

Urinary stones can be classified as calcium-based, struvite, uric acid, or cystine stones, and mixed stone types exist. Stone composition will determine strategies to prevent recurrence.

# Kidney Stones

## Current Diagnosis and Management

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The lifetime risk for nephrolithiasis is estimated between 15% and 25%, and changes in diet and lifestyle may have contributed to increased incidence in women and adolescents. The high rate at which urinary stones recur—and the potential in patients with chronic stone disease for impaired kidney function—should prompt primary care providers to seek a fuller understanding of urinary stone disease.

**K**idney or urinary tract stones (whose presence is referred to as *nephrolithiasis*) are hard, crystalline mineral concretions that form within the kidney or the urinary tract. They are a common problem, with an estimated annual incidence of 1% and a lifetime risk of 15% to 25%; this constitutes a significant health care burden, particularly for people of working age.<sup>1</sup>

Nephrolithiasis is currently more prevalent in men than in women (13% vs 7%, respectively), and it is three to four times more likely to present in white than nonwhite patients.<sup>2</sup> However, recent epidemiologic data suggest an alarming increase in the number of women and adolescents primarily diagnosed with stone disease.<sup>3-6</sup> The pattern of increasing incidence in women can be attributed in part to changes in diet and lifestyle.<sup>4,5</sup> Figure 1<sup>7</sup> represents the prevalence of stone disease, specific to gender and race.<sup>2,4</sup>

Due to kidney stones' relatively common occurrence, the diagnosis, management, and prevention of stone disease have become increasingly relevant for the primary care practitioner. In the course of stone disease management, the clinician should be aware of a vital fact: *Stones have a tendency to recur.*<sup>1</sup> Indeed, evidence suggests that following an initial diagnosis of nephrolithiasis, the probability of kidney stone recurrence increases to nearly 50% after five years.<sup>8</sup>

Even more concerning, evi-

dence from several studies suggests that patients with a history of stone disease have a higher probability of experiencing a significant reduction in renal function (ie, decrease in glomerular filtration rate) and hence end-stage renal disease, when compared with non-stone formers.<sup>9-11</sup> This accentuates the importance of early diagnosis, treatment, and initiation of steps to prevent further recurrence of this condition.

### PATHOGENESIS

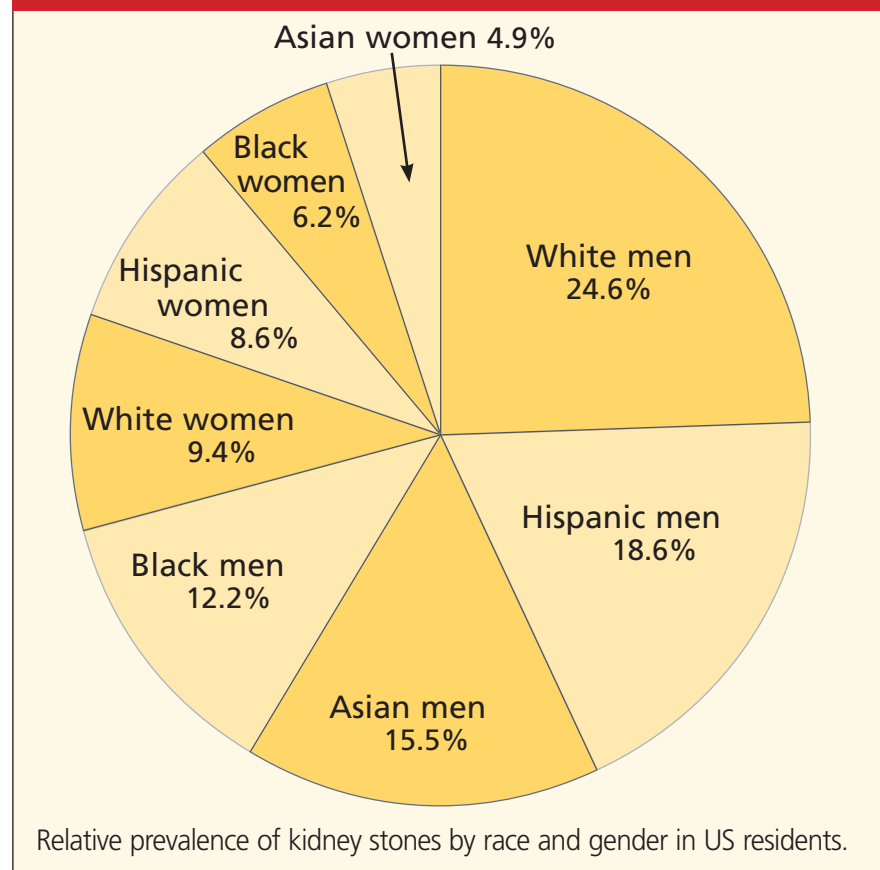
Stones in the urinary tract develop under specific urinary conditions, including supersaturation of the urine with stone-forming ions (ie, calcium, oxalate, uric acid, and phosphate) and deficiency of urinary stone inhibitors (citrate, magnesium, zinc, macromolecules, and pyrophosphate). Stone formation occurs in a mucoprotein matrix that attaches to the renal epithelium. Urine becomes supersaturated as a result of increasing levels of solutes (such as the stone-forming ions) and/or decreasing free water volume. When the concentration of stone-forming ions exceeds solubility in the urine (equilibrium solubility product), these ions can combine to form crystals.<sup>12,13</sup>

Stones are typed based on the ion composition of their crystals (see Table 1,<sup>2,12</sup> page 33). Once crystals are formed, they can also aggregate with other crystals, developing into a calculus.<sup>12</sup> Urinary pH influences ion crystallization: Alkaline urine favors formation of calcium and/or

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**FIGURE 1**

**Ethnic diversity in the prevalence of kidney stones<sup>7</sup>**



Adapted from Romero et al. *Rev Urol.* 2010.<sup>7</sup>

phosphate stones, whereas acidic urine favors uric acid and cystine stone formation.<sup>13</sup>

Kidney stones can be divided into four broad types: *calcium-based*, *struvite*, *uric acid*, and *cystine* stones (see Figure 2, page 33). Among these, calcium-based stones are by far the most common, with nearly 80% of stones composed of calcium compounds (usually calcium oxalate, and rarely calcium phosphate).<sup>4</sup> The etiologies of these four types are vastly different, and prevention of stone formation must be tailored to the stone type. Once stones form, however, the appropriate treatment strategies have many similarities.

### RISK FACTORS

Specific risk factors for stone formation vary widely and are unique to the type of stone. A thorough history, including a family or personal history of stone disease and dietary history,

must be part of the initial work-up when a patient is being evaluated for stone disease; patients with any of these risk factors should be investigated further.

The risk factors for stone disease can be broadly categorized as either individual risk factors or dietary risk factors.

#### Individual Risk Factors

A positive family history increases the risk for stone formation by two- to three-fold. Other individual risk factors include congenital anatomic defects, such as *medullary sponge kidney*, *horseshoe kidney*, and *ureteropelvic junction obstruction* (UPJ).<sup>14-16</sup> These can cause obstruction that leads to urinary stasis, and subsequently to stone precipitation.

Certain systemic disorders (eg, hyperparathyroidism) and situations have also been associated with stone disease and should be considered risk factors. (See Table 2,<sup>12,17</sup> page 34).

In patients who undergo gas-

**TABLE 1**

**Prevalence and Composition of Common Renal Stones<sup>2,12</sup>**

Stone type	Prevalence	Risk factors / contributing factors
Calcium oxalate	36% – 70%	Hypercalciuria Hypercalcemia Hyperoxaluria Hypocitraturia Gouty diathesis Low urine volume
Calcium phosphate	6% – 20%	Distal renal tubular acidosis Hyperparathyroidism Low urine volume Urinary tract infections
Mixed calcium oxalate and phosphate	11% – 31%	(See above)
Struvite (infection-related); includes “staghorn calculi”	6% – 20% (twice as common in women as men)	Urinary tract infections Urea-splitting organisms (eg, <i>Proteus</i> , <i>Klebsiella</i> , <i>Pseudomonas</i> , <i>Staphylococcus</i> )
Uric acid	17%	Gouty diathesis Arthritis Nephropathy Hyperuricosuria Hyperuricosemia Low urine volume Urinary pH < 6 Obesity Myeloproliferative disorders Alcohol abuse Congenital metabolic errors
Cystine	0.5% – 3% (predominantly in children, teens)	Cystinuria Congenital intestinal tract defect Congenital renal transport error
Other	1% – 4%	Use of specific medications (including indinavir, ephedrine, triamterene)

Sources: Schade and Faerber. *Prim Care*. 2010<sup>2</sup>; Johri et al. *Nephron Clin Pract*. 2010.<sup>12</sup>

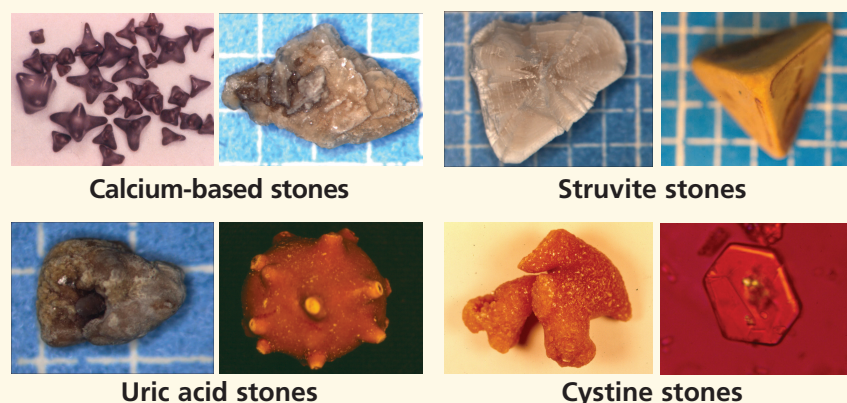
trointestinal bypass surgery, the development of hyperoxaluria, hypercalciuria, and decreased urinary volume are associated with an increased risk for stone formation,<sup>18,19</sup> and these patients should be watched for this development. Obesity and weight gain are directly proportional to nephrolithiasis

risk, especially in women.<sup>4,20</sup>

Environment plays a very important role in stone formation. Persons who live in a hot, arid climate, for example, and those who work outdoors in hot weather are at increased risk for stone formation due to excessive fluid loss from sweating.<sup>2,4,7</sup> (In regions where the risk for kidney

**FIGURE 2**

**Commonly occurring kidney stones**



Courtesy of Louis C. Herring Company Kidney Stone Analysis Laboratory, www.herringlab.com.

stone formation is high, Romero et al<sup>7</sup> predict, nephrolithiasis incidence could rise from 40% to 56% by 2050 as a result of the effects of global warming.)

Lastly, an individual’s ability (or inability) to metabolize calcium salts plays a vital role in the pathogenesis of stone disease. Intestinal calcium absorption is a major determinant of hypercalciuria, as nearly 90% of ingested calcium is absorbed in the intestines. People can broadly be divided into high or low calcium absorbers. Hypercalciuria (mean urinary calcium excretion  $\geq 300$  mg/d in men and  $\geq 250$  mg/d in women on a 1,000-mg/d calcium diet) is detected in 20% to 40% of those with calcium stones.<sup>21-23</sup> Hypocitraturia (mean urinary citrate excretion  $\leq 320$  mg/d) and hyperoxaluria (mean urinary oxalate excretion  $> 45$  mg/d) can also increase the risk for stone formation.<sup>12,24</sup>

**Dietary Risk Factors**

These are primarily related to fluid intake and dietary calcium.<sup>7,17,25,26</sup> Drinking less than 1 L of fluids daily is associated with an increased risk for forming stones; this risk is magnified when the urine volume is also decreased.<sup>7,17,27</sup> Increased dietary intake of animal protein can elevate the risk for formation of uric acid stones as a result of el-

evated urinary calcium and uric acid and decreased urinary citrate.<sup>17</sup>

Low dietary calcium ingestion, resulting in increased oxalate absorption, can also exacerbate the risk for stones.<sup>7,27</sup> By contrast, a diet high in calcium ( $\geq 1,200$  mg/d) reduces the risk for calcium oxalate stone recurrence,<sup>17</sup> although the effectiveness of supplemental calcium has been questioned.<sup>26-28</sup>

Patients who are advised to make specific dietary adjustments should later undergo repeat urine chemistries to determine the effectiveness of these changes.<sup>17</sup>

**CLINICAL PRESENTATION**

Nephrolithiasis typically presents with colicky flank pain, often accompanied by nausea and vomiting.<sup>29</sup> The pain radiates to the ipsilateral groin, and the patient typically has difficulty finding a comfortable position. Nephrolithiasis may also present with chronic, episodic flank pain or may even be asymptomatic.<sup>30</sup>

Physical examination may reveal signs of severe pain, such as tachycardia and hypertension. Presence of fever indicates associated urinary tract infection and possibly pyelonephritis. Some larger stones can cause urinary tract obstruction; if ob-



**TABLE 2**  
Systemic Disorders and Circumstances Associated With Stone Disease<sup>12,17</sup>

Disorder/condition	Associated mechanism(s)
Primary hyperparathyroidism (present in 5% of stone formers)	Hypercalcemia Hypercalciuria
Urinary tract infections	Precipitation of calcium phosphate and magnesium ammonium phosphate in alkaline urine Struvite stones
Distal renal tubular acidosis	Decreased urine ammonium excretion Low urinary pH
Chronic inflammatory bowel disease	Increased oxalate absorption
Gout (doubles the risk for both uric acid and calcium oxalate stones)	Hyperuricemia
Insulin resistance	Decreased urine ammonium excretion Low urinary pH
History of ileostomy	Loss of bicarbonate and fluid Low urine volume Low urinary pH
Prolonged immobilization	Hypercalciuria due to bone loss Potential for urinary stasis
Congenital, surgical, anatomical defects	Urinary stasis

Sources: Johri et al. *Nephron Clin Pract.* 2010<sup>12</sup>; Curhan. *Nephrology Rounds.* 2004.<sup>17</sup>

struction occurs along with a preexisting urinary tract infection, it can potentially lead to pyelonephritis, pyonephrosis, and eventually urosepsis—a potentially life-threatening condition that requires immediate surgical drainage.<sup>31</sup>

Before a diagnosis of renal stones can be confirmed, care should be exercised to rule out the differentials, including abdominal aortic aneurysm, appendicitis, bowel obstruction, cholecystitis, drug-seeking behavior (eg, painkiller addiction), gastritis, mesenteric ischemia, musculoskeletal pain, ovarian abscess, ruptured ovarian cyst, pelvic inflammatory disease, py-

elonephritis, and UPJ.<sup>2,32,33</sup> All patients with suspected nephrolithiasis should be carefully evaluated using laboratory and radiologic investigations.

#### LABORATORY EVALUATION

The goals in this two-step process are to confirm the diagnosis of nephrolithiasis, then to identify the composition of the stones formed and the associated risk factors.

#### Initial Evaluation

Tests include dipstick urine assessment, serum chemistries, and a complete blood count (CBC). Urine dipstick assessment may be positive for blood,

**TABLE 3**  
Evaluation for Stone Disease<sup>2</sup>

Serum studies	Spot urine studies
Creatinine	Spot urine cystine screen (qualitative)
Blood urea nitrogen	Spot protein creatinine ratio (only reliable if creatinine is stable)
Calcium	Fasting urine screen for pH
Phosphorus	Urinary electrolytes
Parathyroid hormone levels	
Bicarbonate	
Fasting blood glucose or A1C	
Vitamin D 25	
Protein content	
Order a 24-hour urine collection; measure the metabolic determinants of stones and calculate supersaturation indices. These will indicate the composition of the stone, after which recommendations can be made, based on these findings.	
The 24-hour urine collection should be repeated at a later date.	

Source: Schade and Faerber. *Prim Care.* 2010.<sup>2</sup>

protein, or leukocyte esterase, indicating stones or fragments of stones present in the urinary tract. While nearly 10% of patients with stone disease exhibit gross hematuria, nearly 90% of patients have microscopic hematuria.<sup>2</sup>

Urine osmolality should be reviewed to assess urine concentration. Serum chemistries should be ordered to evaluate kidney function. Elevated creatinine may indicate acute rather than chronic kidney disease. Electrolytes and carbon dioxide should be measured to evaluate the kidneys' ability to concentrate urine and maintain an acid-base balance. The CBC may reveal mild leukocytosis in nephrolithiasis; presence of significant leukocytosis indicates infection.<sup>2</sup>

#### Secondary Evaluation

This step begins with a thorough review of the patient's medical record and a detailed patient interview to ascertain all risk factors for stone formation (as summarized in Table 1). Specific

studies to be considered are mentioned in Table 3.<sup>2</sup> This evaluation is critical to prevent formation of future stones and the associated complications. In the patient with a history of stone recurrence or stone formation of identified cause, evaluation is needed for three metabolic abnormalities—hypercalciuria, hyperuricosuria, and hypocitraturia—as these conditions predispose patients to recurrent stone formation.<sup>1,25,34</sup>

The patient should also be encouraged to collect stones passed for further clinical evaluation. Infrared spectroscopy or quantitative wet analysis is used to identify the specific composition of the stone.<sup>32,35</sup>

#### Radiologic Evaluation

Radiologic evaluation of stones is currently performed through plain x-rays, ultrasonography, and noncontrast spiral CT.<sup>12,32,33</sup> When a patient presents with acute signs of nephrolithiasis, a plain film x-ray of the kidneys, ureters, and bladder (KUB) is acceptable as the first imaging

**FIGURE 3**

Bilateral staghorn calculi shown on plain x-ray



Figure courtesy of John C. Sabatino, MD.

study, as it is inexpensive and available in most areas.<sup>33</sup> Plain film KUB x-rays will identify calcium oxalate, calcium phosphate, struvite, and cystine stones. However, the sensitivity of plain film x-rays has been documented between 24% and 59%, and stones that overlie a bone may be missed.<sup>32,36</sup> (See Figure 3.)

Hence, because of these limitations and the increasing availability of noncontrast spiral CT, noncontrast spiral CT is now the most commonly used and useful test in the diagnosis of kidney stones (sensitivity, 95% to 100%).<sup>32,36</sup> Spiral CT accurately defines the size as well as the location of stones, and may additionally rule out other differential diagnoses (see Figures 4a, 4b, and 4c). Historically, IV pyelograms and urograms were considered useful in locating urinary tract stones and diagnosing related complications,<sup>12</sup> but these modalities carry additional risks related to IV contrast dye and radiation exposure. As a result, they have been almost completely replaced by noncontrast spiral CT because

of ease of use and reduced risks.<sup>33</sup>

Stones may also be seen on renal ultrasound—particularly uric acid stones, which are radiolucent (see Figure 5). Ultrasound is appropriate for evaluation of patients whose exposure to radiation should be limited, such as children or pregnant women. In addition to plain film x-rays, renal ultrasound may also be useful for surveillance of stones.<sup>12</sup>

### TREATMENT

Nephrolithiasis treatment varies between acute and chronic care. *Acute care* for nephrolithiasis involves management of acute pain and urinary obstruction, as well as patient stabilization. *Chronic care* includes prevention of recurrence and management of risks.

### Acute Management

Patients who present with acute nephrolithiasis most often require fluid administration, aggressive pain management, and treatment for nausea or vomiting.<sup>31,32</sup> Most ureteral stones measuring 5.0 mm or less will

**FIGURE 4**

Findings on noncontrast spiral CT



A. Bilateral staghorn calculi  
B. Left ureteral stone  
C. Left hydronephrosis

Figures courtesy of John C. Sabatino, MD.

typically pass spontaneously within a few weeks,<sup>1,29</sup> but larger stones usually require intervention—in some cases, surgery.

Patients should be hospitalized if they require IV fluids or pain management. Isotonic IV fluids should be given to increase the urine volume and facilitate passage of stones. Care must be

taken to monitor fluids, as patients with kidney stones may have a limited ability to urinate (due to urinary obstruction and/or acute or chronic renal failure). Whenever possible, all urine should be strained to collect any stones for analysis.

One new strategy to assist with stone passage is medical ex-

**FIGURE 5**

Kidney stones shown on ultrasound

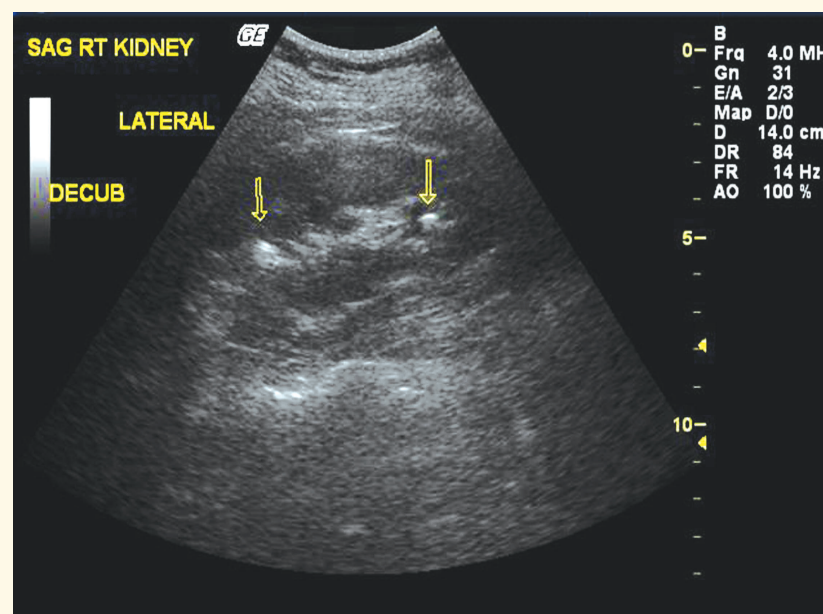
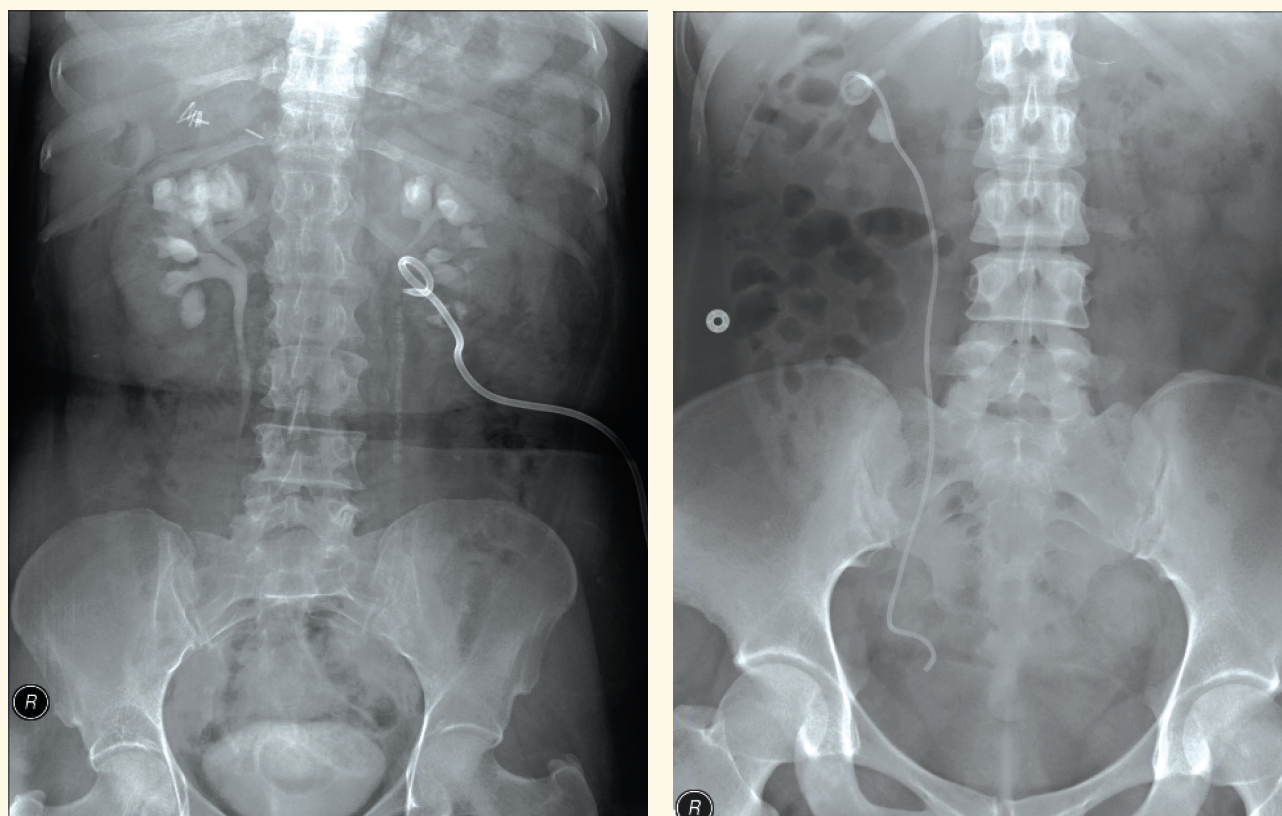


Figure courtesy of John C. Sabatino, MD.



FIGURE 6

For temporary relief of obstruction



A. Nephrostomy tube shown on IV pyelogram

B. Double-J ureteral stent shown on plain x-ray

Figures courtesy of John C. Sabatino, MD.

pulsive therapy (MET), using calcium channel blockers (eg, nifedipine) or  $\alpha$ -blockers (eg, tamsulosin).<sup>1,37</sup> While there is conflicting evidence regarding the efficacy of calcium channel blockers for MET, one meta-analysis revealed a 29% improvement in stone passage with  $\alpha$ -blockers.<sup>1,38</sup>

Pain management can often be accomplished with NSAIDs (eg, ketorolac, diclofenac).<sup>29</sup> Since this class of medications can compromise renal function, however, they must be used with caution. Many patients require narcotic medications to control pain adequately.<sup>39-41</sup> Antiemetic agents (such as the H<sub>1</sub>-receptor blocker dimenhydrinate<sup>42</sup>) should be administered to control nausea and vomiting.

*Surgical and interventional management.* Surgical intervention may be required if stones are too large to pass spontane-

ously (typically  $\geq 8$  mm); if they cause acute renal obstruction; or if they are located at a site with a potential for complications or can lead to persistent symptoms without evidence that they are passing.<sup>1,3</sup> Renal obstruction should be treated aggressively to preserve renal function.

The type of intervention chosen depends on the size and location of the stone, as well as the presence or absence of obstruction. Stones that measure less than 20 mm are commonly treated with extracorporeal shockwave lithotripsy (unless they overlie the sacroiliac joint), whereas patients with larger or more complex stones may require percutaneous nephrolithotomy. Nonobstructive or uncomplicated ureteral stones may be managed medically, whereas obstructive or complicated ureteral stones require placement of a stent or a nephrostomy tube

until they can be removed by endoscopic surgery.<sup>29,43</sup>

Obstruction, which may be partial or complete, is more likely when stone size exceeds 10 mm.<sup>44</sup> Signs of obstruction include sudden-onset, excruciating flank pain that radiates to the groin, along with nausea and vomiting (renal colic). Larger obstructive stones, such as *staghorn calculi* (as shown in Figures 3 and 4a), can present with symptoms of a urinary tract infection, mild flank pain, or hematuria.<sup>33</sup>

Presence of signs of obstruction or infection mandates emergent treatment. Infections of the urinary tract (as serious as pyelonephritis or urosepsis) should be treated with antibiotics: initially with broad coverage, according to the appropriate guidelines for urinary tract infections, then tailored to the results of urine cultures. Ob-

struction can be relieved directly by nephrostomy tubes (and/or stents) or by interventions in which the stone is removed and normal urinary flow is restored.

Typically, endoscopy is used for direct removal of stones that cause obstruction.<sup>44</sup> Nephrostomy tubes and ureteral stents (see Figures 6a and 6b) are placed to relieve obstruction temporarily and provide an alternate route for drainage of urine. The goal is to prevent renal damage until the obstruction can be relieved. Stents can remain in place for several months, but nephrostomy tubes are associated with a higher risk for infection (because they are externalized), and duration of use should be limited to only a few weeks.<sup>12,29,38</sup>

Stents are also associated with infections, but coated stents are available to reduce infection. As with any catheter material inserted into the urinary tract, ureteral stents are a prime location for development of a persistent bacterial biofilm, thus leading to infection. Recent advances in stent manufacturing have included coating stents with various biomaterials to decrease the development of this bacterial biofilm. In a preliminary study in 10 patients using a diamond-like, carbon-coated ureteral stent, Laube et al<sup>45</sup> demonstrated a reduction in formation of this biofilm, hence lowering the probability of stent-induced infection.

### Chronic Stone Management

As previously mentioned, one of the seminal characteristics of stone disease is its ability to recur. After incidental detection of kidney stones through routine diagnostic procedures, the risk for recurrence in patients who do not receive chronic medical management is 30% to 40% within five years.<sup>17,28</sup> In treated patients, by comparison, this risk falls by approximately 50%.<sup>17,26</sup>

Patients with a history of stone recurrence must be evaluated for metabolic defects that precipitate stones, since their risk for chronic kidney disease is increased.<sup>34</sup> All patients with a history of stone disease should be instructed to increase their fluid intake to maintain a daily urinary output of at least 2.5 L, unless contraindications exist.<sup>34</sup>

In patients with *calcium-based stones* who do not benefit from conservative treatment (ie, a low-sodium diet and other dietary modifications), thiazide diuretics may help reduce urinary calcium.<sup>1,46</sup>

*Struvite stones* can be prevented through use of long-term antibiotics to reduce the risk for urinary tract infection and by maintaining urinary pH levels below 6.0.<sup>17,27,34</sup>

For patients with *uric acid stones*, allopurinol may be prescribed to lower uric acid levels; moreover, the solubility of uric acid is greatly increased at higher pH, so it is beneficial to treat these patients with citrate to maintain their urinary pH above 6.0.<sup>47,34</sup>

Ensuring a high urine output ( $\geq 4 \text{ L/d}^{34}$ ) and alkalinizing urine can help prevent recurrence of *cystine stones*.<sup>17,33</sup> Treatment with potassium citrate has been shown to maintain a urinary pH of 6.5 to 7.0.<sup>34</sup>

## CONCLUSION

The ever-increasing significance of nephrolithiasis has mandated an organized and systematic management approach. Indeed, the diagnosis and initial therapy for kidney stones have undergone considerable evolution in recent years. The basic tenets of nephrolithiasis management include early diagnosis and pertinent treatment as well as adequate prophylaxis to prevent subsequent stone recurrence. **CR**

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