Nephrolithiasis

Assistant lecturer :

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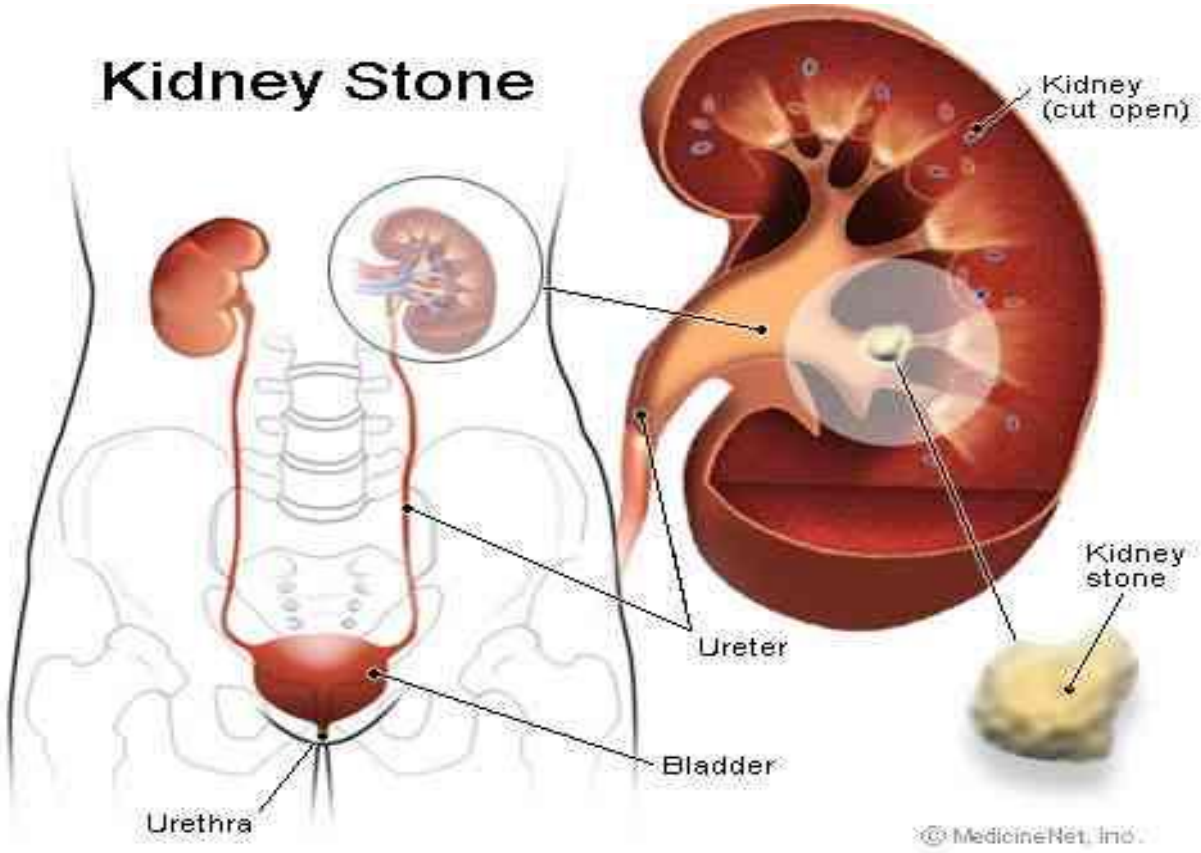
Urinary stones belong to the group of biochemical different inorganic and organic substances with a crystalline or amorphous structure are the major constituents of the stones.

- Urinary stones occur in all parts of the renal collecting system. The peak incidence of urinary calculi is in the third to fifth decades. Stones are more prevalent in men than in women and incidence is increased during the late summer months.


Kidney Stone
Kidney
(cut open)
Kidney
stone
Ureter
Bladder
Urethra
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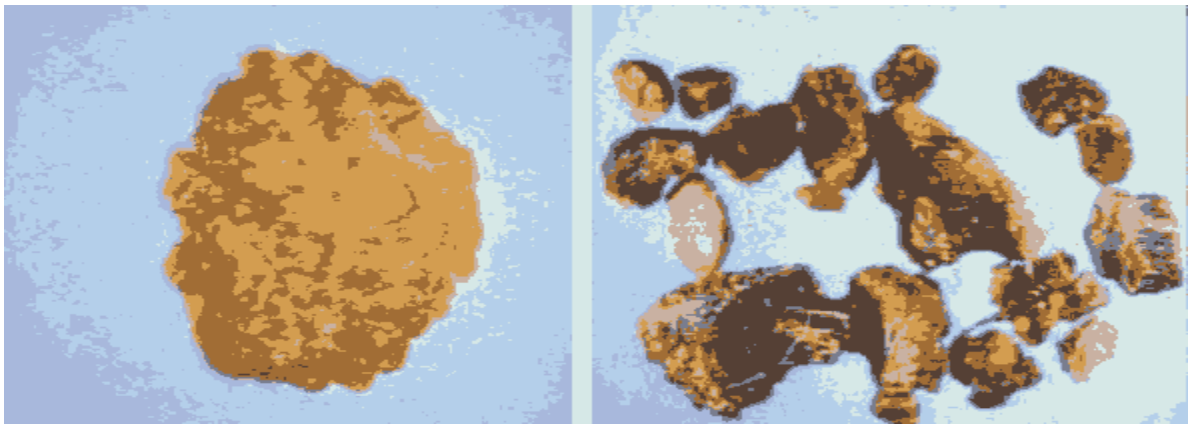
Types of renal stones

- ***Calcium stones:*** are the most common (60%). These stones are composed of mixtures of calcium oxalate (CaOx) and calcium phosphate (CaP), only CaOx or rarely only CaP and higher rates of recurrence in stones with CaP occur. Approximately 50% of people who form a single calcium stone eventually form another within the next 10 years. Calcium stone disease is frequently familial.

Calcium stones

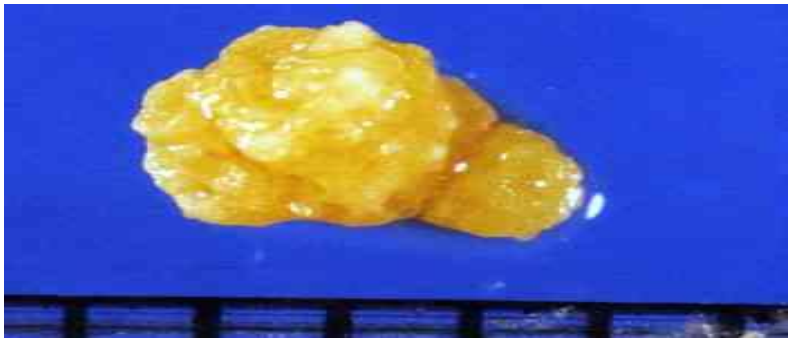
- ***Uric acid stones:*** are radiolucent and are also more common in men. Half of patients with uric acid stones have gout; uric acid lithiasis is usually familial whether or not gout is present.



- ***Struvite stones****:* are composed of a mixture of calcium, ammonium, and magnesium phosphate (triple phosphate stone) are common and potentially dangerous. These stones occur mainly in women or patients who require chronic bladder catheterization and result from urinary tract infection (UTI) with urease-producing bacteria, usually *Proteus* species..

- The stones can grow to a large size and fill the renal pelvis and calyces to produce a “staghorn” appearance. They are radiopaque and have a variable internal density.

- ***Cysteine stones:*** are uncommon; their radioopacity is due to the sulfur content. They appear in the urine as flat, hexagonal plates.



Clinical features

- Pain is the presenting feature of the greatest majority of kidney stones, but if the calculus is embedded within the solid substance of the kidney it may be entirely symptom free. Impaction of the stone at the peliv-ureteric junction, or migration down the ureter itself, produces the dreadful agony of uretric colic;

the pain radiates from loin to groin, is of great severity and is accompanied by typical restlessness of the patient associated with nausea and vomiting, who is quite unable to lie still in bed.

- Haematuria, which may be microscopic or macroscopic, is frequently present.

Clinical investigations:

- *1. Urine:* is tested for the presence of blood.
- *2. Plain abdominal X-ray:* specifically looking at kidneys, ureters and bladder will show the presence of stone in 90% of cases.
- *3. CT scan*: is the investigation of choice to confirm the diagnosis of renal colic since it is rapid, is more sensitive at detecting a stone, and can diagnose alternative pathologies if present (e.g. torted ovarian cyst, ruptured aortic aneurysom).

Investigation of the underlying cause:

- *1. Urine microscopy and culture:* the urine is cultured for bacteria and examined microscopically for the presence of cystine crystals.
- 2. Analysis of the stone, whether passed spontaneously or removed surgically, should be performed.
- *3. Uric acid estimation:* the serum uric acid is raised in gout with its associated uric acid stones.
- *4. Serum calcium:* hypercalcaemia (a value above 2.75mmol/L) is associated suspicious of the presence of a parathyroid tumor, although the incidence of stones due to this cause is low.

Complications:

- 1. *Hydronephrosis*: is a dilatation of the renal pelvis and calyces.
- 2. *Infection :* pyelonephritis, pyonephrosis.
- 3. *Urinary retention:* due to either impaction of calculi in the ureter on each side, or blockage of the ureter in a remaining solitary kidney.

Therapeutic strategies

1. Calicum stones **:**

- ***A. Idiopathic Hypercalciuria*****:** This condition appears to be hereditary. In some patients, primary intestinal hyperabsorption of calcium causes transient postprandial hypercalcemia that suppresses secretion of parathyroid hormone leads to urinary losses of calcium and renal synthesis of 1,25-dihydroxyvitamin D is increased, enhancing intestinal absorption of calcium.

…Vitamin D overactivity, either through high calcitriol levels or excess vitamin D receptor, is a likely explanation for the hypercalciuria in many of these patients. Hypercalciuria contributes to stone formation by raising urine saturation with respect to calcium oxalate and calcium phosphate.

Treatment

- For many years the standard therapy for hypercalciuria was dietary calcium restriction. However, recent studies have shown that low-calcium diets increase the risk of incident stone formation and also likely contribute to the low bone mineral density with increased risk of fracture. Low-sodium and low-protein diets are a superior option in stone formers.

- Thiazide diuretics may be used to lower urine calcium and are effective in preventing the formation of stones.
- Thiazide-induced hypokalemia should be aggressively treated since hypokalemia will reduce urine citrate, increasing urine calcium ion levels.

- ***B. Hyperuricosuria:*** About 20% of calcium oxalate stone formers are hyperuricosuric, primarily because of an excessive intake of purine from meat, fish, and poultry. The mechanism of stone formation is probably due to salting out calcium oxalate by urate . A low-purine diet is desirable but difficult for many patients to achieve. The alternative is allopurinol at a dose of 100 mg bid is usually sufficient.

- ***C. Primary Hyperparathyroidism:*** The diagnosis of this condition is established by documenting that hypercalcemia that cannot be otherwise explained is accompanied by inappropriately elevated serum concentrations of parathyroid hormone.

- Hypercalciuria, usually present, raises the urine super-saturation of calcium phosphate and/or calcium oxalate. Prompt diagnosis is important because Para-thyroidectomy should be carried out before renal damage or bone disease occurs.

- ***D. Hyperoxaluria:*** Urine oxalate comes from diet and endogenous metabolic production, with approximately 40-50% originating from dietary sources . Mild hyperoxaluria (50-80 mg/d) is usually caused by:

- Excessive intake of high-oxalate foods such as spinach, nuts, and chocolate.
- In addition, low-calcium diets may promote hyperoxaluria as there is less calcium binding oxalate in the intestine, increasing the amount of oxalate available for absorption.

- Enteric hyperoxaluria is a consequence of small bowel disease resulting in fat malabsorption e.g. jejunoileal bypass for obesity.
- Hereditary hyperoxaluria states are rare causes of severe hyperoxaluria, often greater than 150 mg per day. Patients usually present with recurrent calcium oxalate stones during childhood. Severe hyperoxaluria from any cause can produce tubule-interstitial nephropathy and lead to stone formation.

- ***E. Hypocitraturia:*** Urine citrate prevents calcium stone formation by creating a soluble complex with calcium, effectively reducing free urine calcium. Hypocitraturia is found in 15-60% of stone formers, either as a single disorder or in combination with other metabolic abnormalities. It can be secondary to systemic disorders, such as renal tubular acidosis (RTA), chronic diarrheal illness, or hypokalemia, or it may be a primary disorder, in which case it is called *idiopathic hypocitraturia*.

- **Treatment :** is with alkali; generally bicarbonate or citrate salts are used. Potassium salts are preferred as sodium loading increases urinary excretion of calcium, reducing the effectiveness of treatment.

- ***2. Uric acid stones:***
- These stones form because the urine becomes supersaturated with undissociated uric acid. In gout, idiopathic uric acid lithiasis, and dehydration, the average pH is often below 5.0. Un dissociated uric acid therefore predominates and is soluble in urine only in concentrations of 100 mg/L.

- Concentrations above this level represent super-saturation that causes crystals and stones .
- Plugging of the renal collecting tubules by uric acid crystals can cause acute renal failure.

Treatment of uric acid stone:

- Supplemental alkali, 1 to 3 mmol/kg of body weight per day, should be given in three or four evenly spaced, divided doses, one of which should be given at bedtime
- Potassium citrate may reduce the risk of calcium salts crystallizing when urine pH is increased, whereas sodium citrate or sodium bicarbonate may increase the risk. If the overnight urine pH is below 5.5, the evening dose of alkali may be raised or 250 mg acetazolamide added at bedtime.

- A low-purine diet should be instituted in those uric acid stone formers with hyperuricosuria.
- Allopurinol may be added to patient regimen.
- If hypercalciuria is also present, it should be specifically treated, as alkali alone could lead to calcium phosphate stone formation.

- ***3. Cystinuria and cystine stones:***
- In this autosomal recessive disorder, proximal tubular and jejunal transport of the dibasic amino acids cystine, lysine, arginine, and ornithine are defective, and excessive amounts are lost in the urine. Clinical disease is due solely to the insolubility of cystine, which forms stones.

Treatment of cystine stones:

- High fluid intake, even at night, is the cornerstone of therapy. Daily urine volume should exceed 3 L.
- Raising urine pH with alkali is helpful, provided the urine pH exceeds 7.5.
- A low-salt diet (100 mmol/d) can reduce cystine excretion up to 40%.

- Because side effects are frequent, drugs such as penicillamine and tiopronin, which form the soluble disulfide cysteine-drug complexes, should be used only when fluid loading, salt reduction, and alkali therapy are ineffective.
- Captopril, which has a free sulfhydryl group to bind cysteine, has been used in a limited number of patients with some success.
- Low-methionine diets have not proved to be practical for clinical use, but patients should avoid protein gluttony.

4. *Struvite stones:*

- These stones are a result of urinary infection with bacteria, usually *Proteus* species, which possess urease, an enzyme that degrades urea to NH3 and CO2. Those in the end will result in the formation of (struvite).

- Chronic *Proteus* infection can occur because of impaired urinary drainage, urologic instrumentation or surgery, and especially with chronic antibiotic treatment, which can favor the dominance of *Proteus* in the urinary tract.

Treatment of struvite stone:

- Complete removal of the stone with subsequent sterilization of the urinary tract is the treatment of choice for patients who can tolerate the procedures.
- Irrigation of the renal pelvis and calyces with hemiacidrin, a solution that dissolves struvite, can reduce recurrence after surgery.

- Newer procedures such as lithotripsy and percutaneous nephrolithotomy, alone or in combination, have largely replaced open surgery.
- Antimicrobial treatment is best reserved for dealing with acute infection and for maintenance of sterile urine after surgery.

- For patients who are not candidates for surgical removal of stone, acetohydroxamic acid, an inhibitor of urease, can be used. But acetohydroxamic acid has many side effects, such as headache, tremor, and thrombophlebitis that limit its use.

Thank you