Kidney Stones

Biochemistry, 4th lecture

Renal Block
430
Biochemistry Team

What are kidney stones?

Renal calculi (kidney stones) are formed in renal tubules, ureter or bladder.

Composed of metabolic products present in glomerular filtrate

These products are in high conc.

Near or above maximum solubility

Conditions causing kidney stone formation:

1-High conc. of metabolic products in glomerular filtrate

2-Changes in urine pH

3-Urinary stagnation

4-Lack of normal inhibitors of stone formation in urine

1- High conc. of metabolic products in glomerular filtrate is due to:

↓urinary volume (with normal renal function)

Restricted fluid intake

↑fluid loss over a long period of time

↑rate of excretion of metabolic products forming stones

↑ plasma volume (that increases filtrate level)

↓ tubular reabsorption from filtrate

2-Change in urine pH is due to:

- Bacterial infection

-↑ precipitation of different salts at different pH:
A persistently **acidic** urine → promotes **uric acid** stones.

-A persistently **alkaline** urine (due to upper urinary tract infection)→ promotes **Mg Ammonium Phosphate** crystals (Struvite stones)

3- Urinary stagnation due to:

Obstruction of urinary flow

Stagnation: Abnormality in the flow.

4- Lack of stone-forming inhibitors:

Inhibitors of stone formation: e.g.:
Pyrophosphate
Glycoproteins

they Inhibit the growth of Ca^{++} oxalate crystals.

In type I renal tubular acidosis, hypocitraturia leads to renal stones.

Types of Kidney Stones:

1-Stones of calcium salts

2-Uric Acid stones

3-Magnesium ammonium phosphate stones

4-Cystine stones

Others (xanthine, etc)

1- Calcium salt stones:

80% of patients with nephrolithiasis form calcium stones:

Mostly: Ca-Oxalate

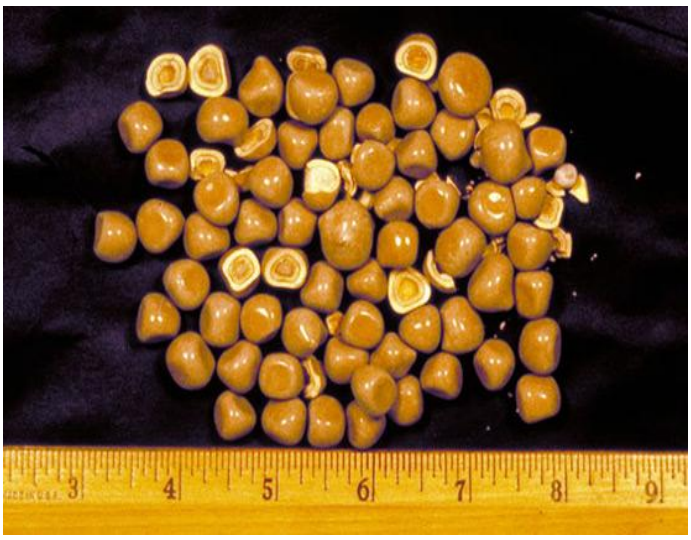
Less often: Ca-Phosphate

Characteristics or General appearance :

White, hard, radioopaque

Calcium PO_4: staghorn in renal pelvis (large)

Calcium oxalate: present in ureter (small)



The type of salt depends on:

1-Urine pH

2-Availability of oxalate

Causes of calcium salt stones:

1-Hypercalciuria :

Daily urinary Ca excretion >6.2 mmol in ♀ & >7.5 mmol in ♂

May or may not be due to hypercalcemia (most often due to 1^{ary} hyperparathyroism)

sometimes, Ca^{++} salts stones are found with no hypercalcemia

2-Hyperoxaluria:

induces formation of calcium oxalates (even with no hypercalciuria)

causes:

exogenous (diet rich in oxalate)
Increased oxalate absorption in fat malabsorption

Primary Hyperoxaluria due to:

inborn errors in childhood.
Urinary oxalate excretion: > 400 mmols/day

-Oxalate can be found in some vegetables, and in beans too.

Conservative lines of treatment:

Treatment of primary condition (i.e. Infection, hypercalcemia, hyperoxaluria)

Reducing oxalates in diet (it is not recommended to reduce calcium in diet)

↑ **Fluid intake** (if no glomerular failure)

Acidification of urine (by dietary changes)
because Calcium salt stones are formed in alkaline urine

> There's no signal drug to treat the disease, e.g. change the diet. (Treat the root)
>
> Acidification: by eating more meat (protein), and eating less vegetables and beans.

2- Uric acid stones.

8% of renal stones contains uric acid

may be associated with **hyperuricemia** (with or without clinical **gout**)

Form in acidic urine

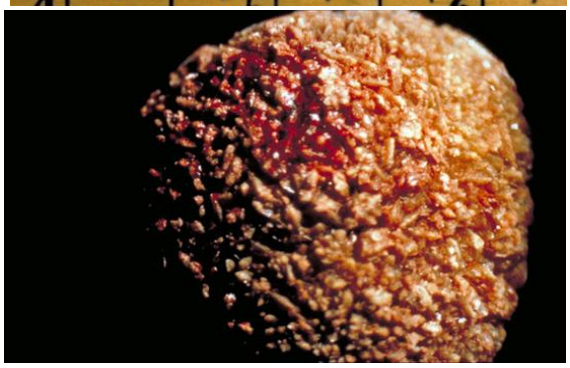
Characteristics or General appearance :

1-small, friable & yellowish

2-may form staghorn (if big)

3-radiolucent (can't be seen be plain X-ray)

4-visualized by ultasonography or I.V. Pyelogram.



Meat -> Tissue -> DNA -> Purine -> Uric acid (explanation of how the uric acid forms, we took it before in Biochemistry)

Friable means: Easily Broken.

We use Pyelogram I.V. because it's Radiolucent.

Treatment:

1-Purine-restricted diet

2-Alkalinization of urine (by dietary changes e.g. potassium citrate)

3-Increased fluid intake

3- Mg ammonium phosphate (struvite) stone.

Struvie = Mineral.
10% of all renal stones

It's associated with chronic urinary tract infection

Because microorganisms that metabolize urea into ammonia such as from Proteus genus (urease splitting oraganism)

it forms in Alkaline urine pH (> 7.0).

Treatment:

Aggressive prevention & treatment of the cause – (urinary tract infection)

Urine acidification –

↑ Fluid intake –

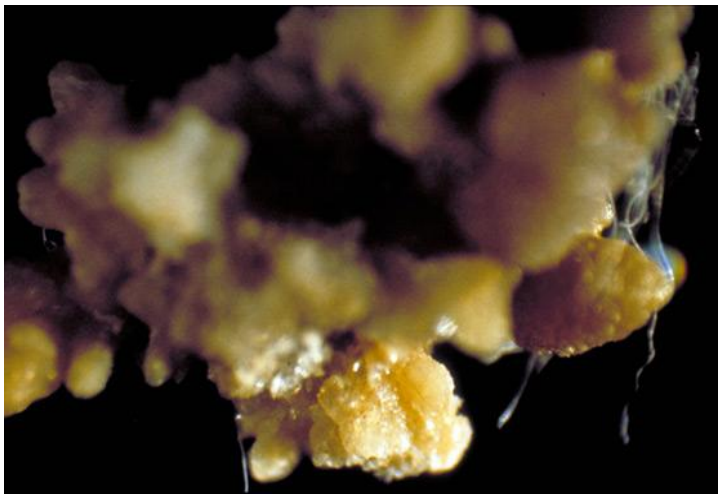
It may require complete stone removal (percutaneous nephrolithotomy) –

4- Cystine stones.

it's a rare type of kidney stone.

occurs in cases of homozygous cystinuria (inborn error of amino acid metabolism)

Soluble in alkaline urine (precipitates by acidic urine).



Treatment:

1-↑ fluid intake

2-Alkalinzation of urine (by dietary changes)

3-Penicillamine. (binds to cysteine to form a compound more soluble than cystine).

Laboratory investigations of kidney stones.

1-If stone has formed (available) and removed (with urine or by surgical intervention):

- Chemical analysis of stone helps to

  Identify the cause

  Advise patient on prevention and future recurrence

2- If stone has not formed (not available):

we will ask for :

- **Blood analysis**: calcium, uric acid, [PTH]

- **Urine analysis**: volume, calcium & oxalate

- **Urine pH**: > 8 suggestive of urinary tract infection (Mg amm. phosph.)

- **Screening of urine for cystine:** qualitative (if +ve: *24* hours urine)

- **Renal tract imaging:** CT, ultrasound & I.V. pyelogram

- there's no major cause for kidney stones, e.g. hypercalcemia may or may not cause it.
- kidney stones appear due to different factors. We could define the etiology by knowing the composition.

- PH is changed mainly by bacteria infection

- to treat the patient we have to change his diet
- ALL patients should increase fluid intake to > 2L/day

Do a complete evaluation in certain patients (those with moderate-high risk:

Middle-aged, White, Males, with + ve Family History

Patients with chronic diarrheal states and/or malabsorption, pathological fractures, osteoporosis, UTIs, or gout.

Patients with certain types of stones:

e.g. stones composed of calcium phosphate (hard stone) or struvite (@ risk for staghorn calculi).

thank you & good luck.