



Preventing kidney stones: Calcium restriction not warranted

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■ ABSTRACT

The traditional wisdom on preventing calcium stones, the most common form of kidney stone, has been to advise patients to limit dietary calcium. Research has proved this wrong, however. Normal dietary calcium intake, along with reduced salt and protein, is now advised. This paper also summarizes the diagnosis and treatment of the less-common forms of kidney stones—struvite, uric acid, and cystine.

THE CONVENTIONAL WISDOM on preventing kidney stones is turning out to be wrong. Since most kidney stones are calcium stones, the conventional wisdom has been to limit calcium intake to prevent recurrence. But evidence shows that this approach actually leads to more stones, and that reducing sodium and protein is more effective.

Kidney stones are common, frequently recur, and are more common in men. The peak incidence is between ages 18 to 45; if a stone occurs for the first time in an older person, a new metabolic disturbance should be considered.

There are basically four types of stones:

- Calcium (calcium oxalate or calcium phosphate)
- Struvite (infection)
- Uric acid
- Cystine.

■ CALCIUM STONES

About three fourths of kidney stones are made of calcium oxalate or calcium phosphate. Urine that is supersaturated with either solute is on the brink of precipitation, and it is not completely understood why some people in this state develop stones and others do not. However, certain factors clearly increase risk.

Risk factors for calcium stones

Low fluid intake. Highly concentrated urine is more prone to crystal formation. People in certain occupations, such as long-distance truck drivers and construction workers on tall buildings, routinely restrict their fluid intake.

Low urinary citrate. Citrate inhibits crystal formation. The normal range for citrate excretion is 300 to 700 mg/day; excretion is lower during prolonged metabolic acidosis, typically in patients with chronic inflammatory bowel disease or renal tubular acidosis.

Excess urinary uric acid (uricosuria) or oxalate (hyperoxaluria). Any additional crystal or solute can trigger precipitation in urine that is already supersaturated with calcium. Uricosuria (> 750 mg/24 hours) may be caused by excess purines from ingestion of large amounts of animal protein.

Hyperoxaluria (> 40 mg/day) can be caused by:

- Increased oxalate production, due to rare congenital disorders
- High dietary oxalate from spinach, rhubarb, nuts, draft beer, cocoa, and chocolate
- Increased oxalate absorption, such as in inflammatory bowel disease and short bowel syndrome: fat malabsorption leads to excess fat binding to calcium in food, leaving dietary oxalate unattached, and when oxalate passes

**To prevent
calcium stones,
limit salt and
protein intake**

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Calcium stones comprise 75% of cases

to the colon, it is efficiently absorbed into the bloodstream

- Low dietary calcium: the lower the dietary calcium, the less calcium oxalate binding occurs, and the more oxalate is absorbed by the colon
- Vitamin C: there is a possible association with doses > 2 g/day.

High urinary calcium (hypercalciuria). Hypercalciuria (> 300 mg/day for men and > 250 mg/day for women, or 4 mg/kg/day for either sex) is one of the most important reasons patients develop kidney stones. It can be caused by:

- Excess calcium in the blood. Hypercalcemia can be due to a variety of causes, including primary hyperparathyroidism, metabolic bone diseases, chronic metabolic acidosis with renal tubular acidosis, and over-ingestion of vitamin D.
- Decreased calcium reabsorption in the renal tubules. This may occur with volume expansion from a high-sodium diet or loop diuretics; phosphate depletion, seen with high-protein diets; hyperparathyroidism; and renal tubular acidosis.
- Unknown factors. Idiopathic hypercalciuria nephrolithiasis is the most common diagnosis for patients with calcium stones. Many have a slightly low serum phosphate level. The diagnosis is made only if there is no hypercalcemia and no known cause for hypercalciuria. It tends to recur and affects men three times more often than women.

High sodium intake. The more sodium in the diet, the more calcium is excreted in urine.¹

■ IS CALCIUM INTAKE A RISK FACTOR?

High calcium intake has long been suspected of increasing the risk of calcium stones, but in fact, the opposite may be the case.

People with hypercalciuria seem to have a regulatory problem with calcium absorption rather than excess calcium intake: at any level of dietary calcium intake they have a higher-than-expected calcium output.² When placed on a low-calcium diet, they go into negative calcium balance and mobilize bone calcium. Abnormal production or activity of 1,25-dihydroxyvitamin D may be part of the explanation.³

Large prospective studies have clearly shown that dietary calcium intake is actually inversely related to the risk of developing kidney stones for both men⁴ and women.⁵ The explanation may be that if one does not consume enough calcium, less is available to bind to dietary oxalate. More oxalate then reaches the colon, where it is absorbed into the bloodstream and excreted as calcium oxalate by the kidney, setting the stage for stone formation.

Interestingly, however, supplemental rather than dietary calcium was found to increase the risk of stones. The explanation may be that supplements are often ingested without food or after a meal low in oxalate.⁵

A recent study⁶ tested the traditionally recommended low-calcium diet for people with hypercalciuria against a diet with normal calcium but low in animal protein and salt. Both diets were associated with a reduction in urinary calcium; however, urinary oxalate excretion rose in the low-calcium group and fell in the normal-calcium group. Twelve of 60 patients developed stones on the normal-calcium, low-protein, low-salt diet, compared with 23 of 60 on the low-calcium diet ($P = .04$).

While it is not clear whether it is protein, salt, or calcium that is the most important dietary factor, a low-calcium diet is clearly not a good treatment plan for people with hypercalciuria or calcium stones.

■ TREATMENT FOR CALCIUM STONES

A small stone (≤ 5 mm in diameter) has an 80% chance of spontaneously passing within 6 weeks. In the meantime, the patient should be advised to drink plenty of fluids and use pain medications as needed.

A large stone (≥ 1 cm) has a low probability of passing, and immediate referral to a urologist is indicated. Treatment of medium-sized stones depends on clinical judgment and the comfort and desires of the patient.

Patients with idiopathic disease should never be sent home thinking that there is nothing they can do to prevent a recurrence. They should be instructed to:

- Increase their fluid intake to at least 8 to 10 glasses of water a day, including one at bedtime



- Eliminate highly salted processed foods and add no extra salt to home-cooked meals
- Eat normal amounts of dairy products and other calcium-rich foods
- Reduce oxalate-containing foods
- Reduce meat protein to 0.8 to 1 g/kg/day.

A thiazide diuretic, amiloride, or both should be prescribed if calcium stones recur.

■ STRUVITE (INFECTION) STONES

Struvite stones, composed of magnesium ammonium phosphate, account for 10% to 15% of kidney stones. They form in the presence of chronic infection with urease-producing bacteria, which split urea and cause persistently alkaline urine. They often form when there is a urinary tract abnormality; for example, patients may complain of chronic problems with bladder emptying or infections.

Urinalysis provides clues to the diagnosis of struvite stones: the urine is alkaline, with a pH of 6.5 to 7, and white cells and bacteria are present.

Struvite calculi frequently form “staghorn” branches, visible on radiographic studies within the collecting ducts of the kidney.

Owing to their size, struvite stones will usually not pass spontaneously and must be removed by surgery or lithotripsy. Treat infection with antibiotics.

■ URIC ACID STONES

Uric acid stones account for 5% to 10% of kidney stones. Risk factors include:

- Dehydration
- Inflammatory bowel disease (because of chronic volume contraction and chronic metabolic acidosis)
- Low urine pH (uric acid is less soluble in acid; acidic urine is sometimes due to a defect in ammonia secretion in the kidney)
- Gout (though fewer than half of uric acid stone-formers have gout).

Uric acid stones are radiolucent and are not visible on plain radiographic films. They can be seen by ultrasonography or computed tomography without contrast.

Treatment of uric acid stones

- Hydration
 - Alkalinization with sodium bicarbonate tablets or citrate solution: give the patient pH paper and instruct him to test his urine every morning and to keep the pH above 6.5.
 - Allopurinol if hyperuricosuria is present.
- Removal of stones or lithotripsy is usually unnecessary.

■ CYSTINE STONES

Fewer than 1% of kidney stones are made of cystine. The condition arises from an autosomal-recessive disorder involving defective gastrointestinal and renal handling of four amino acids: cystine, ornithine, arginine, and lysine. Of these, cystine is the least soluble and forms stones.

Cystine stones usually first arise before age 20, but the first event can occur later, even in old age. The semiluent stones are visible on radiographs, but are not as dense as calcium. The cystine crystal can be seen in the urine: it looks like a benzene ring, unlike any other urine crystal.

Treatment is with hydration and alkalinization of the urine to a pH greater than 6.5. If these methods are unsuccessful, penicillamine is the traditional therapy, but captopril is also effective and causes fewer side effects.

■ WORKUP FOR KIDNEY STONES

Stone analysis should be done if possible. A calcium phosphate stone is a tip-off to renal tubular acidosis and hyperparathyroidism.

Clinical clues

While not diagnostic, certain characteristics are red flags.

Age of onset. If the patient is younger than 20 years, suspect a cystine stone or underlying renal tubular acidosis.

Family history. A strong family history of stones is more common in idiopathic hypercalciuria and cystine stone disease.

Diet. Low fluid intake, excessive use of vitamins C or D, and a diet high in meat and other high-oxalate foods predispose to stones.

Chronic diarrheal disorders. Suspect a calcium oxalate stone.

Stones ≤ 5 mm have an 80% chance of passing spontaneously



Recurrent urinary tract infections.

Suspect a struvite stone.

Ileostomy. Suspect a uric acid stone.

Sarcoidosis or other conditions such as Sjögren syndrome are associated with renal tubular acidosis.

Medical imaging

Look for multiple stones or anatomic abnormalities of the urinary tract. If no stone is visible, consider a calcium stone less than 3 mm in diameter, a uric acid stone, or an obstruction by a tumor or blood clot.

Calcium phosphate or calcium oxalate deposits visible at the renal cortex and medulla interface are described as nephrocalcinosis. If present, suspect renal tubular acidosis, hyperparathyroidism, or (rarely) medullary

sponge kidney.

Laboratory tests

- Chemistry profile for creatinine, calcium, uric acid, phosphorus, and serum parathyroid hormone, especially if hypercalcemia is present
- Electrolytes, looking for renal tubular acidosis (hypokalemia, with low bicarbonate and high chloride and alkaline urine)
- 24-Hour urine for creatinine, calcium, oxalate, sodium, urate, citrate, and volume
- Urine culture
- Urinalysis—check for crystals, pH
- Urine sodium, which gives an idea of dietary sodium. If the level is above 100 mmol per day, the patient must cut back on his sodium intake.

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