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Renal Calculi, Nephrolithiasis

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Continuing Education Activity

Renal calculi are a common cause of blood in the urine (hematuria) and pain in the abdomen, flank, or groin. They occur in 1 in 11 people at some time in their lifetimes, with men affected twice as much as women. Development of the stones is related to decreased urine volume or increased excretion of stone-forming components such as calcium, oxalate, uric acid, cystine, xanthine, and phosphate. Calculi may also be caused by low urinary citrate levels or excessive urinary acidity. Dietary and medications can modify many of the risk factors leading to nephrolithiasis. However, depending on the size and location of the stone, lithotripsy or percutaneous intervention may be required, especially if pyonephrosis develops.

This activity explores the cause, pathophysiology, presentation, and treatment of renal calculi, providing learners with a comprehensive understanding of this condition. The various risk factors contributing to nephrolithiasis and the dietary and medical interventions aimed at prevention and management are explored. Moreover, the course emphasizes the importance of an interprofessional team approach in caring for patients with renal calculi, highlighting effective communication and collaboration among urologists, nephrologists, radiologists, nurses, and dietitians to optimize patient outcomes and provide holistic care.

Objectives:

- Compare the different types of renal calculi and frequency in different patient populations.
- Evaluate the risk factors for renal calculi based on diagnostic testing and detailed patient history.
- Develop a comprehensive treatment plan for patients depending on risk factors, metabolic abnormalities, and results of diagnostic testing.
- Strategize care coordination among interprofessional team members to provide education and improve outcomes for patients affected by renal calculi.

Access free multiple choice questions on this topic.

Introduction

Renal calculi are a common cause of blood in the urine (hematuria) and pain in the abdomen, flank, or groin. They occur in 1 of every 11 people in the United States at some time in their lifetimes, with men affected 2 to 1 over women.[1] Development of the stones is related to decreased urine volume or increased excretion of stone-forming components such as calcium, oxalate, uric acid, cystine, xanthine, and phosphate.

Calculi may also be caused by low urinary citrate levels (an inhibitor of stone formation) or excessive urinary acidity.[2][3][4] Renal calculi may present with excruciating pain, and most patients present to the emergency department in agony. A single event does not cause kidney failure, but recurrent renal calculi can damage the tubular epithelial cells, leading to functional loss of the renal parenchyma.

Etiology

Urolithiasis occurs when solutes crystallize out of urine to form stones. Urolithiasis may occur due to anatomic features leading to urinary stasis, low urine volume, dietary factors (eg, high oxalate or high sodium), urinary tract infections, systemic acidosis, medications, or, rarely, inheritable genetic factors such as cystinuria.[5][6]

Most patients with nephrolithiasis (75%-85%) form calcium stones, most composed primarily of calcium oxalate (monohydrate or dihydrate) or calcium phosphate. The other main types include uric acid (8%-10%), struvite (calcium magnesium ammonium phosphate, 7%-8%), and cystine stones (1%-2%).

The most common causes of urinary stone disease are inadequate hydration and low urine volume. The 4 most common chemical factors contributing to urinary stone formation are hypercalciuria, hyperoxaluria, hyperuricosuria, and hypocitraturia.[7][8]

The 4 major types and causes of renal calculi include:

- Calcium stones: due to hyperparathyroidism, renal calcium leak, absorptive or idiopathic hypercalciuria, hyperoxaluria, hypomagnesemia, and hypocitraturia [9][10]
- Uric acid stones: associated with a pH of less than 5.5, a high intake of purine-rich foods (fish, legumes, meat), or cancer; may also be associated with gout [11][12][13]
- Struvite stones: caused by Gram-negative, urease-producing organisms that break down urea into ammonia [14]
    - Common organisms include *Pseudomonas, Proteus,* and *Klebsiella.*[14] However, *E coli* does not produce urease and is not associated with struvite stones.[14]
- Cystine stones: due to an intrinsic metabolic defect causing the failure of the renal tubules to reabsorb cystine, lysine, ornithine, and arginine;[15] visually opaque and amber

Of these, uric acid and cystine are the most likely stone types that develop recurrences.[16]

Many drugs are known to cause renal stones, including the following:

- Atazanavir
- Guaifenesin
- Indinavir
- Silicate overuse
- Sulfonamide
- Triamterene [17][18][19][20][21][22][23]

There also appears to be a genetic association with the production of renal calculi.[16] In some families, mutations may cause a defect in the renal tubular handling of calcium and other substrates.[24]

Nephrolithiasis risk factors include the following:

- A positive family history of urolithiasis increases the incidence of new stone formation 2.5-fold.
- A prior personal history of urolithiasis increases the risk of new renal stones by 15% during the first year and an average of 50% over the following 10 years.[25]
- Diabetes, hypertension, gout, male sex, metabolic syndrome, and obesity appear to increase the risk of urolithiasis.[26]
- Increased urinary acidity (pH ≤5) promotes uric acid stone formation.[11] This is often associated with chronic diarrhea, gout, and metabolic acidosis.[11]
- Increased oxalate absorption (from malabsorption syndromes, gastro bypass surgery, and similar) can greatly increase urinary oxalate levels and calcium oxalate stone production.[27]

- Low urinary volume due to inadequate fluid intake leads to relative dehydration with increased urinary concentrations and crystal formation.[28]
- Urinary tract infections increase urinary pH in the presence of bacteria that produce urease, predisposing the patient to struvite calculi formation.[14]

In general, higher total dietary fiber, fruit, and vegetable intake, reduced animal meat protein, and a low sodium diet tend to reduce nephrolithiasis. A moderate calcium (dairy) intake is also recommended. Dietary risk factors include the following:

- High salt intake increases urinary osmolarity and promotes hypercalciuria.
- High animal protein (eg, meat) intake increases uric acid production, and hyperuricosuria stimulates proximal renal tubular citrate reabsorption and causes low urinary pH, hypocitraturia, and hypercalciuria.
- High oxalate foods lead to hyperoxaluria.
- Very low dietary calcium intake increases oxalate absorption; excessive calcium intake should also be avoided, as it causes milk-alkali syndrome and hypercalciuria.
- Inadequate fluid intake is a risk factor. (Need sufficient fluids to maintain a minimum of 2000 mL urine output daily.)
- Higher sugar-sweetened beverages: 1 daily sugar-sweetened soda increased urinary stone production by 22% to 33% compared to drinking <1 per week.
- Lower coffee consumption appeared to be a dietary risk factor for nephrolithiasis. (Individuals with the highest caffeinated coffee consumption enjoyed a much lower incidence of nephrolithiasis).
- Fruit juices do not confer substantial benefits. Lemon juice may provide a benefit if ingested in sufficient quantity due to its high citrate content. Cranberry juice has not been confirmed to be helpful.[16][29][30][31][32][33][34][35][36][37][38][39][40]

Epidemiology

Overall, urinary stone prevalence in the United States (US) has increased from 3.8% in 1970 to 8.8% in 2010.[1] This rate has continued to increase and was last reported as 10.1% in 2016.[16][41] Men have a higher rate of stone disease than women, roughly now at 2:1, although the rate of increase among women is increasing faster than among men.[16]

For patients with a history of a previous urinary stone, recurrence rates approach 50% at 10 years. There has traditionally been a high incidence of urinary stones in the Southeastern and South Central US, termed the “stone belt,” which probably reflects the hot weather climate and relative dehydration in these areas.[16][42][43]

Before the development of modern urologic techniques for treatment, mortality from untreated staghorn (infection) calculi was 27% (see **Image.** Staghorn Renal Calculus). Currently, mortality from stone disease is rare, although there is still a significant rate (28%) of renal deterioration with certain stone types, particularly staghorn (struvite or infection) stones and pyonephrosis (obstructive pyelonephritis).[44][45][46]

Kidney stone risk increases with age. The highest incidence is in men aged older than 80 years.[41] The increase in incidence in children and adolescents is rising faster than the general population.[16][47][48] Unlike adults, in the pediatric age group, the highest incidence of nephrolithiasis is among female adolescents.[49] Black and Hispanic populations have the lowest incidence of nephrolithiasis.[50] This does not represent genetic differences but socioeconomic factors, healthcare access, and dietary preferences.[16]

A systematic review demonstrated that first-time stone formers had a 26% median recurrence rate over the following 5 years.[51] A recent comprehensive meta-analysis identified risk factors that increased the likelihood of a kidney stone recurrence.[52] These risk factors were the following:

- Diabetes

- Family history of nephrolithiasis
- Hypertension
- Obesity (higher body mass index)
- Prior personal history of urolithiasis
- Surgical intervention associated with the first or earlier stones
- Uric acid urolithiasis
- White race
- Younger age when diagnosed with nephrolithiasis [16][52]

Nephrolithiasis has been linked to various systemic disorders such as cardiovascular disease, hypertension, chronic renal failure, diabetes, metabolic syndrome, and obesity.[16][53] For example, patients with nephrolithiasis and diabetes have lower average urinary pH levels and greater urinary oxalate than stone formers without diabetes. Both of these conditions significantly increase the risk of calcium oxalate nephrolithiasis.[54]

Patients with nephrolithiasis are also more likely to develop hypertension, atherosclerosis, strokes, myocardial infarctions, and particularly renal failure than the non-stone-forming general population.[16][55][56][57][58][59][60][61][62][63][64][65][66][67] Chronic renal failure and permanent kidney damage are more likely in patients with multiple nephrolithiasis recurrences, primary hyperoxaluria, and renal tubular acidosis, as well as in those with uric acid, struvite (infection), staghorn, and cystine stones.[16][67][68][69][70]

Multiple extracorporeal shockwave lithotripsy treatments do not appear to significantly or permanently affect renal function.[71][72] Global warming and climate change are expected to further increase the incidence of nephrolithiasis in the US by 30% by the year 2050, especially in warmer regions.[16][73] Worldwide, the incidence of kidney stones has increased over the last 3 decades, placing a greater financial burden on global healthcare delivery systems.[1][16][74][75][76]

Pathophysiology

Most urinary stones start as Randall plaque at the junction of the nephron's collecting tubule and the renal pelvis in the papilla. These plaques start in the suburothelium and then gradually grow until they break through the urothelium into the renal pelvis. They form an anchored lithogenic nidus for stone formation.

Once in continuous contact with urine, calcium oxalate layers typically start forming on the calcium phosphate nidus (all Randall plaques are composed of calcium phosphate). Calcium oxalate stones tend to form when the urinary pH is lower than 7.2, while calcium phosphate will form in more alkaline urine.

Hyperparathyroidism and similar metabolic disturbances like renal tubular acidosis typically form stones primarily or significantly composed of calcium phosphate. Overly acidic urine is the primary cause of uric acid stones (not hyperuricosuria).[77][78] Most renal calculi are made of radiopaque calcium compounds followed by radiolucent uric acid. Supersaturation of the urine is the common denominator in all cases of renal calculi.

In some cases, calcium oxalate stones may deposit in the renal papilla. Calcium phosphate stones usually precipitate in the basement membrane of the thin loop of Henle and may erode into the interstitium. The colicky pain is usually due to the dilatation and spasm of the ureter and stretching of the renal capsule.

Calcium oxalate stones are the most common type of renal calculi, comprising 70% to 75% of all urinary stones.[16][79] While chemically identical, they may present as 2 different crystalline forms: calcium oxalate monohydrate (whewellite, very hard) or a dihydrate (weddelite, brittle). These stones typically form in acidic urine but may be found with calcium phosphate, forming the central nidus.

- Calcium oxalate monohydrate calculi are extremely hard and usually present with a smooth, rounded surface. They are typically dark brown.

- Calcium oxalate dihydrate stones will be quite brittle with small, sharp, jagged edges. They are usually yellow to light brown.

Calcium phosphate calculi may be seen as the less soluble carbonate apatite (hydroxyapatite, apatite) and brushite (calcium hydrogen phosphate). They account for about 10% of all renal calculi.[16][79] Hydroxyapatite is more commonly found than brushite and is the calcium salt that forms bone. In general, calcium phosphate stones tend to grow faster and larger than calcium oxalate calculi. These stones are off-white, grayish-white, or yellowish in color. Calcium phosphate stones form in alkaline urine and are typically associated with abnormal metabolic factors, such as hyperparathyroidism and renal tubular acidosis.[10][80]

Uric acid calculi only form in acidic urine, usually with a pH less than 5.5. This acid is the most common composition of bladder stones and is typically radiolucent.[11] Uric acid accounts for 8% to 10% of urinary calculi, and the incidence is increasing worldwide.[16][79] This condition is most closely associated with diabetes, morbid obesity, metabolic syndrome, and older age at presentation.[16]

This is the only kidney stone that can be reasonably expected to dissolve if the urinary pH is sufficiently elevated and maintained.[11] This type of stone is also more likely to form from excessive urinary acidity rather than hyperuricosuria.[11][81] Uric acid stones may be yellow, orange, reddish, or brown, depending on the amount of blood-derived pigment they may have accumulated. Preventive treatment involves urinary alkalinization and possibly allopurinol if there is hyperuricosuria.[11]

Struvite or triple phosphate (calcium, ammonium, magnesium phosphate) stones are always associated with infection and increased pH levels.[14] They frequently form staghorn stones and comprise 7% to 8% of all urinary calculi worldwide.[16] Struvite stones are caused by the action of urease from bacteria, which increase the urinary pH and generate ammonia, leading to triple phosphate precipitation and stone formation.[14][82]

To treat the infection adequately, complete elimination of all stone material is necessary.[14] Struvite stones appear chalky, white, or grayish. Their surface is usually smooth and relatively brittle, as they can be broken relatively easily.

Cystine stones are caused by an uncommon familial genetic defect and account for only 1% to 2% of all urinary stones.[15] They tend to be amber, tan, or yellowish in color with a waxy appearance. Cystine stones may turn somewhat greenish after exposure to air. The stones are not calcified but resistant to shockwave therapy; therefore, laser lithotripsy is usually the preferred treatment.[15] Preventive treatment includes very high levels of hydration (>3 liters of urine/day), urinary alkalinization to a pH of 7.5 or more, and tiopronin, a reducing compound, if necessary.[15]

In addition to cystinuria, several other uncommon inherited disorders cause nephrolithiasis, including adenine phosphoribosyltransferase deficiency (causing dihyroxyadenine calculi), Bartter syndrome, Dent disease, distal tubular acidosis, familial hypomagnesemia with hypercalciuria and nephrocalcinosis, hereditary hypophosphatemic rickets, hereditary xanthinuria, Lesch-Nyhan syndrome, and primary hyperoxaluria.[80][83][84][85][86][87][88][89][90][91]

Natural urinary stone inhibitors include the following:

- Citrate
- Glycosaminoglycans
- Nephrocalcin
- Tamm-Horsfall protein
- Uropontin
- Water (increased urinary volume)

Histopathology

Urine microscopic crystal analysis can help identify the type of kidney stone present. Crystalluria alone does not necessarily indicate the presence of urinary calculi, but it does suggest the type of stone that may be produced.

Crystalluria also indicates that conditions allowing a stone to form are present.

Common types of urinary crystals include the following:

- Calcium oxalate: envelope, spindles, ovals, octahedral, picket fences, or dumbbell-shaped. They often appear to have an "X" in their center. Ethylene glycol toxicity is associated with the picket fence type of urinary crystals.
- Calcium phosphate: colorless, they often appear as plates, stars, stellate, or as a collection of needles or rosettes. These are usually found in more alkaline urine.
- Cystine: hexagonal-shaped crystals. They appear as almost perfect benzene rings under the microscope.
- Struvite (magnesium, ammonium, calcium phosphate): This mineral is found only in alkaline urine and appears coffin-lid-shaped; this is always associated with urinary infections.
- Uric Acid: rhomboidal-shaped crystals, rosettes, or plates. Uric acid crystals are found in acidic urine with a pH of 5.5 or less.[92]

History and Physical

Patients with nephrolithiasis often present with hematuria, as 85% of patients demonstrate at least microscopic hematuria on urinalysis. Renal calculi may often be totally asymptomatic, with the stones being detected unexpectedly on imaging performed for a hematuria evaluation (or for unrelated reasons).[93] Symptoms are unlikely until or unless the calculi become infected or cause some degree of urinary obstruction, either within the kidney, at the ureteropelvic junction, or in the ureter.[94]

Patients with stone disease will also present with acute, severe flank pain (renal colic) that will often radiate to the abdomen and the groin, testicle, or labia.[94] The pain is often sharp, quite severe, and may also be colicky.[94] The pain is often associated with nausea and vomiting due to the embryological origins of the urogenital tract.[94]

Renal colic usually peaks within 90 to 120 minutes, and the pain radiation follows dermatomes T10 to S4.[94] The first phase may wake the patient up from sleep, is quite severe and somewhat intermittent, with excruciating pain coming in waves (renal colic). More constant pain characterizes the second phase, which may last 3 to 4 hours. The third phase is associated with mild pain relief, but intermittent waves of pain (colic) may persist. This phase may last 4 to 16 hours.[94]

The physical exam may reveal costovertebral tenderness and hypoactive bowel sounds. The testis and pubic area may also be tender to touch. Fever is rarely seen in renal colic without infection, but the presence of an elevated body temperature, pyuria, and leucocytosis may indicate infection, pyelonephritis, or pyonephrosis.

If infected, such patients will present with fever, chills, or other systemic signs of sepsis. This condition, called pyonephrosis or obstructive pyelonephritis, is potentially severe and life-threatening, requiring emergency decompression surgery to drain the renal pelvis.[95] The condition can rapidly progress to urosepsis, shock, and death if the renal pelvis is not quickly drained surgically, either percutaneously or cystoscopically, by double-J stenting or nephrostomy tubes.[95]

Patients who are more severely ill, hemodynamically unstable, or who have a large obstructing stone burden generally do better with percutaneous drainage, where manipulation of the infected renal unit and the obstructing stones is minimized.

High-risk factors for nephrolithiasis include the following:

- Bone disorders
- Chronic diarrhea, malabsorption
- Diabetes and obesity (especially in women)
- Excessive Vitamin C ingestion (>1,000 mg/day)
- Family history of kidney stones

- Gastrointestinal disease
- Gastro bypass surgery (especially Roux-en-Y)
- Gout
- Hyperparathyroidism
- Metabolic syndrome
- Obesity
- Prior urinary stones
- Renal failure
- Renal tubular acidosis
- Sarcoidosis (which can increase calcium and vitamin D levels)

Evaluation

Urinalysis should be obtained on every patient with a suspected kidney stone. Hematuria is usually present, but up to 15% of kidney stone patients will not demonstrate microscopic hematuria. The presence of urinary crystals may suggest urolithiasis. Positive nitrites, leukocytes, and bacteria suggest a possible urinary infection, which should be cultured and treated aggressively.[96] Other labs to obtain would include a WBC with differential if the patient is febrile or has a urinalysis suggestive of a possible infection.

KUB (flat plat abdomen including the kidneys, ureters, and bladder) can be obtained to screen for the presence of significant nephrolithiasis but may often miss small stones, calculi hidden by overlying bowel, or radiolucent stones if uncalcified.[97][98]

Ultrasound may be useful for assessing obstruction and resultant hydronephrosis, especially in pregnancy, where x-ray studies are discouraged. Ultrasound can also identify uric acid and other non-calcific renal stones if they are large enough (usually greater than 4 mm), but it can also miss the presence of stones less than 5 mm in size and cannot easily identify ureteral stones (see **Image.** Nephrolithiasis, Ultrasound).

The resistive index, measured using ultrasonography, can suggest ureteral obstruction. The formula is:

- Resistive index=(peak systolic velocity - end-diastolic velocity)/peak systolic velocity
- Values of 0.70 or less are considered normal, while higher values suggest obstructive uropathy.
- Bilateral high resistive indices suggest medical renal disease, while a unilateral high resistive index (0.75 or higher) suggests an obstruction, such as from a stone.
- Once a ureteral stone has been identified, the lower the resistive index, the more likely the stone will pass spontaneously.[99]

A non-contrast abdominal and pelvic computed tomography scan is considered the "gold standard" as it is the most sensitive and reliable test to diagnose urolithiasis and will also provide information regarding obstruction with resultant hydronephrosis.[100][101][102][103][104][105][106]

Even without intravenous contrast, the correct diagnosis can be easily made. If contrast is necessary, performing the non-contrast study first helps eliminate the diagnosis of urinary stones. Obscuring urinary stones with intravenous contrast can make it much more difficult to determine their size, number, or shape, complicating decisions on optimum treatment and possible surgery.[100][101][102][103][104] The initial use of intravenous contrast for computed tomography (CT) scans in patients with abdominal pain is not recommended. In many cases, an atypical abdominal pain will ultimately turn out to be a kidney stone that has moved or the presence of a urological anatomical variant such as a horseshoe kidney.

A KUB should be done immediately before the abdominal CT scan in all cases where urolithiasis is suspected or if the urinalysis shows gross or microscopic hematuria; this will provide vital information helpful in tracking or following the progress of the stone, its degree of calcification, and its shape, which cannot always be reliably determined from the CT scan alone.[97][98]

- Since contrast is used so often in abdominal CT scans, especially when ordered from the emergency department, it is recommended that the KUB be done first to facilitate tracking of any stones discovered. The cost and additional radiation exposure are minimal, and the information provided is extremely helpful to the follow-up clinician/urologist. Once intravenous contrast has been given, a clear x-ray without dye interference is impossible.
- A plain KUB done later in follow-up can easily determine whether a stone seen on the earlier KUB is still present. Otherwise, a repeat CT scan or a surgical retrograde pyelogram can be performed.

Combining a KUB with a renal ultrasound is an effective and reasonable alternative to CT scans as these can show hydronephrosis, measure resistive index, and demonstrate both calcified and non-calcified renal calculi with lower radiation exposure at a lower cost.

European Guidelines for Patients with Renal Calculi

- Check urine for hematuria, pH, and bacteria.
- Obtain a urine culture.
- Order a blood, urea, nitrogen (BUN) test and check serum creatinine.
- Order serum calcium, uric acid, sodium, and potassium levels.
- Order a complete blood count and C-reactive protein test.
- Obtain a coagulation profile in case surgical intervention is necessary.
- Obtain a non-contrast CT scan.[107]

Treatment / Management

Renal calculi can be extremely painful when they cause a ureteropelvic junction or ureteral obstruction or they become infected. Pain control may require opioids, but intravenous (IV) nonsteroidal anti-inflammatory drugs can also be quite effective while avoiding narcotic side effects. As patients with renal colic will often experience nausea and vomiting, IV hydration and antiemetics may be required acutely.

Many stones may be watched conservatively, with intervention planned as an outpatient. Smaller stones (<5 mm) have a greater chance (90%) of passing on their own with medical expulsion therapy (usually tamsulosin, alfuzosin, nifedipine, alfuzosin, silodosin, or mirabegron).[108][109][110][111][112][113][114] Any hint of a urinary tract infection should be treated aggressively with antibiotics, especially in patients with diabetes.[115][116][117]

Study results show that desmopressin can lower the pain of renal calculi. Anecdotal reports also indicate that calcium channel blockers or alpha-blockers can relieve pain due to the relaxation of the ureter and help the distal passage of the stone.[109][110][111] Mirabegron has shown similar efficacy in facilitating ureteral stone passage.[112][113] The urine should be strained for stones, and it should always be sent for chemical analysis.

There are several cases where urgent intervention is required:

- Obstructing stone in a patient with a urinary tract infection, fever, or sepsis—called pyonephrosis or obstructive pyelonephritis
    - This condition requires urgent surgical decompression by urology or interventional radiology.
- Nausea or uncontrolled pain with outpatient care
- An obstructing stone in a solitary kidney

- Any degree of simultaneous bilateral obstruction (can easily lead to renal failure)
- Any degree of obstruction with a rising creatinine

In the case of a urinary tract infection or urosepsis with an obstructing stone, the obstruction should first be relieved with either a ureteral double J stent or nephrostomy tube placement. The decision of which treatment modality is most appropriate should be made by urology in consultation with the patient and family. The more severely ill the patient is, the greater the benefit will be from a nephrostomy tube (at least initially). Definitive stone management can then occur once the infection is no longer active. Patients who are morbidly obese and those who cannot be safely taken off blood thinners may require a double J stent, regardless.

Electively, renal calculi can be surgically managed in several ways:

- Extracorporeal shockwave lithotripsy can break up stones anywhere in the urinary tract but is primarily used in the kidney and upper ureter.[118][119]
- - This is a less invasive therapy but may need to be repeated for larger, harder stones.
  - This is not preferred for stones larger than 2.5 cm.[118]
  - Double J stents may be required for ureteral stones or larger stone burdens (usually >10 mm) where multiple stone fragments could otherwise cause an obstruction (steinstrasse).[118][119][120]
  - Contrary to earlier concerns, multiple extracorporeal shockwave lithotripsy treatments do not appear to significantly or permanently affect renal function.[71][72]
  - - This is not generally recommended for cystine stones.[15]

(See the companion Statpearls reference article on "Renal Extracorporeal Therapy.")[118]

- Ureteroscopy with laser lithotripsy can be used to manage stones endoscopically; this method is preferred for ureteral stones, especially those in the lower ureter.[121][122]
- - This is a reliable treatment modality for stones with a high stone-free rate, but it depends on the surgeon's skill and experience and the kidney's internal anatomy.[121]

(See the companion StatPearls reference article on "Ureteroscopy.")[121]

- For large (>2.5 cm) stones in the renal pelvis, percutaneous nephrolithotomy can be performed and is generally the preferred treatment modality.[123][124]
- Combined treatments (ureteroscopy and percutaneous surgery) may be used in unusually difficult or complex cases.[125]
- Robotic-assisted laparoscopic pyelolithotomy with intracorporeal pyeloscopy can be used selectively in complicated stone cases, such as horseshoe kidneys.[126][127]

24-hour urine testing to identify the underlying chemical risk factors for future stone production so optimal prophylactic therapy can be administered should be carefully discussed with all patients with nephrolithiasis once the acute stone episode has been properly and adequately treated. This testing is strongly recommended by multiple published guidelines, including the American Urological Association.[128][129] Twenty-four-hour urine testing is particularly important if the patient has had multiple stones in the past, has a solitary kidney, has renal failure, or is a high surgical risk.[128][129][130][131]

Testing involves obtaining a basic metabolic panel, a serum uric acid, and a 24-hour urine collection for kidney stone prevention analysis.[130] Patients must understand that this represents their commitment to follow a long-term course of therapy for stone prevention and that no treatment plan is foolproof. An occasional stone may still be produced but is much less likely while on directed prophylaxis. Abnormal urinary chemistry contributing to recurrent stones will be found in over 93% of patients with nephrolithiasis tested by 24-hour urine and blood testing.[16][129]

[132] Physicians evaluating 24-hour kidney stone results should look at the normal ranges and what may be optimal. [130]

For example:

- Optimal 24-hour urinary calcium no more than 250 mg
- Oxalate less than 25 mg
- Citrate more than 600 mg
- Urinary volume of 2500 mL or more
- Urinary uric acid at 600 mg or less [130][133]

While these levels may not be realistically obtainable in every patient, they are used as goals for treatment where the intention is to optimize as many urinary chemistry levels as possible (even if technically within normal limits).[130] [134][135][136]

Analyses of 24-hour urine tests are often complicated but straightforward for most patients.[9][11][15][27][129][130] [137][138][139][140][141]

- Patients with hypercalcemia should have parathyroid hormone levels checked for possible hyperparathyroidism, as these patients typically form calcium phosphate stones.
- Hydration should be optimized to produce at least two liters of urine daily. The optimal urinary volume is 2500 mL daily or more.
- Thiazides are used for hypercalciuria. They may also incidentally increase diuresis (but can also increase serum uric acid levels and decrease urinary citrate.)
- Potassium citrate and sodium bicarbonate are the most used urinary alkalinization agents for hypocitraturia and aciduria.
- Allopurinol will help lower high urinary and serum uric acid levels, but most patients with uric acid stones have aciduria and require urinary alkalinization, usually with potassium citrate.
- Patients with hyperoxaluria can use low-oxalate diets, dietary calcium supplements with higher-oxalate meals, and optimization of all other stone-related urinary chemistries.
- Cystine stone formers are treated with urinary alkalinization (to pH 7.5 or higher), vigorous hydration (sufficient to make 3 liters of urine a day or more), and sometimes with tiopronin, a reducing agent that makes cystine more soluble.
- Patients with struvite stones require infection control and complete removal of all infected stone pieces and fragments. Acetohydroxamic acid, a urease inhibitor, can be helpful in some cases but is associated with significant side effects.

Appropriate thiazide use has been shown to lower hypercalciuric calcium stone disease from 2.94 to 0.05 stones per year (P <0.001), while long-term use of potassium citrate can diminish calcium stone production by 80%.[142] [143] Limiting sodium intake is important to allow thiazide to perform its hypocalciuric action.[144] A daily increase of 100 mEq of sodium will raise urinary calcium excretion by 50 mg.[9] These 3 drugs will be sufficient to treat the majority of patients.

(See the companion StatPearls reference article, "24-Hour Urine Testing for Nephrolithiasis: Guide to Interpretation," for more details on testing, result analysis, and prophylactic therapy.)[130]

Hospital Admission is recommended in the following situations:

- Inadequate pain relief with oral analgesics
- Obstructed pyelonephritis (pyonephrosis)

- Patient with a transplanted kidney and renal calculi
- Presence of renal calculi and pyelonephritis or sepsis
- Intractable nausea and vomiting [141]

Dissolution therapy does not work for calcium stones but may be used to manage uric acid and cystine stones. Uric acid calculi can be dissolved by consistently making the urine alkaline with potassium citrate or sodium bicarbonate.[11][141][145] Potassium citrate is usually preferred due to its lower sodium load. In addition, allopurinol can be used to reduce renal uric acid excretion.[137][141] Cystine stones can be managed with tiopronin, aggressive fluid intake, increased urinary volume, and optimal alkalinization therapy (to a pH of 7.5 or higher).[15][141]

Struvite (triple phosphate) stones can sometimes be managed with acetohydroxamic acid—a urease inhibitor that irreversibly blocks urease.[128][141][146] Acetohydroxamic acid penetrates bacterial cells well, synergizes with many antibiotics, reduces urinary alkalinity levels, lowers ammonia production, and has a high renal clearance. Unfortunately, acetohydroxamic acid also has significant side effects (including hemolytic anemia), cannot be used in renal failure, and is generally considered adjunctive to surgical therapy. This therapy may also be used in patients who are not surgical candidates. Renacidin (hemiacidrin) irrigation may be used cautiously in highly selected cases to dissolve struvite stones, as improper use can be lethal.[14][141][147][148][149][150][151]

Differential Diagnosis

The following may present with similar symptoms to renal calculi:

- Appendicitis
- Benign familial hematuria
- Cholecystitis
- Costochondritis
- Diverticulitis
- Focal nephronia
- Glomerulonephritis
- Hernia
- Lobar pneumonia
- Pelvic inflammatory disease
- Pyelonephritis

Prognosis

Close to 80% to 90% of renal calculi pass spontaneously. About 3% of patients need admission because of pain, inability to pass the stone, sepsis, intractable nausea and vomiting, or dehydration. A few patients may develop urinary tract obstruction and an upper urinary tract infection, resulting in urosepsis, pyelonephritis, or pyonephrosis (obstructive pyelonephritis). Most of these patients require an urgent surgical procedure to bypass the stone or otherwise drain the renal pelvis until the infection is resolved; then, elective surgery can be safely performed to remove the calculus.

The recurrence rate of renal calculi has been reported to be about 50% within 10 years, but some patients form stones far more frequently. Twenty-four-hour urine testing and prophylactic therapy can significantly reduce the recurrence rate of nephrolithiasis, but the results must be properly interpreted, and patients need to be sufficiently motivated to follow treatment suggestions long-term, even if immediate relief is not evident.

Individuals with ongoing metabolic disorders or malignancies are also at greater risk for recurrences. The key for all patients with renal calculi is to stay hydrated as no medical therapy is likely to succeed without adequate hydration

and sufficient urinary fluid output, generally considered at least 2000 mL daily (optimally 2500 mL or more).[133]

Complications

The following are possible complications of nephrolithiasis:

- Abscess formation
- Forniceal rupture
- Hydronephrosis
- Perinephric abscess
- Pyelonephritis
- Pyonephrosis (obstructive pyelonephritis)
- Renal colic
- Renal failure, atrophy, and end-stage kidney disease
- Sepsis and urosepsis
- Ureteral calculi with subsequent colic, obstruction, pain, and scarring
- Urinary extravasation
- Urinoma

Deterrence and Patient Education

Patents should generally avoid diets high in calcium while limiting excessive salt and animal protein intake. A low oxalate diet is also recommended, particularly for patients with hyperoxaluria or recurrent calcium oxalate stones. Overly restrictive dietary calcium is not recommended as it may lead to a lack of intestinal oxalate binding and hyperoxaluria.[27] Twenty-four-hour urine testing for kidney stone prophylaxis is available for any patient with urolithiasis who is interested, willing, and motivated to follow long-term preventive treatment.[128]

Pearls and Other Issues

Other important information to note includes:

- Ultimately, the success of any kidney stone preventive treatment program will depend on the patient's willingness to follow a long-term course of treatment that will involve some level of dietary modifications, medications, personal sacrifice, and lifestyle change, even if immediate benefits are not evident.
- Patients on optimal treatment may still make stones, albeit fewer than otherwise.
- Despite its relatively high oxalate content, increased coffee consumption appears to help reduce nephrolithiasis.[34][35]
- Some patients may only partially follow their therapy regimens, and many will revert to their previous diets and behaviors; however, this can be minimized by warning patients of this natural tendency and seeing patients periodically to remind them of the benefits of maintaining preventive therapy.
- Patients may also develop an over-reliance on drug therapy, leading to failure to maintain the dietary changes and fluid intake goals requested.
- Patients at higher risk of developing chronic renal failure and end-stage renal disease include those with calcium phosphate, cystine, struvite, and uric acid stones, as well as those with primary hyperoxaluria, cystinuria, repeated stone recurrences, larger stones, and staghorn calculi.[16]
- The recommended diet for stone formers would include adequate hydration, low animal protein, low salt, moderate calcium (dairy), and high fiber, fruit, and vegetables (except for high oxalate-containing vegetables

like spinach).[37][128]

- A low calcium diet is not recommended as it leads to increased intestinal oxalate absorption, ultimately forming higher calcium oxalate stones.[16][38][74][152][153]
- Excessive vitamin C intake (>1,000 mg/day) can increase hepatic oxalate production and exacerbate hyperoxaluria.[27][154][155][156][157][158]

Enhancing Healthcare Team Outcomes

The presence of nephrolithiasis places a significant burden on the healthcare system and is a significant cause of patient morbidity. An interprofessional team of nurses, nurse practitioners, physician assistants, dieticians, pharmacists, urologists, nephrologists, and primary care physicians should educate and remind patients about the benefits of preventive therapy. Nutritionists and dieticians are especially important to help motivated individuals have long-term success.[136]

The health care team involved in the care of patients with nephrolithiasis has an absolute obligation to inform patients on preventative dietary changes and how to optimize urine chemistries, particularly in cases of multiple stone recurrences, solitary kidneys, high surgical risk factors, or those in the pediatric age group.[159][160]

Patients with recurrent renal calculi should be referred to a specialist urologist for workup and to rule out an anatomical or metabolic problem. Only through open and effective communication between the team members can the morbidity and recurrence rate of renal calculi be lowered.

Review Questions

- Access free multiple choice questions on this topic.
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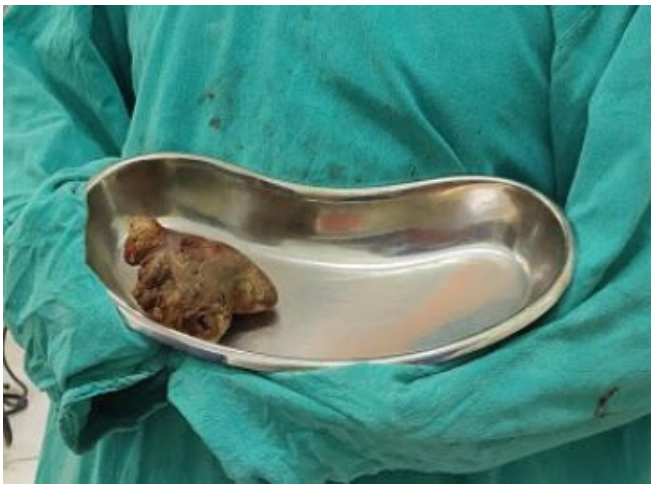
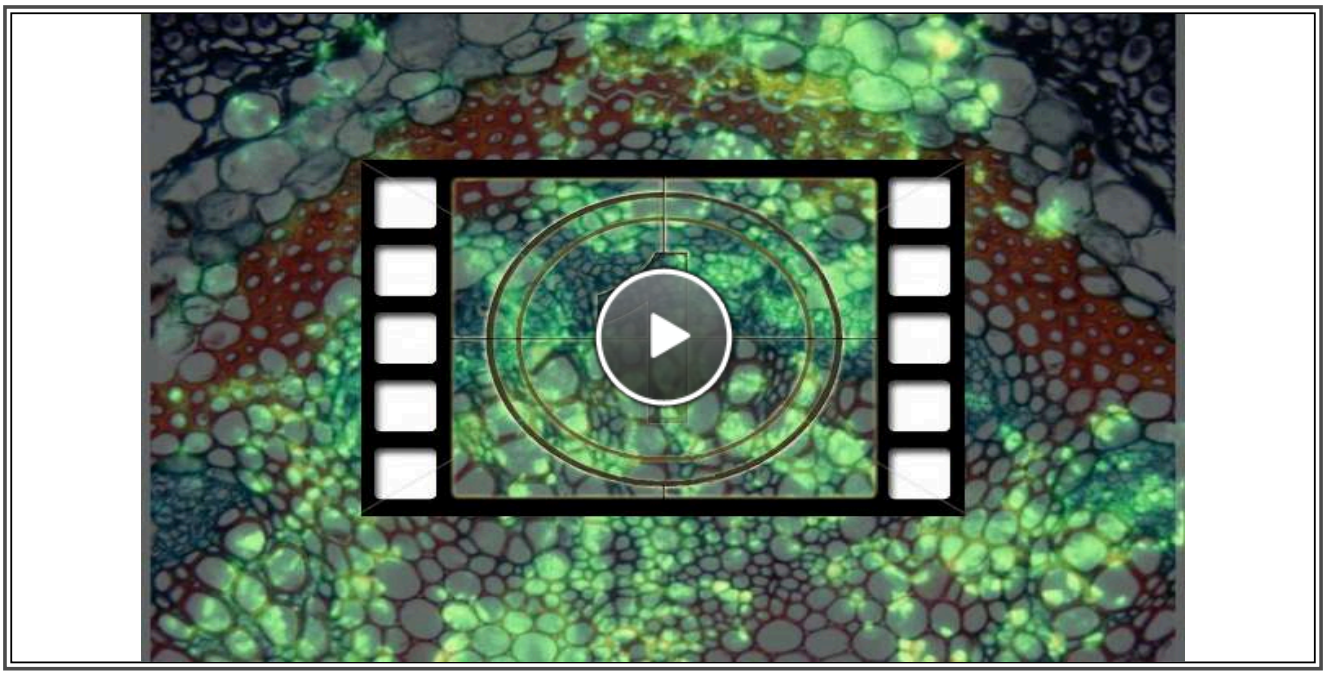
Figures



Staghorn Renal Calculus. These are complex renal stones that occupy a majority of the renal collecting system and are associated with high morbidity. Contributed by S Munakomi, MD



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Nephrolithiasis, Ultrasound. The image shows a normal kidney with grades of hydronephrosis, twinkle artifact, and ureterovesical junction stone. Contributed by MK Herbst, MD

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