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.

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.

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		B- Non-Calcium-Containing	
<u>Stones</u>		<u>Stones</u>	
Calcium oxalate	60%	Uric acid	7%
Hydroxyapatite	20%	Struvite	7%
Brushite (Calcium hydrogen	2%	Cystine	1-3%
phosphate dehydrate)		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)
- **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 ureasplitting 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

- **<u>A. PAIN</u>**: 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.
- **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.
- <u>C. INFECTION</u>: 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. 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.

Diagnosis

1.<u>LABORATORY INVESTIGATIONS</u>

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.
- **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.

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.