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Dietary Management of Idiopathic Hyperoxaluria and the Influence of Patient Characteristics and Compliance

Zeyad R Schwen^a, Julie M Riley^a, Yaniv Shilo^a, and Timothy D Averch^a

^aDepartment of Urology, University of Pittsburgh Medical Center Pittsburgh, Pennsylvania, USA

Abstract

Objectives—To assess the efficacy of dietary management for the treatment of idiopathic hyperoxaluria in a large tertiary care center. Additionally, this study examines the influence of patient factors, compliance, and follow-up on oxalate reduction which has not been previously investigated.

Methods—Retrospectively, 149 kidney stone patients with idiopathic hyperoxaluria who received dietary management at our stone clinic were evaluated. Changes in urinary parameters on 24-hour urine collections were calculated for all patients and those with abnormal values in the overall, short (30–240 days), and long-term (>240 days) time period. Changes in urinary oxalate were evaluated with respect to patient characteristics and compliance measures.

Results—Urine oxalate and supersaturation of calcium oxalate were significantly ($P<0.001$) reduced by 8.9 ± 19.2 mg/d and 1.7 ± 4.3 , respectively. 48.3% reduced their urinary oxalate to normal. Urine oxalate reductions were similar in the short- and long-term periods. Women lowered urine oxalate nearly twice as much as men (12.7 ± 2.0 mg/d vs 6.7 ± 2.2 mg/d, $P=0.022$) and BMI negatively correlated with oxalate reduction (Pearson's $r=-0.213$). Reported noncompliance and keeping follow-up appointments did not affect oxalate, however, there was a significant correlation between increasing urine volume and reducing oxalate (Pearson's $r=-0.21$).

Conclusions—This study confirms that meaningful reductions of urine oxalate and supersaturation of calcium oxalate can be achieved with dietary management of hyperoxaluria on a larger clinical scale. Furthermore, we identified that females and patients with low BMIs had greater urine oxalate reductions and also urine volume may be used by clinicians as a measure of dietary compliance.

Keywords

hyperoxaluria; diet therapy; nephrolithiasis; compliance

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Correspondence to: Zeyad Schwen, Department of Urology, University of Pittsburgh Medical Center, Kaufmann Building, Suite 700, 3471 Fifth Avenue, Pittsburgh, PA 15213-3221, Phone: (513) 324-5851, Fax: (412) 692-4101, zschwen21@gmail.com.

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Introduction

Lifetime prevalence of nephrolithiasis in the United States is estimated to be 10.6–13.0% in men and 7.1% in women^{1,2}. Idiopathic hyperoxaluria is a known risk factor present in over 30% of stone formers which promotes calcium oxalate crystallization and contributes to stone recurrence³. Hyperoxaluria in stone formers is related to an exaggerated absorption of dietary oxalate^{4,5} thus making dietary modification a mainstay of recurrence prevention^{6–8}. Additionally, changes in urinary oxalate exert a greater influence on calcium oxalate supersaturation than do proportional changes in calcium, further stressing the importance of dietary modification to reduce urinary oxalate as a primary strategy for reducing recurrence^{9,10}. The dietary contribution to increased oxalate absorption in idiopathic hyperoxaluria is primarily the result of a high intake of dietary oxalate or oxalate precursors^{7,10} and suboptimal calcium consumption^{11,12}. Therefore, hyperoxaluric patients are counseled to reduce the consumption of oxalate-rich foods, normalize dietary calcium, and maintain a high fluid intake^{6,13}. Multiple studies have shown that traditional dietary management can reduce oxalate excretion in hyperoxaluric stone formers, however the reported oxalate reductions varied considerably, ranging from 5.7 to 14.4 mg/d^{6,7,14–16}. Our primary aim is to address the variation of reported oxalate reductions by examining whether durable reductions in urinary oxalate through dietary management can be achieved for hyperoxaluric patients in a larger patient cohort.

Furthermore, previous studies have not identified how factors such as patient characteristics and compliance influence dietary management of hyperoxaluria which may explain variations in reported oxalate reduction. Identifying determinants of oxalate reduction can help clinicians optimize treatment of hyperoxaluria by targeting modifiable variables that influence outcomes as well as help clinicians better identify non-adherent patients who require additional assistance. In this study, we conducted a quality control review to determine a large kidney stone clinic's performance in reducing the urinary stone risk of patients with idiopathic hyperoxaluria. We then evaluated how patient characteristics, compliance measures, and follow-up affected a patient's ability to reduce urinary oxalate.

Material and Methods

After IRB approval, a retrospective study of our kidney stone clinic was performed. We identified 267 recurrent and first-time kidney stone patients over 18 years of age from our registry with idiopathic hyperoxaluria, defined as a urinary oxalate excretion greater than or equal to 40mg/d and less than 80mg/d in the absence of a previous diagnosis of primary hyperoxaluria (3 patients), enteric hyperoxaluria (24 patients), or cystinuria (5 patients), who were seen in our stone clinic between January 2006 and November 2011. Enteric hyperoxaluria included those with inflammatory bowel disease, celiac disease, gastric bypass surgery, or any bowel surgery. Of the 267 patients, 149 patients (95 males, 54 females) had two or more 24-hour urine samples that were at least 30 days apart. Charts were retrospectively reviewed to determine patient characteristics, previous stone history, family history, stone composition, medical and surgical treatment, dietary management, follow-up appointment history, compliance, and results from 24-hour urine collections analyzed by Litholink Corporation (Chicago, IL, USA).

In our multidisciplinary kidney stone clinic, patients received a baseline metabolic evaluation which includes a 24-hour urine study, basic metabolic panel, serum uric acid, parathyroid hormone, 25(OH) vitamin D, and produced a 3-day diet diary. Patients with hyperoxaluria were counseled on reducing daily oxalate intake to 40–50mg, normalizing calcium consumption to 1,000–1,200 mg per day, drinking 2–3L of fluid daily, and avoiding excessive vitamin C supplementation (>2,000mg/d) by a registered dietitian. Patients were

provided educational material and told to avoid high oxalate foods (>10mg/serving), limit moderate oxalate foods (2–10mg/serving) to 2–3 servings per day, and have no limits on low-oxalate foods (<2mg/d). Patients may also have received additional dietary counseling or pharmacotherapy targeting other abnormalities identified on 24-hour urine collections or general trends seen in diet diaries. Patients with elevated urinary sodium and uric acid were told to limit sodium intake to 2,300 mg and limit daily animal protein to 6oz of meat, respectively. Patients returned for follow-up after 3–4 months with a second 24-hour urine study and diet diary to evaluate treatment performance, compliance, and the need for additional dietary or medical therapy. If significant improvement in urinary risk was achieved, patients were followed yearly to monitor treatment and urinary risk. However, if adjustments were required, patients were followed more frequently until performance was optimized.

For each patient, the cumulative change in urine parameters was calculated by the difference between the last urine sample available and the first urine sample with an oxalate ≥ 40 mg/d. Under- or over- collections on 24-hour studies, calculated by a change of greater than 30% of the mean creatinine/kg ratio from previous collections, were not included. Patients with urinary abnormalities at baseline were then evaluated separately to determine their ability to correct urinary risk factors. Additionally, the change in urine parameters was separated into short- (30 to 240 days from baseline) and long-term periods (> 240 days from baseline). These intervals were created to measure the improvement of urine parameters at the initial follow-up after hyperoxaluria diagnosis and determine if the improvements were sustained in subsequent follow-ups. Change in urine oxalate levels was evaluated with respect to patient characteristics such as gender, age, BMI, previous stone history, and family history. Change in oxalate was also evaluated with respect to patient compliance measures to determine whether performance was dependent on adherence to treatment plans. These compliance measures included reported noncompliance, improvement in urine volume, and ability to keep follow-up appointments. Patients were labeled as noncompliant if it was reported in the physician's note. All patients who visited the kidney-stone clinic were counseled on maintaining an increased fluid intake of 2–3L; therefore, we examined the ability to increase urine volume from the baseline urine analysis as a potential objective measure of adherence to dietary modification.

Statistical analysis was performed to determine the significance of changes in urinary risk parameters using the paired Student's *t*-test to compare the first and last 24-hour urine sample. Delta oxalate was compared to patient characteristics and compliance variables utilizing the Mann-Whitney U test and correlations between delta oxalate and BMI, age, and urine volume were analyzed using Pearson's and Spearman's correlations.

Results

Patient characteristics

Of the 149 patients, the majority were male (63.8%), had a mean age of 54.4 ± 12.9 years, and were, on average, obese (BMI of 31.0 ± 7.9 kg/m²) (Table 1). Only 28.6% of patients had a family history of kidney stones and most patients were recurrent stone formers (79.4%). Upon referral to the stone clinic, 65.1% of patients underwent a surgical procedure.

Urinary Stone Risk

Baseline 24-hour urine collection revealed a mean urinary oxalate of 52.3 ± 14.8 mg/d (Table 2). After management at the kidney stone clinic, patients observed significant reductions of oxalate (8.9 ± 19.2 mg/d) and supersaturation of calcium oxalate (1.7 ± 4.3). There was no overall significant increase in urine volume. Almost half (48.3%) of patients

were able to reduce their urinary oxalate to normal levels below 40 mg/d. When analyzing abnormal urinary values separately, significant improvement was achieved in all relevant parameters with between 24.5–73.5% of patients returning to within normal values (Table 2). Those with abnormal urine volume (<2L/d) saw a significant ($P < 0.001$) increase of, on average, 470 mL with 45.6% of patients reaching a volume of 2L and greater.

Subset analysis of the 24-hour urine collections into follow-up time periods from baseline collection reveals a significant reduction in oxalate and SS CaOx in the short-term period (30–240 days) of 7.5 ± 17.0 mg/d ($P < 0.001$) and 1.2 ± 4.8 ($P < 0.001$), respectively. In the long-term period (>240 days), oxalate and SS CaOx remained significant with a reduction of 8.0 ± 20.5 mg/d ($P < 0.001$) and 1.8 ± 4.1 ($P < 0.001$), respectively.

Factors Affecting Oxalate Reduction

Evaluating the impact of patient characteristics on oxalate reduction revealed that females were able to significantly ($P = 0.022$) reduce oxalate nearly twice that of men (Table 3). Having a family history and a previous metabolic workup for stone disease had no impact on mean oxalate reduction. First time stone formers had a greater mean reduction in oxalate levels compared to recurrent stone formers, though not significant ($P = 0.07$). Analysis of BMI and oxalate reduction revealed a significant negative correlation indicating an association between less obese patients having greater reductions in urinary oxalate.

Examining patient compliance revealed that of the 267 patients with hyperoxaluria, only 55.8% completed a second 24-hour urine collection. 11.4% (17/149) of patients were noted as being noncompliant with dietary or medical recommendations in the physician note and 22.1% (33/149) of patients didn't keep a follow-up appointment. These variables, however, did not seem to affect urine oxalate reduction (Table 4). There was a significant negative correlation between urine volume and oxalate indicating patients who increased their urine volume were more likely to reduce their oxalate.

Comment

Our study has demonstrated that hyperoxaluric stone formers can achieve significant and lasting reductions in urinary oxalate and SS CaOx by medical and dietary management at a large kidney-stone clinic with significant reductions in both the short- and long-term follow-up periods. Examining patient characteristics showed females were better at reducing urine oxalate than men and there was a correlation between lower BMIs and higher reductions in oxalate (Table 3). While noncompliance did not impact urinary oxalate reduction, patients who increased urine volume were more likely to demonstrate greater reductions in urine oxalate (Table 4). Our study provides further clinical evidence of the efficacy of dietary treatment for hyperoxaluric patients in reducing stone recurrence risk and offers a better understanding of the influence of patient characteristics and compliance in reducing urinary oxalate.

Our hyperoxaluric patients observed a mean reduction of 8.9 mg/d of urine oxalate with 48.3% of patients returning to within normal oxalate ranges, confirming dietary modification is an effective option for managing hyperoxaluria. Our results are in agreement with the literature due to our observed magnitude of oxalate reduction fitting within the 5.7–14.4 mg/d range reported in previous studies^{6,7,14–16}. We believe our patient cohort is more representative of expected clinical outcomes because of our larger patient cohort and our inclusion of both recurrent and first time stone formers seeking treatment at our tertiary care kidney stone clinic. Variations in achieved oxalate reductions can likely be explained by a combination of differences in patient population and dietary intake, particularly relating to the targeted daily calcium intake which differed in each study and greatly impacts oxalate

excretion¹². Additionally, multiple studies have identified that urine volume is positively correlated to oxalate excretion which is the result of passive tubular reabsorption of oxalate in the kidney^{5,8}. Thus, total 24-hour oxalate excretion is confounded by concurrent increases in urine output which may also explain differences in oxalate reduction. Because we did not observe an overall significant change in urine volume, likely due to our remarkably high baseline urine volume compared to other studies^{5,6,16}, our reported oxalate reduction was likely unaffected by this factor.

More importantly, our patients experienced a mean reduction in SS CaOx (Table 2) which also agrees with previous studies^{7,14,16}. The observed changes in SS CaOx better characterizes the overall reduction in stone recurrence risk achieved by dietary management^{17,18}. Patients with high (> 10) SS CaOx observed, on average, a reduction of 5.34 with 73.5% of patients returning to below a SS CaOx of 10. While changes in oxalate have significantly greater impact on the SS CaOx^{9,10}, our observations could also be explained by the significant correction of abnormal urinary calcium and volume which are other determinants of SS CaOx.

Understanding the many factors affecting oxalate reduction may explain variations in reported outcomes and provide an opportunity to augment treatment of hyperoxaluric patients. Our results revealed females were able to reduce oxalate twice that of their male counterparts, highlighting potential gender differences in oxalate metabolism. Several studies have identified significantly higher urinary oxalate levels in males in both stone formers and normal population^{19,20}. This is believed to be the result of higher endogenous oxalate synthesis in males, which is a result of larger body size and muscle mass^{11,12}. In addition, a protective effect of female sex hormones has been proposed^{21,22}. Examining gender differences in dietary oxalate absorption in healthy adults yielded no significant difference^{11,12}, however this conclusion cannot be applied to hyperoxaluric stone formers who have abnormal and exaggerated responses to dietary oxalate^{4,5}. Our results may be explained by hyperoxaluric females having a greater proportion of modifiable dietary oxalate compared to males. Creating the threshold definition of hyperoxaluria as > 40 mg/d for both genders filters for females with a more exaggerated response to dietary oxalate than males because females have lower endogenous oxalate production. Thus, dietary modification may be more effective at reducing oxalate for females than males with hyperoxaluria. Additionally, it poses the question of whether the threshold for hyperoxaluria should be lower for females relative to males to reflect gender differences of oxalate metabolism. Doing so would lead to the identification of more females with abnormally high dietary contributions of oxalate who could benefit from targeted dietary management to further reduce stone recurrence risk. Further studies are required to examine this relationship.

Like gender, BMI also appears to influence urine oxalate reduction. Obesity is a major risk factor for hyperoxaluria and is three times more prevalent than in non-obese stone formers^{11,20,23}. Like with gender differences, studies have shown that increased oxalate excretion is associated with larger endogenous production due to greater body size^{11,24}. Also, obese patients have greater urine volumes which also account for increased oxalate excretion due to passive tubular absorption²⁴. Our study identified a significant negative correlation between BMI and oxalate reduction which indicates patients with lower BMIs were able to better decrease urine oxalate. We propose that due to the increasing endogenous production with higher BMIs, patients with lower BMIs have a greater dietary contribution to total oxalate excretion which can be targeted with dietary modification. These results, therefore, indicate that patients with hyperoxaluria who have lower BMIs will respond better to dietary modification. Additionally, our data further supports the theoretical benefit of

including weight loss in treatment plans for obese patients to reduce endogenous oxalate production which would supplement urinary oxalate reduction due to dietary modification.

Patient compliance in kidney stone clinics also remains a significant and largely unstudied factor which may affect treatment outcomes of hyperoxaluric patients. Our clinic experienced significant compliance issues as only 55.8% of patients repeated a second 24-hour urine collection and many failed to keep follow-up appointments. Therefore, due to the retrospective nature of our analysis, our patient population excluded those unwilling to repeat a second 24-hour urine collection which can be viewed a source of bias. Examining the effects of follow-up and reported noncompliance on oxalate reduction yielded no significant difference. Noncompliance reported in the medical note is a poor measure of actual noncompliance as studies have shown that physicians grossly underestimate and poorly identify patients who are non-adherent to treatment which may explain why we were unable to detect a significant difference in compliant versus noncompliant patients²⁵. Moreover, dietary and lifestyle treatments often result in greater noncompliance rates that can reach as high as 70% which suggests our 11.4% reported noncompliance is a considerable underestimation²⁶. Therefore, better methods of assessing true adherence are required in the kidney stone clinic. Our results demonstrating a correlation between urine output and oxalate reduction is evidence that the degree of volume increase may be used by clinicians to better assess patient compliance with dietary modification. This relationship is based on the assumption that patients who increase urine volume are more willing to follow dietary changes to reduce oxalate absorption. Passive tubular absorption of oxalate is a potential confounder, however the increased oxalate excretion from greater urine output was offset by oxalate reduction likely due to dietary modification^{5,8}. While the change in urine volume does not quantitatively measure compliance in hyperoxaluric patients, it certainly should be considered by clinicians as a soft sign for adherence to dietary modification.

Our study had multiple limitations including the retrospective nature of this analysis. Additionally, we were not able to use stone recurrence as a treatment endpoint because our stone clinic is a tertiary care center and reporting of stone recurrence is not reliable. We therefore used recurrence risk as interpreted by urinary analysis to measure treatment performance. Our results, despite our efforts to account for under- and over-collections, may also have been influenced by the very small, but significant decrease in urine creatinine which was not accounted for by fluctuations in weight or BMI. The reduction in creatinine, however, only represented a mean change of 2.9% and likely has minimal impact on our results or conclusions. Another limitation in our study is the inherent variation of 24-hour urine collection results which may lead to misdiagnosis of hyperoxaluria. While our clinic utilizes only a single 24-hour urine collection at baseline which has been shown to be adequate²⁷, some authors argue two urine collections are required^{28,29}. Furthermore, though all patients received the same dietary management to target high urinary oxalate, some patients were on pharmacotherapy such as thiazides, allopurinol, and citrate supplements which may have confounded the effect of dietary management alone.

Conclusions

This study confirms that meaningful reductions of urine oxalate and SS CaOx as well as significant correction of abnormally low urine volume can be achieved with dietary management of hyperoxaluria in a large tertiary care center. Our study demonstrated that females and patients with lower BMIs were able to better reduce urine oxalate, which may be due to a greater contribution of dietary oxalate to total oxalate excretion. Based on this observation, we propose that women may require a lower threshold for hyperoxaluria compared to males which warrants further investigation. Furthermore, our observations highlight the theoretical benefit of targeting weight loss in obese patients to supplement the

reduction of oxalate from dietary modification. Lastly, our study demonstrated that urine volume may be an indicator of dietary compliance and could help clinicians better identify noncompliant patients. By understanding the influence of gender, BMI, and compliance on oxalate reduction, clinicians can better individualize therapy to enhance the prevention of stone recurrence in patients with hyperoxaluria.

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Table 1

Patient Characteristics

Patient Characteristic	Results
N	149
Gender (%)	
Male	95/149 (63.8%)
Female	54/149 (36.2%)
Age (years)	
Mean $\pm$ SD	54.4 $\pm$ 12.9
Median (25%ile, 75%ile)	57 (48, 63)
BMI (kg/m²)	
Mean $\pm$ SD	31.0 $\pm$ 7.9
Median (25%ile, 75%ile)	29.4 (25.1, 35.2)
Family History of Stones (%)	28.6%
Previous Stone History (%)	79.4%
Previous Metabolic Workup (%)	32.5%
Follow-up Time (d)	
Mean $\pm$ SD	430 $\pm$ 364.1
Median (25%ile, 75%ile)	288 (170.0, 567.0)
Treatment	
Surgical (%)	97/149 (65.1%)
ESWL	14/97 (14.43%)
URS	76/97 (78.35%)
PCNL	30/97 (30.93%)
Stone Composition	
Pts with Stone Analysis (%)	103/149 (69.13%)
Calcium Oxalate (%)	68/103 (66.02%)
Calcium Phosphate (%)	10/103 (9.71%)
Uric acid (%)	25/103 (24.27%)

Patient characteristics of 149 eligible kidney stone patients included in analysis with hyperoxaluria (≥ 40 mg/d). Surgical management was based on surgeries after referral to tertiary stone clinic. Stone composition was selected based on majority ($\geq 50\%$ if 2 components, and largest % if >2 components) metabolite composition in stone analysis.

BMI = body mass index, d = day, ESWL = extracorporeal shock wave lithotripsy, Pts = patients, PCNL = percutaneous nephrolithotomy, SD = standard deviation, URS = ureteroscopy.

Table 2

Mean metabolite values from first and last urine studies

Variable ± STD	First Urine Study			Last Urine Study			Delta (last-first)			P	
	# Abnormal	All Pts	Abnormal	All Pts	Abnormal	All Pts	Abnormal	All Pts	Abnormal	Abnormal	% Fixed
Volume (mL)	57/149 (38.3%)	2.3 ± 0.9	1.5 ± 0.3	2.5 ± 1.0	2.0 ± 0.8	0.1	0.47	0.161	<0.001		45.6
Creatinine (mg/d)		1806.4 ± 546.2		1753.8 ± 594.0			-52.7	0.014			
Oxalate (mg/d)	149/149 (100%)	52.3 ± 14.8	52.3 ± 14.8	43.4 ± 17.2	43.4 ± 17.2	-8.9	-8.9	<0.001	<0.001		48.3
Calcium (mg/d)	81/149 (54.3%)	256.9 ± 145.4	358 ± 156.1	234.8 ± 119.5	291.5 ± 207.8	-22.1	-66.5	0.123	<0.001		37.0
pH		6.1 ± 0.6		6.2 ± 0.6		0.1		0.142			
Uric acid (mg/d)	49/149 (32.9%)	0.8 ± 0.3	1.0 ± 0.2	0.7 ± 0.3	0.9 ± 0.2	-0.02	-0.1	0.023	0.002		34.7
Citrate (mg/d)	47/149 (31.5%)	732.8 ± 494.1	282.3 ± 152.9	711.0 ± 485.8	360.1 ± 207.4	-21.8	77.8	0.319	0.038		23.4
Magnesium (mg/d)		121.6 ± 52.2		119.5 ± 51.7		-2.1		0.596			
Sodium (mmol/d)	110/149 (73.8%)	211.3 ± 79.7	243.2 ± 63.2	198.9 ± 87.5	209.8 ± 87.7	-12.5	-33.4	0.025	<0.001		24.5
Potassium (mmol/d)		77.8 ± 30.2		80.6 ± 35.4		2.8		0.668			
SS Ca Oxalate	34/149 (22.8%)	7.7 ± 3.9	13.3 ± 3.7	6.05 ± 3.5	7.9 ± 3.7	-1.7	-5.3	<0.001	<0.001		73.5
SS Ca Phos	39/149 (26.2%)	1.3 ± 1.1	2.8 ± 0.7	1.2 ± 1.0	2.0 ± 1.1	-0.1	-0.8	0.073	<0.001		56.4
SS Uric acid	50/149 (32.9%)	0.9 ± 0.9	2.0 ± 0.8	0.7 ± 0.8	1.1 ± 0.9	-0.1	-0.8	0.061	<0.001		52.0

Mean metabolite values from first and last urine studies of all patients and those with abnormal values.

Abnormal values were defined for volume (<2L), oxalate (40mg/d), calcium (250 mg/d for males, 200 mg/d for females), uric acid (800mg/d for males, 750mg/d for females), citrate (<450 mg/d for males, <550 for females), sodium (150mmol/d), SS CaOx (10), SS Ca Phos (2), SS uric acid (1) Significance was defined as a p 0.05.

Ca Phos = calcium phosphate, Pts = patients, SS = supersaturation, STD = standard deviation.

Table 3

Patient Characteristics Affecting Mean Oxalate Reduction

Variable	Mean Oxalate Reduction (mg/d) ± SE	Significance
Gender		
Male	6.7 ± 2.2	0.022
Female	12.7 ± 2.0	
Family History		
Yes	5.8 ± 5.6	0.961
No	8.6 ± 2.2	
Previous Stone		
Yes	7.8 ± 1.8	0.07
No	15.4 ± 3.5	
Previous Metabolic Workup		
Yes	5.0 ± 3.4	0.218
No	11.7 ± 1.9	
Correlations	Correlation Coefficient	Significance
Age	rho = -.123	0.134
BMI	r = -0.213	0.01

Oxalate reductions were analyzed based on gender, family stone history, previous stone history, and previous metabolic workup.

For continuous variables such as age and BMI, Spearman's rho and Pearson's r correlation coefficients were calculated, respectively. Significance was defined as a p 0.05.

BMI = body mass index, SE = standard error.

Table 4

Compliance Variables Affecting Mean Oxalate Reduction

Variable	N	Mean Oxalate Reduction (mg/d) ± SE	Significance
Noncompliance			
Yes	17	9.6 ± 4.3	0.839
No	132	8.9 ± 1.7	
Kept appointment			
Yes	116	8.5 ± 1.9	0.577
No	33	10.6 ± 2.7	
Correlation	Correlation Coefficient		Significance
Urine Volume	r = -0.21		0.01

Noncompliance as mentioned in the medical note and follow-up appointment history were compared to mean oxalate reduction.

Overall change in urine volume was correlated to mean oxalate reduction using Pearson's r correlation coefficient. Significance was defined as a $p < 0.05$.

SE = standard error.