Diagnosis and Management of Nephrolithiasis

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KEYWORDS

• Nephrolithiasis • Urolithiasis • Management • Diagnosis

KEY POINTS

- Nephrolithiasis can be caused by general surgical conditions, including malabsorption in Crohn's disease, ulcerative colitis, and pancreatitis and can occur in patients after bariatric surgery.
- Removal of a parathyroid adenoma can significantly decrease stone formation in patients with hyperparathyroidism.
- Low-dose unenhanced computed tomography scan has emerged as the gold standard imaging modality in the acute setting, whereas retroperitoneal ultrasound scan is a common option in the nonacute setting.
- Interventions include shock wave lithotripsy, ureteroscopy, percutaneous nephrolithotomy, and, rarely, open or laparoscopic surgery.
- These options vary in likelihood of rendering the patient stone free and in respective contraindications, risks, side effects, and need for additional procedures.

INTRODUCTION

Prevalence

Nephrolithiasis is a common reason for urgent patient presentation for medical or surgical evaluation. The incidence and prevalence of kidney stones has increased in the last decades among adults, adolescents and children. In their lifetime, 7% of females and 11% of males in the United States will be affected by kidney stones.[1]

Disclosure Statement: The authors have nothing to disclose.

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Surg Clin N Am 96 (2016) 517–532

http://dx.doi.org/10.1016/j.suc.2016.02.008 **surgical.theclinics.com**

0039-6109/16/$ – see front matter

Extent and Cost

Nephrolithiasis results in at least 1.2 million emergency department visits in the United States annually and 41,000 surgical interventions.[2,3] The estimated cost of kidney stones in 2007 was $3.8 billion with a projected further increase in cost of $1.2 billion by 2030.[4]

Morbidity and Prognosis

The most common morbidity of a kidney stone is renal colic, a condition resulting in pain, often acute, with need for acute medical and surgical intervention. More severe sequelae include sepsis from an obstructed infected stone and deterioration in renal function. Furthermore, ureteral stones, if left impacted for prolonged periods, can result in ureteral scar and stricture.[5]

Kidney stone recurrence rates vary by the underlying metabolic cause, but on average, after a stone event, 31% recur with another symptomatic kidney stone within 10 years.[6]

Risk Factors

Risk factors for kidney stone formation include increasing age, male sex, race (highest among whites), lower socioeconomic status, obesity, diabetes, and gout disease.[1] Additionally, dietary and endocrine factors are also known to greatly affect risk of kidney stones.[7]

Relevance to the General Surgeon

Likely the most common way for a kidney stone to come to the attention of a general surgeon is as a differential diagnosis for acute abdomen.

Outside of the acute setting, of particular relevance to the general surgeon, is the knowledge that malabsorptive intestinal diseases and conditions, such as Crohn's disease, ulcerative colitis, pancreatitis, and short gut syndrome increase risk of stone formation. The same is true for surgical interventions, such as bariatric surgery, colectomy, and any surgery leading to less absorptive physiology or decreased small bowel length.

Further, hyperparathyroidism, although uncommon (comprising <5%) is an important modifiable cause of renal stones.[8] The general surgeon can substantially decrease the risk of or completely prevent stone formation via surgical removal of an active parathyroid adenoma.

Finally, knowledge of the procedures performed to remove stones is useful for the general surgeon, as there is a known risk of injury to the adjacent structures, including pleura and colon for which intraoperative assistance of the general surgeon may be requested.

RELEVANT ANATOMY AND PATHOPHYSIOLOGY

Anatomy

Anatomically the kidneys are retroperitoneal organs in close proximity to liver, spleen, colon, duodenum, adrenals, diaphragm, and the lowest ribs.[9] This anatomy is relevant when stones are removed percutaneously, as there is potential injury to these organs when a percutaneous needle is introduced and an access tract, often up to 1 cm in diameter, is developed with dilators.

Should a kidney stone move and progress down the ureter, it will encounter 3 classically described areas of decreased luminal diameter. The first is at the ureteropelvic junction, the second occurs where the ureter crosses over the iliac vessels (external

compression), and the third occurs where the ureter passes through the muscle layers of the bladder wall to emerge at the ureteral orifice. These areas of narrowing are the most common sites at which a ureteral stone is likely to become impacted and result in up-stream obstruction.[9]

Pathophysiology

When a solute is added to a solution (such as urine), it dissolves until its saturation point. Because of the presence of crystallization inhibitors, the concentration of clinically relevant crystals such as calcium oxalate can exceed their saturation point, existing in a metastable supersaturated state. From this state, stones may form in urine.[10,11]

In urine, the aforementioned inhibitors include molecules such as citrate, glycoproteins, and magnesium, whereas other factors, such as epithelial cells, urinary casts, red blood cells and even other crystals, can act as nucleating centers in urine, promoting stone formation. The pH can also affect the solubility of solutes in urine.[10]

Alternatively, severe enough super saturation can result in spontaneous crystallization and stone formation in the urine of the renal pelvis (homogenous nucleation).[11]

Given the above, kidney stones can be either intraparenchymal, calyceal, in the pelvis, or in the ureter upon diagnosis.

The size and the location of a kidney stone will affect its natural history and its management. A small renal pelvis or upper pole stone is likely to travel down the ureter and pass spontaneously with or without symptoms, whereas a larger stone or a stone in the dependent lower pole is less likely to do so. Success rates of treatments are also associated with stone location, size, number, and complexity.[12]

Renal stones can have numerous chemical compositions. Calcium-based stones are by far the most common. Frequently, renal stones show a mixture of more than one of the below-mentioned crystals.

Calcium oxalate accounts for 60% to 65% of stones in North America and as high as 90% in India. These findings may result from a combination of decreased fluid intake or relative dehydration, high dietary sodium, conditions that lead to high urinary excretion of calcium, and abnormalities in oxalate handling.[13] These risk factors, as they relate to general surgical conditions, are discussed below.

Calcium phosphate accounts for 10% to 20% of stones in western countries. Calcium phosphate stones may result from states of hypercalciuria (either primary or secondary to hypercalcemia) and disorders of urinary acidification, as calcium phosphate is poorly soluble in alkaline urine.[14] Urinary alkalization occurs in states such as renal tubular acidosis but may also be iatrogenically induced by medications including carbonic anhydrase inhibitors and topiramate.[15]

Uric acid stones account for 5% to 10% of stones. They may be associated with excessive protein intake or abnormalities in protein and uric acid metabolism but form frequently in the absence of either, as the supersaturation of uric acid rapidly increases in the setting of low urine volume or low urine pH. Opposite to calcium phosphate stones, they are poorly soluble in acidic urine and cannot form in alkaline urine.[16]

Ammonium acid urate stones are rare (0.2%–3%) but warrant special mention to the general surgeon. It has been theorized that they form in the setting of gastrointestinal loss of water and electrolytes that cause a volume-depleted state with intracellular acidosis. This state results in elevated urinary ammonia excretion in high enough concentrations to crystalize with urate.[17] In North America these stones are associated with a history of inflammatory bowel diseases or ileostomy diversion, laxative abuse, and morbid obesity.[18]

Other stones include struvite, an ammonium stone composition primarily caused by chronic urinary infection with urease-positive bacteria converting urea to ammonium (1%–14%); cystine stones, caused by a genetic disorder causing an impairment of transport of the amino acid cystine (1%–4%); and other rarer stones (0%–4%).[19]

Stone composition affects surgical treatment and likelihood of recurrence. As an example, harder stones, such as cystine and some calcium phosphate stones, are resistant to shock wave lithotripsy.[20] Struvite stones typically harbor bacteria; if they are infected, they must be completely eradicated.[21] Pure uric acid stones may potentially be resolved by altering urine pH.[22] Finally, the chemical composition affects postsurgical dietary and medical management with regard to decreasing risk of recurrence.

Pathophysiology of General Surgery Conditions Resulting in Stone Formation

Certain gastrointestinal conditions and disease states increase the risk of kidney stone formation. The primary variables that can be altered are elevated urinary excretion of oxalate (stone promoter), decreased urine citrate (stone inhibitor), urine acidification (promotes some stones) and decreased urine volume (less dilution, thus, more supersaturated urine).[23]

The increased urinary excretion of oxalate is thought primarily to be caused by increased enteric uptake, primarily in the setting of fat malabsorption, thus, known as *enteric hyperoxaluria*. As fat is malabsorbed, calcium ions are saponified in it and lost via steatorrhea. Under normal circumstances, the calcium binds to oxalate to form calcium oxalate crystals, which are not absorbed by the intestine and excreted fecally. In the absence of free luminal calcium, however, there is excess free oxalate, which is absorbed by the intestine.[23] At the same time, unconjugated bile salts and long-chain fatty acids have been found to increase oxalate permeability in the intestine, further increasing the uptake of oxalate.[24,25] Additionally, in disease states in which vitamin B6 is malabsorbed, the liver is induced to produce endogenous oxalate.[25] Finally, loss of oxalate degrading bowel flora such as *Oxalobacter formigenes*, either caused by inflammatory bowel conditions or antibiotic use, will increase availability of intestinal oxalate to be absorbed. Because oxalate is predominantly eliminated by renal excretion, increased oxalate uptake leads to increased urinary oxalate and calcium oxalate stone formation.[26,27] In addition to kidney stone formation, enteric hyperoxaluria can lead to renal insufficiency and ultimately end-stage renal disease. The true incidence of end-stage renal disease remains unreported, and most of our current knowledge comes from case series.[28] In one study, 8 of 11 patients presenting with nephropathy secondary hyperoxaluria eventually had end-stage renal failure, emphasizing the gravity of this condition and need for awareness of it in patients with fat malabsorptive conditions.[29]

Citrate is a potent inhibitor of stone formation. Its urinary levels are primarily controlled by acid-base status, as acidosis results in increased citrate utilization by mitochondria; thus, less free citrate is available for urinary excretion.[30] Therefore, any state resulting in chronic subclinical metabolic acidosis can result in low urinary citrate.

Acidosis can also result in excess net renal excretion of acids as a compensation mechanism, lowering the urinary pH and therefore increasing the risk of uric acid and ammonium acid urate stones.

Any state resulting in excessive gastrointestinal fluid losses will result in decreased urine volume and, thus, higher supersaturation of urine crystals and stone formation.[10]

Examples of disorders with increased risk of stone formation

Patients with ileostomy tend to lose excessive fluids. This loss results in more concentrated urine with higher likelihood of solutes being supersaturated. The most common

stones, calcium oxalate, become more common. Additionally, loss of bicarbonate leads to a lower urinary pH, increasing the risk of urine acid and ammonium acid urate stones.[31]

Inflammatory bowel diseases Patients with Crohn's disease and ulcerative colitis are found to have an increased incidence in calcium oxalate stones compared with a healthy population. This increased incidence is thought to be primarily associated with fat malabsorption and resulting enteric hyperoxaluria[32]

Chronic pancreatitis Pancreatic insufficiency often leads to fat malabsorption, resulting in hyperoxaluria and oxalate stone formation.[33]

Bariatric surgery Growing evidence in the literature shows a higher kidney stone rate among patients having undergone bariatric surgery. With increasing postoperative malabsorption, the likelihood of symptomatic stone events increases. Thus, gastric banding and sleeve gastrectomy have stone rates similar to obese controls, whereas the adjusted hazard ratio of a symptomatic stone event was 2.5 patients having undergone Roux-en-Y gastric bypass and 5.2 for the more malabsorptive procedures, such as very long limb Roux-en-Y gastric bypass and biliopancreatic diversion/duodenal switch. The most predominant stone type is calcium oxalate stones.[34]

Hyperparathyroidism Although surgical intervention (such as aforementioned gastric bypass) may lead to kidney stone formation, surgery can also cure the underlying pathology that leads to stone formation. This is exemplified with primary hyperparathyroidism.[35]

Although the reason for stone formation is somewhat uncertain, it is known that parathyroid hormone (PTH) stimulates formation of active 1,25 vitamin D, with stimulation of bone resorption to release calcium and gastrointestinal absorption of calcium, leading to hypercalcemia. Although PTH does increase tubular reabsorption of calcium, net renal calcium excretion increases in part because the filtered load of calcium increases. As a result, kidney stones develop in about 20% of patients with hyperparathyroidism.[36,37]

Parathyroidectomy of a hyperactive adenoma results in a significant decrease in stone formation. Although for the first few years it remains higher, at 10 years it is the same as in the general population.[35]

With this in mind, the International Workshop on the Management of Asymptomatic primary hyperparathyroidism recommends that imaging indicating calcium-containing stones in a primary hyperparathyroidism patient warrants parathyroidectomy.[38]

Similarly, in a stone patient with hypercalcemia, PTH levels should be checked.[7]

PATHOPHYSIOLOGY OF RENAL COLIC AND STONE PASSAGE

Obstruction of urinary flow leads to increased upstream intraluminal pressure. This pressure leads to hydronephrosis, causing stretch and stimulation of nerve endings in the urothelium, resulting in colicky pain. Additionally, the smooth muscle in the ureteral wall contracts in an attempt to expel the stone and can go into spasm.[39]

The afferent nerves of the kidney and ureter enter the spinal cord at the T11 to L1 levels, en route to the central nervous system. These pathways are not specific to the kidney and ureter but are shared with afferent nerves from the gastrointestinal organs, other urinary organs, and genitalia. As such, pain can be perceived by the patient as rising from these organs, making for a presentation of an acute abdomen.[39] Nausea and vomiting may be caused by the common innervation pathway of the renal pelvis, stomach, and intestines through the celiac axis and vagal nerve afferents.[39]

Hematuria, although not always present, is caused by the rough surface of the stone injuring superficial blood vessels in its path.[40]

CLINICAL PRESENTATION AND EXAMINATION

Symptoms

The most common presentations of kidney stone are pain, hematuria, nausea, vomiting, and urinary tract infection (**Box 2**).

The location and nature of the pain can change based on stone location, although there is not a reliable correlation between pain location and stone location. This finding is particularly true in the elderly.[41] Intermittent pain indicates incomplete or intermittent obstruction, whereas constant pain indicates complete obstruction.[42] As mentioned earlier, because of the shared innervation of the ureter with adjacent organs, the pain can be perceived as coming from intestine, groin, bladder, and internal or external genitalia. In particular, ipsilateral testicular, labial, or groin areas are common sites of referred pain from distal ureteral calculi. Although it is classically described that a patient with renal colic is writhing, unable to find a comfortable position, this is not universal finding.[39,41]

Because obstruction is the mechanism by which stones cause pain, nonobstructing stones are not believed to be able to cause pain. Thus, in a patient with acute abdomen or flank or back pain and an imaging finding of a nonobstructing stone or an intraparenchymal stone, an alternate cause for the pain needs to be sought.

Hematuria may be macroscopic but more often is microscopic. The absence of hematuria does not exclude stone. The accuracy of hematuria for predicting stone has been reported as only 62%.[40]

Urgency, frequency, dysuria, and pain at meatus are common findings, as the stone traverses the transluminal bladder wall, thus, irritating the bladder urothelium. The condition can easily be mistaken for cystitis. If a stone passes into the bladder, it often becomes asymptomatic. Because the urethra has a larger diameter then the ureter, the actual passage of stones through the urethra is often less symptomatic and may be fully asymptomatic.[43]

Nausea and vomiting are present in about half of acute cases, making distinction from a gastrointestinal etiology more challenging.[43]

Of particular concern is a kidney stone presentation associated with fevers, chills, and rigors, as upper tract urinary tract infection that is not draining because of an obstructive stone carries a high risk of systemic inflammatory response syndrome and severe sepsis development. In addition to cultures and antibiotics, urinary drainage, in the form of a ureteral stent or a nephrostomy tube, must be performed.[12,42]

Physical Examination

General

In classical descriptions, a patient with renal colic is writhing, unable to find a comfortable position. This is a common finding but not universal.

Vital signs

Renal colic can induce tachycardia and hypertension. Renal colic generally does not cause fevers unless associated with urinary tract infection.[42]

Abdominal and flank examination

Costovertebral angle percussion tenderness is frequently found and is often quite severe. Given the retroperitoneal location of the kidney and the ureter, the abdomen is usually soft, nontender, nondistended, and without signs of peritoneal irritations. Examination findings of the genitalia and groin are normal.[42]

Laboratory Work

Serum chemistry results are most often within normal range. Elevation of creatine can be seen in a solitary kidney or in patients with a baseline decrease in renal function. Creatine elevation may also result from dehydration as a result of colic-related nausea and emesis.[40]

An increase in neutrophils and white blood cells may be noted as stress response or if there is an associated urinary tract infection.[42]

On a urinalysis, microscopic hematuria is common.[40] Although urinary crystals may be noted, crystalluria is a common finding in normal controls and is not diagnostic of urolithiasis.[44] The presence of white cells, leukocyte esterase, and nitrites should raise the suspicion of an underlying infection.[4]

Imaging

Noncontrast computed tomography

Noncontrast, low-dose (or ultra–low-dose) computed tomography (CT) scans have become the gold standard for diagnosis of urolithiasis and has replaced intravenous pyelography (IVP) as such. This type of CT offers high sensitivity (>95%) and specificity (98%) for the detection of stones.[45] This type of CT offers more anatomic stone and renal detail, which is especially relevant when mapping out large branched stones or complex collecting system anatomy. In addition, it offers anatomic information of surrounding organs.[46]

Noncontrast CT avoids the intravenous contrast need for an IVP and allows for better quality imaging in the obese compared with ultrasound scan and in many institutions is often more accessible than ultrasonography.[46]

The main drawback of noncontrast CT is the amount of ionizing radiation, which is of particular concern in frequent stone formers and young patients. The average stone protocol CT scan in North America results in 11 mSv, but ultra–low-dose protocols are available with 1 mSv are available.[47,48]

Retroperitoneal ultrasound scan

Retroperitoneal ultrasound scan offers moderate sensitivity (45%) and specificity (88%–94%), albeit lower than CT scan.[49] Retroperitoneal ultrasound is less expensive than CT; however, it is operator dependent, and there is risk of over- and undercalling stone size.[47] Because there is no radiation, retroperitoneal ultrasound is a reasonable study for the nonacute setting, in follow-up, and in children and pregnant women. The European Association of Urology Guidelines recommends this method as the primary imaging modality, although a CT scan should be obtained for acute flank pain.[12]

Plain radiograph of abdomen and pelvis

Radiograph of the kidney, ureter, and bladder offers lower radiation dose than CT (0.6–1 mSv), with lower sensitivity and specificity. It is easily available and can be routinely use for follow-up if the primary stone is radio-opaque but should not be a primary study in acute circumstances.[12,50,51]

Intravenous pyelogram

Previously the gold standard for kidney stone detection, IVP has essentially been replaced by CT. Low-dose CT scans can now be performed with a similar or less amount of radiation without the need for intravenous contrast. CT can be done in a faster manner, as IVP often requires delayed imaging to allow contrast excretion into a partially obstructed collecting system. IVP still has a role in discerning ureteral stones from phleboliths.

Nuclear functional renal scan

Nuclear functional renal scans have a limited role in renal stone diagnosis and are occasionally used to confirm obstruction when there is doubt. The scans also have a role in assessing differential renal function, as a patient may be best served with observation or nephrectomy in the setting of a stone in a poorly functioning kidney as opposed to complex stone-removing procedures.[52]

MANAGEMENT AND AVAILABLE PROCEDURES

Observation

Small nonobstructing and intraparenchymal stones may potentially neither grow nor move down the ureter and, thus, not cause symptoms. By 3 years, 22% will grow significantly, 28% will cause colic, and another 2% will cause silent obstruction.[53] Observation, with serial imaging to assess for interval growth, is a reasonable alternative in these cases, at least in the short term, as the risk of complications of intervention may not outweigh the benefit.[12]

Medical Expulsive Therapy

Should a small stone (<10 mm) travel down the ureter and cause renal colic, it may pass spontaneously. Success rates are higher and passage time shorter the smaller the stone is and the further it is down the ureter at the time of presentation. Thus, a 1-mm stone has about an 87% chance of passage; a 2- to 4-mm stone, 76%; 5 to 7 mm, 60%; 8 to 9 mm, 48%; and 10 mm or larger, 25%. Similarly, a proximal stone has a 48% chance of passage; a midureteral stone, a 60% chance of passage; and a distal stone, a 75% to 79% chance of spontaneous passage.[54] The smaller and further distal the stone is, the shorter the time of passage, although this may vary from hours to 30 days.

The use of medication to affect ureteral passage rates is known as *medical expulsive therapy* (MET). Alpha blockers inhibit ureteral smooth muscle contraction and peristalsis with decrease basal tone. Calcium channel blockers inhibit calcium influx and prostaglandins, thus, decreasing contractions in the ureter.[55]

MET is found to increase successful passage of a ureteral stone by 44% to 66%.[55] It also decreases pain and number of colic episodes (**Table 1**).[56]

This therapy is a recommended treatment option for the informed patient, both by the American Urology Association and the European Association of Urology.[12,57] Should medical expulsive therapies fail, either because of intractable pain, prolonged course, or sequelae, such as infection or worsening renal function, then surgical intervention is indicated.[12]

Table 1
Medical expulsive therapy versus no medical expulsive therapy

	MET	Controls	Difference
Time of passage (d)	4–30	8–31	3.6[a]
Colic events	23%	40%	17%
Need for auxiliary procedures	12%	33%	11%

[a] weighted mean in meta-analysis.

Data from Fan B, Yang D, Wang J, et al. Can tamsulosin facilitate expulsion of ureteral stones? A meta-analysis of randomized controlled trials. Int J Urol 2013;20:818–30.

AVAILABLE SURGICAL PROCEDURES

- Temporizing drainage
- Shock wave lithotripsy

- Ureteroscopy
- Percutaneous nephrostomy
- Open or laparoscopic stone surgery

PROCEDURE TECHNIQUE

In certain acute settings, such as where a stone is believed to cause an obstructed urinary tract infection or acute deterioration in renal function, urgent decompression of the renal pelvis is needed. This decompression can be done either by placing a percutaneous nephrostomy tube or a ureteral stent cystoscopically.[12]

Shock Wave Lithotripsy

The basis of shock wave lithotripsy (SWL) is to fracture stones using focused shock waves into smaller fragments, which can then be passed spontaneously. Numerous versions of SWL devices (lithotripters) are available, with different means of generating the shock wave. These include electrohydraulic (spark gap), electromagnetic, and piezoelectric shock waves. The shock wave is generated inside the lithotripter and then it is focused to an external point with parabolic reflectors or acoustic lenses. The patient is positioned in such a way that the focal point is on the stone in question. To ensure correct position of the patient and stone, fluoroscopic or ultrasound guidance is used and the patient or lithotripter moved until the stone is in the focal point. Because sound and shock waves are best conducted in liquid, the treatment head of the lithotripter is pushed against the patient's body with water or aqueous gel in between as the medium.[58]

The primary benefit of SWL is that it does not require instrumentation of the patient's urinary tract or placement of a ureteral stent. Many patients poorly tolerate stent because of bladder spams and flank discomfort. However, SWL for many stone locations has a lower likelihood of rendering the patient stone free, as it may be difficult to verify that the stone was fractured into small enough pieces to pass down the ureter spontaneously. Therefore, the need for additional treatments is higher (**Table 2**).[12,57]

SWL does not have a high success rate for very large stones (2 cm or higher) or in certain hard stones, such as cysteine, and certain calcium phosphate stones.

SWL is contraindicated in the setting of obstruction distal to the stone, in patients on anticoagulation, in pregnant patients, and in patients with a known urinary tract infection. Complications include blockage of ureter from fragments (<4%), sepsis (1%), clinically significant bleeding (0.6%), and injury to gastrointestinal organs (1.8%). Long-term effects on hypertension, renal function, and diabetes have also been reported but remain debated.[59]

Table 2
Stone-free rates: SWL versus ureteroscopy from American Urology Association guidelines

Location	SWL (%)	URS (%)
Distal ureter	74	94
Mid ureter	73	86
Proximal ureter	82	81

Ureteroscopy

The basis of ureteroscopy is to advance a small diameter scope (most often 2–3 mm in diameter) in a retrograde manner up the urethra and bladder to the ureter and kidney

and fracture the stone(s) with laser energy via a laser fiber through the scope. The fragments can either be broken down into smaller fragments that can then be extracted with a wire basket or further fractured to submillimeter fragments (dusting) with the plan of having them pass spontaneously.[60]

This method can be achieved either with a semirigid scope in the distal ureter, allowing for better irritant flow and visibility, or a flexible ureteroscope in the more proximal ureter and kidney, allowing for complete inspection of the urinary collecting system.

Ureteroscopy offers superior stone-free rates to those of SWL in most clinical scenarios and, thus, fewer secondary procedures for residual stones.[12,57]

Because ureteroscopy may induce inflammation in the ureter initially after surgery, a ureteral stent is often needed to ensure drainage and prevent colic from temporary obstruction. As mentioned earlier, stent discomfort is common in the form of bladder spasm and flank pain.[60]

There are few contraindications to ureteroscopy other than untreated urinary tract infection. Complications other than stent discomfort are rare, but these included ureteral stricture (1%–2%), ureteral injury (3%–6%), urinary tract infection (2%–4%), and sepsis (2%).[57]

Percutaneous Nephrolithotomy

Both SWL and ureteroscopy are limited by the size of stones that can be treated successfully. In percutaneous nephrolithotomy (PCNL), a percutaneous access tract up to 1 cm in diameter is created through the kidney and allows access to the largest renal stones, which can be fractured accordingly and then removed.

Percutaneous nephrolithotomy allows for the insertion of larger-diameter rigid scopes directly into the renal pelvis. Through these, both suction and more potent energy sources, such as ultrasound and pneumatic lithotripsy, can be applied, greatly facilitating stone fracturing and clearance. In addition, larger flexible scopes can be introduced via the ureter, with improved visibility and working capacity.[61]

The access is obtained under fluoroscopic or ultrasound guidance, either preoperatively by an interventional radiologist or during surgery by the urologist. A posterior approach is typically used to avoid the anterior surrounding organ and to take advantage of the relatively less vascularized watershed area between the anterior and posterior branches of the renal artery.[62–64]

The primary appeal of PCNL is superior stone-free rates compared with SWL and ureteroscopy in large and complex stones (**Table 3**).[12]

Because it involves establishing and dilating a tract through the renal parenchyma, PCNL does carry the risk of bleeding, which may need transfusion (7%) or angioembolization (0.5%). PCNL is, therefore, contraindicated in patients on anticoagulation. Because of posterior access and the low-lying posterior aspect of the pleural sulcus, there is a risk of hydro-, hemo- and pneumothorax (1.5%). The risk is higher if the access is obtained between the ribs, which may be needed in certain circumstances.[12] Treatment of these conditions may require a chest tube as with other causes of these conditions.

Table 3
Ureteroscopy versus PCNL

	PCNL (%)	Ureteroscopy (%)
Stone free	95	84
Complication rate	16	13

Data from De S, Autorino R, Kim FJ, et al. Percutaneous nephrolithotomy versus retrograde intrarenal surgery: a systematic review and meta-analysis. Eur Urol 2015;67:125–37.

Table 4
Ureteral stone management based on American Urology Association guidelines

Stone Size	Primary Option	Secondary Option
<10 mm	Offer MET in a patient informed of the expected course, benefits, and risks, with scheduled follow-up and imaging, proceeding to intervention if MET fails	Either SWL or ureteroscopy
>10 mm	Either SWL or ureteroscopy	—

Data from Preminger GM, Tiselius HG, Assimos DG, et al; EAU/AUA Nephrolithiasis Guideline Panel. 2007 guideline for the management of ureteral calculi. J Urol 2007;178:2418–34.

Although much less frequent (cumulative average in reported series is 0.4%), injuries to surrounding organ, such as the retro-renal colon, duodenum, spleen, or liver are known complications.[12] Colonic injuries have been successfully managed by pulling the nephrostomy tube into the colonic lumen and leaving it to drain. At the same time, a retrograde ureteral stent is placed. This placement diverts the fecal and urinary streams and allows for successful outcomes. However, the need for colectomy and bowel diversion may be required based on the clinical scenario.[63] Successful conservative management of duodenal injury has been described with nasogastric tube decompression, fasting, and parental feeding, but explorative surgery remains the classical approach to any duodenal trauma.[64] In either setting, should the patient develop peritonitis, an explorative laparotomy is mandatory. Splenic injuries secondary to PCNL abide by the general trauma surgery principles and can be treated with observation, angioembolization, or laparotomy, based on the severity of the injury.[65] Liver injury during PCNL is rare and can most often be managed conservatively.[66]

Open or Laparoscopic Surgery for Stone Disease

Surgeries for stone disease include anatrophic nephrolithotomy and pyelolithotomy but are rarely used, given higher morbidity and complication rates, so are thus generally reserved for select cases. These techniques may be considered in rare cases in which SWL, ureteroscopy, and percutaneous nephrolithotomy fail or are unlikely to be successful.[12]

Table 5
Renal stone management based on the European Association of Urology guidelines

Stone Size/Location	Primary Option	Secondary Option
>20 mm	PCNL	Ureteroscopy or SWL
10–20 mm, located in lower pole of kidney	If unfavorable for shockwave: ureteroscopy or PCNL	—
	If favorable[a] for shockwave: ureteroscopy or PCNL	SWL
10–20 mm elsewhere in kidney	Ureteroscopy or SWL or PCNL	—
<10 mm	Ureteroscopy or SWL	PCNL

[a] None of the following: Shockwave-resistant stones, steep infundibular-pelvic angle, long lower pole calyx, or narrow infundibulum.

Data from Türk C, Petřík A, Sarica K, et al. EAU guidelines on interventional treatment for urolithiasis. Eur Urol 2016;69(3):475–82.

Box 1
Overview of diseases seen by the general surgeon that increase risk of kidney stones

Disease/Condition	Metabolic Abnormality	Resulting Stone Type
Colectomy/ileostomy Chronic diarrhea Laxative abuse	1. Dehydration 2. Loss of bicarbonate – renal retention of acid – increased urine pH	1. All types 2. Uric acid stones Ammonium acid urate
Fat malabsorption • Inflammatory bowel disease • Pancreatitis • Bariatric surgery	Saponification of calcium resulting in excess enteric oxalate. Increased per ability to oxalate	Calcium oxalate stones
Altered bowel flora	Loss of oxalate consuming bacteria	Calcium oxalate stone
Hyperparathyroidism	PTH-induced increase in enteric calcium absorption—compensatory hypercalciuria	Any calcium stones

SELECTION OF PROCEDURE

For many ureteral and renal stones, more than one option for surgical treatment may be available. Choices may vary with patient factors, patient preference, and urologist expertise. The American Urology Association and the European Association of Urology have published thorough reviews of outcomes and complications, which are offered in **Tables 1–5** and **Boxes 1–3** regarding the management of ureteral and renal stones.

Box 2
Signs and symptoms of renal colic

- Sudden onset—brought by sudden obstruction to outflow
- Intermittent versus constant
  - Intermittent suggestive of incomplete obstruction
  - Constant suggestive complete obstruction
  - If chronically obstructed may become painless
- Location of pain
  - Flank, lower abdomen, genitalia, groin
- Nausea vomiting
  - Present in about 50% of cases
- Hematuria
  - Present in 64% of cases
- Fever
  - Sign of a confined, nondraining upper tract infection. Must be surgically drained.
- Costovertebral angle tenderness to percussion
- Abdominal and genitalia examination findings often benign
- Laboratory values most often normal. Elevated creatinine and microscopic hematuria common.
- Significantly elevated white count should raise concern of nondraining urinary tract infection

Box 3
Comparison of treatment options

Procedure	Pro	Con
SWL	Avoids instrumentation of the patient Avoids the need of stent placement	Lower stone-free rates More often requires secondary procedures Patient must pass stone fragments, which can block the ureter
Ureteroscopy	Higher stone-free rates Can be performed on an anticoagulant end patient Can be performed on pregnant patients	Commonly requires stent placement Low risk of ureteral injuries
PCNL	Highest stone-free rates	Highest bleeding risk Low, but real risk of injury to adjacent organs
Open or laparoscopic surgery	May be of benefit in rare cases of complex stones in complex anatomy	Most invasive

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