

Hyponatremia and Hypokalemia in Peritoneal Dialysis Patients

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1. Introduction

Imbalance of sodium and potassium in patients on peritoneal dialysis (PD) is not uncommon. Sodium is the major extracellular cation; however, its concentrations are mainly affected by water balance, as well as sodium balance. Potassium is the major intracellular cation, and serum potassium concentrations are very low, compared with its intracellular components. Hyponatremia and hypokalemia are associated with severe consequences, respectively. I will review development of hyponatremia and hypokalemia in incident PD patients, and describe recently emerging evidence related to hyponatremia and hypokalemia in PD patients.

2. Hyponatremia in PD patients

2.1 Hyponatremia in hospitalized patients

Hyponatremia is one of the most common electrolyte disorders in hospitalized patients (Anderson et al., 1985; Zevallos et al., 2001). Daily incidence and prevalence of hyponatremia are approximately 1% and 2.5%, respectively. Two thirds of all hyponatremia was hospital-acquired. Normovolemic state (so-called syndrome of inappropriate secretion of antidiuretic hormone) is the most commonly seen clinical setting of hyponatremia. Hyponatremia, defined as a serum sodium level less than 135 mEq/L, was increased by 20% in inpatients with congestive heart failure (Gheorghide et al., 2007). The fatality rate for hyponatremic patients was 60-fold that for patients without documented hyponatremia (Anderson et al., 1985). Hyponatremia is an independent predictor of death and myocardial infarction in middle-aged and elderly community subjects, respectively (Sajadieh et al., 2009). Hyponatremic patients with congestive heart failure are associated with a 75% increase in 60- to 90-day mortality, compared with normonatremic patients (Gheorghide et al., 2007). Therefore, even mild hyponatremia has a poor prognosis.

2.2 Pathophysiology of hyponatremia in PD patients

The prevalence of hyponatremia is approximately 10% in PD patients (Zevallos et al., 2001). Because fluid and electrolyte balance in these patients is dependent on nonrenal routes, PD-related hyponatremia has been suggested to differ pathophysiologically from non-PD-related hyponatremia. Regulation of a normal serum sodium concentration is dependent on the content of sodium and potassium, as well as total body water. Despite a low rate of

glomerular filtration, the normal kidney can regulate serum sodium concentrations independent of solute and water balance. Whereas, in PD patients without residual renal function, solute and water balance through peritoneal membrane plays an important role in development of hyponatremia (Nolph et al., 1980). Sodium removal by convective transport predominates over diffusive transport in patients on PD, thereby requiring augmentation of ultrafiltration volume in order to increase sodium removal (Nolph et al., 1980). Due to the Gibbs-Donnan effect created by negatively charged proteins in the plasma space, and sodium sieving by aquaporins, the typical dialysate sodium concentration is substantially lower than the plasma sodium concentration (Rippe & Venturoli, 2008). As a result of this sieving, the early part of a PD dwell removes mostly water, and sodium removal occurs later in the dwell as the dialysate Na^+ concentration decreases, creating a higher gradient for diffusive transport of sodium. As a result of its longer dwell times, continuous ambulatory PD removes more sodium per ultrafiltration volume than automated PD (Rippe & Venturoli, 2008).

The serum sodium concentration accounts for the balance of three components: mainly sodium, potassium, and body water. The basis for hyponatremia is a negative balance for sodium plus potassium and/or a positive balance for water. Vasopressin can control water permeability in the normally functioning nephron, which leads to electrolyte-free water retention. In dialysis patients without residual renal function, the role of vasopressin is limited; therefore, solute balance may play a more important role in development of hyponatremia (Zanger, 2010). Hypotonic hyponatremia may occur in PD patients, either from free water retention or loss of electrolyte, such as sodium or potassium. If hyponatremia is accompanied by a quantitatively appropriate gain in weight, a gain of electrolyte-free water is the basis for hyponatremia (Cherney et al., 2001). In the absence of this weight gain, loss of sodium from the extracellular fluid (ECF) space will lead to movement of water into the intracellular fluid (ICF), to restore the equilibrium between intra- and extracellular osmolality. Loss of potassium, along with an ECF anion, such as chloride or bicarbonate, results in potassium efflux from ICF into ECF, with a shift of sodium into the cell for maintenance of electroneutrality (Nguyen & Kurtz, 2004). The net effect is loss of sodium from the ECF, with development of hyponatremia (Cherney et al., 2001). In patients without weight gain, loss of intracellular potassium, with an intracellular anion, such as phosphate, will contribute to development of hyponatremia, due to movement of water from the ICF into the ECF.

2.3 De novo hyponatremia in incident PD patients

In order to control bias on pre-existing hyponatremic conditions, we observed development of hyponatremia in incident PD patients, and evaluated factors contributing to its development. We conducted a 1-year observational study at a single PD center at Gachon University Gil Hospital, South Korea (Lee et al., 2010). Fifty-one incident PD patients were enrolled. All patients were ethnic Koreans and older than 18 years. The same protocol used in our previous report was used in performance of the peritoneal equilibration test (PET) and measurement of dialysis adequacy at 1 and 13 months after the start of PD (Kim et al., 2009). Patients with hyponatremia ($\text{Na}^+ < 135 \text{ mEq/L}$) at month 1 were excluded. At month 13, patients were divided according to their serum sodium levels into hyponatremic ($\text{Na}^+ < 135 \text{ mEq/L}$) and normonatremic ($\text{Na}^+ \geq 135 \text{ mEq/L}$) groups.

Between the two groups, there were no significant differences in baseline demographics of patients beginning PD, including age, sex, height, cause of end-stage renal disease, comorbidity, biocompatible fluid, and CAPD versus automated PD. Sixteen percent of enrolled patients had de novo hyponatremia 13 months after initiation of PD. Initial serum albumin levels of the hyponatremia group were significantly lower than those of the normonatremic group ($p = 0.022$; Table 1). Median levels of initial serum sodium in the hyponatremia group were also slightly lower than those in the normonatremic group; however, the difference was not significant. At month 13, the hyponatremia group showed no significant difference in serum albumin levels, compared to the normonatremic group.

Parameters	Month 1		Month 13	
	HN	NN	HN	NN
Serologic parameters				
C-reactive protein (mg/dL)	0.15	0.13	0.26	0.13
Glucose (mg/dL)	173	130	160	120*
Creatinine (mg/dL)	7.6	7.4	10	7.9
Albumin (mg/dL)	3.1*	3.7	3.4	3.6
Cholesterol (mg/dL)	179	171	200	189
Sodium (mEq/L)	136	140	134*	139
Potassium (mEq/L)	3.7	4.2	3.8	4.3
Chloride (mEq/L)	99	101	95*	101
Total CO ₂ (mEq/L)	23.4	23.0	22.3	25.9
Peritoneal function and adequacy tests				
D/P Cr	0.64	0.67	0.75*	0.66
Maximum dip in DPNa	0.071	0.070	0.091	0.071
Ultrafiltration capacity (mL)	800	875	700	900
Weight (kg)	66.5	65.8	66.5	65.5
nPNA (g/kg/day)	0.81	0.88	0.79	0.88
Total Kt/Vurea	2.05	2.28	2.19	2.12
GFR (L/week/1.73m ²)	44.8	45.1	53.1*	43.8

Table 1. Serum levels of the parameters at months 1 and 13 (Lee et al., 2010). Values are presented as the median. HN, hyponatremia; NN, normonatremia. D/P Cr, dialysate-to-plasma creatinine ratio at 4 hours; Maximum dip in DPNa, the difference between the initial D/P sodium and the lowest D/P sodium; nPNA, protein equivalent of total nitrogen appearance normalized to desirable body weight (generating rate of urea); GFR, Glomerular Filtration Rate (average of creatinine clearance and urea clearance). * P -value < 0.05, hyponatremia group vs. normonatremic group.

No significant differences were observed in serum C-reactive protein, creatinine, cholesterol, potassium, or total CO₂ between the two groups after 13 months. Several parameters changed from month 1 to 13 in PD patients with de novo hyponatremia. Serum albumin levels in the hyponatremia group showed a moderate, but not significant increase ($p = 0.058$), from 3.10 to 3.45 mg/dL. During the 12-month period, no statistical difference was observed in serum glucose, creatinine, cholesterol, potassium, chloride, and total CO₂ levels. At month 1, the two groups showed no significant differences in peritoneal function and adequacy tests. In contrast, at month 13, the D/P Cr was increased to 0.748 in the hyponatremia group, compared with 0.662 in the normonatremic group ($p = 0.007$). The glomerular filtration rate measured at month 13 was slightly higher in the hyponatremia group ($p = 0.021$). No other measure of peritoneal function and adequacy differed between the two groups. Changes in peritoneal function and adequacy tests in PD patients with de novo hyponatremia were observed after 12-month treatments. The D/P Cr showed a significant increase ($p = 0.036$) from 0.644 to 0.748. The glomerular filtration rate tended to be higher at 12 months (44.8 vs. 53.1 L/week/1.73m², $p = 0.063$). A trend toward a decrease in nPNA was seen in the hyponatremia group at month 13; however, the difference was not significant.

We found that the increase in D/P Cr at month 13 was significantly higher in the hyponatremia group. The lower fluid removal during a single PD exchange in high transporters was not only due to a decrease in the ultrafiltration rate, but also an increase in the peritoneal fluid absorption rate (Wang et al., 1998). The decrease in the ultrafiltration rate in high transporters is likely due to a rapid decrease in the osmotic gradient resulting from increased absorption of glucose from the dialysate. The increased fluid absorption rate may be the result of increased peritoneal interstitial hydraulic conductivity, as the peritoneal hydrostatic pressure gradient is unlikely to be higher in high transporters. Therefore, subjects in the hyponatremia group, who were high transporters, showed lower fluid removal, which might lead to free water gain.

Of particular interest, in this study, despite reduced ultrafiltration capacity, there were no changes of body weight in the hyponatremic groups. This finding suggests that hyponatremia in incident PD patients can occur due not only to free water gain, but also additional causes, including catabolic states or malnutrition. Patients with de novo hyponatremia had low levels of initial serum albumin and low levels of nPNA at month 13. Serum albumin and nPNA are markers for nutrition and a well-known predictor of mortality in PD patients; therefore, these conditions may also contribute to de novo hyponatremia (Perez-Flores et al., 2007). Diuretics, which were prescribed to maintain urine output, may also affect development of hyponatremia (Sonnenblick et al., 1993). However, there was no significant difference between the two groups, although approximately half of patients were prescribed diuretics in the current data.

The majority of the patients still had residual renal function since all the enrolled patients were incident cases of peritoneal dialysis. The residual renal function tended to be higher at 12 months in hyponatremic group. Excessive peritoneal ultrafiltration may, by provoking intravascular volume depletion, play a causative role in the decline in residual renal function (Konings et al., 2005; Konings et al., 2003). In the present study, it is possible that increased D/P Cr and, consequently, lower peritoneal ultrafiltration volume in the hyponatremic group might provide a lower risk of intravascular volume depletion and higher probability of relative increase of residual renal function.

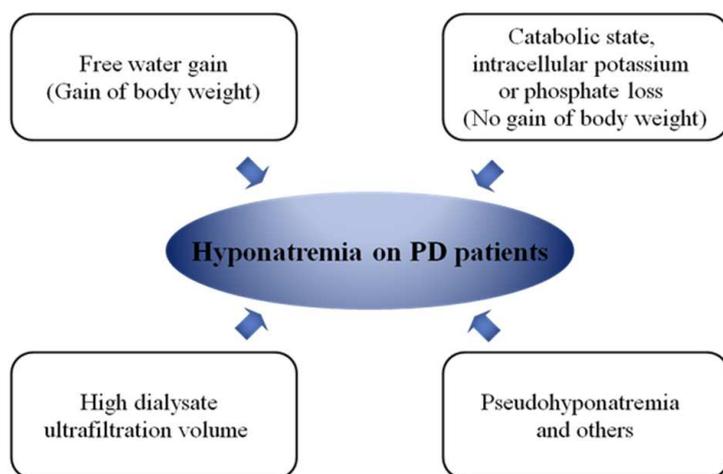


Fig. 1. Pathophysiology of hyponatremia in PD patients

Our findings showed that the maximum dip in D/PNa did not differ between the two groups, while D/P Cr was significantly higher in the hyponatremia group. The sieving coefficient for sodium is higher in high transport patients compared, with low transport patients (Wang et al., 1997). However, no significant differences were found in our study. Discrepancies between studies may be explained by small differences in D/P Cr. The average D/P Cr in the hyponatremia and normonatremic groups classified both as high-average transporters. We observed higher serum glucose levels at month 13 in the hyponatremia group, while no change in glucose or uric acid was observed in the hyponatremia group from month 1 to 13. Hyperglycemia is associated with a decrease in the serum sodium concentration (Roscoe et al., 1975). The expected decrease in the serum sodium concentration is 1.35 mEq/L for every 100 mg/dL increase in the blood glucose concentration (Roscoe et al., 1975). Therefore, higher serum glucose levels in the hyponatremia group were related to the decrease in serum sodium levels.

2.4 Summary of hyponatremia in PD patients

The primary cause of hyponatremia is free water gain by the ECF, which is often paralleled by an increase in body weight. Intracellular potassium or phosphate loss, pseudohyponatremia, and catabolic states are less common causes of hyponatremia (Cherney et al., 2001)(Fig. 1). Some studies have demonstrated a relationship between the incidence of PD-associated hyponatremia and the catabolic state (Zevallos et al., 2001). They reported that tissue catabolism combined with intracellular potassium and phosphate loss may lead to hyponatremia in PD patients. Other studies have shown that the main determinant of PD sodium loss is the net dialysate ultrafiltration volume (Uribarri et al., 2004). Treatment with icodextrin-based dialysis solution regimes has also been implicated as a risk factor for hyponatremia (Gradden et al., 2001). Increased serum NT-Pro-BNP, a predictor of mortality in PD and hemodialysis patients, was found to play a role in development of hyponatremia, water balance disturbance, anemia status, and hypoalbuminemia in the PD patient group (Adachi & Nishio, 2008).

3. Hypokalemia in PD patients

3.1 Pathophysiology of hypokalemia in PD patients

A third of PD patients, in whom potassium removal by PD does not explain the occurrence of hypokalemia, are frequently hypokalemic (Oreopoulos et al., 1982; Rostand, 1983). Some studies have noted that 36% of PD patients have a serum potassium level less than 3.5 mEq/L at some time during their course and that 20% require potassium supplementation (Spital & Sterns, 1985). In addition, hypokalemia is an independent predictor of mortality in PD patients (Szeto et al., 2005).

Potassium homeostasis is maintained by two different balances: the internal balance, representing potassium redistribution between intracellular and extracellular compartments, and the external balance, representing potassium interchange between the organism and the environment (Adrogué & Wesson, 1992). Since fluid and electrolyte balance in these patients is dependent on nonrenal routes, PD-related hypokalemia also differs pathophysiologically from non-PD-related hypokalemia. Daily consumption of potassium exceeds daily elimination with PD; therefore, PD fluid has never contained potassium. Enhanced large intestine secretion of potassium in direct proportion to dietary potassium intake, which is an additional route of potassium removal, plays an important role in maintenance of potassium balance in patients with renal insufficiency (Bastl et al., 1977; Mathialahan et al., 2005).

Figure 2 shows the pathophysiology of hypokalemia in PD patients. Serum potassium levels in PD patients are associated with nutritional status and severity of coexisting comorbid conditions. Hypokalemia in PD patients is due to a shift of potassium into the intracellular space, probably due to insulin release during the continuous dwell of the dialysis solution containing glucose (Tziviskou et al., 2003). Thus, cellular uptake may play an important role in the pathogenesis of hypokalemia. Muscle biopsy studies have shown higher intracellular potassium content in PD patients, compared with hemodialysis patients (Lindholm et al., 1986). Ongoing loss of potassium in dialysate may be another important factor contributing to hypokalemia (Szeto et al., 2005). The main driving force for elimination of potassium is the diffusive gradient between blood and PD fluid (Brown et al., 1973; Nolph et al., 1980).

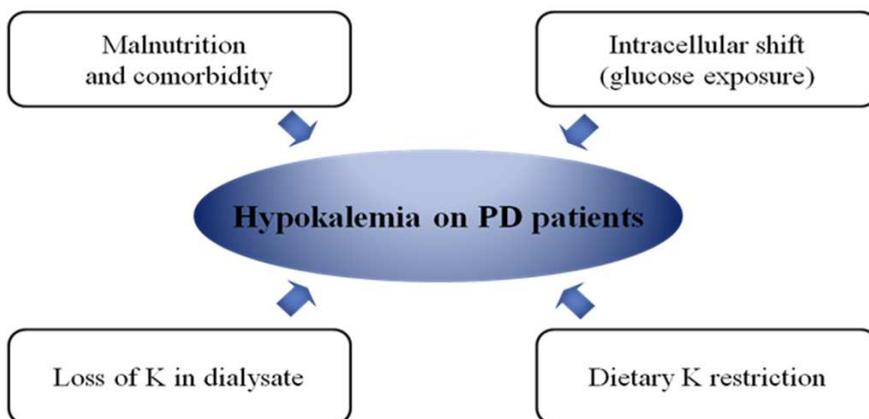


Fig. 2. Pathophysiology of hypokalemia in PD patients

Due to the low concentration of serum potassium, removal of convective fluid, under most circumstances, contributes little to potassium removal (Brown et al., 1973; Nolph et al., 1980). In addition, end stage renal disease patients are instructed to restrict potassium-rich foods, such as fruits and vegetables.

3.2 De novo hypokalemia in incident PD patients

This study (Jung et al., 2009) was undertaken in order to investigate clinical features and factors related to de novo hypokalemia in incident PD patients over the 1-year observational period using the same protocol (Lee et al., 2010), which is designed for control of bias on pre-existing hypokalemic conditions.

Eighty-two incident PD patients who were normokalemic at month 1 of PD were enrolled in the study. According to the plasma potassium levels at month 13, patients were divided into hypokalemic ($K^+ < 3.5$ mEq/L) and normokalemic ($3.5 \text{ mEq/L} \leq K^+ < 5.5$ mEq/L) groups. Eight patients who showed hyperkalemia at month 13 were excluded. Blood, peritoneal function tests, and dialysis adequacy and nutritional status data were taken at months 1 and 13. The two groups, those with and those without hypokalemia, did not differ significantly in age, sex, height, and causes of end-stage renal disease. Medication history of ACE inhibitors or angiotensin II receptor blockades and diuretics were not significantly different between the two groups. No statistical differences were found between the two groups with respect to the Davies comorbidity index, biocompatible fluid, and continuous ambulatory PD versus automated PD.

The incidence of hypokalemia in patients starting PD was 7.3% over the 1-year observation period. Whereas the initial serum potassium and albumin levels at month 1 did not differ between the two groups, serum albumin level in the hypokalemia group showed a significant decrease from 3.1 to 2.9 mg/dL at month 13 ($p = 0.014$) (Table 2). In contrast, no significant differences were observed in serum C-reactive protein, glucose, cholesterol, and total CO_2 between the two groups at month 13. At month 1, the hypokalemia group showed higher serum phosphorus levels and lower cholesterol levels.

At month 1, the two groups showed no significant differences. nPNA at month 1 was also similar between the hypokalemic and normokalemic groups. However, at month 13, the nPNA was significantly lower in the hypokalemia group, compared with that in the normokalemic group. Other measurements determined for peritoneal function, including D/P Cr and daily glucose exposure, did not differ between the two groups.

In the current study, the incidence of hypokalemia in our PD patients, 7.3% in one year, appears relatively low, compared with that of previous reports (Kim et al., 2007; Oreopoulos et al., 1982; Rostand, 1983). This is probably because our result was from newly starting PD patients. We demonstrated an association between development of hypokalemia in incident PD patients and decreases in serum albumin and nPNA. This poor nutritional status can lead to de novo hypokalemia. In PD patients, insulin hormone, stimulated by the continuous glucose peritoneal dwell, can generate an increase of potassium redistribution into the intracellular compartment (Tziviskou et al., 2003). In the present study, the presence of diabetes and daily glucose load did not differ between the two groups, suggesting that intracellular potassium redistribution may not be the main mechanism of hypokalemia in our PD patients. In a cross-sectional study, the serum potassium level of PD patients was correlated with ultrafiltration volume (Kim et al., 2007). However, in our longitudinal study, no difference in ultrafiltration volume was observed between the two groups at month 13.

This finding is consistent with the previous study showing that convective fluid removal contributes little to potassium elimination (Brown et al., 1973; Nolph et al., 1980).

	Month 1		Month 13	
	HK	NK	HK	NK
Serologic parameters				
C-reactive protein (mg/dL)	0.53	0.15	0.36	0.18
Glucose (mg/dL)	158	138	137	127
Creatinine (mg/dL)	7.6	7.6	8.6	9.4
Albumin (mg/dL)	3.1	3.7	2.9*	3.7
Cholesterol (mg/dL)	125*	163	163	174
Sodium (mEq/L)	136	139	136	139
Potassium (mEq/L)	4.1	4.2	3.4*	4.5
Chloride (mEq/L)	101.5	102.0	98.0	101.0
Total CO ₂ (mEq/L)	19.5	23.0	23.5	22.1
Calcium (mg/dL)	8.5	8.5	8.6	8.9
Phosphorus (mg/dL)	5.6 *	4.2	3.7	4.6
Peritoneal function and adequacy tests				
D/P Cr	0.68	0.67	0.69	0.67
Ultrafiltration capacity (mL)	675	850	800	800
Weight (kg)	63.8	66.0	66.0	63.6
nPNA (g/kg/day)	0.89	0.91	0.66*	0.91
Total Kt/Vurea	2.37	2.05	1.94	1.98
GFR (L/week/1.73m ²)	47.0	41.1	48.5	42.6
Glucose exposure (g/day)	130	120	163	120

Table 2. Serum parameters at months 1 and 13 (Jung et al., 2009). Values are presented as median values. HK, hypokalemia; NK, normokalemia. D/P Cr, dialysate-to-plasma creatinine ratio at 4 hours; nPNA, protein equivalent of total nitrogen appearance normalized to desirable body weight (generating rate of urea); GFR, Glomerular Filtration Rate (calculated as the mean of the values for creatinine and urea clearances) * *P*-value < 0.05, hypokalemia group vs. normokalemic group.

3.3 Summary of hypokalemia in PD patients

Ten to 58% of patients on PD are known to develop hypokalemia ($K^+ < 3.5$ mEq/L)(Khan et al., 1996; Rostand, 1983; Spital & Sterns, 1985). The wide range of prevalence of hypokalemia

may depend on dietary consumption of potassium according to the study population or ethnicity. Compared with Caucasians, Asians and African Americans tend to have lower daily potassium intake (Gao et al., 2009; Kant et al., 2007; Szeto et al., 2005). In a group of 266 Chinese PD patients, prevalence was reported as 20.3%, and hypokalemia was found to be an independent predictor of mortality, although causes of death in the hypokalemic group did not differ from those in the normokalemic group (Szeto et al., 2005). In other Asian PD patients, hypokalemia can be considered a nutrition marker, such as serum albumin or normalized protein nitrogen appearance (nPNA) (Chuang et al., 2009; Szeto et al., 2005). Therefore, hypokalemia is associated with poor clinical outcomes, including malnutrition and death. New evidence points to a link between hypokalemia and the risk of peritonitis. Hypokalemia and malnutrition may increase the incidence of Enterobacteriaceae peritonitis, which is caused by impairment of gut mobility, small bowel overgrowth with colonic flora, and bacterial translocation across the bowel wall (Berg, 1992; Casafont et al., 1997; Chuang et al., 2009). Particularly in diabetic patients, hypokalemia may be associated with impaired bowel motility and bacterial overgrowth (Shu et al., 2009). A study of 140 Asian PD patients found a higher incidence of peritonitis in hypokalemic patients, compared with normokalemic patients, and the incidence of peritonitis due to Enterobacteriaceae was found to be higher in the hypokalemic group, compared with the normokalemic group (Chuang et al., 2009). Both hypokalemia and markers of malnutrition may be independent risk factors for development of peritonitis. Further studies are warranted for determination of whether correction of hypokalemia can result in reduced risk of peritonitis.

4. Conclusion

Hyponatremia occurs frequently in patients undergoing PD. Therefore, one must understand its pathophysiology, since the therapeutic strategy differs from that of non-PD-related hyponatremia. Incidence of PD-associated hyponatremia is mainly related to fluid overload. Tissue catabolism combined with intracellular potassium and phosphate loss may also lead to hyponatremia. Sodium loss through peritoneal membrane is associated with the dialysate ultrafiltration volume, although that may be of little effect. Hypokalemia also develops commonly in PD patients. Serum potassium levels in PD patients may be influenced by nutritional status and severity of coexisting comorbid conditions. Development of hypokalemia can be due to a shift of potassium into the intracellular space, which is related to insulin release during the continuous dwell of the dialysis solution containing glucose. Ongoing loss of potassium in dialysate may be another important factor contributing to hypokalemia; however, potassium loss may not be related to ultrafiltration volume. Recently, hypokalemia has been considered as a risk factor for peritonitis in PD patients. This common electrolyte imbalance in PD patients is associated with severe consequences, morbidity, and mortality. Therefore, hyponatremia and hypokalemia should be monitored more carefully in these patients.

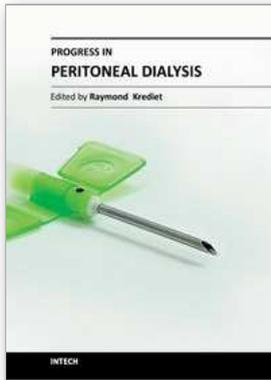
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