



Hypokalemia in peritoneal dialysis patients in Thailand: the pivotal role of low potassium intake

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Abstract

Objective Hypokalemia is highly prevalent in chronic peritoneal dialysis (PD) patients worldwide, particularly in Thailand. This study aims to investigate the major determinants of hypokalemia in Thai PD patients.

Methods A cross-sectional study was performed in chronic PD patients at 4 PD centers in Bangkok, Thailand. Hypokalemia was defined if the average serum potassium level during the last 3 consecutive visits was < 3.5 mEq/L. Patients and/or their caregivers were asked to perform a 3-day dietary food record and take pre- and post-meal pictures following the instructed protocol. Daily dietary nutrients, including potassium, were estimated by a single dietician using INMUCAL-N software. Total potassium excretion was determined by 24-h PD effluents and urine collection. Intracellular and extracellular water values (ICW and ECW, respectively) were measured by electrical bioimpedance assay (BIA) to indirectly explore the role of intracellular potassium shift in hypokalemia.

Results Among 60 eligible PD patients, 19 (31%) had hypokalemia. Hypokalemic patients had significantly lower dietary potassium intake (24.4 ± 11.1 vs. 30.5 ± 9.4 mEq/day, $p = 0.031$) and lower total potassium excretion (28.5 ± 8.4 vs. 36.7 ± 11.2 mEq/day, $p = 0.006$) compared to normokalemic patients. Both groups had comparable values of ICW and ECW. On logistic regression, there was no significant correlation between hypokalemia and daily PD exchange volume, total Kt/Vurea, residual renal function, concurrent medications (insulin, diuretics, renin–angiotensin–aldosterone inhibitor, and beta-blockers) or ICW. Low dietary potassium was an independent risk factor for hypokalemia.

Conclusion Low dietary potassium intake, rather than increased potassium excretion or intracellular shift, is the major contributing factor of hypokalemia in Thai chronic PD patients. Dietary intervention or potassium supplement protocol should be implemented.

Keywords Peritoneal dialysis · Hypokalemia · Dietary potassium · Food record

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Introduction

The prevalence of hypokalemia among chronic peritoneal dialysis (PD) patients from previous studies varied from 10 to 36% [1, 2]. The importance of hypokalemia has recently become a major concern in Thailand after the release of data from the Thailand Peritoneal Dialysis Outcomes and Practice Patterns Study (PDOPPS). The study demonstrated that Thailand had the highest prevalence of hypokalemia (46%) [3]. Since potassium is critical for maintaining cellular function, serum potassium alteration inevitably causes cellular malfunction, particularly of cardiac muscle cells. Indeed, hypokalemia induces cardiac problems, including arrhythmias and decreased cardiac output [4]. Hypokalemia

is also associated with malnutrition, severe comorbidities, and decreased patient survival [5–10]. Moreover, hypokalemia may affect gastrointestinal motility, resulting in peritonitis through intestinal bacterial overgrowth and subsequent transmural migration of enteric organisms [11–13].

In general, serum potassium changes are mainly contributed by alteration of the nominator, total body potassium, rather than the denominator, total body water. Regulation of potassium in the human body or potassium homeostasis is a balance between external and internal factors. In case of PD patients, the former includes renal excretion, dialysate excretion, and potassium intake, while the latter involves potassium distribution across the cells, which is mainly affected by insulin and catecholamine [14]. As most of the prior studies did not directly address the leading causes of hypokalemia, and the reasons behind the high prevalence of hypokalemia in Thailand remain uncertain, this study aims to conduct a detailed investigation of the major determinants of hypokalemia in Thai PD patients.

Materials and methods

Patient selection

From May to December 2018, a cross-sectional study on Thai PD patients undergoing PD for more than 3 months was performed in 4 PD centers in Bangkok, including King Chulalongkorn Memorial Hospital, Bangkok Metropolitan Administration General Hospital, Taksin Hospital, and Banphaeo Hospital. Only stable patients aged 18–70 years were eligible. Patients with any of the following criteria were excluded from the study: (1) inability to provide detailed dietary records; (2) diarrhea or vomiting in the past 1 week; (3) peritonitis within 1 month; (4) unwillingness to participate in this study; (5) bed-ridden state or terminal illness. Patient's demographic data and data on dietary potassium intake, residual renal potassium excretion, and PD potassium excretion were collected. All participating patients provided written informed consent. The study protocol was approved by the ethics committee of King Chulalongkorn Memorial Hospital and each participating center.

Demographic and clinical characteristics

Collected data included demographics, comorbidities, current medications, dialysis-related information, and serum biochemistries. The degree of comorbidity was assessed by Davies comorbidity score, which comprises seven comorbid conditions, i.e., malignancy, ischemic heart disease, peripheral vascular disease, left-ventricular dysfunction, diabetes mellitus, systemic collagen vascular disease, and others (chronic obstructive pulmonary disease, liver cirrhosis,

and asthma) [15]. Regarding the PD prescription, daily dialysate exchange volume, hypertonic dialysate, and mode of PD (continuous ambulatory PD or automated PD) were reviewed. Peritoneal membrane transport status was also retrieved by reviewing recent peritoneal equilibration test (PET) results within the past 3 months. Dialysis adequacy data were expressed as the Kt/Vurea. The 24-h collection of dialysate effluent and urine were used to calculate peritoneal and renal Kt/Vurea, respectively. The total Kt/Vurea was calculated as the sum of the peritoneal and renal Kt/Vurea. All laboratory tests were performed at the Nephrology unit, King Chulalongkorn Memorial Hospital.

Detection of hypokalemia and measurement of serum potassium

Hypokalemia was defined if the average serum potassium level during the last 3 consecutive visits, including the study enrollment visit, was <3.5 mEq/L [2]. To avoid factitious change in serum potassium during blood collection and handling, a protocol collection was emphasized to trained nurses in brief as follows: applying the tourniquet during venous puncture for no longer than 1 min, completing air dry after cleaning the puncture area with 70% alcohol, using a 20–22 gauge needle, placing the bevel of the needle face-up, drawing blood only from an antecubital region of the arm, pulling a syringe plunger gently, filling the blood into sterile sodium citrate tube and mixing thoroughly by inverting the tube slowly 3–4 times, separating the serum by centrifugation at 150 g for 5 min, and shipping the separated serum in an ice-shield box to the central laboratory. An indirect ion-selective electrode method was used to determine serum potassium levels.

Estimation of dietary potassium intake

At study entry, patients and/or their caregivers were trained to perform a 3-day dietary food record by dietitians [16]. They received intensive education to take pre- and post-meal photographs of all foods the patients had taken with the instructed protocol using their camera. Single study dietitian integrated information from 3-day dietary records and food photographs to estimate daily dietary intakes of each patient, including dietary protein intake (DPI), dietary energy intake (DEI), and dietary contents, particularly potassium, using Institute of Nutrition, Mahidol University the calculation of nutrients (INMUCAL-N) software, the database for food composition analysis, based on the Thai food composition table developed by Mahidol University [17, 18]. Nutritional information of most Thai food could be found in the software's database.

Measurement of total potassium excretion

The 24-h dialysate and urine samples were collected at study entry to determine the total potassium excretion. Both specimens were tested for potassium levels by the same technique as mentioned previously for serum potassium. Peritoneal potassium excretion (mEq) equaled 24-h dialysate potassium concentration (mEq/L) \times 24-h dialysate volume (L), while renal potassium excretion (mEq) equaled 24-h urine potassium concentration (mEq/L) \times 24-h urine volume (L). Total potassium excretion was then calculated by the summation of peritoneal potassium excretion (mEq) and renal potassium excretion (mEq).

Measurement of potassium distribution across the cell

The patients' volume status as well as intracellular and extracellular water (ICW and ECW, respectively) status was determined by multiple-frequency electrical bioimpedance assay (BIA) (Body Composition Monitor [BCM], Fresenius Medical Care, Germany) to explore the role of intracellular potassium shift in hypokalemia. BIA was performed by placing electrodes on the wrist and ankle unilaterally with patients lying in a supine position to ensure equilibration of body fluid. Patients were measured without dialysate in the peritoneum. Utilizing multiple-frequency BIA, total body water (TBW) could be measured and subcategorized into ECW and ICW. Expected normal ECW for each patient could be estimated using their weight and body composition. Overhydration (OH) indicated the difference between measured ECW and expected ECW. The results were considered valid only if the operative quality score of the BIA measurement was over 90%.

ICW has previously been demonstrated to correlate directly with serum potassium in PD [1]. In this regard, ICW serves as an indirect marker of intracellular and, supposedly, total body potassium, given the majority of potassium resides in the cells. To explore the effect of intracellular potassium shift on serum potassium, the proportion of ICW in relation to ECW as ECW/ICW ratio was also calculated. The hypothesis was that if hypokalemia was accountable by the intracellular shift of potassium, not potassium depletion, the intracellular pool of potassium represented by ICW would be increased relative to the ECW, resulting in a decreased ECW/ICW ratio.

Statistical analysis

Statistical analysis was performed using STATA version 14.0 (StataCorp, College Station, TX). Results were expressed as mean \pm SD or percentage unless otherwise specified. To compare differences between the hypokalemic

and normokalemic groups, *t* test and Mann–Whitney *U* test were used for continuous variables, while the Chi-square test or Fisher's exact test were used for categorical variables. The association between clinical characteristics and hypokalemia were analyzed using logistic regression. Variables with *p* value $<$ 0.20 in a univariable regression analysis were incorporated into the multivariable model. Statistical significance was determined at an alpha of less than 0.05.

Results

Demographic features of subjects

Table 1 illustrates the baseline clinical and laboratory characteristics of the 60 participating chronic PD patients. The distribution of serum potassium levels is depicted in Fig. 1. Hypokalemia, according to the above criteria, was observed in 31%. Comparing between PD patients in normokalemic and hypokalemic group, there were no significant differences in age, gender, DM, concurrent medications (insulin, ACEi/ARB, beta-blockers, loop diuretics, and spironolactone), dialysis characteristics, residual renal function, hemoglobin, serum albumin, serum phosphate, total Kt/Vurea, and total CCr between the two groups except higher incidence of high peritoneal membrane transporter status in hypokalemic patients (Table 1).

Dietary pattern, potassium excretion, and volume status

Table 2 details the dietary pattern of all participating patients in the study. The distribution of daily dietary potassium intake is shown in Fig. 2. Of note, daily dietary potassium intake was significantly lower in hypokalemic patients compared to normokalemic patients (24.4 ± 11.1 vs. 30.5 ± 9.4 mEq/day, $p = 0.031$). Moreover, the daily potassium excretion was also lower in the hypokalemic group compared to the normokalemic group (28.5 ± 8.4 vs. 36.7 ± 11.3 , $p = 0.006$). There was no significant difference in ICW and ECW/ICW values between the two groups (16.0 ± 3.5 vs. 17.3 ± 5.3 L and 1.0 ± 0.2 vs. 0.9 ± 0.2 L, $p = 0.335$ and 0.151 , respectively).

Association between clinical characteristics and hypokalemia

By univariable analysis, every 10 mEq increase in daily dietary potassium intake was associated with lower risk of hypokalemia (odds ratio [OR] 0.52, 95% CI 0.29–0.96, $p = 0.028$), while the use of insulin, loop diuretic, hypertonic glucose PD solution, high peritoneal membrane transport, and residual urine volume of $>$ 100 mL/day were not significantly

Table 1 Baseline clinical characteristics

	Total (N=60)	Normoka- lemia (N=41)	Hypokalemia (N=19)	<i>p</i> value*
Age, years	55 ± 17	56 ± 20	53 ± 11	0.591
Male gender (%)	45%	34%	21%	0.759
Body mass index (kg/m ²)	22.4 ± 4.3	22.3 ± 4.4	22.7 ± 13.2	0.731
Diabetes mellitus (%)	33%	32%	37%	0.695
Davies comorbidities (%)				
0	78%	80%	74%	0.818
1	17%	15%	21%	
2	5%	5%	5%	
Medications (%)				
ACE inhibitor, ARB	47%	46%	46%	0.941
Spironolactone	2%	0%	5%	0.139
Beta-blocker	38%	34%	47%	0.327
Loop diuretics	40%	34%	53%	0.174
Insulin	20%	15%	32%	0.127
Potassium supplement	50%	48%	56%	0.573
Supplement dose (mEq/day)	19.1 ± 9.8	20.2 ± 10.0	17.0 ± 9.7	0.410
RRF > 100 mL/day (%)	60%	59%	63%	0.734
PD vintage (months)	30.2 ± 28.4	30.7 ± 26.4	29.2 ± 33.1	0.853
Dialysis characteristics				
Total dialysis exchange volume (L/day)	9.7 ± 2.2	9.8 ± 1.6	9.6 ± 3.2	0.710
Hypertonic PDF use (%)	22%	22%	22%	0.982
APD (%)	35%	42%	21%	0.123
High transporter (%)	28%	17%	57%	0.043
Serum potassium (mEq/L)	3.9 ± 0.6	4.2 ± 0.5	3.2 ± 0.3	<0.001
Serum albumin (g/dL)	3.5 ± 0.5	3.4 ± 0.5	3.6 ± 0.5	0.256
Serum phosphorus (mg/dL)	4.5 ± 1.7	4.4 ± 1.6	4.8 ± 1.8	0.376
Hemoglobin (g/dL)	10.3 ± 1.6	10.1 ± 1.6	10.7 ± 1.5	0.165
Total (Kt/Vurea)	2.2 ± 0.6	2.2 ± 0.5	2.2 ± 0.8	0.804
Total CCr (L/week)	68.5 ± 37.8	74.4 ± 25.2	77.3 ± 56.2	0.225
PCR (g/day)	58.9 ± 12.8	60.7 ± 12.3	55.3 ± 13.2	0.129

Data are expressed in mean ± SD or percentage as appropriate

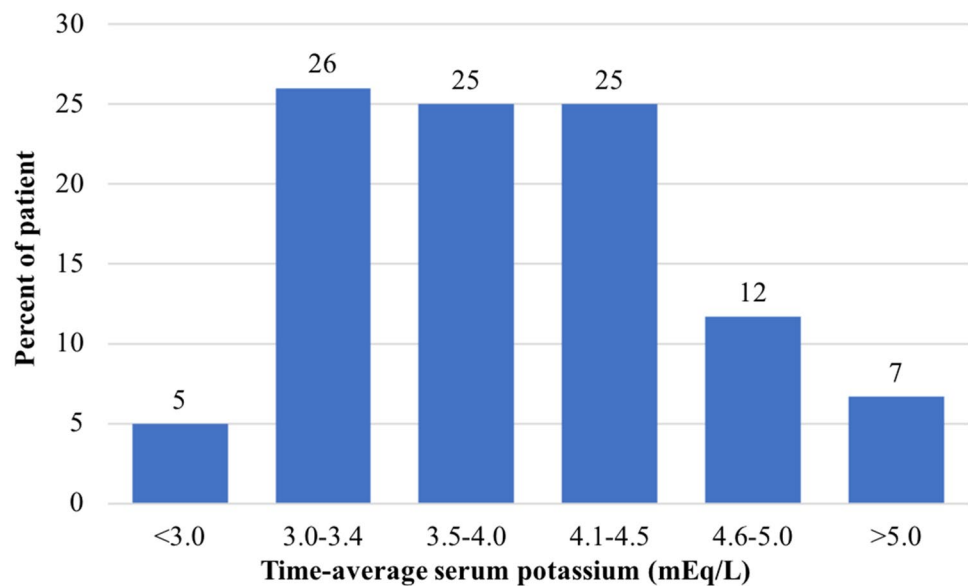
ACE angiotensin-converting enzyme, APD automated peritoneal dialysis, ARB angiotensin II receptor blockers, CCr creatinine clearance, HIV human immunodeficiency virus, PD peritoneal dialysis, PDF peritoneal dialysis fluid, PCR protein catabolic rate, RRF residual renal function

*Comparison between hypokalemic and normokalemic groups

associated with hypokalemia (Table 3). Multivariable analysis was performed by utilizing variables with *p* value < 0.200 in the univariable analysis, including insulin treatment, loop diuretic use, and high peritoneal membrane transport into consideration. Again, every 10 mEq increment in daily dietary potassium was still the sole parameter that was independently associated with decreased risk of hypokalemia (adjusted OR 0.85, 95% CI 0.73–0.99, *p* = 0.045).

Discussion

The present study demonstrated that hypokalemia was identified in 31% of the participating Thai PD patients. When compared with the normokalemic group, hypokalemic patients had a significantly higher prevalence of high peritoneal membrane transport and consumed diets

Fig. 1 Distribution of serum potassium of the participating PD patients ($N=60$)**Table 2** Dietary pattern, potassium excretion, and volume status

	Total ($N=60$)	Normokalemia ($N=41$)	Hypokalemia ($N=19$)	p value*
Dietary potassium intake (mEq/day)	28.6 ± 10.3	30.5 ± 9.4	24.4 ± 11.1	0.031
Dietary sodium intake (mg/day)	1731.8 ± 965.8	1715.9 ± 173.5	1768.1 ± 124.2	0.850
Dietary phosphate intake (mg/day)	445.3 ± 164.7	444.1 ± 25.5	448.1 ± 40.6	0.932
Dietary calorie intake (kcal/day)	1088.5 ± 335.4	1091.7 ± 55.4	1081.2 ± 69.6	0.912
Dietary calorie intake (kcal/kg/day)	19.5 ± 7.4	19.0 ± 1.2	20.4 ± 1.8	0.522
Dietary protein intake (g/day)	49.6 ± 18.4	49.8 ± 2.9	49.3 ± 4.2	0.930
Dietary protein intake (g/kg/day)	0.89 ± 0.05	0.87 ± 0.06	0.94 ± 0.10	0.536
Urinary potassium excretion (mEq/day)	5.9 ± 9.1	7.1 ± 10.5	3.3 ± 3.9	0.132
Dialysate potassium excretion (mEq/day)	28.3 ± 8.2	29.7 ± 7.5	25.3 ± 9.0	0.051
Total potassium excretion (mEq/day)	34.1 ± 11.1	36.7 ± 11.3	28.5 ± 8.4	0.006
TBW (L)	32.4 ± 7.3	32.6 ± 7.1	32.1 ± 8.1	0.790
ECW (L)	15.6 ± 3.5	15.3 ± 2.5	16.1 ± 5.0	0.436
ICW (L)	16.9 ± 4.8	17.3 ± 5.3	16.0 ± 3.5	0.335
ECW/ICW	0.9 ± 0.2	0.9 ± 0.2	1.0 ± 0.2	0.151
OH (L)	$2.1 (1.3-3.2)$	$1.9 (1.1-3.0)$	$2.1 (1.3-5.9)$	0.352

Data are expressed in mean \pm SD, median (interquartile range), or percentage where appropriate

ECW extracellular water, ICW intracellular water, K potassium, TBW total body water, OH overhydration

*Comparison between hypokalemic and normokalemic groups

with lower potassium contents. By multivariable logistic regression analysis, low dietary potassium intake was an independent risk factor of hypokalemia, and an increase in dietary potassium intake by every 10 mEq/day decreased the risk of hypokalemia by 15%.

Hypokalemia is not uncommon in PD patients and may put patients at increased risks of cardiovascular diseases, PD-related peritonitis, and death [12, 19, 20]. Remarkably, high prevalence of hypokalemia in Thailand according to data from PDOPPS [3] and the present study raises concern

for an increased risk of unfavorable outcomes among Thai PD patients. However, the data on crucial patient outcomes are not available in this cross-sectional study. The hypokalemia prevalence of 31% in this study slightly lower compared with previous reports [3, 21] might be, in part, resulted from increasing awareness of the condition among Thai PD care providers suggested by the prescription of continuous oral potassium supplement in as high as 50% of the patients.

As stated earlier, potassium homeostasis is the primary determining factor of serum potassium levels. Indeed,

Fig. 2 Distribution of daily dietary potassium intake of the participating PD patients ($N=60$)

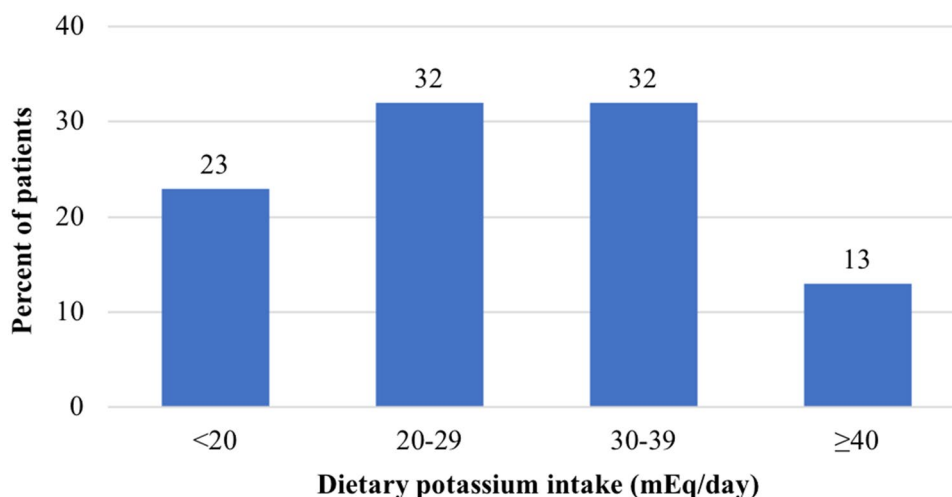


Table 3 Association between clinical characteristics and hypokalemia by logistic regression

Characteristics	Univariable analysis		Multivariable analysis	
	Odd ratio (95% CI)	<i>p</i> value	Odd ratio (95% CI)	<i>p</i> value
Insulin	2.69 (0.73–9.86)	0.137	1.71 (0.73–9.86)	0.717
Loop diuretic	2.14 (0.71–6.49)	0.176	1.05 (0.71–6.49)	0.973
Hypertonic PDF use	1.26 (0.36–4.48)	0.712	–	–
High transporter	6.67 (0.95–46.56)	0.056	14.35 (0.95–215.89)	0.054
Urine > 100 mL/day	1.21 (0.40–3.72)	0.734	–	–
Dietary potassium, every 10 mEq/day	0.52 (0.29–0.96)	0.028	0.85 (0.73–0.99)	0.045

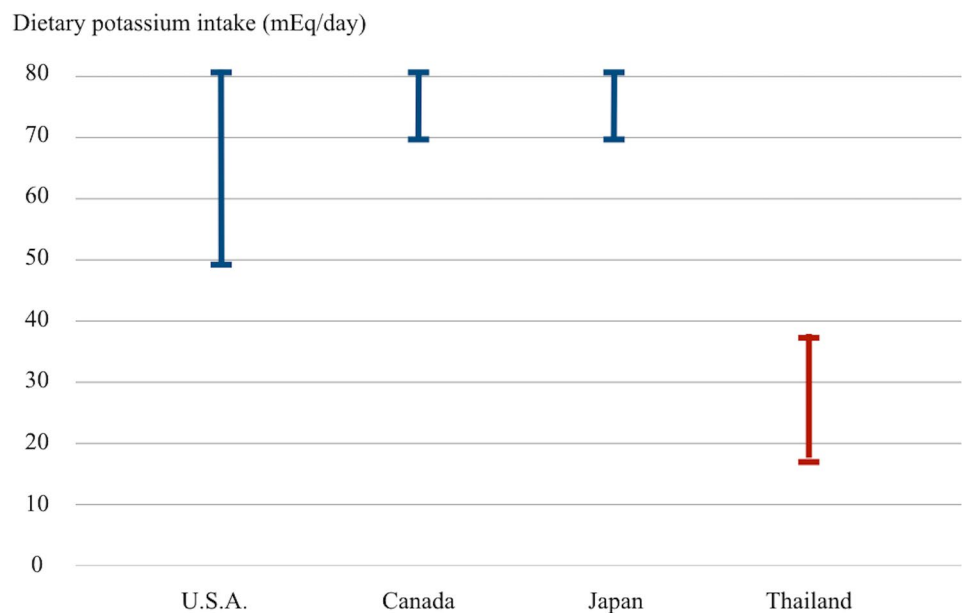
CI confidence interval, PDF peritoneal dialysis fluid

physiological potassium homeostasis is governed by two different balances: the internal balance, including potassium distribution between the intracellular and extracellular compartments, and external balance, which comprises uptake from outside and potassium loss. Although certain previous studies attributed hypokalemia in PD to inadequate potassium intake [2, 22], the evidence is still lacking. In the present study, dietary potassium intake in hypokalemic patients is significantly lower than in the normokalemic group (Table 2). Of interest, dietary potassium intake in both hypokalemic and normokalemic patients in the present study was much lower than that of the recommended values [23, 24]. The results of studies in Chinese patients [1, 25] were in agreement with the present study, in that the amount of dietary potassium intake in both Chinese and Thai patients is less than Japanese, American, and Canadian patients approximately by half (Fig. 3) [3, 26, 27]. Although a traditional Thai diet is rich in vegetables making general Thai patients exposed to foods with high potassium, the cooking method usually involves extensive boiling and frying, resulting in a substantial reduction in potassium content in the dishes. The significantly low dietary potassium intake would explain the high prevalence of hypokalemia in Thai

PD patients, according to PDOPPs [3]. Besides low dietary potassium intake, the nutritional assessment also suggested that PD patients in Thailand also had caloric and protein intake below the recommended level [24, 28], highlighting the significance of inappropriate dietary patterns overall.

Increased potassium removal through PD effluent and urine as a cause of hypokalemia is another possibility. Nevertheless, the present study demonstrated lower potassium excretion in patients with lower serum potassium levels, suggesting that potassium was in a steady state where the potassium intake must equal potassium output to maintain potassium balance. Therefore, the concept of total body potassium depletion leading to decreased potassium excretion is consistent with this observation, rather than increased potassium excretion as a cause of potassium depletion or hypokalemia.

The role of the intracellular potassium shift was indirectly evaluated in this study. First, factors well established to cause intracellular potassium shift, including diabetes or insulin use and the use of hypertonic PD solution, were not significantly associated with hypokalemia. Second, as most potassium in the human body is located in the cells, the precise determination of total body potassium requires

Fig. 3 Daily dietary potassium intake stratified by country [3, 26, 27]

measurement of intracellular potassium, for example, utilizing percutaneous skeletal muscle biopsy [29]. However, such a method is complicated and not feasible in clinical practice. Body composition analysis by BIA, on the other hand, was simple and could potentially serve this purpose. ICW, which has been demonstrated to significantly correlate to serum potassium in a prior report by Yu et al. [1], was not different between normokalemic and hypokalemic groups in the present study. Moreover, suppose that the lower serum potassium among patients with hypokalemia is mediated by intracellular shift. In that case, a lower ECW/ICW ratio is expected in hypokalemic patients, because, without body potassium depletion, the shift of potassium into cells would increase ICW and, to some degree, cause a slight decrease in ECW. However, our results showed the opposite trend of slightly higher ECW/ICW ratio among hypokalemic patients than normokalemic patients, although not reaching statistical significance (Table 2). This finding provides indirect evidence against the concept of intracellular shift as the major mechanism of hypokalemia in Thai PD patients.

The present study has several strengths. This is a pioneer work to examine macro- and micronutrient data by employing pre- and post-meal photography in addition to the standard 3-day dietary food record, which would help establish

the most accurate dietary data. Moreover, interpretation of the acquired dietary information by a single dietitian who was utterly blind to each patient's serum potassium status helped to decrease interobserver variability and interpretation biases. Finally, this study was performed in Thailand, where hypokalemia is exceptionally high for an uncertain reason. Depicting the significant association between low dietary potassium intake and hypokalemia provide strong evidence that inadequate dietary potassium was the critical mechanism of hypokalemia in PD patients, at least in Thailand (Table 4).

Nevertheless, our study has limitations. This study only included patients living in Bangkok whose dietary patterns may not represent the diets of patients living from other regions of Thailand, given that there is a great variety of local foods nationwide. Some non-significant associations observed in this study might result from limited statistical power, since the number of participating patients was relatively small.

In summary, we investigated the mechanism of hypokalemia in Thai PD patients and demonstrated that low dietary potassium intake, rather than increased potassium excretion or intracellular shift, is an independent risk factor of hypokalemia in Thai PD patients. Dietary intervention to improve

Table 4 Comparison of dietary intake between the present study and recommendations

Nutritional intake	ISRNM 2013[23, 24]	KDOQI 2000[24, 28]	Present study
Potassium (mEq/day)	Not usually an issue	40–80	28.6 ± 10.3
Calories (kcal/kg/day)	30–35	30–35	19.5 ± 7.4
Protein (g/kg/day)	> 1.2	> 1.2	0.9 ± 0.4
Sodium (mEq/day)	1800–2300	1800–2300	1731.8 ± 965.8
Phosphate (mg/day)	800–1000	800–1000	445.3 ± 164.7

patients' food intake or oral potassium supplement protocol should be implemented to ensure appropriate serum potassium level and potentially improve treatment outcomes in this particular group of patients.

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Author contributions All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by MV and PP. The first draft of the manuscript was written by MV, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials The present manuscript has no associated depository data.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

Ethical approval The study protocol was approved by the ethics committee of King Chulalongkorn Memorial Hospital and each participating center.

Consent to participate Each participating patient provided informed consent.

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