Urolithiasis/Nephrolithiasis: What’s It All About?

Joan Colella  
Eileen Kochis  
Bernadette Galli  
Ravi Munver

The term nephrolithiasis (kidney calculi or stones) refers to the entire clinical picture of the formation and passage of crystal agglomerates called calculi or stones in the urinary tract (Wolf, 2004). Urolithiasis (urinary calculi or stones) refers to calcifications that form in the urinary system, primarily in the kidney (nephrolithiasis) or ureter (ureterolithiasis), and may also form in or migrate into the lower urinary system (bladder or urethra) (Bernier, 2005). Urinary tract stone disease has been documented historically as far back as the Egyptian mummies (Wolf, 2004).

Prevalence

As much as 10% of the U.S. population will develop a kidney stone in their lifetime. Upper urinary tract stones (kidney, upper ureter) are more common in the United States than in the rest of the world. Researchers attribute the incidence of nephrolithiasis in the United States to a dietary preference of foods high in animal protein (Billica, 2004).

Age and Gender

The literature reflects the incidence of kidney (renal) stone formation to be greater among white males than black males and three times greater in males than females. Although kidney stone disease is one-fourth to one-third more prevalent in adult white males than black males, black males demonstrate a higher incidence of stones associated with urinary tract infections caused by urea-splitting bacteria (Munver & Preminger, 2001).

Kidney stones are most prevalent between the ages of 20 to 40, and a substantial number of patients report onset of the disease prior to the age of 20 (Munver & Preminger, 2001; Pak, 1979, 1987). The lifetime risk for kidney stone formation in the adult white male approaches 20% and approximately 5% to 10% for women. The recurrence rate for kidney stones is approximately 15% in year 1 and as high as 50% within 5 years of the initial stone (Munver & Preminger, 2001; Spirnak & Resnick, 1987).

PATHOPHYSIOLOGY OF NEPHROLITHIASIS

Any factor that reduces urinary flow or causes obstruction, which results in urinary stasis or
reduces urine volume through dehydration and inadequate fluid intake, increases the risk of developing kidney stones. Low urinary flow is the most common abnormality, and most important factor to correct with kidney stones. It is important for health practitioners to concentrate on interventions for correcting low urinary volume in an effort to prevent recurrent stone disease (Munver & Preminger, 2001; Pak, Sakhae, Crowther, & Brinkley, 1980).

Contributing Factors Of Nephrolithiasis

Sex. Males tend to have a three times higher incidence of kidney stones than females. Women typically excrete more citrate and less calcium than men, which may partially explain the higher incidence of stone disease in men (National Institutes of Health [NIH], 1998-2005).

Ethnic background. Stones are rare in Native Americans, Africans, American Blacks, and Israelis (Menon & Resnick, 2002).

Medical history. Past medical history may provide vital information about the underlying etiology of a stone’s formation (see Table 1). A positive medical history of skeletal fracture(s) and peptic ulcer disease suggests a diagnosis of primary hyperparathyroidism. Intestinal disease, which may include chronic diarrheal states, ileal disease, or prior intestinal resection, may be a predisposition to enteric hyperoxaluria or hypocitraturia. This may result in calcium oxalate nephrolithiasis because of dehydration and chemical imbalances (see Figure 1). Irritable bowel disease or intestinal surgery may prevent the normal absorption of fat from the intestines and alter the manner in which the intestines process calcium or oxalate. This may also lead to calculi or stone formation. Patients with gout may form either uric acid stones (see Figure 2) or calcium oxalate stones. Patients with a history of urinary tract infections (UTIs) may be prone to infection nephrolithiasis caused by urea-splitting bacteria (Munver & Preminger, 2001). Cystinuria is a homozygous recessive disease leading to stone formation. Renal tubular

<table>
<thead>
<tr>
<th>Table 1. Factors Contributing to Stone Development</th>
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<tr>
<td>• Congenital kidney defects (medullary sponge disease)</td>
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<tr>
<td>• Excess parathyroid hormone</td>
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<tr>
<td>• Medications (ephedrine, guaifenesin, indinavir, allopurinol)</td>
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<td>• Gout</td>
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<td>• Hypertension</td>
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<td>• Colitis, inflammation of colon, and chronic diarrheal states</td>
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<td>• Irritable bowel disease or intestinal surgery</td>
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<td>• Renal tubular acidosis</td>
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<td>• Crohn’s disease results in dehydration and low citrate.</td>
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<td>• Arthritis (skeletal disease)</td>
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<td>• Urinary tract infections</td>
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<td>• Past medical history of kidney stones</td>
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<td>• Obesity and high body mass index</td>
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<td>• Prolonged inactivity</td>
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<td>• Anatomic factors – Ureteropelvic junction (UPJ) obstruction</td>
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<td>• Horseshoe or ectopic kidney</td>
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<td>• Autosomal dominant polycystic kidney disease</td>
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<tr>
<td>• Vesicoureteral reflux</td>
</tr>
<tr>
<td>• Calyceal diverticula</td>
</tr>
</tbody>
</table>

Source: Tanagho & McAninch, 2004
Acidosis is a familial disorder that causes kidney stones in most patients who have this disorder.

**Dietary habits.** Fluid restriction or dehydration may cause kidney stone formation. Dietary intake that is high in sodium, oxalate, fat, protein, sugar, unrefined carbohydrates, and ascorbic acid (vitamin C) has been linked to stone formation. Low intake of citrus fruits can result in hypocitraturia, which may increase an individual’s risk for developing stones.

**Environmental factors.** Fluid intake consisting of drinking water high in minerals may contribute to kidney stone development. Another contributing factor may be related to geographical variables such as tropical climates (NIH, 1998-2005). Stone formation is greater in mountainous, high-desert areas that are found in the United States, British Isles, Scandinavia, Mediterranean, Northern India, Pakistan, Northern Australia, Central Europe, Malayan Peninsula, and China (Menon & Resnick, 2002). Affluent societies have a higher rate of small upper tract stones whereas large struvite (infection) stones occur more commonly in developing countries (see Figure 3). Bladder stones are more common in underserved countries and are likely related to dietary habits and malnutrition (Menon & Resnick, 2002).

**Medications.** Medications such as ephedrine, guaifenesin, thiazide, indinavir, and allopurinol may be contributory factors in the development of calculi (see Drug-Induced Nephrolithiasis).

**Occupations.** Occupations in which fluid intake is limited or restricted or those associated with fluid loss may be at greater risk for stone development as a result of decreased urinary volume.

**CLINICAL PRESENTATION**

Symptoms may vary and depend on the location and size of the kidney stones or calculi within the urinary collecting system. In general, symptoms may include acute renal or ureteral colic, hematuria (microscopic or gross blood in the urine), urinary tract infection, or vague abdominal or flank pain. A thorough history and physical examination, along with selected laboratory and radiologic studies, are essential to making the correct diagnosis. Small nonobstructing stones or “silent stones” located in the calyces of the kidney are sometimes found incidentally on x-rays or may be present with asymptomatic hematuria. Such stones often pass without causing pain or discomfort.

**Kidney Stone Symptoms**

Stones in the kidneys can become lodged at the junction of the kidney and ureter (ureteropelvic junction), resulting in acute ureteral obstruction with severe intermittent colicky flank pain. Pain can be localized at the costovertebral angle. Hematuria may be present intermittently or persistently and it may be microscopically or gross.

**Ureteral Stone Symptoms**

Stones that can pass into the ureter may produce ureteral colic, which is an acute, sharp, spasm-like pain located in the flank. Hematuria may be present. Stones moving down the ureter to the pelvic brim and iliac vessels will produce spasms with intermittent, sharp, colicky pain radiating to the lateral flank and around the umbilical region.

As a stone passes through the distal ureter, near the bladder, the pain remains sharp but with a waxing and waning quality. Relief is offered when the spasm subsides or the pain may intensify and radiate to the groin, testicles, or labia. Nausea, vomiting, diaphoresis, tachycardia, and tachypnea may be present and patients are typically uncomfortable.

**Bladder Stone Symptoms**

Once a stone enters the bladder, dysuria, urgency, and frequency may be the only symptoms experienced. Immediate relief of symptoms occurs once the stone passes out of the bladder.

**Kidney Stone Complications**

Occasionally, stones can injure the kidneys by causing infection, resulting in fever, chills, and loss of appetite or urinary obstruction. If a UTI accompanies the urinary obstruction, pyelonephritis or urosepsis can occur. If stones are bilateral, they can cause renal scarring and damage, resulting in acute or chronic renal failure.

**CALCIUM NEPHROLITHIASIS**

**Hypercalciuria**

Eighty to eighty-five percent of calculi or stones diagnosed in the United States are idiopathic (spontaneous and without recognizable cause) or primary. These stones are comprised of calcium and are due to excess calcium excretion in the urine, usually exceeding 200 mg/24-hour collection (see Table 2).

**Absorptive hypercalciuria.** The primary abnormality in absorptive hypercalciuria is increased absorption of calcium. Absorptive hypercalciuria Type I is more severe and characterized by a high urine calcium level,
with high or low dietary calcium intake. There is a normal serum level of calcium and phosphorus and normal or low serum level of parathyroid hormone.

Absorptive hypercalciuria Type II is a mild to moderate form of hypercalciuria and less severe than Type I. Type II hypercalciuria only occurs with high calcium intake. There is normal urinary calcium excretion while fasting or on a restricted calcium diet (Munver & Preminger, 2001).

Renal hypercalciuria. Renal hypercalciuria or “renal leak mechanism” is thought to be caused by impairment in renal tubular reabsorption of calcium (Munver & Preminger, 2001; Pak, 1979). The loss of calcium in the urine leads to stimulation of the parathyroid function, causing elevated 1,25-vitamin D and increased intestinal absorption to maintain serum levels of calcium.

Primary hyperparathyroidism. Excess parathyroid hormone (PTH) results in increased bone resorption of calcium from bone and accounts for less than 5% of stones. Parathyroid hyperplasia or adenomas secrete excess parathyroid hormone causing increased intestinal absorption of calcium, increased 1,25-vitamin D3, and increased bone demineralization and calcium release from bone. Laboratory tests reveal elevated parathyroid hormone and serum calcium levels.

Less frequent causes of hypercalciuria include chronic immobilization, metastatic cancer to bone, multiple myeloma, and vitamin D intoxication. Calcium stones have been described as appearing spiculated, dotted, mulberry, or jackstone in appearance.

Hyperoxaluria

Hyperoxaluria is defined by urinary oxalate excretion in excess of 45 mg/day (Munver & Preminger, 2001). The cause of these calcium stones can be related to primary or secondary factors.

Primary hyperoxaluria. Type I hyperoxaluria is a rare autosomal recessive disorder that begins in childhood in which a defect of the hepatic enzyme alanine-glyoxylate aminotransferase (AGT) causes increased urinary excretion of oxalic, glycolic, and glyoxylic acids (Danpure, 1994; Menon & Mahle, 1982; Munver & Preminger, 2001). This condition is characterized by nephrocalcinosis, oxalate deposition in tissues, and renal failure resulting in death before age 20, if untreated. Diagnosis is made through percutaneous liver biopsy and evaluation of the amount and distribution of AGT in liver specimens.

Type II hyperoxaluria is a very rare deficiency of the hepatocytic enzymes (D-glycerate dehydrogenase and late reductase) resulting in increased urinary oxalate and glycerate excretion. This results in the development of nephrocalcinosis, tubulointerstitial nephropathy, and chronic renal failure.

Secondary hyperoxaluria (dietary). Approximately 80% of urinary oxalate is synthesized within the liver and a small percentage (20%) from dietary intake. Overindulgence of diets rich in oxalates can contribute to hyperoxaluria through intestinal absorption of oxalate. This includes foods such as rhubarb, green leafy vegetables, spinach, cocoa, beer, coffee or tea, or excess ascorbic acid (vitamin C) intake.

Enteric hyperoxaluria. The primary site of oxalate absorption is the distal colon. Intestinal malabsorption can cause excess oxalate absorption due to various diseases such as chronic diarrhea/short bowel inflammatory disease, gastric or small bowel resection surgery. Additional secondary causes can be the result of low urinary output from intestinal fluid loss, low urinary citrate due to hypokalemia and metabolic acidosis, and low magnesium levels due to impaired intestinal magnesium absorption.

Hyperuricosuria

Hyperuricosuria (excessive urinary uric acid) accounts for 10% of calcium stones. There is a genetic predisposition, common in men, for stone development due to high uric acid levels and excess uric acid excretion, which results in hyperuricosuria. Excessive uric acid excretion can be found in primary gout, and secondary conditions of purine overproduction including myeloproliferative disorders such as acute leukemia, glycogen storage disease, and malignancy. Hyperuricosuria is often caused by excess intake of purine in meat, fish, and poultry. This purine diet causes a low uri-
nary pH. The features of hyperuricosuric calcium oxalate nephrolithiasis include elevated urinary uric acid (>600 mg/day), a normal serum calcium level, normal urinary calcium and oxalate levels, normal fasting and calcium load response, and urinary pH typically <5.5.

**Hypocitraturia**

Hypocitraturic calcium nephrolithiasis may exist as an isolated abnormality (10%) or more commonly in combination with other metabolic disorders (50%) (Menon & Mahle, 1982; Pak, 1987; Pak, 1994). Acid-base status, acidosis in particular, is the most important factor affecting the renal handling of citrate, with increased acid levels resulting in diminished endogenous citrate production. Low urinary citrate causes the urinary environment to become supersaturated with calcium salts, promoting nucleation, growth, and aggregation, resulting in stone formation.

**Distal renal tubular acidosis.** A more common cause of hypocitraturia is distal renal tubular acidosis (RTA). Acidosis impairs urinary citrate excretion by enhancing renal tubular reabsorption of citrate as well as by reducing its synthesis (Pak, 1982). Distal RTA can be complete or incomplete. In both forms, hypercalcuiuria and profound hypocitraturia may be associated. In combination with alkaline urine, the patient is at risk for developing calcium oxalate or calcium phosphate stones (Munver & Preminger, 2001; Preminger, Sakhaee, Skurla, & Pak, 1985).

**Chronic diarrheal syndrome.** Chronic diarrheal syndrome causes a loss of alkali in the form of bicarbonate through the gastrointestinal tract resulting in metabolic acidosis with subsequent impairment in citrate synthesis (Munver & Preminger, 2001; Rudman et al., 1980). The decreased citrate production causes a lower urinary concentration of citrate. Patients with chronic diarrheal syndrome may have additional risk factors for stone formation such as low urine volumes and hyperoxaluria.

**Thiazide-induced hypocitraturia.** Thiazide diuretics can produce hypokalemia (low potassium) leading to intracellular acidosis. This acidic state inhibits the synthesis of citrate, resulting in hypocitraturia. The essential mechanism is the inhibition of citrate production, which is a consequence of chronic acidosis (Nicar, Peterson, & Pak, 1984).

**Idiopathic hypocitraturia.** Mechanisms that account for hypocitraturia in this condition include a high animal protein diet (with an elevated acid-ash content), strenuous physical exercise (causing lactic acidosis), high sodium intake, and intestinal malabsorption of citrate.

**Hypomagnesuria**

Magnesium, an inhibitor of calcium nephrolithiasis, increases the solubility product of calcium oxalate and calcium phosphate. Hypomagnesuria is defined as urinary magnesium excretion <50 mg/day. Many patients with nephrolithiasis will report a limited intake of magnesium-rich foods such as nuts and chocolate, suggesting the dietary basis of this condition.

**Gouty Diathesis**

Gouty diathesis (predisposition to uric acid or calcium stones) may appear in a latent or an early phase of classic gout, or it may manifest fully with gouty arthritis and hyperuricemia. Patients develop renal stones composed purely of uric acid, uric acid in combination with calcium oxalate or calcium phosphate, or stones that reveal only calcium oxalate or calcium phosphate. Some patients may form uric acid or calcium stones (Khatchadourian, Preminger, Whitson, Adams-Huet, & Pak, 1995). The invariant feature of this condition is persistently acidic urine (pH <5.5) and no specific cause has been detected for the low urinary pH.

**NON-CALCIUM NEPHROLITHIASIS**

**Uric Acid Stones**

Uric acid stones may form in the presence of gouty diathesis or in secondary causes of purine overproduction. Secondary causes of these stones can include chronic diarrheal states such as ileostomy, ulcerative colitis, and Crohn’s disease. These chronic diarrheal states predispose uric acid precipitation, acidic urinary pH due to bicarbonate loss in stool or urinary ammonium excretion defects, and reduced urinary volume (see Table 3).

**Cystine Stones**

Cystine stones are due to a rare, congenital condition resulting in large amounts of cystine (an amino acid) in the urine. Cystinuria causes cystine stones, requiring lifelong therapy (Urology Channel, 1998). This disorder typically presents during childhood and adolescence (Bernier, 2005). The diagnosis should be suspected for patients with an early onset of nephrolithiasis, a significant family history, or recurrent stone disease. A positive sodium-nitroprusside urine test or the presence of flat, hexagonal crystals in urinary sediment provides a presumptive diagnosis of cystine stone disease.

**Struvite Stones (Infection Stones)**

These stones are caused by UTIs, which affect the chemical balance of the urine, raising the pH. Urea-splitting bacteria (for example, Proteus, Klebsiella, and Pseudomonas) release chemicals into the urinary tract, neutralizing acid in the urine, enabling the bacteria to grow quickly and
Low Urine Volume

Low urine output is defined as <1 liter/day. The typical etiologies of nephrolithiasis are low fluid intake and reduced urine volume. Other possible causes of low urine volume include chronic diarrheal syndromes that result in large fluid losses from the gastrointestinal tract and fluid loss from perspiration, or evaporation from lungs or exposed tissue. Stone formation may be initiated by a low urine output, providing a concentrated environment for substances such as calcium, oxalate, uric acid, and cystine to begin crystallization.

No Pathological Disturbance

In approximately 35% of the stone-forming population, no identifiable risk factors for stone formation can be found (Levy, Adams-Huet, & Pak, 1995). This group includes individuals with normal serum calcium and PTH, normal fasting and calcium load response, normal urine volumes, normal pH, calcium, oxalate, uric acid, citrate, and magnesium levels in the presence of calcium nephrolithiasis.

Drug-Induced Nephrolithiasis

Ephedrine calculi. Ephedrine and its metabolites (norephedrine, pseudoephedrine, and norpseudoephedrine) are sympathomimetic agents that have been used for the treatment of enuresis, myasthenia gravis, narcolepsy, and rhinorrhea (Powell, Hsu, Turk, & Hruska, 1998). In addition to numerous side effects, ephedrine and its derivatives have been associated with the production of urinary stones (Blau, 1998). The diagnosis of these calculi is similar to that of other radiolucent calculi. Twenty-four hour urine metabolic analyses can aid in identifying ephedrine or its respective metabolites.

Guaifenesin calculi. Guaifenesin sulfate (Crixivan®) is currently one of the most frequently used protease inhibitors used against human immunodeficiency virus, the virus that causes AIDS. The incidence of calculi in patients taking indinavir ranges from 3% to 20% (Schwartz, Schenkman, Armenakas, & Stoller, 1999). Indinavir calculi are radiolucent when they are pure, and are radiopaque when they contain calcium.

Xanthine calculi. These stones occur due to a rare hereditary condition with xanthine oxidase deficiency (see Figure 4). The deficiency in this enzyme results in decreased levels of serum and urinary uric acid. Acidic urine causes crystal precipitation, resulting in stone formation (Bernier, 2005). These stones are also seen in patients treated with iatrogenic inhibition of xanthine oxidase with xanthine oxidase inhibitors for hyperuricosuria such as allopurinol.

OTHER CAUSES OF NEPHROLITHIASIS

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Guaifenesin calculi. Guaifenesin is a widely used expectorant that has been recently associated with nephrolithiasis. Guaifenesin calculi are radiolucent and present in patients who ingest this medication in excess. Twenty-four hour urine metabolic analysis can aid in the identification of guaifenesin or b-2-methoxyphenoxy-lactic acid.

Indinavir calculi. Indinavir sulfate (Crixivan®) is currently one of the most frequently used protease inhibitors used against human immunodeficiency virus, the virus that causes AIDS. The incidence of calculi in patients taking indinavir ranges from 3% to 20% (Schwartz, Schenkman, Armenakas, & Stoller, 1999). Indinavir calculi are radiolucent when they are pure, and are radiopaque when they contain calcium.

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DIAGNOSIS OF KIDNEY STONES

Urolithiasis can mimic other etiologies of visceral pain. It is imperative to consider causes of surgical abdomens such as appendicitis, cholecystitis, peptic ulcer, pancreatitis, ectopic pregnancy, and dissecting aneurysm in patients who present with abdominal pain. Initial assessment includes a thorough history and physical examination, basic serum and urine chemistries, and a radiologic imaging study. First-time stone formers may benefit from a more detailed laboratory
evaluation to identify causal factors for stone formation. Multiple or recurrent stone-formers (metabolically active stone formers) require a more comprehensive laboratory evaluation (NIH, 1998-2005).

METABOLIC EVALUATION

The primary objective of a diagnostic evaluation of nephrolithiasis should be to efficiently and economically identify the particular physiological defect present in the patient to enable the selection of specific and rational therapy. The evaluation should be able to identify the metabolic disorders responsible for recurrent stone disease, including cystinuria, distal renal tubular acidosis, enteric hyperoxaluria, gouty diathesis, and primary hyperparathyroidism.

A detailed history and physical examination are imperative for both first-time stone-formers and recurrent stone-formers. Past medical history emphasis should include information about previous UTIs, diet and fluid intake, medications including vitamin intake, bowel disease, gout, renal disease, bone or parathyroid disease, and bowel surgery.

First-time stone-formers may undergo an abbreviated diagnostic evaluation such as stone analysis, urinalysis, culture and sensitivity, and a comprehensive metabolic panel, which includes serum calcium, uric acid, and phosphorus. Recurrent stone-formers and first-time stone-formers are at risk for recurrence. Both will benefit from an extensive diagnostic evaluation such as stone analysis, urinalysis, culture and sensitivity, comprehensive metabolic panel, which includes serum calcium, uric acid, and phosphorus, parathyroid hormone level, and 24-hour urine collections (random and after being on a special diet). Patients at risk include children, middle-aged white males with a family history of stones, and patients with intestinal disease (chronic diarrheal or malabsorptive states), gout, nephrocalcinosis, osteoporosis, pathologic skeletal fractures, or urinary tract infection. Stones composed of cystine, struvite, or uric acid should undergo a complete metabolic workup (Pre-minger, Peterson, Peters, & Pak, 1985).

Urine Assessment

Urinalysis with urine culture and sensitivity are mandatory tests. Reports may reveal microscopic or gross hematuria and pyuria with or without infection. Increase or decrease in urine pH and the presence of crystals may give clues to whether the stone is alkaline or acidic. A cyanide nitroprusside test will screen for suspected cystinuria.

Two 24-hour urine collections should be performed evaluating calcium, sodium, phosphorus, magnesium, oxalate, uric acid, citrate, sulfate, creatinine, pH, and total volume. The first 24-hour urine should be a random specimen. The second 24-hour urine should be obtained after the patient has been on a sodium, oxalate, and calcium-restricted diet.

Serum Assessment

Complete blood count (CBC) may reveal an elevated white blood count (WBC) suggesting urinary systemic infection, or depressed red blood cell count suggesting a chronic disease state or severe ongoing hematuria. Serum electrolytes, BUN, creatinine, calcium, uric acid, and phosphorus assess current renal function, dehydration, and the metabolic risk of future stone formation. An elevation in PTH level will confirm a diagnosis of hyperparathyroidism.

Radiologic Assessment

Intravenous pyelography (IVP). Intravenous pyelography (urography) has long been considered the primary diagnostic study of choice for identifying urinary tract calculi. The IVP provides anatomical and functional information, identifies the precise size and location of a stone, the presence and severity of the obstruction, and renal or ureteral abnormalities. For these reasons, the IVP has been among the most important diagnostic tests that may enable successful management decisions.

Computed Tomography (CT) Scan

CT scan (with and without contrast) is believed to be the best radiographic examination for acute renal colic as it creates images of the urinary tract and shows delayed penetration of intravenous contrast through the obstructed kidney. The delayed penetration of the contrast through an obstructed kidney is the hallmark of acute urinary obstruction. The CT findings indicative of acute urinary obstruction secondary to a stone would include renal enlargement, hydronephrosis, ureteral dilatation, perinephric stranding, and periureteral edema (Katz, Lane, & Sommer, 1996; Smith, Verga, Dalrymple, McCarthy, Rosenfield, 1996). Other conditions that can mimic ureteral colic can be identified as well as anatomic abnormalities and obstruction. For many reasons, the CT scan is considered superior to an IVP in detecting both renal and ureteral calculi, and is routinely performed on most patients in which a diagnosis of urolithiasis is
Radionuclide Imaging

Renal scan is considered the gold standard for assessing renal function, especially in the setting of recurrent or long-standing nephrolithiasis. It is noninvasive, does not require any special preparation or bowel preparation, exposes the patient to minimal radiation, and is nearly free of allergic complications.

Plain X-Rays

Plain abdominal X-rays entailing a flat plate radiograph of kidney, ureter, and bladder (KUB) will identify renal stones that are radiopaque (Department of the Navy Bureau of Medicine and Surgery, 2004). Abdominal X-rays are helpful in documenting the number, size, and location of stones in the urinary tract and the radiopacity may provide information on the type of stones present. Plain abdominal films can be useful in identifying nephrocalcinosis, suggestive of hyperparathyroidism, primary hyperoxaluria, renal tubular acidosis, or sarcoidosis.

Renal Ultrasound

Ultrasonography can be used as a screening tool for hydronephrosis or stones within the kidney or renal pelvis. A renal ultrasound can also determine the amount of renal parenchyma present in an obstructed kidney, in addition to the presence of stones. The ultrasound can be used in combination with plain abdominal radiograph to determine hydronephrosis or ureteral dilation (Wolf, 2004). This may be helpful in assessment during pregnancy (see Table 4).

### Table 4.
#### Diagnostic Tests for Urinary Stones

<table>
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<tr>
<th>Diagnostic Tests</th>
<th>Rationale of Testing</th>
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<tr>
<td>Urinalysis</td>
<td>Evaluate presence of blood or infection</td>
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<tr>
<td>Intravenous pyelogram (IVP)</td>
<td>Evaluate urinary tract obstruction</td>
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<tr>
<td>Chemistry</td>
<td>Evaluate for stones</td>
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<tr>
<td>Creatinine</td>
<td>Evaluate anatomy of urinary stone</td>
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<tr>
<td>BUN</td>
<td>Evaluate kidney function</td>
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<tr>
<td>Electrolytes</td>
<td>Evaluate presence of dehydration and electrolyte imbalances</td>
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<tr>
<td>Calcium, PTH</td>
<td>Diagnose hyperparathyroidism</td>
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<tr>
<td>CBC</td>
<td>Evaluate presence of infection</td>
</tr>
<tr>
<td>Renal ultrasound</td>
<td>Evaluate presence of stone</td>
</tr>
<tr>
<td>Cystoscopy</td>
<td>Evaluate urinary tract obstruction</td>
</tr>
<tr>
<td>CT scan (non-contrast)</td>
<td>Same as IVP</td>
</tr>
<tr>
<td>Abdominal X-rays</td>
<td>Evaluate size, shape, and stone location</td>
</tr>
</tbody>
</table>

### Table 5.
#### Stone Management Guidelines

- Depends on size and location of the stone
- Presence or absence of associated infection
- Presence of one or two kidneys
- Degree of symptoms
- Majority will pass spontaneously with no residual damage
- Stones <4 mm pass spontaneously in approximately 90% of patients
- Stones 4-6 mm pass in approximately 50% of patients
- Stones >8 mm pass without intervention in 20% of patients
- Stones >8 mm will pass in only 20% of patients and usually require surgical intervention
- If obstruction and infection present, emergent decompression of upper urinary collecting system (kidneys and upper ureter) required

### MEDICAL MANAGEMENT

Effective kidney stone prevention is dependent on the stone type and identification of risk factors for stone formation (see Tables 5 & 6). An individualized treatment plan incorporating dietary changes, supplements, and medications can be developed to help prevent the formation of new stones. Certain con-
Conservative recommendations should be made for all patients regardless of the underlying etiology of their stone disease. Patients should be instructed to increase their fluid intake in order to maintain a urine output of at least 2,000 ml/day. Patients should also limit their dietary oxalate and sodium intake, thereby decreasing the urinary excretion of oxalate and calcium. A restriction of animal proteins is encouraged for patients with “purine gluttony” and hyperuricosuria.

Hypercalciuria (General) Besides treating underlying disease, management of hypercalciuria includes:

- Low calcium diet (about 400 mg calcium).
- Distilled water, if high calcium content in water supply.
- Limit vitamin C (<0.5g/day).
- High sodium intake.
- Thiazide diuretics.
- Cellulose phosphate.
- Orthophosphate.

### Table 6. Selected Medical Management of Urinary Stones

<table>
<thead>
<tr>
<th></th>
<th>Drug</th>
<th>Dosage</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Calcium Oxalate</strong></td>
<td>Thiazides</td>
<td>25 mg qd</td>
<td>Hypokalemia, frequent urination, sexual dysfunction</td>
</tr>
<tr>
<td></td>
<td>Potassium citrate</td>
<td>20-60 mEq in 3-4 divided doses daily</td>
<td>Abdominal discomfort, N/V/D</td>
</tr>
<tr>
<td></td>
<td>Sodium citrate</td>
<td>2-3 g after meals and HS</td>
<td>GI upset and hyperkalemia</td>
</tr>
<tr>
<td></td>
<td>Orthophosphate</td>
<td>1-2.5 gm/day in divided doses</td>
<td>Gas, diarrhea</td>
</tr>
<tr>
<td></td>
<td>Cellulose sodium phosphate</td>
<td>5 gms TID</td>
<td>Dyspepsia, loose bowel movements</td>
</tr>
<tr>
<td></td>
<td>Calcium carbonate/citrate</td>
<td>1 gm 4-5x/day</td>
<td>Constipation, gas, increase calcium leak, nausea</td>
</tr>
<tr>
<td></td>
<td>Cholestyramine</td>
<td>2-4 g/packet/day</td>
<td>Constipation, abdominal pain, gas, Gl upset</td>
</tr>
<tr>
<td><strong>Calcium Phosphate</strong></td>
<td>Potassium citrate</td>
<td>20-60 mEq in 3-4 divided doses daily</td>
<td>Abdominal discomfort, N/V/D</td>
</tr>
<tr>
<td></td>
<td>Sodium citrate</td>
<td>2-3 m unit doses</td>
<td>GI upset and hyperkalemia</td>
</tr>
<tr>
<td></td>
<td>Citric acid</td>
<td>After each meal and HS</td>
<td></td>
</tr>
<tr>
<td><strong>Uric Acid Stones</strong></td>
<td>Potassium citrate</td>
<td>60 mEq in 3-4 divided doses daily</td>
<td>Abdominal discomfort, N/V/D</td>
</tr>
<tr>
<td></td>
<td>Sodium bicarbonate</td>
<td>325 mg-2 gm qid or 48 mEq</td>
<td>CHF, cirrhosis, possible increase calcium-type stones</td>
</tr>
<tr>
<td></td>
<td>Allopurinol</td>
<td>300 mg/day</td>
<td>Rash, diarrhea, increase liver enzymes, nausea</td>
</tr>
<tr>
<td><strong>Cystine Stones</strong></td>
<td>Penicillamine</td>
<td>300 mg TID</td>
<td>Rash, kidney damage, N/V/D, tinnitus, loss of taste</td>
</tr>
<tr>
<td></td>
<td>Captopril</td>
<td>25 mg qd</td>
<td>Proteinuria, rash, hypotension, cough dizziness</td>
</tr>
<tr>
<td></td>
<td>Tiopronin</td>
<td>800-1,000 mg/day in divided doses</td>
<td>Jaundice, kidney damage, decreased RBCs in bone marrow</td>
</tr>
<tr>
<td></td>
<td>Sodium bicarbonate</td>
<td>325 mg-2 gm qid or 48 mEq</td>
<td>CHF, cirrhosis, possibly increase in calcium-type stones</td>
</tr>
<tr>
<td><strong>Struvite Stones</strong></td>
<td>Acetohydroxamic acid</td>
<td>250 mg 3-4x/day</td>
<td>Headache, depression, N/V/D, DVT, hemolytic anemia, sweating</td>
</tr>
</tbody>
</table>

N/V/D = nausea, vomiting, diarrhea; DVT = deep vein thrombosis
Absorptive Hypercalciuria – Type I

Thiazides are commonly used for the management of absorptive hypercalciuria Type I as these medications stimulate calcium reabsorption in the distal nephron, preventing formation of kidney stones by reducing the amount of calcium in the urine. Thiazides force a mandatory increase in urinary volume but can cause electrolyte disorders. Side effects include decreased level of potassium, frequent urination, sexual dysfunction, and increased triglycerides.

Less-common medications used for treatment include orthophosphate, sodium cellulose phosphate, and urease inhibitors. Orthophosphate and sodium cellulose phosphate reduce the absorption of calcium from the intestines thereby reducing calcium in the urine. The urease inhibitors dissolve crystals and struvite kidney stones and prevent formation of new crystals. Side effects can include a bad taste in the mouth, diarrhea, and dyspepsia.

Neither sodium cellulose phosphate nor thiazide corrects the basic, underlying physiologic defect in absorptive hypercalciuria. Sodium cellulose phosphate should be used in patients with severe absorptive hypercalciuria Type I (urinary calcium >350 mg/day) or in those resistant to or intolerant of thiazide therapy. In patients with absorptive hypercalciuria Type I, who presently have bone loss, thiazide may be the medication of first choice. Sodium cellulose phosphate may be substituted for short-term therapy when thiazide action is decreased.

Potassium supplementation (Urocit-K®, Polycitra-K® crystallor syrup) should be added when using thiazide therapy to prevent hypokalemia and decrease urinary citrate excretion. A typical treatment program might include chlorthalidone 25 mg/day. Potassium citrate 15 to 20 mEq twice/day should be provided with both of these diuretics. Side effects include abdominal discomfort, nausea, and vomiting.

Absorptive Hypercalciuria – Type II

In absorptive hypercalciuria Type II, specific drug therapy may not be necessary since the physiologic defect is not as severe as in absorptive hypercalciuria Type I. Many patients show disdain for drinking fluids and excreting concentrated urine. A low intake of calcium (400-600 mg/day) and a high intake of fluids (sufficient to achieve a minimum urine output of >2 liters/day) would be acceptable treatment. Normal urine calcium excretion would be restored by dietary calcium restriction alone, and the increase in urine volume would help reduce urinary saturation of calcium oxalate.

Renal Hypercalciuria

Thiazides are indicated for the treatment of renal hypercalciuria. This diuretic can correct the renal leak of calcium by augmenting calcium reabsorption in the distal tubule and by causing extracellular volume depletion and stimulating proximal tubular reabsorption of calcium.

Hyperoxaluria

Oral administration of large amounts of calcium (0.25 g to 1.0 g four times/day) or magnesium has been recommended for controlling enteric hyperoxaluria. A high fluid intake is recommended to assure adequate urine volume in patients with enteric hyperoxaluria. Calcium citrate may theoretically have a role in the management of enteric hyperoxaluria. This treatment may lower urinary oxalate by binding oxalate in the intestinal tract. Calcium citrate may also raise the urinary citrate level and pH. Side effects are constipation, gas, and increased calcium leak. Cholestyramine is also another method used to treat calcium oxalate stones. Cholestyramine binds to bile in the intestines which limits the amount of oxalate absorbed from the intestines, therefore less oxalate is excreted in the urine. Side effects include constipation, abdominal pain, gas, and heartburn.

Hyperuricosuria

Allopurinol (300 mg/day) is the drug of choice in patients with hyperuricosuric calcium oxalate nephrolithiasis (with or without hyperuricemia) because of its ability to reduce uric acid synthesis and lower urinary uric acid by inhibition of the enzyme xanthine oxidase. The usual dose is 300 mg/day; however, the dosage should be reduced in patients with renal insufficiency. Side effects are rash, diarrhea, and increased liver enzymes.

Potassium citrate represents an alternative to allopurinol in the treatment of this condition. Use of potassium citrate in hyperuricosuric calcium oxalate nephrolithiasis is warranted since citrate has an inhibitory activity with respect to calcium oxalate (and calcium phosphate) crystallization, aggregation, and agglomeration. Potassium citrate (30 to 60 mEq/day in divided doses) may reduce the urinary saturation of calcium oxalate.

Hypocitraturia

For patients with hypocitraturic calcium oxalate nephrolithiasis, treatment with potassium citrate can restore normal urinary citrate, thus lowering urinary saturation of calcium and inhibiting crystallization of calcium salts.

Distal Renal Tubular Acidosis

Potassium citrate therapy is able to correct metabolic acidosis and hypokalemia found in patients with distal RTA. It will
also restore normal urinary citrate levels, although large doses (up to 120 mEq/day) may be required for severe acidosis. Since urinary pH is generally elevated in patients with RTA, the overall rise in urinary pH is small. Citrate is a significant urinary calcium stone inhibitor that retards crystallization of calcium oxalate and calcium phosphate. Potassium citrate binds to calcium in the urine, preventing formation of crystals and raising the urinary citrate level and pH. It will effectively alkalinize the urine, which makes it useful in the treatment, dissolution, and prevention of uric acid stones. Urinary pH should be monitored periodically during citrate therapy because of excessive alkalinization. Side effects are mucous loose stools and minor GI complaints. Sodium citrate and citric acids are other alkalinizing agents used to prevent kidney stones by inhibiting stone formation through alkalinization.

**Cystinuria**

The objective for treatment of cystinuria is to reduce the urinary concentration of cystine to a level below its solubility limit (200-250 mg/liter). The initial treatment program includes a high fluid intake and oral administration of soluble alkali (potassium citrate) at a dose sufficient to maintain the urinary pH at 6.5 to 7.0. When this conservative program is ineffective, d-penicillamine or alpha-mercaptopropionylglycine (1,000 to 2,000 mg/day in divided doses) has been used. Potassium citrate is absorbed to prevent uric acid stones as it binds to calcium in urine, preventing formation of crystals. Sodium bicarbonate makes the urine less acidic, which makes uric acid or cystine kidney stone formation less likely. Possible side effects include increased formation of calcium-type stones, fluid retention, and sodium in blood. Urinary pH should be monitored periodically during citrate therapy because excessive alkalinization may occur, which can increase the risk of calcium phosphate precipitation and stones. Side effects are mucous loose stools and minor GI complaints. Sodium citrate and citric acid are other alkalinizing agents used to prevent kidney stones by inhibiting stone formation through alkalinization.

**Gouty Diathesis**

The major objective in the management of gouty diathesis is to increase the urinary pH above 5.5, preferably to a level between pH 6.0 and 6.5. Potassium citrate is the drug of choice in managing patients with gouty diathesis. Potassium citrate is an adequate alkalinizing agent, capable of maintaining urinary pH at approximately 6.5 at a dose of 30 to 60 mEq per day in two divided doses.

**Drug-Induced Nephrolithiasis**

*Ephedrine calculi.* There are no limited studies that address the management of these calculi. As with other calculi, a urine output of at least two liters/day is recommended.

*Guaifenesin calculi.* As with ephedrine calculi, there are no limited studies regarding pharmacologic management of these calculi.

*Indinavir calculi.* Initial measures in the management of these calculi should focus on hydration and analgesia as well as drug discontinuation and substitution with another protease inhibitor.

*Xanthine calculi.* The medical management of xanthine calculi is limited because the solubility of these calculi is essentially invariable within physiologic pH ranges. Currently the recommendation includes a fluid intake of at least three liters/day. If significant quantities of other purines are present in the urine, then urinary alkalinization with potassium citrate in the range of 6.0 to 6.5 is indicated to prevent hypoxanthine or uric acid calcu.

**NURSING MANAGEMENT**

The nurse conducts a comprehensive nursing assessment to include all contributing factors such as dietary history and fluid intake, family history, environmental factors, medical history (diabetes, hypertension, hyperparathyroidism, inflammatory bowel disease, bowel resection, Crohn’s disease, UTIs), social history, review of systems, and surgical history. Next, the nurse counsels the patient on pertinent findings elicited during the comprehensive nursing assessment and provides followup counseling to support dietary and lifestyle changes and monitor outcomes and compliance. In our institution, a *Kidney Stone Disease in Adults* teaching pamphlet is given to patients at the
Nursing Interventions
The nurse should:

- Perform pain assessments to include Visual Analog, numerical, or Wong-Baker scales as appropriate for patient population to assess level of pain and effectiveness of outcome with pain interventions.
- Provide pharmacological education. Narcotics are usually used liberally, such as parenteral (IM/IV) narcotics (ketorolac, [Toradol®], mepereidine [Demerol®], morphine, and oral narcotics/analgiesic combinations (Department of the Navy Bureau of Medicine and Surgery, 2004). Use of narcotic medication needs to be explained as well as side effects, such as nausea, vomiting, constipation, and caution with driving or operating machinery.
- Review bowel patterns and suggest interventions to prevent constipation due to pain medication.
- Assess contributing factors of dehydration such as nausea, vomiting, and diarrhea and administer antiemetics, such as metoclopramide (Reglan®), prochlorperazine (Compazine®), granisetron (Kytril®), or ondansetron (Zofran®). Administer antidiarrheal agents such as loperamide (Imodium®), diphenoxylate, atropine (Lomotil®), or paregoric and assess effectiveness of outcomes. If severe nausea and vomiting occur, patients must be aware that prevention of dehydration and electrolyte imbalance, may require IV hydration, prescription of anti-emetics, and solutions such as such as Gatorade® or Pedialyte® to replace electrolytes lost via the GI tract.
- Assess for vital signs checking for orthostatic hypotension (lowering of blood pressure and increase in pulse with positional changes) and monitoring patient weights.
- Encourage increases in daily fluid intake, especially water, and monitor outcomes of interventions through patient voiding history and 24-hour urine reports. The most important lifestyle change to prevent stones is drinking more fluids, especially water up to 2 quarts/day.
- Educate the patient on completing a voiding diary to track daily urine output.
- Educate the patient on the importance of completing laboratory tests ordered, especially 24-hour urines. This can become an imposition on the patient’s quality of life, especially if he is active and working.
- Educate the patient on collecting urine specimens and straining urine.
- Educate the patient on diagnostic testing, including required dietary or bowel preparation to reduce anxiety.
- Educate the patient on the importance of weight loss, maintaining weight loss, and daily exercise.
- Provide counseling on health promotion and maintenance, stressing the importance of followup care to evaluate causes of stone formation in an effort to prevent future recurrences.

Preventative Health Maintenance/Lifestyle Changes
Effective kidney stone prevention depends upon the stone type and identifying risk factors for stone formation. An individualized treatment plan incorporating dietary changes, supplements, and medications can be developed to help prevent the formation of new stones. If kidney stones develop despite increasing fluid intake and making changes to diet, medications can be prescribed to help dissolve the stones or to prevent formation of new stones.

As a health care provider, it is imperative that causes of stone formation be investigated to prevent future occurrences that may lead to permanent kidney damage. Patient education and counseling are vital to effective care, and can be provided by the urologic nurse to promote lifestyle changes in this patient population. Weight management is a critical factor in managing stone formation.

Table 7.
Recommended Dietary Lifestyle Changes

| 1. Increase water intake to ensure urine output of 2,000 ml. |
| 2. Maintain adequate (1,000 mg) calcium intake from dietary sources. |
| 3. Avoid calcium in pill form. |
| 4. Avoid foods with added vitamin D (and vitamin C if recommended). |
| 5. Avoid certain antacids with a calcium base. |
| 6. Limit coffee, tea, and cola to 1-2 cups/day. |
| 7. Eat less meat, fish, and poultry (decreased protein). |
| 8. Limit dietary sodium and oxalate intake. |

Source: NIH, 2003
Kidney Stone Disease in Adults

Overview

Kidney stones are one of the most painful disorders to afflict humans. This ancient health problem has tormented people throughout history. Scientists have even found evidence of kidney stones in an Egyptian mummy estimated to be more than 7,000 years old.

Kidney stones are also one of the most common disorders of the urinary tract. It is estimated that 10 percent of all people in the United States will have a kidney stone at some point in time. Men tend to be affected more frequently than women.

Most kidney stones pass out of the body without any treatment by a doctor. Cases that cause lasting symptoms or other complications may be treated by various techniques, most of which do not involve major surgery. Research advances also have led to a better understanding of the many factors that cause stones to form.

Introduction to the Urinary Tract

The urinary tract, or system, consists of the kidneys, ureters, bladder, and urethra. Among other important functions, the kidneys remove extra water and waste from the blood, converting it to urine.

Narrow tubes called ureters carry urine from the kidneys to the bladder, a triangle-shaped chamber in the lower abdomen. Like a balloon, the bladder's elastic walls stretch and expand to store urine. They flatten together when urine is emptied through the urethra to outside the body.

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Kidney Stone Development

A kidney stone develops from crystals that separate from urine and build up on the inner surfaces of the kidney. Normally, urine contains chemicals that keep the crystals from forming. These chemicals do not work for everyone, however, and some people form stones. If the crystals remain tiny, they will travel through the urinary tract and pass out of the body in the urine without even being noticed.

Kidney stones may contain various combinations of chemicals. The most common type of stone contains calcium in combination with either oxalate or phosphate. These chemicals are part of a person’s normal diet and make up important parts of the body, such as bones and muscles.

A less common type of stone is caused by infection in the urinary tract. This type of stone is called a struvite, or infection stone. Much less common are the uric acid stone and the rare cystine stone.

Urolithiasis is the medical term used to describe stones occurring in the urinary tract. Other frequently used terms are urinary tract stone disease and nephrolithiasis. Doctors also use terms that describe the location of the stone in the urinary tract. For example, ureterolithiasis is a stone found in the ureter. To keep things simple, the term "kidney stones" is used throughout this entire document.

Gallstones and kidney stones are not related. They form in different areas of the body. If a person has a gallstone, he or she is not necessarily more likely to develop kidney stones.

Cause

Doctors do not always know what causes a stone to form. While certain foods may promote stone formation in people who are more prone to them, scientists do not believe that eating any specific food causes stones to form in people who are not susceptible.

A person with a family history of kidney stones may be more likely to develop stones. Urinary tract infections, kidney disorders such as cystic kidney diseases, and metabolic disorders, such as hyperparathyroidism, also are linked to stone formation.
In addition, more than 70 percent of patients with a hereditary disease called renal tubular acidosis develop kidney stones.

Cystinuria and hyperoxaluria are two other rare, inherited metabolic disorders that often cause kidney stones. In cystinuria, the kidneys produce too much of the amino acid cystine. Cystine does not dissolve in urine and can build up to form stones. With hyperoxaluria, the body produces too much of the salt oxalate. When there is more oxalate than can be dissolved in the urine, the crystals settle out and form stones.

Absorptive hypercalciuria occurs when the body absorbs too much calcium from food and empties the extra calcium into the urine. This high level of calcium in the urine causes crystals of calcium oxalate or calcium phosphate to form in the urinary tract.

Other causes of kidney stones are hyperuricosuria, a disorder of uric acid metabolism, gout, excess intake of vitamin D, and blockage of the urinary tract. Certain diuretics, or water pills, or calcium-based antacids may increase the risk of forming kidney stones by increasing the amount of calcium in the urine.

Calcium oxalate stones may also form in people who have a chronic inflammation of the bowel or who have had ostomy, or intestinal bypass, surgery. As mentioned above, struvite stones can form in people who have had a urinary tract infection.

At-Risk Groups

For some unknown reason, the number of people in the United States with kidney stones has been increasing over the past 20 years. White people are more prone to kidney stones than are African Americans. Although stones occur more frequently in men, the number of women who get kidney stones has been increasing over the past 10 years, causing the ratio to change. Kidney stones strike most people between the ages of 20 and 40. Once a person gets more than one stone, he or she is more likely to develop others.

Symptoms

Usually, the first symptom of a kidney stone is extreme pain. The pain begins suddenly when a stone moves in the urinary tract, causing irritation or blockage. Typically, a person feels a sharp, cramping pain in the back and side in the area of the kidney, in the lower abdomen, or groin. Additional symptoms of kidney stones include:

- Nausea and vomiting.
- Blood in the urine.
- Frequent need to urinate.
- Burning during urination.

If fever and chills accompany any of these symptoms, an infection may be present and your doctor should be contacted immediately.
Diagnosis

Sometimes "silent" stones -- those that do not cause symptoms -- are found on x-rays taken during a general health exam. These stones would likely pass unnoticed.

More often, kidney stones are found on an X-ray, CT scan, or sonogram taken on someone who complains of blood in the urine or sudden pain. These diagnostic images give the doctor valuable information about the stone's size and location. Blood and urine tests help detect any abnormal substance that might make stones form.

The doctor may decide to scan the urinary system using a special x-ray test called an intravenous pyelogram, or IVP, or CT scan.

Together, the results from these tests help determine the proper treatment. IVP x-rays will miss some stones. CT scan will often call things stones that are not.

Occasionally, a patient will need both an IVP and CT scan or a repeat of the first test to confirm the presence of stones.

Treatment

Fortunately, most stones can pass through the urinary system with plenty of water - two to three quarts a day - to help move them along. In most cases, a person can stay home during this process, taking pain medicine as needed. The doctor usually asks the patient to save the passed stone or stones for testing.

Some type of surgery may be needed to remove a kidney stone if the stone does not pass after a reasonable period of time and causes constant pain, is too large to pass on its own, blocks the urine flow, or causes ongoing urinary tract infection.

Surgery may also be required if damage is done to the kidney tissue, or if the stone causes constant bleeding or has grown larger (as shown on follow up X-ray studies).

Until recently, surgery to remove a stone was very painful and required a recovery time of four to six weeks. Today, treatment for these stones is greatly improved. Many options exist that do not require major surgery.
Extracorporeal shockwave lithotripsy (ESWL) is the most frequently used surgical procedure for the treatment of kidney stones. ESWL uses shock waves that are created outside of the body to travel through the skin and body tissues until the waves hit the dense stones. The stones become sand-like and are easily passed through the urinary tract in the urine.

There are several types of ESWL devices. One device positions the patient in the water bath while the shock waves are transmitted. Other devices have a soft cushion or membrane on which the patient lies. Most devices use either x-rays or ultrasound to help the surgeon pinpoint the stone during treatment. For most types of ESWL procedures, some type of anesthesia is needed.

In some cases, ESWL may be done on an outpatient basis. Recovery time is short, and most people can resume normal activities in a few days.

Complications may occur with ESWL. Most patients have blood in the urine for a few days after treatment. Bruising and minor discomfort on the back or abdomen due to the shock waves are also common. To reduce the chances of complications, doctors usually tell patients to avoid taking aspirin and other drugs that affect blood clotting for several weeks before treatment.

In addition, the shattered stone fragments may cause discomfort as they pass through the urinary tract in the urine. In some cases, the doctor will insert a small tube called a stent through the bladder into the ureter to help the fragments pass. Sometimes the stone is not completely shattered with one treatment and additional treatments may be required.

Sometimes a procedure called percutaneous nephrolithotomy is recommended to remove a stone. This treatment is often used when the stone is quite large or in a location that does not allow effective use of EWSL.

In this procedure, the surgeon makes a tiny incision, or opening, in the back and creates a tunnel directly into the kidney. Using an instrument called a nephroscope, the stone is located and removed. For large stones, some type of energy probe may be needed to break the stone into small pieces. Generally, patients stay in the hospital for several days and may have a small tube called a nephrostomy tube left in the kidney during the healing process.

One advantage of percutaneous nephrolithotomy over ESWL is that the surgeon removes the stone fragments instead of relying on their natural passage from the kidney.
Although some ureteral stones can be treated with ESWL, **ureteroscopy** may be needed for middle and lower ureter stones. No incision is made in this procedure. Instead, the surgeon passes a small instrument called a ureteroscope through the urethra and bladder into the ureter. The surgeon then locates the stone and either removes it with a cage-like device or shatters it with a special instrument that produces a form of shock wave (EHL) or with a laser device. A small stent may be left in the ureter for a few days after treatment to help the lining of the ureter heal.

### Prevention

People who have had more than one kidney stone are likely to form another. Therefore, prevention is very important. To prevent stones from forming, their cause must be determined. The urologist will order laboratory tests, including urine and blood tests. A complete medical history will be taken, along with information about the patient's work and eating habits. If a stone has been removed, or if the patient has passed a stone and saved it, the lab can analyze the stone to find out its chemical make-up.

A patient may be asked to collect urine for 24 hours after a stone has passed or been removed. The sample is used to measure urine volume and levels of acidity, calcium, sodium, uric acid, oxalate, citrate, and creatinine, a byproduct of how the body uses protein. The doctor will use this information to determine the cause of the stone. A second 24-hour urine collection may be needed to see if the prescribed treatment is working.

### Lifestyle Changes

The most important lifestyle change to prevent stones is to drink more liquids -- water is best. Someone who has had stones before should try to drink enough liquids throughout the day to produce at least two quarts of urine in every 24-hour period.

Patients with too much calcium or oxalate in the urine may need to eat fewer foods containing calcium and oxalate. Not everyone will benefit from a low-calcium diet, however. Some patients who have high levels of oxalate in their urine may benefit from extra calcium in their diet. Patients may be told to avoid food with added vitamin D and certain types of antacids that have a calcium base.

Patients who have a very high level of acid in their urine may need to eat less meat, fish, and poultry. These foods increase the amount of acid in the urine.

To prevent cystine stones, patients should drink enough water each day to reduce the amount of cystine that escapes into the urine. This is difficult because more than a gallon of water may be needed every 24 hours, a third of which must be drunk during the night.

### Medication

The doctor may prescribe certain medications to prevent calcium and uric acid stones. These drugs control the amount of acid or alkali in the urine, key factors in crystal formation. The drug Allopurinol may also be useful in some cases of hypercalciuria and hyperuricosuria.
Another way a doctor may try to control hypercalciuria, and thus prevent calcium stones, is by prescribing certain diuretics, such as Hydrochlorothiazide. These drugs decrease the amount of calcium released by the kidneys into the urine.

Some patients with absorptive hypercalciuria may be given the drug Sodium cellulose phosphate. This drug keeps calcium from leaking into the urine.

If cystine stones cannot be controlled by drinking more fluids, the doctor may prescribe the drug Thiola. This medication helps reduce the amount of cystine in the urine.

For **struvite stones** that have been totally removed, the first line of prevention is to keep the urine free of bacteria that can cause infection. The patient's urine will be tested on a regular basis to be sure that bacteria are not present.

If struvite stones cannot be removed the doctor may prescribe a new drug called Aetohydroxamic acid (AHA). AHA is used along with long-term antibiotic drugs to prevent the infection that leads to stone growth.

**Surgery**

To prevent calcium stones that form in hyperparathyroid patients, a surgeon may remove all of the parathyroid glands (located in the neck). This is usually the treatment for hyperparathyroidism as well. In most cases, only one of the glands is enlarged. Removing the gland ends the patient's problem with kidney stones.

**Research on Kidney Stones**

The Division of Kidney, Urologic, and Hematologic Diseases of the National Institutes of Diabetes and Digestive and Kidney Diseases (NIDDK) funds research on the causes, treatments, and prevention of kidney stones. The NIDDK is part of the federal government's National Institutes of Health.

New drugs and the growing field of lithotripsy have greatly improved the treatment of kidney stones. Still, NIDDK researchers and grantees seek to answer questions such as:

- Why do some people continue to have painful stones?
- How can doctors predict who is at risk for getting stones?
- What are the long-term effects of stones?
formation and prevention of future occurrences as evidenced by a study at Brigham and Women’s Hospital, Boston (Guttman, 2005). Researchers evaluated the correlation of obesity and weight gain and the risk of developing kidney stones. The findings indicated obesity was a contributing factor in stone development since, as we age, the majority of weight gain is from fat tissue not bone or muscle. The risk of developing stones increased by 71% to 109% among younger and older women in the highest weight, BMI, and waist circumference and 33% to 48% in men. These findings support the need for health care providers to emphasize the importance of exercise and weight management in a prevention program. Dietary recommendations for stone formers are discussed in detail by Krieg (2005).

Conclusion

With appropriate diagnosis and treatment of specific disorders resulting in nephrolithiasis, a remission rate greater than 80% can be obtained (see Table 8). In patients with mild to moderate severity of stone disease, virtually total control of stone disease can be achieved with a remission rate greater than 95% (Preminger, Harvey, & Pak, 1985). The need for surgical stone removal may be reduced dramatically or eliminated with an effective prophylactic program. Selective pharmacologic therapy also has the advantage of overcoming nonrenal complications and averting certain side effects that may occur with nonselective medical therapy. It is clear that selective medical therapy alone cannot provide total control of stone disease. A satisfactory response requires continued dedicated compliance by patients to the recommended program and a commitment of the physician to provide long-term followup and care with the intention of improving quality of life by eliminating the symptoms caused by urinary tract stones.

References


### Table 8. Quick Reference

<table>
<thead>
<tr>
<th>Type of Stone</th>
<th>Cause</th>
<th>Clinical Findings</th>
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<tr>
<td><strong>Absorptive Hypercalciuria</strong> Type I</td>
<td>Increased absorption of calcium, unknown (high or low dietary calcium intake)</td>
<td>High urine calcium level Normal serum calcium Normal serum phosphorus Normal of low serum parathyroid hormone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal urine calcium</td>
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<td><strong>Absorptive Hypercalciuria</strong> Type II</td>
<td>Excess calcium intake Excess vitamin D intake</td>
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<td><strong>Renal Hypercalcuria</strong> Decreased renal absorption</td>
<td>Impaired renal tubular re-absorption of calcium → decreased urine calcium → increased intestinal calcium absorption → PTH stimulation</td>
<td>Elevated 1, 25-vitamin D3</td>
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<td></td>
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<td>Increased 1, 25-vitamin D3 Increased bone demineralization Increased parathyroid hormone and ↑ serum calcium</td>
</tr>
<tr>
<td><strong>Renal Hypercalcuria</strong> Increased bone absorption</td>
<td>Excess parathyroid hormone, increased ↓ bone resorption of calcium</td>
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<tr>
<td><strong>Hyperoxaluria</strong> Primary Type I</td>
<td>Autosomal recessive disorder Defect in hepatic enzyme alanine-glyoxylate Aminotransferase (AGT)</td>
<td>Increased urine levels of oxalic, glycolic, and glyoxylic acid Oxalate tissue deposits Renal failure</td>
</tr>
<tr>
<td><strong>Hyperoxaluria</strong> Type II</td>
<td>Rare Deficiency of hepatic enzymes D-glycerate dehydrogenase and glyoxylate reductase</td>
<td>Increased urine level of oxalate and glycerate excretion Nephrocalcinosis Tubular interstitial neuropathy Chronic renal failure</td>
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<tr>
<td><strong>Secondary Dietary Hyperoxaluria</strong></td>
<td>Excess dietary intake oxalate foods Excess intake ascorbic acid (vitamin C)</td>
<td>Increased urine oxalate</td>
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<tr>
<td><strong>Enteric Hyperoxaluria</strong></td>
<td>Intestinal malabsorption (distal colon) Excess oxalate absorption Chronic diarrheal states Bowel surgery</td>
<td>Decreased urine citrate Hypokalemia Metabolic acidosis Decreased urine volume Decreased potassium Decreased magnesium</td>
</tr>
<tr>
<td><strong>Hyperuricosuria</strong></td>
<td>Increased uric acid levels Excess uric acid excretion Excess purine diet Genetic predisposition Purine overproduction Acute leukemia Malignancy Glycogen storage disease</td>
<td>Normal serum calcium Normal urinary calcium Normal urinary oxalate Normal fasting and calcium load response Acidic urine Increase urine uric acid</td>
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<tr>
<td><strong>Hypocitraturia</strong></td>
<td>Disruption of acid base balance Increased acid levels Decreased endogenous citrate production</td>
<td>Decreased urine citrate</td>
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Table 8. (continued)
Quick Reference

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<thead>
<tr>
<th>Type of Stone</th>
<th>Cause</th>
<th>Clinical Findings</th>
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<tbody>
<tr>
<td>Hypomagnesuria</td>
<td>Low magnesium prevents inhibition of solubility product of calcium oxalate and calcium phosphate</td>
<td>Low urine magnesium excretion &lt;50 mg/day</td>
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<tr>
<td>Uric Acid</td>
<td>Purine overproduction</td>
<td>Acidic urine</td>
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<td>Uric acid precipitation</td>
<td>Low urine volume</td>
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<tr>
<td>Cystine</td>
<td>Rare</td>
<td>Positive sodium-nitroprusside</td>
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<td></td>
<td>Congenital condition</td>
<td>Flat, hexagonal crystals in urine sediment</td>
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<tr>
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<td>Large amounts of cystine in urine</td>
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<tr>
<td>Struvite</td>
<td>UTIs-urea-splitting bacteria (Proteus, Klebsiella, Pseudomonas)</td>
<td>Bacteria in urine and culture and sensitivity</td>
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<td></td>
<td>Neurogenic bladder</td>
<td>Increased WBCs in urinalysis</td>
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<td>Triamterene ephedrine</td>
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<tr>
<td>Xanthine</td>
<td>Rare</td>
<td>Decreased level serum uric acid</td>
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<td>Hereditary deficiency in xanthine oxidase</td>
<td>Decreased level urine uric acid</td>
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<td>Acidic urine</td>
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Mineral & Electrolyte Metabolism, 13, 257-266.


Urolithiasis/Nephrolithiasis
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Additional Readings

