The Effects of Potassium and Magnesium Supplementations on Urinary Risk Factors of Renal Stone Patients

SUDJAI JAIPAKDEE, MSc*,
VITOON PRASONGWATANA, MSc, MD*,
AMORN PREMGAMONE, MD, MPH**,
SIRIRAT REUNGJUI, MD***,
PIYARATANA TOSUKHOWONG, MSc****,
KRIANG TUNGSANGA, MD****,
SUNTHON SUWANTRAI, BSc*
CHEDSADA NOPPAWINYOOWONG, B PHARM, MBA*****,
SRINOI MASKASAME, DVM, MPH, MSPD**,
POTE SRIBOONLUE, MSc, MCH*

Abstract

The effects of potassium and magnesium supplementation on urinary risk factors for renal stone disease were studied in 61 renal stone patients. The subjects were divided into four groups and supplemented for a period of one month with potassium chloride (KCl, Group 1), potassium sodium citrate (K Na citrate, Group 2), magnesium glycine (Mg glycine, Group 3) and potassium magnesium citrate (K Mg citrate, Group 4) with a daily dose of 42 mEq potassium, 21 mEq magnesium or sodium and 63 mEq citrate, accordingly. The results showed that serum potassium and magnesium of all four groups normalized after the supplementation. Though urinary potassium significantly increased in all three groups supplemented with elemental potassium containing solutions [i.e. KCl ($p < 0.001$), K Na citrate ($p < 0.001$) and K Mg citrate ($p < 0.001$)] only K Na citrate and K Mg citrate, caused a significant increase in urinary pH and citrate but decrease in calcium. Supplementation with Mg glycine in Group 3 although caused a significant increase in urinary magnesium, its effects on urinary pH, citrate and calcium, however, were similar to KCl, in that they caused a significant decrease in urinary pH without any change in urinary citrate or calcium. Supplementation with K Mg citrate in Group 4 seems to have given the best results, as far as lowering stone risk factors in that it caused an increase in urinary pH, potassium and citrate and decreased calcium excretions similar to K Na citrate in Group 2. In addition, K Mg citrate also caused the enrichment of urine with magnesium, another inhibitor of calcium-containing stones. Although the four supplements had no effect on urinary saturation of calcium oxalate salt, their effects on the saturations of brushite ($CaHPO_4 \cdot 2H_2O$), octacalcium phosphate ($Ca_8H_2(PO_4)_6 \cdot 5H_2O$) and uric acid were clearly associated with changes in urinary pH. Therefore, in Group 1 and 3, subjects having a decrease in urinary pH, also experienced a significant increase in uric acid saturation. Though the saturation of brushite and octacalcium phosphate in Group 2 and 4 and the sodium acid urate in Group 2 were significantly increased, these urinary risk factors could be

overcome, however, by the concomitant increase in urinary citrate. The present results demonstrate that for those stone vulnerable subjects having a high risk of potassium and magnesium depletion, to obtain the best therapeutic results, they should be provided supplementations of both potassium and magnesium together and also in the forms that would result in the delivery of an alkali loading effect.

Key word : Potassium Magnesion Citrate Supplementation, Renal Stone, Stone Risk Factors

JAIPAKDEE S,
PRASONGWATANA V, PREMGAMONE A, et al
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* Department of Biochemistry,
** Department of Community Medicine,
*** Department of Internal Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002,
**** Department of Medicine and Biochemistry, Faculty of Medicine, Chulalongkorn University, Bangkok 10330,
***** Manufacturing Pharmacy Unit, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand.

The low status of potassium and magnesium among renal stone patients residing in rural Northeast Thailand is well documented(1-4). The main metabolic abnormalities include hypocitraturia associated with hypokaliuria and hypokalemia(1). Though urinary excretion of calcium among these patients is within the normal range, calcium loading reveals that most of them (60%) could be classified as having absorptive hypercalciuria(5). An increased excretion of calcium is an important risk for calcium stone formation, but with the low excretion of citrate, an important calcium stone inhibitor, the combination of conditions would increase the risk of forming stones. Furthermore, it has been shown that supplementation with potassium not only corrected the low potassium status, but also caused a decrease in calcium excretion with a concomitantly increase urinary citrate(6-9).

Metabolism and regulation of potassium and magnesium are interrelated: subjects at risk of being potassium depleted should be checked for magnesium depletion(10-12). It has been demonstrated that in the event of a combined reduction of tissue potassium and magnesium, supplementation with extra potassium alone, tissue potassium is not restored(11). However, magnesium supplementation alone does lead to restoration of both magnesium and potassium levels(12). Magnesium deficiency theoretically increase risk of calcium stone forming because magnesium itself forms a solute complex with oxalate and lower calcium oxalate salt saturation(13-15). It has also been demonstrated that magnesium may be involved in citrate metabolism as it causes an increase in urinary excretion of citrate after the administration of magnesium salts(14,15).

Due to the widespread low potassium and magnesium status among these stone subjects and their main metabolic defects are hypocitraturia and probably absorptive hypercalciuria, the authors consider the benefits of treatment with potassium and magnesium supplementation. It has been demonstrated that in supplementing with different forms of potassium and magnesium, though aiming to receive the same amount of elemental potassium or magnesium for each subject, it could bring about different results (8,15,16). So, the authors compared the results of supplementations with four different salts of potassium and magnesium on urinary risk factors for stone disease.

MATERIAL AND METHOD

Experimental subjects

Studies were conducted in an outpatient setting with 61 rural subjects who had renal stones as detected using a mobile ultrasound unit. The authors tested 34 males and 27 females between the ages of 35 and 65 with normal renal function (serum creatinine

< 2.3 mg/dl). We excluded the patients with chronic medical illnesses, previous urinary tract surgery or renal insufficiency from this study. A urinalysis of the first morning urine specimen with urine strip (Multistix 10 SG, Bayer Corporation, USA) yielded normal results for all subjects. Those who showed results of trace or +1 leucocytes were first treated with a full dose of antibiotics for a period of about one month before inclusion in the study. This research project was approved by the Ethical Committee of the Faculty of Medicine, Khon Kaen University and informed consent was obtained from each participant.

Study protocol

The subjects were randomly allocated into four groups according to the types of treatment. Group 1 (n = 16), Group 2 (n = 15), Group 3 (n = 16) and Group 4 (n = 14) were supplemented for a period of one month with potassium chloride (KCl), potassium-sodium citrate (K Na citrate), magnesium glycine (Mg glycine) and potassium-magnesium citrate (K Mg citrate), respectively. Each subject received a daily dose of 42 mEq potassium for Group 1, of 42 mEq potassium, 21 mEq sodium and 63 mEq citrate for Group 2, of 21 mEq magnesium for Group 3 and of 42 mEq potassium, 21 mEq magnesium and 63 mEq citrate for Group 4. All other medications were withdrawn during the study period. Subjects were allowed to eat freely and to perform their daily activities during the supplementation except that, each morning and each evening they had to go for their supplements given by the village health volunteers at the health centers located near their home. Two 24-hour urine samples and a clotted blood sample were collected from each subject, both before and after supplementation. Urine samples were preserved during collection with Thymol. The collection time for some females was adjusted to avoid their menstrual cycles.

Analysis of serum and urine

Serum was analyzed for creatinine by the modified Jaffe's method, electrolytes by an autoanalyzer and magnesium by atomic absorption spectrophotometry. After the measurement for volume and pH, urine samples were analyzed for sodium and potassium by flame photometry, calcium and magnesium by atomic absorption spectrophotometry, phosphate by the technique of Fiske and Subbarow[17], creatinine by modified Jaffe's method and uric acid by the uricase technique. Citrate was analyzed using an enzyme kit from Boehringer-Mannheim Company. An indirect method as described by Sriboonlue et al was used to estimate urinary oxalate concentration[18].

Estimation of urine saturation

Nomogram methods as described by Marshall and Robertson were used to estimate the levels of urine saturation with respect to calcium oxalate, calcium phosphate [brushite ($CaHPO_4 \cdot 2H_2O$) and octacalcium phosphate ($Ca_8H_2(PO_4)_6 \cdot 5H_2O$)], uric acid and sodium acid urate[19]. The saturation of each salt in the urine was expressed as an absolute value of-$\log_{10}$ (activity product).

RESULTS

Demographic characteristics

Demographic data of the participating subjects are summarized in Table 1. The male to female ratio among the four groups was unequal, Group 1 (4 : 12), Group 2 (7 : 8), Group 3 (14 : 2) and Group 4 (9 : 5). All other data including age, body weight, height and body mass index (BMI), however, were balanced.

Baseline serum and urine chemistries and urinary saturation levels of stone-forming constituents

Table 2 shows biochemical compositions of serum and 24-h urine as well as the saturation of

Table 1. Clinical data of the four groups study. Group 1 supplemented with KCl, Group 2 with K Na citrate, Group 3 with Mg glycine and Group 4 with K Mg citrate.

	Group 1	Group 2	Group 3	Group 4
No. of subjects	16	15	16	14
Male : female	4 : 12	7 : 8	14 : 2	9 : 5
Age (year)	51.50 ± 10.13	49.93 ± 7.75	48.14 ± 8.92	51.64 ± 9.15
Body weight (kg)	59.81 ± 9.14	62.27 ± 8.96	59.31 ± 10.32	60.29 ± 6.17
Height (m)	1.62 ± 5.78	1.65 ± 9.48	1.65 ± 2.22	1.65 ± 5.92
BMI	22.90 ± 3.34	22.89 ± 3.39	22.86 ± 3.57	22.01 ± 1.61

urine with the five stone-forming constituents, i.e., calcium oxalate, brushite, octacalcium phosphate, uric acid and sodium acid urate of the four studied groups before supplementations. Though some parameters determined were lower than those reported in Western countries, particularly for urinary levels of potassium, magnesium and citrate (see also Table 4), they were similar to the authors' previous reports[1-4]. In comparison, among the four groups, the mean values of all parameters were similar.

Effects of supplementation

Effects on serum and urine composition

The effects of the four supplemental regimens on serum and urine composition are shown in Table 3 and on metabolic abnormalities in Table 4. Though the four supplements gave different changes in a number of serum parameters (Table 3), the hypokalemia in all four groups and hypomagnesemia in Group 4 (Table 4) were ultimately resolved. It is interesting to note the significant increase in serum potassium ($p < 0.001$) in Group 3 supplemented with Mg glycine. Taking into consideration the five urinary parameters related to the supplements and stone risk factors (i.e. pH, potassium, magnesium, calcium and citrate) supplementation with KCl (Group 1) caused a significant decrease in pH ($p < 0.001$) but an increase in potassium ($p < 0.001$). By contrast, supplementation with K Na citrate (Group 2), resulted in significant changes in the four values: increases in pH ($p < 0.001$), potassium (< 0.001) and citrate ($p < 0.001$) and a decrease in calcium ($p < 0.001$). Though as expected Mg glycine caused a significant increase in magnesium ($p < 0.022$), it also caused a significant decrease in the pH ($p < 0.001$) of Group 3 subjects. Supplementation with K Mg citrate, on the other hand, gives the best results in that it rise to beneficial changes in all interested parameters; increases in pH ($p < 0.004$), potassium ($p < 0.001$), magnesium ($p < 0.005$) and citrate ($p < 0.001$) and a decrease in calcium ($p < 0.005$).

Effects on urinary saturation

Saturation of calcium oxalate salt in the urine did not significantly alter in any of the four supplementation regimens, even in subjects from Groups 3

Table 2. Baseline serum and 24-hour urine composition and saturation levels of urine of the four groups before supplementation with KCl (Group 1), K Na citrate (Group 2), Mg glycine (Group 3) and K Mg citrate (Group 4).

	Group 1	Group 2	Group 3	Group 4
Serum				
Creatinine (mg/dl)	1.06 ± 0.20	1.15 ± 0.18	0.96 ± 0.16	0.95 ± 0.16
Potassium (mmol/L)	4.55 ± 0.34	4.40 ± 0.3	4.50 ± 0.42	4.27 ± 0.37
Sodium (mmol/L)	140 ± 1.6	139 ± 1.9	140 ± 1.9	140 ± 2.2
Magnesium (mg/dl)	0.89 ± 0.07	0.84 ± 0.09	0.81 ± 0.07	0.83 ± 0.10
Bicarbonate (mmol/L)	20.5 ± 1.5	18.3 ± 2.2	17.8 ± 1.2	17.6 ± 1.7
Chloride (mmol/L)	107 ± 2.1	105 ± 3.1	102 ± 2.1	106 ± 2.5
24-hour urine				
Volume (ml)	1,517 ± 666	1,436 ± 691	1,460 ± 174	1,426 ± 148
pH	5.79 ± 0.50	5.46 ± 0.36	5.84 ± 0.61	5.81 ± 0.35
Sodium (mmol/d)	78.7 ± 45.9	78.9 ± 39.2	90.6 ± 35.0	72.4 ± 25.5
Potassium (mmol/d)	22.7 ± 12.6	22.4 ± 9.7	19.6 ± 10.0	19.8 ± 4.1
Magnesium (mmol/d)	2.69 ± 0.78	3.00 ± 1.1	2.68 ± 0.92	2.09 ± 0.46
Calcium (mmol/d)	3.54 ± 1.4	3.70 ± 1.5	3.04 ± 1.7	3.45 ± 1.2
Phosphate (mmol/d)	13.0 ± 3.6	16.5 ± 5.2	18.7 ± 10.3	15.8 ± 4.0
Oxalate (mg/d)	24.4 ± 9.6	20.4 ± 7.7	27.0 ± 8.8	19.5 ± 6.4
Uric acid (mmol/d)	2.53 ± 0.66	2.21 ± 0.75	2.75 ± 0.99	2.49 ± 0.69
Citrate (mmol/d)	1.65 ± 1.3	1.47 ± 1.1	1.44 ± 0.97	1.62 ± 0.86
Creatinine (g/d)	0.83 ± 0.2	0.95 ± 0.5	0.83 ± 0.29	0.74 ± 0.15
Urine saturation ($-\log_{10}$ activity product)				
Calcium oxalate	7.55 ± 0.23	7.60 ± 0.27	7.55 ± 0.25	7.55 ± 0.11
Brushite	6.76 ± 0.52	6.98 ± 0.55	6.77 ± 0.62	6.57 ± 0.33
Octacalcium phosphate	49.7 ± 2.4	50.7 ± 2.6	49.7 ± 2.6	48.5 ± 1.3
Uric acid	8.82 ± 0.40	8.68 ± 0.17	8.87 ± 0.48	8.85 ± 0.32
Sodium acid urate	4.34 ± 0.59	4.43 ± 0.38	4.19 ± 0.31	4.23 ± 0.30

Table 3. Statistical analysis of changes in serum and 24-hour urine composition and in levels of urine saturation after supplementation with KCl (Group 1), K Na citrate (Group 2), Mg glycine (Group 3), and K Mg citrate (Group 4).

	Group 1	Group 2	Group 3	Group 4
Serum				
Creatinine (mg/dl)	↑*	NS	NS	NS
Potassium (mmol/L)	↑***	↑***	↑***	NS
Sodium (mmol/L)	NS	NS	NS	NS
Magnesium (mg/dl)	↓**	NS	NS	NS
Bicarbonate (mmol/L)	↓***	↓***	NS	↑***
Chloride (mmol/L)	↓***	NS	↑*	↓**
24-hour urine				
Volume (ml)	NS	NS	NS	NS
pH	↓***	↑***	↓***	↑**
Sodium (mmol/d)	NS	NS	NS	NS
Potassium (mmol/d)	↑***	↑***	NS	↑***
Magnesium (mmol/d)	NS	NS	↑*	↑**
Calcium (mmol/d)	NS	↓***	NS	↓**
Phosphate (mmol/d)	↑*	NS	NS	NS
Oxalate (mg/d)	NS	NS	NS	NS
Uric acid (mmol/d)	NS	↑*	NS	NS
Citrate (mmol/d)	NS	↑***	NS	↑***
Creatinine (g/d)	↑**	↑*	NS	NS
Urine saturation				
Calcium oxalate	NS	NS	NS	NS
Brushite	↓***	↑**	↓*	↑**
Octacalcium phosphate	↓***	↑**	↓**	↑**
Uric acid	↑*	↓**	↑**	↓**
Sodium acid urate	NS	↑**	↓*	NS

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, ↑ increase, ↓ decrease

and 4 with a significant decrease in urinary-Ca concentration. This is probably due to the unchanged urinary excretion of oxalate, the counterpart ion (Table 2). Urinary saturation with respect to brushite and octacalcium phosphate significantly decreased in Groups 1 ($p < 0.001$ for both) and 3 ($p < 0.05$ for brushite, $p < 0.004$ for octacalcium phosphate), and significantly increased in Groups 2 ($p < 0.01$ for brushite, $p < 0.004$ for octacalcium phosphate) and 4 ($p < 0.01$ for both) after the supplementation. These changes seemed to parallel the changes in urinary pH, where it decreased in Groups 1 and 3 and increased in Groups 2 and 4. The urinary saturation of uric acid also exhibited an association with the change in urinary pH where it increased when the pH decreased in Groups 1 ($p < 0.05$) and 3 ($p < 0.008$), and decreased when the pH increased in Groups 2 ($p < 0.006$) and 4 ($p < 0.004$). Saturation of sodium urate salt increased only in the urine of Group 2 subjects supplemented with K Na citrate ($p < 0.01$).

DISCUSSION

Potassium deficiency has been shown to be a cause of low urinary citrate excretion[9]. In supplementation with KCl in stone patients with normal potassium status, Sakhaee et al demonstrated an increase in urinary excretions of only potassium but not of citrate[8]. Though the presented stone subjects were in a state of potassium depletion, KCl supplements did not result in an increase in urinary citrate. The results confirm the authors' previous observation where the administration of KCl for a week caused a significant increase in both serum and urinary potassium but not urinary citrate (1).

A study in experimental rats, showed both potassium deficiency and chronic metabolic acidosis caused an increase in citrate oxidation in the renal tubular cells resulting in an increased tubular citrate reabsorption and hypocitraturia[20,21]. Although chronic potassium deficiency is a cause of intracellular acidosis[22], potassium administration in the form

Table 4. Prevalence of metabolic abnormalities before and after supplementations with KCl (Group 1), K Na citrate (Group 2), Mg glycine (Group 3), and K Mg citrate (Group 4), before supplementation (B) and after supplementation (A).

	Group 1		Group 2		Group 3		Group 4	
	B (%)	A (%)	B (%)	A (%)	B (%)	A (%)	B (%)	A (%)
Hypokalemia (< 3.5 mmol/L)	6.3	0	13.3	0	12.5	0	14.3	0
Hypomagnesemia(< 0.65 mmol/L)	0	0	0	0	0	0	7.1	0
Hypokaliuria (< 30 mmol/day)	81.3	0	93.3	13.3	93.8	100	100	7.1
Hypomagnesiuria (< 3 mmol/day)	62.5	43.8	53.3	73.3	68.8	18.8	100	50
Hypocitraturia (< 1 mmol or 200 mg/day)	31.3	18.8	46.7	13.3	37.5	31.3	21.4	0

of KCl does improve potassium status but probably does not correct the acidosis. Furthermore, KCl may aggravate the condition due to the accompanying chloride load. This was clearly seen when potassium was supplemented as alkali in the forms of K citrate [7,8] or K Na citrate in the present study where serum and urine potassium, urinary pH and citrate excretion were significantly increased. The citraturic response is largely dependent on the systemic alkali load introduced, which impairs the renal tubular citrate reabsorption[7,8]. It has been shown that the correction of intracellular acidosis is attributable to *in vivo* oxidation of absorbed citrate[20], which was partly exhibited in the significant increase in urinary pH in the present study.

It is well recognized that depletions of potassium and magnesium are always coexistent[10-12]. From the results of muscle specimen analysis, stone patients in Northeast Thailand may be deficient in both potassium and magnesium[3]. This was partly confirmed by the high prevalence of hypokaliuria and hypomagnesiuria in the subjects of all four groups. In Group 3 stone subjects, after the administration of Mg glycine, the status of magnesium clearly improved as seen from the increase in urinary excretion of magnesium. However, elemental magnesium alone, as potassium, seemed to have little effect on citraturic response. The explanation may be due to preexisting intracellular acidosis not yet corrected as in the case of KCl administration. To correct the coexisting depletions of potassium and magnesium, whatever the causes, and to raise urinary pH and citrate excretion concomitantly, Koenig et al, recently introduced a new compound, K Mg citrate[16]. It has been demonstrated that K Mg citrate could prevent not only the recurrence of uric acid stone similar to K citrate, but it could prevent the recurrence of hypocitraturic calcium nephrolithiasis more effectively due to the concurrent increase in both magnesium and citrate excretion[16,23].

Increase potassium excretion has been demonstrated to cause the reduction in urinary calcium concentration, a risk factor for calcium stone formation (6-8). Though Group 1 subjects could successfully increase their urinary potassium, it had no effect on urinary calcium. This finding supports the previous observation made by Sakhaee et al[8]. In the case of Groups 2 and 4, the increase in urinary potassium was well accompanied with a decrease in urinary calcium. This indirectly suggests that intracellular acidosis not only caused the increase in citrate oxidation, but also prevented tubular reabsorption of calcium.

Though the urinary calcium concentration significantly decreased after supplementation with K Na citrate and K Mg citrate, the nomogram-estimated urine saturation indicated that they could not offer increased protection against crystallization of calcium oxalate in the urine. This is probably due to the unchanged urinary oxalate ion, where its concentration in urine is only about 1/20 of the counterpart calcium ion. Therefore, when giving rise as an ion activity product of the two ions (a measurement of saturation level) would result in a nonsignificant change as suggested by Robertson and Hughes[24]. Supplementation with KCl and Mg glycine, on the other hand, clearly increased the risk of uric acid stone development due to the significant increase in urinary saturation of uric acid from the decreased urinary pH. Though urinary saturation of brushite and octacalcium phosphate was increased from the supplementations of K Na citrate and K Mg citrate, this would be overcome by the increase in urinary excretion of citrate. Similarly, increased urinary citrate would protect against sodium acid urate crystallization in the Group 2 subjects.

In conclusion, the present results indicate when supplementing potassium and magnesium to depleted subjects, the forms of the drugs significantly affect results. Administration of potassium and magnesium should be provided with alkali loading in order to correct for both potassium and magnesium depletions and the concomitant intracellular alkalosis leads to a reduction in tubular citrate oxidation and reabsorption. Furthermore, this alkali loading effect will also increase tubular reabsorption of calcium resulting in the reduction of urinary calcium concentration.

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ผลของการเสริมแร่ธาตุโพแทสเซียมและแมกนีเซียมต่อปัจจัยเสี่ยงในปัสสาวะของผู้ป่วยโรคนิ่วไต

สุดใจ ใจภักดี, วทม, วิฑูรย์ ประสงค์วัฒนา, วทม, พบ*,*
*อมร เปรมกมล, พบ, MPH**, ศิริรัตน์ เรืองจุ้ย, พบ***,*
*ปิยะรัตน์ โตสุโขวงค์, วทม****, เกรียง ตั้งสง่า, พบ****,*
สุนทร สุวรรณไตรย์, วทบ, เจษฎา นพวิญญูวงค์, ภบ, MBA*****,*
*ศรีน้อย มาศเกษม, สพบ, สม, MSPD**, พจน์ ศรีบุญลือ, วทม, MCH**

ได้ทำการศึกษาผลของการเสริมแร่ธาตุโพแทสเซียมและแมกนีเซียมต่อปัจจัยเสี่ยงในปัสสาวะของ ผู้ป่วยโรคนิ่วไต จำนวน 61 คน ทำโดยการแบ่งผู้ป่วยออกเป็น 4 กลุ่ม แล้วเสริมเป็นระยะเวลา 1 เดือนด้วย โพแทสเซียมคลอไรด์ (กลุ่ม 1) โพแทสเซียม โซเดียมซิเทรต (กลุ่ม 2) แมกนีเซียมไกลซีน (กลุ่ม 3) และโพแทสเซียม แมกนีเซียมซิเทรต (กลุ่ม 4) โดยให้ได้รับวันละ 42 mEq สำหรับโพแทสเซียม 21 mEq สำหรับแมกนีเซียมหรือโซเดียม และ 63 mEq สำหรับซิเทรต หลังการเสริมพบว่าโพแทสเซียมและแมกนีเซียมในซีรัมของประชากรทุกกลุ่มมีค่ากลับเป็นปกติ สำหรับค่าโปตัสเซียมในปัสสาวะเพิ่มขึ้นอย่างมีนัยสำคัญ พบได้ทุกกลุ่มที่ทำการเสริมด้วยยาที่มีแร่ธาตุโพแทสเซียมเป็นองค์ประกอบ แต่พบว่าเฉพาะกลุ่มที่ได้รับเสริมด้วยยาที่มีซิเทรตเป็นองค์ประกอบ (กลุ่ม 2 และ 4) เท่านั้น ที่มีผลทำให้มีการเปลี่ยนแปลงเพิ่มขึ้นของค่า pH และเพิ่มขึ้นของค่าซิเทรต และพบค่าแคลเซียมในปัสสาวะลดลงซึ่งไม่พบในกลุ่มที่ได้รับโปตัสเซียมกับแมกนีเซียมคลอไรด์ (กลุ่มที่ 1 และ 3) การเสริมด้วยแมกนีเซียมไกลซีนในกลุ่ม 3 แม้จะทำให้ระดับแมกนีเซียมในปัสสาวะเพิ่มขึ้น ($p < 0.022$) แต่ผลต่อค่า pH ค่าซิเทรต และค่าแคลเซียมของปัสสาวะกลับคล้ายกลุ่ม 1 ที่เสริมด้วยโพแทสเซียมคลอไรด์ คือ มีผลทำให้ pH ของปัสสาวะลดลง ($p < 0.001$) โดยไม่มีผลใด ๆ ต่อการเปลี่ยนแปลงของค่าซิเทรตและแคลเซียม กลุ่มที่ได้รับโปตัสเซียมแมกนีเซียม ซิเตรท พบว่าให้ผลดีที่สุดในแง่ของผลต่อการลดปัจจัยเสี่ยงโรคนิ่วไตในปัสสาวะ คือทำให้เกิดการเพิ่มขึ้นของ pH โพแทสเซียม ซิเทรต และแมกนีเซียมในปัสสาวะ และการลดลงของแคลเซียมอย่างมีนัยสำคัญ ซึ่งแมกนีเซียมเป็นสารยับยั้งนิ่วชนิดนิ่วแคลเซียมออกซาเลต สำหรับผลต่อความอิ่มตัวของปัสสาวะถึงแม้การเสริมในทั้ง 4 กลุ่มจะไม่มีผลทำให้ระดับความอิ่มตัวของเกลือแคลเซียมออกซาเลตลดลง แต่ผลของการเสริมที่มีต่อระดับความอิ่มตัวของแคลเซียมฟอสเฟต (บรัสไซต์และอ๊อกตะแคลเซียม-ฟอสเฟต) และกรดยูริก กลับพบว่ามีความสัมพันธ์กันอย่างชัดเจนกับค่า pH ของปัสสาวะ นั่นคือกลุ่ม 1 และ 3 หลังการเสริมที่มีค่า pH ของปัสสาวะลดลงจะมีค่าความอิ่มตัวของกรดยูริกเพิ่มขึ้น ในขณะที่ความอิ่มตัวของบรัสไซต์และอ๊อกตะแคลเซียม

ฟอสเฟต ในกลุ่ม 2 และกลุ่ม 4 และของโซเดียมยูเรตในกลุ่ม 2 จะมีค่าเพิ่มขึ้นอย่างมีนัยสำคัญ เมื่อ pH ของปัสสาวะเพิ่มขึ้น อย่างไรก็ตามการเพิ่มขึ้นของปัจจัยเสี่ยงนี้ก็จะถูกชดเชยด้วยการมีค่าซิเทรตในปัสสาวะเพิ่มขึ้น ผลการศึกษาทั้งหมดแสดงให้เห็นว่ากลุ่มประชากรที่เป็นนิ่วไตที่มีความเสี่ยงสูงที่จะมีภาวะพร่องทั้งโพแทสเซียมและแมกนีเซียมนั้น เพื่อที่จะให้การรักษาได้ผลที่ดีที่สุด ควรทำการเสริมแร่ธาตุทั้งสองร่วมกันและให้อยู่ในรูปที่มีผลของการโหลดด่างร่วมด้วย

คำสำคัญ : การเสริมแร่ธาตุโพแทสเซียมและแมกนีเซียม, โรคนิ่วไต, ปัจจัยเสี่ยงโรคนิ่วไต

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* ภาควิชาชีวเคมี,
** ภาควิชาเวชศาสตร์ชุมชน,
*** ภาควิชาอายุรศาสตร์, คณะแพทยศาสตร์ มหาวิทยาลัยขอนแก่น, ขอนแก่น 40002
**** ภาควิชาชีวเคมีและภาควิชาอายุรศาสตร์, คณะแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย, กรุงเทพ ฯ 10330
***** หน่วยยาน้ำ, คณะแพทยศาสตร์ มหาวิทยาลัยขอนแก่น, ขอนแก่น 40002