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Treatment of Moderate to Severe Hypokalemia in Patients Undergoing Peritoneal Dialysis by Intraperitoneal Administration of a Hyperkalemic Dialysate

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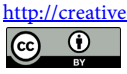

Abstract

Background: Hypokalemia is very common in peritoneal dialysis patients. The aim of this study was to investigate the effect of a peritoneal dialysate with a high concentration of potassium in the treatment of this electrolyte disorder. **Patients & Methods:** For two years, among 62 patients with end-stage renal disease under peritoneal dialysis in our unit, 10 cases of moderate (serum potassium ≥ 2.3 mmol/L to ≤2.9 mmol/L) to severe hypokalemia (serum potassium < 2.3 mmol/L) were recorded. All of them came to the hospital as part of the monthly follow-up and not at a special clinical event. None had any acid-base disorder, and all of them were taking a β-blocker, heparin (2/10), non-steroidal anti-inflammatory drugs (3/10), and eplerenone (3/10). They all had severe hypoalbuminemia. Peritoneal dialysis fluid of 2 L/bag, with 20 mmol/L potassium, was administered every 6 hours according to the protocol, *i.e.*, until blood potassium levels detected immediately before the next exchange bags were higher than 4 mmol/L. **Results:** The mean ± SD serum potassium of our patients at the beginning of the diagnosis of hypokalemia was 2.51 ± 0.39 mmol/L (5 patients with moderate and 5 with severe). It took 3 - 6 exchange bags to achieve the target blood potassium (>4 mmol/L). Generally, we gave a total of 120 - 240 mmol of potassium in the dialysate (from 3 bags of 2 L with 20 mmol/L potassium, to 6 bags of the same solution). Only 2 patients complained of pain during the filling of the abdomen (the first in one exchange and the second in all exchange bags). **Conclusion:** It is concluded that intraperitoneal administration as a dialysis fluid with a content of 20 mmol/L po-

tassium is effective, safe, and well tolerated for the restoration of blood potassium levels in patients with hypokalemia undergoing peritoneal dialysis.

Keywords

Hypokalemia, Peritoneal Dialysis, Intraperitoneal Potassium Administration, Beta-Blockers, Heparin, Insulin, Icodextrin, Eplerenone, ACE Inhibitors

1. Introduction

Hypokalemia is found in 1% of healthy people who do not take any medication. Even though during one hemodialysis session patients lose larger amounts of potassium (K^+) compared to Peritoneal Dialysis (PD) patients [1], hypokalemia in PD patients is more common (10% - 36%) [1]-[6]. Another important difference between hemodialyzed and PD patients is the fact that in the hemodialyzed K^+ is removed quickly, causing large changes in the blood levels, while in PD patients the dialytic removal of potassium is slower [6].

Hypokalemia in PD patients is attributed to many causes. First, in the dietary intake, both due to the content of food (in those patients it is often poor in K^+), and to the way of cooking [7]. Of course, it may be owing to the redistribution, where the continuous secretion of insulin contributes, due to the presence of glucose [8], but also due to alkalosis [9], which moves K^+ intracellularly. It may also be due to skin and renal losses (in those with residual renal function) [7], but also to peritoneal losses, in those who are treated with more than 4 PD exchange bags/24 hours (where there are continuous potassium losses by diffusion) [7], [10]. Some medications may also affect blood K^+ levels, some of which insert K^+ intracellularly (*i.e.*, insulin, catecholamines, thyroxine) and others that prevent the transcellular movement of K^+ (*i.e.*, β-blockers, heparin), or that do not allow its renal or intestinal elimination from the body (angiotensin-converting enzyme inhibitors-ACE, angiotensin receptor blockers-ARB, etc.). However, hypokalemia is generally found in PD patients who are treated with higher volumes of PD fluid/24 hours (*i.e.*, those who are exposed to higher concentrations of glucose, but also in those who achieve higher doses of clearance) [6] [11] [12].

In this prospective study, we recorded patients with moderate to severe hypokalemia who were treated with solutions given intraperitoneally with significantly greater concentrations of K^+ than those reported in the literature.

2. Patients-Methods

2.1. Patients

Prospectively, all patients aged > 18 years old who were on PD for more than 6 months and had moderate to severe hypokalemia (as defined above) were included in the study. Everyone came to the hospital at the standard monthly appointment for follow-up and not because they had any clinical event or symptoms.

The medications they were taking were recorded, and those that could affect serum K^+ levels—ACE inhibitors, ARBs, heparin, potassium-sparing and other diuretics, β-blockers, and Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)—were recorded. Also recorded were the presence or absence of diabetes mellitus, the type of peritoneal membrane transport characteristics, the type of PD exchange bags used daily (glucose content), the number of exchange bags used per 24 hours, and whether the patients were malnourished at the visit (based on serum albumin levels < 3.5 g/L, as well as clinically). In addition to serum K^+, their acid-base status was determined at the visit. Blood K^+ and glucose levels were determined, and any side effects from intraperitoneal administration of K^+ were recorded before each PD exchange bag.

Table 1. Patient demographics (age, sex), with or without diabetes, with malnourishment, residual renal function, peritoneal dialysis method, type of peritoneal membrane transport characteristics, kind of peritoneal dialysis fluid used daily, and ultrafiltration per 24 hours (APD = Automated Peritoneal Dialysis, CAPD = Continuous Ambulatory Peritoneal Dialysis, PD = Peritoneal Dialysis).

Parameter	Results
Age (years)	68.8 ± 9 (range 54 - 83)
Sex (F/M)	7/3
Diabetes mellitus (yes/No)	5/5
Malnourishment (clinically-laboratory) (Yes/No)	7/3
Residual renal function (urine volume > 400 ml/24 hours) (Yes/No)	4/6
Method of PD	1 APD/9 CAPD
Peritoneal membrane transport characteristics of patients	3 Low 5 Low average 1 High 1 High average
PD exchange bag of 2 L/24 hours (CAPD)	7 patients: 3 isotonic + 1 icodextrin 1 patient: 3 isotonic + 1 hemi-hypertonic 1 patient: 4 hemi-hypertonic + 1 icodextrin 1 patient: 4 isotonic
PD solution L/24 hours (APD)	11
Ultrafiltration/24 hours	<1000 ml 2 patients 1001 - 1500 ml 4 patients 1501 - 2000 ml 4 patients

We excluded from the study those who had peritonitis in the previous 3 months, diarrhea, vomiting, those who consistently had 1 - 2 PD exchange bags/24

hours, and those who changed their PD treatment in the last 3 months or had an acute heart attack. Patients with metabolic alkalosis and sepsis and those with hypokalemia following a remarkable dose of insulin or adrenaline were also excluded.

2.2. Methods

Hypokalemia was diagnosed by the results of biochemical testing (the initial determination of K^+ was done with an ion-selective analyzer), but blood potassium and glucose during the PD exchange bag were determined from the blood gas samples. It was considered that there was malnourishment (protein-energy wasting) in a patient when his/her serum albumin was <3.5 g/L or when it was clinically evident (presence of edema without the existence of hyperhydration) who, from his history, appears not to be consuming enough food for his needs.

According to the protocol, in each case of hypokalemia, PD fluid with a content of K^+ = 20 mmol/L (achieved by inserting 3 amp of KCI 10% of 10 ml in each 2 L PD bag fluid) was infused. This solution bag was administered every 6 hours until blood K^+ was detected before the next exchange bag to be more than 4 mmol/L (blood samples were taken with a gas syringe before each PD exchange bag to determine blood K^+ and glucose).

3. Results

Among 62 patients who were under PD during a period of 2 years, 10 with moderate to severe hypokalemia (K^+ from 2.1 to 3.0 mmol/L) were recorded. Patients' age, sex, type of peritoneal dialysis (automated or continuous), number of PD exchange bags/24 hours, ultrafiltration volume/24 hours, type of peritoneal membrane transport characteristics, kind of bag solution, existing residual renal function, presence or absence of diabetes, and existence of malnourishment are shown in Table 1.

All patients were taking β-blockers, heparin 2/10, NSAIDs 3/10, eplerenone 3/10, and ACE inhibitors 3/10. All had severe hypoalbuminemia (serum albumin 29 ± 7 g/L, range from 13 - 35 g/L). In terms of symptoms, only 2/10 had weakness and discharge, one had anorexia, and one had a headache. No one had any acid-base disorder (pH normal range from 7.39 - 7.49), and their blood bicarbonate levels ranged from 20.9 - 32.3 mmol/L.

In our patients with hypokalemia, 3 - 6 exchanges of 2 L PD bags with a content of K^+ 20 mmol/L had to be made to restore the K^+ of the blood to levels higher than 4 mmol/L. Only 2 patients complained of pain when the infused fluid was in the abdomen (in the second one, after well stirring of the bag, no pain reappeared). No case of hyperkalemia was noted after the infusion of the exchange bag. The number of PD exchange bags needed for each patient to restore the blood levels of K^+, the blood K^+ levels, and the glucose concentration after each change are shown in Table 2.

Table 2. Changes in blood K^+ levels and glucose levels after each exchange bag of PD solution.

Exchange bags	1st	2nd	3rd	4th	5th	6th
n patients	n = 10	n = 10	n = 10	n = 9	n = 5	n = 4
Blood K^+ (mmol/L)	2.51 ± 0.39	2.86 ± 0.23	3.10 ± 0.45	3.54 ± 0.56	3.64 ± 0.45	4.35 ± 0.61
Blood glucose (mmol/L)	6.77 ± 1.67	6.55 ± 1.55	6.94 ± 2.05	6.94 ± 2.05	6.33 ± 1.83	8.55 ± 1.39

4. Discussion

Serum K^+ levels are relatively stable in PD patients, compared with hemodialyzed patients [1]. However, many studies have shown that hypokalemia is more common in patients undergoing PD (PD solutions do not contain K^+) than in those on hemodialysis [3] [8] [13] [14]. Pai *et al.* recorded symptomatic hypokalemia in 5% of their PD patients who had to visit the emergency department (ED) [15], while Szeto *et al.* found serum $K^+ < 3.5$ mmol/L in 20% of their patients [16]. In general, however, 10% - 30% of patients on PD need K^+ supplementation [2] [16]-[18].

Dietary potassium contributes to its balance in the body. Of course, malnutrition is also associated with hypokalemia [16], as we have seen from the elevated serum C-Reactive Protein (CRP) levels in hypokalemic patients and in those with decreased serum albumin levels [5] [13] [16] [19] [20]. Others have found a link between hypokalemia and malnutrition [5] [16], including us, as our patients (7/10) were clinically malnourished and all had serum albumin < 3.5 g/L (range from 1.9 to 3.5 g/L). It is noted that malnutrition, which is relatively common in PD patients, when maintained for months, is also associated with an increased incidence of peritonitis, because it is accompanied by overhydration, which leads to swelling of the intestinal mucosa, which is associated with easier endotoxin passage through the intestinal wall [13] [21]. This may be true because hypokalemia is associated with decreased intestinal motility [16] [20]. In the diet of malnourished patients, it should be borne in mind that each gram of protein contains 1 mmol of K^+ [5] [17] and that malnutrition indicates reduced food intake [5].

Additional factors that may potentially have an influence on serum potassium levels in PD patients include the influence of glucose-containing PD solutions on transcellular movement of potassium. Hypokalemia may therefore be due to redistribution, because of the continuous insulin secretion due to the presence of glucose [8], which is confirmed by muscle biopsies that showed a higher concentration of K^+ in PD patients compared to those on hemodialysis [22], something that others disagree with [4]. Of course, no correlation was found between the incidence of hypokalemia and the presence of diabetes mellitus [5], something that we also found, since 7/10 of our patients did not have diabetes. However, what should be emphasized is that even though many of our patients were taking medications that do not allow intracellular movement of K^+ because they do not allow Na^+-K^+-ATPase to function properly (10/10 patients received β-blockers and heparin 2/10), they had low blood K^+ levels, which means that the hypokalemia in

some of them was more significant.

Also, catecholamines and thyroxine, which stimulate Na^+-K^+-ATPase, may be responsible for hypokalemia; however, there were no such cases among our patients. Redistribution can, of course, be caused by blood acidification with a negative effect on Na^+-K^+-ATPase (for each decrease in pH by 0.1, the serum K^+ increases by 0.8 mmol/L) [9].

Potassium is removed by PD with diffusion [13] [23]; therefore, removal is affected by blood K^+ concentration [18]. PD removes 1.5 K^+ mmol/hour [24], and 4 PD exchanges bags of 2 L remove 20 - 30 mmol K^+ daily [13] [25], an amount that is quite less than that taken in by food from each patient/24 hours (with the Western diet, people take in on average 60 - 90 mmol K^+/24 hours) [12].

Another cause of hypokalemia in PD patients is an increase in the clearance provided [7] [10] and an increase in ultrafiltration with solutions with high glucose concentration [6] [11] [12]. Most of our patients (8/10) were treated with 4 PD exchange bags of 2 L/24 hours, one with 5 exchange bags of 2 L, and one was in APD with 11 L dialysate/24 hours, so there is no question of increased clearance. The glucose concentration of the PD bags affects insulin secretion and the redistribution of K^+ between intracellular and extracellular spaces [14], so it also affects blood K^+ levels (perhaps in 2 of our patients this parameter played a role, since one was on 4 and the other on 1 semi-hypertonic PD exchange bag).

Icodextrin dialysate can affect serum K^+ because it increases PD ultrafiltration (which means an increase in serum K^+ concentration) [13], does not cause hyperglycemia like glucose solution, and improves intestinal motility, which contributes to better dietary food intake and K^+ [26] [27] and does not affect appetite negatively, as conventional glucose dialysate solutions do. Yi *et al.* studied serum K^+ levels for 2 years in 255 patients on PD (116 on overnight glucose solution and 139 on icodextrin dialysate). The incidence of hypokalemia noted in the two groups was similar, with mean serum K^+ levels being higher with icodextrin dialysate compared with glucose solution ($p < 0.05$). This study showed that icodextrin in chronic use during the night exchange is associated with increased serum K^+ levels and in these individuals, hypokalemia is less common compared to those who use only glucose solutions. This effect of icodextrin dialysate is attributed to better nutritional status than to redistribution of K^+ into the intracellular space [14]. We do not agree with these results, since 8/10 of our patients received icodextrin and nevertheless had a significant degree of hypokalemia.

Hypokalemia in our patients may also have been due to urinary loss of K^+ (in patients with residual renal function) [7]. This may have been present in some of our patients, as 4/10 had residual renal function.

Some studies have found that ACE inhibitors affect serum K^+ levels in PD patients [2] [28] [29], as well as aldosterone receptor inhibitors (3/10 were taking eplerenone). This is thought to occur through regulation of intestinal K^+ excretion [30]. Specifically for spironolactone, it was found to be associated with a significant increase in serum K^+ after 2 months of administration, without any side ef-

fects [31]. At the same time, Valencia *et al.* used spironolactone in PD patients who became hypokalemic because of diarrhea at a dose of >65 mg/24 hours and found a change in serum K^+ after 12 months of treatment, with 20% developing hyperkalemia [32], although others did not find any change in serum K^+ in 12 PD patients with hypokalemia who received 25 mg spironolactone/24 hours [33]. The effectiveness of spironolactone in improving hypokalemia is attributed to its effect on the epithelial cells of the colon, where it inhibits the receptors of mineralocorticoids, preventing the loss of K^+ through the large intestinal epithelium [34]. Of course, when there is residual renal function, the action of spironolactone in the distal renal tubules is added, with an additional benefit in improving K^+ levels.

Despite the small number of patients in our study, it is impressive that everyone was taking β-blocker, 3/10 eplerenone, 2/10 heparin, 3/10 NSAIDs, 3/10 ACE inhibitors, and 8 icodextrin dialysate, each of which could somehow increase blood K^+ concentration, but all of them had moderate to severe hypokalemia.

The low blood K^+ concentration can affect the myocardial membrane resting potential, repolarization, and stimulus conduction velocity, which has an impact on mortality. Severe hypokalemia in PD patients may be manifested by muscle disorders (muscle weakness, rhabdomyolysis) [17], cardiac arrhythmia, weak peripheral pulse, orthostatic hypotension, decreased cardiac output [35], and in the intestine with constipation, which favors the occurrence of peritonitis episodes [35] [36].

In PD patients, low serum K^+ levels are positively correlated with all causes of death, but also with cardiovascular events [6]. It is also associated with sudden deaths in patients with cardiovascular disease [37]. In fact, hypokalemia has been found to be an independent prognostic risk factor for survival [16] and a risk factor for cardiovascular causes of death [38]. Hypokalemia in PD patients is generally associated with reduced survival [5] [16]. In fact, a large study of 10,468 PD patients revealed that low serum K^+ levels contributed significantly to the occurrence of death in them, compared to hemodialyzed patients. It was also found that patients with serum $K^+ < 4$ mmol/L were 2 times more likely to die than those with serum $K^+ > 5.5$ mmol/L [38].

Xu *et al.* considered that patients on PD with serum K^+ 3.0 - 3.5 mmol/L can restore their levels with a diet rich in K^+, while when the blood levels are 2.5 mmol/L intravenous administration is required [6]. This is important because hypokalemia is dangerous for the development of arrhythmias and is associated with sudden deaths, while restoring serum potassium with foods rich in K^+ is not as easy and as fast as required.

In Australia, it is recommended to administer a PD exchange bag with a K^+ concentration of 3 mmol/L when the serum K^+ is 3 - 5 mmol/L and a 4 mmol/L solution when the serum K^+ is <3 mmol/L. In our country, in general, the dose of K^+ given intraperitoneally to PD patients with hypokalemia corresponds (usually 5 ml of 10% KCI is added to every 2 L of PD solution). In contrast to this conservative treatment, Spital & Sterns, who gave intraperitoneal K^+ to five PD pa-

tients with hypokalemia in only one PD exchange bag of 2 L, found that it was well tolerated at a density of 20 mmol/L, while administration of a solution of 40 mmol/L in one PD exchange bag caused severe abdominal pain in some of them [2]. We administered a PD solution with 20 mmol/L K^+, continuously in each exchange bag until the K^+ of the blood concentration became > 4 mmol/L, which restored the blood K^+ levels gradually, without significant side effects (abdominal pain during infusion of the dialysate was found in only two of our patients, and after good mixing of the solution in one of them it did not recur), with no hyperkalemia being found in any of our patients.

Amirmokri *et al.* were, of course, more daring and, in 5 patients on Continuous Ambulatory Peritoneal Dialysis (CAPD) with severe hypokalemia, they gave K^+ intraperitoneally at a dose of 60 - 80 mmol K^+ in a 2 L PD exchange bag, *i.e.*, 30 - 40 mmol/L. Specifically, K^+ was added to a PD dialysate of 2.5% glucose during the night exchange, and serum K^+ was determined after 2, 4, 6 - 8, and 20 - 24 hours of solution infusion in the abdomen. They found an increase in serum K^+, on average from 3.2 mmol/L (range 2.6 - 3.6 mmol/L) to 4.1, 4.2, 4.1, and 4.2 mmol/L, respectively, during the reported blood sampling hours [23], without side effects. It is noted that such a high concentration of K^+ in a PD bag was administered for the first and last time.

It is concluded that intraperitoneal administration of K^+ at a concentration of 20 mmol/L in each PD exchange bag, until the concentration of K^+ increases to levels greater than 4 mmol/L, is safe and is not accompanied by any side effects.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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