The Most Important Metabolic Risk Factors in Recurrent Urinary Stone Formers

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Purpose: To evaluate different urinary factors contributing to idiopathic calcium stone disease for determining appropriate medical treatments.

Materials and Methods: Two 24-hour urine samples were collected from 106 male recurrent idiopathic calcium stone formers and another 109 randomly selected men as the control group matching for age.

Results: Cases had significantly higher mean urine oxalate, calcium, uric acid, and chloride in comparison with the healthy controls ($P < .001$). After necessary adjustment, only mean urine levels of oxalate and uric acid were higher in stone formers than those in controls. The mean value of supersaturation for calcium oxalate was significantly higher in patients than the controls ($P = .001$); whereas supersaturation for calcium phosphate and uric acid did not reach statistical significe *(*$P = .675$ and $P = .675$, respectively). Hyperoxaluria and hypercalciuria were among the most frequent abnormalities. After categorizing urine parameter values into four quartiles, the risk of stone formation was found to increase as the urine calcium, oxalate, uric acid, chloride, and citrate rise. In contrast, the risk of stone formation decreased with the increase of urine potassium.

Conclusion: Oxalate seems to play the most important role as urinary stone risk factor in our population followed by calcium and uric acid. In addition to the risk factors, it seems that supersaturation as the sum of all risk factors probably has a high predictive value.

Keywords: kidney calculi, risk factors, calcium oxalate, hypercalciuria, hyperoxaluria

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INTRODUCTION

Nephrolithiasis is a common disorder with the estimated prevalence of up to 15% in lifetime, influenced by many factors, such as age, gender, race, and geographical location resulting in various incidence rates in different countries.[1,2] The reported incidence rate of urolithiasis varies between 68 to 720 per 1 000 000 population depending on the countries or regions in which the studies were performed.[3] In a population-based study in Iran, the pooled yearly incidence of urolithiasis was 136/100 000.[3]

Calcium is a principal component of urinary calculi, forming nearly 80% of all stones.[4] Stones containing calcium are mainly in the form of calcium oxalate with the relative occurrence rate of 60% followed by hydroxyapatite (20%) and brushite (2%).[5]

Not only genetic and environmental factors, but also metabolic ones are implicated in the pathogenesis of stone formation.[6-9] Therefore, metabolic evaluation of recurrent stone formers might

recognize urolithiasis risk factors,[10,11] leading to proper medical and dietary therapies to prevent stone formation.[12]

It has been shown that lifestyle, diet, race, environment, climate, drinking water quantity, and soil factors can affect stone formation; therefore, the risk factors for Iranian stone formers might be different from that of other ethnicities or races.

In this study, we evaluated the contributory urinary factors to the idiopathic calcium stone disease in male stone formers.

MATERIALS AND METHODS

In this case-control study, 106 men with recurrent urinary calcium stones were recruited. They were selected from our outpatient stone clinic and were in the age range of 30 to 55 years. The study was approved by the medical ethics committee, and all of the subjects gave their informed consents.

The participants had no documented metabolic, gastrointestinal, liver, renal, cardiovascular, or endocrinological disorders. We excluded all the patients with urinary tract anomalies, urinary tract infection, first single stone presentation, obesity (Body Mass Index > 30), and those who were on medication that may affect calcium metabolism. Furthermore, those patients with the excretion of cystine crystals in their urine were also excluded from this study.

"Recurrent stone former" was defined as either a recurrent stone event or an increase in the stone size during the past five years confirmed by imaging modalities. The type of stones was documented either by stone analysis (69 patients) or by disappearance of the opaque material on conventional radiography after stone passage or intervention for stone removal (37 patients).

Two 24-hour urine samples with one week to one month apart were collected from all the subjects. Moreover, the patients were requested to collect their urine at least 30 days after the last stone episode, which was defined either by an intervention for stone removal or spontaneous stone passage. Furthermore, we asked the patients to be on their normal diets and avoid taking any medication that could influence urinary excretion rates of the stone forming materials two weeks prior to the urine collections.

The control group consisted of 109 healthy men with the age range of 30 to 55 years. They volunteered to participate in this study. All the control subjects had no renal or urinary stone disease confirmed by ultrasonography. They were instructed to collect two 24-hour urine samples similar to the case group. The 24-hour urine samples were collected in polyethylene containers with Hydrochloric Acid 6N or Boric Acid as preservative and were stored at -20° C until analysis.

Urine volume, urine specific gravity, creatinine, urea, phosphate, calcium, oxalate, citrate, sodium, potassium, chloride, magnesium, uric acid, pH (in fresh urine), urinary supersaturation of calcium oxalate, calcium phosphate, and uric acid were measured using standard methods as indicated in brackets: sodium (flame photometry; constant velocity (CV) 1.9%), potassium (flame photometry; CV 1.5%), chloride (colorimetric assay; CV 2.1%), pH (reflectance photometry), specific gravity (refractometry), protein (sulfosalicylic acid, quantitative; CV 2.1%), creatinine (Jaffe's kinetic, CV 1.8%), calcium (Arsenazo, colorimetric, CV 2.4%), oxalate (enzymatic colorimetric, LTA, Milano, Italy, CV 3.57%), uric acid (enzymatic uricase; CV 4.2%), citrate (enzymatic colorimetric, LTA, Milano, Italy, CV 3.18%), magnesium (Calmagite, colorimetric; CV 2.9%), urea (urease, GLDH; CV 2.6%), chloride (colorimetric assay; CV 2.3%), and inorganic phosphate (phosphomolybdate; CV 2.5%). Lithorisk software was utilized to calculate the supersaturation.

We measured calcium, phosphate, magnesium, and oxalate in the Hydrochloric Acid 6N containing urine, and the other parameters in the Boric Acid containing urine, according to National Committee for Clinical Laboratory Standards.[13]

We further categorized 24-hour urine chemistries into four quartiles, and class intervals were defined by combining both groups. Thereafter, these new categorical variables were compared between the two groups using the Chi-square test as well as multivariate logistic regression to obtain age-adjusted odds ratios (for each categorical

variable, the first quartile served as the reference category). We adjusted the urine chemical values for body weights and urine creatinine levels in all the analyses. The mean of two 24-hour urine chemistries was calculated and applied. A *P* value of less than .05 was considered statistically significant.

RESULTS

The characteristics of the cases and controls are summarized in Table 1.

Although the controls were selected matching for age, the difference between two groups was statistically significant. To overcome this mismatch, the 24-hour urine chemistries were compared by covariances analysis in which "age" was the covariate. In the case group that the linear age relationship was important, the adjusted means and their confidence intervals were reported.

Of 69 patients who had stone analysis, 64 were calcium oxalate stone formers, while 1 had calcium phosphate stone, and the remaining 4 patients were mixed calcium stone formers.

Stone formers had significantly higher urine oxalate, calcium, uric acid, chloride in comparison to the healthy controls ($P < .001$). Tendencies towards statistical significance were observed with higher values of creatinine and lower values of urine potassium when the cases were compared with the healthy controls ($P = .064$ and $P = .075$, respectively). Citrate, however, had strong linear correlation with age and did not differ between the two groups after the adjustment made for age ($P = .892$). The mean value of calcium oxalate supersaturation was significantly higher in the patients compared to the controls ($P = .001$) (Table 2).

The mean 24-hour urine values were further adjusted according to the measured body weights and urine creatinine (Tables 3 and 4).

Excretion of oxalate, chloride, and uric acid

Table 1. Characteristics of the participants

Variable		Case	Control	*P*
Age (y)	Mean	43.42 (SD: 6.925)	38.36 (SD: 6.907)	<.001
	Median	44.00	36.00	
	Range	30 to 55	30 to 55	
Weight (kg)	Mean	78.44 (SD: 9.861)	78.53 (SD: 10.540)	.950
	Median	79.50	78.00	
	Range	50 to 104	54 to 104	
Height (cm)	Mean	172.59 (SD:6.606)	174.38 (SD: 7.536)	.066
	Median	173.00	175.00	
	Range	160 to 190	155 to 190	
Body Mass Index (kg/m^2)	Mean	26.3115 (SD: 2.74583)	25.8258 (SD: 3.00603)	.218
	Median	26.7946	26.4463	
	Range	18.41 to 29.94	17.83 to 29.98	

Table 2. Comparison of the mean values of urinary parameters in the cases and the controls

Variable	Case (n = 106)	Control (n =109)	*P*
Urine pH (first morning)	5.9525 (SD:0.48195)	5.9817 (SD:0.47957)	.88
Volume (mL/24hr)	1842.1 (SD: 771.09)	1338.2 (SD: 448.67)	.026*
Phosphorus (g/24hr)	0.63 (SD:0.26)	0.59 (SD: 0.23)	.218
Creatinine (g/24hr)	1.78 (SD:0.55)	1.65 (SD: 0.55)	.064
Calcium (mg/24hr)	221.46 (SD: 109.89)	156.26 (SD: 73.30)	<.001
Oxalate (mmol/24hrs)†	0.4584 (SD:0.1860)	0.3366 (SD: 0.1284)	<.001
Sodium (meq/24hr)	220.83 (SD: 88.66)	211.73 (SD: 69.53)	.235
Uric acid (mg/24hr)	464.43 (SD: 239.00)	352.1 (SD: 205.86)	<.001
Magnesium (mg/24hr)	97.88 (SD:48.80)	91.03 (SD: 43.59)	.275
Urea (mg/24hr)	19.83 (SD:6.62)	18.96 (SD: 6.25)	.354
Chloride (mmol/24hr)	196.82 (SD: 73.67)	167.96 (SD: 52.04)	<.001
Citrate (mg/24hr)	487.55§ (SE: 23.646)	461.20§ (SE: 23.298)	.892*
Potassium (meq/24hr)	47.69 (SD: 20.27)	53.90 (SD: 21.59)	.075
Calcium oxalate supersaturation	7.52 (SD: 4.69)	5.80 (SD: 2.68)	.001
Calcium phosphate supersaturation	1.63 (SD: 1.78)	1.59 (SD: 1.56)	.675
Uric acid supersaturation	0.56 (SD: 0.61)	0.48 (SD:0.40)	.273

*Adjusted for age
†Oxalate (mg/24hr): Case: 41.30 (SD:16.76), control: 30.32 (SD: 11.57)
§Adjusted mean

were significantly higher in the case group when the mean 24-hour urine values were compared adjusted for body weights ($P < .001$, $P < .004$, and $P < .001$, respectively). All other 24-hour urine parameters did not show any significant differences between the patients and the controls, except for potassium that was marginally higher in the control group ($P = .051$) (Table 3).

When we adjusted the results for the urine creatinine, the cases had significantly higher urine oxalate and uric acid, and lower potassium excretion than the controls ($P < .001$, $P = .011$, and $P < .001$, respectively). No other significant differences were observed (Table 4).

In terms of concentration, the mean value of urea was higher in the control group ($P = .002$), yet other parameters did not differ significantly when the two groups were compared (Table 5).

Hyperoxaluria and hypercalciuria were the most frequent abnormalities. Since there is a lack of data in our country regarding the normal values for the urine parameters, we examined the risk of stone formation according to the categories of absolute amounts of 24-hour urinary excretion (Table 6). To determine the odds ratio of recurrent urinary stone formation due to variables with significant effect, logistic regression model was used, in which presence or absence of urolithiasis (dependent variable) as well as risk factors, including calcium, oxalate, uric acid, chloride, and citrate (independent variables) were assessed. We found that the risk of stone formation would increase with rising urine calcium [$P = .026$; OR (95%CI):1.471 (0.971 to 1.632)], oxalate [$P = .015$; OR (95%CI): 2.061 (1.543 to - 2.753)], uric acid [$P = .028$; OR (95%CI): 1.668 (1.269 to 2.193)], chloride [$P = .034$; OR (95%CI): 1.407 (1.083 to 1.829)], and citrate [$P = .026$; OR (95%CI): 1.660 (1.265 to 2.178)]. In contrast, stone formation risk would decrease with an increase in urine potassium [OR (95%CI): 0.766 (0.592 to 0.990)]. For other urine parameters, no significant association was found. Since the risk of stone formation in a patient is the consequent of inhibitory as well as

Table 3. Comparison of the mean values of the urine parameters adjusted for weight in the case and control groups

Variable	Case (n = 106)	Control (n = 109)	*P*
Phosphorus (g/kg)	8.08 (SD: 3.28)	7.55 (SD: 3.06)	.323
Calcium (mg/kg)	2.88 (SD: 1.54)	2.02 (SD: 1.00477)	.296*
Oxalate (mmol/kg)	0.0059 (SD: 0.002391)	0.00436 (SD: 0.001740)	<.001
Sodium (meq/kg)	2.84 (SD: 1.16)	2.74 (SD: 0.96)	.336
Uric acid (mg/kg)	5.95 (SD: 3.02)	4.54 (SD: 2.65)	<.001
Magnesium (mg/kg)	1.26 (SD: 0.64)	1.18 (SD: 0.6)	.401
Urea (mg/kg)	0.25 (SD: 0.086)	0.24 (SD: 0.08)	.491
Chloride (mmol/kg)	2.54 (SD: 0.98)	2.17 (SD: 0.71)	.004
Citrate (mg/kg)	6.95 (SD: 3.77)	5.28 (SD: 2.32)	.918
Potassium (meq/kg)	0.61 (SD: 0.26)	0.69 (SD: 0.28)	.051

*Adjusted for age

Table 4. Comparison of the mean values of the urine parameters adjusted for urine creatinine in the case and control groups

Variable	Case (n = 106)	Control (n = 109)	*P*
Phosphorus (g/mg Cr)	0.35 (SD: 0.09)	0.36 (SD: 0.10)	.219
Calcium (mg/mg Cr)	0.13 (SD: 0.06)	0.10 (SD: 0.05)	.432†
Oxalate (mmol/mg Cr)	0.0003 (SD: 0.00012)	0.0002 (SD: 0.00009)	<.001
Sodium (meq/mg Cr)	0.13 (SD: 0.05)	0.14 (SD: 0.04)	.566
Uric acid (mg/mg Cr)	0.27 (SD: 0.13)	0.22 (SD: 0.11)	.011
Magnesium (mg/mg Cr)	0.05 (SD: 0.02)	0.06 (SD: 0.02)	.539
Urea (mg/mg Cr)	0.01 (SD: 0.002)	0.01 (SD: 0.002)	.057
Chloride (mmol/mg Cr)	0.11 (SD: 0.04701)	0.11 (SD: 0.04152)	.335
Citrate (mg/mg Cr)	0.34 (SD: 0.21)	0.28 (SD: 0.14)	.658
Potassium (meq/mg Cr)	0.03 (SD: 0.01)	0.03 (SD: 0.01)	<.001

*Cr indicates creatinine.
†Adjusted for age

Table 5. Concentration of parameters in 24-hour urine of cases and controls

Variable	Case (n = 106)	Control (n = 109)	P
Calcium (mg/dL)	13.62 (SD: 9.74)	12.12 (SD: 5.25)	.287
Oxalate (mmol/dL)	0.09 (SD: 0.01)	0.027 (SD: 0.01)	.99*
Sodium (meq/dL)	13.24 (SD: 5.36)	16.74 (SD: 5.19)	.940
Uric acid (mg/dL)	27.07 (SD: 14.58)	27.08 (SD: 14.82)	.618
Magnesium (mg/dL)	6.04 (SD: 3.62)	7.13 (SD: 3.40)	.095
Urea (mg/dL)	1.21 (SD: 0.54)	1.49 (SD: 0.48)	.002
Chloride (mmol/dL)	11.50 (SD: 3.82)	12.95 (SD: 2.84)	.839
Citrate (mg/dL)	32.76 (SD: 19.25)	32.85 (SD: 15.95)	.794
Potassium (meq/dL)	2.89 (SD: 1.42)	4.29 (SD: 1.89)	.117*

*Adjusted for age

Table 6. Stone formation trend according to the categories of 24-hour urinary excretion

Variable		Number of Cases	Number of Controls	Odds Ratio	Odds Ratio for Trend (95%CI)
Creatinine (g/24hr)	Q1, Cr < 1.340	25	31	Ref	1.471 (0.971 to 1.632)
	Q2, Cr: 1.34 to 1.700	24	31	1.048 (0.468 to 2.346)	
	Q3, Cr: 1.701 to 2.050	28	25	1.442 (0.645 to 3.226)	
	Q4, Cr > 2.050	29	22	1.945 (0.854 to 4.427)	
Calcium (mg/24hr)	Q1, Ca < 110	18	36	Ref	1.682 (1.277 to 2.216)
	Q2, Ca: 110 to 174	20	34	1.004 (0.435 to 2.318)	
	Q3, Ca: 174 to 245.5	29	26	1.954 (0.862 to 4.428)	
	Q4, Ca > 245.5	39	13	4.587 (1.893 to 11.115)	
Oxalate (mmol/24hr)	Q1, Ox < 0.265	14	40	Ref	2.061 (1.543 to - 2.753)
	Q2, Ox: 0.266 to 0.364	20	34	1.587 (0.661 to 3.810)	
	Q3, Ox: 0.365 to 0.497	34	20	5.273 (2.173 to 12.793)	
	Q4, Ox > 0.497	38	15	7.266 (2.927 to 18.039)	
Sodium (meq/24hr)	Q1, Na < 163	28	28	Ref	1.087 (0.840 to 1.405)
	Q2 Na: 163 to 207.5	26	26	0.883 (0.393 to 1.986)	
	Q3 Na: 207.5 to 262	21	21	0.697 (0.307 to 1.582)	
	Q4 Na > 262	31	31	1.415 (0.627 to 3.197)	
Uric Acid (mg/24hr)	Q1 UA < 214.5	18	36	Ref	1.668 (1.269 to 2.193)
	Q2 UA: 214.5 to 353	22	32	1.134 (0.489 to 2.630)	
	Q3 UA: 353 to 572	29	25	2.197 (0.959 to 5.032)	
	Q4 UA > 572	37	16	4.401 (1.846 to 10.489)	
Magnesium (mg/24hr)	Q1 Mg < 55.5	26	28	Ref	1.039 (0.804 to 1.341)
	Q2 Mg: 55.5 to 92.5	26	29	0.890 (0.397 to 1.994)	
	Q3 Mg: 92.5 to 123.5	28	26	1.070 (0.479 to 2.391)	
	Q4 Mg > 123.5	26	26	1.067 (0.475 to 2.396)	
Urea (mg/24hr)	Q1 Urea < 15	27	27	Ref	1.193 (0.923 to 1.541)
	Q2 Urea: 15 to 19.55	22	36	0.542 (0.242 to 1.214)	
	Q3 Urea:19.55 to 23.5	25	25	0.969 (0.425 to 2.209)	
	Q4 Urea > 23.5	32	21	1.480 (0.657 to 3.338)	
Chloride (mmol/24hr)	Q1 Cl < 134.5	21	34	Ref	1.407 (1.083 to 1.829)
	Q2 Cl: 134.5 to 170	23	30	1.407 (0.619 to 3.202)	
	Q3 Cl: 170 to 214.5	26	28	1.524 (0.679 to 3.422)	
	Q4 Cl > 214.5	36	17	3.081 (1.333 to 7.120)	
Citrate (mg/24hr)	Q1 Cit < 336.5	16	38	Ref	1.660 (1.265 to 2.178)
	Q2 Cit: 336.5 to 410	20	34	1.194 (0.514 to 2.773)	
	Q3 Cit: 410 to 584.5	35	19	3.784 (1.624 to 8.820)	
	Q4 Cit > 584.5	35	18	3.603 (1.536 to 8.453)	
Potassium (meq/24hr)	Q1 K < 36	32	24	Ref	0.766 (0.592 to 0.990)
	Q2 K: 36 to 48	28	26	0.582 (0.257 to 1.318)	
	Q3 K: 48 to 61.5	27	25	0.710 (0.314 to 1.6050	
	Q4 K > 61.5	19	34	0.383 (0.168 to .872)	

Q indicates quartile; and 95% CI, 95% confidence interval.

provoking factors, it could be shown better by supersaturation; thus, the result about the citrate is not conflicting with its inhibitory role.

DISCUSSION

In this study, the stone formers had significantly higher urine oxalate excretion not only in terms of absolute amount, but also after adjustments for body weight and urine creatinine. In addition, hyperoxaluria was one of the most frequent abnormalities in our study. After categorizing the absolute amounts of oxalate into 4 quartiles (Table 6), the risk of stone formation increased from the third quartile (ie, Ox > 32.80 mg/24hr or Ox > 0.365 mmol/24hr). These findings indicate the very important role of oxalate as a risk factor for stone formation in our population.

Robertson and Hughes re-examined the hypothesis of the superior role of mild hyperoxaluria over hypercalciuria in stone formation pathogenesis. The result of their study in Arabian Peninsula indicated that the prevalence of calcium containing stones was higher in that area than the western countries. Of note, none of their subjects had hypercalciuria, and hyperoxaluria was strongly associated with urolithiasis.[14]

Some other studies have also reported higher mean urinary excretion of oxalate in stone formers compared with the individuals who are not stone formers.[15-17] Although Curhan and colleagues first reported no significant differences in the mean values of urine oxalate in their cases and controls,[18] in the second cycle of urine collection after increasing their sample size, they found that the mean urine oxalate was significantly higher in the cases than the controls, and that the risk of stone formation escalated with increasing urine oxalate.[19] However, Netelenbos and associates reported that hyperoxaluria did not influence the risk for active stone formation.[20]

Although hyperoxaluria was one of the most common risk factors in the studies by Serra and coworkers[6] and Curhan and colleagues,[18] it was the second most common risk factor in the study by Hess and associates,[21] but not very common in the Thai stone formers.[15] Hess and colleagues included both men and women in their study and this might have influenced the frequency of hyperoxaluria in their study compared to ours that was carried out in men only.[21] This justification could further be supported by the results of the study by Curhan and coworkers, where they reported that hyperoxaluria was not the most frequent abnormality among their female cohorts.[18] As a result, differences in oxalate and calcium intake as well as genetic factors could be in part responsible for different values in various populations.

In our study, the mean calcium excretion (only absolute amount) was significantly higher in the stone formers than the controls, which is consistent with other studies.[6,16,18,19,22] Furthermore, hypercalciuria was one of the most common risk factors that is in agreement with studies carried out by other researchers.[12,18,21,23] In addition, when categorizing the urine calcium amounts into 4 quartiles (Table 6), risk of stone formation increased in the fourth quartile (with Ca > 245.5mg/24hr). Curhan and colleagues reported that the relative risk of stone formation significantly increased for urine calcium values of over 250mg/24hr in the male cohort,[18] which is very similar to the results of the present study.

Mean urine uric acid was significantly higher among our cases that is in accordance with some studies.[16,24,25] After adjusting the uric acid value for body weight and urine creatinine, we reached approximately the same results. In addition, after categorizing uric acid absolute amounts into four quartiles, the risk of stone formation increased in the fourth quartile, supporting the existing belief that uric acid increases the risk of calcium oxalate stone formation.[20,26]

The mean values of magnesium did not differ between the groups that is compatible with the study by Deshmukh and Khan.[16] In the study by Curhan and coworkers,[18,19] although male cases had higher urine magnesium than the controls, the differences did not seem to be clinically significant.

In our study, the risk of stone formation increased unexpectedly with the rise in urine citrate levels from the third quartile (Cit > 410 mg/24hr) that is inconsistent with the studies of Netelenbos and colleagues[20] and Curhan and Taylor.[19] In

Curhan and associates' first study,[18] lower urine citrate was not associated with the increased risk of stone formation in the male cohort. Generally, hypocitraturia has happened to be an isolated abnormality in up to 10% of the patients with calcium stones and has been associated with other risk factors in 20% to 60% of the patients.[12,27-29] In our study, 13.2% of the patients were hypocitratuic.

One possible explanation for this discrepancy with the inhibitory role of citrate is that its effect as stone formation inhibitor could be overcome by calcium, and especially oxalate promotive effect in our study. Furthermore, although Khan and Hackett reported that urine magnesium decreases urinary saturation of calcium oxalate through increasing urinary pH and citrate,[30] two randomized trials did not show any clinical advantages in stone formers who were taking magnesium oxide compared with those who took placebo or received no treatment at all.[31,32] This leaves us with the unclear role of magnesium in stone formation.

Based on our findings, the cases found to have a tendency toward showing a statistical significance in lower mean urine potassium over the controls and it is in accordance with the literature.[16,18,19,22] This was also true even after adjusting the value of potassium for body weight and urine creatinine (Tables 3 and 4). Furthermore, after categorizing potassium absolute amounts into four quartiles, we observed that potassium was inversely associated with the risk of stone formation beginning from the fourth quartile ($K > 61.501$ meq/24hr). All these could demonstrate the role of potassium in preventing stone formation. Although not statistically significant, our patients had higher mean urine sodium and phosphate excretion in agreement with the study of Curhan and Taylor (male cohort.)[19]

According to our results, it seems that absolute amounts of urinary risks are more important than adjustment of these urinary measurements with urine creatinine levels and body weights.

The mean values of calcium oxalate supersaturation were significantly higher in the case group than the controls in our study. Higher calcium oxalate supersaturation in calcium stone formers were also reported in several studies.[15,17,33,34] According to the study by Coe and associates, supersaturation is a useful index to follow the treatment response in stone formers.[35] Siener and coworkers also reported that after medical treatment for calcium oxalate in stone formers, those who remained stone-free had significant decrease in calcium oxalate supersaturation.[17]

CONCLUSION

In our study, oxalate was realized to play the most important role as a urinary stone risk factor in our population followed by calcium and uric acid. It seems that the absolute amount of parameters is a stronger and more determinant factor in stone formation than their concentration and the adjusted values for body weight and urine creatinine.

In comparison to other studies, some discrepancies were observed in our results that could be justified due to the fact that our sample group was very homogeneous in terms of gender, age range, and exclusion of primarily stone formers. Furthermore, the differences in lifestyle, environment, and races are believed to be other explanatory factors to this issue.

According to our findings, since supersaturation is not influenced by geographical, environmental, and nutritional factors, it could be considered as the final and aggregative index for the evaluation of the stone formers. It seems that seeking an index that could accommodate all involving elements in stone formation is necessary, and supersaturation by definition could be one of them. As our studied population was highly selective, the results cannot be generalized to other groups of patients.

CONFLICT OF INTEREST

None declared.

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