REVIEWS

APPROACH TO THE METABOLIC IMPLICATIONS OF PERITONEAL DIALYSIS IN ACUTE KIDNEY INJURY

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During the 1970s and 1980s, peritoneal dialysis (PD) was widely accepted as the standard treatment for acute kidney injury (AKI). However, advances in the techniques of extracorporeal blood purification gradually reduced its use, making PD an underused modality in this context. Although PD for AKI is an underutilized modality worldwide, it is frequently used in developing countries because of its lower cost and minimal infrastructure requirements. Recent studies have shown that PD administered continuously through a flexible catheter and cycler is an effective treatment in AKI because it ensures adequate fluid status and metabolic control. However, the use of PD in AKI has several limitations, such as the need for an intact peritoneal cavity and, in emergency situations such as severe fluid overload and severe hyperkalemia, an efficacy that is lower than that with extracorporeal blood purification techniques. Metabolic, infectious, and mechanical disorders related to PD are also limitations.

Among the metabolic complications of PD are hyperglycemia, hyponatremia, protein loss into the dialysate, and hypercatabolism. Hyperglycemia is caused by the use of dialysate containing high concentrations of glucose. Hyponatremia is a result of short dialysate dwell times during the rapid exchanges of high-volume PD. Protein loss into the dialysate can reach 48 g daily, worsening the nutrition status of patients already depleted by AKI. Severe hypercatabolism caused by PD remains controversial and occurs because PD methods cannot provide an adequate dialysis dose for AKI patients.

Few studies have assessed the metabolic implications of PD in AKI patients. Evaluation of these implications is relatively simple, imposes no additional costs, and can

provide information about the severity of the disease. Evaluation could also guide the selection of therapeutic, dialytic, and nutrition measures, preventing metabolic complications. The present manuscript describes the metabolic implications of PD and reviews the literature on how to prevent metabolic complications.

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Acute kidney injury (AKI) is a complex disorder defined as a rapid decline in glomerular filtration rate resulting in retention of metabolic waste products and in disturbances of fluid, electrolyte, and acid–base balance (1). This common disorder affects approximately 5% – 7% of all hospital inpatients and up to 30% of patients in intensive care units (2). In AKI, prognosis is poor, and mortality ranges from 40% to 80% (2–6) despite advances in the management of intensive care unit patients and improvements in dialysis techniques (2). Several factors have contributed to this current trend, especially the greater number and severity of comorbidities found in today's patients.

The great impact of AKI on mortality might be related to its metabolic and immunologic consequences, and to its induction of a proinflammatory state, with cytokine release and increased production of stress mediators that lead to insulin resistance and increased proteolysis, glycogenolysis, gluconeogenesis, and lipolysis. As a result, patients with AKI develop hyperglycemia, hypertriglyceridemia, and hypercatabolism (7).

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Patients with AKI might require dialysis. Two major types of renal supportive therapy are currently available for patients with AKI: peritoneal dialysis (PD) and hemodialysis (HD). Both can be performed intermittently or continuously (2–7). Because evidence for the superiority of either method for AKI patients is still lacking, the choice of treatment has so far depended on the experience of the nephrologist and the clinical condition of the patient at presentation (1).

PD FOR AKI

The first modality of renal replacement therapy used for AKI was PD. In the 1970s, PD was widely accepted for AKI treatment, but its use progressively declined in favor of HD (8–10). Multicenter studies have showed variable use of PD in AKI, with values ranging from 3% to 30% (11–13).

Several techniques used in chronic patients have been adapted for acute patients. Intermittent PD (IPD) is characterized by short exchanges, 20–30 per session, performed manually through a rigid catheter, with the dialysate remaining in the peritoneum for short periods of time (30 minutes) in sessions of 16–24 hours, two or three times weekly (14,15). Although widely used, IPD is inadequate for critical and hypercatabolic AKI patients because of its low small-solute clearance (16).

Chronic equilibrated PD is a modified form of continuous ambulatory PD (CAPD). It consists of 4–6 manual exchanges daily, with long dwell times (4–6 hours), using a flexible catheter. It is frequently used in developing countries because of its lower cost and minimal infrastructure requirements. However, clearance of small molecules might be insufficient, leading to metabolic inadequacy (16,17).

In tidal PD (TPD), only a portion of the dialysate is drained from the peritoneal cavity after the initial PD fluid exchange. The drained volume is replaced by fresh dialysate (the tidal volume), with each cycle leaving a variable amount of dialysate (residual volume) in constant contact with the peritoneal membrane until the end of the dialysis session, when the fluid is drained as completely as possible. Originally, TPD was designed to improve the efficiency of the dialysis technique by shortening fluid inflow and outflow times and optimizing the duration of dialysate contact with the peritoneal membrane (16,17). However, compared with other PD modalities, TPD achieves higher small-solute clearances only when very high dialysate flow rates—more than 20 L per session—are used (16,17). In a prospective randomized trial in patients with AKI (18), TPD provided higher solute clearances than chronic

equilibrated PD (creatinine: 9.94 ± 2.93 mL/min vs 6.74 ± 1.63 mL/min; urea: 19.85 ± 1.95 mL/min vs 10.63 ± 2.62 mL/min; $p = 0.001$).

Continuous-flow PD (CFPD) uses a fixed intraperitoneal volume and fast, continuous movement of dialysate into and out of the peritoneal cavity. The dialysate inflow and outflow occur simultaneously through two peritoneal access routes. Dialysate flow rates up to 300 mL/min can be used. As a result, high volumes of sterile dialysis fluid are required. Compared with chronic equilibrated PD, CFPD maximizes the transperitoneal solute concentration gradient, which enhances solute transport and clearance. Small-solute clearances during CFPD have been reported to be 3–8 times those achieved during conventional automated PD (19–23). However, CFPD requires large dialysate volumes and still has not been tested in adult AKI patients. Furthermore, because CFPD uses two peritoneal access routes, it has been associated with a higher rate of catheter-related mechanical complications (16).

High-volume PD (HVPD)—which has a mortality rate similar to that of other dialysis modalities (conventional daily HD and nocturnal HD)—has lately become an option for promoting adequate metabolic and fluid control in selected AKI patients. An increasing number of publications concerning its use, indications, and limitations in AKI patients are now available (9,10,24–26). In HVPD, a large volume of dialysate is delivered through a flexible catheter by a cyclor in short exchanges (18–22 daily, with 30–60 minutes of dwell time) during 24-hour continuous sessions 7 days per week (8,24,25,27). Ponce *et al.* showed that HVPD provides a Kt/V of 0.5 per session, with adequate metabolic control from the fourth day of treatment and mortality similar to that in patients treated with conventional and prolonged daily HD: that is, 55%–60% (9,24–27).

Figure 1 shows the PD modalities used in AKI.

The placement and maintenance of the catheter in the peritoneal cavity, the presence of low-pH hyperosmolar solution, and the use of the peritoneum as a semipermeable membrane may give rise to PD-related mechanical, infectious, and metabolic complications (29).

METABOLIC IMPLICATIONS OF PD

Few studies have looked at the metabolic repercussions of PD in patients with AKI. Some of the major complications, such as protein loss into dialysate, glucose absorption, protein catabolism, and electrolyte disorders can aggravate the already critical metabolic status of AKI patients and thus increase the impact of AKI on mortality.

Glucose Absorption: Glucose is the main osmotic agent in PD. The amount of glucose absorbed during PD significantly differs from patient to patient because of differences in peritoneal membrane permeability. Fast transporters of low molecular weight solutes can absorb glucose more quickly than slow transporters do. Among patients undergoing CAPD, 60% – 80% of the glucose instilled is absorbed (30).

In automated PD, glucose absorption is reduced because the number of cycles is higher and the dwell times are shorter. Perez and colleagues (31), assessing the effects of cycle frequency in 18 APD patients, observed that the percentage of glucose absorbed dropped with higher cycle frequency: 46.8% ± 14.2% for 5 two-liter exchanges, 38.7% ± 11.4% for 7 two-liter exchanges, 32.8% ± 11.1% for 9 two-liter exchanges, and 39.3% ± 15% for 14 L of TPD.

Figure 1 — Peritoneal dialysis modalities used in acute kidney injury. Adapted from Ponce *et al.* (28). IPD = intermittent peritoneal dialysis (PD); CEPD = chronic equilibrated PD; HVPD = high-volume PD; TPD = tidal PD; CFPD = continuous-flow PD.

Glucose absorption from dialysate can easily be calculated provided that the 24-hour drained dialysate is measured:

$$\text{Glucose absorption (g)} = [\text{glucose concentration of infused dialysate} \times \text{infused dialysate volume}] - [\text{glucose concentration in drained dialysate (g/L)} \times \text{drained dialysate volume}]$$

where the glucose concentrations are given in grams per liter, and the volumes are given in liters.

In patients with AKI, a classic study aimed to estimate the uptake of glucose, because values had ranged from 40% to 50% (32). But the calculation used formulae developed for chronic renal failure patients that had not been validated for acute patients. A study by Góes *et al.* (33) in 31 AKI patients treated with 208 HVPD sessions observed that absorption of glucose remained at approximately 35.3% ± 10.5% during treatment, and that glucose uptake was higher in patients with a higher Acute Tubular Necrosis Individual Severity Score and with more inflammation (higher C-reactive protein).

Table 1 shows glucose absorption rates in the various PD modalities.

The absorption of glucose from dialysate might cause or worsen hyperglycemia (18,25,27), which is indeed a common complication in hospitalized patients. Hyperglycemia is found in 25% of inpatients and in up to 90% of critically ill patients in the intensive care unit (34).

Hyperglycemia and insulin resistance are expected complications in AKI patients. Several studies have suggested that kidney function plays an important role in glucose homeostasis, which is regulated by insulin. As consequence, decreased kidney function can lead to insulin resistance. Uremia itself has been also associated with decreased hepatic and peripheral glucose uptake and a reduced number of glucose transporters of adipocytes (35,36). A number of studies in both human

TABLE 1
Glucose Absorption Estimates by Peritoneal Dialysis (PD) Modality

Modality	Reference	(cycles)	Infused (mL)	Glucose (%)	Absorption [g (% of infused)]	Kcal
CAPD	Koehnlein <i>et al.</i> , 2009 (30)	6	2000	1.5	98 (60)	392
APD	Perez <i>et al.</i> , 2000 (31)	9	2000	1.5	98 (40)	392
Acute PD (IPD)	Daugirdas <i>et al.</i> , 2001 (32)	24	2000	1.5	292 (45)	1167
HVPD	Góes <i>et al.</i> , 2013 (33)	24	2000	1.5	227 (35)	908

CAPD = continuous ambulatory PD; APD = automated PD; IPD = intermittent PD; HVPD = high-volume PD.

and animal models have suggested that hyperglycemia and hyperinsulinemia may exert profound effects on the acute inflammatory response and oxidative stress pathways, which are also important predictors of outcome in critically ill AKI patients (36).

Bochicchio *et al.* (37) studied the association between glycemic control and mortality in critically ill patients and noted that the hyperglycemic group had a risk for mortality that was higher by a factor of 2.2, even when adjusted for age and Acute Tubular Necrosis Individual Severity Score. Basi *et al.* (36) showed that hyperglycemia in AKI patients was an independent predictor of death even after adjustment for age, race, severity of illness score, cortisol (as a marker of hormonal stress), and nutrition status.

To avoid or reduce hyperglycemia because of overfeeding in patients treated with PD, glucose absorption from dialysate should be taken into consideration when calculating their total energy intake. Additionally, when hyperglycemia is detected, blood glucose should be monitored frequently (approximately every 6 hours), and intravenous or intraperitoneal administration of insulin (or both) should be considered (16). In studies of patients undergoing HVPD, glucose levels were maintained between 130 mg/dL and 170 mg/dL using both intravenous and intraperitoneal insulin (27).

Glucose absorption from the dialysate also seems to play a role in obesity and lipid profile abnormalities in PD patients. High levels of cholesterol, triglycerides, and low-density lipoprotein cholesterol (with reduced high-density lipoprotein cholesterol) and gain of weight and body fat are often associated with PD in patients with chronic kidney disease (38); however, such glucose absorption-related complications are not observed in AKI patients who receive treatment only until their kidneys recover function.

Protein Losses: Protein loss might play an important role in PD, especially in the presence of peritonitis. According to studies conducted in the 1970s and 1980s, protein loss into dialysate ranges from 0.5 g/L to 4 g/L during intermittent PD, reaching 20 – 200 g after 24 – 48 hours of dialysis, with no decline in plasma levels of albumin and total proteins (38–40).

In stable CAPD patients, protein loss averages 5 g daily (41). Tjiong *et al.* (42) concluded that, during APD, 24-hour protein loss into dialysate range between 4.2 g and 9.7 g (median: 6 g) and are similar to the loss occurring with nightly intermittent PD (8.5 hours) and continuous PD with a cycler (15.5 hours).

Earlier studies performed by our group in AKI patients on HVPD (25,27) showed considerable protein loss into

peritoneal dialysate (median: 21.7 g daily) that worsens in the presence of peritonitis or ascites. However, a recent study performed by Góes *et al.* (33) in 31 AKI patients treated with 208 HVPD sessions showed a lower protein loss (about 4.23 ± 1.58 g daily) that was associated with glucose uptake and removal of sodium. Greater protein loss was also observed in patients with peritonitis and ascites.

In a study of the factors associated with albumin loss in chronic kidney disease patients undergoing PD treatment, Kaysen (43) reported that, although peritoneal albumin loss and dialysate C-reactive protein were strongly and independently associated with serum albumin, no relation was observed between serum C-reactive protein and albumin loss into dialysate. However, Balafa *et al.* (41) observed that high albumin clearance was associated with fast transport status ($p < 0.0001$) and the presence of peripheral arterial disease ($p = 0.04$).

Still, evaluating factors that alter protein loss through the peritoneum, Westra *et al.* (44) suggested that increasing the number of nighttime exchanges and lengthening the duration of dwell times might increase protein loss into dialysate in APD patients ($r = 0.80$, $p < 0.01$). Unlike Balafa *et al.* (41), those authors showed that the relationship between frequency of nighttime cycling and protein loss persisted even after adjustment for dialysate-to-plasma ratio of creatinine, demonstrating no relation with peritoneal transport (44).

The peritoneal clearance of proteins was associated with mortality in a study by Perl *et al.* (45). Increased peritoneal clearance of proteins was associated with older age and higher pulse pressure, and a tendency toward higher protein loss was observed in patients with diabetes, peripheral vascular disease, and ischemic heart disease.

Protein loss through dialysis might cause or increase malnutrition and hypoalbuminemia in PD patients (46–48). The prevalence of malnutrition among chronic kidney disease patients is 20% – 50% (49). In a cohort of patients with AKI, 58% were identified as malnourished by subjective global assessment. Furthermore, malnutrition as diagnosed by subjective global assessment represented a significant risk factor for death, independent of the severity of the underlying illnesses (50).

In general, albeit significantly, protein loss through dialysis does not seem to limit the use of PD in AKI patients. Studies in HVPD patients have shown no decrease in serum albumin despite such protein loss (24,25,27).

To avoid malnutrition in AKI patients on PD, greater daily oral intake of protein (1.5 g per kilogram of body weight) has been recommended (16).

Alterations in Protein Catabolism: The most important alteration in the nutrition status of AKI patients is hypercatabolism, which is multifactorial (51,52). This condition increases protein degradation and reduces protein synthesis because of inadequate transport of amino acids into cells. This inadequacy in amino acid transport has been associated with insulin resistance and uremia. Other factors that contribute to protein catabolism include endocrine abnormalities, metabolic acidosis, systemic inflammatory response syndrome, protease release, and loss and inadequate supply of nutrition substrates (53,54). Thus, AKI alone cannot be held responsible for such alterations: underlying diseases and renal replacement therapy itself are also involved (2).

Because daily small-solute clearance is lower in PD than in HD, the question of whether PD can affect metabolic control in critically ill patients arises (16). Some studies have reported inadequate metabolic control in critically ill patients on PD. However, those studies used rigid peritoneal catheters, manual exchanges, and short dwell times, which are known to be associated with poor solute clearance and worse dialysis outcomes (55,56). On the other hand, TPD, CFPD, and HVPD were demonstrated to supply enough Kt/V for adequate metabolic control (serum urea < 120 mg/dL) and moderate catabolism in AKI patients (16).

Despite providing better clearance, PD is still a limited method in terms of dialysis dose. Because there is no satisfactory marker of dialysis adequacy in AKI, the Kt/V used for chronic patients was extrapolated to AKI patients, although it has limitations in situations of hypercatabolism and fluid overload. During HVPD, the maximum Kt/V attained (about 0.5 per session) could hinder control of urea levels in severely hypercatabolic patients (57). However, studies of HVPD have shown that blood urea nitrogen and plasma creatinine were significantly reduced at treatment onset, becoming stable (blood urea nitrogen: ~50 mg/dL; creatinine: ~4 mg/dL) after 4 sessions (9,25,57).

Nitrogen balance (NB) is the “gold standard” for assessing the degree of protein anabolism and catabolism (7). Nonetheless, only a few investigators have used this measure in AKI patients.

Nitrogen balance is the difference between nitrogen intake and nitrogen output (losses of urea and non-urea nitrogen). The non-urea nitrogen losses (not correlated with dietary intake) are estimated using the Maroni formula (58), which takes body weight into account. Urea nitrogen loss (also called urea nitrogen appearance) is calculated as the sum of urinary urea nitrogen plus the variation in body urea and dialysate urea nitrogen, as follows (3):

$$\text{Non-urea nitrogen loss (g)} = 0.031 \text{ g nitrogen} \times \text{body weight (kg)}$$

$$\begin{aligned} \text{Urea nitrogen appearance} = & [\text{urinary urea} \\ & \text{nitrogen concentration (g/day)} \times \\ & \text{urinary volume (L)}] + \\ & \{[\text{variation of blood urea nitrogen (g/L) in} \\ & 24 \text{ hours} \times 0.6 \times \text{body weight (kg)}] + \\ & [\text{variation of body weight in 24 hours} \times \\ & \text{final weight (kg)} / 100]\} + \\ & [\text{dialysate urea nitrogen (g/L)} \times \\ & \text{dialysate volume (L)}]. \end{aligned}$$

Some studies stated that a positive NB is a parameter of nutrition associated with improved patient outcomes and that, when a positive balance cannot be achieved, minimization of nitrogen deficit is the next best option (59).

In stable patients, the aim is to achieve a positive NB between 4 g and 6 g daily. Unfortunately, critically ill patients are rarely capable of maintaining a positive balance, especially when the situation of stress is not resolved (59).

Some studies have linked NB with prognosis in hospitalized patients. Scheinkestel *et al.* (59), in a prospective study of critically ill anuric patients requiring continuous renal replacement therapy, showed that NB was directly associated with hospital outcome and intensive care unit outcome. For every 1 g increase in the daily NB, the probability of survival increased by 21%.

Ponce *et al.* (57), in a study of 204 AKI patients treated with HVPD, found that NB increased after 3 sessions and stabilized at about 1 g daily after 5 sessions. The 1-g increase in NB observed after 3 HVPD sessions was associated with risk of death (odds ratio: 0.88; 95% confidence interval: 0.81 to 0.98; $p = 0.01$). Notably, these same authors had already demonstrated in prior studies the possibility of maintaining a positive NB in patients receiving HVPD (9,24,25).

Sodium Disorders: Few studies have evaluated sodium balance in AKI patients treated with PD. Physiologically, the dialysate sodium concentration declines during the initial phase of a dwell with hypertonic solutions, and then gradually rises. The minimum value is usually reached after 1 – 2 hours. It is likely that this so-called sieving of sodium is caused by transcellular water transport through the ultrasmall pores (aquaporin 1). However, other mechanisms—such as temporal binding of sodium in the interstitial tissue—cannot be excluded with certainty. Water transport rates are high during the initial phase of a hypertonic exchange. The decrease in dialysate sodium is therefore a dilutional phenomenon, which implies that, during short dwells with hypertonic dialysate, much more water than sodium is removed from the extracellular volume, an effect that can lead to hyponatremia (60–62).

This hindrance in the removal of sodium compared with the removal of water is not clinically important during CAPD because the increment in the concentration gradient is counteracted by increased diffusion of sodium. However, during short dwells, as are often applied in APD, much more water than sodium can be removed from the extracellular volume (60,61). In this context, it is likely that the dialysate sodium concentration does not have time to equilibrate with the plasma concentration given the short dwell times used in HVPD. As result, the amount of water removed from the body would proportionally exceed the amount of sodium removed, leading to hypernatremia.

Several studies have evaluated the influencers of sodium removal. A study by Rodríguez–Carmona and Fontán (63) showed that APD is often associated with low rates of sodium removal. For any degree of ultrafiltration, removal of sodium was better in CAPD than in APD. The same study also showed that a nocturnal exchange with icodextrin and complementary daytime exchanges enhanced the removal of sodium in APD.

Choices for optimizing the gradient of sodium removal include lowering the sodium concentration or increasing the dextrose concentration in the dialysate, thus increasing convective loss of sodium (60).

In the context already described, AKI patients treated with short-dwell HVPD would be expected potentially to develop hypernatremia (≥ 145 mEq/L). However, our study of patients with AKI receiving HVPD showed variable removal of sodium that was higher in patients with higher serum sodium, suggesting that HVPD does not cause hypernatremia and can correct that condition when necessary. Sodium removal also correlated positively with ultrafiltration, AKI index, and urea nitrogen appearance, and correlated negatively with age. Thus, younger patients, those who were more catabolic, those achieving more ultrafiltration, and those who had a worse prognosis experienced higher sodium removal (33).

Observational data in large PD cohorts show that low peritoneal removal of sodium and water is associated with lesser survival, regardless of residual renal function and inflammation (64–66). Similarly, an increase in sodium and fluid removal has been associated with lesser mortality in several studies (64,67,68).

However, a recent study hypothesized that the relationship between low sodium removal and mortality can be confused with parameters of dietary intake and nutrition in PD patients. Thus, patients with poorer dietary intake experience lower sodium removal and worse prognosis because of malnutrition (69).

TABLE 2
Main Metabolic Implications of Peritoneal Dialysis (PD)

Implication	Complication	Cause	Preventive action
Glucose absorption	Hyperglycemia	Glucose as PD osmotic agent	Use intravenous or intraperitoneal insulin
	Inadequate energy intake	Absent or inadequate estimate of glucose absorption	Proper quantification of glucose in dialysate
Protein loss	Malnutrition	Peritoneal semipermeable membrane	Minimum protein intake of 1.5 g/kg daily
	Hypoalbuminemia	Peritonitis	
Na removal	Hypernatremia	Difficulty of removing Na by HVPD because of rapid changes	Monitor blood Na Lower dialysate sodium Increase ultrafiltration
K removal	Hypokalemia	Considerable loss of K when using a large volume of dialysate with no K	Add K to PD bags
Catabolism	No control of hypercatabolism	Limited peritoneal Kt/V	Protein intake adequate to catabolism, aiming at neutral or positive nitrogen balance

HVPD = high-volume PD.

Potassium Disorders: Standard PD solutions do not contain potassium, which is lost during PD by diffusion and convection. In general, after a 4- to 6-hour dwell time, serum and dialysate potassium concentrations are similar (29). As a result, 10% – 58% of PD patients either develop hypokalemia (<3.5 mEq/L) or require potassium supplementation to maintain normal serum levels (70).

Hypokalemia has been identified as a risk factor for peritonitis and death in chronic PD patients (71). Chuang and colleagues (72) found a greater incidence of peritonitis in hypokalemic than in normokalemic patients (6.9% vs 2.1%, $p < 0.001$). Hypokalemia is one of the causes of reduced gastrointestinal motility, resulting in bacterial overgrowth and transmural migration of enteric organisms. It is also a sign of malnutrition, which alters immune defenses and bowel loops, leading to peritonitis.

Szeto *et al.* (71) concluded that, compared with patients having no hypokalemia, those with hypokalemia had a significantly worse actuarial survival after adjustment for confounding factors. The authors noted that serum potassium in these patients was associated with nutrition status and severity of coexisting comorbid conditions.

Potassium loss can be quite high in acute PD, because each 2-L exchange has the potential to remove 8 mEq per hour. Such removal might cause serious potassium depletion and cardiovascular instability. Those consequences might be prevented by adding potassium to the dialysis solution (3 – 4 mEq/L).

Table 2 summarizes the metabolic implications of PD for AKI patients, including causes and preventive strategies.

SUMMARY

Peritoneal dialysis can be used as a modality of renal replacement therapy to treat AKI. Techniques such as HVPD, CFPD, and TPD are effective for the treatment of a select group of AKI patients, but few studies have addressed the metabolic implications of these treatments. Assessment of such implications is relatively simple, does not require additional costs, and provides diagnostic and nutrition information on protein loss, glucose absorption, sodium removal, and degree of protein catabolism. In addition, the adequacy of dialysis and nutrition can easily be determined so as to prevent metabolic complications such as hyperglycemia and hypercatabolism. Further studies are needed to assess the metabolic implications of PD in patients with AKI.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

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