UROLITHIASIS

Urinary calculi are the third most common affliction of the urinary tract, exceeded only by urinary tract infections and pathologic conditions of the prostate.

The nomenclature associated with urinary stone disease arises from a variety of disciplines. .

Before the time of von Struve, the stones were referred to as guanite, because magnesium ammonium phosphate is prominent in bat droppings.

The history of the nomenclature associated with urinary stone disease is as intriguing as that of the development of the interventional techniques used in their treatment.

Urinary stones have plagued humans since the earliest records of civilization.

The etiology of stones remains speculative.

Advances in the surgical treatment of urinary stones have outpaced our understanding of their etiology.

Without such follow-up and medical intervention, stone recurrence rates can be as high as 50% within 5 years.

Renal & Ureteral Stones
Etiology

Theories to explain urinary stone disease are incomplete.

Renal & Ureteral Stones
Etiology

Stone formation requires supersaturated urine. Supersaturation depends on urinary pH, ionic strength, solute concentration, and complexation.

Renal & Ureteral Stones
Etiology

The activity coefficient reflects the availability of a particular ion.

Renal & Ureteral Stones
Etiology

Concentrations above this point are metastable and are capable of initiating crystal growth and heterogeneous nucleation.

Renal & Ureteral Stones
Etiology

Multiplying 2 ion concentrations reveals the concentration product.

The concentration products of most ions are greater than established solubility products.

Renal & Ureteral Stones

Etiology

Crystal formation is modified by a variety of other substances found in the urinary tract, including magnesium, citrate, pyrophosphate, and a variety of trace metals.

Renal & Ureteral Stones
Etiology

The nucleation theory suggests that urinary stones originate from crystals or foreign bodies immersed in supersaturated urine.

Renal & Ureteral Stones

Etiology

Additionally, many stone formers' 24-h urine collections are completely normal with respect to stone-forming ion concentrations.

Renal & Ureteral Stones
Etiology

This theory does not have absolute validity since many people lacking such inhibitors may never form stones, and others with an abundance of inhibitors may, paradoxically, form them.

Crystal Component

Stones are composed primarily of a crystalline component.

Crystals of adequate size and transparency are easily identified under a polarizing microscope.

Crystal Component

Multiple steps are involved in crystal formation, including nucleation, growth, and aggregation.

Crystal Component

A crystal of one type thereby serves as a nidus for the nucleation of another type with a similar crystal lattice.

Crystal Component

How these early crystalline structures are retained in the upper urinary tract without uneventful passage down the ureter is unknown.

The theory of mass precipitation or intranephronic calculosis suggests that the distal tubules or collecting ducts, or both, become plugged with crystals, thereby establishing an environment of stasis, ripe for further stone growth.

Crystal Component

This explanation is unsatisfactory; tubules are conical in shape and enlarge as they enter the papilla, thereby reducing the possibility of ductal obstruction.

Crystal Component

The fixed particle theory postulates that formed crystals are somehow retained within cells or beneath tubular epithelium. Randall noted whitish-yellow precipitations of crystalline substances occurring on the tips of renal papillae as submucosal plaques.

Crystal Component

These can be appreciated during endoscopy of the upper urinary tract.

Matrix Component

The amount of the noncrystalline, matrix component of urinary stones varies with stone type, commonly ranging from 2% to 10% by weight.

Matrix Component

Histologic inspection reveals laminations with scant calcifications.

Matrix Component

The role of matrix in the initiation of ordinary urinary stones as well as matrix stones is unknown.

Urinary Ions
Calcium

Calcium is a major ion present in urinary crystals.

Diuretic medications may exert a hypocalciuric effect by further decreasing calcium excretion.

Oxalate

Oxalate is a normal waste product of metabolism and is relatively insoluble.

Oxalate

Once absorbed from the small bowel, oxalate is not metabolized and is excreted almost exclusively by the proximal tubule.

Oxalate

Normal excretion ranges from 20 to 45 mg/d and does not change significantly with age.

Oxalate

Hyperoxaluria may develop in patients with bowel disorders, particularly inflammatory bowel disease, small-bowel resection, and bowel bypass.

Oxalate

The unbound oxalate is readily absorbed.

Phosphate

Phosphate is an important buffer and complexes with calcium in urine.

Phosphate

The small amount of phosphate filtered by the glomerulus is predominantly reabsorbed in the proximal tubule.

Uric Acid

Uric acid is the by-product of purine metabolism.
The pH of uric acid is 5.75.

Uric Acid

Rarely, a defect in xanthine oxidase results in increased levels of xanthine; the xanthine may precipitate in urine, resulting in stone formation.

Uric Acid

This results from a deficiency of adenine phosphoribosyltransferase (APRT).

Sodium

Although not identified as one of the major constituents of most urinary calculi, sodium plays an important role in regulating the crystallization of calcium salts in urine.

Sodium

This reduces the ability of urine to inhibit calcium oxalate crystal agglomeration.

Citrate

Citrate is a key factor affecting the development of calcium urinary stones.

Citrate

Metabolic stimuli that consume this product (as with intracellular metabolic acidosis due to fasting, hypokalemia, or hypomagnesemia) reduce the urinary excretion of citrate.

Magnesium

Dietary magnesium deficiency is associated with an increased incidence of urinary stone disease.

Magnesium

The exact mechanism whereby magnesium exerts its effect is undefined.

Sulfate

Urinary sulfates may help prevent urinary calculi. They can complex with calcium.

Stone Varieties

Calcium Calculi

Calcifications can occur and accumulate in the collecting system, resulting in nephrolithiasis.

Eighty to eighty-five percent of all urinary stones are calcareous.

Calcium Calculi

Hyperuricosuria is identified as a solitary defect in 8% of patients and associated with additional defects in 16%.

Calcium Calculi

Finally, decreased urinary citrate is found as an isolated defect in 17% of patients and as a combined defect in an additional 10%.

Calcium Calculi

Symptoms are secondary to obstruction, with resultant pain, infection, nausea, and vomiting, and rarely culminate in renal failure.

Calcium Calculi

Most patients with nephrolithiasis, however, do not have obvious nephrocalcinosis.

Calcium Calculi

Nephrocalcinosis may result from a variety of pathologic states.

Calcium Calculi

Disease processes resulting in bony destruction, including hyperparathyroidism, osteolytic lesions, and multiple myeloma, are a third mechanism. Finally, dystrophic calcifications forming on necrotic tissue may develop after a renal insult.

Absorptive Hypercalciuric Nephrolithiasis

Normal calcium intake averages approximately 900-1000 mg/d.

Absorptive Hypercalciuric Nephrolithiasis

This results in an increased load of calcium filtered from the glomerulus.

Absorptive Hypercalciuric Nephrolithiasis

Absorptive hypercalciuria can be subdivided into 3 types.

Absorptive Hypercalciuric Nephrolithiasis

Urinary calcium excretion returns to normal values with therapy.

Symptoms & Signs at Presentation

Symptomatology

1) **Pain**
2) **Hematuria**
3) **Pyuria**

12% of men and 5% of women will suffer from renal stones by the age of 70 years.

The majority of patients with nephrolithiasis are those from 25 up to 55 years.

By localization there can be stones of the:

-Calices

-

Upper-tract urinary stones usually eventually cause pain.

The character of the pain depends on the location.

Radiation of pain with various types of ureteral stone.

Upper right: Midureteral stone. Same as above but with more pain in the lower abdominal quadrant.

Pain

Renal colic and noncolicky renal pain are the 2 types of pain originating from the kidney.

Pain

This pain is due to a direct increase in intraluminal pressure, stretching nerve endings.

Pain

Renal colic does not always wax and wane or come in waves like intestinal or biliary colic but may be relatively constant.

Pain

In the ureter, however, local pain is referred to the distribution of the ilioinguinal nerve and the genital branch of the genitofemoral nerve, whereas pain from obstruction is referred to the same areas as for collecting system calculi (flank and costovertebral angle), thereby allowing discrimination.

Pain

The vast majority of urinary stones present with the acute onset of pain due to acute obstruction and distention of the upper urinary tract.

Pain

The stone burden does not correlate with the severity of the symptoms. Small ureteral stones frequently present with severe pain, while large staghorn calculi may present with a dull ache or flank discomfort.

Pain

The pain frequently is abrupt in onset and severe and may awaken a patient from sleep.

Pain

This movement is in contrast to the lack of movement of someone with peritoneal signs; such a patient lies in a stationary position.

Renal Calyx

Stones or other objects in calyces or caliceal diverticula may cause obstruction and renal colic.

Renal Calyx

Radiographic imaging may not reveal evidence of obstruction despite the patient's complaints of intermittent symptoms.

Renal Calyx

Caliceal calculi occasionally result in spontaneous perforation with urinoma, fistula, or abscess formation.

Renal Calyx

Effective long-term treatment requires stone extraction and elimination of the obstructive component.

Renal Pelvis

Stones in the renal pelvis > 1 cm in diameter commonly obstruct the ureteropelvic junction, generally causing severe pain in the costovertebral angle, just lateral to the sacrospinalis muscle and just below the 12th rib.

Renal Pelvis

Symptoms frequently occur on an intermittent basis following a drinking binge or consumption of large quantities of fluid.

Renal Pelvis

Partial or complete staghorn calculi that are present in the renal pelvis are not necessarily obstructive.

Upper and Mid Ureter

Pain associated with ureteral calculi often projects to corresponding dermatomal and spinal nerve root innervation regions.

Upper and Mid Ureter

The pain of upper ureteral stones thus radiates to the lumbar region and flank.

Upper and Mid Ureter

Stones or other objects in the upper or mid ureter often cause severe, sharp back (costovertebral angle) or flank pain.

Distal Ureter

Calculi in the lower ureter often cause pain that radiates to the groin or testicle in males and the labia majora in females.

Distal Ureter

Stones in the intramural ureter may mimic cystitis, urethritis, or prostatitis by causing suprapubic pain, urinary frequency and urgency, dysuria, stranguria, or gross hematuria.

Bowel symptoms are not uncommon.

In women the diagnosis may be confused with menstrual pain, pelvic inflammatory disease, and ruptured or twisted ovarian cysts.

Distal Ureter

Strictures of the distal ureter from radiation, operative injury, or previous endoscopic procedures can present with similar symptoms.

Hematuria

A complete urinalysis helps to confirm the diagnosis of a urinary stone by assessing for hematuria and crystalluria and documenting urinary pH.

Infection

Magnesium ammonium phosphate (struvite) stones are synonymous with infection stones.

Infection

All stones, however, may be associated with infections secondary to obstruction and stasis proximal to the offending calculus.

Infection

Uropathogenic bacteria may alter ureteral peristalsis by the production of exotoxins and endotoxins.

Infection

Local inflammation from infection can lead to chemoreceptor activation and perception of local pain with its corresponding referral pattern.

Pyonephrosis

Presentation is variable and may range from asymptomatic bacteriuria to florid urosepsis. Bladder urine cultures may be negative.

Pyonephrosis

Radiographic investigations are frequently nondiagnostic.

Pyonephrosis

If unrecognized and untreated, pyonephrosis may develop into a renocutaneous fistula.

Xanthogranulomatous Pyelonephritis

Xanthogranulomatous pyelonephritis is associated with upper-tract obstruction and infection.

Xanthogranulomatous Pyelonephritis

Open surgical procedures, such as a simple nephrectomy for minimal or nonrenal function, can be challenging owing to marked and extensive reactive tissues.

Associated Fever

Costovertebral angle tenderness may be marked with acute upper-tract obstruction; however, it cannot be relied on to be present in instances of long-term obstruction.

Associated Fever

If retrograde manipulations are unsuccessful, insertion of a percutaneous nephrostomy tube is required.

Nausea and Vomiting

Effective ureteral peristalsis requires coaptation of the ureteral walls and is most effective in a euvolemic state.

Special Situations
Pregnancy

Renal colic is the most common nonobstetric cause of acute abdominal pain during pregnancy.

Special Situations
Pregnancy

The increased filtered load of calcium, uric acid, and sodium from the 25-50% increase in glomerular filtration rate associated with pregnancy has been thought to be a responsible factor in stone development.

Special Situations
Pregnancy

Initial investigations can be undertaken with renal ultrasonography and limited abdominal x-rays with appropriate shielding.

Special Situations
Pregnancy

Treatment requires balancing the safety of the fetus with the health of the mother.

Obesity

Ultrasound examination is hindered by the attenuation of ultrasound beams.

Obesity

Standard lithotripters have focal lengths less than 15 cm between the energy source and the F2 target, frequently making treatment of obese patients impossible.

Obesity

A preplaced heavy suture eases removal of such sheaths.

Obesity

Postoperative prophylaxis for thromboembolic complications should be considered.

There are numerous theories of origination and development of urolithiasis, however, any of them does not explain completely its origin.

The known role in the etiology of urolithiasis is played by the disturbance of urate, phosphate, oxalic exchange, however, it is not to be overestimated.

It is possible to divide the numerous factors contributing to the formation of stones, into exogenous and endogenic, and the latter into general and local (connected directly with changes in the kidney).

The formation of phosphate stones is promoted also by fractures of tubular bones.

The uric acid is the end product of purine exchange.

To the internal causes, contributing to originating urolithiasis, we also attribute disturbance of a normal function of the gastrointestinal tract (chronic gastritis, colitis, peptic ulcer).

The local factors of lithogenesis

70-80% of all stones are Ca containing. The major factor in urolithiasis in children and adults is the production of insoluble calcium salts of oxalic acid.

Three conditions which contribute to the formation of struvite stones are the following:

Congenital anomalies

There are four types of urate urolithiasis:

Idiopathic urate urolithiasis

Formation of stones of uric acid depends on:

- **pH of urine**

Anatomical Pathology

- Degree of obstruction of the urinary paths
- Expressiveness of inflammatory process, which, as a rule, accompanies the disease

Complications of urolithiasis

The most often complication of nephrolithiasis is the inflammatory process in the kidney, that may clinically proceed in the acute or chronic form.

Both chronic pyelonephrosis and pyonephrosis, as well as hydronephrosis owing to urolithiasis can entail a nephrogenic arterial hypertention.

The most severe complication of urolithiasis is prerenal anuria with the development of acute renal failure.

Diagnostics

The diagnosis of urolithiasis is established, first of all, on the basis of the patient's complaints and anamnesis.

Laboratory research

It is necessary to remember, that the absence of pathological changes of urine does not allow to eliminate nephrolithiasis, as the stone can desely obturate the urinary paths, and the investigated urine is excreted from a contralateral kidney.

Ultrasound investigation

X-ray examination

Retrograde ureteropyelography

Computed tomography

Differential diagnosis

Treatment

Conservative treatment

Indications for surgical intervention:

1. Urinary obstructions with progressing damage of the kidney
2. Persistent infection despite antibiotics
3. Uncontrollable pain
4. Impairment of renal function
5. A relapsing gross hematuria

Instrumental methods of treatment

Percutaneous nephrolithotomy

Extracorporeal shock wave lithotripsy (ESWL)

The indications for open surgical treatment are:

Pains depriving the patient of capability normally to live and to work