Stones are aggregates composed of varying amounts of crystalloid and organic matrix.
Theories to explain urinary stone disease are :
**The nucleation theory** suggests that urinary stones originate from a nidus (or nucleus) of crystals or foreign bodies immersed in supersaturated urine.
**The crystal inhibitor theory** claims that calculi form owing to the absence or low concentration of natural stone inhibitors, including magnesium, citrate, pyrophosphate which has been shown to be an efficient inhibitor of crystal nucleation and aggregation.

**STONES COMPONENTS** :

**A. CRYSTAL COMPONENT**: The addition of further crystals to the saturated solution will cause the crystals to precipitate.
**B. MATRIX COMPONENT :**The amount of the non-crystalline matrix component of urinary stones varies with stone type, commonly ranging from 2% to 10% by weight. It is composed predominantly of protein**.** It may serve as a nidus for crystal aggregation or as a naturally occurring glue to adhere small crystal components.

## RENAL AND URETERAL STONES

The lifetime prevalence of kidney stone disease is estimated at 1% to 15%, with the probability of having a stone varying according to age, gender, race, and geographic location. A higher prevalence of stone disease is found in hot, arid, or dry climates.
Staghorn calculi are those stones that fill the major part of the renal collecting system. Typically, they occupy the renal pelvis and branch into most of the calyces, mimicking the horns of a deer or stag . Most staghorn stones are composed of struvite
Ureteral stones usually become impacted at three distinct sites where the caliber of the ureter narrows: the PUJ, the iliac vessels, and the ureterovesical junction.

### Stone Composition and Relative Occurrence :

| A- Calcium-Containing Stones | | B- Non–Calcium-Containing Stones | |
|---|---|---|---|
| Calcium oxalate | **60%** | Uric acid | **7%** |
| Hydroxyapatite | **20%** | Struvite | **7%** |
| Brushite (Calcium hydrogen phosphate dehydrate) | **2%** | Cystine | **1-3%** |
| | | Others : as Xanthine, Indinavir | **< 1%** |

## A. CALCIUM CALCULI

Calcium nephrolithiasis is most commonly due to elevated urinary calcium, elevated urinary uric acid, elevated urinary oxalate, or a decreased level of urinary citrate. 80% to 85% of all urinary stones are calcareous.

***Types and Etiology of Clcium Clculi :***

**1-hypercalciuric nephrolithiasis:** When urinary calcium (>150–200 mg/24 h) ;types including:

**A. Absorptive hypercalciuric nephrolithiasis—**is secondary to increased calcium absorption from the small bowel, predominantly from the jejunum.

**B. Resorptive hypercalciuric nephrolithiasis—**is associated with hypercalcemia that secondary to hyperparathyroidism.

**C. Renal-induced hypercalciuric nephrolithiasis—**is due to an intrinsic renal tubular defect in calcium excretion.

**2. Hyperuricosuric calcium nephrolithiasis—**Hyperuricosuric calcium nephrolithiasis is due to an increase in urinary urates (>600 mg/24 h in women and >750 mg/24 h in men) that makes a nidus for calcium crystals aggregation.

**3. Hyperoxaluric calcium nephrolithiasis—**is secondary to increased urinary oxalate levels (>40 mg/24 h).

**4. Hypocitraturic calcium nephrolithiasis—**Citrate is an important inhibitor of urinary stone disease. conditions which decrease the excretion of citrate (<320 mg/24 h) will facilitate calcium stone formation.

**B. NON-CALCIUM CALCULI**

**1. Struvite—**Struvite stones are composed of magnesium, ammonium, and phosphate (MAP). They are found most commonly in women and may recur rapidly. They frequently present as renal staghorn calculi. Struvite stones are infection stones associated with urea-splitting organisms, like *Proteus, Pseudomonas, etc...* that results in an alkaline urinary pH .

**2. Uric acid—**Uric acid stones compose 7% of all urinary calculi and are usually found in men. Patients with gout, myeloproliferative diseases, or rapid weight loss, and those treated for malignant conditions with cytotoxic drugs have a high incidence of uric acid lithiasis. Elevated uric acid levels are frequently due to dehydration and excessive purine intake. Patients present with an acidic urinary pH.

**3. Cystine—**Cystine lithiasis is secondary to an inborn error of metabolism associated with cystinuria.

**4. Others —**like **Xanthine stones** and **Indinavir stone** ( indinavir is Protease inhibitors used in treatment of patients with AIDS ).

## Symptoms & Signs at Presentation

**A. PAIN** : Renal colic usually is caused by stretching of the collecting system or ureter, while non-colicky renal pain is caused by distention of the renal capsule. Local mechanisms such as inflammation, edema, hyperperistalsis, and mucosal irritation may contribute to the perception of pain in patients with renal calculi.

**B. HEMATURIA :** A complete urinalysis helps to confirm the diagnosis of a urinary stone by assessing for hematuria and crystalluria and documenting urinary pH. Patients frequently admit to intermittent gross or microscopic hematuria **.**

**C. INFECTION :** Magnesium ammonium phosphate (struvite) stones are synonymous with infection stones. They are commonly associated with *Proteus, Pseudomonas,* etc…. , Calcium phosphate stones are the second variety of stones associated with infections.

**D. ASSOCIATED FEVER :** The association of urinary stones with fever is a relative medical emergency. Signs of clinical sepsis are variable and include fever, tachycardia, hypotension, and cutaneous vasodilation. Fever associated with urinary tract obstruction requires prompt decompression. This may be accomplished with a retrograde catheter (double-J, or an insertion of a percutaneous nephrostomy tube is required.

**E. NAUSEA AND VOMITING :** Upper-tract obstruction is frequently associated with nausea and vomiting. Intravenous fluids are required to restore a euvolemic state. Intravenous fluids should not be used to force a diuresis in an attempt to push a ureteral stone down the ureter.

## Evaluation

### A. DIFFERENTIAL DIAGNOSIS

Urinary stones can mimic other retroperitoneal and peritoneal pathologic states. A full differential diagnosis of the acute abdomen should be made, including acute appendicitis, ectopic and unrecognized pregnancies, ovarian pathologic conditions including twisted ovarian cysts, diverticular disease, bowel obstruction, biliary stones with and without obstruction, peptic ulcer disease, acute renal artery embolism, and abdominal aortic aneurysm**.**

### B. HISTORY AND PHYSICAL EXAMINATION

A thorough medical history and abdominal examination should be done to exclude other causes of abdominal pain.

## C. INVESTIGATIONS :

### 1.LABORATORY INVESTIGATIONS

Including urinalysis, 24-hour urine collections to determine abnormally high levels of calcium, uric acid, oxalate, magnesium, or citrate in patients with recurrent stones, Serum calcium determinations are useful in patients with calcium urolithiasis.

### 2. RADIOLOGIC INVESTIGATIONS:

**A. Computed tomography—**Noncontrast spiral CT scans are now the imaging modality of choice in patients presenting with acute renal colic. It can visualized urinary stones and their associated effects as hydronephrosis and it can images other peritoneal and retroperitoneal structures and helps when the diagnosis is uncertain.

**B. KUB films and directed ultrasonography—**A KUB film and renal ultrasound are effective in establishing a diagnosis. Stones that appear opaque in KUB include calcareous stones, struvite and cystine atones , while lucent stones include uric acid , and xanthine stones.

**C. Intravenous pyelography—**An IVP can document simultaneously nephrolithiasis and upper-tract anatomy.

**D. Retrograde pyelography—**Retrograde pyelography occasionally is required to delineate upper-tract anatomy and localize small or radiolucent offending calculi.

**E. Magnetic resonance imaging—**MRI is a poor study to document urinary stone disease.

### 3. STONE ANALYSIS

Recurrent stone formation have arisen the necessity of both chemical stone analysis and metabolic evaluation which gives helpful information for such an investigation that can aid with preventive therapy.

## Intervention

### A. CONSERVATIVE OBSERVATION

**1- Fluid intake :** One mainstay of conservative management is the forced increase in fluid intake to achieve a daily urine output of 2 L .

**2-Selective medical therapy :** may be used in the following conditions:

**A**-Patients with hyperuricosuria should be instructed to decrease dietary purine intake. Allopurinol can decrease uric acid production and may be ideal for those patients with a history of gout.

**B**-Thiazides are first-line therapy for the treatment of renal induced hypercalciuria.

**C**-Citrates are effective therapy for the management of hypocitraturia.

**D**-Treatment options for cystinuria consists of aggressive fluid intake, urinary alkalinization, salt avoidance, and the use of a cystine-binding agent.

**E**-Struvite calculi are best managed with surgical removal and can be avoided with the use of antibiotic prophylaxis.

**3-Dissolution agents:** Oral alkalinizing agents include sodium or potassium bicarbonate and potassium citrate are especially effective with pH-sensitive calculi as in uric acid and cystine lithiasis. Struvite stone dissolution requires acidification of urine and may be achieved successfully with specific solutions.

### B. RELIEF OF OBSTRUCTION

Urinary stone disease may result in significant morbidity and possible mortality in the presence of obstruction, especially with concurrent infection. A patient with obstructive urinary calculi with fever and infected urine requires emergent drainage by retrograde placement of a double-J ureteral stent or a percutaneous nephrostomy tube.

Most ureteral calculi pass and do not require intervention. Spontaneous passage depends on stone size, shape, location, and associated ureteral edema . Ureteral calculi 4–5 mm in size have a 40–50% chance of spontaneous passage. In contrast, calculi >6 mm have a <5% chance of spontaneous passage.

The vast majority of stones that pass do so within a 6- week period after the onset of symptoms. Ureteral calculi discovered in the distal ureter at the time of presentation have a 50% chance of spontaneous passage, in contrast to a 25% and 10% chance in the mid- and proximal ureter, respectively.

**C. EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY**

The concept is using shock waves to fragment stones through an energy source to create the shock wave that a transferred from outside to inside the body. Most patients harboring small renal or ureteric calculi can be treated satisfactorily with SWL.

**D. URETEROSCOPIC STONE EXTRACTION**

Ureteroscopic stone extraction is highly efficacious for lower ureteral calculi, Stone-free rates are dependent on stone size and location, length of time the stone has been impacted, and the experience of the operator. Laser system is the best variety of lithotrites can be used through an ureteroscope.

**E. PERCUTANEOUS NEPHROLITHOTOMY**

Percutaneous removal of renal and proximal ureteral calculi is the treatment of choice for large (>2.5 cm) calculi and those resistant to ESWL .

**F. OPEN AND LAPROSCOPIC STONE SURGERY**

Open stone surgery is the classic way to remove calculi. The morbidity of the incision, the possibility of retained stone fragments, and the ease and success of less invasive techniques have made these procedures relatively uncommon .

## Prevention

In general, 50% of patients experience recurrent urinary stones within 5 years without prophylactic intervention. Fluids should be encouraged during mealtime, after meals, and at nighttime. Life style changes should be encouraged with alterations in physical activity may significantly reduce the incidence of recurrent nephrolithiasis.

Sudies have confirmed the advantage of a diet with reduced animal protein (meat) intake , a diet high in fruits and vegetables and dietary sodium restriction in stone formers.

Metabolic evaluation , in addition to prophylactic medications that inhibit stone formation are required for those patients with recurrent calculi.

## BLADDER STONES

Bladder calculi usually are a manifestation of an underlying pathologic condition, including voiding dysfunction or a foreign body. Voiding dysfunction may be due to a urethral stricture, benign prostatic hyperplasia, bladder neck contracture, or flaccid or spastic neurogenic bladder, all of which result in static urine. Foreign bodies can serve as nidi for stones . Most bladder calculi are seen in men.

A solitary bladder stone is the rule, but there are numerous stones in 25% of patients , Patients present with irritative voiding symptoms, intermittent urinary stream, urinary tract infections, hematuria, or pelvic pain. A large percentage of bladder stones are radiolucent (uric acid). Ultrasound of the bladder identifies the stone.

Cystolitholapaxy allows most stones to be broken and subsequently removed through a cystoscope. Cystolithotomy can be performed through a small abdominal incision.

## URETHRAL STONES

Urethral calculi usually originate from the bladder and rarely from the upper tracts. Most ureteral stones that pass spontaneously into the bladder can pass through the urethra unimpeded. Urethral stones may develop secondary to urinary stasis, secondary to a urethral diverticulum, near urethral strictures, or at sites of previous surgery.