NEPHROLITHIASIS

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Epidemiology

- Incidence 1:1000 per year
- Peak onset 20 - 35 years of age
- Male:Female 3 - 4 : 1
- In their lifetime
  - 2 - 5% of the Asian population
  - 8 - 15% of North Americans and Europeans
  - 20% of Saudi Arabians will develop a kidney stone

Epidemiology

- Soucie et al performed a cross sectional study to investigate the geographic variability in the rates of stone formation
- 1,167,009 men and women in the U.S Stones were 2x as prevalent in the southeast
- Ambient temps and sunlight indices were independent predictors of stone formation

Epidemiology

- Curhan et al studied the influence of FH on stone formation
- 17.2% of men vs. 6.4% + family history
- 1986 - 1994 : 795 incident cases of stones
- RR of stone formation in men with +FH 2.57 (95% CI 2.19 - 3.02)

Epidemiology

- Serio and Fraioli confirmed hereditary predisposition
- 22.5% of patients who developed stones in Italy between 1993 - 1994 had a positive FH in one or both of their parents

nephrolithiasis

- Nephrolithiasis is a common disorder, affecting ~10% of individuals in Western countries
- a recurrence rate of 50% at 5–10 years
- requires the related frequent need for urological treatments and it is a significant cause of morbidity.
- Although ‘common’ forms of calcium oxalate nephrolithiasis and idiopathic hypercalciuria are complex polygenic disorders, with several genes contributing to their pathogenesis in as high as 50% of cases

Family-based or case-control studies

Single-candidate genes evidenced the possible involvement of gene polymorphisms in stone formation :

- **Calcium-sensing receptor (CASR)**
- **Vitamin D receptor (VDR),**
- **Osteopontin (OPN)**
- **Claudin 14 (CLDN14)**

gene as a possible major gene of nephrolithiasis.

Specific phenotypes were related with these genes

CASR gene in normocitraturic patients,

VDR gene in hypocitraturic patients with severe clinical course,

CLDN14 gene in hypercalciuric patients.

The pathogenetic weight of these genes remains unclear, but an alteration of their expression may occur in stone formers.

- (Kidney International :2011

Table 2 | Hereditary diseases causing nephrolithiasis and/or nephrocalcinosis

Gene	Locus/ inheritance mode	Protein	Cellular defect	Tubular defect	Disorder	Ref.
CLCN5	Xp11.22 X-linked recessive	Chloride channel 5 on the endosome membrane	Impaired acidification of the endosome fluid in proximal tubular cells.	Multiple reabsorption defects in the proximal tubule. Stones, nephrocalcinosis and possible end-stage renal failure.	Dent's syndrome	Cho *et al.*[6] and Scheinman *et al.*[7]
OCRL1	Xq26.1 X-linked recessive	Phosphatidylinositol 4,5-bisphosphate 5-phosphatase	Accumulation of phosphatidylinositol 4,5-bisphosphate in proximal cells, followed by actin polymerization, and tight and aderens junction defects.	Multiple reabsorption defects in the proximal tubule. Stones, nephrocalcinosis and possible end-stage renal failure. Hydrophthalmia, cataract, mental retardation.	Dent's syndrome 2 Lowe syndrome	Cho *et al.*[6]
CLDN16	3q27 Autosomal dominant	Claudin 16	Alteration of tight junction ion selectivity in the thick ascending limb of Henle loop.	Urinary loss of magnesium and calcium, nephrocalcinosis, and progressive kidney failure in homozygotes. Heterozygotes may produce kidney stones.	Familial hypomagnesemia with hypercalciuria and nephrocalcinosis	Muller *et al.*[8]
CLDN19	1p34.2 Autosomal dominant	Claudin 19	Alteration of tight junction ion selectivity in the thick ascending limb of Henle loop.	Renal wasting of magnesium and calcium, nephrocalcinosis and progressive kidney failure in homozygotes. Macular colobomata, myopia, and nystagmus.	Familial hypomagnesemia with hypercalciuria and nephrocalcinosis with ocular impairment	Konrad *et al.*[9]
ATP6N1B	7q33-q34 Autosomal recessive	β-Subunit ATP6N1B of the H-pump	Defect of the proton secretion and urine acidification in the α-intercalated cells of the collecting duct.	Hypokalemic hyperchloremic acidosis with nephrocalcinosis and kidney stones.	Distal tubular acidosis	Smith *et al.*[10]
ATP6B1	2cen-q13 Autosomal recessive	Subunit ATP6B1 of the H-pump	Defect of the proton secretion and urine acidification in the α-intercalated cells of the collecting duct.	Hypokalemic hyperchloremic acidosis with nephrocalcinosis and kidney stones. Neural deafness.	Distal tubular acidosis with progressive neural deafness	Karet *et al.*[11]
SLC4A1	17q21-q22 Autosomal dominant	Anion exchanger	Decreased bicarbonate reabsorption at the basolateral membrane of the α-intercalated cells of the collecting duct.	Hypokalemic hyperchloremic acidosis, nephrocalcinosis and kidney stones. Kidney stones and incomplete tubular acidosis in heterozygotes.	Distal tubular acidosis	Bruce *et al.*[12]
SLC34A3	9q34 Autosomal recessive	NPT2c sodium-phosphate cotransporter	Reduced phosphate reabsorption and increased calcitriol synthesis in the proximal tubular cells.	Severe rickets and kidney stones caused by renal loss of phosphate, hypophosphatemia, and hypercalciuria.	Hypophosphatemic rickets with hypercalciuria	Bergwitz *et al.*[13] and Tencza *et al.*[14]
CASR	3q13.3-q21 Autosomal dominant	Calcium-sensing receptor (activating mutations)	Inhibition of calcium reabsorption in the ascending limb of Henle loop.	Hypercalciuria and hypocalcemia. Hyperphosphatemia and hypophosphaturia. Renal hypopotassemia if very potent effect of the mutation.	Autosomal-dominant hypoparathyroidism Bartter syndrome type 5	Pearce *et al.*[15]
SLC12A1	15q15-q21.1 Autosomal recessive	NKCC2 sodium-potassium-chloride transporter	Decreased sodium, potassium and chloride reabsorption in the ascending limb of Henle loop.	Renal hypokalemia, alkalosis, hypercalciuria, secondary aldosteronism and nephrocalcinosis.	Bartter syndrome type 1	Puricelli *et al.*[16]
KCNJ1	11q24 Autosomal recessive	ROMK1 potassium channel	Decreased sodium, potassium and chloride reabsorption in the ascending limb of Henle loop.	Renal hypokalemia, alkalosis, hypercalciuria, secondary aldosteronism and nephrocalcinosis.	Bartter syndrome type 2	Puricelli *et al.*[16]

Gene mutations cause phenotypic alterations at protein, cellular, and body level that are described in the table. All these mutations cause a protein loss-of-function with the exception of *CASR* gene mutations that cause nephrolithiasis in the presence of activating mutations.

Natural History Recurrence Rates

- 40 % in 2 - 3 years
- 55% in 5 - 7 years
- 75% in 7 - 10 years
- 100% in 15 - 20 years

Stone Formation

Urine saturation

Supersaturation

Crystal nucleation

Aggregation

Retention and growth

Saturation

- Saturation level - specific concentration of a salt = solubility product and no more salt can be dissolved
- Crystals form in supersaturated urine
- Saturation is dependent on chemical free ion activities of the components of a stone

Chemical free ion activities

- concentration of the relevant ions
- urine pH
- complexation with substances in the urine

Nucleation

- Homogeneous - supersaturation reaches a formation product and nuclei form in free solution
- Heterogeneous - crystal growth occurs on the surface of dissimilar but complimentary crystal or foreign substances

Clinical Presentation

- Renal colic begins suddenly and intensifies over 15 - 30 minutes
- Associated with nausea & vomiting
- Pain passes from the flank anteriorly to the groin
- At the ureterovesicular junction, urinary frequency and dysuria may occur
- Microscopic hematuria> 75%, gross - 18%

Patient Evaluation: Basic Single Stone Former

- Stone history
- Medical disease: skeletal disease, IBD, UTI's, HIV, granulomatous disease
- Meds: Lasix, glucocorticoids, theophylline, calcium, vitamins A, C, and D
- Family history
- Lifestyle/occupation
- Diet/fluids: protein, coffee, tea, dairy

Patient Evaluation: Basic Single Stone Former

- Physical Examination
- Laboratory data
  - Urinalysis, urine for cystine
  - Urine culture -
  - Blood tests
    - electrolytes, creatinine, calcium, phosphorous, uric acid, intact PTH if calcium elevated

Patient Evaluation: Complete

- All patients with "metabolically active" stones, children, people in demographic groups that don't usually form stones
- Metabolically active stone: grow in size, number, or are passed within 1 year of f/up
- Basic evaluation and 24 hour urine for volume, calcium, oxalate, sodium, phosphorous, uric acid, citrate, cystine

Patient Evaluation: Complete

- Optimal values of 24-hr urine constituents:
  - volume > 2 - 2.5 L/day
  - calcium < 4 mg/kg or < 300 mg Γ,< 250 mg E
  - uric acid < 800 mg Γ, < 750 mg E
  - citrate > 320 mg
  - sodium < 200 meq
  - phosphorous < 1100 mg
  - pH > 5.5 and < 7.0

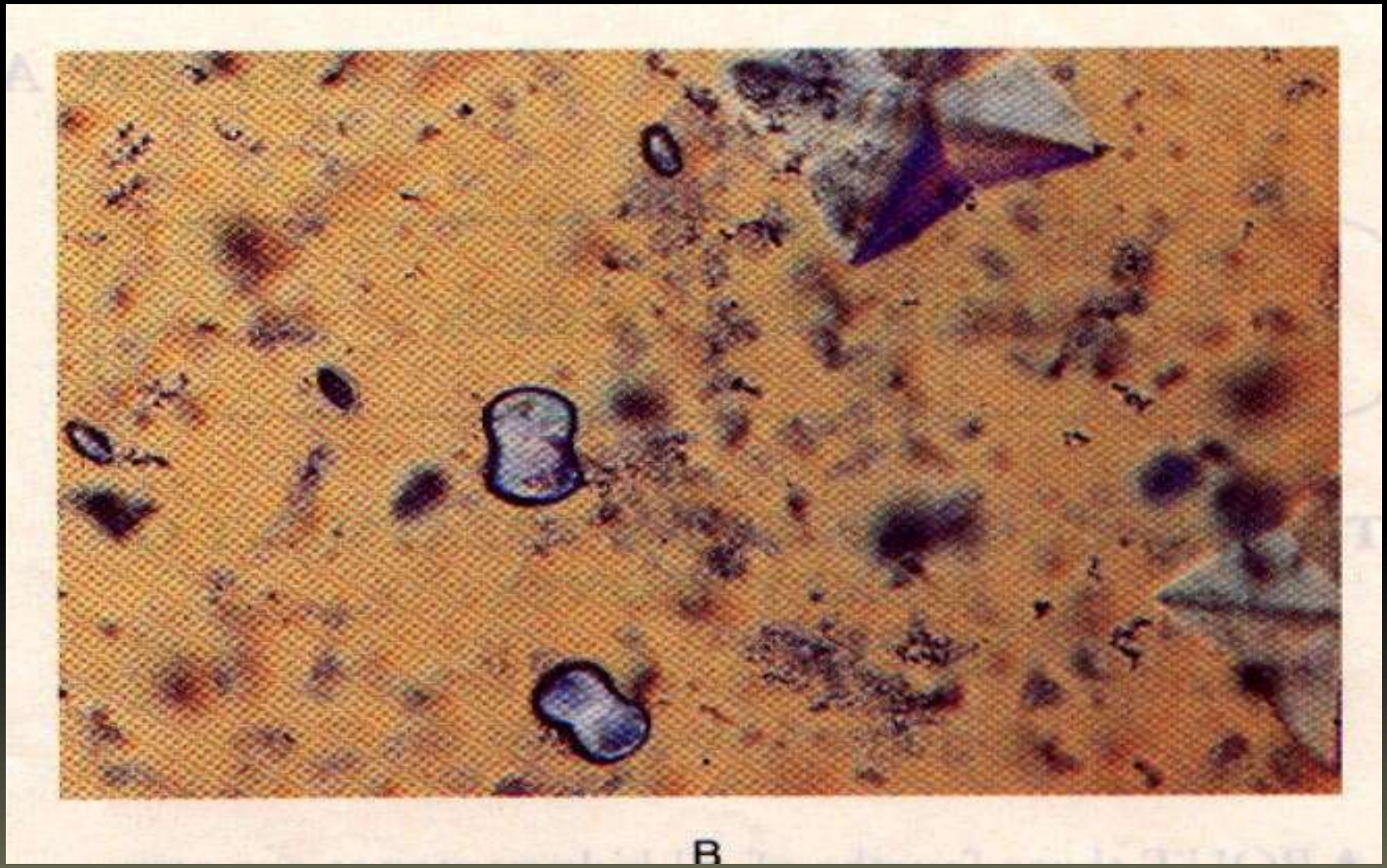
Etiology

Calcium oxalate & calcium phosphate	37%
Calcium oxalate	26%
Calcium phosphate	7%
Uric Acid	5 - 10 %
Struvite	10 - 20%
Cystine	2%

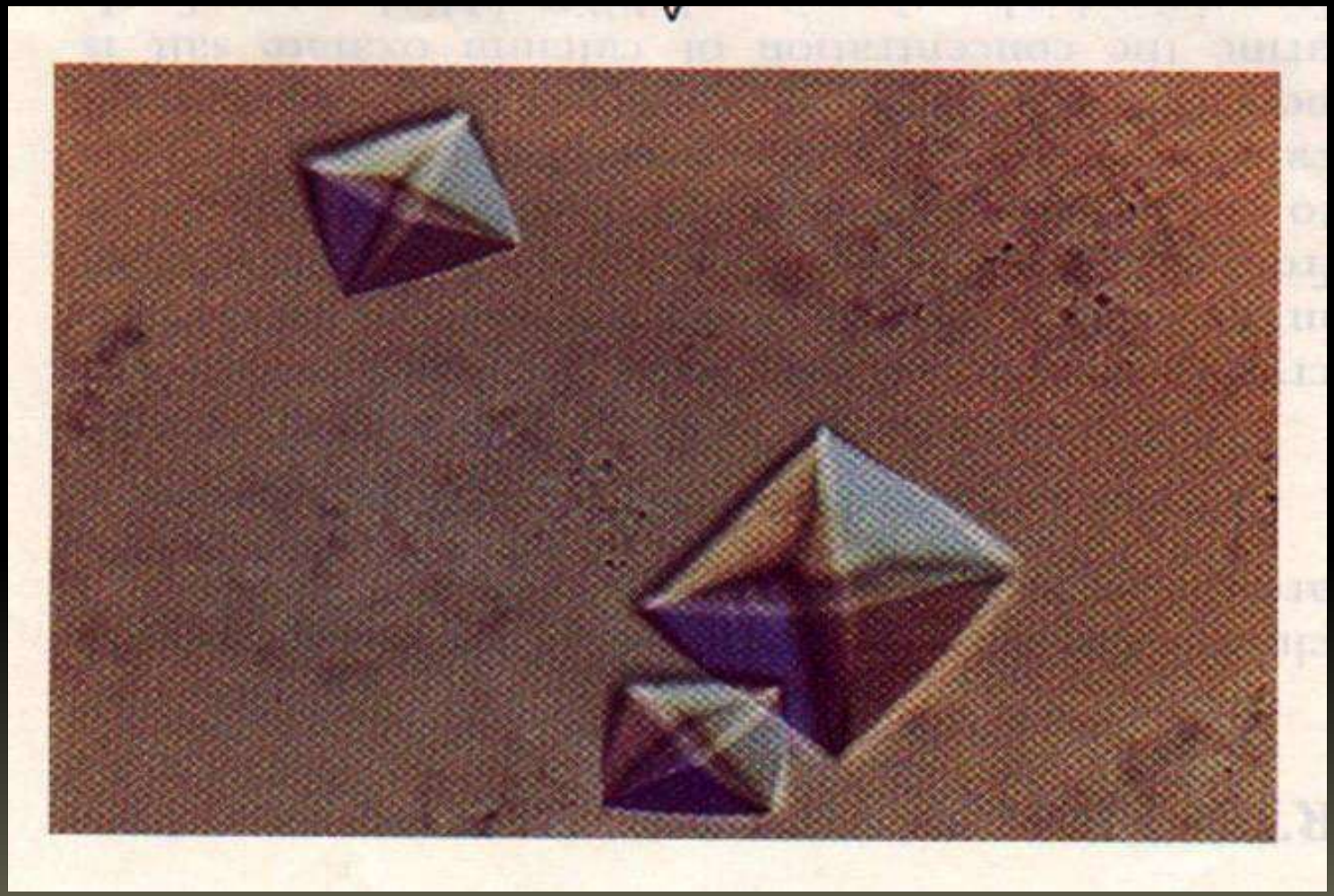
Calcium Stones

- 70 - 75% of all stones
- Calcium oxalate - brown, gray, or tan
- Calcium oxalate monohydrate - dumbbell
- Calcium oxalate dihydrate - pyramidal
- Calcium phosphate - white or beige
- Calcium phosphate - elongated (brushite)

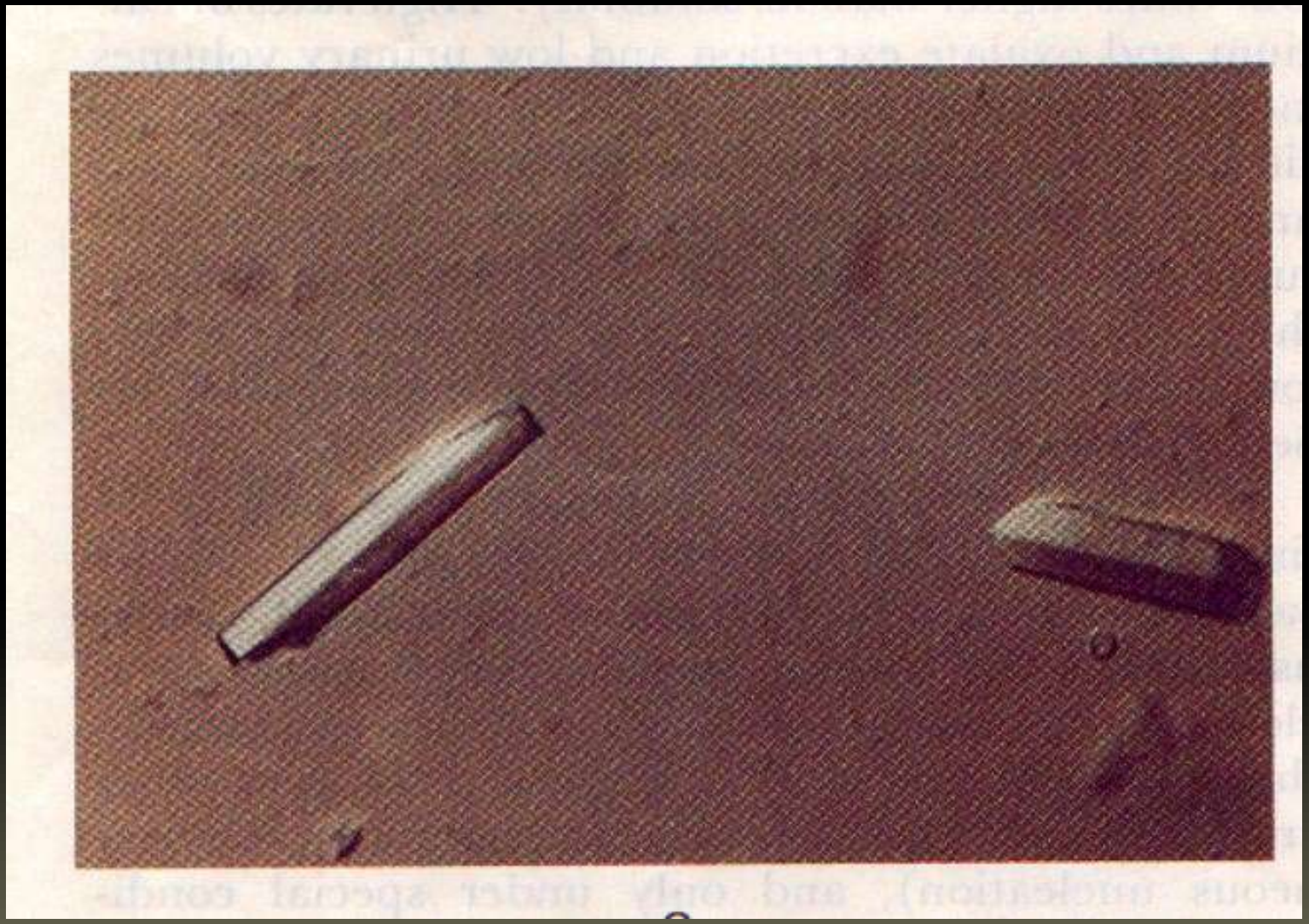
Calcium oxalate monohydrate



Calcium oxalate dihydrate



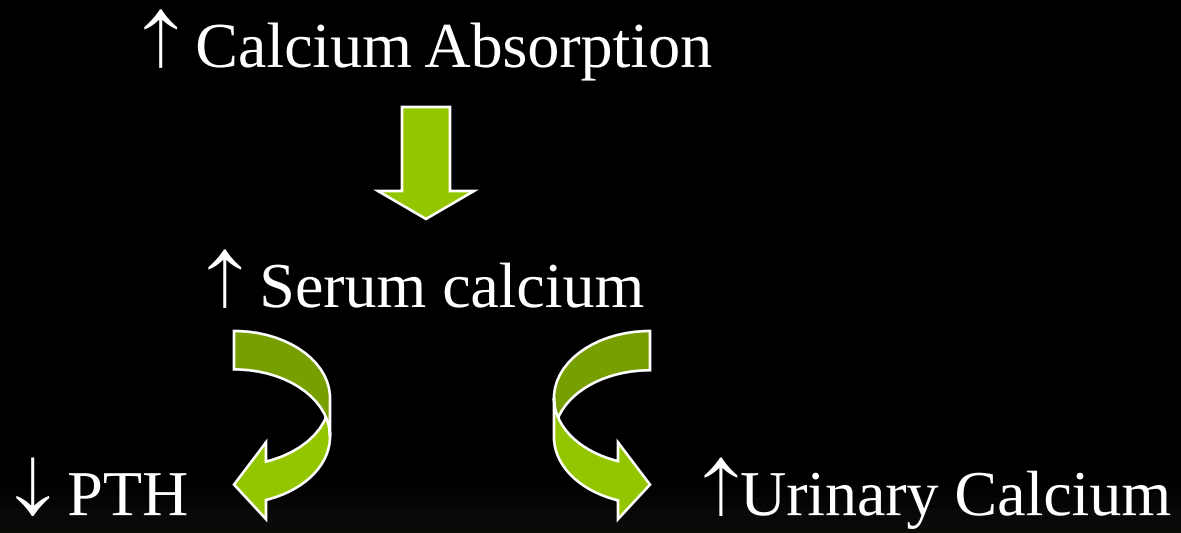
Calcium phosphate brushite



Calcium Stones Pathophysiology

Hypercalciuria	40 - 50%
Hypocitraturia	20 - 30%
Hyperoxaluria	5%
Unclassified	25%

Calcium Stones
Hypercalciuria - Absorptive



Calcium Stones
Hypercalciuria - Absorptive

- Urine calcium exceeds 250 mg/day in females, 300 mg/day in males
- Most common cause of hypercalciuria
- familial, 50% of 1st degree relatives, M=F
- Patients have a higher incidence of reduced bone mineral density
- Ca++oxalate & Ca++phosphate stones

Calcium Stones
Hypercalciuria - Absorptive

- Pak et al have proposed two subtypes
- Type I - hypercalciuria on random and low calcium diets
- Type II - normocalciuria on restricted diets
- Etiology unknown
- Some patients have high serum calcitriol

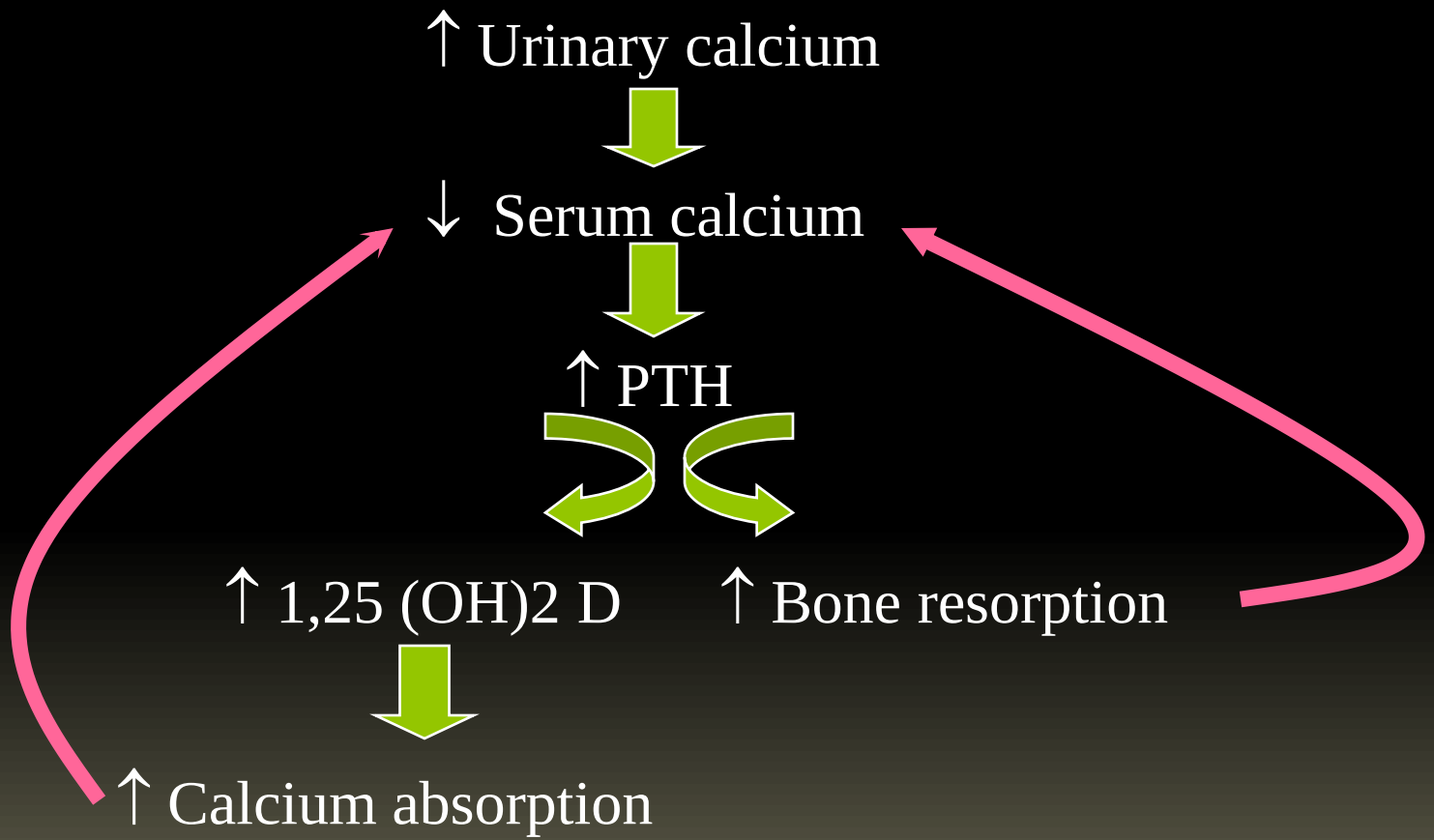
Rx: Calcium Stones Idiopathic Hypercalciuria

- Maintain urine volume > 2 liters/day
- Thiazide diuretics
- Potassium citrate
- Sodium restriction (2 - 3 grams/day)
- Protein restriction (0.8 - 1.0 g/kg/day)

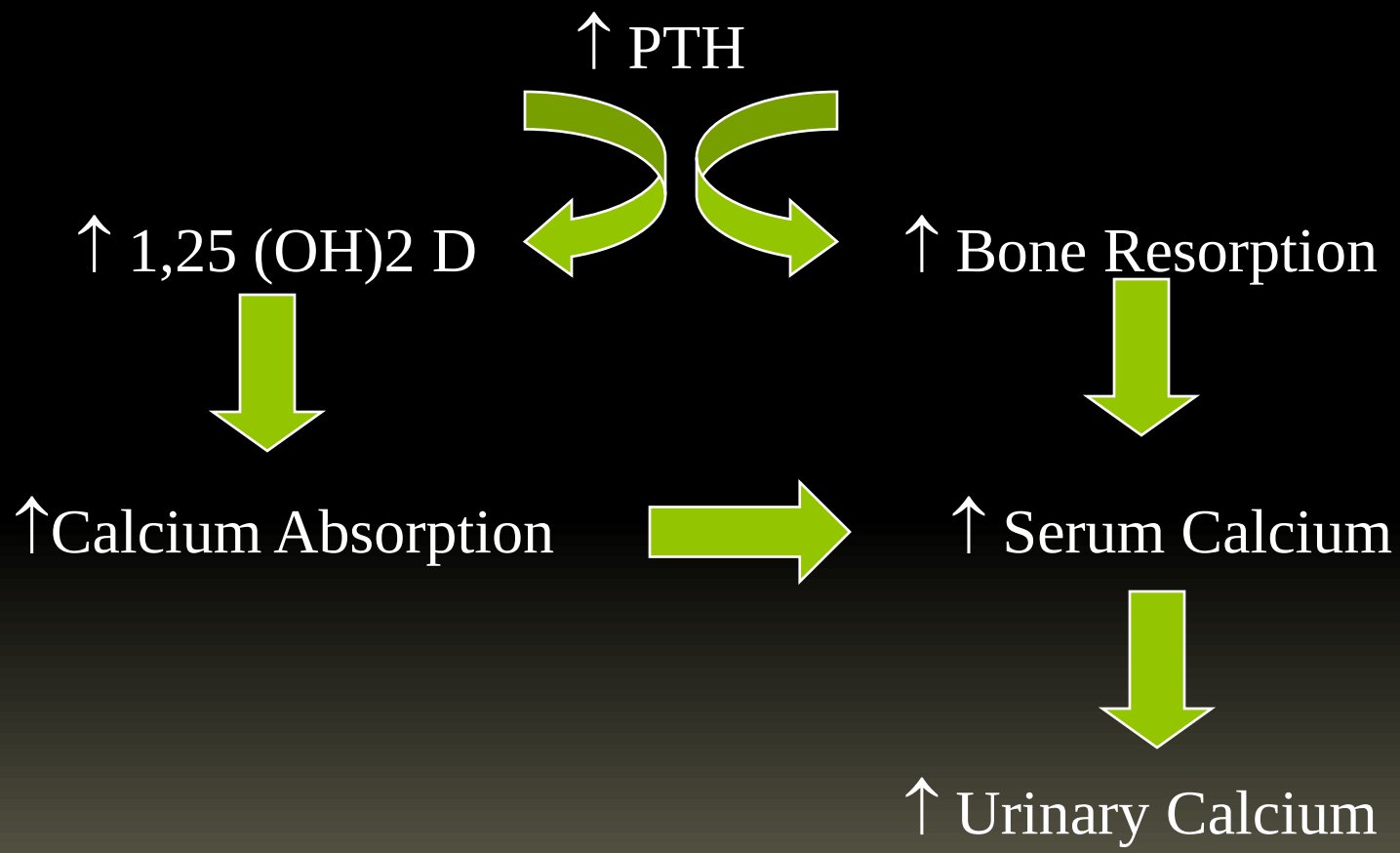
Hypercalciuria
Dent's Disease

- Rare, X-linked inheritance
- Tubular defect which causes renal phosphate wasting(PO_4 <2.9 mg/dl)
- Stimulates vitamin D, ↑ intestinal calcium absorption, ↑ urinary calcium
- Rx: Phosphorus replacement

Calcium Stones
Renal leak



Calcium Stones
Hypercalciuria - Resorptive



Calcium Stones Hyperparathyroidism

- 85% adenoma, 15% multigland hyperplasia
- Urine pH tends to be higher than in idiopathic hypercalciuria so the fraction of calcium phosphate stones is higher
- Rx: Parathyroidectomy

Calcium Stones
Hypocitraturia

- Occurs alone (10%) or with other abnormalities (50%)
- More common in females
- May be idiopathic or secondary
- Acidosis reduces urinary citrate by ↑ tubular reabsorption

Calcium Stones
Hypocitraturia

- Associated with distal RTA, metabolic acidosis of diarrhea, & consumption of a diet rich in meat
- Tend to form calcium oxalate stones (except Type I RTA)
- Rx: ↓ dietary protein, alkali (K citrate or K bicarbonate), avoid sodium bicarbonate

Calcium Stones
Renal Tubular Acidosis

- 2/3rds of patients with Type I RTA have nephrocalcinosis or nephrolithiasis or both
- Mechanism for stone formation
  - Hypocitraturia due to acidosis
  - Hypercalciuria: acidosis ↑ bone resorption
  - Alkaline urine pH: defect in H+ excretion

Calcium Stones
Renal Tubular Acidosis

- Form calcium phosphorus stones largely due to the alkaline urine pH which decreases the solubility of calcium phosphate complexes
- Rx: Alkali (↓calciuria, ↑ citraturia), thiazides if hypercalciuria persists

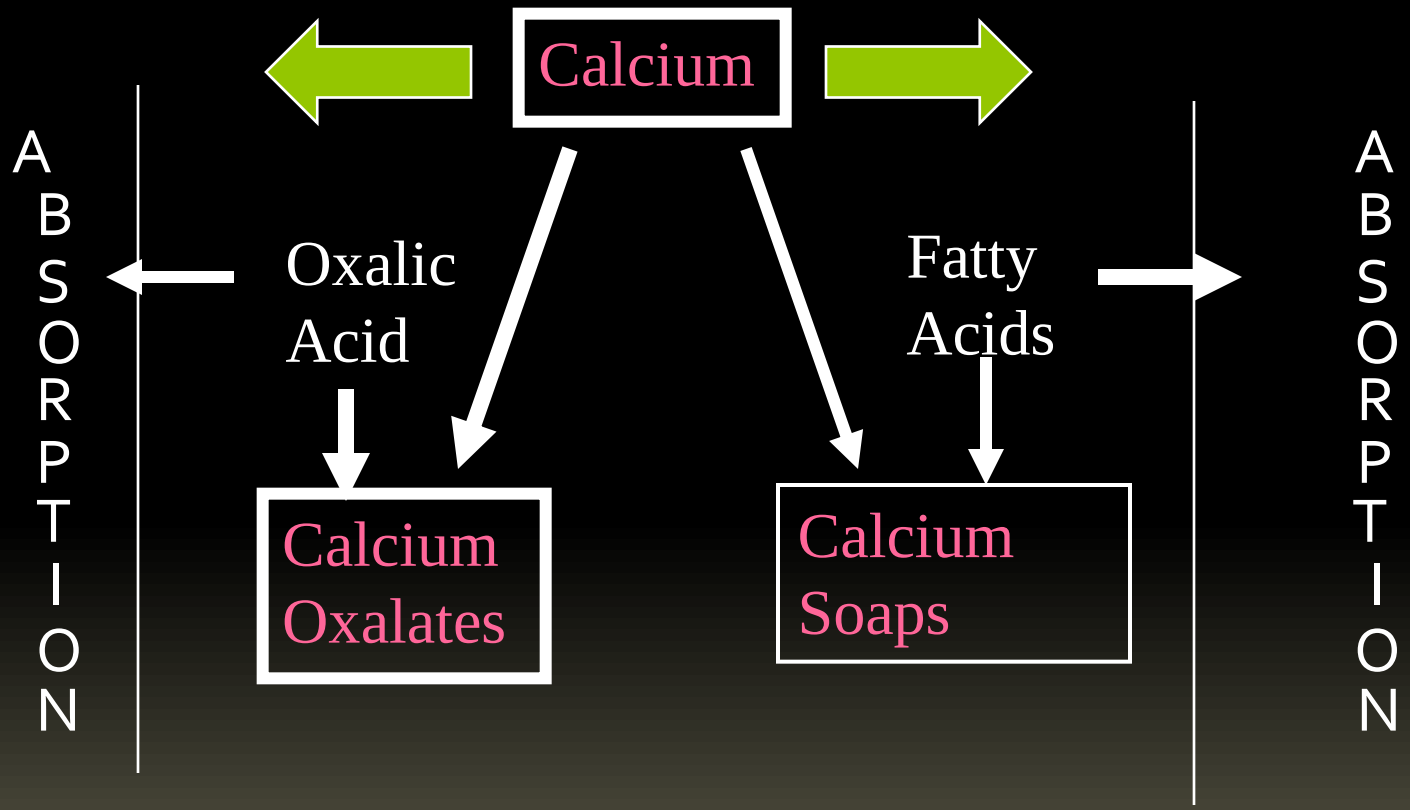
Calcium Stones
Hyperoxaluria

- Most patients with calcium oxalate stones excrete normal urinary oxalate: <40 mg/day
- Excessive urinary oxalate
  - Dietary hyperoxaluria
  - Endogenous hyperoxaluria
  - Enteric hyperoxaluria
- Normal people absorb < 5% of dietary oxalate
- Foods rich in oxalate (spinach, chocolate, beets, peanuts) can ↑ absorption 25 - 50%
- Low calcium diets ↑ urinary oxalate excretion

Calcium Stones
Endogenous hyperoxaluria

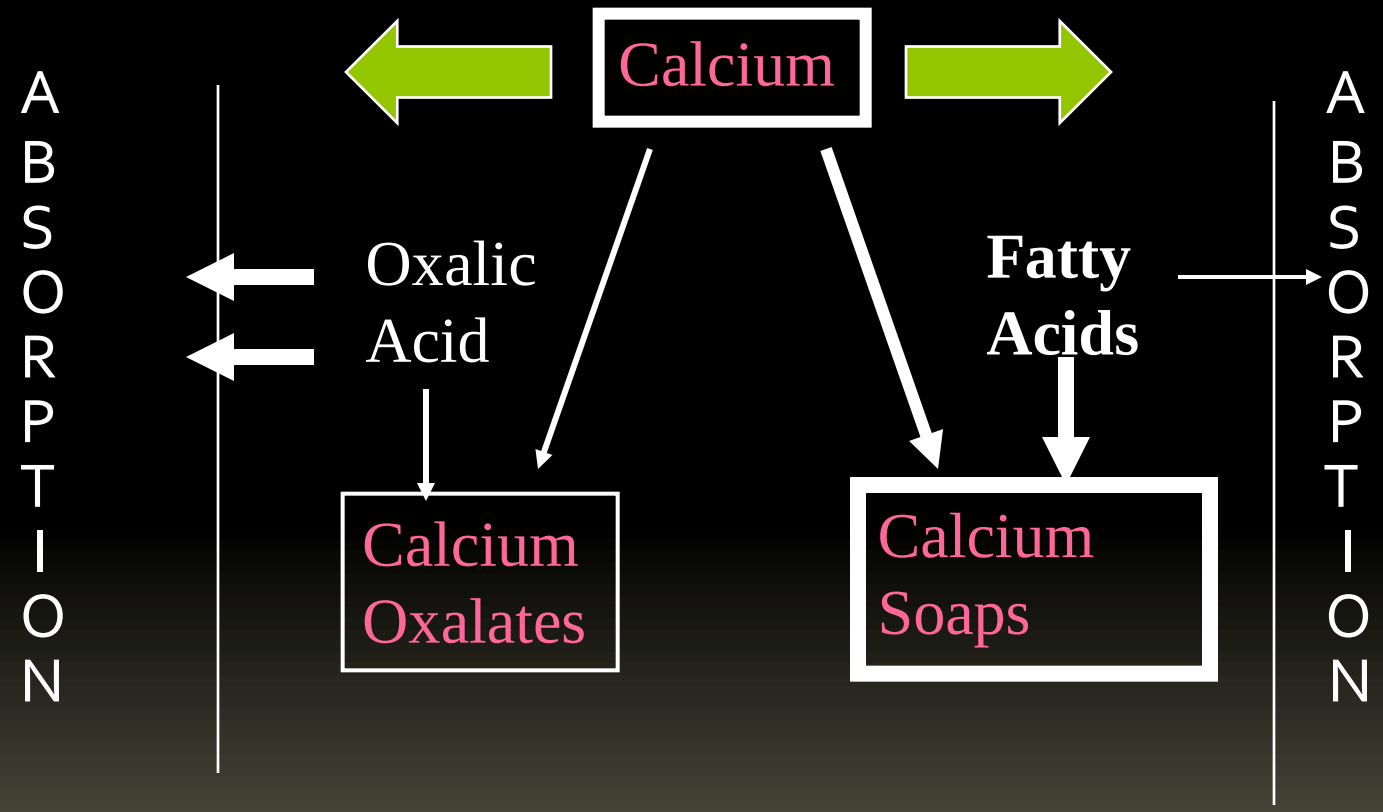
- Primary hyperoxaluria - enzymatic deficiencies that result in massive oxaluria
- Widespread calcium oxalate deposition in tissues (bone marrow, blood vessels, heart, and renal parenchyma)
- Rx: ↑ P.O. fluids, pyridoxine and orthophosphate ↓urinary crystallization

Calcium Stones
Enteric Hyperoxaluria



NORMAL

Calcium Stones
Enteric Hyperoxaluria



ENTERIC HYPEROXALURIA

Calcium Stones
Enteric Hyperoxaluria

- Treatment
  - Decrease dietary oxalate and fat
  - Oral Calcium supplements
  - Cholestyramine
  - Increase fluid intake
  - Oral citrate

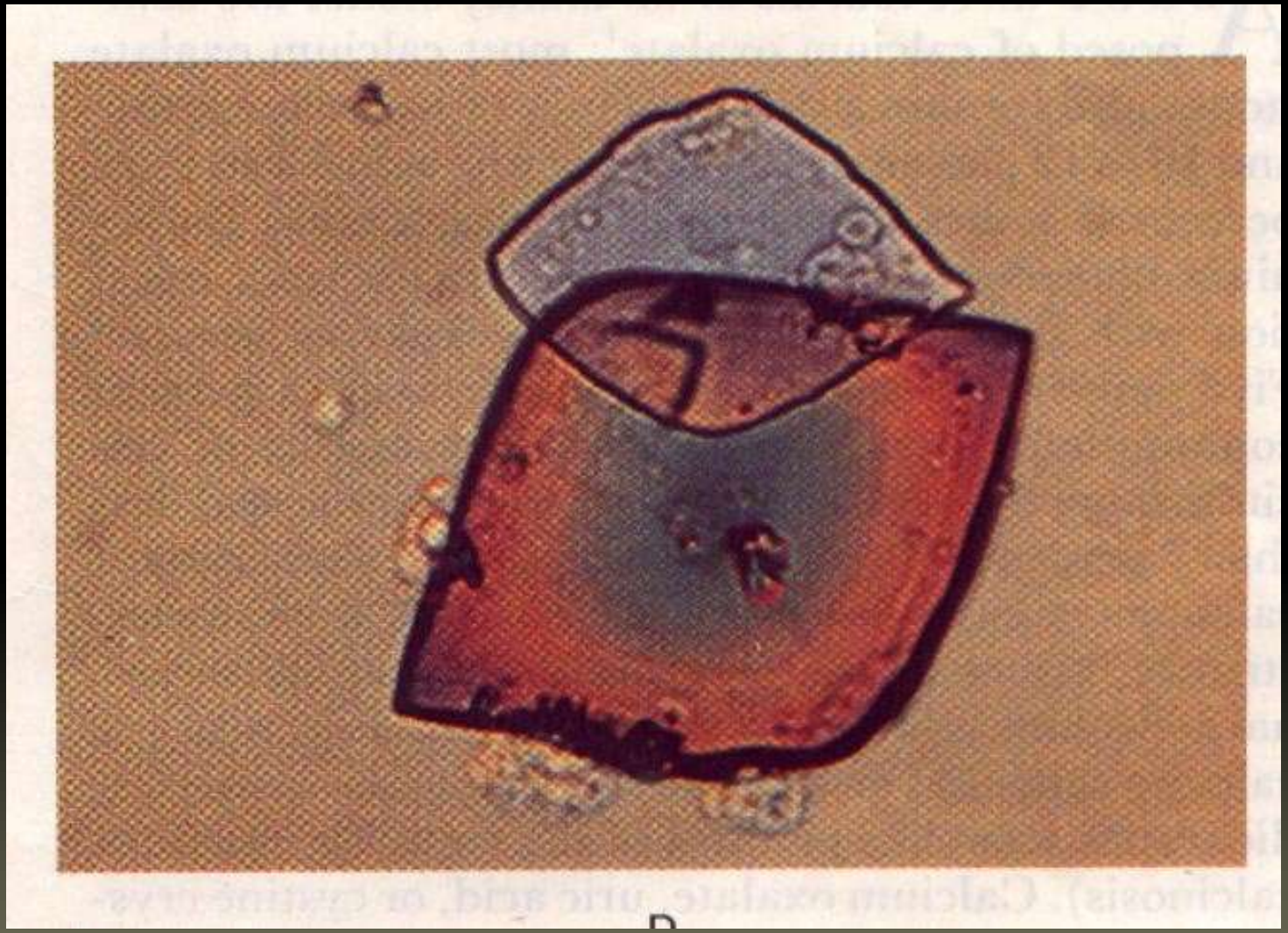
Calcium Stones Hyperuricosuria

- Increased frequency of hyperuricosuria in patients who form calcium stones
- Urate crystals serve as a nidus for calcium oxalate nucleation & comprise 4 - 8% of the cores of calcium stones
- Rx: Allopurinol, Potassium citrate

Uric Acid Stones

- Smooth, white or yellow-orange
- Radiolucent
- Crystals form various shapes: rhomboidal, needle like, rosettes, amorphous

Uric acid



Uric Acid Stones

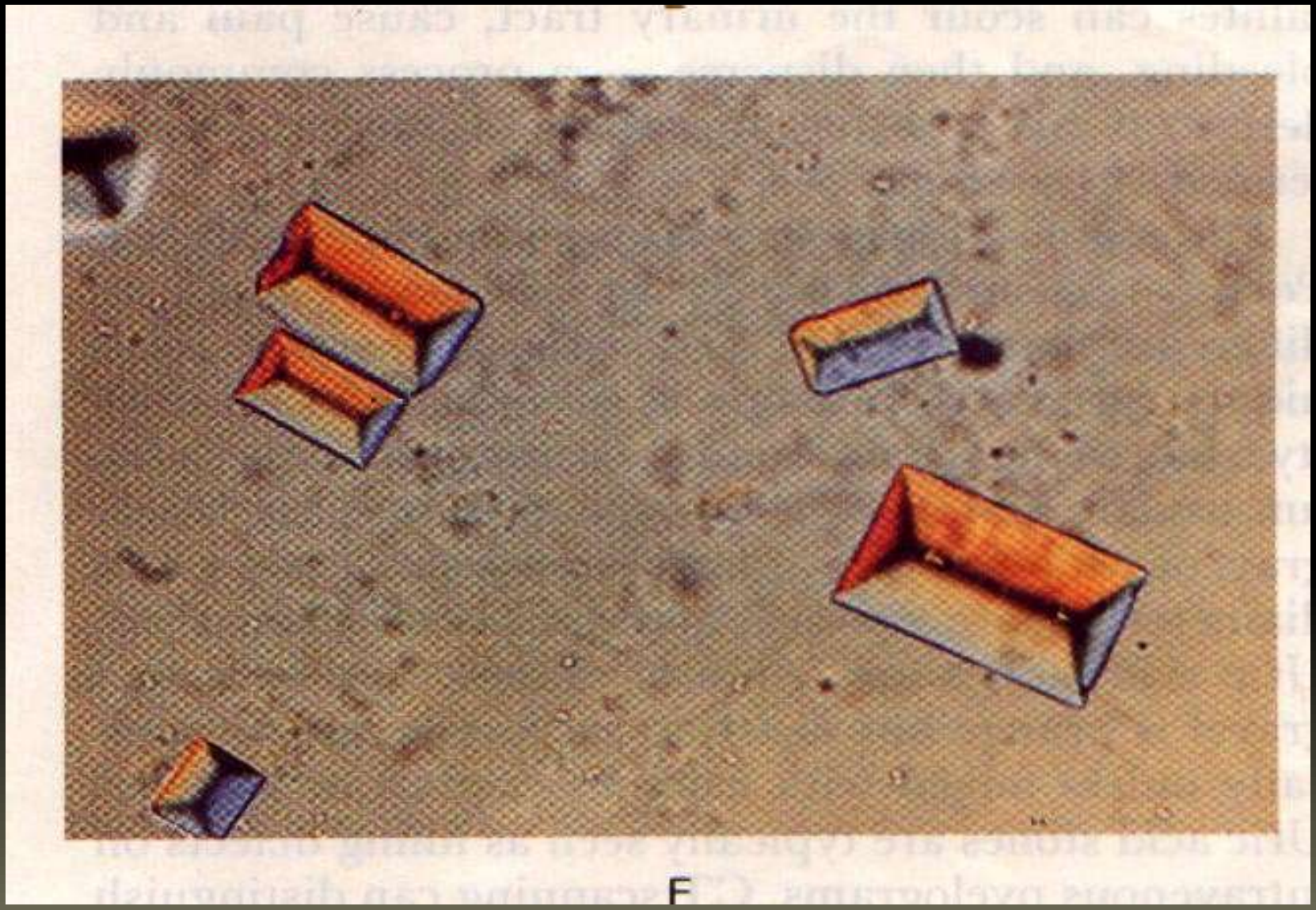
- Main determinants of uric acid stone formation
  - Urinary pH < 5.5, ↓ urine volume
  - Hyperuricosuria
    - Genetic overproduction
    - Myeloproliferative disorders
    - High purine diet
    - Drugs

    Rx: ↑ fluid intake, ↓ purine diet, urinary alkalinization (pH 6.5 - 7.0) with potassium citrate , allopurinol to reduce 24 hour uric acid excretion

Struvite Stones - Magnesium Ammonium Phosphate

- More common in women than men
- Most common cause of staghorn calculi
- Grow rapidly, may lead to severe pyelonephritis or urosepsis and renal failure
- Light brown or off white
- Gnarled and laminated on X-ray

Struvite



Struvite Stones
Infection Stones

- Caused in part by infections by organisms with urease (Proteus, Klebsiella, Pseudomonas, and Serratia)
- Hydrolysis of urea yields ammonia & hydroxyl ions, consumes H+ & thus↑urine pH
- ↑ urine pH increases saturation of struvite
- Rx: Remove existing stones (harbor causative bacteria) with ESWL or PUL
- Prolonged antibiotics
- Acetohydroxamic acid (urease inhibitor) - use limited because of side effects

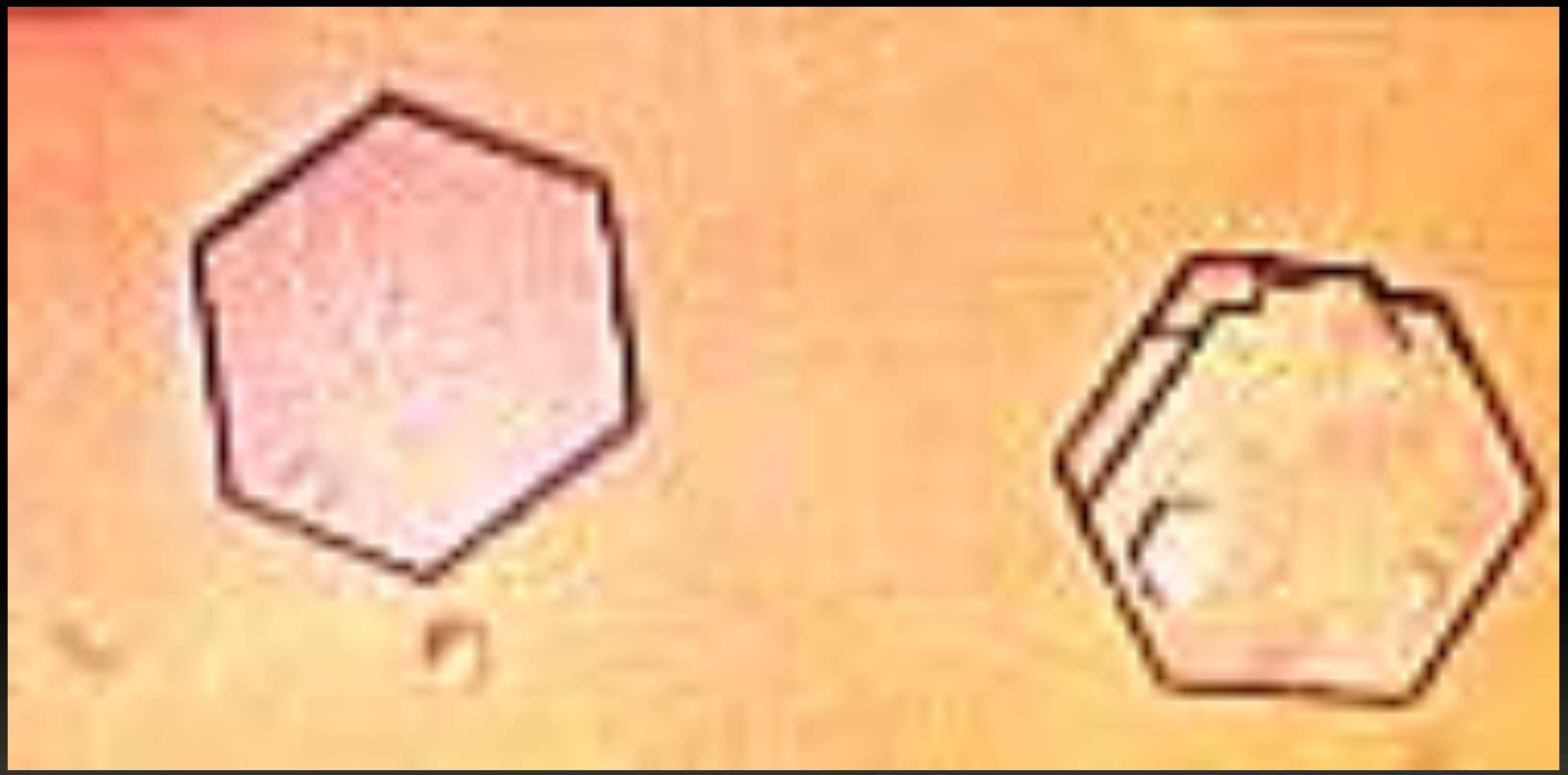
Cystine Stones

- Hereditary disorder caused by a tubular defect in dibasic amino acid transport, autosomal recessive
- Excrete excessive amounts of cystine, ornithine, lysine and arginine
- Cystine is soluble in the urine to a level of only 24 - 48 mg/dl
- In affected patients, the excretion is 480 - 3500 mg/day
- Nephrolithiasis usually occurs by the 4th decade

Cystine Stones

- Hexagonal, radiopaque, greenish-yellow
- Often present as staghorn calculi or multiple bilateral stones

Cystine



Cystine Stones

- Treatment
  - estimate urine volume to maintain solubility (240 - 480 mg/l)
  - urine pH > 7.5
  - Restrict dietary sodium (60 meq/d)
  - Troponin or D-penicillamine bind cystine and reduce urine supersaturation
  - Urological: removal difficult (2nd hardest) -PUL often required

Nephrolithiasis
Treatment - Calcium Stones

- *Peale, Roehborn and Pak performed a meta-analysis to determine the efficacy of different drug therapies for stone disease*
- 14 randomized, controlled trials, 6 drugs tx.
- Treatment arms: Thiazide diuretics (7), allopurinol (4), magnesium (2), alkali citrate (3), phosphate (3), non-thiazide diuretic (1)

Nephrolithiasis Treatment

- Significant reduction in stone recurrence was found in 5/7 of the thiazide trials and the indapamide trial
- Two remaining trials with no differences had mean follow up < 2 years
- The phosphate and the magnesium trials showed no treatment benefit
- Only 1/4 of the allopurinol studies showed a significant benefit
- In that study however patients were selected for hyperuricosuria and normocalcuria

Nephrolithiasis Treatment Alkali Citrate

- *Barcelo et al : K citrate vs. placebo in patients with hypocitraturia*

  Significant ↑ remission 72% vs. 20%

- Ettinger et al: K-Mg-citrate vs. placebo

  Significant ↑ remission 87% vs. 36%

- Hofbauer et al: Na-K-citrate Ø difference
- Meta-Analysis on 11/14 trials
- Risk difference of -22.6% for patient receiving treatment : 95% CI - 29.0 % to - 16 % , $P < 0.001$

Nephrolithiasis Treatment

- *Lee et al performed a retrospective analysis to assess the efficacy of K citrate based tx*
- 439 patients, 3 groups (regular prophylaxis, intermittent prophylaxis, no prophylaxis)
- Stone recurrence was significantly ↓ in group I (7.8% vs. 30% vs. 46%, $p<0.001$)

Nephrolithiasis Treatment - Non-specific

- *Curhan et al: prospective cohort study to assess whether ↑ dietary ca++ and supplemental calcium ↑ risk of stones*
- ↑ dietary calcium relative risk = 0.65 (p = 0.005, 95%CI, 0.50 - 0.83)
- Relative risk of supplemental calcium was 1.20 (p = 0.03)

Nephrolithiasis Treatment

- Sodium and sucrose intake were associated with an ↑ risk of stones
- Potassium and fluid were associated with ↓ risk of stones (RR 0.65 and 0.61 respectively)

Urological Treatment of Nephrolithiasis

- Extracorporeal shock wave lithotripsy (ESWL)
  - Complications
    - pain, steinstrasse (filling of the ureter with fragments of stone), bruising, perinephric hematoma, pancreatitis, urosepsis, ↑ BP
- Percutaneous Urolithotomy
- Ureteroscopic lithotripsy or extraction

Urological Treatment of Nephrolithiasis - Ureteral

- Most ureteral stones < 5mm pass spontaneously
- Stones ≥ 7mm in size have a poor chance of passing
- Stones in the distal ureter that stop progressing should be removed via ureteroscope or EWSL

Urological Treatment of Nephrolithiasis - Ureteral

- Stones in the proximal ureter that stop progressing should be pushed upward into the renal pelvis, then disrupted with EWSL
- If above fails, PUL

Urological Treatment of Nephrolithiasis - Renal Pelvis

- Stones that are < 2 cm and > 5 mm - EWSL
- Stones > 2 cm or exceed 1 cm and are in the lower poles require PUL

Principles of Stone Prevention

- prevent supersaturation
  - water! water and more water enough to make 2L of urine per day
  - prevent solute overload by low oxalate and moderate Ca intake and treatment of hypercalcuria
  - replace "solubilizers" i.e… citrate
  - manipulate pH in case of uric acid and cystine
- flush! forced water intake after any dehydration

Principles of Medical Management

- monitor stone burden with periodic kub
- instruct patient on adequate water consumption (enough to produce 2L of urine in 24 hrs.)
- instruct in low oxalate and modified calcium diet
- if hypercalcuric treat with hydrochlorothiazide (monitor urinary Ca)

Principles of Medical Management (2)

- if hyperuricosuric
  - allopurinol if serum uric acid elevated
  - alkalinize urine if serum level is normal
- if active Ca stone former not aided by diet, hctz add K citrate
- if magnesium ammonium phosphate stone after reduction of burden treat aggressively with antibiotics.

Metabolic Evaluation

- The first stone or infrequent (no problem for 10 years) no work up needed.
- more than one isolated stone event:
  - serum Ca, P, Uric Acid (repeat 2-3 times)
  - 24 hr urine for Ca. P, Uric Acid
  - serum parathormone if serum Ca is high
  - urine culture
- If above is normal then obtain
  - urine citrate, urine oxalate

Clinical Risk Factors

- occupation
- family history
- diet
- hydration
- small bowel disease (i.b.d.)
- medical conditions causing hypercalcuria
- medical conditions causing aciduria

Pathogenesis of Renal Stones (cont.)

- investigations show that the formation of a stone is similar to the development of a crystalline mass in vitro
- given that stone formation is an example of crystallization one could predict:
  - the necessity for a supersaturated state in urine
  - the occurrence of spontaneous crystallization
  - the need for the earliest polycrystalline state to be arrested in the u.t. allowing time for growth

Pathogenesis of Renal Stones (cont.)

- 99% of renal stones (in western hemisphere) are composed of:
  - calcium oxalate 75% (mono or di hydrate)
  - calcium hydroxyl phosphate (15%)(apatite)
  - magnesium ammonium phosphate 10% (struvite)
  - uric acid 5%
  - cystine 1%

Pathogenesis of Renal Stones

- all urinary stones are composed of 98% crystalline material and 2% mucoprotein
- the crystalline component(s) may be found "pure" or in combination with each other.
- the common characteristic that all crystalline components share, is that they have a very limited solubility in urine

prevention

Prevention

- Patient Education
  - Hydration
    - Drink 3 liters of fluid per day (14 cups)
    - Water
    - Lemonade (citrate decrease stone formation)
  - Diet
    - Low sodium
    - Watch amounts of oxalate
    - Low protein
  - Exercise/Increase Activity
- Medication

http://3.bp.blogspot.com/_-gcaht5yp_0/SdINrCVuqdI/AAAAAAAAAGw/xeEk4-F3z_I/s320/foods+rich+in+oxalate+2.gif

4 main types of kidney stones:

1. Calcium stones: They occur in 2 forms: calcium oxalate (the most common kidney stone) and calcium phosphate;
2. Uric acid stones form when the urine is acidic. A diet rich in purines increases uric acid in urine;
3. Struvite stones form in infected bladders or kidneys;
4. Cystine stones are rare and hereditary.

calcium oxalate stones

- Limit salt : Salt (sodium) increases the amount of calcium in urine. Limit sodium intake to less than 2300mg per day

  To reduce salt /sodium: Buy fresh foods, or foods without salt: fruit, fresh vegetables, fresh meats, chicken, fish, eggs, porridge, rice, and pasta.

- Most sodium (75%) comes from processed foods. Limit processed foods high in salt
- "Low salt" or "no added salt" foods are the best choices. Look for food with sodium content less than 150 mg per serve as a guideline
- Do not add salt at the table or in cooking

Include adequate calcium in diet

- When diet is very low in calcium, body will absorb more oxalate.
- Hence having sufficient calcium is important.
- Dietary calcium is important for your

  bones and teeth.
- Adequate dietary calcium intake of 1200mg per day is recommended.
- Include 3 choices daily from the following list: 250mL milk , 200mL high calcium milk (e.g. Physical); 200g yoghurt; 40g cheese (2 slices); 125mL (1/2 cup) ricotta cheese.

Include only a moderate amount of animal protein

- Limit protein from meat, seafood, fish, chicken, eggs to a moderate serve (120-150g) daily (cooked) (note 60g meat = 2 eggs).

Vitamin supplements

- Avoid vitamin C supplements (known as ascorbic acid, calcium ascorbate).
- Avoid Vitamin D supplements or cod liver oil
- Vitamin B supplements have not shown to be harmful to people with kidney stones

Choose whole meal and wholegrain foods

- Eating foods higher in fibre can decrease the risk of stone formation by reducing the amount of calcium and oxalate you absorb.
- Choose wholemeal and wholegrain breads, flour, cereals, pasta, biscuits and crackers.

uric acid stones

- 1. **Limit high purine foods:** Red meat, organ meats, gravies and meat extracts , Mackerel, anchovies, herring , hellfish
- 2. **Limit alcohol intake** Alcohol interferes with the removal of uric acid from your body.
- 3. **Limit sugar** sweetened food and drinks
- 4. **Increase fruit and vegetable intake** : Eating less animal-based protein and eating more fruits and vegetables will help decrease urine acidity which will help reduce the chance of stone formation