

An Imaging Review on Urinary Tract Calcifications

SUSHIL. G. KACHEWAR, DEVIDAS. S. KULKARNI

ABSTRACT

The urinary tract is one of the most important excretory systems in the human body. It consists of the kidneys, ureters, bladder and the urethra. The existence of calcium and other minerals in the excretory contents makes it prone to calcifications in the conditions of stasis of these contents, in a conducive biochemical environment.

The calcifications in the renal parenchyma - Nephrocalcinosis can be seen in a number of metabolic diseases which present with renal impairment or failure. The calcifications in the lumen of the dilated renal calices, pelvis, ureters, and the bladder or even in the urethra can be seen as stones which impair or obstruct the normal flow of urine. Urinary

tract calcifications are seen not only in benign conditions, as has been described above, but also in malignant conditions like Wilm's tumour in children or renal cell carcinoma in adults. Inflammatory conditions like Xanthogranulomatous Pyelonephritis can also have renal caliceal or pelvic calculi.

The mode of presentation of the patients with urinary tract calcifications is different in different entities. A correct and a timely diagnosis is therefore a must for treating this malady. It is the single largest contributor of the cases of acute abdomen globally. This imaging review focuses on the calcifications in the urinary tract and the roles of various imaging modalities in their diagnosis, treatment and follow up.

Key Words: Urinary System, Excretory System, Calculi, Calcifications, Benign Calcifications, Malignant Calcifications

INTRODUCTION

The human urinary tract acts as a perfect host to many calcification processes, the determinants of which are multi-factorial and hence are its health impacts. The most common form of a urinary tract calcification is a calculus. Depending upon its calcium content, it could be radio-opaque when it is made of calcium oxalate or it may be slightly radiolucent if it is composed of cystine. These calculi are located within the lumen of the excretory system.

The next type is where the renal cortex shows calcifications, as in nephrocalcinosis. This calcification may be focal or diffuse and it may even demonstrate a thin rim which outlines the cortex. The renal parenchymal calcifications may also be seen in renal neoplasms. These may either be central or peripheral. At times, the arcuate or the parenchymal vessels may also be calcified. The thin walls of the renal cysts may also get calcified, giving an eggshell appearance.

The ureteral calculi are usually longitudinally oriented along the long axis of the ureters. They are uniformly radiopaque. In the pelvis, their closest differential diagnosis is the presence of phleboliths which have a lucent centre. The urinary bladder calculi come in all shapes and sizes. Typically, they are multi-

laminated, multi faceted and sometimes, even spiculated. Schistosomiasis (Bilharziasis) is one infective aetiology which can cause mural calcifications in any portion of the urinary tract. Tuberculosis is another infective condition with a potential to cause widespread calcifications when it afflicts the urinary system. Usually, in the infective conditions, calcifications occur only when the causative agent is dead or entombed. But not so with tuberculosis, as it may also be associated with active tuberculosis. Primary amyloidosis is the closest differential diagnosis of a sub-mucosal calcification of the renal pelvis, the distal ureters and the bladder, in addition to bilharziasis and tuberculosis which have already been mentioned above.

With the widespread availability of good ultrasound machines in neonatal intensive care units (NICUs), renal calcifications have been found to be more common, especially in Extremely Low-Birth Weight (ELBW) infants. Although, these renal calcifications resolve spontaneously in most of the NICU patients; they may sometimes be associated with persistent abnormalities in the renal function if hypercalciuria continues and when they are associated with the inborn errors of metabolism, they can progress to a chronic renal injury, as are seen in cases with primary hyperoxaluria. The presence and the extent of

the urinary tract calcifications, irrespective of their aetiology, can be determined by the use of various imaging modalities like Plain Radiography, Computerized Tomography (CT scan), MRI Scan, Ultrasound scan and Intravenous Urography.

The health impacts of this entity are such that the patients may sometimes be totally asymptomatic, even in the presence of large calcifications and they may sometimes cry in pain when even a small calculus gets obstructed and causes colic. Those who do not respond to a medical management must be treated with surgery or they must be followed up intensely with the aim of retaining as much of the functional parenchyma as possible.

THE PATHO-PHYSIOLOGY OF THE URINARY TRACT CALCIFICATIONS

Urinary tract calculi are formed when the urine is supersaturated with salt and minerals such as calcium oxalate, struvite (ammonium magnesium phosphate), uric acid and cystine[1]. Studies have suggested that the initial factor which is involved in the formation of a stone may be the presence of nanobacteria that form a calcium phosphate shell. The other factor that leads to stone production is the formation of Randall's plaques. Calcium oxalate precipitates form in the basement membrane of the thin loops of Henle; these eventually accumulate in the sub epithelial space of the renal papillae, leading to the formation of a Randall's plaque and eventually a calculus. Epidemiologically [2] speaking, men are more commonly affected than women, with a male to female ratio of 3:1. The difference between the sexes is gradually being eroded. This is thought to be due to lifestyle-associated factors such as obesity and a western diet. The peak age for developing stones is between 30 and 50 years and a recurrence is common.

In the entire urinary tract; the region which is most commonly and significantly affected by calcifications are the kidneys. The processes of the calcifications in the kidneys, followed by the calcifications in the ureters and then the calcifications in the urinary bladder, will be discussed.

[I] Calcifications In The Kidney

1. Renal calculus Disease

Although renal calculus is the most common cause of a calcification within the kidney [3], other causes like nephrocalcinosis, renal cysts, renal masses and a host of miscellaneous conditions need a mention under this broad umbrella. Around 12% of the living population is expected to experience an episode of urinary tract calculi at some time or the other during their lifetime [4]. The situations of those who are afflicted are grim, as a recurrence is the rule in a majority of them, if not all.

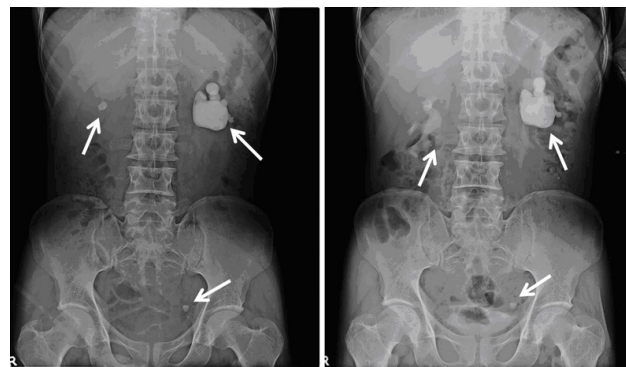
The different types of urinary tract calculi can be summarized

[4], based on their chemical compositions and their frequencies of occurrence, as has been shown in [Table/Fig-1].

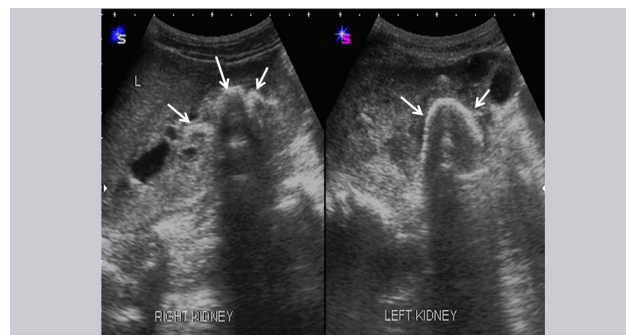
Sr No	Primary Composition	Occurrence
1	Calcium	70-80
1A	Calcium Oxalate-Phosphate	30-40
1B	Calcium Oxalate	20-30
1C	Calcium Phosphate	5-10
2	Struvite	15-20
3	Uric Acid	5-10
4	Cystine	1-3

[Table/Fig-1]: Chemical composition and percentage wise occurrence of calculi in urinary system

Any imbalance between the inhibitors and the promoters of crystal formations and the presence or the absence of hypercalcaemia and hypercalciuria, are important mechanisms which explain the formation of calculi in the urinary system. The stasis or stagnation of urine in the urinary tract due to any reason, hastens the crystallization process and it is hence associated with the development of urinary tract calculi. Locationwise, they may be seen in the calices and in the renal pelvis or when they are sufficiently large, they may also be seen in the lumen of both [Table/Fig-2 and 3] on plain radiographs or on ultrasound imaging.



[Table/Fig-2]: X-ray KUB and in IVU film showing right renal caliceal, left renal pelvic and left lower ureteric calculus



[Table/Fig-3]: Ultrasound image showing bilateral large renal caliceal calculi

Thus, the commonest urinary tract calculi are composed of calcium as the base, that combines with oxalate, phosphate, or both in different proportions and result in a single unit.

The calcium metabolism may thus get deranged, leading to the formation of calculi.

The following are the notable 5 metabolic derangements which lead to the genesis of calcium calculi [4] in the urinary system:

A] Hypercalciuria and hypercalcaemia may be seen in conditions like primary hyperparathyroidism, sarcoidosis, hypervitaminosis D, malignant neoplasms, Paget's disease the milk-alkali syndrome, immobilization, hyper- and hypothyroidism and adrenal insufficiency and in cases of renal transplantation.

B] Hypercalciuria without hypercalcaemia is seen in idiopathic hypercalciuria, renal tubular acidosis, acetazolamide therapy, sarcoidosis, malignant neoplasms, immobilization, Paget's disease, Cushing's syndrome and medullary sponge kidney.

C] Disordered oxalate metabolism may be seen as in primary hyperoxaluria, as well as in secondary or acquired hyperoxaluria.

D] Calcium oxalate stone formation which is associated with hyperuricosuria

E] Idiopathic conditions which lead to calcium calculi formation, mainly due to the absence or paucity of a crystallization inhibitor.

2. Renal Parenchymal Calcifications (Nephrocalcinosis)

In nephrocalcinosis, an abnormal deposition of calcium occurs in the renal parenchyma, either in the medulla (medullary nephrocalcinosis) or in the renal cortex (cortical nephrocalcinosis) [3, 5]. [Table/Fig-4] shows bilateral diffuse nephrocalcinosis. Uniform medullary nephrocalcinosis is most commonly seen with hyperparathyroidism or distal renal tubular acidosis (type 1) [1]. Asymmetric medullary nephrocalcinosis is commonly seen in a medullary sponge kidney. While cortical nephrocalcinosis is secondary to chronic glomerulonephritis or acute cortical necrosis ; it may also be seen in a transplanted kidney as a result of a chronic rejection.

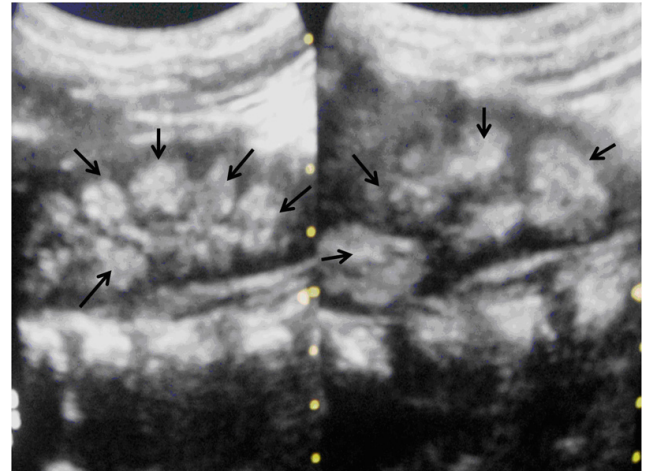
3. Calcifications in the Renal Cysts

The cystic lesions of the kidneys like isolated simple cysts, cysts of autosomal dominant polycystic kidney disease, hydatid cysts, etc may develop calcifications of their walls due to urinary stasis or local tissue reactions [6].

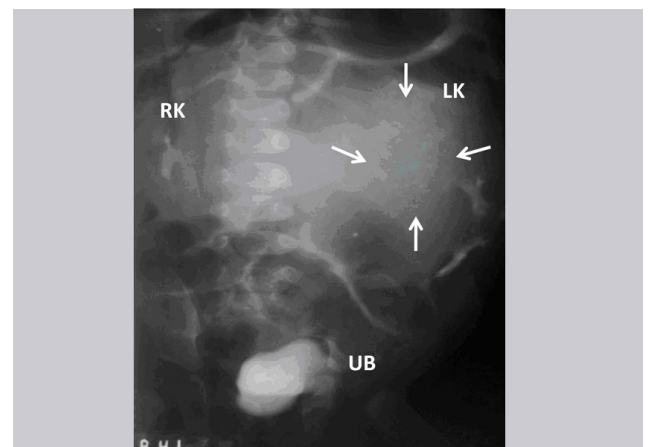
4. Solid Renal Masses

Nearly 20% of the renal cell carcinomas show calcifications [7]. Wilm's tumour which is classically seen in paediatric patients, might also show calcifications [Table/Fig-5]. When the calcification is in the centre of the mass, it is suggestive of

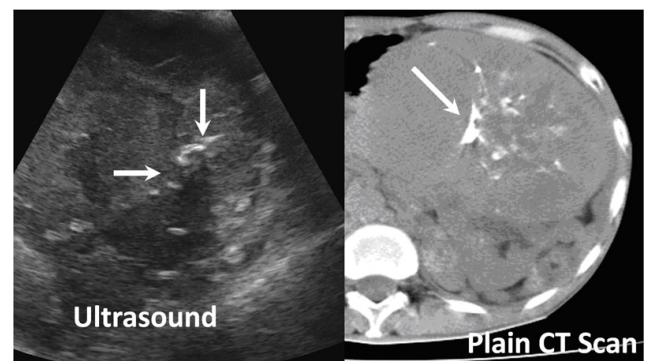
a malignancy in around 87% of the cases. Calcifications are also seen in renal cell carcinoma [Table/Fig-6]. However 20% of the masses with predominant or only peripheral calcifications are found to be malignant [7].



[Table/Fig-4]: Ultrasound image showing bilateral diffuse renal parenchymal calcifications in Nephrocalcinosis



[Table/Fig-5]: Intravenous Urography study showing enlarged left kidney due to a solid soft tissue mass showing central tiny calcifications-Wilm's tumor



[Table/Fig-6]: Ultrasound and CT scan image showing calcifications in the center of a left renal cell carcinoma

5. Calcifications in Renal Tuberculosis

In renal tuberculosis, the calcifications may be local or diffuse and they are referred to as the “putty” kidney [3]. 7.1%- 24% [8] of the patients with renal tuberculosis demonstrate calcifications in the urinary tract.

The exact cause of the calcifications in genitourinary tuberculosis is still not clear and there is no evidence to support the different pathogeneses of the tuberculous calcifications. Although many times, the precipitating factors that are known to be associated with calculus disease, such as a prolonged bed rest, a high calcium intake, recurrent urinary tract infections, obstructive uropathy and hypercalciuria are found, there is no common denominator [9].

The calcification process in the renal tissues is believed to be a retrogressive change in which there is deposition of the insoluble calcium salts, especially phosphate and carbonate, in the cells or the degenerative tissues. Thus, it is an excessive development in the process of chronic inflammation. This deposition of calcium is purely due to local changes and it is unrelated to the variations in the blood calcium levels [10].

It has also been suggested that the phosphate ions which are liberated by the disintegration of nucleoproteins and phospholipids in the necrosed tissues may attract calcium and lead to the formation of lime salts which contain carbonate and phosphate in the same proportions as bone [11].

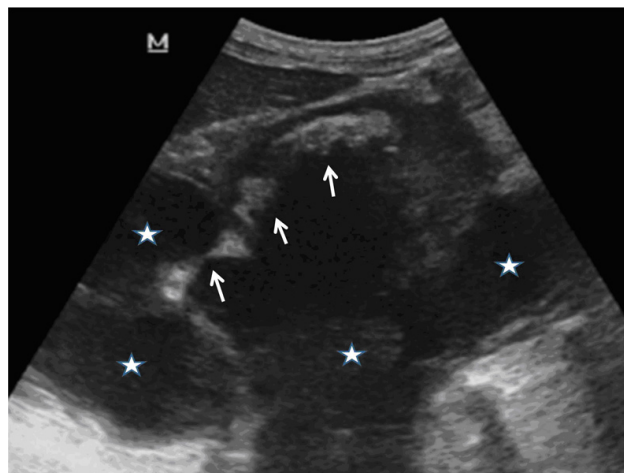
There seems to be a general agreement that a calcification is usually preceded by a hyaline stage. The damage to the cell membranes, which causes an increased permeability, leads to a disturbance in the intra-cellular cations and accumulation of calcium in the damaged cells. This in turn, impairs the oxidation, so that the cellular respiration is impaired and the cell dies.

Although a calcification is unusual in the early stages of the disease, nearly every end stage tuberculous kidney contains a calcification. The calcification may take a variety of patterns, which in most of the cases, is not specifically diagnostic of tuberculosis [12].

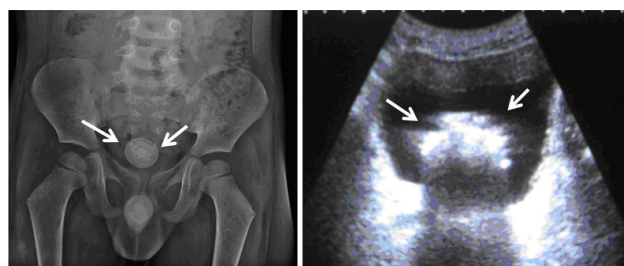
6. Miscellaneous Causes of the Renal Calcifications

Calcified renal vessels may be seen in Diabetes mellitus, hyperparathyroidism and in severe atherosclerotic disease [3]. A dystrophic calcification occurs in a subcapsular haematoma [13].

In Xanthogranulomatous Pyelonephritis [Table/Fig-7], the larger calculi become fragmented, which causes obstruction and leads to swelling and enlargement of the kidneys [14]. The renal abscesses may also get calcified. Sub-mucosal calcifications are also seen in the renal pelvis in primary amyloidosis and in bilharziasis; in addition to tuberculosis [15].



[Table/Fig-7]: Ultrasound image of Xanthogranulomatous Pyelonephritis -larger calculi become fragmented, that causes obstruction and leads to swelling and enlargement of the kidney



[Table/Fig-8]: Plain Radiograph and Ultrasound showing a large lamellated calculus in the bladder

[II] Calcifications In The-Ureter

The commonest cause of the calcifications in the ureter is the displaced renal calculi. Hence, chemically they are the same as the renal calculi [Table/Fig-2]. Usually, the sites of anatomic narrowing are the locations where the ureteral stones become impacted. A further accumulation of crystalline material on the ureteral stone may significantly enlarge its size [3]. A ureteral diverticulum or ureteroceles are the other favourable sites for ureteral calculi formation due to stasis [6]. The ureteral walls may sometimes become calcified in schistosomiasis or tuberculosis [6] and in an amyloid infiltration or a tumour of the ureteral wall [15].

[III] Calcifications In The Urinary Bladder

The bladder calculi [Table/Fig-9] usually consist of a mixture of calcium oxalate and calcium phosphate, which is similar to the calcifications in the kidneys. The calculi which are in the lumen may be seen in bladder outlet obstructions as well as in the vesical diverticuli [16].

The walls of the bladder show calcifications in the patients with transitional cell carcinoma, urachal carcinoma, schistosomiasis, alkaline incrustrated cystitis, cytotoxic cystitis, radia-

tion therapy, and tuberculosis [16]. The foreign bodies which are within the bladder or elsewhere within the urinary tract may calcify in response to the urine exposure [16].

THE DETERMINANTS OF THE URINARY TRACT CALCIFICATIONS

The multiple risk factors that promote the potential of a susceptible individual to develop urinary calcifications are:

1. Anatomical anomalies in the kidneys and/or in the urinary tract e.g. a horseshoe kidney, ureteral strictures.
2. A family history of renal stones.
3. Hypertension.
4. Gout.
5. Hyperparathyroidism.
6. Immobilization.
7. Relative dehydration.
8. Metabolic disorders which increase the excretion of solutes, e.g. chronic metabolic acidosis, hypercalciuria, hyperuricosuria.
9. A deficiency of citrate in the urine.
10. Cystinuria (an autosomal-recessive aminoaciduria).
11. Drugs, especially thiazide diuretics and calcium/vitamin D supplements.
12. More common occurrence in hot climates.
13. An increased risk of stones in the higher socio-economic groups.
14. Being associated with the melamine-contaminated infant milk formula [17].

The renal calculi which are formed in the sufficiently larger structures (renal cysts or hydronephrotic areas) are free to assume any shape. However, the calculi which have only smaller cavities for formation and growth, like the caliceal diverticula, are small and of similar sizes, owing to the lapidary effect [3]. A striking pattern which is called "milk of calcium", is most commonly seen in a structure that communicates directly with the collecting system, such as a caliceal diverticulum, or in an obstructed portion of the collecting system [3].

In the patients who suffer from recurrent urinary tract infections in alkaline urine, calculi which are composed of calcium which is mixed with magnesium, ammonium, and phosphate are formed; they have the shape of a stag's antlers and are therefore referred to as 'Staghorn calculi' [3]. The Staghorn calculi are often less opaque than the pure calcium stones.

Most of the urinary tract calculi are radiopaque as a result of

their calcium content. The calcium oxalate stones may occur in either a pure monohydrate or a dehydrate form. Like the pure calcium phosphate stones, the pure calcium oxalate monohydrate stones tend to be small and they are highly radiopaque for their size. The calcium oxalate dihydrate stones may be spiculated [3].

The cystine stones may have small sizes or they may be like the staghorn calcifications [3]. They are less radiopaque than the calcium stones because of their higher sulphur content, that produces the opacity. The cystine stones may grow quite rapidly.

Nephrocalcinosis

In asymmetric medullary nephrocalcinosis which is commonly seen in medullary sponge kidneys, the calcifications develop within the papillary tips in the dilated collecting ducts. In cortical nephrocalcinosis, the renal cortex may be outlined by a thin rim of calcifications, thus producing a "tramline" appearance. Spotty calcifications which are due to a preferential calcium deposition in the necrotic glomeruli, are seen in chronic glomerulonephritis.

Calcifications In Renal Cysts

The calcifications which are within a cyst wall have an "egg-shell" appearance 6 as they are usually thin and peripheral [16]. The calcified hydatid cysts too are calcified in their walls.

Calcifications In Renal Tuberculosis

These can be either homogenous or heterogenous. The classic radiological features in tuberculosis are described as smudgy, with indistinct borders and as speckled, when they are sharply defined with calcific densities of varying sizes. The calcifications which are at the boundary, between the necrotic and the viable tissues, appear curvilinear [12]. In putty kidney, a dense calcification replaces the totally destroyed caseated kidney and it is usually associated with an obstruction. Xanthogranulomatous Pyelonephritis may also be associated with renal tuberculosis and it shows an enlarged kidney which is filled with pus and fragmented calculi.

Calcifications In The Ureters

Morphologically, they are the same as the calcifications in the kidney. Those in the lumen are usually elongated and they have the same longitudinal axis as that of the ureteral axis.

Following an extra-corporeal shock wave lithotripsy of the large-volume renal or ureteral stones, the accumulated stone fragments may be identified radiographically as they pass through the ureter. This collection of stone fragments is referred to as a "steinstrasse" (stone street) [18].

The commonest differential diagnosis of the ureteral calculi in the pelvis is a phlebolith. The ureteral calculi are usually single

or few and they have a uniform radiopacity. They are often angulated along the course of the distal ureter. The phleboliths tend to be multiple and they are often lucent, centrally. The calcifications which are within the ureteral wall, give a tram track like appearance.

Calcifications In The Urinary Bladder

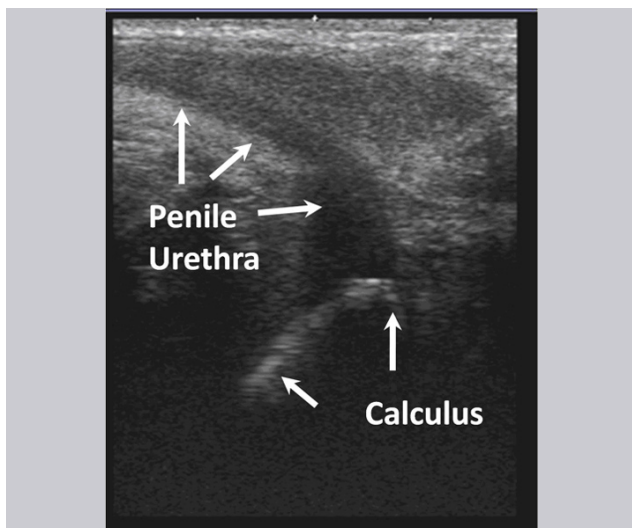
The vesicle calculi can be laminated, faceted, spiculated, or seed like in appearance [16]. A dumbbell stone is an unusual bladder stone that initially forms in the diverticulum and extends through the communication with the bladder into the bladder lumen [16].

A urachal carcinoma is also commonly associated with a tumour calcification, which typically occurs at the dome of the bladder and it may be identified on plain radiographs [19].

The enlarged, calcified prostate gland may protrude into the bladder and give a false appearance of a vesicle calculus.

The foreign bodies which are within the bladder or elsewhere within the urinary tract may calcify in response to a urine exposure [16] and they have characteristic appearances as per their original shapes.

The calcifications in the urethra are usually of a renal or a prostatic origin and they migrate into the lumen. When they are impacted inside [Table/Fig-9], there is dilatation of the urethra, prior to the sight of an obstruction.



[Table/Fig-9]: Ultrasound showing stone impacted in distal penile urethra.

THE HEALTH IMPACTS OF THE URINARY TRACT CALCIFICATIONS

The spectrum of the health impacts of the urinary tract calcifications is very broad. On one end of the spectrum, lies a perfectly asymptomatic individual, although he may be har-

bouring calcifications in the urinary tract. These might be in the lumen or in the walls of the tract. Somewhere in between, is an individual with excruciating pain, following an impaction of the calculus in the lumen of the tract. At the farther end of the spectrum, is an individual with a totally calcified tract, which has become non-functional as well and it is referred to as an end stage kidney.

The clinical findings thus usually depend on the site and the size of the urinary tract calcification. The patients may be asymptomatic or they may have pain, dysuria, frequency, hesitation and gross haematuria, or even a sudden cessation of voiding, which is accompanied by pain in the perineum.

The clinical presentation

- Many stones are asymptomatic and they are discovered during the investigations which are done for other conditions.
 - The classical feature of renal colic (or ureteric colic) is a sudden, severe pain. It is usually caused by the stones in the kidney, the renal pelvis or the ureter, which causes dilatation, stretching and spasm of the ureter. In most of the cases, no cause is found.
1. The pain starts in the loin at about the level of the costovertebral angle (but sometimes lower) and moves to the groin, with tenderness of the loin or the renal angle, sometimes with haematuria.
 2. If the stone is high and if it distends the renal capsule, then it will be in the flank, but as it moves down, the pain will move anteriorly and down towards the groin.
 3. A stone that is moving, is often more painful than a stone that is static.
 4. The pain radiates down to the testis, scrotum, labia or the anterior thigh.
 5. Whereas the pain of the biliary or the intestinal colic is intermittent, the pain of the renal colic is more constant, but there are often periods of relief or just a dull ache before it returns. The pain may change as the stone moves. The patient is often able to point to the place of the maximal pain and this has a good correlation with the current site of the stone.

Other symptoms which may be present, include: rigors and fever, dysuria, haematuria, urinary retention, nausea and vomiting.

Imaging studies are necessary for their confirmation. The initial imaging study of choice is a KUB (kidneys, ureters and bladder) radiograph which is obtained by using conventional radiography. Although it demonstrates almost all the calculi, the uric acid and the ammonium urate stones are non-opaque.

Hence comes the role of ultrasound, which in addition to confirming the existence of the calculus, gives information about its shape and size and on whether the proximal obstruction was caused by it or not. Serial follow ups during the medical treatment and post the surgical follow up for an adequate removal, can also be done.

Intravenous Urography demonstrates the excretory function of the kidney and the extent of the physiological obstruction which is caused by the calcifications in the lumen of the urinary tract. An unenhanced spiral CT scan is highly sensitive in diagnosing the calculi along the urinary tract and it has largely replaced intravenous pyelography and cystography. Cystoscopy is the most common test which is used to confirm the presence of the bladder calculi.

The treatment of the small calculi is done usually by medications. A symptomatic treatment is advised in cases of pain. An intervention may be indicated when there is acute urinary retention, gross haematuria, recurrent infections or obstructive uropathy and in cases with an unsuccessful medical treatment. Most of the bladder stones are removed via cystoscopy, which includes the use of lithotripsy to fragment the stones. The larger stones may require a suprapubic surgery.

THE INVESTIGATIONS, AS SUGGESTED BY THE EUROPEAN ASSOCIATION OF UROLOGY, INCLUDE [19]

- **The basic analysis should include:**
 1. Stick testing of the urine for red cells (which is suggestive of urolithiasis), white cells, nitrites (both are suggestive of infections) and pH. A pH of above 7 suggests urea-splitting organisms such as *Proteus* spp., whilst a pH of below 5 suggests uric acid stones.
 2. A mid-stream specimen of urine for microscopy (pyuria suggests infection), culture and sensitivities.
 3. Blood for FBC, CRP, renal function, electrolytes, calcium, phosphate, urate and creatinine.
 4. The prothrombin time and the international normalized ratio if an intervention is planned.
- A non-enhanced CT scanning is now the imaging modality of choice and it has replaced an Intravenous Pyelogram (IVP). An ultrasound scanning may be helpful in differentiating the radio-opaque from the radiolucent stones and in detecting the evidence of an obstruction.
- Plain X-rays of the kidney, ureter and the bladder (KUB) are useful in watching the passage of the radio-opaque stones (around 75% of the stones are of calcium and so will be radio-opaque).
- The European Association of Urology's guidelines on uro-

lithiasis recommend stone analysis for:

1. All the first-time stone formers.
2. All the patients with recurrent stones who are on a pharmacological preventing therapy.
3. The patients who have had an early recurrence after a complete stone clearance.
4. A late recurrence after a long stone-free period (the stone composition may change).

The patient must be encouraged to try to catch the stone for analysis. This may mean urinating through a tea strainer and a filter paper such as a coffee filter or a gauze.

MANAGEMENT [19]

The initial management can either be done on an inpatient or on an urgent outpatient basis, and it will usually depend on how easily the pain can be controlled.

- **The indications for hospital admissions:**

1. Fever.
2. Solitary kidney.
3. A known non-functioning kidney.
4. Inadequate pain relief or a persistent pain.
5. Inability to take adequate fluids due to nausea and vomiting.
6. Anuria.
7. Pregnancy.
8. Poor social support.
9. Inability to arrange an urgent outpatients department follow-up.
10. The people who are over the age of 60 years should be admitted if there are concerns on the clinical conditions or diagnostic certainties (a leaking aortic aneurysm may present with identical symptoms).

- **The indications for an urgent outpatients appointment:**

1. The pain has been relieved.
2. The patient is able to drink large volumes of fluid.
3. Adequate social circumstances.
4. No complications are evident.

THE INITIAL MANAGEMENT OF AN ACUTE PRESENTATION

- Non-steroidal anti-inflammatory drugs (NSAIDs), usually in the form of diclofenac IM or PR, should be offered as a first-line treatment for the relief of the severe pain of renal

colic. NSAIDs are more effective than opioids for this indication and they have fewer tendencies to cause nausea. A Cochrane review concluded that if opioids are needed, the choice should not be pethidine [20].

- Anti-emetics and a rehydration therapy should be provided if they are needed.
- A majority of the stones will pass spontaneously but they may take 1-3 weeks; the patients who have not passed a stone or who have continuing symptoms should have the progress of the stone monitored at a minimum of weekly intervals, to assess the progression of the stone.
- The conservative management may be continued for up to three weeks unless the patient is unable to manage the pain, or if he or she develops signs of an infection or obstruction.
- A medical expulsive therapy may be used to facilitate the passage of the stone. It is useful in the cases where there is no obvious reason for an immediate surgical removal. Calcium-channel blockers (e.g. nifedipine) or alpha-blockers (e.g. tamsulosin) are given. A corticosteroid such as prednisolone should be occasionally added when an alpha-blocker is used, but it should not be given as a monotherapy [21].

Managing the patients at home

- All the patients who are managed at home should drink a lot of fluids and, if possible, void the urine into a container or through a tea strainer or gauze, to catch any identifiable calculus.
- Analgesia: Paracetamol is safe and effective for mild-to-moderate pain; codeine can be added if more pain relief is required. Paracetamol and codeine should be prescribed separately so that they can be individually titrated.
- The patients who are managed at home should be offered a fast-track investigation which is initiated by the hospital on the receipt of a faxed letter which is compiled by the general practitioner.
- The patients should ideally receive an appointment for radiology within seven days of the onset of the symptoms.
- An urgent urology outpatient appointment should be arranged for, within one week, if the renal imaging shows a problem which requires intervention.

Surgical Treatment [2]

- Approximately 1 in 5 stones will not pass spontaneously and they will require some form of intervention.
- If the ureter is blocked or if it could potentially become blocked, e.g. when a larger stone will fragment, following other forms of therapy, a JJ stent is usually inserted

by using a cystoscope. It is a thin hollow tube with both ends coiled (pigtail). It is also used as a temporary holding measure, as it prevents the ureter from contracting and it thus reduces the pain, buying time until a more definitive measure can be undertaken.

The procedures for removing the stones include:

1. Extracorporeal Shock Wave Lithotripsy (ESWL): Shock waves are directed over the stone to break it apart. The stone particles will then pass spontaneously.
 2. Percutaneous Nephrolithotomy (PCNL): It is used for the larger stones (>2 cm), staghorn calculi and also for the cystine stones. The stones are removed at the time of the procedure by using a nephroscope.
 3. Ureteroscopy: This involves the use of laser to break up the stone and it has an excellent success rate in experienced hands.
 4. Open surgery: It is rarely necessary and it is usually reserved for the complicated cases or for those in whom all of the above have failed, e.g. multiple stones.
- The aim of the management of the calcification in renal tuberculosis should be to retain as much functioning renal tissue as possible. The small lesions can be kept under re-view on an annual basis and managed conservatively, provided there is no increase in their sizes. Most of the small calcified lesions remain unchanged for more than 20 years [9].
 - Removal of the non-functioning kidneys with extensive calcification and excision of the larger areas of calcification is recommended [9]. A partial nephrectomy is indicated if there is a localized polar lesion which contains a calcification that has failed to respond to 6 weeks of chemotherapy or an area of calcification which slowly increases in size.

Complications [2]

- The complete blockage of the urinary flow from a kidney decreases the Glomerular Filtration Rate (GFR) and, if it persists for more than 48 hours, it may cause irreversible renal damage.
- If the ureteric stones cause symptoms after four weeks, there is a 20% risk of complications, which include deterioration of the renal function, sepsis and ureteric strictures.
- The infections can be life-threatening.
- A persisting obstruction predisposes to pyelonephritis.
- Prognosis [2]
- Most of the symptomatic renal stones are small (less than 5 mm in diameter) and they pass spontaneously.

- The stones which are less than 5 mm in diameter pass spontaneously in up to 80% of the people.
- The stones which are between 5 mm and 10 mm in diameter pass spontaneously in about 50% of the people.
- The stones which are larger than 1 cm in diameter usually require an intervention (an urgent intervention is required if a complete obstruction or an infection is present).
- Two-thirds of the stones that pass spontaneously will do so within four weeks of the onset of the symptoms.
- A stone that has not passed within 1-2 months is unlikely to pass spontaneously.

The following features predispose to recurrent stone formation:

1. The first attack before 25 years of age.
2. A single functioning kidney.
3. A disease that predisposes to stone formation.
4. Abnormalities of the renal tract.

Prevention [2]

The recurrence of renal stones is common and therefore, the patients who have had a renal stone should be advised to adapt several lifestyle measures which will help in preventing or delaying a recurrence:

- Increase the fluid intake to maintain the urine output at 2-3 litres per day.
- Reduce the salt intake.
- Reduce the amount of meat and animal protein which is eaten.
- Reduce the oxalate intake (the foods which are rich in oxalate include chocolate, rhubarb and nuts) and the urate-rich foods (e.g. offal and certain fish).
- Regularly drink cranberry juice: this increases the citrate excretion and it reduces the oxalate and phosphate excretions.
- Maintain the calcium intake at normal levels (lowering the intake increases the excretion of calcium oxalate).
- Depending on the composition of the stone, a medication to prevent further stone formation is sometimes given, e.g. thiazide diuretics (for the calcium stones), allopurinol (for the uric acid stones) and calcium citrate (for the oxalate stones).

Thus, the health impacts of the urinary tract calcifications thus affect the physical, mental, emotional and the economical well being of an individual as well as his/her family.

REFERENCES

- [1] Worcester EM, Coe FL. Clinical practice. Calcium kidney stones. *N Engl J Med.* 2010; 363(10):954-63.
- [2] Macneil F, Bariol S; Urinary stone disease - assessment and management. *Aust Fam Physician.* 2011; 40(10):772-75.
- [3] Baker SR, Elkin M. Plain film findings in the urinary tract. In: Baker SR, ed. *The abdominal plain film.* Norwalk, Conn: Appleton & Lange., 1990; 327-78.
- [4] van Arsdalen KN. Pathogenesis of renal calculi. *Urol Radiol.* 1984; 6:65-73.
- [5] Lalli AF. Renal parenchyma calcifications. *Semin Roentgenol.* 1982; 17:101-12.
- [6] Daniel WW Jr, Hartman GW, Witten DM, Farrow GM, Kelalis PP. Calcified renal masses: a review of ten years experience at the Mayo Clinic. *Radiology.* 1972; 103:503-08.
- [7] Babaian RJ, Lucey DT, Fried FA. Significance and evaluation of calcifications associated with renal masses. *Urology.* 1978; 12:108-11.
- [8] Crenshaw JL. Renal tuberculosis with calcification. *J Urol.* 1930; 23: 515.
- [9] Won- SH, Lau WY. The surgical management of non-functioning tuberculosis kidneys. *Journal of Urology.* 1980; 124: 187-91.
- [10] Gow JG. Genitourinary tuberculosis. In: Campbell's Urology, 7th ed. Philadelphia, Saunders. 1998; 807-36.
- [11] Cosbie Ross J. Calcification in genitourinary tuberculosis. *Br J Urol.* 1970; 42: 656-60.
- [12] Cappel DF. Disturbances of nutrition (calcification). In: Muir's text-book of pathology, 8th ed. *ELBS and Edward Arnold publishers.* 1964; 116-21.
- [13] Page AC, Hammonds JC, Wells IP. Early calcification of haematoma in the renal pelvis. *Br J Urol.* 1989; 63:98-99.
- [14] Goldman SM, Hartman DS, Fishman EK, Finizio JP, Gatwood OM, Siegelman 55. CT of Xanthogranulomatous pyelonephritis: radiologic-pathologic correlation. *AJR.* 1984; 142:963-69.
- [15] Thomas SD, Sanders PW III, Pollack H. Primary amyloidosis of urinary bladder and ureter: causes of mural calcification. *Urology.* 1977; 9:586-89.
- [16] Banner MP, Pollack HM. Urolithiasis in the lower urinary tract. *Semin Roentgenol.* 1982; 17:140-48.
- [17] Garcia Lopez FJ, Quereda C; Melamine toxicity: one more culprit in calcium kidney lithiasis. *Kidney Int.* 2011; 80(7):694-96.
- [18] Dyer RB, Zagoria RJ, Auringer ST, et al. Radiologic contribution to the management of patients undergoing extracorporeal shock wave lithotripsy. *CRC Crit Rev Diagn Imaging.* 1988; 28:295-330.
- [19] Guidelines on Urolithiasis, *European Association of Urology.* (2011).
- [20] Holdgate A, Pollock T; Non-steroidal anti-inflammatory drugs (NSAIDs) versus opioids for acute renal colic. *Cochrane Database Syst Rev.* 2005; (2):CD004137.
- [21] Seitz C, Liatsikos E, Porpiglia F, et al. Medical therapy to facilitate the passage of stones: what is the evidence? *Eur Urol.* 2009; 56(3):455-71.

AUTHOR(S):

1. Dr. Sushil. G. Kachewar
2. Dr. Devidas. S. Kulkarni

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Rural Medical College (RMC), PIMS, Loni, India.
2. Professor, Rural Medical College (RMC), PIMS, Loni, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sushil Kachewar,
MD, DNB (Radio-diagnosis), Associate Professor, Rural Medical College, PIMS, Loni, India.
Email: sushilkachewar@hotmail.com
Ph: 9921160357

FINANCIAL OR OTHER COMPETING INTERESTS:

None.

Date of Submission: **May 05, 2012**

Date of Peer Review: **Oct 28, 2012**

Date of Acceptance: **Nov 01, 2012**

Date of Publishing: **Apr 01, 2013**