Medical Management of Nephrolithiasis
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At the Cleveland Clinic, we have several kidney stone specialists who work closely with our Urology colleagues to optimize the management of patients with nephrolithiasis. Today I would like to review our approach to the medical management of calcium stones.

The incidence of kidney stones has been steadily increasing over the past few decades, especially in women. It is thought that the increased incidence of obesity and the use of bariatric surgery is contributing to this rise.

Calcium stones, mainly oxalate, continue to be the most common stones encountered.

We take a multi-faceted approach to the kidney stone patient: we address nutritional issues and metabolic abnormalities that increase the risk of kidney stones as well as focusing on the type of stone involved.

The work-up of the patient presenting after their first stone is generally limited to routine dietary counseling and review of clinical risk factors for recurrent stone disease. If the stone is unavailable for analysis, management is based on the assumption that the stones are calcium-based.

Dietary counseling forms the foundation for the management of every stone patient. Water intake should exceed 2 liters per day to maintain urine flow and increase solute solubility. In order to minimize nocturnal urinary concentration, patients should drink water before bedtime to force nighttime urination, at which time they should drink more water. I recommend patients avoid caffeine, tea and grapefruit juice. Lemon and cranberry juices are considered safe. Diet should include a 2000mg sodium restriction and avoidance of calcium restriction. A high sodium diet leads to increased urine calcium excretion. And as we discuss later, a low calcium diet can increase oxaluria. Patients who have a diet high in animal fats should decrease their meat intake to reduce the acid load. Borghi in the New England Journal of Medicine in 2002 demonstrated that this diet decreased stone recurrence significantly compared to a low calcium diet.

Clinical features that are associated with nephrolithiasis should be identified. Patients with Crohn's disease or who have had bariatric surgery may have malabsorption syndromes that can cause volume depletion and low urine volume, acidosis, hyperoxaluria and hypocitraturia. Development of stones at a young age or a family history of stones suggests an RTA or another familial process.

Risk of recurrence after the first kidney stone increases with time, with 50% recurrence at 5 to 10 years. Asymptomatic stones diagnosed incidentally may become symptomatic in about 50% of patients at 5 years.

Any patient who has multiple or recurrent stones, non-calcium stones, metabolic or systemic abnormalities increasing their stone risk, required urologic intervention or developed acute kidney injury with their first stone, or is otherwise concerned about reducing the risk of recurrence should undergo a more extensive work-up.

This work-up consists of a 24 hour urine panel to include sodium, creatinine, citrate, calcium, oxalate, uric acid, total volume and in select cases cystine. Serum chemistry, PTH, uric acid and vitamin D levels should also be checked.
Any lab abnormality identified should be addressed.

High urinary sodium excretion or urine volume <2L per day requires dietary counseling.

With hypocitraturia, systemic acidosis and hypokalemia should be treated. Potassium citrate supplementation is required at doses of up to 6-8 grams per day. This is best taken two to three times daily with meals to avoid dyspepsia. As this slide details, systemic acidosis impairs citrate excretion. Thus, if citrate is not tolerated, potassium bicarbonate may be used. Sodium bicarbonate should be used with caution due to the sodium load.

For hyperoxaluria, or a daily excretion of more than 40mg, dietary oxalate restriction and cessation of vitamin C supplements are recommended. Dietary oxalate restriction has been shown to cut oxalate excretion by up to 50%. Malabsorption syndromes should be treated. Citrate supplements may be used in a patient with acidic urine.

While calcium supplementation remains controversial, dietary calcium restriction is no longer recommended because calcium is the main cation that binds dietary oxalate in the gut, preventing it from being absorbed. Malabsorption syndromes can lead to binding of calcium by fatty acids, increasing unbound oxalate available for absorption. The development of hyperoxaluria after bariatric surgery is just now being investigated but appears to be a real phenomenon based upon retrospective studies.

In hypercalciuria, loop diuretics should be stopped. Hypercalcemia and hyperparathyroidism should be ruled out. If hypercalciuria is found to be idiopathic, thiazide diuretics are used to decrease urinary calcium. Again, acidic urine may be treated with citrate supplements.

Hyperuricosuria increases the risk of both calcium and urate stones, and can occur in the absence of hyperuricemia. Allopurinol therapy can decrease stone incidence.

It may take up to 3 serial urine collections to accurately identify a urinary abnormality. Even if the urine studies remain normal, treatments targeting sodium, oxalate or citrate may still lead to decreased stone recurrence.

Remember that cystinuria can also present with calcium stones.

Calcium phosphate stones tend to be recurrent and do not respond well to external lithotripsy. They are associated with RTA’s and increased calcium and oxalate excretion.

After implementing a change in therapy, a repeat 24-hour urine collection should be repeated 6-8 weeks later. This will assess the impact of your interventions and can confirm your patient is drinking enough water and is restricting sodium intake. Serial imaging can be done with ultrasound or x-ray every 2-3 years if the patient remains asymptomatic.

Finally, if a patient presents with acute renal colic, there are several things you can do to assist with stone passage. A non-enhanced renal CT should be obtained since the probability of passing a stone depends on its size and location. Pain control can often be accomplished with NSAID’s rather than narcotics. Tamsulosin has been shown to expedite stone expulsion from the ureter. You should consider referring the patient to a urologist if they are in intractable pain, develop moderate hydronephrosis or acute kidney injury, develop pyelonephritis or are unable to pass the stone after 2-4 weeks of medical management.

This concludes our review of the medical management of nephrolithiasis. The Cleveland Clinic Department of Nephrology and Hypertension is available to assist you with the medical management of your patients with recurrent stone disease.