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The effect of dietary counselling in reducing sodium consumption among hypercalciuric stone formers and its impact on metabolic risk factors

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Abstract

Introduction: Elevated urinary sodium from excessive dietary consumption may lead to hypercalciuria and increase urinary stone risk. Our study aimed to assess the impact of targeted dietary counseling on normalizing urinary sodium levels in hypercalciuric stone patients.

Methods: A retrospective analysis of a prospectively collected metabolic stone clinic database was performed. Patients with hypercalciuria and elevated urine sodium on 24-hour urine collection were counselled by the attending nephrologist, urologist or a registered dietician to limit their dietary sodium to < 2g/day intake. Repeat metabolic testing was performed at least 6 months later. Logistic regression was used to determine correlations between elevated urinary calcium and sodium to other urinary abnormalities, and to evaluate the effect of normalizing urinary sodium on urinary parameters.

Results: Metabolic evaluations from 1184 patients were analyzed. Ninety-eight patients with concomitant hypercalciuria and hypernatriuria were predominantly male (67.3%) and had a higher median body mass index than the entire cohort. The presence of elevated urinary sodium was associated with hyperuricosuria ($p<0.001$) and hyperphosphaturia ($p < 0.001$). In follow-up, 59.4% corrected their urinary sodium, and 43.8% also corrected their urinary calcium. Patients who corrected their urinary sodium were also more likely to have normal urinary values for volume ($p = 0.045$), oxalate ($p = 0.004$), and urate ($p = 0.008$).

Conclusions: Targeted dietary counseling can be associated with normalizing both elevated urinary sodium and calcium levels in stone patients and may reduce the need for pharmacotherapy for the treatment of hypercalciuria.

Keywords: Hypercalciuria; Dietary counseling; Renal calculi; Urinary sodium

Abbreviations

Sodium (Na)
Calcium (Ca)
Metabolic stone clinic (MSC)
Body mass index (BMI)
24-hour urine collection (24-HUC)
Parathyroid hormone (PTH)
Interquartile range (IQR)
Confidence interval (CI)

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Introduction

The prevalence of urolithiasis is on the rise worldwide [1]. The United States National Health and Nutrition Examination Survey (NHANES) published in 2022 showed that the overall prevalence of urolithiasis had increased from 6.3% in 1988-1994 to 11% in 2015-2018, as well for all age, gender and racial ethnic groups [2]. Recurrence rates after the first stone event range from 30-50% within 10 years [3]. Not surprisingly, 81% of patients report a motivation to implement prophylactic measures to prevent another stone episode [4].

Calcium oxalate composition accounts for 70-80% of all urolithiasis, and the main metabolic abnormalities associated with calcium (Ca) stone formation include dehydration, hypercalciuria, hypernatriuria, hypocitraturia, and hyperoxaluria [5]. The role of diet in the pathogenesis of kidney stones is well known and dietary indiscretion has been proposed as one of the reasons for increasing rates of stone disease especially among younger adults and females [6]. Among Ca stone formers, elevated urinary sodium (Na) related to excessive dietary intake is associated with an 61% increase in stone recurrence [7].

Excess dietary Na consumption has a well-known link to Ca stone formation. Among Ca stone formers, high Na intake leads to a decrease in water and Na absorption in the proximal convoluted tubule, increasing Ca excretion and diminishing citrate excretion, with both factors increasing the potential risk of stone formation. It has been estimated that every 100mmol (2300mg) of Na excreted in the urine results in a urinary Ca increase of 1mmol (40mg) [8].

American data from the NHANES 1999-2016 survey reported that the mean Na intake of the average American was 3232mg per day, with 77% of the population consuming higher than the recommended amount of Na [9]. Even more concerning, the population adherence to dietary sodium guidelines decreased by 11% from 1999-2000 to 2015-2016 [9]. Excessive dietary Na intake has been identified as one of the leading diet risk factors for cardiovascular morbidity and mortality [10]. In fact, the World Health Organization has identified diet Na intake reduction as one of nine important global health targets for disease prevention, with the goal to reduce consumption by 30% by 2025 [11]. While the cardiovascular benefits of reducing dietary Na are well known even to many patients, the impact on stone prevention is less familiar. Several studies have demonstrated the potential to reduce urinary Ca with the aid of Na dietary restriction [8,12,13].

Goal specific dietary advice based on metabolic assessment has been shown to be effective in modifying patients' risks of further stones [14]. To provide specific dietary counselling, all patients at risk of recurrence should have a metabolic evaluation although data suggests that only 8% of them are offered this evaluation [15]. Most physicians are aware of the role of diet in stone disease prevention but may not be the best members of the health care team to provide nutritional advice. Numerous barriers have been identified including a lack of in-depth nutrition education, time constraints during appointments, and a conflicting body of nutritional information [16]. As a result, patients may not receive the most detailed or up-to-date nutritional advice. Reliance on the internet as a credible source of dietary stone prevention information may not be ideal either [17]. We aimed to evaluate if targeted nutritional education provided by a knowledgeable health care professional could result in patient's lowering their urinary Na and normalizing their urinary Ca levels.

Methods

We performed a retrospective analysis of a prospectively collected metabolic stone clinic (MSC) database including patients seen from September 2001 to October 2021. Western University Institutional Review Board approval was obtained. Patients referred to the MSC had risk factors for stone recurrence according to current guidelines.[18] All patients underwent an in-depth metabolic evaluation including history, physical examination, bloodwork (serum Na, Ca urate, Parathyroid hormone (PTH), vitamin D, creatinine, urea, bicarbonate, chloride, potassium, and phosphate levels), and a single 24-hour urine collection (24-HUC; which included urinary volume, Na, Ca, creatinine, phosphate, urate, urea, citrate, oxalate) and a urinalysis. All patients evaluated in the MSC were first seen by a urologist, patients with complex metabolic abnormalities or renal insufficiency were additionally assessed by a nephrologist, and all patients were offered one-on-one dietary counselling with a registered dietician at the same appointment. General dietary advice was provided, as well as specific recommendations based on the patient's metabolic test results from the attending nephrologist, urologist and/or registered dietician. All patients received guidance both verbally and in a written form instructing them to restrict their Na intake to less than 2000 mg/day, based on current recommendations [16].

Patients with a history of elevated urinary Ca (> 7.5 mmol/day) and elevated urinary Na levels (> 217 mmol/day) were identified. Patients were excluded if they had non-adequately collected 24-HUC based on urinary creatinine levels (reference values are listed on Appendix - Table S1) or were on thiazide diuretics. In patients newly diagnosed with both hypercalciuria and hypernatriuria, thiazides diuretics were not prescribed until dietary modifications were initially attempted. Follow up 24-HUC and blood work were performed at least 6 months after dietary counselling.

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Descriptive statistics were used to assess demographics and normally distributed variables were noted as means (± standard deviation) and non-normally distributed variables as medians (interquartile range (IQR)). Chi-square and Fisher's exact test were used to proportion comparison between groups. Logistic regression was utilized to identify urinary abnormalities that were predictors of hypercalciuria and hypernatriuria and to analyze the association of correcting urinary Na levels to changes in other urinary parameters (Ca, oxalate, urate, citrate, and phosphate). Statistical significance was considered for *p*-values < 0.05 with 95% confidence interval (CI). All analyses were performed using IBM SPSS® Statistics version 26.0 (IBM Corp. Released 2019. IBM SPSS Statistics for Windows, Version 26.0. Armonk, NY: IBM Corp.).

Results

Between September 2001 and October 2021, 1813 24-HUC from 1184 patients were analyzed (Figure 1). Median age of the entire cohort was 52.53 years (IQR 41.8 – 62.11years) with 55.4% male and 44.6% female patients. Median BMI was 28.81 Kg/m^2 (IQR 21.97 – 33.23Kg/m^2) and 76% of the patients had a documented consult with a dietician. The remaining 24% of patients were provided specific dietary advice from the attending urologist or nephrologist.

Stone composition was known in 69.6% of the patients and was predominantly Ca-based in 79.2% of patients. The most common metabolic abnormality in the cohort was low urinary volume (< 2000ml) in 58.9% of patients, followed by hypercalciuria (24.6%), hypernatriuria (23.4%), and hypocitraturia (21.9%).

Among the entire cohort, ninety-eight patients presented with hypercalciuria and concomitant hypernatriuria (Figure 1), with a mean age of 51.51 years (± 12.08years) and median BMI of 31.64Kg/m^2 (IQR 28.56 – 34.95Kg/m^2). The gender distribution of these patients differed from the overall cohort; 67.3% of patients were male and 32.7% were female. Registered dietician consultation was performed in 78% of these patients. Stone composition was known in 83.5% of these patients and, in 82.2% of these patients, the stones were Ca-based. Thirty-one patients (31.6%) had low urinary volume, 38.8% had hyperphosphaturia and 18.9% had hyperoxaluria. Urinary abnormalities in the cohort are described in table 1.

A larger proportion of patients with hypercalciuria and hypernatriuria were male *(p=0.013),* presented an adequate urinary volume (>2L/day) ($p<0.001$), normal urinary citrate ($p<0.001$), hyperphosphaturia ($p<0.001$), and hyperuricosuria ($p<0.001$) compared to patients without hypercalciuria and hypernatriuria (Figure 1).

The follow up 24-HUC was analyzed in the 98 patients to evaluate the changes in urinary parameters after dietary counselling. The median time between initial and follow up 24-HUC was 274 days (range 210 - 393 days). After dietary counseling, 59.4% of patients corrected their urinary Na levels, and 43.8% corrected both their urinary Na and Ca. There was no difference in the proportion of patients who corrected their urinary Na based on whether the dietary advice was provided by a physician (urologist or nephrologist) or the dietician

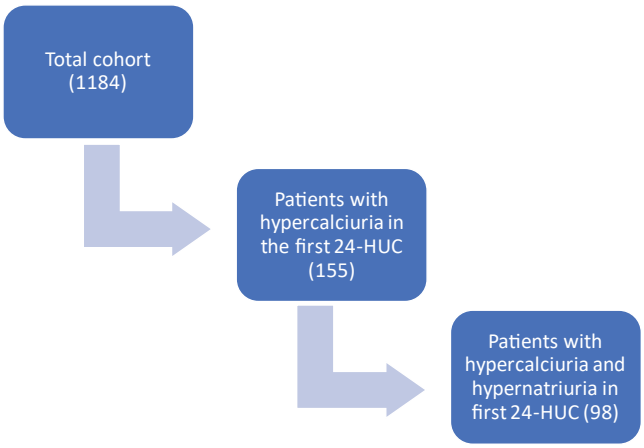

Figure 1: Flowchart demonstrating inclusion of patients for analysis.

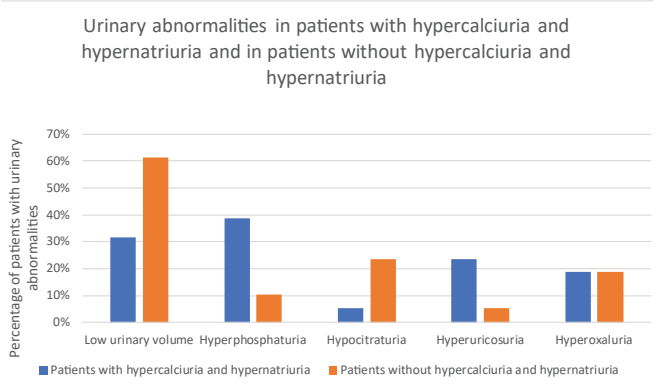

Figure 2: Percentage of urinary abnormalities in patients with concomitant hypercalciuria and hypernatriuria compared to patients without these abnormalities

Table 1: 24-hour urinary parameters in the initial visit and in the follow up visit for patients with hypercalciuria and hypernatriuria

Urinary parameter	Initial collection [median (IQR)]	Follow up collection [median (IQR)]
Volume (ml/day)	2316 (1822 – 2809)	2330 (1749 – 3012)
Sodium (mmol/day)	268 (241 – 305)	197 (152 – 254)
Calcium (mmol/day)	10.5 (9.1 – 12.8)	8.2 (6.2 – 10.9)
Phosphate (mmol/day)	41 (31.7 – 48.2)	37 (25 – 46)
Urate (mmol/day)	4.8 (3.6 – 5.9)	4.4 (3.2 – 5.5)
Oxalate (mmol/day)	370 (249 – 486)	402 (301 – 511)
Citrate (mmol/day)	3.5 (2.4 – 4.6)	3.5 (2.3 – 5.1)

IQR=Interquartile Range

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Table 2: Logistic regression comparing 24-HUC parameters in patients who normalized their elevated urinary Na and patients with persistent hypernatriuria.

Urinary parameter	Exp. B	S.E	Wald	Sig	95% Confidence Interval	
					Lower	Upper
Volume	2.621	0.48	4.035	0.045	1.024	6.712
Oxalate	4.138	0.489	8.428	0.004	1.586	10.793
Phosphate	1.576	0.344	1.093	0.296	0.672	3.696
Urate	6.429	0.698	7.101	0.008	1.636	25.262
Citrate	1.71	0.847	0.401	0.527	0.325	8.977
Calcium	1.045	0.422	0.11	0.916	0.457	2.39

Exp.B=Odds Ratio; S.E.=Standard Error of Regression; Wald=Wald chi-square Test;Sig=Sigmoid Function

Table 3: Comparison of urinary parameters of patients who normalized their urinary sodium in the follow up 24-HUC and patients with persistent hypernatriuria.

Urinary parameter	Normalized urinary sodium in the follow up 24-HUC (median (IQR))	Patients with persistent hypernatriuria (median (IQR))
Volume (ml)	2200 (1588 – 2577)	2450 (2000 – 3175)
Calcium (mmol/day)	8.2 (6.2 – 10)	8.4 (6.6 – 12.7)
Phosphate (mmol/day)	34 (23 – 45)	40 (31 – 50)
Urate (mmol/day)	3.8 (3.1 – 5.0)	5.2 (4.0 – 6.0)
Oxalate (mmol/day)	367 (264 – 467)	464 (378 – 584)
Citrate (mmol/day)	3.5 (2.4 – 5.2)	3.1 (2.1 – 4.8)

(p=0.441). Patients who corrected their urinary Na were more likely to have normalized other parameters including urinary volume (OR 2.621, 95% CI, 1.024–6.712, p=0.045), normal urinary oxalate (OR 4.138, 95% CI, 1.586–10.793, p=0.004), and normal urinary urate (OR 6.429, 95% CI, 1.636–25.262, p=0.008) than patients with persistent hypernatriuria (Table 2). Urinary Ca, phosphate, citrate, and serum parameters did not show significant differences between groups. Table 3 shows the median urinary parameters in the follow up 24-HUC for patients who corrected their urinary Na and patients with persistent hypernatriuria.

Discussion

It has been reported that there is a 30-50% risk of a stone recurrence within 10 years of the initial symptomatic event [19]. Given this, patients are typically quite motivated to seek prevention strategies. The American Urological Association (AUA), the European Association of Urology (EAU) and the Canadian Urological Association (CUA) guidelines all recommend an in-depth metabolic evaluation including serum biochemistry and 24-HUC for patients at high risk of recurrent kidney stones [20,21]. Evidence suggests that at least one 24-HUC has the potential to change the preventive approach in 45% of patients [22]. Specific dietary modifications based on the results of the metabolic evaluation can play a role in correcting urinary abnormalities and aid in the prevention of new stones [8]. A 'dietary-first' approach potentially avoids the long list of side-effects of thiazide-type medications most used to reduce hypercalciuria including electrolyte disturbances, dehydration, renal impairment, glucose intolerance, elevated urate levels and allergic reactions. This approach also allows for medication to be more appropriately prescribed based on individual need and risk factors [23].

Urinary stone formation is a result of an imbalance between crystal formation and inhibitory factors in the urine. The supersaturation of a component increases crystal formation and consequently, precipitation. Several ions and macromolecules are involved in this process, such as citrate and glycosaminoglycans acting as crystal inhibitor factors [24]. One of the most common mechanisms for Ca stone formation is hypercalciuria as it promotes an increase in crystal Ca salts in the urine (mainly oxalate and phosphate) and decreases crystal inhibitory factor concentrations [25]. Hypercalciuria may occur for several reasons including heightened gastrointestinal Ca absorption, increased bone resorption, or due to a renal leak of Ca. Excess Na dietary intake causes higher urinary Na levels [27] leading to an increase in Ca excretion due to a common Na/Ca transport mechanism and also to a more modest decrease in urinary citrate, which are both risk factors for the development of urolithiasis [28].

Berto F, et al., J Food Sci Nutr Res 2024
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In this single institution retrospective review of our multidisciplinary MSC patient population, the metabolic test results of those patients identified to have both hypercalciuria and hypernatriuria were re-evaluated after dietary counselling was provided. Within this cohort, 76% of patients had a consultation with a registered dietitian. The dietician had access to the patients' medical and dietary histories and laboratory results. The remaining 24% of patients received dietary counselling from the attending nephrologist or urologist alone. All patients regardless of who provided the dietary education, received a pamphlet with written dietary target recommendations including a daily dietary sodium target of <2000mg.

A larger proportion of patients with hypernatriuria and hypercalciuria were male (p = 0.013) and male to female rate was 2:1 which is slightly higher than the reported in the literature [29]. Various theories have been proposed to explain the disparity between genders. Hormones might have a role in this dissimilarity. Studies suggest that post-menopausal women with Ca oxalate stones may have a lower risk to develop kidney stones if their estrogen levels are higher [29]. This difference also could be related to dietary factors influencing in urinary parameters and intrinsic differences in urinary metabolism according to gender [29]. The mechanism responsible for hypercalciuria in men occurs mostly in the proximal tubule where a decrease in Ca reabsorption can be observed. In women, this phenomenon is predominant in the distal tubule [26]. Women also have higher urinary pH and citrate levels than men which would act as protective factors [29].

The median BMI of the entire cohort was 28.81Kg/m^2 while in patients with hypercalciuria and hypernatriuria it was 31.64Kg/m^2. The median BMI for the entire cohort falls into the overweight category but, in the combined hypercalciuric/hypernatriuric patients the median BMI would fulfill the diagnosis of obesity. This variance might be linked to dietary factors as foods with high amounts of Na are also high in calories, and sodium intake has been directly linked to daily caloric intake [9].

After the initial 24-HUC, the 98 patients with hypercalciuria and hypernatriuria received dietary recommendations and were offered a consult with a dietitian, and 78% of these patients attended. A 24-HUC was repeated and in this second analysis, 59.4% of the patients were able to correct their hypernatriuria, and among this group of patients 43.8% also corrected the hypercalciuria. Compared to patients with persistent hypernatriuria, patients who had their urinary Na corrected, were more likely to have normal urinary volumes (p=0.045), normal urinary oxalate (p=0.004), and normal urinary urate (p=0.008).

The dietary counseling our patients received was directed by the results of the 24-HUC Na and Ca levels which the dietician, attending nephrologist or urologist reviewed with the patients. It may be the case that patients are more likely to respond to counselling that is informed by objective laboratory results 'confirming' a dietary issue, than they would by general dietary advice without supportive clinical data to highlight a problem. This is an important consideration as the impressive reductions achieved in urinary Na and Ca levels in response to a dietary intervention may require this approach to be replicated.

This study also demonstrates that patients with hypernatriuria are more likely to present with other urinary abnormalities, suggesting this may be a marker for suboptimal dietary practices in general. In addition, patients who were able to correct their urinary Na, were also more likely to demonstrate improvements in their other urinary parameters suggesting they were compliant with other dietary changes.

Dietary counselling is a relatively low-cost intervention for stone prevention, which may obviate the need for medical therapy. Our results suggest regardless of whether the dietary advice is given by the attending nephrologist, urologist or the dietician, correction of previously noted 24 HUC abnormalities is possible when clear target recommendations are provided.

Our study has several limitations. Although the data was prospectively collected, it was analyzed retrospectively and clinical outcomes, such as number of stone events, were not assessed. Among those who corrected their urinary Na and Ca values after dietary counselling, some may have undertaken other self-directed interventions that were not accounted for as the diet changes themselves were not recorded. In addition, medication changes by the patients' other health care providers that could alter urinary parameters may have occurred after the initial evaluation that were also not captured in our data set. Furthermore, as all patients received some type of dietary counselling, there was no comparison group of patients who did not receive any counselling. Longer-term follow-up will also be required to ensure the dietary changes are sustained. It has been shown that ongoing compliance requires a commitment and adherence to lifestyle changes which can be challenging [30].

Conclusions

Excessive dietary Na intake is a major health concern, linked to many adverse health outcomes including recurrent urinary stone formation. Urinary stone prevention strategies can include both diet and lifestyle modifications as well as medical prophylaxis. In this study we demonstrated elevated urinary Na is a common finding in stone formers and is frequently seen in association with hypercalciuria and other metabolic risk factors. Detailed counseling provided by either the urologist, nephrologist, or a registered dietician regarding Na intake can be associated with normalizing both elevated urinary Na and Ca levels in patients and may reduce the need for pharmacotherapy for the treatment of hypercalciuria in some patients.

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