Epidemiology and pathophysiology

Renal calculi are predominantly a western affliction with a prevalence of 1 in 1,000. They have a higher lifetime incidence in men with a male to female ratio of 3:1. A family history of renal stones increases the risk threefold. They generally present in the third to fifth decades of life. 12% of the population develops stones by the age of 70. They most commonly present with pain and haematuria but may also cause urgency, urinary tract infection or acute renal impairment. 2-3% of the population will experience renal colic in their lifetime. Of those with first time stones, 70% have a specific metabolic disorder.

Factors influencing the development of renal calculi include metabolic disorders that result in increased urine levels of calcium, oxalic acid, uric acid or citric acid. Genetic causes include familial renal tubular acidosis and mutations in the CLCN5 gene. An increase in renal calculi formation is also seen in those with renal structural abnormalities such as the presence of a horseshoe kidney or pelvic kidney.

Extrinsic factors include a warm climate promoting fluid loss and leading to low urine volume and increased solute concentration in the urine. Dehydration from any cause such as from Crohn’s disease or diarrhoea has a similar effect. Dietary causes include a large intake of dietary protein resulting in an increase in uric acid excretion. Excessive amounts of tea or fruit juice may elevate urinary oxalate level.

Infection, sedentary lifestyle and stress are other known contributing factors. Stasis of urine also permits precipitation of organic matter and minerals. The presence of any foreign body in the bladder serves as a nidus for infection and calculus formation.

Calculi may also be drug-induced such as indinavir calculi (a protease inhibitor used in the treatment of HIV), high disease of vitamin C, vitamin D or long-term use of antacids.

Pathophysiology and stone make-up

Salt crystals precipitate out of supersaturated urine and adhere to renal tubular epithelial cells. Randall’s plaques, which are soft tissue calcifications in the interstitium of the renal papilla, serve as a substrate for renal calculi to grow and protrude into the tubular lumen. Almost all ureteral and urinary bladder calculi are thought to originate in the renal medulla or intrarenal collecting system.

There are many different types of stones, 80% of which are calcium stones. Stones may be pure calcium oxalate, mixed calcium oxalate and phosphate or pure calcium phosphate. 10% of stones contain no calcium and are primarily composed of uric acid or cystine.

Approximately half of calcium stones form as a result of hypercalcemia with aetiologies including hyperparathyroidism, milk-alkali syndrome, hypervitamin D, paraneoplastic, Cushing’s disease and sarcoidosis. Calcium stone formation in those with normal serum calcium may result from obstruction, urinary tract infections, calyceal diverticulum, renal tubular acidosis, medullary sponge kidney and idiopathic hypercalciuria. Citrate is an inhibitor of calcium salt formation by forming a soluble complex with calcium and stone formation may also result from hypocitraturia.

Oxalate forms an insoluble complex with calcium resulting in precipitation of crystals. Oxalate stones may be congenital or acquired. The congenital form due to familial oxaluria is rare and affected patients are usually of Mediterranean extraction. Acquired forms of hyperoxaluria include hyperabsorption of oxalate as seen with inflammatory bowel disease, coeliac sprue, pancreatic insufficiency and small bowel bypass surgery.

10-15% of calculi are struvite stones. They are more common in women (3:1), those with a neurogenic bladder or urinary diversion such as ileal conduit. Infectious organisms such as proteus or klebsiella can break down urea to make ammonia, leading to formation of magnesium ammonium phosphate (struvite) stones. They tend to form staghorn calculi with the calculus seen to fill the expanded renal collecting system. These stones are difficult to eliminate with medical treatment because hard stone forms around a nucleus of bacteria, protecting them from antibiotic therapy. Given their large size and configuration, lithotripsy can be difficult to perform and these stones often require nephrectomy in order to eliminate the infectious stone cycle.

Uric acid calculi make up 5-10% of all renal calculi. These may result from hyperuricaemia from excessive dietary intake, gout, myeloproliferative disease or chemotherapy. If serum uric acid is normal, they may be idiopathic. These are radiolucent on plain radiographs but visible on CT.

Cystinuria is an autosomal recessive disorder that is characterised by renal collecting system stone formation. Cystine stones make up 1% of all renal calculi and are non-radiopaque.

Another autosomal recessive disorder that may lead to renal stone formation is xanthinuria. It is very rare and its exact incidence is unknown. Xanthine stones form early in childhood. These stones are also non-radiopaque.

Almost all stones are seen on CT with the exception of matrix stones and indinavir stones, both of which are of soft tissue attenuation. Matrix calculi are rare. These are composed of mucoprotein and mucopolysaccharide, and are soft and pliable. These stones are radiolucent, even on CT. 4-13% of those treated with indinavir, a protease inhibitor for HIV, develop nephrolithiasis. Calculi form because indinavir is poorly soluble and crystals act as nidi for stone formation. Withdrawal of indinavir may be necessary.

CT KUB

Calcium, oxalate and struvite stones are radiopaque on plain radiography, while cystine is semi-opaque. Uric acid, xanthine, matrix and indinavir stones are radiolucent. All stones apart from indinavir and matrix stones are seen on CT. All stones are echogenic on ultrasound.

CT has replaced excretion urography with sensitivity and
proximal hydronephrosis, hydroureter and perinephric or couteretic junction, confirming an intravesical location. Dislodge a stone that may be lying dependently at the vesicoureteric junction or has just passed. Performing non-crossed the iliac vessels. Occur at the ureteropelvic junction or where the ureter at or near the vesicoureteric junction. They also commonly in the pelvis is incompletely surrounded by soft tissue. Comet tail sign described in phleboliths where calcification is due to intravenous urogram, doesn’t require patient preparation and iodinated contrast is not used, allowing ease of use in those with renal impairment. In addition, the use of dual energy CT has enabled the determination of stone composition. Limitations of CT KUB (kidneys, ureters and bladder) include the lack of information provided about renal function, a failure to identify the more rare radiolucent stones, occasional difficulties in distinguishing hydronephrosis from an extrarenal pelvis, difficulties in differentiating calculi from vascular calcifications and a poor correlation between CT estimate of stone size and actual stone size following passage.

Newer, low dose non-contrast CT imaging techniques have been shown to detect the presence of calculi with a similar sensitivity and specificity to standard dose CT scans, but they may be less sensitive in the detection of small stones less than 2mm in diameter. Low dose CT technique of 110mAs results in a radiation dose of approximately 2.9mSv (almost equivalent to the dose received from IVP technique) and standard technique of 220mAs dose approximately 5.7mSv.

**Radiologic signs of nephrolithiasis**

In the pelvis it may be difficult to differentiate renal calculi from vascular calcification. The presence of a rim sign is useful in determining if calcification is due to a ureteral calculus or lies outside the ureter. The rim sign is the presence of soft tissue surrounding the calcification, due to a thickened oedematous ureteral wall. This contrasts with the comet tail sign described in phleboliths where calcification in the pelvis is incompletely surrounded by soft tissue. Obstructing ureteral calculi are most commonly located at or near the vesicoureteric junction. They also commonly occur at the ureteropelvic junction or where the ureter crosses the iliac vessels. It may sometimes be difficult to tell if a stone is at the vesicoureteric junction or has just passed. Performing non-enhanced helical CT in the prone position is of value to dislodge a stone that may be lying dependently at the vesicoureteric junction, confirming an intravesical location.

Secondary signs of the presence of renal calculi include proximal hydronephrosis, hydroureter and perinephric or periureteral fat stranding. An obstructed kidney may appear enlarged and of reduced attenuation on CT.

**Dual energy CT**

Dual energy CT looks at the ability of the stone to block a photon at two different energies, which is unique to its composition. By imaging at different energies, the nature of the stone can be inferred by its response. This is important in treatment, assessment of therapy response and in formulating strategies to prevent recurrence, as certain stones are more amenable to lithotripsy or alkalinisation of the urine, eg uric acid stones are more malleable and less likely to fracture with the use of lithotripsy but can be dissolved by alkalinising the urine. Struvite and lower density calculi may be more susceptible to extracorporeal shock wave lithotripsy (ESWL). The efficacy of ESWL decreases with higher stone attenuation values. Hard stones composed of calcium oxalate or cysteine may be fragmented using percutaneous lithotripsy.

**Conclusion**

A CT reporting template should include location, size and volume of the calcui as well as density, as this may determine stone composition. Larger stones, measuring >5mm in diameter, are less likely to spontaneously pass than stones <5mm. In cases where ESWL is being considered for treatment, information on distance of the stone from the adjacent skin surface is necessary to decide if shock waves will be successful in fragmenting the calculus. In addition, the radiologist should assess for the presence of secondary signs and any complications related to the presence of renal calculi, while eliminating any alternative diagnosis and evaluating for concomitant pathology.

**References**

Figure 3
Non-contrast axial CT demonstrating mild right sided hydronephrosis, perinephric fat stranding and reduction in attenuation of the right kidney secondary to more distal ureteric obstruction.

Figure 4
Dual energy CT: Example of stone analysis.