>
<td>4</td>
<td>Lower permeability to urea</td>
</tr>
</tbody>
</table>
effective treatment modality. However, decreased solute transport is not associated with type 1 UFF, and clearance of urea and creatinine remains adequate.

In type 2 UFF, by contrast, the osmotic gradient driving ultrafiltration does not dissipate. Rather, the decreased permeability of the peritoneal membrane impairs urea and creatinine clearance and little ultrafiltration occurs.

Two additional types of UFF also have been described. Type 3 UFF exists as a result of high lymphatic reabsorption of fluid and solutes. Type 4 UFF is believed to be due to abnormal water channels. The prevalence of these types of UFF is uncertain, and presently they cannot be diagnosed without research techniques.

### Other Etiologies of Decreased Drain Volume

Additional causes of decreased drain volume include hydrothorax and bends or kinks in the catheter. In the course of traversing the subcutaneous fat and muscle tissue, occasionally the catheter may bend. This problem usually is detected early following catheter placement and should be considered if flow is improved when pressure is applied to the subcutaneous tunnel. Replacement of the catheter generally is required to rectify this problem. As described previously, a hydrothorax results in decompression of intra-abdominal fluid into the chest. Patients with hydrothorax may slowly or rapidly accumulate fluid, with presenting symptoms determined by the rate of accumulation.

- What are treatment options for this patient?

### Table 5. Classification of Ultrafiltration Failure

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Possible Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 High permeability, low UF</td>
<td>Rapid exchanges</td>
</tr>
<tr>
<td>Type 2 Low permeability, low UF</td>
<td>May need HD</td>
</tr>
<tr>
<td>Type 3 High lymphatic reabsorption</td>
<td>More exchanges, CCPD, or NIPD</td>
</tr>
<tr>
<td>Type 4 Water channels</td>
<td>NIPD or CCPD, may need HD</td>
</tr>
</tbody>
</table>

CCPD = continuous cycling peritoneal dialysis; HD = hemodialysis; NIPD = nightly intermittent peritoneal dialysis; UF = ultrafiltration.
TREATMENT OPTIONS

Figure 2 presents a diagnostic and treatment algorithm for patients with persistent volume overload despite undergoing dialysis. Understanding the pathophysiology of this patient’s problem facilitates decisions regarding treatment options. The patient’s underlying problem is the high permeability of her peritoneal membrane to solutes, combined with inadequate ultrafiltration of fluid (type 1 UFF). For patients with type 1 UFF, reducing dwell times may maintain the osmotic gradient necessary for ultrafiltration without compromising urea and creatinine clearance. Changing the patient’s treatment modality to nightly intermittent peritoneal dialysis (NIPD) may achieve this result. Patients who do not have preserved intrinsic renal clearance (ie, creatinine clearance of 3 to 5 mL/minute) also may need to perform 1 or 2 short exchanges during the daytime. Patients who cannot use a cycler (eg, because the machine makes frequent alarm sounds that disturb their sleep) may need to be converted to hemodialysis with a trial of CCPD or CAPD in the future.

CASE 2 RESOLUTION

The patient prefers to remain on PD, and her treatment is changed to NIPD. The new treatment regimen consists of 8 exchanges during a 10-hour period with alternating 2.5% and 4.25% dextrose solutions in 2-L volumes. Her pain at the completion of the drain phase of her exchanges resolves without intervention and is believed to have resulted from catheter irritation.

After 1 month on the NIPD regimen, the patient’s fluid balance improves but her outpatient laboratory results consistently reveal morning serum glucose levels greater than 200 mg/dL. The patient is started on an oral hypoglycemic agent that improves her glucose control. However, over the next 3 months, she has progressive UFF and is converted to hemodialysis.

CASE PATIENT 3: DECREASED EFFECTIVE SOLUTE CLEARANCE

INITIAL PRESENTATION

A 45-year-old man presents to his nephrologist with symptoms of fatigue, occasional nausea, and an inability...
to focus thoughts for more than a few minutes at a time. The patient is concerned that these symptoms are similar to those he experienced prior to initiation of his CAPD therapy.

**History**

The patient began therapy with CAPD 2 years ago following an episode of membranous nephropathy. He denies any change in diet and uses only prescribed medications.

**Physical Examination**

Physical examination reveals a blood pressure of 130/90 mm Hg, pulse of 80 bpm, and normal temperature. Otherwise, the examination is unremarkable and unchanged from previous visits. The insertion site of his PD catheter is without erythema, and his abdomen is soft and nontender. There is 2+ pitting edema of the legs.

**Initial Laboratory Testing**

Initial laboratory studies reveal the following: hemoglobin, 10.6 mg/dL; BUN, 130 mg/dL; and creatinine, 9.6 mg/dL. Electrolytes are normal. BUN and creatinine levels 7 months ago were 65 mg/dL and 5.1 mg/dL, respectively.

- What are the reasons for decreased effective solute clearance in peritoneal dialysis?

**CAUSES OF DECREASED EFFECTIVE SOLUTE CLEARANCE**

Decreased effective solute clearance may be the result of an inadequate PD prescription, changes in the peritoneal membrane, decline in residual renal function, patient body size, and patient noncompliance with the treatment regimen.

**Inadequate Treatment Modality and Prescription**

In patients on PD, the dialysis modality and prescription should be reassessed regularly to ensure that solute clearance and ultrafiltration are adequate. In many patients, the initial modality and prescription are correct but become inadequate due to intervening complications, such as altered peritoneal membrane characteristics or noncompliance with the treatment regimen.

PD is likely to be adequate if a patient’s Kt/V value for urea is at least 2.0/week and creatinine clearance is at least 60 L/week/1.73 m². Kt/V is a dimensionless term that denotes the adequacy of urea removal per week; Kt/V indicates the total volume of plasma cleared of urea per week, normalized to total body water. These clearance parameters are achieved through a combination of the patient’s residual renal function and the efficacy of the dialysis treatments. A change in the peritoneal membrane or a decline in residual renal function over time will require changes in the dialysis prescription or modality.

**Changes to the Peritoneal Membrane**

Disorders of the peritoneal membrane also may contribute to a decline in dialysis efficiency. Under normal conditions, solute clearance in PD is a direct function of the volume of fluid removed. In most patients, the D/P creatinine concentration increases slowly to a maximum value at 4 to 6 hours of dwell time. At that point, the volume of fluid removed will be the key determinant of the quantity of creatinine removed (Figure 3). As noted in the discussion of case patient 2, a decline in peritoneal membrane permeability to solutes may lead to a decline in solute clearance, even if fluid removal is apparently adequate. The slow movement of creatinine from blood to peritoneal fluid is the limiting feature. This defect may result from several factors, including an inordinately high uptake of solute into the peritoneal lymphatics and sclerosing encapsulating peritonitis. The latter is a relatively rare and serious condition associated with a high mortality and is characterized by severe intra-abdominal adhesions that result in declining surface area for peritoneal transport and, ultimately, episodes of bowel obstruction.

**Decline in Residual Renal Function**

Loss of residual renal function can be monitored through measurements of a patient’s weekly creatinine clearance. For example, a patient who maintains a glomerular filtration rate of 5 mL/minute will have a weekly creatinine clearance of approximately 50 L/week. For patients on PD, creatinine clearance per exchange is a complex result of the rate of equilibration of glucose across the peritoneal membrane (Figures 3–6). If such a patient has 5 2-L exchanges per day, her average creatinine clearance per exchange will be approximately 1 L of creatinine per liter of fluid exchanged or approximately 70 L/week. Therefore, a patient whose peritoneal transport characteristics decline in a manner that leads to less efficient dialysis (ie, high transporter or low transporter based on PET results) and who has a simultaneous decline in residual renal function may experience a total weekly creatinine clearance of less than 60 L/week and become uremic.

**Patient Body Size**

Patient body size also affects the suitability of PD as a dialytic modality. Large patients may not achieve
Figure 3. Relationship between dialysate and plasma creatinine (D/P creatinine ratio) over time. The dialysate initially is free of creatinine, and the rate of entry of creatinine across the peritoneal membrane is most rapid because the concentration gradient is the greatest. As the gradient dissipates over time, the rate of entry slows until equilibration is achieved. The clearance of creatinine may be plotted across the peritoneum as it is across the kidney: Creatinine clearance = U × V/P, where U represents dialysate creatinine concentration (mg/dL), V is dialysis volume (L), and P is plasma creatinine concentration (mg/dL). Therefore, the limiting factors influencing creatinine removal are the rate of equilibration and the volume of fluid removed.

Figure 4. Dissipation of the glucose gradient in the peritoneal space over time. Glucose at hypertonic concentration (1.5% or 4.25%) is absorbed across the peritoneal membrane, thereby reducing the osmotic gradient for fluid movement across the peritoneal membrane from the plasma. In individuals who are high transporters (see Figure 1), dissipation of the osmotic gradient occurs more quickly than normal and reduces the flow of fluid across the membrane. A reduced flow across the membrane results not only in a deficit of fluid removal and potential fluid overload, but it also may limit solute removal and reduce creatinine clearance.

Figure 5. Rate of fluid removal in the average patient on peritoneal dialysis. The osmotic gradient between peritoneal fluid and plasma is greatest during the beginning of an exchange, and the rate of fluid removal is therefore greatest during that time. As glucose is absorbed and the gradient begins to diminish, the rate of fluid removal slows and asymptotically approaches 0 by 4 hours.

Figure 6. Creatinine clearance across the peritoneal membrane over time. The close parallel between the curves for solute entry into the peritoneal fluid (Figure 3) and fluid accumulation (Figure 5) defines the creatinine clearance curve. Any reduction in solute diffusion (eg, defect in the membrane) or fluid removal (eg, rapid dissipation of the glucose gradient) will compromise creatinine clearance and efficacy of dialysis.
adequate clearances using the recommended dwell volumes or number of exchanges. Patients weighing greater than 176.4 lb (80 kg) may need 3-L volumes and 5 exchanges per day if using CAPD. If such patients are using CCPD, the duration of cycling may need to be increased by 10% as well as supplemented by daytime exchanges.

Patient Noncompliance

Some patients who receive an adequate initial dialysis prescription and appropriate modality of PD treatment may manifest a decline in adequacy of dialysis over time, as seen in case patient 3. Therefore, the prescribing physician must be certain that the patient actually is receiving the dialysis treatment as prescribed. For example, chronic depression is a significant problem for both PD and hemodialysis patients and may lead patients to miss treatments or receive shortened treatments. Similarly, because CAPD is self-administered, patients who skip exchanges will manifest signs of uremia with no apparent cause. Noncompliance can be assessed using a PET, and results showing no evidence of deterioration in peritoneal function suggest that a patient is noncompliant.

CASE 3 RESOLUTION

A PET is performed and reveals a D/P urea ratio of 0.58, an ultrafiltration volume of 100 mL, and a residual renal clearance of urea of 2 mL/minute. The patient is classified as a low transporter (ie, low membrane permeability). Hemodialysis is initiated through a tunneled dialysis catheter for 6 weeks. A trial of CAPD is subsequently attempted, but the patient’s Kt/V is only 1.2/week. An arteriovenous graft is placed, and the patient is later evaluated and medically cleared to be listed for a cadaveric renal transplant.

REFERENCES