

Nutrition and Kidney Stones, Part One

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Recently, an excellent review on kidney stones came across my desk.¹ The highlights of this article are the basis of this two-part series.

Ask anyone who has passed a kidney stone what it felt like, and even the most laid-back person will suddenly become animated. Ask them if they look forward to having the experience again, and the answer is almost universal; that is, they would not wish the experience on their worst enemy.

Kidney stones (nephrolithiasis) occur in a little over 10 percent of North Americans.² When a patient informs you that they have kidney stones, or have had them in the past, and the patient wants nutritional advice, the first step is a record request from the physician who managed their problem. This is because your advice will vary depending on the pathophysiology of the kidney stone. You need to determine the cause, which could be hypercalciuria, hyperoxaluria, hyperuricosuria, hypocitraturia, hypomagnesuria, or renal tubular acidosis.

Hypercalciuria

Hypercalciuria can generally be divided into two types: idiopathic and absorptive. Idiopathic hypercalciuria shows elevated urinary calcium levels with normal calcium serum levels. Absorptive hypercalciuria shows calcium elevations in both the urine and serum.

Other types of hypercalciuria include resorptive and renal. Resorptive hypercalciuria accounts for 5 percent of all cases. It is caused by subclinical hyperparathyroidism and has the classic findings of bone demineralization and elevated fasting urinary calcium levels. Renal hypercalciuria is due to the kidneys' decreased ability to reabsorb calcium. This causes the body to increase vitamin D synthesis. Fasting urinary calcium levels are normal.

A recent paper showed that keeping dietary calcium at normal RDA levels and lowering sodium, protein and oxalates with high levels of fluid and citrus consumption was superior to a low-calcium diet in reducing the risk of recurrent stones.³ All patients with hypercalciuria should be referred to rule out hyperparathyroidism.¹

Next month, in part two, we will review the other metabolic defects that promote stone formation, including hyperoxaluria, hyperuricosuria, hypocitraturia, hypomagnesuria, and renal tubular acidosis.

Dietary Treatment for Hypercalciuria*

Low:

Sodium -	Less than 2,000 mg/day
Protein -	RDA level (USA): No more than 0.8 grams per kilogram of body weight a day

Oxalate - Spinach, rhubarb, strawberries, chocolate, nuts, tea, wheat bran, beet greens

Normal:

Calcium - RDA level (USA): Depending on age and sex will range from 900 milligrams to 1300 milligrams a day from all sources (food and supplements)

High:

Citrus fruit - 3 servings a day

Fluids - Enough to produce at least 64 ounces of urine daily

*Please note: These are general guidelines only and will vary depending on a patient's specific need.

References

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