HHS Public Access

Author manuscript

Urolithiasis. Author manuscript; available in PMC 2016 November 18.

Published in final edited form as:

Urolithiasis. 2016 February ; 44(1): 27–32. doi:10.1007/s00240-015-0839-4.

Lowering urinary oxalate excretion to decrease calcium oxalate stone disease

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Abstract

Dietary modifications should be considered as a first line approach in the treatment of idiopathic calcium oxalate nephrolithiasis. The amounts of oxalate and calcium consumed in the diet are significant factors in the development of the disease due to their impact on urinary oxalate excretion. There are a number of strategies that can be employed to reduce oxalate excretion. The consumption of oxalate-rich foods should be avoided and calcium intake adjusted to 1000–1200 mg/day. To encourage compliance it should be emphasized to patients that they be vigilant with this diet as a deviation in any meal or snack could potentially result in significant stone growth. The evidence underlying these two modifications is outlined and other strategies to reduce urinary oxalate excretion are reviewed.

Introduction

Calcium oxalate stones will only develop when calcium oxalate crystals form in the urinary tract due to the supersaturation of the fluid with calcium and oxalate ions. With this premise in mind it leads to the notion that the formation of stones could be prevented if the oxalate concentration is lowered sufficiently. This is safe and feasible given that oxalate does not appear to form any functional role in humans and is derived from the diet and as an unneeded by-product of metabolism. A case–control comparison of the urinary oxalate excretion in stone formers and controls revealed that a difference of 5 mg of oxalate could be associated with an approximate 70–100 % increase in stone risk in some groups [1].

Why is it that a clear path for eliminating calcium oxalate stones has not emerged despite many decades of research? The reasons for this impasse are twofold: (1) there are currently no established medical therapies or dietary protocols that will decrease the endogenous synthesis of oxalate, and (2) it is virtually not possible to completely eliminate oxalate from the diet or prevent its intestinal absorption. While elimination of stones may not be possible at the moment by reducing urinary oxalate to a very low level, some experimental studies

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Conflict of interest The authors declare no conflict of interest.

Ethical approval All studies conducted by the investigators involving human participants were in accordance with the ethical standards of the Institutional Review Boards.

Informed consent Informed consent was obtained from all participants in studies conducted by the investigators.

have shown that the absorption of dietary oxalate can be lowered to levels that should decrease stone recurrence. Furthermore, hope is emerging that novel therapeutic strategies that decrease the expression of key enzymes can attenuate endogenous oxalate synthesis. We will review these developments, suggest ways current approaches can be improved, and offer insight into new therapeutic pathways that may emerge.

Unique handling of oxalate in the body

The major ions that contribute to the formation of calcium stones are calcium, phosphate, oxalate and citrate. Oxalate is unique amongst these ions in that the amount absorbed and excreted in urine is under little biological control. In contrast calcium and phosphate are subject to hormonal regulation in their absorption and excretion whereas the excretion of citrate is largely determined by renal transport and metabolism. Gastrointestinal oxalate absorption is not known to be subject to regulation and compared to the other ions its absorption is low and <15 %. The oxalate absorbed is practically all excreted in urine. A small portion (<10 %) may be secreted back into the intestine [2] and if plasma oxalate levels rise above 4 μM, renal secretion of oxalate is thought to be stimulated [3].

A much overlooked factor in calcium oxalate stone disease is that the growth of kidney stones is not likely to be constant, but a process that responds to transient sharp increases in oxalate concentration. It is likely to occur when the kidney is flooded with stone-forming salts 3–6 h after a meal and calcium oxalate crystals form. Ingesting a spinach salad which is known to have a high oxalate and low calcium content once a month, for instance, could result in substantial stone growth over a 12-month period. This risk for rapid stone growth associated with the infrequent ingestion of high-oxalate foods may have contributed to the inability of the food frequency questionnaire analysis to adequately identify the actual magnitude of stone risk associated with dietary oxalate. However, despite these limitations, a positive association between kidney stone risk and the consumption of oxalate rich foods has been demonstrated in two large epidemiologic cohorts [4].

Dietary oxalate and intestinal oxalate absorption

We have shown that dietary oxalate makes an important contribution to urinary oxalate excretion [5]. We estimated that on average it contributed 40–50 % of the oxalate excreted. This is now widely accepted but its role in stone formation is still not fully appreciated for several reasons. Intake of dietary oxalate can be difficult to determine due to conflicting incomplete food oxalate tables, large differences in the oxalate content of foods as a result of varietal differences and climate effects, and inherent errors in estimating food intake using dietary records, dietary recall or food frequency questionnaires. These issues are compounded by complex and variable food combinations in recipes. These factors can also make it challenging to control dietary oxalate in experimental studies. To counteract these influences in dietary oxalate studies we measure the oxalate content of all food used as well as determining the oxalate content of a homogenate of all of the food eaten on a particular day. The intake of calcium has to be controlled as it will influence oxalate absorption as discussed below. The absorption of dietary oxalate in the gut is also quite variable for reasons not well understood [6]. This absorption appears to vary extensively both within and

between individuals receiving soluble oxalate loads. Another consideration is the bioavailability of oxalate in food. If foods contain more soluble (non-crystalline oxalate), a greater portion of this oxalate will be absorbed through the gastrointestinal tract and this will augment oxalate excretion [7].

Dietary calcium and urinary oxalate excretion

The amount of dietary calcium in the diet has a significant effect on urinary oxalate excretion, which increases when calcium intake is low and decreases when calcium ingestion is elevated. Lemann and associates reported that daily urinary oxalate declined as dietary calcium increased in normal subjects, although dietary oxalate was not controlled [8]. We have also confirmed that urinary oxalate excretion is higher when calcium intake is low (400 mg/day) in healthy adults consuming a carefully controlled metabolic diet [5]. This effect of dietary calcium is hypothesized to be due to the genesis of crystalline calcium oxalate in the intestinal lumen as dietary calcium increases, thus limiting intestinal oxalate absorption and reducing its urinary excretion. In contrast, more oxalate will be soluble with diminished calcium intake, thus augmenting net oxalate gastrointestinal absorption and urinary oxalate excretion. The influence of daily calcium intake on oxalate absorption was further studied by von Unruh et al. who assessed the absorption of a soluble oxalate load with varying amounts of daily calcium intake (200, 360, 600, 1200 and 1800 mg/day) in healthy subjects [9]. The calcium sources for the lower calcium intake sequences were dietary while those for intakes of 1200 and 1800 mg were from both diet and supplements. There was a linear inverse relationship between daily calcium intake and oxalate absorption which ranged between 1.7 and 16.9 %.

Oxalate synthesis in the body

Oxalate is generated in the liver and to lesser extent the kidney from the metabolism of the amino acids, hydroxyproline, glycine, phenylalanine and tryptophan, and the dialdehyde, glyoxal [10–13]. It is possible that systemic fluctuations in hormones and metabolite concentrations influence these synthetic pathways as some of our studies in experimental animals have shown. Glucagon and alanine are two such potential modifiers we have identified [14, 15]. Our studies on urinary oxalate excretion on a zero-oxalate diet show that moderate fluctuations in endogenous oxalate synthesis can occur [16]. The mean intra-individual coefficient of variation (CV) in 24 h oxalate excretion on days 3–5 of the diet was 15.8 % and the inter-individual CV 23.5 %.

Vitamin C can breakdown to oxalate in tissues as it performs its antioxidant function. An association between vitamin C intake and kidney stone formation was reported by Taylor et al. in a prospective study of ~50,000 male health professionals [17]. Men with an intake >218 mg/day had a 31 % higher risk of forming stones than those consuming <105 mg/day ($P = 0.01$). Supplemental vitamin C also increased stone risk and an intake >1000 mg/day increased risk by 16 % compared to those not taking supplemental vitamin C. In a similarly sized study in Swedish males, supplemental vitamin C intake was associated with a nearly twofold increase in stone risk [18]. This risk appeared to be associated with the number of supplemental tablets taken each day. These two large studies support vitamin C intake as a

significant risk factor for the development of kidney stones. Support for this effect being due to oxalate formation was provided by Taylor and Curhan in epidemiological studies of stone formation in male health professionals and female nurses [19]. They reported that vitamin C had the greatest effect on urinary oxalate excretion. Compared with participants consuming <90 mg/day, participants with an intake >1000 mg/day had a urinary oxalate excretion that was 6.8 mg/day higher when adjusted for confounding variables. Others have reported that the consumption of 1 g or more can significantly increase urinary oxalate excretion and even lead to oxalate nephropathy [20–22]. These results suggest that calcium oxalate stone formers should cease taking supplemental vitamin C and should decrease their intake of vitamin C-rich foods.

Decreasing dietary oxalate lowers urinary oxalate excretion

Our studies with normal subjects consuming diets controlled in their oxalate and calcium contents have indicated that an intake of food-derived oxalate in the range of 100–750 mg/day results in an increase of 2 mg of urinary oxalate per 100 mg of oxalate consumed [5, 23]. Our studies with diets containing less than 100 mg oxalate per day further showed that a larger percentage of the dietary oxalate was absorbed, presumably because much more of it was present as the soluble oxalate anion [5]. With a diet of ultra-low oxalate content (10 mg/day) it was estimated that 25 % (2.5 mg) was absorbed. Calcium oxalate stone formers appear to have a similar response to dietary oxalate. Lieske et al. reported that stone formers with hyperoxaluria had a 36 % reduction in urinary oxalate excretion when they consumed a low oxalate (80–100 mg/day) and normal calcium (1000 mg/day) diet [24].

Dietary calcium lowers oxalate absorption

The impact of calcium intake on oxalate absorption and oxalate excretion previously profiled was mainly in normal subjects and with soluble oxalate loads.

We previously showed that this held true with food-derived oxalate but used a surrogate for oxalate absorption, contribution of dietary oxalate to the urinary oxalate pool. This study was also done in healthy, non-stone forming adults [5]. There is inferential evidence that this relationship holds true in stone formers. In the dietary study of Borghi and associates, urinary oxalate excretion increased 11 % over the 5 year follow-up in the low calcium (400 mg/day) cohort and it decreased 19 % over this interval in the normal calcium (1200 mg/day), low protein/salt group [25]. This study was conducted in recurrent, male, calcium oxalate stone formers with hypercalciuria. The oxalate consumption was thought to be similar in both groups as they were advised to restrict their intake of oxalate-rich foods. The same group assessed the benefits of a normal calcium, low sodium/animal protein diet in calcium oxalate stone formers with hyperoxaluria. Daily oxalate intake was modest before this intervention and during it, 143 and 121 mg. They reported a 7.3 mg/day adjusted decline in urinary oxalate excretion with this dietary intervention. The aforementioned studies clearly suggest that strategies to reduce urinary oxalate excretion through dietary modifications must be at least dually controlled with a reduction in oxalate intake and maintenance of normal calcium consumption.

Colonization with *Oxalobacter formigenes* decreases oxalate absorption and promotes intestinal oxalate secretion

Oxalobacter formigenes is an intestinal organism that is unique in relying on oxalate as a carbon source for energy and growth [26, 27]. In Western societies it appears to colonize the gut of 30–40 % of the population [28]. Stone formers are colonized at approximately half of this rate and those not colonized are 70 % more likely to develop a kidney stone [28]. Its mode of action appears to be twofold. Firstly, the degradation of oxalate in the intestine decreases its absorption particularly when calcium intake is low [23, 29]. Secondly, it may promote oxalate secretion into the gut [2]. We have previously shown in two individuals that they can be colonized by ingesting live organisms [30]. Furthermore, four individuals showed a 37–40 % reduction in oxalate excretion in a 6 h period after ingesting *O. formigenes* with an oxalate load. These results suggest that colonizing stone forming individuals that are non-colonized could be an inexpensive way of decreasing stone risk, particularly that associated with oxalate ingestion. The biopharmaceutical company, Oxthera (www.oxthera.com) is currently conducting a clinical trial in patients with primary hyperoxaluria to determine whether consuming large doses of *O. formigenes* will decrease urinary oxalate excretion.

Other strategies to lower urinary oxalate excretion

Other methods have been utilized in an attempt to reduce oxalate excretion. One study demonstrated that the administration of a lactic acid bacterial probiotic reduced oxalate excretion in patients with idiopathic hyperoxaluria [31]; although this has not been substantiated by others [24]. The administration of fish oil should be further explored to confirm its effects and mode of action [32]. Pyridoxine therapy has been recommended for patients with hyperoxaluria. However, it has only been demonstrated to be beneficial in certain patients with type 1 primary hyperoxaluria [33]. Its utilization in patients with idiopathic hyperoxaluria has not been substantiated with well-designed studies [34]. In theory, it could benefit such patients as it may promote the activity of alanine glyoxylate aminotransferase, which diverts glyoxylate away from oxalate synthesis or potentially stimulates other aminotransferases.

Blocking the synthesis of oxalate in metabolic pathways is an obvious therapeutic approach for decreasing urinary oxalate excretion in individuals with calcium oxalate stone disease. The pharmaceutical company Merck invested substantial amounts in developing inhibitors of glycolate oxidase, a liver-specific enzyme that converts glycolate to glyoxylate, the immediate precursor of oxalate [35, 36]. More recently this enzyme has re-emerged as a target for therapeutic siRNAs to block oxalate synthesis in the rare disease, primary hyperoxaluria. More information on this approach can be obtained at company websites (www.alnylam.com and www.dicerna.com). As of this date, however, no medical therapies other than pyridoxine supplementation exist that will decrease endogenous oxalate synthesis. We have suggested that hydroxyproline dehydrogenase is good therapeutic target for blocking oxalate synthesis resulting from hydroxyproline breakdown in individuals with primary hyperoxaluria [37]. We also anticipate that such a therapeutic approach will also be

effective in reducing idiopathic calcium oxalate stone formation. In addition, limiting consumption of hydroxyproline-rich foods may prove to be beneficial.

Increasing the intake of magnesium to bind oxalate in the gut has also been considered as a way to decrease oxalate absorption. Magnesium does decrease the absorption of oxalate provided as a soluble oxalate load [38], but a randomized clinical trial in stone patients did not see any effect of supplemental magnesium on stone formation [39].

Conclusions

There are many approaches that can be taken to reduce urinary oxalate excretion that are outlined in Table 1. Decreasing the amount and concentration of oxalate in urine is an effective strategy for decreasing the recurrence of calcium oxalate stone disease with appropriate patient compliance. The simplest approach for stone formers to implement this strategy is to avoid oxalate-rich foods and to increase their consumption of calcium with each meal. This strategy was endorsed in the American Urological Association Guidelines document on medical and dietary prevention of kidney stones [40]. Drinking more fluids will also decrease the oxalate concentration in urine. For maximal effects using food tables and other nutrient sources to adjust oxalate intake to <100 mg and calcium to 1000–1200 mg/day is advised. Future advances may include ensuring colonization with *O. formigenes* and the development of medical therapies that attenuate endogenous oxalate synthesis.

Acknowledgments

Funding This work was supported by NIH grants DK62284, DK73732 and DK87967.

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Table 1

Recommendations for lowering urine oxalate

Recommendation	How to accomplish
Decrease oxalate intake	Avoid or limit oxalate-rich foods including spinach, rhubarb, beets, nuts, chocolate, wheat bran, sesame seeds Consult table of food oxalate content https://regepi.bwh.harvard.edu/health/Oxalate/files/Oxalate%20Content%20of%20Foods.xls
Consume 1000–1200 mg calcium/day	Note the calcium content of dairy products and other calcium-enriched foods usually consumed. Ensure that a calcium-rich food is consumed with a meal known or suspected to contain significant amounts of oxalate
Determine whether colonized with *O. formigenes*	Not easily accomplished as yet. Wait for commercial development of a test to determine colonization status and procedure for colonization
Decrease vitamin C consumption	Avoid supplements and excessive intake of vitamin C-rich products such as orange juice
Decrease urinary oxalate concentration	Increase fluid intake and decrease dietary oxalate absorption by the above strategies