

# NEPHROLOGY

# Rounds™

## Diagnosis and Management of Stone Disease

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Kidney stones are a major cause of morbidity. The lifetime prevalence of symptomatic urolithiasis is approximately 10% in men and 5% in women<sup>1,2</sup> and more than \$2 billion is spent on treatment each year.<sup>3</sup> Notably, incidence rates of stone disease are rising in the US and other countries.<sup>1,4-6</sup> Prompt diagnosis and individualized intervention are required to reduce the morbidity and expense of stone disease.

### Diagnosis of stone disease

#### *Clinical and laboratory manifestations*

Symptomatic urolithiasis classically presents with unilateral flank pain of sudden onset. The pain is precipitated by the passage of a kidney stone from the renal pelvis to the ureter, and is due to ureteral spasm. Because the waxing and waning pain of a symptomatic stone does not completely remit, the term “renal colic” is technically inaccurate.<sup>7</sup> The pain is often severe enough to prompt the patient to seek medical care at an emergency department, and can be accompanied by nausea and vomiting. The location of the pain depends on the location of the kidney stone: a stone in the upper ureter may cause pain to radiate anteriorly to the abdomen; whereas, a stone in the lower ureter can cause pain to radiate to the ipsilateral testicle in men or to the ipsilateral labium in women. If the stone is lodged at the ureterovesical junction (UVJ), the patient may experience urinary frequency and urgency. Less commonly, urolithiasis can manifest as gross hematuria without pain.

On physical examination, the patient will be in obvious pain and may constantly adjust position in an unsuccessful attempt to alleviate the discomfort. Ipsilateral CVA tenderness may be present. Signs and symptoms of sepsis can occur in cases of obstruction with infection.

Serum chemistries are usually normal, but leukocytosis may be present due to stress or infection.<sup>8</sup> Although the urinalysis will often reveal hematuria and pyuria (and occasionally crystalluria),<sup>8</sup> the absence of red cells in the urine does not exclude a stone, particularly in cases where a stone causes complete ureteral obstruction. A study of 397 patients presenting with acute, symptomatic urolithiasis found that 9% did not have hematuria.<sup>9</sup>

#### *Imaging*

Helical computed tomography scan (HCT) is the preferred radiographic test to confirm or exclude the diagnosis of symptomatic urolithiasis.<sup>10</sup> HCT does not require radiocontrast and can visualize uric acid stones (traditionally considered “radiolucent”).<sup>10</sup> Typically, the HCT will show a ureteral stone or evidence of recent passage (eg, perinephric stranding or hydronephrosis). Although published sensitivity and specificity values obtained from direct comparison of HCT and intravenous urography (IVU) do not exist, HCT can detect small stones that may be missed by IVU.<sup>11,12</sup>

Few studies have compared HCT to ultrasound (US). However, in patients presenting with presumed renal colic, the sensitivity of HCT was 96% compared with 61% for US; the specificity for each was 100%.<sup>13</sup> Although US has the advantage of avoiding radiation, it can only image the kidney and proximal ureter. Thus, ureteral stones can be missed on US. A retrospective study comparing 123 US examinations with HCT found that the sensitivity for US was 24% and the specificity 90%.<sup>14</sup> US may also miss renal stones that are <3 mm in size.<sup>14</sup>

The conventional abdominal x-ray (KUB) is inadequate for diagnosis. KUB can miss a stone in the ureter or kidney (even when radio-opaque), and provides no information on obstruction or recent stone passage. For the evaluation of residual stone fragments in patients who have undergone percutaneous nephrostolithotomy, HCT is more sensitive than KUB (100% versus 46%).<sup>15</sup>

AS PRESENTED IN THE ROUNDS OF  
THE NEPHROLOGY DIVISION OF  
BRIGHAM AND WOMEN'S HOSPITAL  
BOSTON, MASSACHUSETTS



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***Nephrology Rounds* is approved  
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## Differential diagnosis

In general, a kidney stone must pass into the ureter to cause pain. Therefore, the isolated presence of a renal stone on radiographic imaging is an inadequate explanation for acute abdominal or flank pain.<sup>8</sup> The differential diagnosis of a patient with suspected renal colic includes musculoskeletal pain, herpes zoster, acute cholecystitis, duodenal ulcer, appendicitis, diverticulitis, pyelonephritis, abdominal aortic aneurysm, gynecologic disease, and ureteral obstruction due to blood clot, sloughed papilla, or ureteral stricture.<sup>7,8</sup>

## Management in the acute setting

### Medical treatment

Because renal colic is excruciating, analgesia is a primary goal in the acute setting. Randomized controlled trials suggest that parenteral nonsteroidal anti-inflammatory drugs (NSAIDs) are as effective as narcotics in treating renal colic.<sup>16</sup> Newer medications that may be effective include antispasmodics,<sup>17</sup> trigger point injection with lidocaine,<sup>18</sup> desmopressin,<sup>19</sup> and NSAIDs combined with nitrates.<sup>20</sup> However, data on the utility of these interventions are limited.

Medical therapy has also been directed at treating kidney stones or hastening ureteral stone passage. Alkalinization of the urine may dissolve uric acid stones<sup>21</sup> and some experts believe that volume expansion will increase the likelihood of stone passage.<sup>22</sup> Although more trials are needed, alpha-blockers and calcium-channel blockers also may facilitate the passage of ureteral stones.<sup>23-25</sup>

### Surgical treatment

Larger and more proximal ureteral stones are less likely to pass spontaneously and are more likely to require urologic intervention. If a stone does not pass rapidly, the patient can be sent home with oral analgesia and instructions to return for fever or uncontrollable pain. Most urologists prefer to wait several days before intervention unless there is evidence for infection, low likelihood of spontaneous passage (eg, size is >6 mm), the presence of an anatomic abnormality that would prevent passage, or unrelenting pain.<sup>26</sup> Infection in the setting of obstruction is a surgical emergency and mandates emergency drainage.

The initial urologic approach may be directed at the relief of obstruction (generally by cystoscopic placement of a ureteral stent) rather than stone removal. Anatomy of the urologic tract, availability of technology, experience of the urologist, and the size, location, and composition of the stone determine the best option for stone removal. Extracorporeal shock wave lithotripsy (ESWL) is the least invasive option, and is most effective for smaller calcium stones (<1 cm) located in the renal pelvis or proximal ureter.<sup>27-29</sup> Cystoscopic stone removal by basket extraction or fragmentation is invasive, but effective, and can now be used to remove stones in the proximal ureter or kidney.<sup>30</sup>

Percutaneous nephrostolithotomy is more invasive, but may be necessary for large stone burdens or stones that cannot be removed cystoscopically; this is the gold standard for making a patient stone-free.<sup>31</sup> It is rare that a patient requires open ureterolithotomy or nephrolithotomy.

## Clinical and metabolic evaluation

### The first stone

Disagreement exists about the benefit of performing a full clinical and metabolic evaluation after the first episode of urolithiasis.<sup>32</sup> Assuming the patient is willing to participate in a diagnostic work-up and adhere to treatment recommendations, we encourage a full evaluation for all stone formers for the following reasons.

- First, although recurrence rates are uncertain, the chance of passing a second calcium stone may be as high as 30% to 50% after 5 years.<sup>1,33,34</sup>
- Second, treatable systemic diseases of clinical importance, such as osteoporosis and primary hyperparathyroidism, may be diagnosed during the metabolic evaluation.<sup>35</sup>
- Third, allowing a stone former to progress to recurrence may hinder the effectiveness of subsequent prophylaxis.<sup>36</sup>
- Finally, some analyses suggest that medical prevention may be cost saving.<sup>37</sup>

For individuals suffering from a large stone burden or at very high risk of recurrence, little disagreement exists about the utility of a complete evaluation. Specifically, patients with a large first stone (eg, >10 mm), multiple stones on initial imaging, recurrent disease, or disease requiring invasive intervention should be offered a complete evaluation.

### Stone composition

Because treatment recommendations vary by stone type, every effort should be made to retrieve a passed stone for chemical analysis. Approximately 80% of kidney stones contain calcium and the majority of calcium stones consist primarily of calcium oxalate.<sup>27</sup> Although most calcium oxalate stones contain some calcium phosphate, only 5% of kidney stones have hydroxyapatite or brushite (calcium monohydrogen phosphate) as their main constituent.<sup>27,38</sup> About 10% of calcium stones contain some uric acid.<sup>27</sup> Other types of stones, such as pure uric acid, struvite, and cystine, are less common but merit careful attention because of recurrence risk.

### History and laboratory testing

Evaluation should be directed toward identifying risk factors for stone formation with the goal of devising appropriate, individualized therapy (Table 1 delineates risk factors for calcium oxalate urolithiasis). The evaluation should start with a detailed history, which will provide information crucial for treatment recommendations. The following information should be elicited:

<b>Table 1: Risk factors for calcium oxalate stone formation</b>	
<b>Urinary risk factors</b>	
<b>High levels</b>	<b>Low levels</b>
Calcium	Citrate
Oxalate	Total volume
Uric Acid	
<b>Dietary risk factors</b>	
<b>High intake</b>	<b>Low intake</b>
Sodium	Fluid
Animal protein	Calcium (dietary)
Sucrose	Potassium
Calcium (supplemental)	Phytate
Vitamin C	Magnesium
Oxalate	
<b>Other risk factors</b>	
Obesity	
Gout	
Anatomic abnormalities (eg, medullary sponge kidney)	

dietary intake and medication use prior to the stone event, family history of stone disease, total number of stones, evidence of residual stones, number and types of procedures, and types and success of previous preventive treatments. More severe disease will lower the threshold for early medical intervention in addition to recommended dietary changes.

The metabolic evaluation should include a determination of serum electrolytes, creatinine, calcium, phosphorus, and uric acid. Although usually normal, a low serum bicarbonate should prompt consideration of type 1 renal tubular acidosis, which is classically associated with calcium phosphate stones. Intact parathyroid hormone should be measured if the serum calcium is elevated or in the high-normal range, if the serum phosphorus is low, or if the urinary excretion of calcium is elevated.

A urinalysis should be performed as part of the initial evaluation. A urine pH >7 with phosphate crystals suggests calcium phosphate or struvite stones. The presence of hexagonal cystine crystals is pathognomic for cystinuria. Uric acid or calcium oxalate crystalluria can be seen in normal subjects and is, therefore, less informative.

The cornerstone of the metabolic evaluation is the 24-hour urine collection. In order to tailor initial intervention and determine response to treatment, 24-hour urine collections are necessary, even if the stone composition is known. The factors that should be measured include total volume, creatinine (to assess the adequacy of collection), calcium, oxalate, citrate, uric acid, sodium, potassium, phosphorus, and pH. Estimates of relative supersaturation, based on measurements of the urine factors above, are offered by some laboratories and should be obtained whenever possible. The relative supersaturation can guide the selection of, and response to, therapy and is particularly useful if the composition of the stone is

unknown. Because of controversy over the clinical importance of Pak's classic schemata,<sup>39</sup> most clinicians do not attempt to categorize individuals with idiopathic hypercalciuria into absorptive, resorptive, or renal subtypes.

The stone-forming patient should wait at least 6 weeks before performing a 24-hour urine collection because individuals frequently alter their dietary habits immediately after an episode of urolithiasis.<sup>40</sup> In addition, 2 collections are necessary at baseline because of substantial day-to-day variability in urinary parameters: a single 24-hour urine collection is insufficient.<sup>41</sup>

### *The normal metabolic evaluation*

Because kidney stones can remain asymptomatic for many years before they pass into the ureter and cause renal colic, the actual time of stone formation is usually unknown. Therefore, the metabolic evaluation may be completely normal. In this case, no intervention is required. However, repeat imaging in one year to assess active stone formation, in conjunction with another 24-hour urine, is warranted in patients who present with severe disease.

## **Prevention of stone recurrence – calcium oxalate urolithiasis**

### *Dietary recommendations*

Although dietary intervention has been shown to decrease the rate of calcium stone recurrence in randomized controlled trials,<sup>42,43</sup> controversy and uncertainty exist about the benefit of many dietary modifications. These controversies, as well as the available evidence supporting our dietary recommendations, have been summarized by our group in a previous edition of *Nephrology Rounds* and elsewhere.<sup>44</sup>

Dietary advice should be based on the results of the 24-hour urine collection. For example, dietary oxalate restriction or discontinuation of vitamin C supplementation is unnecessary in a calcium oxalate stone former with a low urinary excretion of oxalate. Dietary recommendations are listed in Table 2. Of note, there is no evidence that dietary calcium restriction alone is helpful in preventing the formation of calcium stones and there is substantial evidence that it may be harmful. Observational data showing an inverse relation between dietary calcium and the risk of incident kidney stones suggests that dietary calcium may bind to oxalate in the gut, thereby limiting intestinal oxalate absorption (and subsequent urinary oxalate excretion). Indeed, the inhibitory effect of calcium ingestion on urinary oxalate excretion has been demonstrated in oxalate loading studies.<sup>45,46</sup> The role of calcium supplements deserves comment because their use is so common. A patient with calcium urolithiasis who wishes to continue calcium supplementation should collect 24-hour urine samples on and off the supplement. If the urinary supersaturation of the calcium salt in question increases during the period of supplement use, the supplement should be discontinued or at least the dosage should be reduced.

**Table 2: Dietary and pharmacologic treatments according to urinary abnormality**

Urinary abnormality	Dietary Changes	Medication
High calcium	Adequate dietary calcium intake Reduce animal protein intake Reduce sodium intake <2.4 g/d Reduce sucrose intake	Thiazide
High uric acid	Reduce purine intake	Allopurinol
High oxalate	Avoid high oxalate foods Adequate dietary calcium intake	Colestipol? High dose pyridoxine?
Low citrate	Increase fruit and vegetable intake Reduce animal protein intake	Alkali (eg, K citrate)
Low volume	Increase total fluid intake	Not applicable

Most stone formers will be encouraged to increase their fluid intake. Based on the urine volume, the patient should be instructed on how many additional 8 oz glasses of water to drink each day with the goal of producing over 2 liters of urine daily. Decreasing purine intake (meat, chicken, and seafood) will reduce the urinary excretion of uric acid. For low urinary citrate, the patient should increase intake of potential alkali (fruits and vegetables) and decrease intake of acid-producing foods such as animal protein.

### Drug therapy

Medications (Table 2) are indicated for the stone-forming patient whose urinary abnormalities persist despite attempted lifestyle changes. Because the goal of therapy is to prevent the additional formation and growth of calcium oxalate stones, and because an existing calcium stone will not dissolve, the passage of another stone does not necessarily reflect therapeutic failure. As with dietary modification, the 24-hour urine collection is essential to select intervention and to gauge the success or failure of treatment.

Thiazide diuretics can lower urinary excretion of calcium by 150 mg/day or more, and treatment with a thiazide may reduce the rate of stone recurrence by up to 90%.<sup>47, 48</sup> The diuretic dose is usually started at 25 mg/day of chlorthalidone or hydrochlorothiazide (or its equivalent), but many patients will require 50 to 100 mg/day to achieve satisfactory reductions in urinary calcium excretion. Without dietary sodium restriction, the reduction in urinary calcium excretion obtained with treatment may be inadequate. In addition, serum potassium levels should be closely monitored during therapy because hypokalemia can

result in a decrease in urinary citrate excretion. Of interest, thiazide diuretics may be beneficial even in patients without overt hypercalciuria.<sup>47, 48</sup>

Calcium stone formers with hyperuricosuria may be treated with allopurinol (100 to 300 mg per day). Allopurinol may reduce new stone formation by up to 80% in individuals with isolated hyperuricosuria.<sup>49</sup> Theoretically, alkali therapy with potassium citrate may also be beneficial, since raising the urine pH will convert uric acid to the more soluble urate salt (thereby decreasing the potential formation of a uric acid nidus). However, no trials have addressed this intervention.

Urinary citrate excretion can be increased by administration of an alkali load in the form of potassium citrate or potassium bicarbonate (30 to 80 mEq per day).<sup>50, 51</sup> In one study, stone recurrence in a group of hypocitraturic patients treated with potassium citrate decreased from 1.2 to 0.1 per patient year (versus no change with placebo).<sup>51</sup>

To date, no satisfactory drug treatment exists to decrease the urinary excretion of oxalate. For patients with increased intestinal absorption of oxalate secondary to bowel disease, clinicians sometimes administer oxalate binders such as calcium carbonate or colestipol. Experimental therapies include the oral administration of oxalate consuming bacteria and the administration of high dose pyridoxine (to reduce the endogenous production of oxalate).

### Prevention of stone recurrence – other stone types

For the less common stone types, little data exist to support the role of specific dietary recommendations, and trial evidence for medical therapies is generally absent. Therefore, the following recommendations are based on the pathophysiology of the different stone types and clinical experience.

#### Uric acid stones

Decreasing the consumption of meat, chicken, and seafood will decrease purine intake and uric acid production and will also reduce the amount of endogenous acid production from the metabolism of sulfur-containing amino acids. Higher intake of fruits and vegetables, which are high in potential base (such as citrate), may raise urine pH and reduce the risk of uric acid crystal formation.

Alkali supplementation is the most effective treatment for pure uric acid stones.<sup>21</sup> If the urine pH is maintained at  $\geq 6.5$  (which may require 90 to 120 mEq of supplemental alkali per day), uric acid stones will dissolve. Lower doses of alkali can be administered to prevent new uric acid stone formation. If the patient has marked hyperuricosuria and is unable to maintain an elevated urine pH, allopurinol is the second line choice.

### Cystine stones

Restricting dietary sodium may reduce the urinary excretion of cystine. Because the solubility of cystine increases as urinary pH rises, fruit and vegetable consumption may be beneficial. There is little evidence to support the dietary restriction of proteins high in cystine. However, reducing animal protein intake may be beneficial by increasing urine pH.

Medications such as tiopronin and penicillamine increase the solubility of filtered cystine, but have a high frequency of adverse side effects. Supplemental alkali may provide some benefit by increasing urinary pH, but is rarely sufficient as the sole treatment.

### Struvite stones

Struvite stones only form in the setting of upper urinary tract infection with urease-producing bacteria. Struvite stones can be very large (“staghorn calculi”) and usually require urologic intervention. In addition to complete removal of all residual fragments, prevention of recurrent urinary tract infection is crucial to prevent recurrence. Acetohydroxamic acid inhibits urease, but has frequent and serious side effects.

### Calcium phosphate stones

Information on dietary factors related to calcium phosphate stone formation is limited. Patients with type 1 renal tubular acidosis (RTA) may benefit from the administration of alkali supplementation, generally in the form of potassium citrate. However, alkali supplementation should be used with caution, since an increase in urinary pH can increase the risk of calcium phosphate crystal formation. Thiazides can be used to reduce the excretion of urinary calcium, using a similar approach to that recommended for calcium oxalate stones.

### Conclusion

Urolithiasis is common, costly, and painful. Advances in imaging and urologic techniques have improved the diagnosis and management of stone disease in the acute setting. Because recurrence is common, a clinical and metabolic evaluation should be offered to patients willing to adhere to specific dietary and/or pharmacologic recommendations. Even if stone composition is known, a thorough evaluation requires at least two 24-hour urine specimens collected at least 6 weeks after resolution of an acute episode. The initial choice of dietary or medical intervention should be tailored to the individual patient on the basis of stone type, if known, and the 24-hour urine results. Subsequent 24-hour urine collections are necessary to gauge the adequacy of treatment. Prevention of stone recurrence is an achievable goal with individualized therapy and regular follow-up.

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## Upcoming Scientific Meetings

16-19 February 2006

### 7<sup>th</sup> International Conference on New Trends in Immunosuppression and Immunotherapy

Berlin, Germany

CONTACT: Secretariat Kenes International, +41 22 908 0488

Fax: +41 22 732 2850

Email: immuno@kenes.com

Web site: www.kenes.com/immuno

26-28 February 2006

### 26<sup>th</sup> Annual Dialysis Conference

San Francisco, CA

CONTACT: Elaine Rogers

Tel.: 573 882-9973 or 882-4105

573 882-5666

E-Mail: dialysis@health.missouri.edu

Website: <http://www2.muhealth.org/~dialysis/>

1-4 March 2006

### 3<sup>rd</sup> NephroAsia 2006

Singapore, Singapore

CONTACT: NephroAsia 2006 Congress Secretariat

Tel.: 65-62-990-200

Fax: 65-62-517-555

Email: nephroasia@nkfs.org

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*Dr. Taylor and Dr. Curhan have no disclosures to declare related to the enclosed CME program.*

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This publication is made possible by an educational grant from

**Amgen Inc.**

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