CME CREDIT

# CALCIUM STONES: CALCIUM RESTRICTION NOT WARRANTED

A low-calcium diet, traditionally prescribed for patients with calcium kidney stones, may actually promote stone formation. Sounder advice would be to drink more fluids, decrease salt, protein, and oxalate intake, and perhaps even increase calcium intake.

Renal stones are a common reason patients visit their physician. Recurrence is common: 40% of patients have a second event within 2 to 3 years, increasing to 75% within 7 to 10 years, and 100% within 15 to 20 years. Calcium stones are the most common variety, representing 75% of all stones. The majority are composed of calcium oxalate; calcium phosphate stones are less common.

## RISK FACTORS FOR CALCIUM STONES

Various factors may promote stone formation through a common pathway. Even in normal persons, the concentration of calcium oxalate in the urine is four to five times higher than its solubility. Fortunately, most people have adequate amounts of substances in the urine such as citrate which inhibit crystal formation and promote the solubility of calcium oxalate.

### Inadequate fluid volume

The single most common factor that promotes stone growth is inadequate fluid intake, ie, a low volume of concentrated urine. Patients with a urine volume less than 2 L/day can often avoid further events by increasing their fluid intake. Hyperuricosuria also promotes calcium stone formation; the mechanisms are not clear, but uric acid crystals may serve as a nidus for the growth of calcium oxalate or calcium phosphate crystals.

### Hypercalciuria

A number of factors can increase calcium excretion; however, most patients with calcium stones have idiopathic hypercalciuria.

A high sodium intake promotes increased urinary calcium excretion. Individuals who have hypercalciuria are, in general, more sensitive to sodium than are normal subjects and also tend to have a higher sodium intake.

Data from World War II show that when a country undergoes protein deprivation, the frequency of renal stones decreases, and when a country consumes more protein the frequency of renal stones increases. Patients with stones excrete more calcium than normal subjects do at any level of protein intake, however.

Prolonged hypercalcemia increases the chance of hypercalciuria, as do metabolic diseases that increase the entry of calcium into the circulation from bone or from the gut, even in the absence of hypercalcemia. Any condition that decreases the renal tubular reabsorption of calcium can also produce hypercalciuria. These include renal tubular acidosis, chronic phosphate depletion, and protein loading. Volume expansion directly increases calcium excretion because calcium reabsorption in the tubule is linked to sodium reabsorption; anything that diminishes sodium reabsorption also diminishes calcium reabsorption. Finally, loop diuretics promote calcium excretion, as opposed to thiazide diuretics, which promote calcium reabsorption.

However, the benefit of thiazide diuretics is lost if the patient's salt intake is high. I always ask my patients if they salt their food before tasting it. Any patient who responds "yes" to this question almost always ingests more than 10 to 15 g of salt per day. Careful instruction can usually reduce the salt intake and make thiazide diuretics more effective.

At any level of dietary calcium intake, patients

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with calcium stones excrete more calcium than subjects without stones. Therefore, high calcium intake does not necessarily cause calcium stones.

### The phosphate hypothesis

Why would patients with idiopathic hypercalciuria excrete more calcium? One hypothesis holds that they have a defect in the way their kidneys handle phosphate. A decrease in tubular maximum reabsorption of phosphate would render them slightly hypophosphatemic, though their serum phosphate level would not be below normal limits. A reduction in serum phosphorus would stimulate production of vitamin D, which increases absorption of calcium from the gut. Decreases in serum phosphate are also associated with decreased distal tubular calcium reabsorption. Finally, chronic hypophosphatemia increases bone calcium resorption.

Evidence for this hypothesis is provided by studies in patients with calcium stone disease and decreased bone density. These patients have increased urinary calcium excretion even when fasting and not receiving any dietary calcium. Further, dietary studies provide evidence that patients with calcium stones have increased vitamin D production or decreased vitamin D metabolism, even when calcium intake is high.

### Hyperoxaluria

The most common causes of hyperoxaluria are inflammatory bowel disease and dietary calcium restriction. Normally, calcium and oxalate bind in the gut, leaving less free oxalate available to be absorbed and hence excreted in the urine. Anything that diminishes the amount of calcium in the gut will result in less calcium oxalate binding, leaving more oxalate available for absorption and excretion. Patients with inflammatory bowel disease often have fat malabsorption; the fat ties up the calcium, leaving more oxalate unattached, so that more oxalate is absorbed and excreted.

A recent prospective study supports the concept that calcium in the gut may protect against calcium oxalate stones. Some 45,619 men with no history of kidney stones completed a questionnaire regarding diet, medications, and medical history. After 4 years, 505 men had developed kidney stones. The majority (71.5%) had calcium stones. Higher calcium intake actually reduced the risk for kidney stones. Protein intake increased the likelihood of kidney stones slightly, but the increase was not statistically significant. High potassium intake reduced the risk, as did higher fluid intake.

## RECOMMENDATIONS

If a patient has recurrent calcium stones, two 24-hour urine samples should be obtained to measure the excretion of calcium, creatinine, uric acid, oxalate, citrate, and sodium. If there is hypercalciuria, known causes such as hypercalcemia, renal tubular acidosis, hyperparathyroidism, or use of corticosteroids should be ruled out. Dietary sodium, protein, and oxalate should be reduced, and calcium should not be restricted. A thiazide diuretic should be instituted. If there is hyperuricosuria, the patient should follow a diet low in purine and could be given allopurinol if diet alone is inadequate.

Elevated rates of oxalate excretion (> 40 mg/day) should prompt a search for enteric oxaluria, which can be treated with calcium supplements, and for primary hyperoxaluria, which may respond to pyridoxine. The patient should begin a low oxalate diet and avoid vitamin C.

If there is hypocitraturia, one should initiate oral citrate therapy and continue to measure the excretion of citrate. No matter what type of stone is present, the patient should increase fluid intake to 3 L/day.

PHILLIP M. HALL, MD
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## SUGGESTED READING

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