The Role of Diet in the Prevention of Common Kidney Stones

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Current dietary recommendations for patients who form kidney stones are discussed. Focusing on the most common kidney stone types, calcium oxalate and uric acid, the rationale for dietary changes are described based on the renal and urine physiology.

Introduction

Kidney stone formers may feel doomed to a life of unpredictable flank pain, periodic surgical intervention, and concomitant loss of work and daily pleasures. Indeed, if untreated, those who have formed one calcium oxalate stone have a 50% chance of forming additional stones within 10 years (Menon & Resnick, 2002). With appropriate education, patients can exercise some control over stone disease and reduce their chances of forming stones through dietary modifications and medication.

General dietary recommendations appropriate for patients who form the most common metabolic stone types — calcium oxalate and uric acid — will be discussed in this article. Patients with a tendency to form cystine and brushite stones may also benefit from some of the same dietary recommendations, but dietary management is a small part of an even more complex treatment regimen in these instances. Regardless of stone type, recommendations for dietary modifications are most accurate when tailored to the results of urine stone risk profiles, or "24-hour urine" studies. These studies typically provide total urine volume, urine calcium, sodium, citrate and uric acid, as well as pH, and supersaturation of critical compounds, among other measurements. The values, if properly interpreted, allow the clinician to observe the patient's specific abnormalities, recommend medication and/or dietary modification, and track progress through followup studies.

As with many bodily processes, stone formation is a complicated and multi-factorial process. Yet, there is much still to be understood about stone formation. For example, we know that stone formation runs in families, but while all humans form calcium oxalate crystals, most do not form stones (Lemann, 2002). And while for years calcium stone formers were instructed to restrict dietary calcium, there is now significant evidence against this recommendation (Borghi et al., 2002). These observations illustrate the sometimes counterintuitive and always complex nature of stone formation and the need for ongoing investigation.

Stone recurrence is frustrating for patients who have made changes in their lives and yet still form stones. While diet alone cannot always control the disease, dietary measures can absolutely help supplement other therapies, and for some patients are the primary tool for stone prevention. As a component of the medical management for stone disease, the goal of therapy should be to improve those factors thought to contribute to stone formation in the urinary tract, and thereby reduce the chance of forming stones, even if the disease is not eliminated.

Urine Supersaturation and Stone Formation

Urine volume plays a pivotal role in the process of stone formation. In particular, low volume, highly concentrated urine contributes to the supersaturation of elements normally found in the urine, such as calcium oxalate. Simply put, when the solute exceeds the solvent's ability to dissolve it, precipitation of crystals can occur. Consider an attempt to dissolve sugar in water: a tablespoon of sugar is readily dissolved in a glass of water, but eight tablespoons of sugar in that same glass will not completely dissolve, resulting in the accumulation of crystals in the bottom of

the glass. In this scenario, the water is saturated with sugar; the solvent, water, can dissolve no more solute. It has exceeded the point of saturation, and is supersaturated.

Fortunately, urine has the unique quality of holding more solute in suspension than does water and so can accept large concentrations of solute without precipitation. The ability of urine to keep such large concentrations in solution is, in part, due to the presence of protective organic molecules like citrate, as well as the presence of charged ions which alter the solubility (Menon & Resnick, 2002). Despite the fact that calcium oxalate can be present in urine in concentrations 7 to 11 times its solubility in water (Menon & Resnick, 2002), the point exists at which calcium oxalate exceeds the unique properties of urine; crystals will then form and possibly aggregate to form stones.

Understanding saturation principles in the urine, is it not clear that methods of prevention shall be focused on both an increase in *solvent* and a reduction of *solute*? These two concepts are the basis for the dietary changes described below.

Assessment of Dietary Patterns

As dietary history is not part of the typical urologic patient history forms, the nurse can obtain this information through patient interview. Important information includes the patient's intake of fluids throughout the day, environmental factors promoting dehydration, special diets such as now-popular high-protein diets, and a propensity to consume packaged or restaurant foods which are typically very high in sodium. What does the patient drink, how much is consumed, and how is fluid intake distributed through the day? Does he or she work in a hot or dry environment (such as a hot factory, outside work in the summer)? Does he or she prepare fresh foods at home or tend to eat convenience foods?

Nurses' dietary interview can occur before or after metabolic testing. Absent metabolic testing, this interview has even greater import as it is the only source of information about dietary habits. Asking patients to keep a 24-hour diet record may help identify patterns of which even the patient was unaware. Sources of dietary sodium can be obvious or insidious. The excess intake of a patient who consumes a bag of microwave popcorn every night is more apparent than the intake of a patient who drinks sports drinks after a daily workout; these are both very high in sodium, but the latter is less often recognized.

If the nurse has at his or her disposal a 24-hour urine study, dietary anomalies may be more specifically exposed and documented. This author views 24-hour urine studies as "vice recognition software;" the numbers show actual urine output, and indicate dietary sodium, protein, and oxalate excess. Patients with low urine volumes may believe their results are incorrect. They may say "I drink all the time!" and yet the output is low. Here the role of dietary counseling is critical; the insightful nurse helps the patient identify volume consumed, sources of insensible loss, and ways to ensure increased urine volume.

Dietary Changes to Prevent and Reduce Stone Formation

1. Increase Fluid Intake

Increasing urine volume can reduce supersaturation, and is widely known to help prevent stone formation. Recommendations for urinary output vary, but there is general agreement that it should exceed two liters per day, while some even encourage urinary outputs in excess of three liters per day (Menon & Resnick, 2002; Sakhaee, Zerwekh, & Pak, 1980). A key point is that the dilution of urine is necessary "24/7," or all day, every day. A patient who voids the recommended two liters a day between the hours of 8 am and 10 pm, but only 300 milliliters during the remaining 10 hours

of the day will have saturated urine overnight, with the possibility of precipitation and aggregation during the sleeping hours. Patients must accept the necessity of getting up at least twice at night to urinate, and should consume more water each time they rise to void.

Stress to patients that it is not the quantity of fluid consumed that is important, but rather the fluid voided that should be measured. Patients living in hot or dry conditions, or who exercise and perspire significantly, will need to drink even more liquid to maintain adequate urine output.

Many patients ask what fluids are recommended, and which are prohibited. The simple answer is that water is best. For those with excessive urinary oxalates, black tea should be eliminated because black tea is a high-oxalate beverage. Curhan, Willett, Rimm, Speizer, and Stampfer (1998) found, in a retrospective study of previously non-stone forming women from the Nurses' Health Study, that the type of beverages consumed proved relevant for stone formers. Of the 17 beverages studied, and after correcting for other contributing factors, those who drank one daily 8 ounce glass of grapefruit juice had a 44% increased risk of a stone event in the 8-year period, while the risk was decreased by 8% to 10% for each daily 8 ounce serving of coffee (both caffeinated and non-caffeinated), tea, or wine. A prospective study had similar conclusions for men, additionally showing that beer had a protective effect and apple juice increased the risk of stone events (Curhan, Willett, Rimm, Speigelman, & Stampfer, 1996). Also, a study published by Massey and Sutton (2004) showed a modest positive relationship between caffeine intake and urinary calcium levels in stone formers and non-stone formers, so caffeinated beverages should be limited in stone formers. In summary, stone formers should drink more water and avoid excess caffeine, black tea, and grapefruit and apple juices.

What do these studies mean for patient education? Water is the best beverage for stone formers. It is non-caloric, non-caffeinated, and contains insignificant amounts of solutes. In initial attempts to increase patients' fluid intake, it may be appropriate to advise them to drink whatever they can consume in large quantities. However, warning them of side effects of sugared and caffeinated beverages in large quantities is important. The results discussed above indicate that consumption of alcoholic beverages is unlikely to increase stone risk. Water that tastes good (filtered, reverse osmosis, bottled) may be easier to consume than tap water, so encourage patients to seek a source of good-tasting water. There is no clear agreement on the impact of drinking water's mineral content on lithogenesis; "hard water" may not be problematic for most patients (Menon & Resnick, 2002). Again, water in large quantities should be the focus of prevention. Lemonade is often recommended, as it supplies dietary citrate, a stone inhibitor and pH buffer when excreted later in the urine.

Encourage patients to set consumption goals, carry water with them at all times, and strive for pale urine throughout the day and night. Some patients describe an initial physiologic resistance to increased fluid intake which eases as their bodies and minds learn the new habit of extra fluid intake and output. According to Parks, Goldfischer, and Coe (2003), aims by clinicians to increase patients' urinary volumes often fall short, and follow-up metabolic studies showed an average increase in urine output of only 0.3 liters per 24 hours. This increase was associated with a curious increase in sodium intake. High urine volumes should be the goal of all patients who form stones. In this instance, more is definitely better. Most patients find that after forcing fluids for a couple of months, their bodies crave fluids and their habit is to drink more.

2. Consume Adequate Calcium

High urine calcium, hypercalciuria, is associated both with formation of kidney stones and with osteoporosis. Sufficient calcium intake is required for the growth and maintenance of the skeleton in children and adults. Reducing urine calcium should be a goal for stone formers, but not via dietary restriction. While reduced dietary calcium can decrease urine calcium (Lemann, 2002), calcium restriction is no longer advisable for patients who form calcium kidney stones as this puts

them at risk of bone disease, namely osteoporosis. Recall that bones are in a constant process of resorption and formation; adequate calcium is required for the ongoing rebuilding of bone material.

Several recent studies have shown, in fact, that adequate calcium intake is associated with *decreased* stone formation. Curhan, Willett, Knight, and Stampfer (2004) found that in previously non-stone forming younger women, higher intake of dietary calcium was related to lower risk of kidney stone formation. Additionally, a 5-year randomized clinical trial of men with a history of calcium oxalate stones found that a normal calcium, decreased sodium, and decreased animal protein diet was more effective for reducing stone events than was a restricted calcium diet (Borghi et al., 2002). So, adequate calcium plus decreased sodium and protein intake had a significantly more protective effect against stones than decreased calcium intake alone.

Why might increased dietary calcium reduce the risk of calcium stone formation? Calcium and oxalate bind in the gut *and* in the urine to form a nonabsorbable compound. Low dietary calcium permits greater free oxalate to be absorbed in the gut and excreted in the urine, which may be counterproductive for calcium oxalate stone formers. Restricted calcium intake results in increased urinary oxalates, a risk for stone formation (Menon & Resnick, 2002). This is a proposed cause of the association between reduced calcium intake and increased supersaturation of calcium oxalate (Lemann, 2002).

Clearly, strong research evidence now supports adequate calcium intake for patients who form kidney stones. Low-fat dairy products, green leafy vegetables, broccoli, fortified foods, and almonds are excellent sources. Patients should consume enough dietary calcium to meet (but not exceed) the United States Recommended Daily Allowance (RDA) of calcium, which ranges from 1,000 to 1,200 milligrams daily for adults. The recommendations are the same for men and women, but vary by age group (see Table 1). Patients should avoid calcium supplements in favor of calcium-rich foods; a patient with intolerance to dairy products may supplement, but should not exceed the RDA for his/her age group.

3. Limit Dietary Oxalates

Oxalate is found in many foods, but there is considerable variability in the amount, which depends upon where the food is grown. Likewise, individual absorption of oxalate also varies, which makes adequate calcium intake critically important. Nonetheless, oxalate restriction should be attempted. The highest levels of oxalate are found in chocolate, nuts, beans (including soybeans), rhubarb, spinach, beets, and black tea. A thorough oxalate list can be found on the Web site of the Oxalosis and Hyperoxaluria Foundation (http://www.ohf.org/diet.html). This list is exhaustive and may be overwhelming to patients. Stress that reduction of high oxalate foods is the goal for typical stone formers rather than strict avoidance of all oxalate-containing foods (which would be very difficult). Followup 24-hour urine studies will demonstrate the adequacy of patients' restriction.

Though only 10% to 20% of urinary oxalates come from dietary sources (Morton, Iliescu, & Wilson, 2002), dietary reduction is commonly advised for calcium oxalate stone formers. It has been suggested that because there is much less oxalate in the urine than calcium in the urine, urinary oxalate concentration is much more critical to the formation of calcium oxalate crystals than is the urinary calcium concentration; reducing urine oxalates may have a more powerful effect on stone formation than can reduction of urine calcium (Morton et al., 2002). Patients with calcium oxalate stones, particularly those with documented hyperoxaluria, should avoid foods high in oxalates. Vitamin C is a precursor to endogenous production of oxalates, so some clinicians recommend avoiding mega-doses of vitamin C. The rare genetic condition of primary hyperoxaluria is only slightly impacted by dietary reduction, and causes serious medical problems besides kidney stones.

4. Limit Sodium Intake

Because calcium and sodium compete for reabsorption in the renal tubules, excess sodium intake and consequent excretion result in loss of calcium in the urine. High-sodium diets are associated with greater calcium excretion in the urine (Lemann, 2002). Metabolic studies often reveal exceptionally high urine calcium over 24 hours, related to patients' exceptionally high sodium excretion. Patients may deny salt intake, stating, "I never salt my food!" They quite likely are ignorant of hidden sodium sources in the diet. Sodium is a common preservative in canned and frozen foods, and is endemic in restaurant foods. Instruction on careful inspection of food labels and wise food choices helps patients identify and reduce sodium in their diets.

A notable dietary "ah-ha!" was the admission by one patient that, on the day of 24-hour urine testing, she ate a full jar of pickles to reduce stress, and then *drank the brine*; needless to say, her urine sodium was very high on the day of her stress mitigation.

The role of the nurse or dietician in shedding light on sources of sodium cannot be underestimated. Repeated, persistent inquiry into dietary habits may be necessary. The goal of therapy should be a "no added salt diet," or the equivalent of 2,000 mg per day or less of dietary sodium. Reduction of dietary sodium is difficult and disappointing to patients. They may believe they have made significant reductions and sacrifices, while their urine sodium remains high. Consultation with a registered dietician may help the patient achieve the specific goal of a sodium intake of 2,000 milligrams or less per day.

5. Limit Animal Protein

The effect of excess animal protein (purine) is most obvious for the uric acid stone former. Uric acid, a byproduct of purine metabolism, is excreted in large quantities in the urine. Excess protein creates urine with high total urine uric acid, potentially high supersaturation of urine uric acid, and a low pH, necessary for formation of uric acid stones. There is no inhibitor of uric acid crystal formation (Menon & Resnick, 2002), so dietary measures focus on reducing uric acid and increasing urine volume. Reduction of animal protein to 12 ounces per day for adults is recommended. This is plenty to meet the dietary needs of most Americans, many of whom typically consume several more ounces of animal protein daily than is recommended. Protein from plant sources (beans, legumes, etc.) can be substituted as a dietary alternative without negative consequences. Calcium oxalate stone formers reducing their animal protein should note the oxalate content of substitute proteins.

The role of excess protein in promoting calcium stone formation is less obvious, but equally important. High dietary protein is associated with increased urinary calcium. Thus, there is a link between meat consumption and both uric acid *and* calcium stone formation. In fact, vegetarians form stones at one-third the rate of those eating a mixed diet (Lemann, 2002). A study of 18 hypercalciuric stone formers found that a 15-day protein restriction had many positive effects on urinary markers of stone risk. Namely, significant decreases were seen in urine calcium, urine uric acid, urine phosphate, and urine oxalate. And, for unclear reasons, a beneficial increase in urinary citrate was observed (Giannini et al., 1999). Citrate is a known inhibitor of calcium oxalate crystal formation and also increases pH, which can prevent uric acid stones. Clearly, the benefits of protein restriction for stone formers are many.

6. Weight Loss

A relationship between weight, body mass index and risk of calcium oxalate stone formation was established in a retrospective study of health professionals. Curhan and colleagues (1998) found that "the prevalence of stone disease history and the incidence of stone disease were directly associated with weight and body mass index. However, the magnitude of the associations was

consistently greater among women" (p. 1645). The value of weight loss for stone prevention has not been proven, but given the benefits of weight loss for general health, it is certainly worth mentioning to overweight patients who form stones.

Educational Resources

There are excellent resources on the Internet for patients seeking nutritional information. One stellar example is NutritionData (www.nutritiondata.com). Here patients can search by general food category, like "pickle," to view the standard sodium content, as well as a plethora of additional information regarding vitamin and mineral content, calories, suggested healthier substitutes, and even the individual amino acid compositions of each protein. The site also provides detailed information about thousands of specific brand items from grocery and fast food restaurants. Under "Tools," patients can search within food categories like "dairy products" for choices highest in calcium and lowest in sodium. This site is complex and may be overwhelming to patients without good computer and Web skills, but is extraordinarily comprehensive; unfortunately, this site does not list oxalate content. For that purpose, refer patients to www.ohf.org.

For patients without Web access, nurses might find it helpful to review a general nutrition book for charts and diagrams to help patients understand nutrition content. Show patients a sample food label from a can of soup so they know where to find sodium content on foods at home. For a simple list of high-oxalate foods, visit http://www.gicare.com/pated/edtgs29.htm.

Conclusion

The dietary measures discussed have value particularly for patients who form the most common types of kidney stones: calcium oxalate and uric acid. That said, they may be insufficient to control the various metabolic abnormalities present in individual patients. The most effective management of kidney stones includes in-depth metabolic studies, recommendations tailored to patients regarding medications and dietary changes, and follow-up to ensure changes are having the desired effect. Urine studies should be repeated to judge progress approximately 6 to 8 weeks after initial metabolic testing recommendations are implemented. Once a stable state is reached in which the patient's urine demonstrates decreased risk of stone formation, metabolic testing should be performed (along with an x-ray to check for stone growth) at least annually to monitor stone risk. The cycle of stone formation can be altered, and in some cases broken, with the aid of effective dietary management.

Every patient need not make all of these changes to his/her diet, but in the absence of patient-specific urine studies, none of these recommendations is harmful. Aside from oxalate consumption, the dietary recommendations for calcium oxalate and uric acid stone formers are the same (see Table 2). Assessing patients' dietary habits can shed light on potential areas of improvement. For example, a receptive uric acid stone former on a high-protein diet for weight loss could benefit from counseling on the effects of this diet on his/her stone disease.

Of course, talking about dietary changes is easier than actual implementation. Encourage patients to make changes at a realistic pace. Praise even modest progress and stress the value of striving for improvement rather than perfection. The role of the nurse here is clear. Who better to educate, provide encouragement and reinforcement for patients with kidney stones than the urologic nurse?

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