Review Article

Calcium intake and urinary stone disease

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Abstract: Calcium homeostasis is a complicated and incompletely understood process that is primarily regulated through an interaction between the intestines, kidneys, and bones. Intestinal calcium absorption is determined by many factors including the amount of regular calcium intake, as well as vitamin D and parathyroid hormone levels. Intestinal calcium absorption is likely different between stone formers and non-stone formers, with higher levels of calcium absorption in those with a history of stones independent of their calcium intake. We no longer recommend dietary calcium restriction as this may lead to bone demineralization and an increase in stone formation. Practitioners need to continue to educate patients to maintain moderate dietary calcium intake. The effect of calcium supplementation on stone formation is currently controversial. It is likely that large doses of supplemental calcium, especially if taken separate from a meal, may lead to stone formation. When necessary, stone forming patients should be encouraged to take their calcium supplements with a meal and their stone disease should be monitored.

Keywords: Intestinal calcium absorption; hypercalciuria; dietary calcium intake; calcium supplementation; kidney stones; nephrolithiasis

Submitted Mar 25, 2014. Accepted for publication Jun 16, 2014.
doi: 10.3978/j.issn.2223-4683.2014.06.05
View this article at: http://dx.doi.org/10.3978/j.issn.2223-4683.2014.06.05

Introduction

As 80-90% of kidney stones are composed of calcium in the form of either calcium oxalate or calcium phosphate (1), excess calcium excretion in the urine has been a primary focus in preventing stone recurrence (2). Calcium metabolism and calcium homeostasis are complex processes that involve multiple organ systems. A thorough understanding of the interaction between the various players in calcium homeostasis is critical to understanding the abnormalities and dysregulation that can arise when calcium is not managed appropriately by the intestines, vitamin D and parathyroid hormones, bones, and the kidneys. Calcium intake is perhaps one of the most misunderstood factors related to urinary stone formation and there is controversy as to whether there are differences in the effect of dietary and supplemental calcium on kidney stone formation.

Intestinal calcium absorption

The ingestion of calcium is the only route by which humans can acquire this important element (3). About 30-40% of dietary calcium is absorbed from the gastrointestinal tract with the majority occurring in the small intestine (4). The amount of calcium absorbed in the intestine depends on habitual calcium intake. When calcium intake is low, calcium is actively transported in the duodenum and a larger portion of calcium is absorbed by an active process leading to a greater fractional absorption of calcium (5,6). The active, transcellular process of calcium absorption is regulated by calcitriol [1,25-$(OH)_2$-vitamin D_3] (7,8). When calcium intake is high, passive calcium absorption is the primary absorptive process occurring in the jejunum and ileum leading to a lower fractional absorption of calcium (4-6,9). Thus the process of adaptation of the intestines allows the fractional absorption of calcium to vary widely between individuals, ranging from 10-70% (10-12).

Many factors influence calcium absorption including age, gender, race, pregnancy/lactation/menopausal status, obesity, the timing and amount of calcium taken, vitamin D and parathyroid hormone levels, and the other nutritional

factors consumed with the calcium (3). Calcium is absorbed in the ionic state and thus intake of calcium with compounds that complex calcium such as oxalate, phosphate, sulfate, citrate, fiber, and fats reduce the bioavailability of calcium for potential absorption (3,13).

Vitamin D is the major regulator of intestinal calcium absorption and is obtained from either the diet or exposure to sunlight (14). Vitamin D_2 and D_3 are ultimately converted to the physiologically active calcitriol [1,25-$(OH)_2$-vitamin D_3] which is the most potent stimulator of intestinal absorption of calcium. Calcitriol also acts on the bone, along with parathyroid hormone, to promote bone demineralization by stimulating osteoclasts (13). Parathyroid hormone does not directly affect intestinal calcium absorption, but it stimulates synthesis of calcitriol, which leads to enhanced intestinal calcium absorption.

Intestinal calcium absorption likely plays a role in kidney stone formation. Patients with excess calcium losses in the urine tend to have higher intestinal calcium absorption (15,16). Radioactive calcium isotopes have been used to evaluate the relationship between intestinal fractional calcium absorption and elevated urinary calcium levels (17-19). Recently, we evaluated data from the prospective multicenter study of osteoporotic fractures where more than 5,400 women underwent oral radioactive calcium assay to evaluate intestinal calcium absorption (20). In this study, fractional calcium absorption was independently associated with nephrolithiasis. Women with a history of kidney stones tended to have higher fractional calcium absorption at each level of dietary and supplemental calcium intake. Also, increasing calcium intake was associated with lower fractional calcium absorption, with no difference between dietary or supplemental calcium sources.

It is therefore likely that calcium absorption differs between stone formers and non-stone formers. This may be due to primary difference in baseline intestinal absorption of calcium, leading to greater calcium absorption and then greater urinary excretion and subsequent stone formation. On the other hand, differences in intestinal calcium absorption are very closely related to calcium intake, and thus it is possible that the differences in calcium absorption between stone formers and non-stone formers are secondary to differences in past calcium intake.

Hypercalciuria

Hypercalciuria is the most common abnormality in calcium stone formers, occurring in 35-65% of subjects, and may lead to supersaturation of urinary calcium salts (21-24). Several mechanisms can lead to hypercalciuria, as calcium homeostasis is primarily regulated through a complex interrelated interaction between the intestines, kidney, and bones (25,26). Historically, research from Pak *et al*. in 1974 classified the type of hypercalciuria based on the organ system responsible including intestinal absorptive, renal leak, or parathyroid resorptive hypercalciuria (27). Hypercalciuria not explained by the classification scheme was determined to be idiopathic, a category which represents at least 50% of stone formers (25). Idiopathic hypercalciuric stone forming patients likely have a more generalized systemic abnormality with some degree of dysregulation of calcium homeostasis in the intestine, kidney, and bone.

The dysregulation of calcium balance is complicated. One might expect that patients with primary hyperparathyroidism might be the most straightforward. Nephrolithiasis formation in these patients is largely attributed to hypercalciuria (21,28-30). Elevated serum parathyroid hormone levels increase serum calcium levels due to increased intestinal absorption, bone resorption, and renal reabsorption of calcium (31). Despite the increased parathyroid hormone mediated renal reabsorption of calcium in the distal nephron, the excess serum calcium load overwhelms the ability of the kidney to reclaim calcium. Thus, one would expect to find hypercalciuria in all patients with primary hyperparathyroidism, increasing the risk of nephrolithiasis. However, only 20% of patients with primary hyperparathyroidism form stones (29), and up to 35% of stone forming patients with primary hyperparathyroidism do not have hypercalciuria (32). Though urinary calcium levels do decrease significantly after parathyroidectomy, there are no apparent differences in the magnitude of the decrease in stone formers and non-stone formers (32).

This example serves to demonstrate that our understanding of calcium homeostasis remains somewhat limited. While Pak's classification system potentially simplifies the causes of excess urinary calcium loses, it has not dramatically changed the understanding of the mechanisms of nephrolithiasis formation or changed the management of stone disease. It also has not lead to more effective prevention of stones, and thus, use of the classification system is not recommended in clinical practice (20,33,34).

Dietary calcium

For many years patients were advised to decrease their calcium intake in an attempt to limit the diet-dependent

intestinal absorptive hypercalciuria, as dietary calcium restriction was one of the mainstays of therapy for prevention of stone recurrence (35,36). Large, prospective observational studies were the first to demonstrate the potential risks of a low calcium intake. Using data from more than 45,000 men in the Health Professionals Follow-up Study, Curhan *et al*. were the first to demonstrate in their 1993 article that low dietary calcium intake potentially increased the risk of stones by more than 51% compared to men with the highest dietary calcium intake after adjusting for multiple potential confounders (37). These findings were later confirmed with a similar effect in both younger and older women in the Nurses Health Studies II and I respectively (38,39), and more recently in the women in the observational arm of the Women's Health Initiative (40). Dietary calcium intake is likely a protective factor against stone formation and this is likely the case whether dietary calcium comes from dairy or non-dairy sources (41).

The inverse relationship between low dietary calcium intake and an increase in stone formation is likely due to a secondary increase in urinary oxalate. Oxalate absorption occurs throughout the intestinal tract (42). When calcium and oxalate are consumed at the same meal, a calcium-oxalate complex forms within the intestinal tract limiting the intestinal absorption and subsequent urinary excretion of free oxalate (36,43). However, with dietary calcium restriction, free oxalate becomes increasingly available for intestinal absorption, leading to greater urinary excretion of oxalate (43-47).

This was ultimately tested in a randomized control trial in 2002 of men with hypercalciuria and a history of recurrent stones, where men were prescribed a low salt, low animal protein, moderate calcium (1,200 mg daily) diet and were compared to men prescribed a low calcium diet (400 mg daily) (48). Both groups were advised to decrease dietary oxalate intake and after 5 years of follow up the risk of stone recurrence was more than 50% lower in men on the normal calcium diet. While urinary calcium levels decreased in both groups, urinary oxalate levels increased in men on the low calcium diet and decreased in men on the normal calcium diet. Though it is suggestive, this study did not directly assess the independent effect of dietary calcium on urinary oxalate and stone recurrence as multiple dietary changes were made in both groups.

In addition, dietary calcium restriction may lead to bone demineralization. Patients with a history of kidney stones have an increased risk of bone mineral density problems and osteoporotic fractures, and these risks are likely compounded in patients with low dietary calcium intake (49,50). A population-based cohort study demonstrated that patients with a history of kidney stones had nearly a 4-fold increased risk of vertebral osteoporotic fractures (51). Cross-sectional data has demonstrated an association between dietary calcium restriction and decreased bone mineral density in stone formers (52).

As a result of the increased risk of stone formation and bone demineralization, dietary calcium restriction is now no longer recommended for patients with hypercalciuria (53). Almost 25 years later, the end result of such a focus on intestinal absorptive hypercalciura as a potential source for kidney stone formation, still leads many patients to intentionally decreasing their dietary calcium intake. As the prevalence of stone disease increases, continued efforts are needed to educate patients that modest dietary calcium intake between 800-1,200 mg daily is recommended for stone formers.

Calcium supplementation

The effect of calcium supplements on stone risk is currently controversial. As a part of the Women's Health Initiative Calcium and vitamin D randomized control trial, more than 36,000 postemenopausal women were randomized to receive placebo or 500 mg of calcium carbonate plus 200 units of vitamin D_3 twice daily (54). The mean total calcium intake for the women receiving calcium and vitamin D was 2,100 mg of daily calcium consisting of an average of 1,100 mg daily from dietary calcium plus the additional 1,000 mg of supplemental calcium. After 7 years of follow up, there was a 17% increased risk of stone formation in the women randomized to calcium and vitamin D. The lower end of the 95% confidence intervals approached the null hypothesis but was statistically significant (HR 1.02-1.34). Interestingly, when only the women with greater than 80% compliance were analyzed, the risk was similar in magnitude, but no longer statistically significant (55). The risk of stone formation was similarly increased (20%) among women in the Nurse's Health Study I observational study who reported taking supplemental calcium (39). In this observational cohort, 67% of the women taking calcium supplements took them separate from a meal or with a low-oxalate meal. Given the increased risk of stone formation, this may indicate that the relationship between calcium supplementation and a meal may be important.

In a separate study, postmenopausal women with a total calcium intake of over 2,400 mg daily, 800 mg/day from

diet and 1,600 mg daily from calcium supplementation, had significantly higher rates of hypercalciuria compared to subjects receiving placebo during this 1-year study, though no stone events occurred during the study (56). Thus, calcium intake at high amounts may increase urinary calcium levels.

In contrast to the studies above, prospective observational studies such as the Nurse's Health Study II, and the Health Professionals Follow Up Study demonstrated no increased risk of nephrolithiasis with calcium supplementation (37,38,57).

The timing of calcium supplementation is likely critically important. Whether obtained from dietary or supplemental sources, calcium present in the intestinal tract will bind oxalate leading to decreased oxalate absorption and subsequent urinary excretion. As calcium is always in relative excess in the urine, urinary oxalate is likely more important than even large increases in urinary calcium (43,58). To further evaluate the effect of timing of calcium intake, Domrongkitchaiporn *et al*. performed a randomized, crossover, diet controlled study in young, healthy, male navy privates where subjects received either 1,000 mg of calcium carbonate with three meals daily (3,000 mg total), or 3,000 mg of calcium carbonate at bedtime (59). Urinary calcium levels increased similarly between the two groups, but the urinary oxalate levels were significantly decreased when the calcium supplementation was taken with a meal. Despite the increase in urinary calcium, the protective effect on urinary oxalate prevented the supersaturation of calcium oxalate from increasing. The authors concluded that calcium supplements should be taken with meals in order to avoid increasing the risk of calcium oxalate nephrolithiasis.

These data suggest that calcium supplements, especially if medically necessary for the prevention or treatment of osteopenia and/or osteoporosis, should be taken with a meal, rather than between meals or at bedtime. Patients with a history of kidney stones, who are taking calcium supplements, should have their urine monitored when they begin this therapy and if the activity of their stone disease increases. Patients should be encouraged to use dietary calcium sources whenever possible, and if hypercalciuria does occur then the timing, type and dosage of calcium supplementation should be evaluated and the hypercalciuria treated if it is felt to be a contributing factor to stone formation.

Summary

Calcium is a very important mineral for cellular function. Calcium homeostasis is a complicated process which we do not completely understand. Dysregulation of calcium homeostasis is also complex and may lead to primary or secondary changes in intestinal calcium absorption. Alternatively, patients with a history of kidney stone formation may have abnormal intestinal calcium absorption either due to an inherent predisposition, or due to differences in calcium intake.

There is level 1 evidence that dietary calcium intake is a protective factor against stone formation. We no longer recommend dietary calcium restriction as it may lead to increased stone formation potentially through increased oxalate absorption, and may cause bone demineralization. Further efforts are needed to educate patients not to restrict calcium intake. The data on supplemental calcium intake is currently controversial. In cases where calcium supplementation is medically necessary, patients should be encouraged to take their calcium supplements with a meal and should be monitored for changes in the activity of their stone disease.

Acknowledgements

None.

Footnote

Conflicts of Interest: The author has no conflicts of interest to declare.

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Cite this article as: Sorensen MD. Calcium intake and urinary stone disease. Transl Androl Urol 2014;3(3):235-240. doi: 10.3978/j.issn.2223-4683.2014.06.05

