# UROLITHIASIS

# *Metabolic etiology in Urinary Lithiasis*

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# BACKGROUND

- Urinary stone disease continues to occupy an important place in everyday urological practice. The average lifetime risk of stone formation has been reported in the range of 5-10%.
- A predominance of men over women (approx. 3:1) can be observed with an incidence peak between the fourth and fifth decade of life.
- Recurrent stone formation is a common problem with all types of stones and therefore an important part of the medical care of patients with stone disease.

# Theories of **Stone Formation**

- **A. Nucleation Theory**
- **B. Stone Matrix Theory**
- **C. Inhibitor of Crystallization Theory**

Most investigators acknowledge that these 3 theories describe the 3 basic factors influencing urinary stone formation. It is likely that more than one factor operates in causing stone disease. A generalized model of stone formation combining these 3 basic theories has been proposed.

# RISK FACTORS

- *Start of disease early in life: <25 years*
- *Stone containing brushite*
- *Only one functioning kidney*
- *Disease associated with stone formation*:

- hyperparathyroidism
- renal tubular acidosis (partial/complete)
- jejunoileal bypass
- Crohn’s disease
- intestinal resection
- malabsorptive conditions
- sarcoidosis
- hyperthyroidism

# RISK FACTORS

- ***Medication associated with stone formation:***

**- calcium supplements**
**- vitamin D supplements**
**- acetazolamide - ascorbic acid in megadoses ( > 4 g/day)**
**- sulphonamides - triamterene**
**- indinavir**

- ***Anatomical abnormalities associated with stone formation:***

**- tubular ectasia (medullary sponge kidney)**
**- pelvo-ureteral junction obstruction**
**- calix diverticulum, calix cyst**
**- ureteral stricture**
**- vesico-ureteral reflux**
**- horseshoe kidney**
**- ureterocele**

# Etiology (according Capital and I. Pogo Elko).

- A). Disorders of urinary tract:
- congenital abnormalities those favor to apostasies;
- obstructive processes;
- neurogenic duskiness of the urinary tract;
- inflammative and parasitogenic damages;
- foreign bodies of urinary tract;
- traumatic injuries.
- B) Liver and digestive tract disorders:
- latent and manifested hepathopathiy;
- hepatogenic gastritis;
- colitis, etc.
- C) Endocrine diseases
- hyperparathyreoidism;
- hyperthyroidism;
- hypopituitaric diseases;
- D.) Infect focuses of the urogenital system.
- E) Metabolism disorders.
- essential hypercalciuria;
- disorders of membranes for colloid substances diffusion;
- renal rickets, etc
- F) Injuries those leads to continuous immobilization
- fractures of the vertebral column and limbs
- osteomyelitis
- diseases of the bones and joints
- chronic diseases of the visceral organs and nervous system.
- G) Climate and geographical causes.
- dry and hot climate with a high vaporization
- decrease water supply
- iodine deficiency
- H) Disorders of nutrition and vitamins balance:
- retinole and oscorbine acid deficiency in food.
- Excessive amount of the ergocalciferole in organism.

# Renal Calculi


- 1 Coral calculus
- 2 Coral calculi fragment
- 3 Calculi, which are impregnated with blood pigments

# Aetiology:

**1-Geography:** tropical area, mountainous and Mediterranean countries.

**2-Climate:** direct relationship between temp and stone incidence.

**3-low Water intake:**

**4-Diet:**

**5-Socio-economic status:**

# Metabolic & endocrinal factors:

**I- $Ca^{++}$ oxalate & $Ca^{++}$ phosphate calculi:**

- **Hypercalcemia (hyperparathyroidism)**
- **Hypercalciuria.**
- **Hyperoxaluria: primary (congenital), secondary (enteric disease) or idiopathic (dietary).**
- **Hypocitraturia: .**
- **Hyperuricosuria:**
- **Renal tubulal acidosis:**
- **associated with hypercalciuria & hypocitraturia.**

**II- Uric acid calculi:**

Hyperuricemia and hyperuricosuria.

**III-Cystine calculi:** hereditary cystinuria.

**IV-Xanthine calculi:**hereditary xanthinuria

# Absorptive hypercalciuric nephrolithiasis

- Absorptive hypercalciuric nephrolithiasis Secondary to increased calcium absorption from the small bowel (jejunum). results in an increased load of calcium filtered from the glomerulus. result is suppression of parathyroid hormone, decreased tubular reabsorption of calcium, culminating in hypercalciuria (>4 mg/kg).

- Type 1 Absorptive hypercalciuria independent of diet Cellulose phosphate
- Type 2 Absorptive hypercalciuria dietary dependent common cause of urinary stone disease.
- Type 3 Absorptive hypercalciuria secondary to a phosphate renal leak serum phosphate 1, 25-dihydroxyvitamin D synthesis absorption of phosphate and calcium from the small bowel renal excretion of calcium Orthophosphate

# Resorptive hypercalciuric nephrolithiasis

- Primary hyperparathyroidism present with nephrolithiasis. Parathyroid adenoma Inc.urinary phosphorus.
- decrease in plasma phosphorus,
- inc in plasma calcium
- decrease in urinary calcium. Hypercalcemia
- renal damage limits the concentrating ability of the kidney and impairs the kidney's ability to acidify urine.

# Renal-induced hypercalciuric nephrolithiasis

- due to an intrinsic renal tubular defect in calcium excretion. Ca+2 excretion serum Ca+2 PTH Absorption Ca+2 in gut

# Hyperuricosuric calcium nephrolithiasis

- dietary intake of purines endogenous uric acid production. elevated urinary uric acid levels >600 mg/24 h in women >750 mg/24 h in men

# Hyperoxaluric calcium nephrolithiasis

- secondary to increased urinary oxalate levels (>40 mg/24 h) inflammatory bowel disease chronic diarrheal state

# Hypocitraturic calcium nephrolithiasis

- Citrate = inhibitor Inc metabolic demands on the mitochondria of renal cells  dec. citrate intracellular metabolic acidosis fasting bacteria during a urinary tract infection.

# NONCALCIUM CALCULI

- 1. Struvite— compose of MAP Women Proteus,Pseudomonas, Providencia, Klebsiella, Staphylococci, and Mycoplasma. pH (>7.19) MAP crystals precipitate.

- 2. Uric acid— Men Gout, rapid weight loss Elevated uric acid levels are frequently due to dehydration and excessive purine intake

- 3. Cystine— Inborn error of metabolism: abnormal intestinal (small bowel) mucosal absorption renal tubular absorption of dibasic amino acids, cystine, ornithine, lysine, and arginine. chromosome 2p.16 recently to 19q13.1

# NONCALCIUM CALCULI

- 4. Xanthine— Def. xanthine oxidase. catalyzes the oxidation of hypoxanthine  xanthine  uric acid

- 5. Indinavir— most common protease inhibitor that results in radiolucent stones tannish red Indinavir calculi are the only urinary stones to be radiolucent on noncontrast CT scans.

# THANK YOU FOR ATTENTION