CHAPTER 182

Correction of Fluid, Electrolyte, and Acid-Base Derangements by Peritoneal Dialysis in Acute Kidney Injury

Raymond T. Krediet and Isaac Teitelbaum

OBJECTIVES

This chapter will:

1. Describe the key components of an acute peritoneal dialysis prescription in patients with acute kidney injury in the intensive care unit.
2. Explore the role of acute peritoneal dialysis in the management of patients with volume overload in the intensive care unit.
3. Discuss the correction of electrolyte abnormalities with the use of acute peritoneal dialysis.
4. Summarize the role of acute peritoneal dialysis in the correction of acid-base derangements.
5. Detail potential metabolic complications of acute peritoneal dialysis in the intensive care unit.

Acute kidney injury (AKI) is a common problem in the intensive care unit (ICU) and represents a clinically diverse entity. It is associated with significant morbidity, mortality, and financial expenditure.[1–2] Management involves the appropriate control of fluid balance, electrolyte status, and acid-base balance and the initiation of renal replacement therapy when appropriate. Numerous treatment options are available, although there is no consensus in the literature on the best method or ideal dialysis dose in the setting of AKI in the ICU. Peritoneal dialysis (PD) should be considered a viable option for the treatment of selected patients with AKI in the ICU.[3] A number of studies have shown that patients' survival is similar compared with those treated with hemodialysis.[4,5] Furthermore, PD is indicated especially in developing countries, where it is often the only available dialysis modality.[6] This chapter details the role of PD in correction of fluid, electrolyte, and acid-base derangements in the patient with AKI.

PRESCRIPTION FOR ACUTE PERITONEAL DIALYSIS

Physical Aspects

Once the decision is made to initiate PD, a prescription must be formulated on the basis of the particular clinical situation and therapeutic goals (Table 182.1). The details of the therapy instituted, in addition to fluid balance, should be recorded meticulously on flow sheets to facilitate future decisions about the patient's PD regimen. The PD prescription should be reviewed frequently and appropriate adjustments made on the basis of the patient's clinical parameters and laboratory investigations.

The length and the technique of PD must be determined. Session length can vary greatly depending on the cause and duration of AKI as well as the presence or absence of underlying chronic kidney disease. The length of the dialysis sessions also depends on the goals of fluid and solute removal. PD can be performed intermittently or continuously, either manually or with an automated cycling device. Techniques available for the treatment of AKI include acute intermittent PD, continuous equilibrating peritoneal dialysis, and high-volume PD.

Other components of the dialysis prescription include determination of the appropriate *exchange volume* and the dwell time. The exchange volume is influenced by several factors, including technique of PD being used, concomitant medical problems such as the presence of hernias or respiratory disease, the estimated size of the patient's peritoneal cavity, and any noted leakage of dialysate around the PD catheter. For example, in acute intermittent PD, an exchange volume for an average-size person without respiratory failure may be 2 L, whereas in a larger patient, it may be as much as 3.5 L. On the other hand, an exchange volume for a small person with acute respiratory distress syndrome may be only 0.5 to 1.5 L to prevent compromise of diaphragmatic excursion and respirations. Dialysis solutions should be warmed to body temperature before infusion to avoid discomfort and to enhance solute transport. The temperature of the dialysate can be especially advantageous in the management of hyperthermia and hypothermia.[7]

The *inflow period* is the time required to instill the dialysate into the peritoneal cavity. For manual exchanges, gravity is the primary determinant of this period, although the exchange volume, elevation of the dialysate bag, and presence of inflow resistance also play roles. To maximize the efficiency of PD, the inflow period must be kept to a minimum. A typical inflow period is 10 to 15 minutes. The *outflow period* is defined as the time needed to drain the peritoneal cavity of the effluent dialysate, which averages 20 to 30 minutes. This period consists of an initial fast segment lasting a few minutes, in which time approximately 80% of dialysate is drained; this segment is followed by a slower segment in which the remainder is emptied. Like the inflow period, the outflow period also must be kept to a minimum and is determined primarily by gravity. The time between the inflow and outflow period is referred to as the *dwell time:* the period in which the exchange volume remains in the peritoneal cavity. The standard dwell time for acute PD is approximately 30 minutes, the time in which the gradients for fluid and urea are most favorable. For continuous equilibrating PD, the usual dwell time ranges from 3 to 6 hours; for high-volume PD dwell times of various duration can be applied, but the total number of exchanges is divided over 24 hours during several days.

TABLE 182.1

The Various Components of an APD Prescription

COMPONENTS OF AN APD PRESCRIPTION	EXAMPLES
Length of dialysis session	≥24 hr, depending on the clinical need
Dialysate composition	Glucose concentration dependent on the hydration state
Dialysate additives	For example, heparin, insulin, antibiotics
Exchange volume	For example, 2 L
Number of exchanges	Dependent on serum urea, potassium, creatinine
Inflow period	10–15 min
Dwell time	20–30 min or longer with a CAPD scheme
Outflow period	20–30 min

APD, Automated peritoneal dialysis; *CAPD,* continuous ambulatory peritoneal dialysis.

PRESCRIPTION FOR ACUTE PERITONEAL DIALYSIS

Dialysis Solution

Once the technique of PD is decided upon, consideration must be given to the composition of the dialysate to be used for treatment. Standardized peritoneal dialysis solutions are commercially available. They are electrolyte solutions that also contain glucose (in the form of dextrose) as the osmotic agent and lactate as the buffer. The electrolytes are sodium, chloride, magnesium, and calcium. The glucose/dextrose concentrations are 1.36/1.5% (75 mmol/L), 2.27/2.5% (125 mmol/L), and 3.86/4.25% (214 mmol/L). The glucose concentration determines the osmotic strength of the dialysate by crystalloid osmosis. Every millimole of glucose creates an osmolarity of 1 mosmol/L; the small differences in the concentrations, expressed by weight, are dependent only on using the European or U.S. pharmacopeia but are identical on a molecular basis. The glucose concentration determines the degree of ultrafiltration obtained. Therefore the initial choice of glucose concentration is determined in part by the patient's volume status. In patients with more severe volume overload, a reasonable choice of glucose solution would be 3.86/4.25%; for patients who are hemodynamically unstable with only slight volume overload, an initial dialysis glucose solution of 1.36/1.5% or 2.27/2.5% may be more appropriate. Glucose-containing solutions can provide a substantial source of caloric intake in the critically ill patient and may require an intensive insulin regimen to prevent the development of hyperglycemia.[8] On the other hand, the use of glucose-containing PD solutions should be used with caution in the patient with severe respiratory failure, in whom administration of such solutions may worsen respiratory failure through the greater production of carbon dioxide.

Standard dialysate solutions use lactate (35–40 mmol/L) as the bicarbonate-generating base because of its high stability in the presence of calcium and magnesium. Bicarbonate buffered solutions are available, but not in all parts of the world. They consist of a two-compartment bag system to keep the bicarbonate separate from the calcium and magnesium until just before administration. In patients with reduced lactate metabolism, such as in those with hepatic failure, lactic acidosis, or severe septic shock, lactate-buffered solutions should be replaced by bicarbonate-buffered ones if possible.

Another approach to PD is through the simultaneous use of glucose-based and amino acid–based dialysates that are mixed immediately before administration with an automated device. The absorption of amino acids during a dwell is higher than that of glucose because of their lower molecular weight. This approach has the theoretic advantage of reducing amino acid loss and improving nitrogen balance, but their use is limited because of the nitrogen load. They can lead to mild acidosis and to elevations in urea concentrations,[9] which may not be well tolerated in a critically ill patient with AKI. A mixture of amino acids with glucose improved net-protein balance in stable adult PD patients,[10] but this mix has not been studied in adults with AKI. However, in a retrospective analysis of the use of PD solutions with amino acids in children with AKI, Vande Walle et al.[11] found that glucose reabsorption and protein loss were significantly lower with mixed amino acid solutions than with glucose alone, but without a difference in serum albumin levels.

Icodextrin is another agent that can induce ultrafiltration in PD. It is a mixture of high-molecular-weight glucose polymers that creates a stable colloid osmotic (oncotic) pressure gradient despite being isotonic. Icodextrin is especially effective during long dwells (8 hours or more), because the absorption of the polymers is limited because of their high molecular weight. Despite its importance in chronic kidney failure,[12,13] the role of icodextrin in AKI is limited, because mainly short dwells are applied in this situation.

The standard dialysate contains calcium in concentrations ranging from 1.25 to 1.75 mmol/L (2.5–3.5 mEq/L). Such concentrations of calcium typically result in the movement of calcium from the PD solution to the extracellular fluid, potentially helping to support blood pressure in the critically ill patient. Standard dialysate does not contain potassium, but it contains sodium (132 mEq/L/mmol/L) and magnesium (0.5–1.5 mEq/L/0./25–0.75 mmol/L). Other agents, such as heparin, insulin, antibiotics, and potassium, may be added to the dialysate as the clinical situation dictates.

FLUID REMOVAL

Fluid balance is a critical component of the care of patients in the ICU who have AKI. In fact, in a study by Ronco et al.[14] collecting data from 345 nephrology centers on five continents, continuous extracorporal renal replacement therapy (CRRT) was used in 52% of centers for indications other than AKI. Of these, the two most commonly reported conditions, which accounted for approximately half of the uses, were congestive heart failure and fluid overload. Overall fluid balance depends on the amount of fluid intake, which is countered by the amount removed by any remaining urine output and ultrafiltration. The titration of fluid removal with PD is not as easily achieved as that with hemodialysis or CRRT, because fluid removal occurs with PD through the crystalloid osmotic gradient for water from the patient's blood to the peritoneal cavity. Ultrafiltration generally is well tolerated with less hemodynamic instability than seen in other forms of renal replacement therapy because of the continuous nature of the therapy and the fact that no extracorporeal circulation is applied. This improved hemodynamic stability at least theoretically may lessen the insult to the acutely damaged kidneys. Fluid removal with PD also may permit the administration of

parenteral nutrition to help counter protein losses through the peritoneum, even in patients who otherwise may not have been able to tolerate the fluid load.

Large amounts of fluid can be removed in patients treated with acute PD, through a combination of larger fill volumes, higher number of exchanges, and greater tonicity of the dialysate. When this technique is employed, close attention must be paid to plasma sodium because hypernatremia may result from a phenomenon known as *sodium sieving*. This confusing terminology is used for the observation that the dialysate Na^+ concentration decreases during the first hour of a 3.86/4.25% glucose/dextrose–containing dialysis solution. This decrease occurs as a consequence of transcellular water transport through the endothelial water channel aquaporin-1, which is not accompanied by sodium transport because the latter is transported only through the interendothelial pores. Consequently, more water than sodium is removed. This can lead to marked hypernatremia in the setting of repeated rapid exchanges with hypertonic glucose solutions. After this initial phase of rapid water transport through aquaporin-1, it decreases because of peritoneal glucose absorption, while dialysate sodium increases continuously through diffusive and convective transport into the peritoneal cavity. Therefore sodium sieving is marked especially during short dwell times.

Although acute PD has the capacity to remove large amounts of fluid, its use probably should be avoided in cases of severe volume overload with impending respiratory failure in patients who are not supported by mechanical ventilation, because instillation of a large volume of fluid into the abdominal cavity may restrict respiratory excursion. On the other hand, patients with congestive heart failure (CHF) refractory to medical management may benefit from the use of acute PD. The acute treatment prescription of patients with CHF consists of initial small volumes, such as 0.5 to 1 L of hypertonic glucose solution, with short dwell times, 1 to 2 hours.[15] PD as treatment of CHF improves fluid status and reduces pulmonary capillary wedge pressure, increases cardiac output, and improves hyponatremia and quality of life; however, it is not known whether this therapy affects survival. Some reviews on the use of PD in CHF have been published recently.[16,17]

ELECTROLYTE ABNORMALITIES

PD can be used to correct electrolyte abnormalities in blood urea, creatinine, potassium, and plasma sodium concentrations. This modality enables the continuous correction of electrolyte imbalances with the gradual removal of nitrogenous waste products without the risk of dysequilibrium syndrome. In a prospective randomized study, Arogundade et al.[18] managed 40 patients with AKI, who were matched for age and clinical diagnosis, with either intermittent PD or hemodialysis. These investigators found that patients treated with PD had significant reductions in concentrations of urea (from 29 mmol/L to 13 mmol/L), creatinine (from 1694 μmol/L to 796 μmol/L), and potassium (from 4.8 mmol/L to 3.3 mmol/L). There were no significant differences in the reduction of these parameters between the two treatment groups. However, the hemodialysis group required more blood transfusions, and their treatment involved overall greater cost of dialysis and total cost of hospitalization compared with the PD group.

Acute PD may not be preferred, although it may be the only option in some developing countries for cases of life-threatening hyperkalemia. Nonetheless, it is beneficial in managing cases of hyperkalemia other than those that are directly life-threatening. PD not only allows for the gradual removal of potassium but also enhances the intracellular movement of potassium through the correction of metabolic acidosis and the stimulation of insulin production by the administration of intraperitoneal glucose. It is equally important to remember that with longer durations of dialysis, hypokalemia may ensue because standard PD solutions do not contain potassium. Hypokalemia can be corrected through the addition of potassium to the dialysate fluid or with oral or peripheral administration of a potassium supplement.

PD also can be used for the correction of sodium abnormalities associated with anuria and the inappropriate use of intravenous administration of fluids. In a study by Inagaki et al.[19] correction of severe abnormalities of plasma sodium was obtained through the use of individualized PD solutions. These researchers were able to successfully lower the plasma sodium concentration to 138 mEq/L/mmol/L in a severely hypernatremic patient with an initial value of 170 mEq/L/mmol/L by the administration of hypotonic (Na^+ 70 mEq/L/mmol/L) peritoneal solution. Likewise, in two patients with hyponatremia (plasma sodium concentrations of 113 mEq/L/mmol/L and 121 mEq/L/mmol/L), the authors raised plasma sodium concentrations to the normal range through the administration of PD solutions with a sodium concentration of 190 mEq/L/mmol/L. Similarly, hypercalcemia can be ameliorated through the use of dialysate either without calcium or with a lower calcium concentration than that of a standard solution.

ACID-BASE DERANGEMENTS

Correction of metabolic acidosis with PD in the setting of AKI occurs through the gain of alkali from the absorption of either lactate or bicarbonate from the PD dialysate. In the case of lactate-buffered solutions, lactate absorbed into the bloodstream can be converted to bicarbonate via the enzyme pyruvate dehydrogenase, which is found principally in the liver and muscles. A randomized study has examined the efficacy of lactate and bicarbonate-buffered solutions in the correction of metabolic acidosis in patients either with or without shock. Thongboonkerd et al.[20] studied a group of 20 subjects requiring acute PD who were classified further by the presence or absence of shock. They then were assigned randomly to treatment with either bicarbonate- or lactate-buffered PD solutions with an average exchange volume of 1.4 L with a dwell time of 30 minutes. By cycle 12, subjects in the shock group treated with bicarbonate-buffered solutions had a more rapid improvement and significantly higher blood pH (7.30 vs. 7.05) and plasma bicarbonate (21 vs. 14) values. These improvements remained statistically significant between the two groups through cycle 36. Overall plasma lactate levels were significantly lower in the group receiving the bicarbonate-buffered solution in the patients with shock (3.6 mmol/L vs. 5.2 mmol/L) and without shock (2.9 mmol/L vs. 3.4 mmol/L). However, the patients without shock had comparable improvements in blood pH and serum bicarbonate with either solution, and peritoneal urea and creatinine clearances were similar in all of the subgroups. Results of this study suggest that AKI associated with poor perfusion states, such as shock, lactic acidosis, and multiple-organ failure, preferably should be managed with bicarbonate-buffered solutions, when available. In line with findings in this study, Inagaki et al.[19] reported successful treatment of two cases of lactic acidosis with

a fluid mixed with distilled water, 10% sodium chloride, and 7% sodium bicarbonate. This therapy allowed for the diffusion of lactic acid from the extracellular fluid into the PD fluid and diffusion of bicarbonate from the PD fluid into the extracellular space.

PD also can be used to correct metabolic alkalosis. The study by Inagaki et al.[19] also examined nine patients who had either AKI or chronic renal failure and in whom hemodialysis was unable to correct metabolic alkalosis. The authors found that by administering normal saline as the primary component of the dialysate, they were able to correct the metabolic alkalosis through the shift of bicarbonate from the extracellular space into the PD solution and of chloride from the PD solution into the bloodstream.

CONCLUSION

Acute PD is an often-overlooked therapy for dialytic support in patients with AKI in the ICU. When used in the appropriate clinical setting, this modality has the capacity to correct fluid balance disturbances, electrolyte abnormalities, and acid-base derangements. One of the keys to successful implementation of this form of renal replacement comes from the careful attention to all components of the PD prescription along with meticulous monitoring of the therapy. Furthermore, it has the distinctive advantage of better hemodynamic stability secondary to its continuous nature without the need for anticoagulation.

Key Points

1. The peritoneal dialysis prescription for acute renal failure must be individualized to fulfill the specific and often changing needs of the critically ill patient, particular attention being paid to volume status, respiratory status, and the presence of any metabolic abnormalities.
2. Bicarbonate-buffered dialysate solutions are preferred over lactate-buffered solutions in the setting of impaired lactate metabolism such as that seen in shock, lactic acidosis, and hepatic failure.
3. Short dwell times, which often are used in the clinical setting of volume overload, may place the patient at risk for significant hypernatremia as a result of sodium sieving.
4. Peritoneal dialysis may be efficacious in settings other than acute renal failure in the intensive care unit, such as for treatment of hypothermia, hyperthermia, and congestive heart failure.
5. Through individualized prescription, peritoneal dialysis is a viable and safe option for the correction of severe abnormalities of serum sodium and calcium concentrations, metabolic acidosis, and metabolic alkalosis.

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A complete reference list can be found online at ExpertConsult.com.

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