



URINARY STONE DISEASE: ETIOLOGY, PATHOGENESIS, AND TREATMENT

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Abstract: Urolithiasis (UL) is one of the most common diseases, with worldwide increasing incidence and prevalence. The pathogenesis of calcium oxalate (CaOx) UL, which accounts for >80% of all urinary stones, is only incompletely understood.

Objective

Our aim was to review trends in epidemiology and current concepts for the pathogenesis, treatment of urinary stone disease.

Etiology (Causes)

Urinary stone disease (urolithiasis) results from crystallization of minerals in the urinary tract. Major causes include metabolic, dietary, environmental, and anatomical factors.

- Dehydration – concentrated urine increases crystal risk
- Diet – high oxalate, salt, animal protein; low citrate
- Metabolic abnormalities – hypercalciuria, hyperoxaluria, hyperuricosuria, cystinuria, hyperparathyroidism
- Urinary tract infections (Proteus, Klebsiella) – struvite stones
- Anatomical abnormalities – urinary stasis
- Genetic predisposition
- Medications – loop diuretics, topiramate, indinavir

Pathogenesis

Stone formation involves supersaturation, nucleation, crystal growth, and retention within the urinary tract.

- a. Supersaturation – urine contains excessive calcium, oxalate, uric acid, or cystine.
- b. Nucleation – crystals aggregate around a nucleus (cell debris or bacteria).
- c. Growth and aggregation – crystals enlarge and form masses.
- d. Retention – stones remain in kidney (nephrolithiasis), ureter (ureterolithiasis), or bladder (cystolithiasis).

Common stone types:

Stone Type	Composition	Common Cause
Calcium oxalate	Calcium + oxalate	Hypercalciuria, dehydration
Uric acid	Uric acid	Gout, high purine diet,



		acidic urine
Struvite	Magnesium ammonium phosphate	UTI with urease-producing bacteria
Cystine	Cystine	Genetic cystinuria

Clinical Manifestations

- Severe flank pain (renal colic) radiating to groin
- Hematuria
- Nausea and vomiting
- Dysuria, urinary frequency
- Fever and chills (if infection)

Diagnosis

- Urinalysis: crystals, blood, infection
- Imaging: CT KUB (best), ultrasound, X-ray KUB
- Blood tests: serum calcium, uric acid, electrolytes
- Stone analysis for composition

Treatment

a. Conservative / Medical management:

- Hydration (2.5–3 L/day)
- Pain relief (NSAIDs, opioids)
- Alpha-blockers (tamsulosin) to facilitate stone passage
- Antibiotics if infection

b. Stone-specific medical therapy:

Stone Type	Treatment
Calcium stones	Thiazides, low sodium diet
Uric acid stones	Alkalinization (potassium citrate), allopurinol
Cystine stones	High fluid, alkalinization, penicillamine
Struvite stones	Surgery + antibiotics

c. Surgical / procedural options:

- Extracorporeal shock wave lithotripsy (ESWL)
- Ureterscopy with laser lithotripsy
- Percutaneous nephrolithotomy (PCNL)
- Open or laparoscopic surgery (rare)



6. Prevention

- Hydration (2–3 L/day)
- Diet modification: low salt, low oxalate, normal calcium
- Treat underlying metabolic or endocrine disorders
- Regular urine and metabolic follow-up

Conclusions

The lithogenesis of key stones is multifactorial. Lifestyle and dietary choices are important contributing factors. The pathogenesis and pathophysiology of CaOx stones is still incompletely understood. Recent evidence suggests a primary interstitial apatite crystal formation that secondarily leads to CaOx stone formation.

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