Urine Potassium Per Hour as a Marker for Renal Potassium Losses

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Background: *Hypokalemia, serum potassium (K) < 3.5 mEq/L, is a serious and common clinical problem. The traditional diagnosis of renal potassium losses is 24-hr urine potassium ($24U_K$) ≥ 20 mEq/day during hypokalemia. Immediate replacement of potassium is often required to prevent complication but may normalize serum K during 24-hr urine collection and render the test inconclusive.*

Material and Method: *The authors examined the ability of urinary indices including $24U_K$, transtubular potassium gradient (TTKG), fractional excretion of potassium (FE_K), urine potassium-creatinine ratio ($U_{K/Cr}$) and spot U_K and introduced urine potassium per hour during the first 8 hours (U_K/hr) as a novel index for evaluation of hypokalemia during treatment. Any serum K level ≥ 4 mEq/L during urine collection was defined as normalized serum K. In the present study, the final classification of renal K losses in non-normalized 24-hr serum K group was made when $24U_K \geq 20$ mEq/day. In normalized group, the final classification of renal or non-renal K losses was based on the majority of the results of four urine indices including TTKG, FE_K, $U_{K/Cr}$, and spot U_K.*

Results: *Of 61 patients (renal:non-renal = 50:11), 51% and 18% met the criteria of normalized 24-hr and 8-hr serum K. Over all, the U_K/hr ≥ 0.9 mEq/hr can indicate renal K losses with a sensitivity of 96% and specificity of 72.7% compared with the $24U_K \geq 20$ mEq/day of 100% and 54.5%, respectively. In a subgroup of normalized 24-hr serum K, the sensitivity and specificity of U_K/hr = 95.5% and 77.8% whereas $24U_K$ = 100% and 44.4%, respectively.*

Conclusion: *U_K/hr is a new practical, simple, and reliable marker that can be applied to evaluate hypokalemic patients during treatment with comparable sensitivity and specificity with $24U_K$.*

Keywords: *Hypokalemia, Urine potassium per hour (U_K/hr), 24-hour urine potassium ($24U_K$), Transtubular potassium gradient (TTKG), Fractional excretion of potassium (FE_K), Urine potassium-creatinine ratio ($U_{K/Cr}$)*

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Hypokalemia is a commonly encountered clinical problem[1]. Immediate treatment is sometimes necessary to avoid a life threatening condition[2]. The etiology of hypokalemia is often not immediately obvious at initial presentation. Most hypokalemia results from potassium depletion induced by abnormal losses of potassium either due to renal or non-renal losses[3-7]. The kidney plays a major role in long-term potassium homeostasis[8-10]. The measurement of urinary potassium excretion is very helpful in differentiation of renal from non-renal K losses and assignment of appropriate treatment. 24-hour urine potassium ($24U_K$) collection is often used as the gold standard[9,11]. Urinary K (U_K) excretion of more than 20 mEq/24-hr during hypokalemia is indicative of excessive renal K losses[12,13]. In contrast, non-renal K wasting is suspected when urinary K excretion is less than 20 mEq/24-hr[11,14]. However, this is not feasible in many cases. It is sometimes difficult to obtain complete 24 hr urine collection. Moreover, the serum K levels during 24-hour urine collection might not be persistently low due to treatment. Excess of potassium from treatment would result in excretion of K into urine. This may affect the total excretion of urinary potassium and makes the interpretation of 24-hour urine K results uncertain[12,15].

Many authors tried to employ other non-invasive indices such as transtubular potassium concentration gradient (TTKG)[16], fractional excretion of potassium (FE_K)[17], urine potassium-creatinine ratio

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($U_{K/Cr}$)[18] and spot U_K[13] to determine the diagnosis. These parameters are simpler but may be less accurate compared to a 24-hour urine collection, since these parameters assess potassium excretion only at a single point in time. No study has been formally evaluated the accuracy of these markers in a large group of patients. Hence, the authors conducted a study to validate the previously known urine indices and introduced a new urine index that is both simple and reliable in indicating renal potassium losses in hypokalemic patients during treatment. In the present study, the authors evaluated the urine potassium per hour during the first 8 hours (U_K/hr) and compared this value with 24U_K and other urinary indices.

Material and Method

Subjects

All untreated hypokalemic patients were recruited from the emergency room, medicine and surgery departments. Initially, the etiology of hypokalemia was unknown. All enrolled patients followed the protocol of the present study. Hypokalemia was defined as a serum K level below 3.5 mEq/L. The exclusion criteria were serum creatinine > 1.2 mg/dL, previous renal disease, patients with shock, congestive heart failure, nephrotic syndrome, or cirrhosis. All participants gave their written informed consent after receiving oral and written information concerning the present study according to the Declaration of Helsinki II. The study protocol was approved by the Ethical Committee for Research Involving Human Subjects of the Ramathibodi Hospital, Mahidol University (ID 08-48-16).

The final diagnosis of the cause of hypokalemia in each patient was assigned after the clinical course and careful investigations. The definite diagnoses of various diseases causing hypokalemia were as follows. Hypomagnesemia was defined by serum magnesium < 1.4 mEq/L on two occasions. Solute diuresis was defined by (1) the urine osmolality above 300 mOsm/kg and (2) total solute excretion above 900 mOsm/day. The diagnosis of diabetic ketoacidosis was based on (1) blood glucose ≥ 250 mg/dL, (2) ketosis, and (3) metabolic acidosis (arterial pH < 7.3). The diagnosis of a hyperglycemic hyperosmolar state was based on (1) blood glucose ≥ 600 mg/dL, (2) hyperosmolality (serum osmolality > 350 mOsm/kg), and (3) absence of or mild acidosis and ketonemia. The diagnosis of hyperaldosteronism was a plasma aldosterone level > 160 pg/mL (normal 10-160 pg/mL in a supine position) on two occasions. Thyrotoxic periodic paralysis was defined by (1) hyperthyroidism [(i) serum TSH < 0.3 μIU/mL (normal 0.3-4.0) and (ii) free T4 > 2.0 ng/dL (normal 0.9-2.0) or T3 > 160 ng/dL (normal 80-160), or total T4 > 12 μg/dL (normal 4.0-12.0)], (2) attacks occurring suddenly with generalized weakness and preserved consciousness, and (3) episodes association with hypokalemia. The diagnosis of distal renal tubular acidosis was based on (1) hyperchloremia metabolic acidosis with normal GFR (> 60 mL/min), and (2) abnormal long acid loading test which was defined as failure to acidify urine (pH > 5.5) and urine excretion of ammonia < 50 mEq/day in the presence of systemic acidosis (blood pH < 7.35) [11].

Method

Patient characteristics were documented. Blood samples were initially collected from patients for determination of electrolytes, blood urea nitrogen, creatinine, calcium, phosphate, magnesium, albumin, uric acid, and osmolality. Serum K was measured every 8 hours. The first voiding urine was collected for measuring TTKG, FE_K, $U_{K/Cr}$, and spot U_K. Then, 24 hour urine was collected every 8 hour interval for volume, potassium, sodium, creatinine, calcium, phosphate and magnesium measurements.

After the first voiding urine collection, the potassium supplements were given. The treatment protocol for mild hypokalemia (K 3.0 to < 3.5 mEq/L) was oral potassium chloride (KCl) elixir 20 mEq every 8 hours. In moderate hypokalemia (K 2.5 to < 3.0 mEq/L), oral KCl elixir 40 mEq every 8 hours plus intravenous KCl infusion at the rate 5 mEq/hr were given. In severe hypokalemia (K < 2.5 mEq/L) or symptomatic hypokalemia (*e.g.* arrhythmias), oral KCl elixir 40 mEq every 8 hours plus intravenous KCl infusion at the rate 10 mEq/hr were given with electrocardiographic monitoring. Nevertheless, the judgment of the potassium supplement rate was based on the clinician's decision against clinical setting and the risk of hyperkalemia.

The U_K/hr during the first 8 hours was calculated by using the concentration of urine potassium during the first 8 hours (mEq/L) multiplied by urine volume during the first 8 hours (L) and divided by 8. TTKG, FE_K and $U_{K/Cr}$ were calculated from the equations [(urine/serum K)/(urine/serum osmolality)][16], [(urine/serum K)/(urine/serum creatinine)][17] and (urine K/urine creatinine)[18], respectively. Patients who had any serum K levels ≥ 4 mEq/L during urine collection were considered as a normalized serum K group, otherwise they were

considered as a non-normalized serum K group. The cutoff values to indicate renal or non-renal K losses of $24U_K$, TTKG, FE_K, $U_{K/Cr}$ and spot U_K were 20 mEq/day[12], 2.5[16], 6.5%[17], 2.5 mmol/mmol[18] and 20 mEq/L[13], respectively.

In the present study, the final classification of renal or non-renal K losses in each patient was as follows. In non-normalized 24-hr serum K group (serum K persistently < 4 mEq/L throughout 24 hours), the final classification of renal K losses was made when $24U_K \geq 20$ mEq/day. In normalized 24-hr serum K group, the final classification of renal or non-renal K losses was based on the majority of the results of four urine indices including TTKG, FE_K, $U_{K/Cr}$, and spot U_K. In case that the final classification of the patient was equivocal (renal:non-renal = 2:2), the patient would be classified as a non-conclusive group. The final classification of each patient was correlated with the authors' clinical diagnosis.

Statistical analysis

The mean ± standard deviation (SD) was used to describe the baseline characteristics and the mean ± standard error of mean (SEM) was calculated for patient diagnostic measures. The statistical significance of the difference in these characteristics across those with renal and non-renal potassium losses was examined by using Mann Whitney U-test or t-test (continuous variable and depend on the data distribution) and Chi-square or Fishers' exact test (categorical variable). The cutoff point of all urine indices were calculated from the receiver-operating characteristic (ROC) curve and the coordination table of the curve for the value of the highest sensitivity and specificity. The difference was considered significant if the p-value was < 0.05.

Results

Seventy-five patients with hypokalemia were included in the present study. Sixty-one patients completed the present study and included 19 males (31.2%) and 42 females (68.9%). The baseline characteristics are summarized in Table 1. Of 61 patients, 50 (82%) were classified as renal K losses and 11 (18%) as non-renal K losses. No patient was classified as a non-conclusive group in the present study. There were no significant differences in the mean serum K level at presentation, age and serum creatinine concentration between the two groups ($p > 0.05$). Of 61 patients, only 30 (49.2%) met the criteria for non-normalized 24-hr serum K. In contrast, 50 (82%) patients met the criteria for non-normalized 8-hr serum K (Table 1). The number of patients between renal and non-renal potassium losses in the non-normalized 24-hr serum K group was the factor that was significantly different ($p = 0.02$). Total 24-hr K supplement in renal K loss group was higher than that of non-renal K loss group ($p = 0.01$).

In the present study, drug induced renal potassium wasting (*e.g.* amphotericin B, aminoglycoside, ifosphamide, etc) was the leading cause of renal K losses (Table 2). For non-renal K losses, the major cause of disease was diarrhea. Two of 11 (18.2%) patients in the non-renal K loss group were due to cellular potassium shift (thyrotoxic periodic palalysis).

Table 1. Baseline characteristics of the patients

	Total	Mean ± SD		
		Renal	Non-renal	p-value
Number	61 (100%)	50 (82%)	11 (18%)	
Age (mean ± SD, year)	50.9 ± 16.7	52.0 ± 16.4	45.8 ± 18.0	NS
Sex (F:M)	42:19	34:16	8:3	NS
Serum K at presentation (mean ± SD, mEq/L)	2.94 ± 0.43	2.88 ± 0.41	3.20 ± 0.22	NS
Serum creatinine (mean ± SD, mg/dL)	0.8 ± 0.3	0.8 ± 0.3	0.9 ± 0.2	NS
Number of patients in non-normalized 24-hr serum K^+	30/61 (49.2%)	28	2	0.02
Number of patients in non-normalized 8-hr serum K^{++}	50/61 (82%)	41	9	NS
Total 24-hr K supplement (mean ± SD, mEq/day)	94 ± 85	104 ± 89	49 ± 47	0.01

$^+$ Non-normalized 24-hr serum K = patients who had serum K level < 4 mmol/L throughout 24 hours
$^{++}$ Non-normalized 8-hr serum K = patients who had serum K level < 4 mmol/L throughout the first 8 hours
NS = Not significant

Table 2. The definite causes of hypokalemia in all patients

Renal losses	50 cases
Hypomagnesemia	8
Diuretics	9
Drug induced	12
Distal renal tubular acidosis	2
Solute diuresis/DKA*/Hyperosmolar	9
Vomiting	3
Hyperaldosteronism	7
Non-renal losses	11 cases
Diarrhea	5
Low intake	4
Thyrotoxicosis periodic paralysis	2
Total	61 cases

* DKA = diabetic ketoacidosis

TTKG can only be interpreted correctly when urine osmolality is higher than serum osmolality[16]. In this case, TTKG could be interpreted in 52 out of 61 patients. As expected in every urine index, the urine excretion of potassium in the renal K loss group was higher than that of the non-renal K loss group ($p < 0.05$) (Table 3).

Scatter plots were used for data visualization to explore the cutoff values for each diagnostic measure compared with final classification (Fig. 1A). Of 61 patients, fifty-five (90.2%) had $24U_K \geq 20$ mEq/day. The other six (9.8%) had $24U_K < 20$mEq/day (Fig. 1A). In the group in which $24U_K \geq 20$ mEq/day, 50 out of 55 (90.9%) patients had a final classification of hypokalemia from renal K losses. The other five (9.1%) were classified as non-renal K losses. All of the latter were in the normalized 24-hr serum K group. The sensitivity and specificity of 24 $U_K \geq 20$ mEq/day to indicate renal K losses were 100% and 54.5%, respectively (Table 3).

The best diagnostic cutoff value for U_K/hr in the present study was 0.9 mEq/hr, from the optimal point of the ROC curve (Fig. 2B). Of 61 patients, 51 (83.6%) had $U_K/hr \geq 0.9$ mEq/hr. The other 10 (16.4%) had $U_K/hr < 0.9$ mEq/hr (Fig. 1B). In the group in which $U_K/hr \geq 0.9$ mEq/hr, 48 out of 51 (94.1%) patients had a final classification of renal K losses. The other three (5.9%) were classified as non-renal K losses. All of the latter were in the normalized 8-hr serum K group. The sensitivity and specificity of this new index was 96% and 72.7%, respectively. A comparison of the areas under the ROC curve between $24U_K$ and U_K/hr are shown in Fig. 2. U_K/hr tended to have higher AUC than $24U_K$ (0.86 vs. 0.84). In the subgroup of patients with normalized 24-hr serum K, the authors identified the reduction of $24U_K$ specificity from total to subgroup, 54.5% to 44.4%, with the increase of U_K/hr specificity from 72.7 to 77.8% (Table 3, 4).

Table 3. Sensitivities and specificities of various urine indices in predicting renal K losses in all patients

Index	Diagnosis (mean ± SEM)		p-value	Cutoff value	Reference	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
	Renal (R)	Non-renal (NR)							
$24U_K$ (mEq/day) R:NR = 50:11	54.0 ± 4.0	26.6 ± 7.0	0.003	20	Narins et al. 1982	100	54.5	90.9	100
U_K/hr (mEq/hr) R:NR = 50:11	3.5 ± 0.3	1.3 ± 0.6	0.007	0.9	This study	96	72.7	94.1	80
TTKG *R:NR = 43:9	6.8 ± 0.8	2.7 ± 0.5	0.001	2.5	Ethier et al. 1990	95.3	44.4	89.1	66.7
FE_K(%) **R:NR = 49:11	22.4 ± 2.5	11.4 ± 4.7	0.03	6.5	Elisaf and Siamopoulos 1995	93.9	63.6	92	70
$U_{K/Cr}$ (mmol/mmol) **R:NR = 49:11	9.1 ± 1.4	3.4 ± 1.1	0.02	2.5	Lin et al. 2004	85.7	54.5	89.4	46.2
Spot U_K (mEq/L) R:NR = 50:11	27.5 ± 2.3	13.2 ± 2.3	0.001	20	Halperin and Kamel 1998	62	81.8	93.9	32.1

$24U_K$ = 24 hour urine potassium; U_K/hr = urine potassium per hour; TTKG = transtubular potassium concentration gradient; FE_K = fractional excretion of potassium; $U_{K/Cr}$ = urine potassium-creatinine ratio; Spot U_K = spot urine potassium; PPV = positive predictive value; NPV = negative predictive value; SEM = standard error of mean; R = renal; NR = non-renal

* Interpreted only when urine osmolality is higher than serum osmolality

** Missing one value of serum creatinine

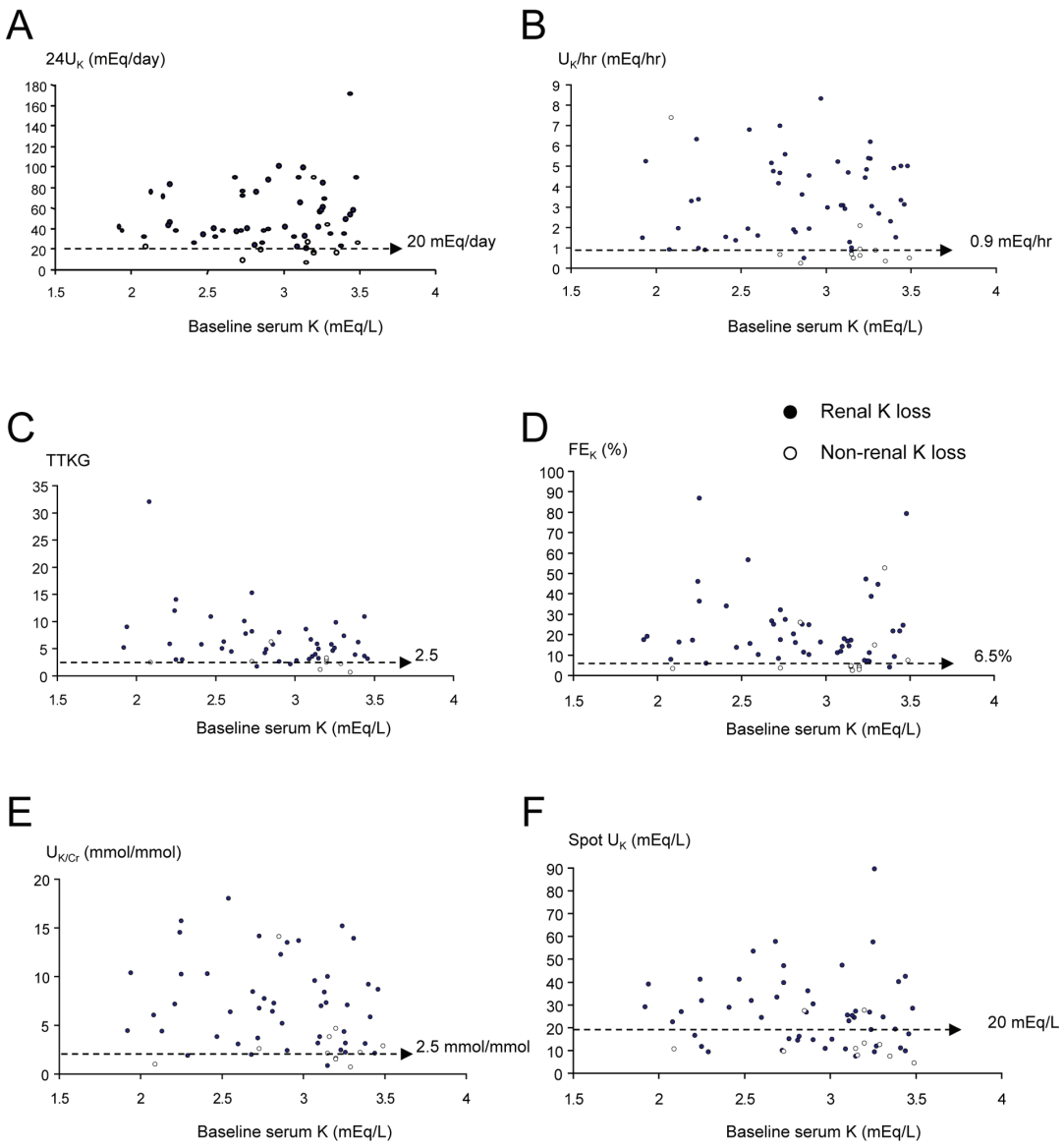

Fig. 1 Demonstrates the urine indices and baseline serum potassium level in all patients
(A) 24 hour urine potassium ($24U_K$), (B) urine potassium per hour (U_K/hr), (C) Transtubular potassium concentration gradient (TTKG), (D) Fractional excretion of potassium (FE_K), (E) Urine potassium-creatinine ratio ($U_{K/Cr}$) and (F) Spot urine potassium (spot U_K). The dash line indicates the cutoff value (from reference studies) to discriminate renal from non-renal potassuim losses for each urine index
● = Renal K loss, o = Non-renal K loss

In another view, of hypokalemic patients who had a final classification of non-renal K losses, only 6 out of 11 (54.5%) patients had $24U_K$ < 20 mEq/day whereas 8 out of 11 (72.7%) had U_K/hr < 0.9 mEq/hr. Two patients diagnosed as cellular K shift (thyrotoxic periodic paralysis) were misdiagnosed by using $24U_K$ and U_K/hr. Moreover, TTKG and spot U_K were unreliable for diagnosis of cellular K shift. Only $U_{K/Cr}$ and FE_K could be used to identify these two patients correctly.

For other indices, TTKG, FE_K, $U_{K/Cr}$, and spot U_K were evaluated for their accuracy by using the previously mentioned cutoff point referred to in many studies (Table 3).

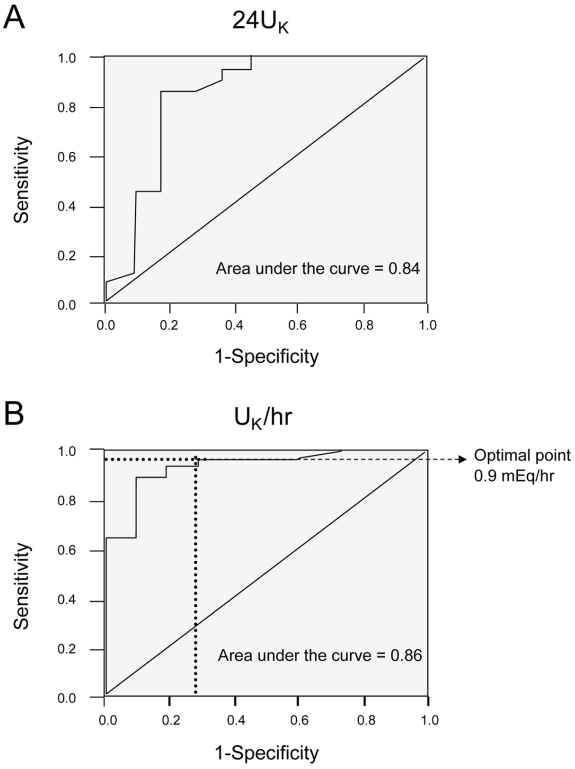

Fig. 2 Demonstrates receiver-operating characteristic (ROC) curve and area under the curve. (A) 24 hour urine potassium ($24U_K$), (B) urine potassium per hour (U_K/hr). The dash line indicates the optimal cutoff value to discriminate renal from non-renal potassium losses, (cutoff value = 0.9 mmol/hr)

To identify the best cutoff value for each urine index in the present study, the optimal point of the ROC curves were plotted. The best cutoff values for 24 U_K, TTKG, FE_K, $U_{K/Cr}$ and spot U_K were 23 mEq/day, 2.7, 5.5%, 3.0 mmol/mmol, and 10.5 mEq/L, respectively. With the new cutoff values, the new sensitivity and specificity of each urine index were 94%, 93%, 95.9%, 85.7% and 90%, respectively and 63.6%, 66.7%, 63.6%, 72.7% and 45.6%, respectively (data not shown).

Discussion

Hypokalemia is a common clinical problem arising from diverse etiologies[19]. Even mild or moderate hypokalemia increases the risks of morbidity and mortality in patients especially with cardiovascular diseases[2]. As a result, when hypokalemia is identified, the underlying cause should be sought and treated. The traditional approach to distinguish between renal and non-renal causes of hypokalemia is based on a 24-hour urine potassium[9,12,14]. In the setting of hypokalemia, a urine potassium that is more than 20 mEq/day suggests that there is renal cause of potassium wasting[9,12,14]. Levels below these values indicate that there are non-renal causes for potassium depletion. However, obtaining the 24 hr urine potassium is sometimes difficult in clinical practice because it takes too long for urine collection. Most important, therapy with potassium must be given promptly in most situations[15,20]. Hence, serum potassium during 24-hour urine collection might not be persistently low because of potassium supplementation. This situation makes the interpretation unclear. To avoid this limitation, the authors proposed a new index that assessed urine potassium for a period of time, not only one time point like TTKG[16], FE_K[17], $U_{K/Cr}$[18], or spot U_K[13] and more practical than the 24 hour urine collection[12]. In the present study, the authors evaluated the roles of U_K/hr in the first eight hours to discriminate renal from non-renal K losses compared with other urine indices.

Ideally, a 24-hr urine potassium collection should be performed without any potassium supplement. In the reality, this strategy may not be safe for the patients since the complications of

Table 4. Sensitivities and specificities of $24U_K$ and U_K/hr in predicting renal K losses in subgroup of normalized 24-hr serum K patients

Index	Diagnosis (mean ± SEM)		p-value	Cutoff value	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
	Renal (n = 22)	Non-renal (n = 9)						
$24U_K$ (mEq/day)	53.7 ± 7.4	28.9 ± 8.5	0.039	20	100	44.4	81.5	100
U_K/hr (mEq/hr)	3.3 ± 0.4	1.5 ± 0.8	0.049	0.9	95.5	77.8	91.3	87.5

$24U_K$ = 24 hour urine potassium, U_K/hr = urine potassium per hour
PPV= positive predictive value, NPV= negative predictive value, SEM = standard error of mean

hypokalemia are sometimes severe and sometimes life threatening[2]. Thus, evaluation of hypokalemia during treatment is essential in clinical practice. From the data shown in Table 3, 24 U_K and U_K/hr were the markers that had high sensitivity and specificity in predicting renal potassium losses in the present study. Regarding their accuracies in predicting renal K losses, U_K/hr > 0.9 mEq/hr can indicate renal K losses with a sensitivity of 96% and a specificity of 72.7% whereas $24U_K \geq 20$ mEq/day had a sensitivity and specificity of 100% and 54.5%, respectively. A low specificity of $24U_K$ may result from serum K levels that were not persistently low during the 24-hr urine collection. Only 30 out of 61 (49.2%) of 24-hr urine collections met the criteria for non-normalized serum K (serum K persistently < 4 mEq/L throughout 24 hours) whereas 50 out of 61 (82%) of 8-hr urine collection met the criteria for non-normalized serum K (serum K persistently < 4 mEq/L throughout the first 8 hours). This data indicated the chance that the authors would collect a complete $24U_K$ during a persistently low serum potassium period and the chance that the authors could reliably interpret these data was generally less than a half. In contrast with $24U_K$, the shorter duration in urine collection of U_K/hr resulted in a greater number of patients who met the criteria for non-normalized serum K and made the data more reliable for interpretation. Interestingly, U_K/hr is a new index that is much more practical, convenient and takes less time than the conventional 24-hr urine collection. This marker not only had comparable sensitivity, but also had a higher specificity than $24U_K$ especially in cases that serum K was not persistently low throughout the 24-hr urine collection.

Based on Ethier J et al[16], TTKG is a simple index to evaluate renal K losses. However, it still has some limitations. One must rely on another index (osmolality) to calculate the TTKG. Moreover, TTKG is invalid if the urine osmolality is lower than the plasma osmolality. In the present study, the authors could use only 85% of TTKG values because nine patients had the urine osmolality lower than the plasma osmolality. When compared with U_K/hr, the number of patients that can be interpreted by U_K/hr was more than those by TTKG and U_K/hr was more reliable with a higher specificity in predicting renal K losses.

For FE_K, $U_{K/Cr}$, and spot urine K, although these noninvasive parameters are simple to use, they are less accurate compared with $24U_K$ or U_K/hr, because the former parameters assess potassium excretion only at one time point. In the present study, although the authors used the best cutoff value to determine the best sensitivity and best specificity of each marker, FE_K, $U_{K/Cr}$, and spot U_K still had low sensitivities, specificities and area under the curve of ROC when compared with U_K/hr.

U_K/hr had a limitation in diagnosis of patients who had hypokalemia from thyrotoxic periodic paralysis. From the authors' previous study[21], it was found that nine out of 11 of the authors thyrotoxic periodic paralysis patients recovered from hypokalemia within 24 hours. Six out of 11 patients recovered from hypokalemia within eight hours. The authors speculated that both a rapid change of transcellular potassium from hypokalemia to normokalemia or even hyperkalemia and potassium supplementation from treatment might increase the urine potassium excretion and, thus, made the test inaccurate. In TPP patients, the authors suggest that the clinical setting and awareness of the disease are important for the definite diagnosis.

In conclusion, the present study demonstrated that U_K/hr is a new practical, simple and reliable marker that can be applied to evaluate renal K losses in hypokalemic patients during treatment with comparable sensitivity and specificity with $24U_K$.

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Potential conflicts of interest

None.

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การใช้ปริมาณโปแทสเซียมในปัสสาวะต่อชั่วโมงเป็นดัชนีในการบ่งบอกภาวะการสูญเสียโปแทสเซียมทางไต

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ภูมิหลัง: *Hypokalemia เป็นภาวะที่ระดับความเข้มข้นของโปแทสเซียมในเลือดต่ำกว่า 3.5 mEq/L ซึ่งเป็นภาวะที่อันตรายและพบได้บ่อยในเวชปฏิบัติทั่วไป วิธีมาตรฐานในการวินิจฉัยภาวะการสูญเสียโปแทสเซียมทางไต คือ การตรวจพบปริมาณโปแทสเซียมในปัสสาวะตลอด 24 ชั่วโมง ($24U_K$) ≥ 20 mEq/day ในขณะที่ผู้ป่วยอยู่ในภาวะ hypokalemia ตลอดเวลา การให้โปแทสเซียมทดแทนในทันทีนั้นมักจะมีความจำเป็น เพื่อป้องกันภาวะแทรกซ้อนต่าง ๆ ที่อาจจะเกิดขึ้น แต่ก็มักจะทำให้ระดับความเข้มข้นของโปแทสเซียมในเลือดกลับสู่ระดับปรกติในระหว่างการเก็บ $24U_k$ และทำให้ไม่อาจจะแปลผลค่า $24U_k$ ที่เก็บได้*

วัสดุและวิธีการ: *คณะผู้นิพนธ์ศึกษาความสามารถของดัชนีต่างๆของปัสสาวะได้แก่ $24U_k$, transtubular potassium gradient (TTKG), fractional excretion of potassium (FE_k), urine potassium-creatinine ratio ($U_{k/cr}$) และ spot U_k พร้อมกับแนะนำดัชนีตัวใหม่คือการวัดปริมาณ urine potassium per hour ในระหว่าง 8 ชั่วโมงแรก (U_k/hr) เพื่อใช้เป็นดัชนีในการประเมินภาวะ hypokalemia ในระหว่างการรักษาการตรวจพบระดับโปแทสเซียมในเลือดที่ ≥ 4 mEq/L ในระหว่างการเก็บปัสสาวะให้ถือว่าระดับโปแทสเซียมได้ถูกแก้ไขเข้าสู่ภาวะปรกติแล้ว ในการศึกษานี้ถ้า $24U_k$ ≥ 20 mEq/วัน ในกลุ่มที่ระดับโปแทสเซียมในเลือดไม่ได้ถูกแก้ไขเข้าสู่ภาวะปรกติภายใน 24 ชั่วโมง จะถูกจัดกลุ่มขั้นสุดท้ายว่าเป็น hypokalemia จาก renal loss ในกรณีของกลุ่มที่ระดับโปแทสเซียมในเลือดได้ถูกแก้ไขเข้าสู่ภาวะปรกติภายใน 24 ชั่วโมง การจัดกลุ่มขั้นสุดท้ายจะขึ้นกับผลลัพธ์ส่วนใหญ่ของดัชนีทางปัสสาวะ 4 ตัว ได้แก่ TTKG, FE_k, $U_{k/cr}$, และ spot U_k*

ผลการศึกษา: *ในผู้ป่วย 61 คน พบว่าเป็น hypokalemia จาก renal:non-renal = 50:11 ในจำนวนนี้พบว่า 51% ของผู้ป่วยมีระดับโปแทสเซียมในเลือดกลับเข้าสู่ระดับปรกติในเวลา 24 ชั่วโมง ในขณะที่ 18% ของผู้ป่วยมีระดับโปแทสเซียมกลับเข้าสู่ระดับปรกติในในเวลา 8 ชั่วโมง โดยภาพรวม U_k/hr ที่ ≥ 0.9 สามารถบอกภาวะการสูญเสียโปแทสเซียมทางไตได้ด้วยความไว 96% และความจำเพาะ 72.7% เมื่อเทียบกับ $24U_k$ ที่ ≥ 20 mEq/วัน สามารถบอกได้ถูกต้องด้วยความไว 100% และความจำเพาะ 54.5% ตามลำดับ ในกลุ่มย่อยที่ระดับโปแทสเซียมในเลือดได้กลับสู่ระดับปรกติใน 24 ชั่วโมง พบว่าความไวและความจำเพาะของ U_k/hr = 95.5% และ 77.8% ตามลำดับ ในขณะที่ความไวและความจำเพาะของ $24U_k$ = 100% และ 44.4% ตามลำดับ*

สรุป: *U_k/hr เป็นดัชนีตัวใหม่ที่สะดวกใช้ง่าย และเชื่อถือได้ในการประเมินภาวะ hypokalemia ในผู้ป่วยระหว่างการรักษา และมีความไวและความจำเพาะใกล้เคียงกับ $24U_k$*