

# Diet Interventions for Calcium Kidney Stone Disease

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## ABSTRACT

Kidney stone disease is a common condition with an increasing prevalence. Diet is an important, modifiable risk factor of an individual's risk of developing kidney stone disease, particularly for those without genetic causes of kidney stone disease. Prospective and epidemiological evidence suggest that adequate fluid intake, limited sodium ingestion, and sufficient calcium and potassium intake can decrease the risk of developing kidney stones. Metabolic risk factors for KSD found on 24-hour urine studies can be used to tailor dietary modifications recommended to reduce subsequent risk of kidney stone formation.

**KEYWORDS:** calcium kidney stones; nephrolithiasis; nutrition

## INTRODUCTION

Kidney stone disease (KSD) is a common condition in industrialized nations, impacting up to 15% of males and 5% of females.<sup>1</sup> The treatment of symptomatic kidney stone disease has significant morbidity and economic cost.<sup>2</sup> As the prevalence of KSD is rising, likely related to changing dietary and lifestyle factors, these burdens are growing.<sup>3</sup> With approximately four of five kidney stones being calcium-based, interventions aimed at reducing the risk of developing, or re-developing, these stones seems to hold the most promise at reducing these burdens. In this review, we will address diet as a modifiable risk factor in calcium-based KSD.

## RISK FACTORS FOR CALCIUM KIDNEY STONE FORMATION

The urinary solubility of the constituent parts of calcium oxalate and calcium phosphate stones is what helps determine the risk of developing them. This solubility can be estimated using a computer-based equation to determine the supersaturation for each compound and is determined by factors such as the urinary concentration of the compounds' ions; the pH of the urine; and the concentration of inhibitors of crystal formation, such as citrate. Data from large, United States based epidemiologic studies, Nurses' Health Study (NHS) I and II as well as the Health Professionals Follow-up Study (HPFS), demonstrate an association of elevated supersaturation of

calcium oxalate and calcium phosphate with stone risk in all sexes; in women, there is also an increased association of KSD and elevated uric acid supersaturation.<sup>4</sup> Calcium oxalate stones tend to form in acidic urine (pH 5) whereas calcium phosphate stones, composed of brushite and/or hydroxyapatite, tend to form in more alkaline urine (pH 6 or higher). In patients with KSD, analysis of 24-hour urine collection for supersaturation of stone forming crystals and other nutrients is recommended by the American Urologic Association (AUA) to assist in creation of a therapy plan.<sup>5</sup>

## FLUID INTAKE

Of any dietary intervention, the strongest data associating diet and kidney stone risk is in the consumption of fluids without added sugar. Mechanistically, the greater the fluid consumption, the more solvent (urine) exists for the solute (calcium, oxalate, phosphate, etc.) to be diluted in, decreasing the concentration of the solute. This decreased solute concentration necessarily reduces the supersaturation, decreasing the risk of subsequent kidney stone development. This principle has been demonstrated across several studies.

A meta-analysis of studies examining dietary, fluid, and supplement intake amongst those with a known history of any nephrolithiasis found increased water intake of at least 2–2.5 liters daily decreased KSD recurrence risk by at least 60%.<sup>6</sup> A small, prospective study of patients with documented calcium kidney stone disease randomized subjects to either no additional treatment or counseled to consume enough water such that urine output was at least two liters daily. Those who consumed the high water volumes were found to have significant decreases in the supersaturation of calcium oxalate, brushite, and uric acid as well as a significantly decreased risk of kidney stone recurrence over the five year study period.<sup>7</sup> Analysis of subjects from HPFS and NHS studies found total urine volume, a marker of higher fluid intake, was inversely associated with risk of kidney stone disease.<sup>8</sup> Similarly, subjects from the large United Kingdom-based epidemiologic study, the UK Biobank, with the highest fluid intake had the lowest risk of kidney stone development.<sup>9</sup>

The type of fluid ingested may also be important in modifying stone risk. Data from the UK Biobank suggested that those who consume more tea, coffee, and alcohol had

decreased kidney stone development risk, although there was interestingly no association between water intake and kidney stone risk.<sup>9</sup> Using data from the HPFS of men with no history of kidney stone disease at the start of the study, the authors found consuming coffee, tea, beer, and wine to be associated with decreased risk of developing kidney stones over the subsequent six years whereas regular consumption of apple juice and grapefruit juice may have increased it.<sup>10</sup> Data from the populations of the NHS I & II and the HPFS demonstrated that those who consumed the most sugar sweetened beverages, such as cola and punch, had a significantly increased risk of kidney stone formation as compared to those who consumed the least of these items.<sup>11</sup> A meta-analysis of men with a history of kidney stone disease found a significant association between reduction in soft drink intake and decreased risk of kidney stone recurrence.<sup>6</sup>

In the setting of the data above, the AUA recommends that all patients with a history of kidney stones ingest enough fluid to allow for a urine output of at least 2.5 liters per day.<sup>5</sup> The European Association of Urology (EAU) similarly advises consumption of 2.5–3 L fluid daily, of which water is the preferred fluid, so that the daily urine output is 2–2.5 L daily.<sup>12</sup>

## HYPERCALCIURIA

Urinary calcium concentration is impacted by intake of several different nutrients. Dietary sodium, potassium, and citrate can all play roles in modulating urinary calcium excretion.

### Sodium

In the nephron 80–85% of the filtered calcium load is reabsorbed in the proximal tubule and loop of Henle, largely via passive transport set up by sodium chloride and water reabsorption.<sup>13</sup> Thus, the more sodium reabsorbed, the more calcium will be reabsorbed, decreasing urinary calcium excretion. A major driver of proximal tubule sodium reabsorption is extracellular volume state. In states of volume depletion, more sodium is reabsorbed to expand extracellular volume, and in states of volume excess, less sodium is reabsorbed. Thus, a diet high in sodium, leading to expanded extracellular volume, will enhance urinary sodium and, therefore, calcium excretion. This physiologic principle has been practically demonstrated in several studies.

In a small study of patients without hypercalciuria, 24-hour urinary calcium excretion was found to be positively correlated with dietary sodium intake and independent of intestinal calcium absorption.<sup>14</sup> A retrospective analysis of the impact of one week of a combined low sodium and low calcium diet in patients with recurrent calcium oxalate stones found significant reduction in urinary sodium and calcium were achieved with these dietary changes.<sup>15</sup> Thirdly, a short, prospective study of patients with a documented history

of calcium KSD in the setting of hypercalciuria tested the impact of 2–3 L water daily versus 2–3 L water plus a diet where table salt and high sodium foods were restricted. After three months, those on the low sodium diet had greater reductions in urinary calcium and oxalate.<sup>16</sup> Notably, subsequent stone formation was not directly measured in any of these studies.

Given the association of sodium-restricted diets and decreased risk of KSD, both the AUA and EAU have recommended that patients with kidney stone disease limit sodium intake, although to different degrees. The AUA recommends a target of no more than 2,300 mg (100 mEq) sodium intake per day.<sup>5</sup> The EAU, citing the absence of robust prospective clinical trials of sodium reduction and its relationship to calcium kidney stone disease, recommend dietary sodium intake to not exceed a higher value of 3–5 g daily.<sup>12</sup>

### Potassium

Increases in dietary potassium leading to decreases in urinary calcium has been observed in several studies; however, the exact mechanism has not yet been elucidated. In a small but elegant study, healthy volunteers were each given standardized diets and supplemented with sodium chloride, sodium bicarbonate, potassium chloride, and potassium bicarbonate over separate periods. As expected, urinary calcium increased during times where sodium chloride supplements were provided. However, sodium bicarbonate, potassium chloride, and potassium bicarbonate all led to decreased urinary calcium excretion, with the decrease in urinary calcium greater for each of the potassium salts, suggesting that potassium independently decreases urinary calcium.<sup>17</sup> Furthermore, a small, prospective study of postmenopausal women designed to assess the impact of supplemental potassium citrate on sodium driven hypercalciuria found that potassium citrate was able to prevent the increase in urinary calcium driven by a high sodium diet.<sup>18</sup> Data from NHS and HPFS both have found that increased potassium intake is associated with decreased risk of KSD.<sup>19,20</sup>

## HYPOCITRATURIA

Citrate is a powerful inhibitor of calcium-based kidney stone formation, working via multiple mechanisms. Citrate creates relatively soluble calcium-citrate complexes in the tubule, decreasing the concentration of free calcium available for crystal formation with oxalate or phosphorus. Citrate also prevents aggregation of both calcium oxalate and calcium phosphate crystals that have formed through binding to the crystal surface itself.<sup>21,22</sup> Furthermore, citrate, when converted to bicarbonate, reduce bone resorption and enhances renal calcium reabsorption in the distal nephron.<sup>23</sup> In the diet, citrate is found in fresh fruits and vegetables, with citrus fruits – in particular lemons – being excellent sources of citrate.<sup>21</sup>

In a study using both simulated and natural urine, higher lemon juice concentrations were found to have a dose dependent inhibitory effect on calcium oxalate crystallization.<sup>24</sup> In human subjects, patients with hypocitraturia and history of calcium oxalate KSD were treated with potassium citrate in effort to restore normal urine citrate concentration and increase urinary pH. Over an average of 2.2 years, only 11% had recurrence of kidney stones with this treatment.<sup>25</sup> Another small study of patients with calcium KSD and hypocitraturia were trialed on lemonade therapy – 4 ounces of lemon juice diluted in water to create 2L lemonade – in lieu of pharmacotherapy. These patients all had increased urinary citrate and non-significant decrease in urinary calcium with similar urine volumes as compared to before the intervention.<sup>26</sup>

A study randomizing subjects to the addition of 2 ounces of lemon juice twice daily to 2–2.5 L water intake or no addition found that those taking lemon juice had decreased kidney stone recurrence after one year. This benefit was not seen after two years of the study in the setting of low adherence (48%) to the lemon juice at year two.<sup>27</sup> Thus, for patients preferring to avoid pharmacotherapy, or in those experiencing untoward gastrointestinal side effects of pharmacotherapy, lemonade therapy may be a reasonable option to pursue.

As the addition of citrate may lead to urinary alkalinization, there is a theoretical concern of increased calcium phosphate crystallization at higher urine pH. In a small, prospective cross-over study examining the impact of potassium citrate and citric acid on stone risk among calcium phosphate stone formers, Doizi et al failed to find any difference in urinary stone risk parameters among the three study groups.<sup>22</sup> Thus, citrate supplementation appears to be safe even among calcium phosphate stone formers.

### Animal Protein

Consumption and subsequent breakdown of animal protein increases daily titratable urinary acid load leading to hypocitraturia.<sup>28</sup> Indeed, the data from the HPFS suggests that the amount of animal protein consumed has a positive correlation with risk of developing symptomatic KSD.<sup>20</sup> Additionally, a small, prospective cross-over study suggests the type of animal protein does not matter with regards to kidney stone risk. Subjects on a standard diet with meat consisting of beef, chicken, or fish did not have any significant difference on urinary uric acid, pH, citrate, or oxalate content despite higher serum uric acid during periods on the chicken and fish diets.<sup>29</sup>

To reduce KSD risk, the AUA recommends patients with calcium stones and low urinary citrate to increase fruit and vegetable consumption and decrease intake of non-dairy animal protein.<sup>5</sup>

### HYPEROXALURIA

Oxalate is absorbed in the gut in its soluble form and excreted in stool when in its crystalline, calcium oxalate form. Additionally, there may be some degree of the gut microbiota degrading oxalate as an energy source, decreasing its absorption. Increased soluble oxalate, be it from diet, increased enteric absorption in the setting of malabsorptive states such as following Roux-en-Y bypass, and increased endogenous production all increase the filtered load, and directly lead to an increased supersaturation of calcium oxalate in the urine.<sup>30</sup> Although there are no studies demonstrating an association of decreased oxalate ingestion and decreased KSD risk, both the AUA and EUA recommend patients with calcium oxalate KSD and hyperoxaluria, particularly enteric hyperoxaluria, limit dietary oxalate intake.<sup>5,12</sup>

### Calcium

Calcium ingested during a meal can complex with oxalate, leading to formation of non-absorbable crystalline calcium oxalate. However, if taken away from a meal, such as during calcium supplementation, a larger proportion of the ingested calcium and oxalate can be absorbed, leading to a greater delivery of calcium to the nephron. This was demonstrated in a small, prospective study of healthy male volunteers given 3g calcium carbonate supplementation daily, either as 3g at bedtime or 1g thrice daily with meals. Although both protocols increased urinary calcium excretion, those who took the supplement with meals had significantly decreased urinary oxalate compared to those with increased calcium oxalate supersaturation when taking the supplement at bedtime.<sup>31</sup>

A randomized, prospective trial of 120 men with history of calcium oxalate nephrolithiasis and hypercalciuria compared low calcium or normal calcium diets added to a decreased sodium and low animal protein diet. Those on the normal calcium diet had approximately a 50% reduction in risk of kidney stone recurrence.<sup>32</sup> Data from the NHS and HPFS also suggests an inverse association between dietary calcium and kidney stone risk.<sup>19,20,33</sup> However, supplemental calcium use was associated with a higher risk of kidney stones in older women, perhaps due to timing of the intake being away from dietary oxalate ingestion.<sup>19</sup> Interestingly, supplemental calcium was not associated with increased risk of KSD in younger women.<sup>33</sup>

The AUA also recommends dietary calcium intake for 1,000–1,200 mg daily with the caveat that calcium should be ingested at meals.<sup>5</sup> The EAU advises dietary calcium intake of 1,000–1,200 mg daily. They do not advise supplemental calcium except in the setting of enteric hyperoxaluria.<sup>12</sup>

### Vitamins

Vitamin B6 and vitamin C both impact total body oxalate metabolism. Vitamin B6 decreases oxalate production

whereas vitamin C can be metabolized into oxalate, therefore increasing the serum concentration.<sup>34</sup>

In analysis of women in the NHS, those who took at least 40 mg daily vitamin B6 were associated with decreased subsequent kidney stone risk.<sup>34</sup> In analysis of the NHS and HPFS, vitamin C intake was not found to be associated with increased risk of kidney stones in females but was in males.<sup>34,35</sup> Specifically, the increased risk was found in those who consumed supplements of vitamin C, particularly at doses over 700 mg daily, as high levels of diet-derived vitamin C did not lead to increased risk. Although the AUA considers vitamin C data controversial,<sup>5</sup> EAU advises those with calcium oxalate KSD avoid “excessive” vitamin C intake.<sup>12</sup>

## DIETARY SUPPLEMENTS

### Probiotics

Over the past several years, the increasing work examining the relationship between disease states and the composition of the gut microbiota has included several studies aimed at KSD risk factors. Small studies have demonstrated an association between certain gut bacteria and urinary citrate and oxalate.<sup>36,37</sup> However, manipulation of the microbiome with probiotics to promote bacteria with favorable associations has yet to be proven clinically effective. In a small, prospective trial of patients with calcium oxalate KSD and hyperoxaluria, two different probiotic preparations did not result in a change in urinary oxalate excretion or calcium oxalate supersaturation whereas a restriction of dietary oxalate to 100 mg daily did.<sup>38</sup> Another small, prospective study using probiotics in patients with hyperoxaluria similarly did not lead to changes in urinary oxalate excretion after four weeks of use.<sup>39</sup>

## CONCLUSION

For calcium kidney stone prevention, it is generally recommended to maintain adequate oral hydration while avoiding sugar sweetened drinks, to restrict dietary sodium and animal protein intake, and to optimize dietary potassium and citrate intakes. We should also emphasize that dietary interventions should be individualized based on patient’s medical history and urinary stone risk profiles.

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