# UROLITHIASIS
# (Renal calculi, Stones)

# Urolithiasis

- Definition: formation of stones in renal collecting system from crystals contained in urine
- Common, seen in 5 per 1000 population
- Pathogenesis: Most are due to Idiopathic hypercalciuria
  - Metabolic disturbance
    - Hyperthyroidism (10%) and other causes of Hypercalcemia like tubular resorption defect
  - Environmental disturbance
- Renal stones are more common in USA than bladder stones

# Renal stones

- Calculus formation at any level of collecting system
- Most arise from kidneys
- Calcium Oxalate stones
- Mixed stones
- Uric acid, Cystine, Others

# Calculi form

- when there is increased excretion of solutes such as calcium

- when urine alkalinity, acidity, stasis, and/or concentration are favorable.

# Four main types of calculi

- (1) *most stones (about 70%) are calcium containing*, composed largely of calcium oxalate or calcium oxalate mixed with calcium phosphate;

- (2) another 15% are so-called *triple stones* or *struvite stones*, composed of magnesium ammonium phosphate;

- (3) *5% to 10% are uric acid stones*; and
- (4) *1% to 2% are made up of cystine*

- **CALCIUM (OXALATE or PHOSPHATE) 70%** → CA↑↑↑
- **MAGNESIUM AMMONIUM PHOSPHATE 20%** → Bact.
- **URIC ACID 10%** → U.A. ↑↑↑

# Risk factors

- Hypercalciuria in the absence of hypercalcemia
  - Most common metabolic abnormality
  - Due to increased gastrointestinal reabsorption of calcium
- Decreased urine volume concentrates the urine
- Reduced urine citrate
  - Citrate normally chelates calcium
- Primary hyperparathyroidism
- Diets high in dairy products (contain phosphate) or oxalates
- Urinary infections due to urease producers (e.g., *Proteus*)

# Renal Stones: Composition

| % | Composition | Mechanism of Formation |
|---|---|---|
| 75 | Calcium oxalate ± calcium phosphate | -Hypercalciuria with/without hypercalcemia<br>-Hyperoxalaturia (vegetarians)<br>-Hyperuricuria (uric acid crystals forms a core for calcium deposits) |
| 15 | Mg/$NH_4$ phosphate (triple or struvite) Usually accompanied by apatite $Ca_{10}(PO_4)_6$ | Alkaline urine due to UTI, e.g., *Proteus*, etc (infectious stones) |
| 5 | Uric acid | -Hyperuricosuria with/without hyperuricemia<br>-Acidic urine (pH<5.5) |
| 1 | Cystine | Hereditary cystinuria |

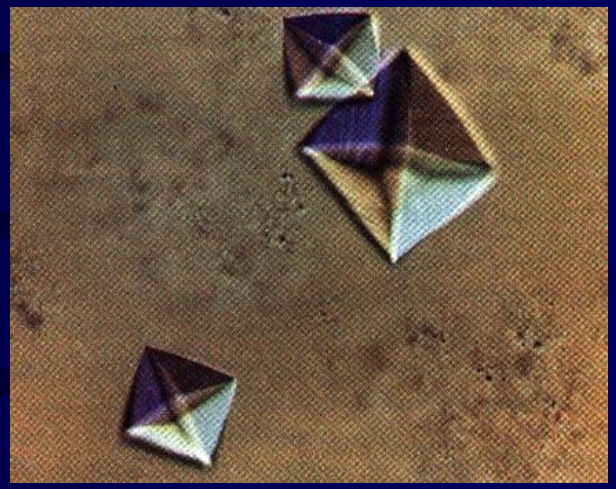
# Calcium Oxalate Crystals and Stones

- Most common stones
- ~40% patients will have hypercalciuria
- Idiopathic hypercalciuria accounts for most cases
- Secondary Calciuria may be seen
  - ~10% have hyperparathyroidism or hypervitaminosis D
  - ↑ Absorption from gut
  - Thyrotoxicosis and Cushing's Disease
  - Osteolytic tumors
  - Renal tubular Acidosis, Medullary sponge kidney
  - ↑ Vitamin D
  - Hyperuricuria
- Hypotheses for Pathogenesis
  1. Fatty acid defects in membranes
  2. Receptor defects

# Calcium Oxalate stones

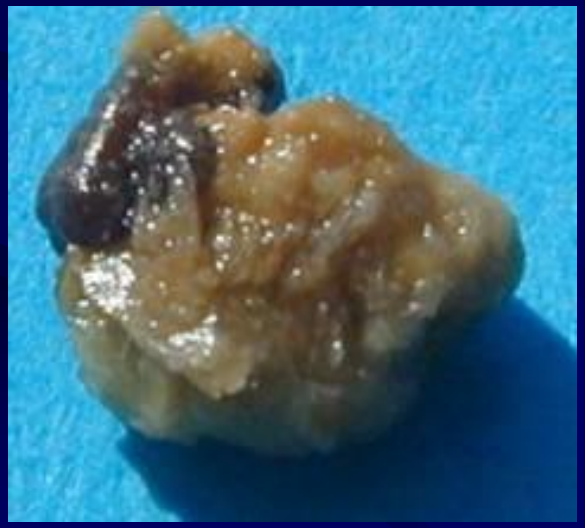
- 75% of all renal stones
- About half the time occur when there is hypercalciuria.
- Only about 10% of the time do they appear as a consequence of hypercalcemia.
- Increased incidence in pure vegans and Crohn's disease
- Supersaturation of urine with salts
- Radio-opaque

# Calcium Crystals and Stones



Calcium Oxalate

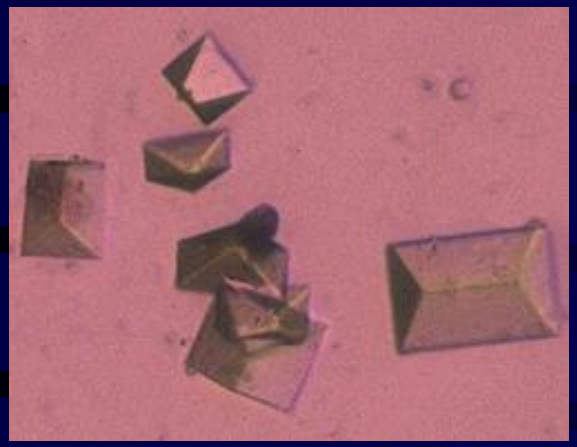
Calcium Phophate

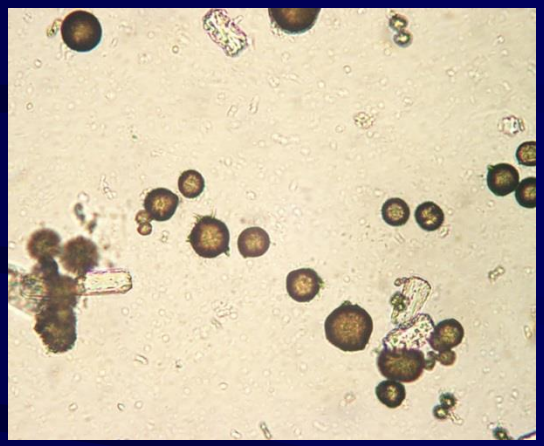


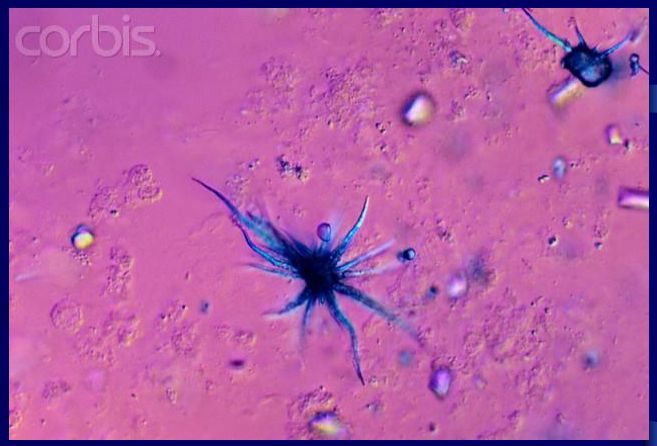
# Struvite Crystals and Stones

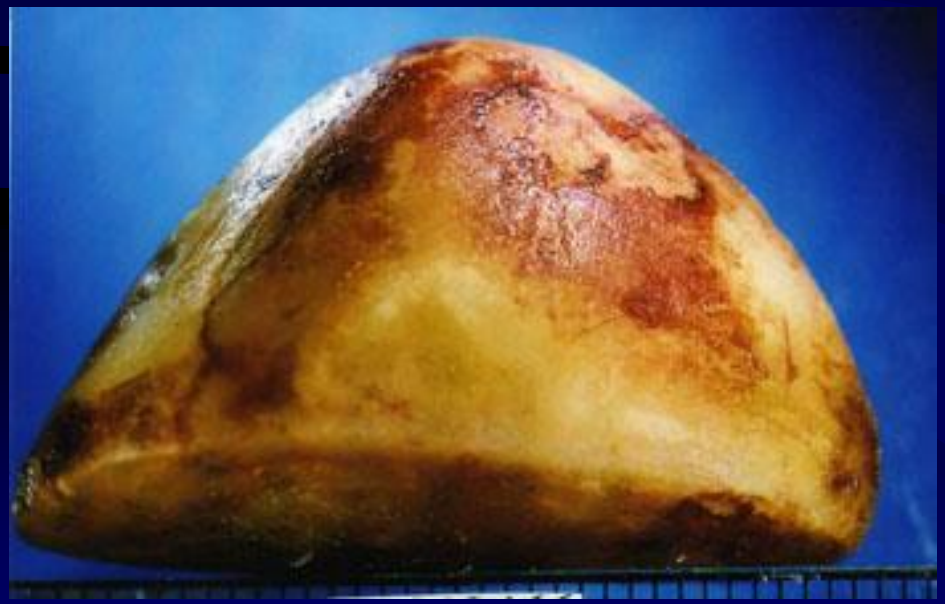
- Magnesium Ammonium Phosphate stones
- Associated with UTI esp *Proteus*
- *Proteus* breaks down urea to ammonia producing alkaline conditions
- Added sluggish flow of urinecauses precipitation in the pelvicalyceal system → Staghorn stones
- Over time these may cause
  - Hydronephrosis
  - Irritation →squamous metaplasia →squamous carcinoma

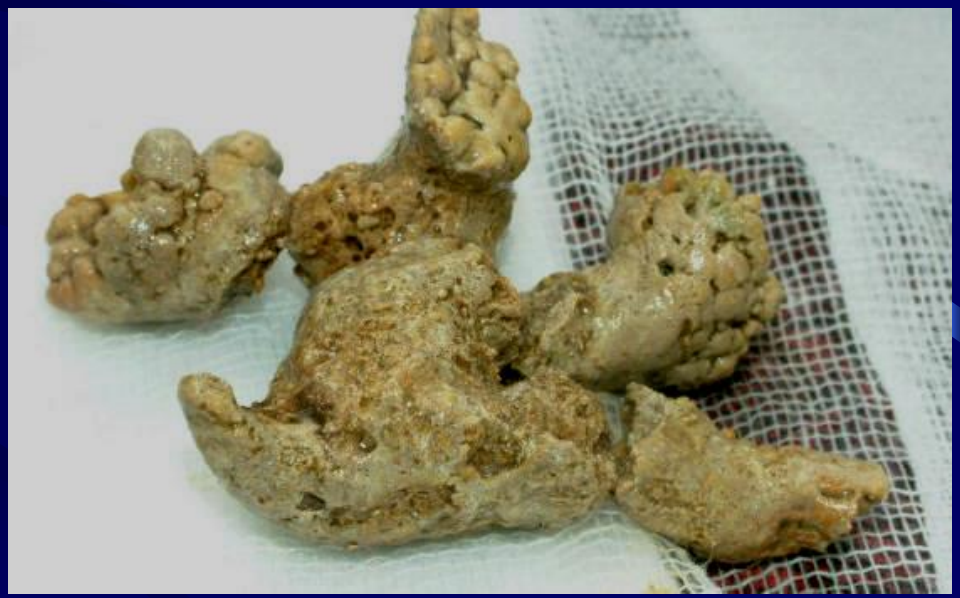
# Struvite Crystals and Stones





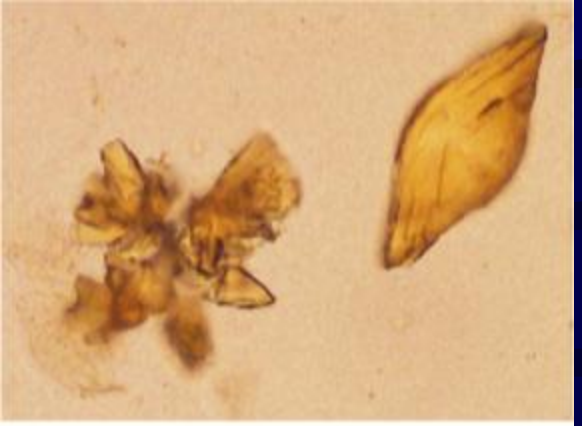




# Uric Acid Crystals and Stones

- Uric acid crystals are seen in urine BUT stones are rarely formed
- Uricuria may be due to
  - X's dietary intake
  - Gout -20% may form stones, usually there is nephropathy with crystaluria
  - Glycogen storage disease, Lesch-Nyhan Syndrome
  - Ileostomy
  - Treatment of Leukemia and other tumors
- Heat, dehydration and unusually acidic urine may lead to stone formation



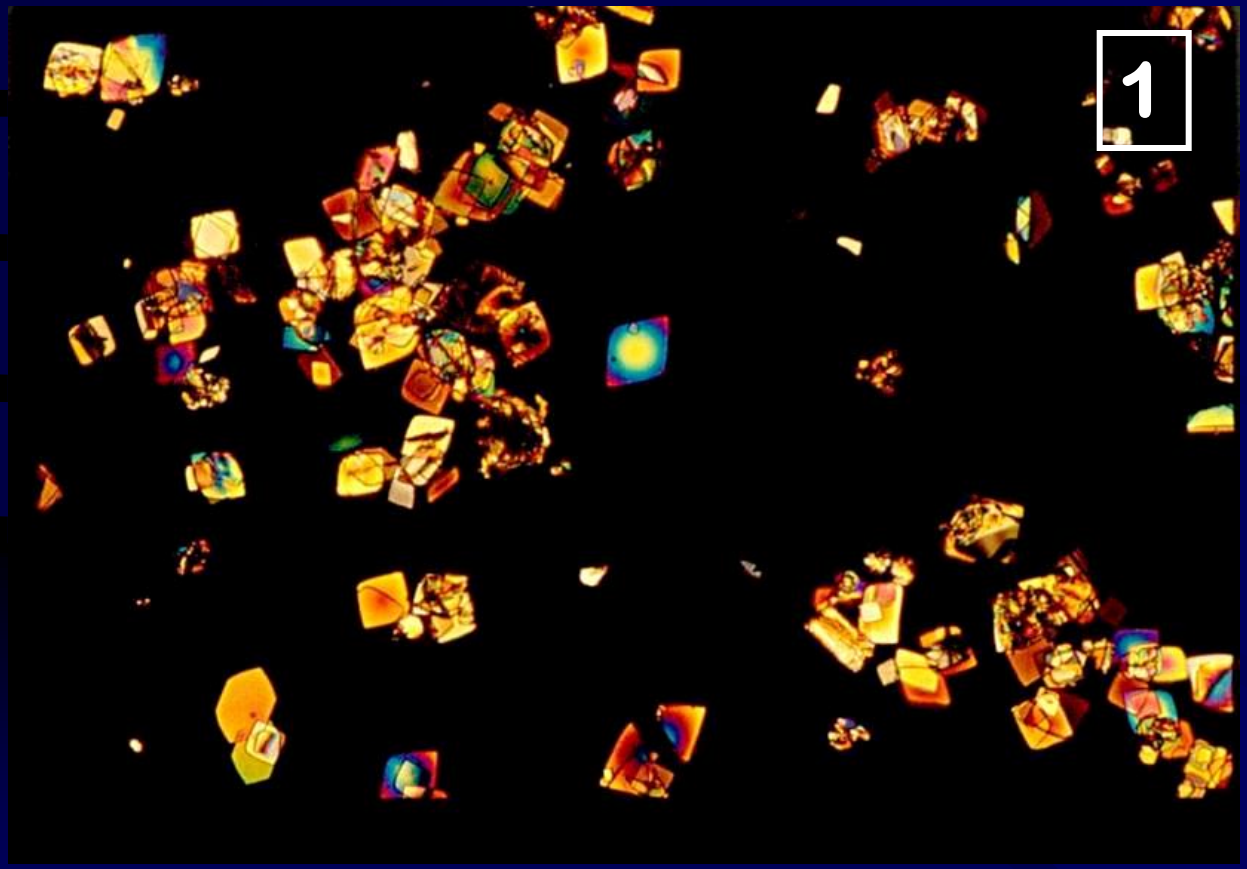

# Uric Acid Stones

- Gout
- Leukemia – Rapid cell turnover states
- About 50% of patients do not have Hyperuricemia
- Unknown mechanisms

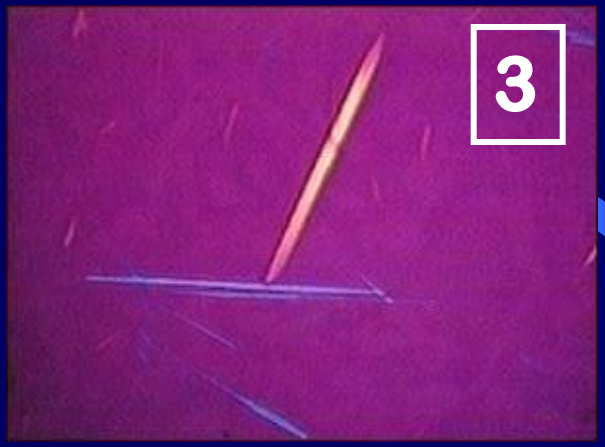
- More stones in acidic urine

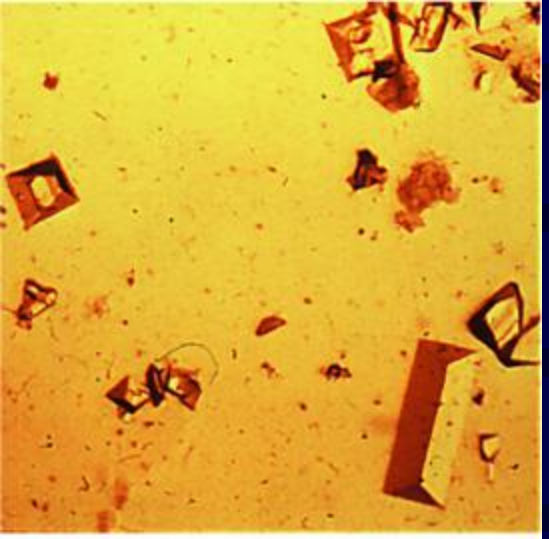
- Few are Radio-lucent

# Uric Acid Crystals (1) and Stones (2), Urate Crystals (3) Polarized Light







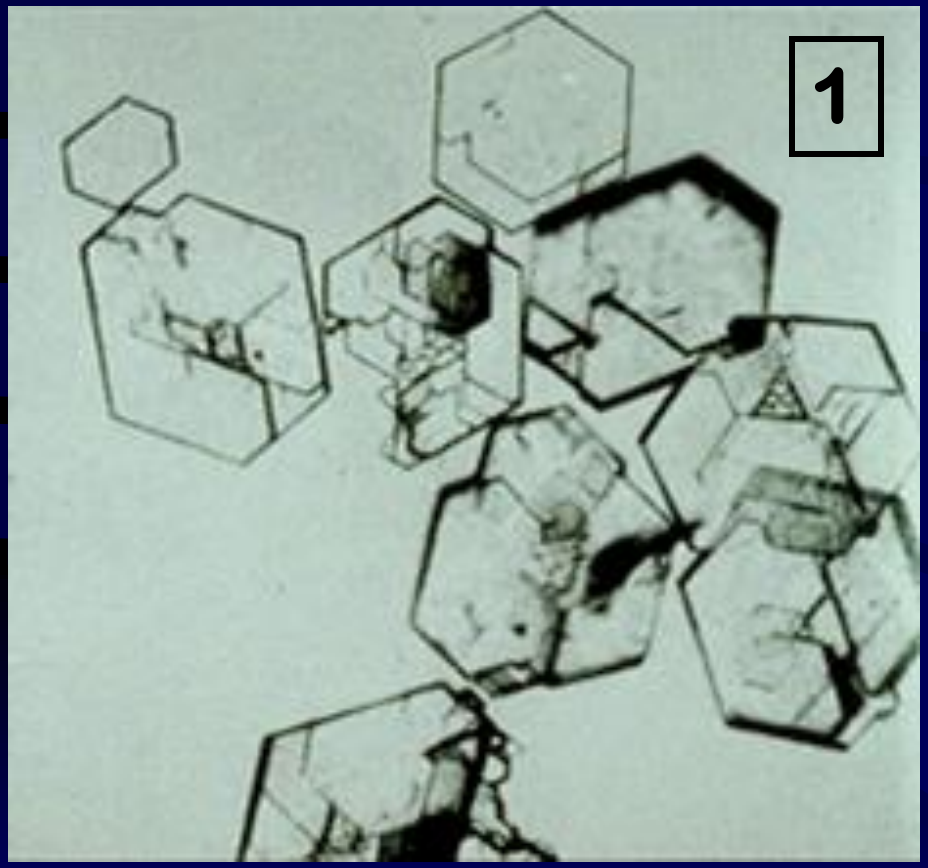


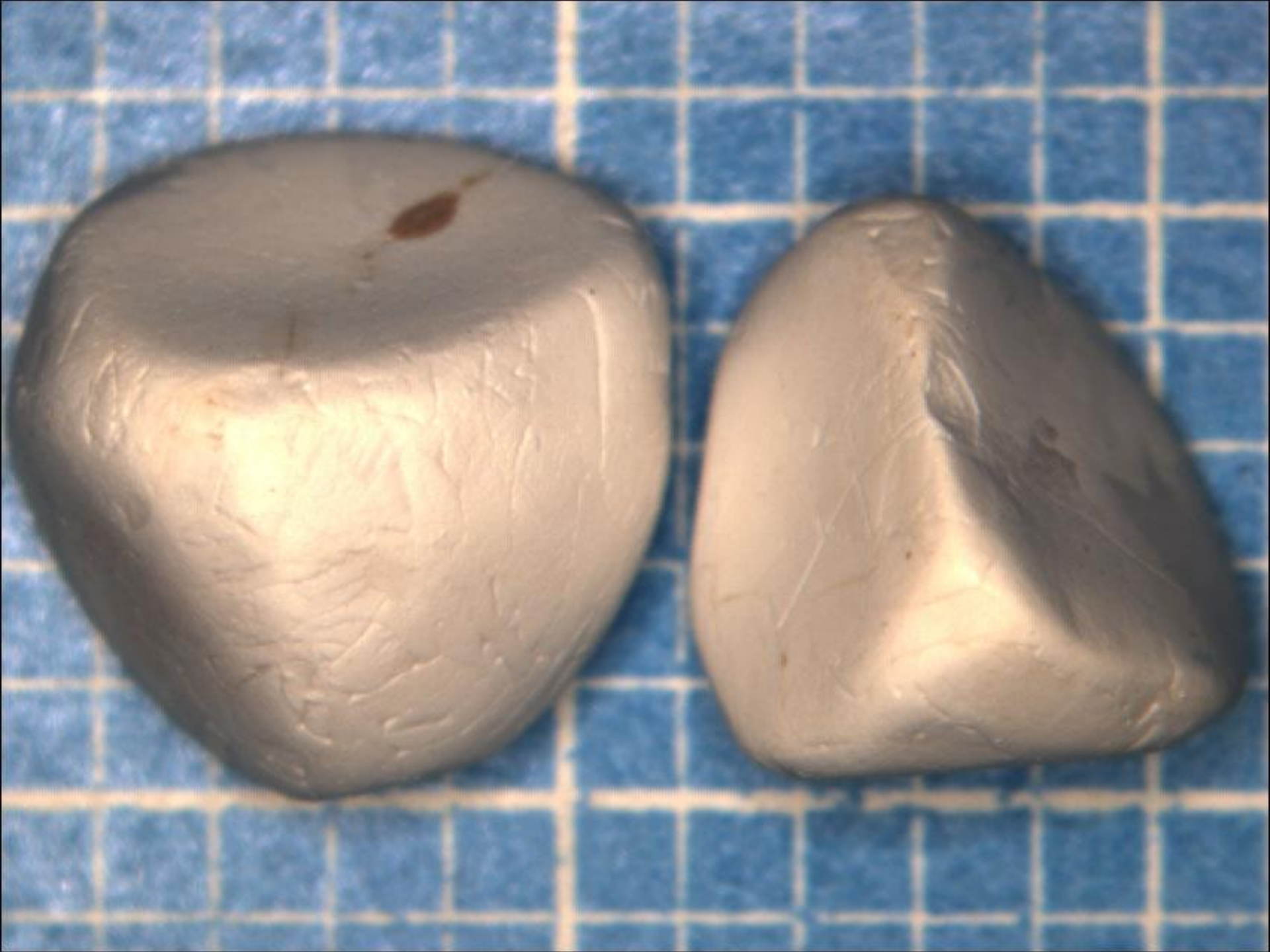

# Mixed Stones

- Magnesium Ammonium Phosphate stones
- Struvite stones
- Alkaline urine, Infection are predisposing factors
- Proteus vulgaris- urea splitting
- Split urea to ammonia favor their formation.
- Vit- A deficiency → Desquamation
- Radio- Opaque

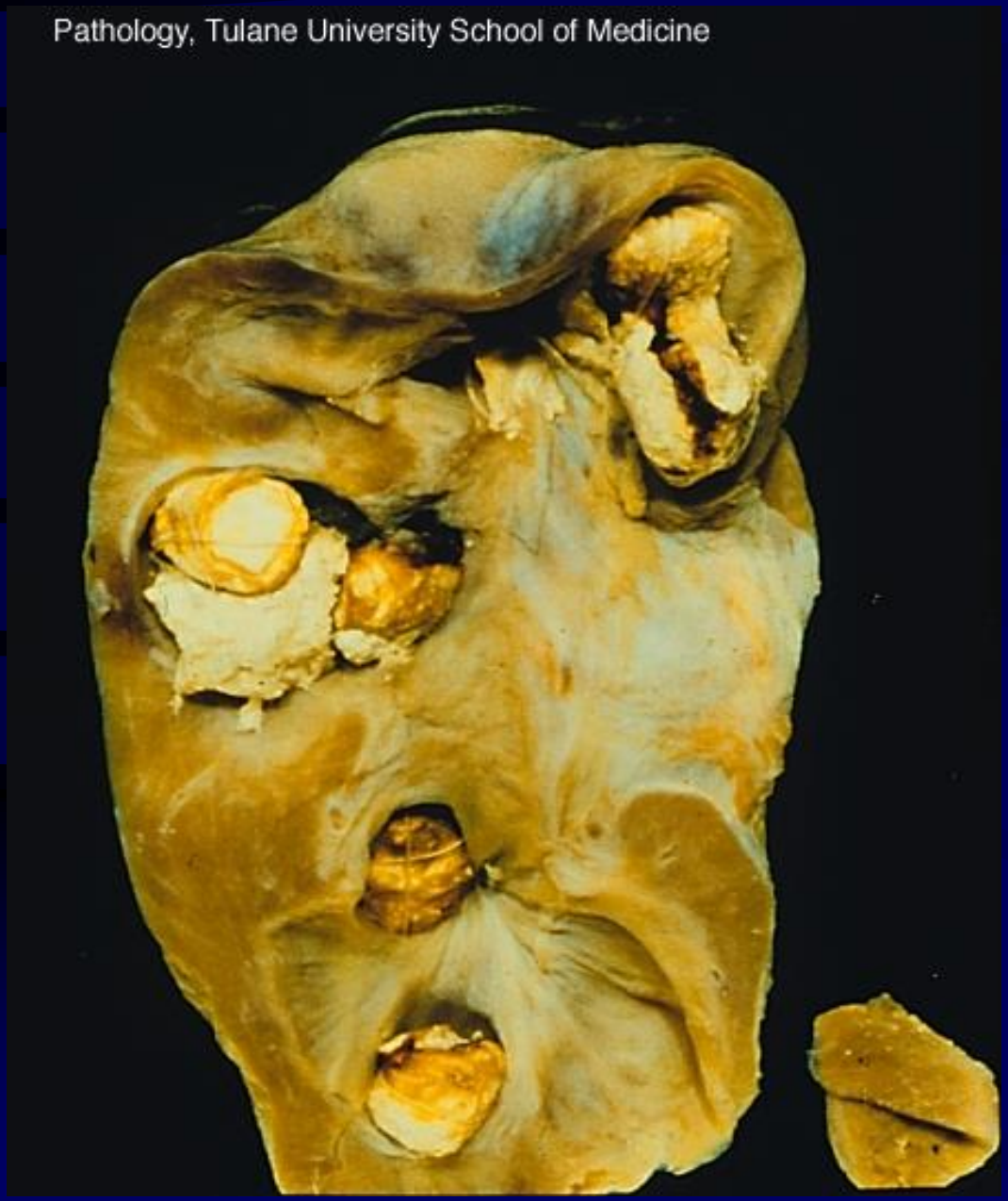
# Rare Urolithiasis

## Cystine Crystals (1) and Stones (2)




Broken Struvite Stone With Ruler




Stones may be surgically removed from kidneys; size of individual stones may vary from 0.5 cm-more

A Nephrectomy specimen with abundant stones
The normal cortex is replaced by fibrous tissue and the cortico-medullary junction is lost

# Urolithiasis vs Nephrocalcinosis

- Urolithiasis: calculus (stone) formation in the calyces, pelvis, and bladder
- Usually unilateral
- Stones are usually small, but can be large ("staghorn stones")

- Nephrocalcinosis: crystals inside the nephrons

# Nephrocalcinosis

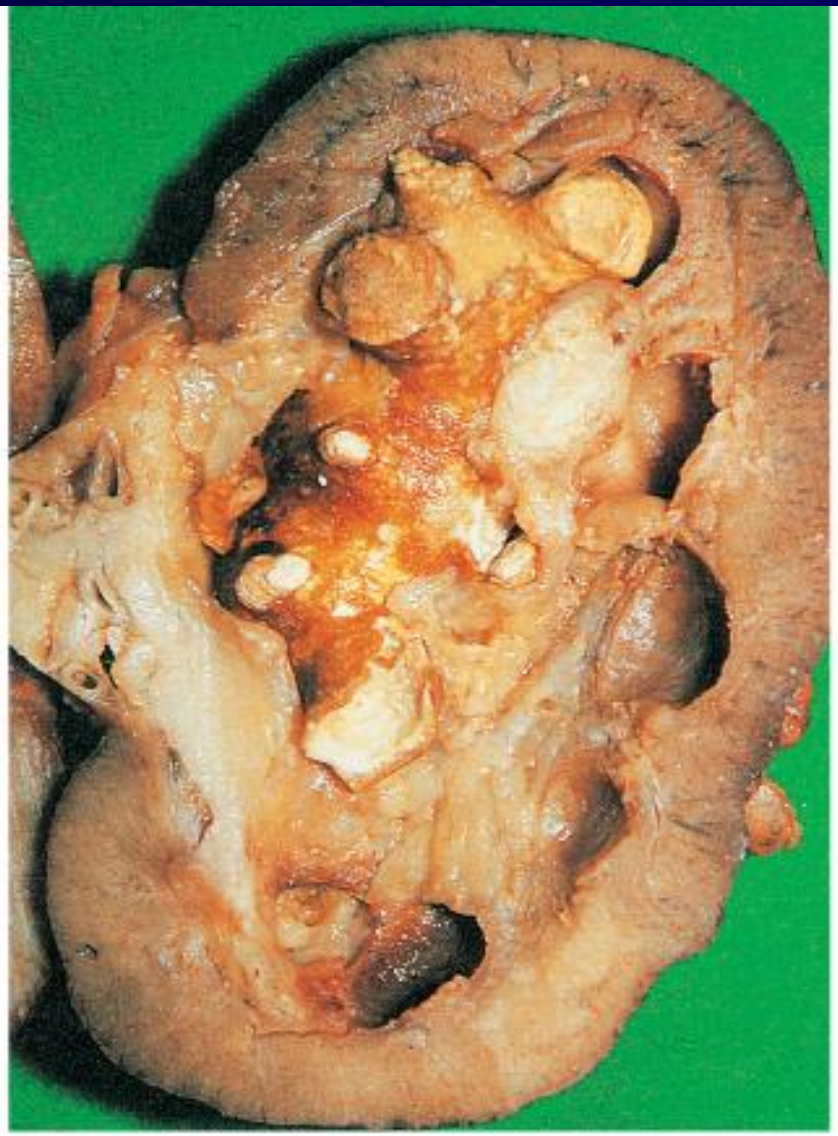
- Definition: Increased calcium content of the kidneys
- 3 categories
  - Chemical nephrocalcinosis:
    - increased concentration of calcium in renal cells, especially the tubular epithelium
  - Microscopic nephrocalcinosis:
    - Calcium precipitates in crystalline form as oxalate and/or phosphate, but it is only seen with the aid of a microscope
  - Macroscopic nephrocalcinosis:
    - large areas of calcification can be seen
- May be associated with renal calculi

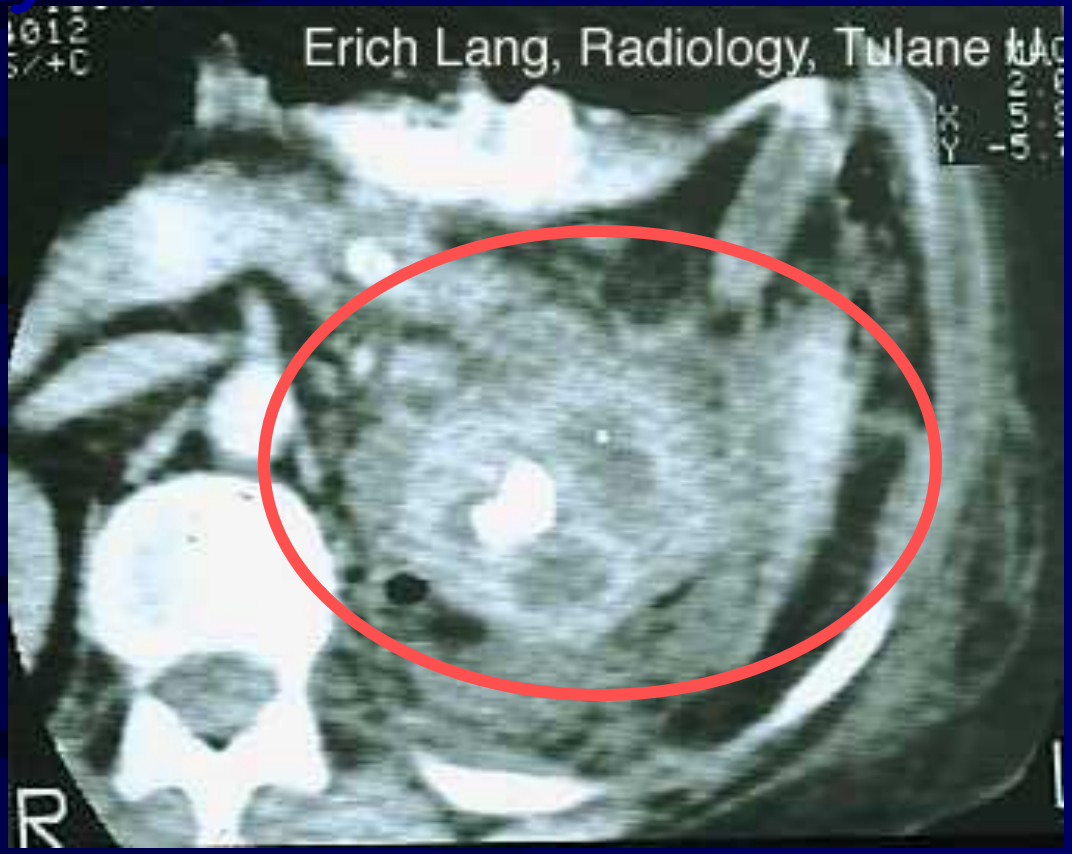
# Nephrocalcinosis

## Pathology

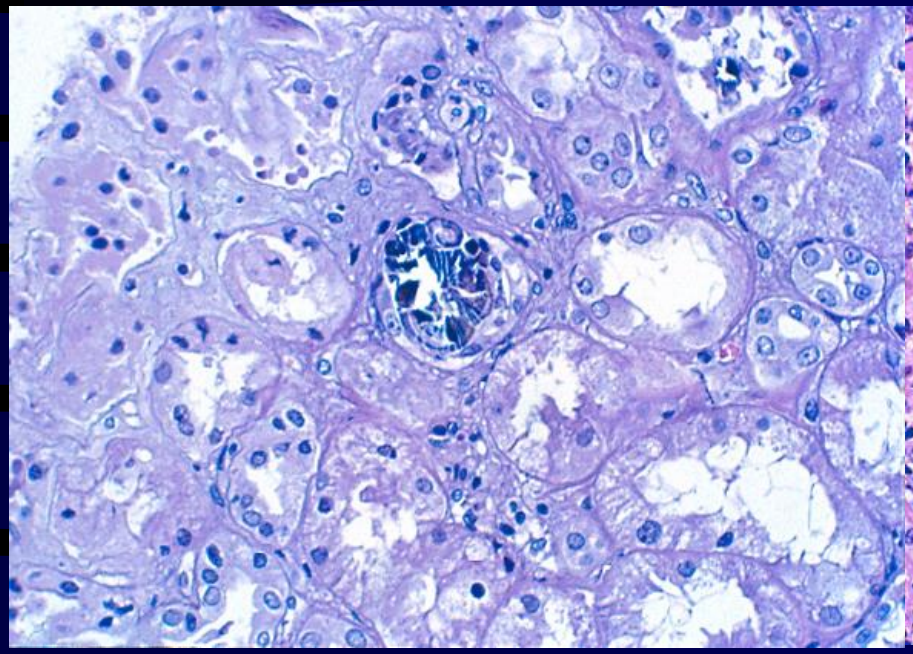
- Medullary Nephrocalcinosis:
  - Exclusive involvement of the medullary pyramids.
  - Associated with Hypercalcemia

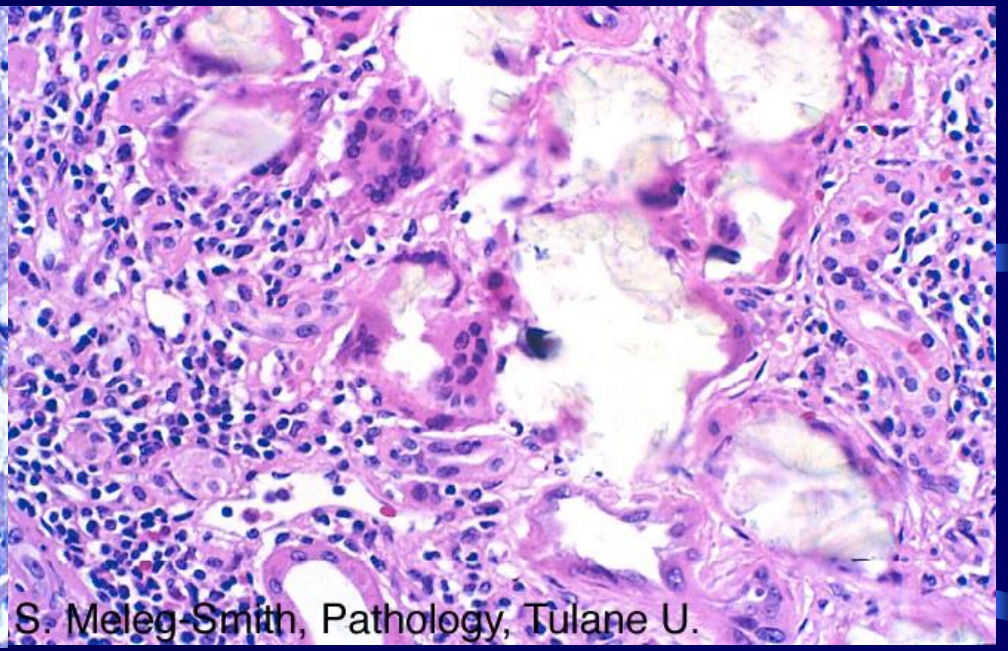
- Cortical Nephrocalcinosis:
  - Rarer and involves all the renal parenchyma.
  - Frequently associated with severe metabolic defects, such as primary hyperoxaluria or end-stage renal failure.
  - Other causes include haemolytic uraemic syndrome, chronic pyelonephritis, vesicoureteral reflux, renal transplantation and polycystic kidney disease

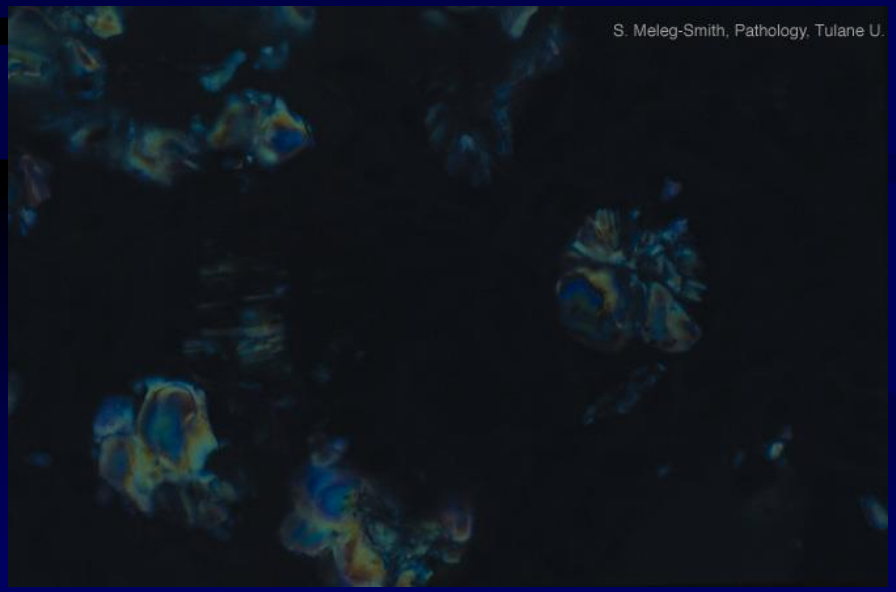




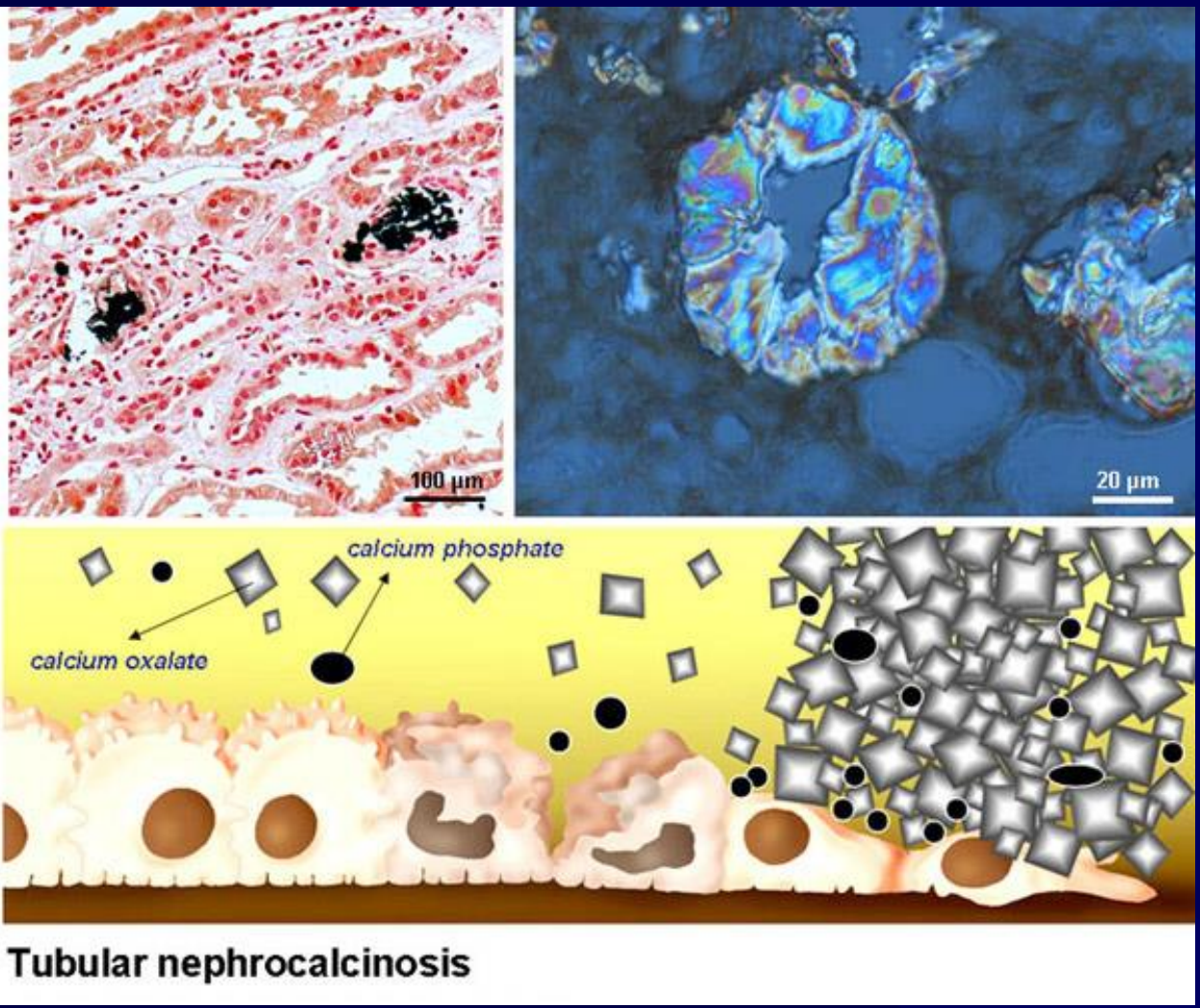

Cortico-medullary phase CT shows dilated calices (hydronephrosis) and a large calculus. There is also loss of parenchymal thickness. Note striation and fluid in the perirenal space, and marked thickening of the posterior renal fascia, findings suggestive of a perirenal abscess. In view of the infection, this is a case of pyonephrosis





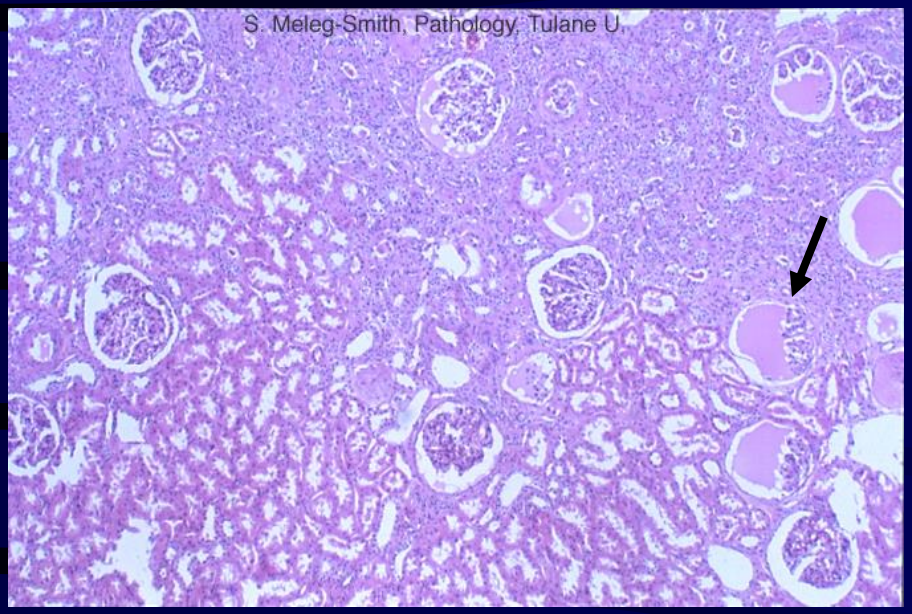



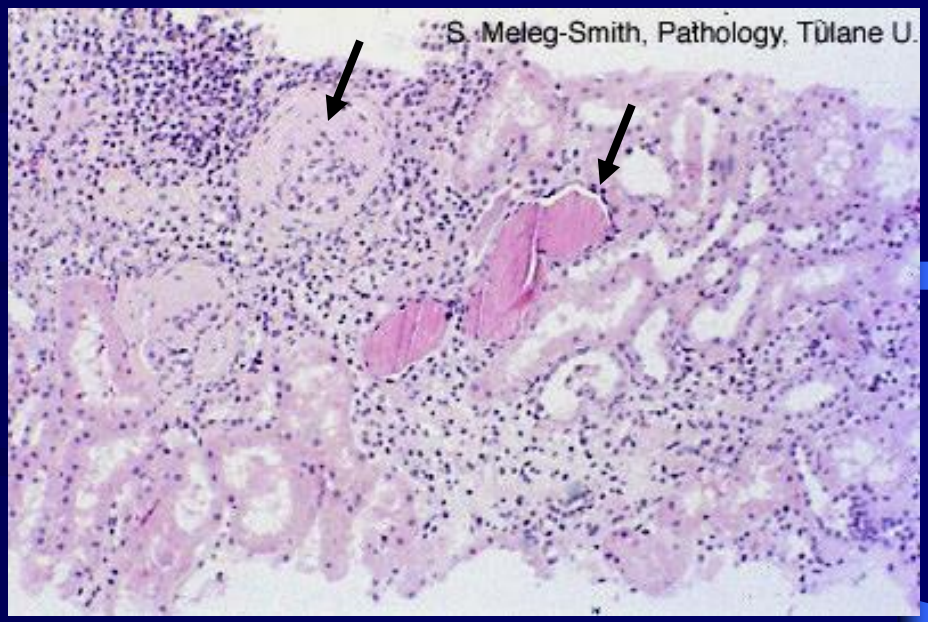

**Basophilic crystals in the tubular lumen are calcium (Left hand panel)**
**Empty" irregular areas are surrounded by foreign body giant cells (Right panel)**

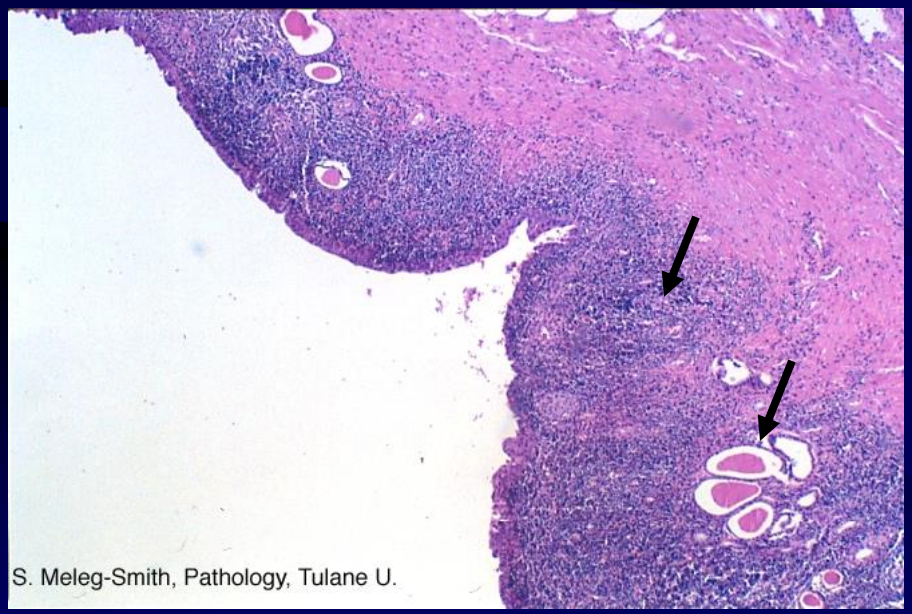



Tubular nephrocalcinosis

Top left: Retention of calcium oxalate crystals in the renal tubules after kidney transplantation. Top right: Close-up image of calcium oxalate crystals plugging the renal tubules of a primary hyperoxaluria patient with end-stage renal failure. Obstruction of the renal tubules leads to tubular necrosis and loss of the total nephron mass. In preterm infants, tubular nephrocalcinosis may lead to reduced renal function in adulthood, while in renal transplant patients it may have a negative impact on long-term graft survival.



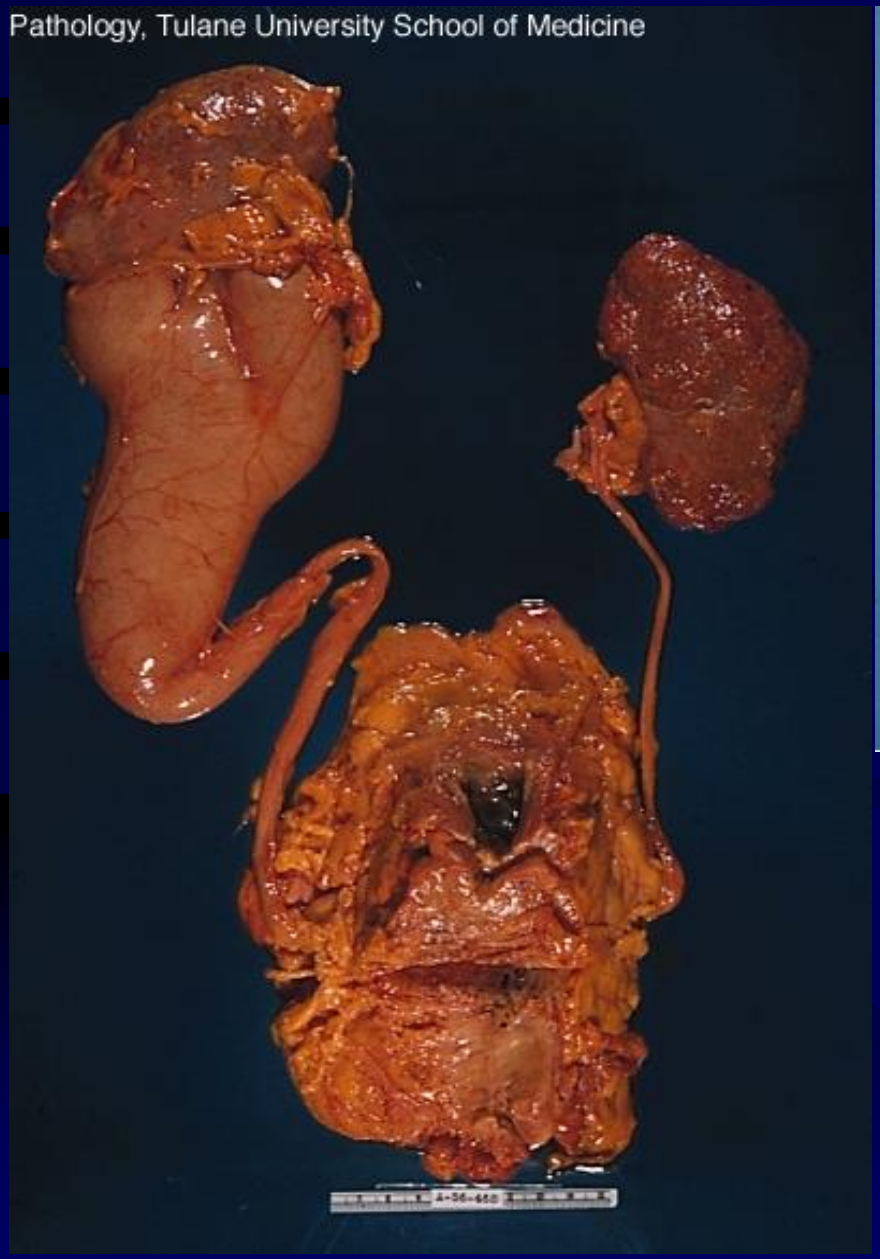



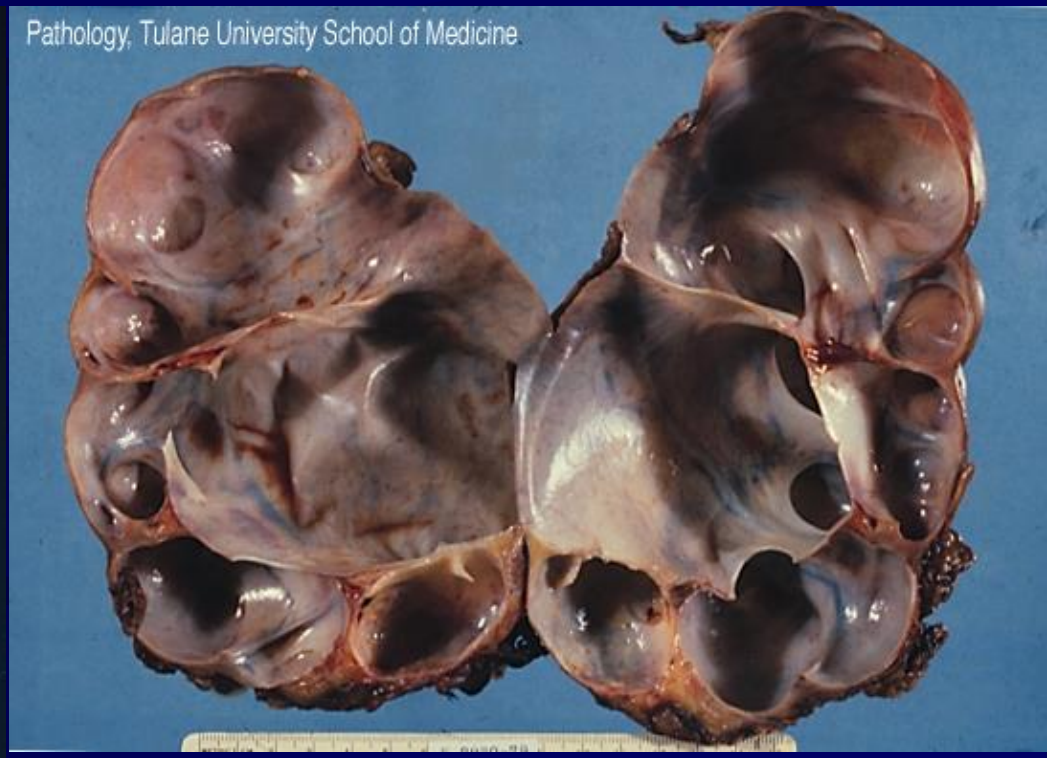




Backed up fluid is seen in the bowman's capsule
Glomeruli are replaced by global sclerosis
Interstitial fibrosis and inflammation are observed
Tubules in the center of the field are distended by proteinaceous material

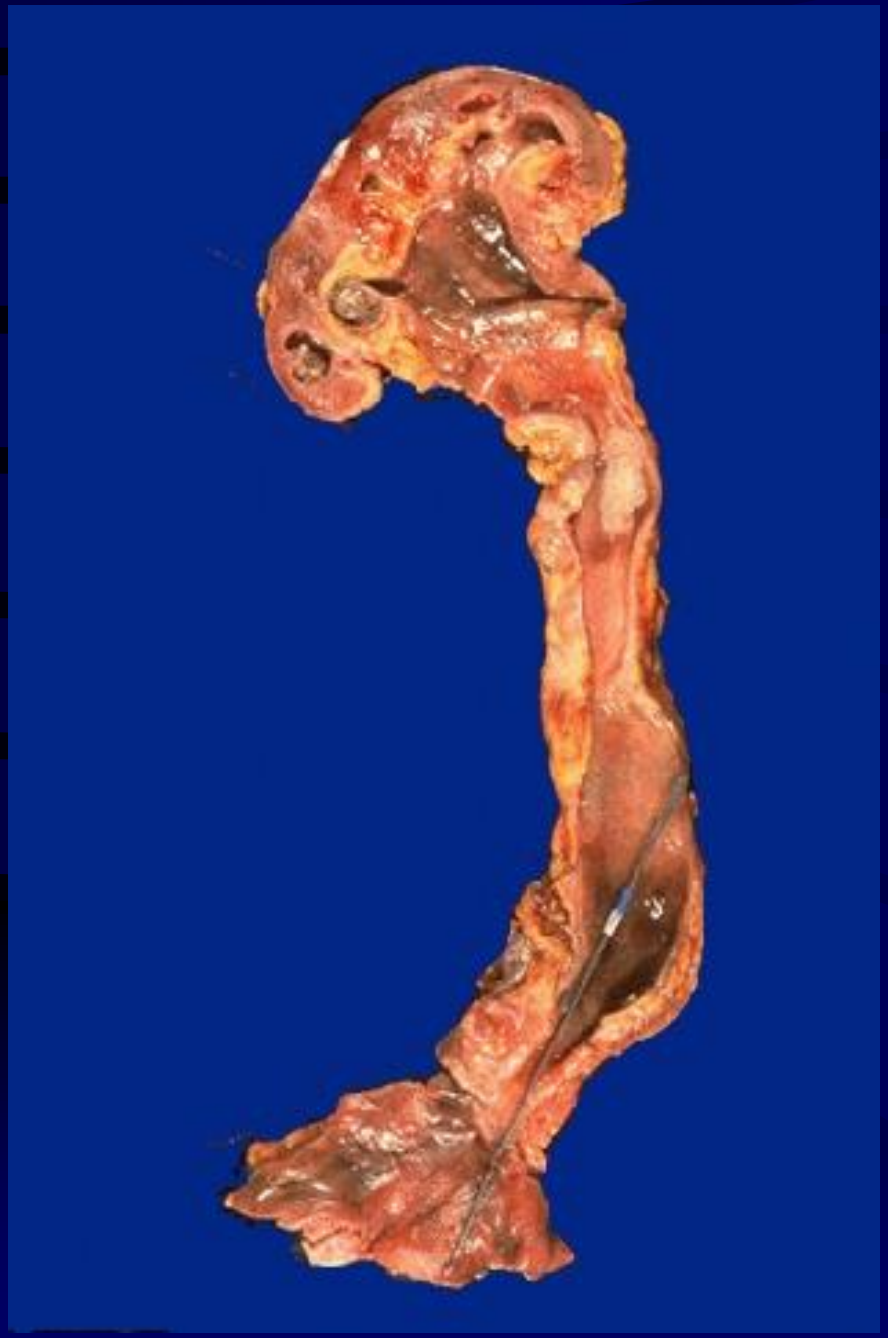
# Complications -Obstructive Uropathy and Hydronephrosis

- Pathogenesis
  - Obstruction: uni- or bilateral, usually partial
  - Pelvis and calyceal system are dilated due to back pressure
  - Ureteric perstalsis is increased
  - Initially the trapped filtrate is reabsorbed by lymphatics and vascular route
  - Continued obstruction ultimately reduces glomerular blood flow and GFR
  - Prolonged back pressure leads to Hydronephrosis




**Hydroureter (Left) and Hydronephrosis (Right)**



A long-standing obstruction (probably congenital) at the ureteral orifice through which the metal probe passes led to the marked hydroureter and hydronephrosis seen here.

# Complications -Obstructive Uropathy and Hydronephrosis

- Causes
  - Congenital anomalies: Uretero-pelvic stenosis, Ureteropelvic reflux etc
  - Tumors: Carcinoma of bladder and Prostate
  - Hyperplastic lesions: Benign prostatic hyperplasia (BPH)
  - Calculi (Stones)
  - Spinal cord damage with bladder paralysis
  - **Gravid uterus**

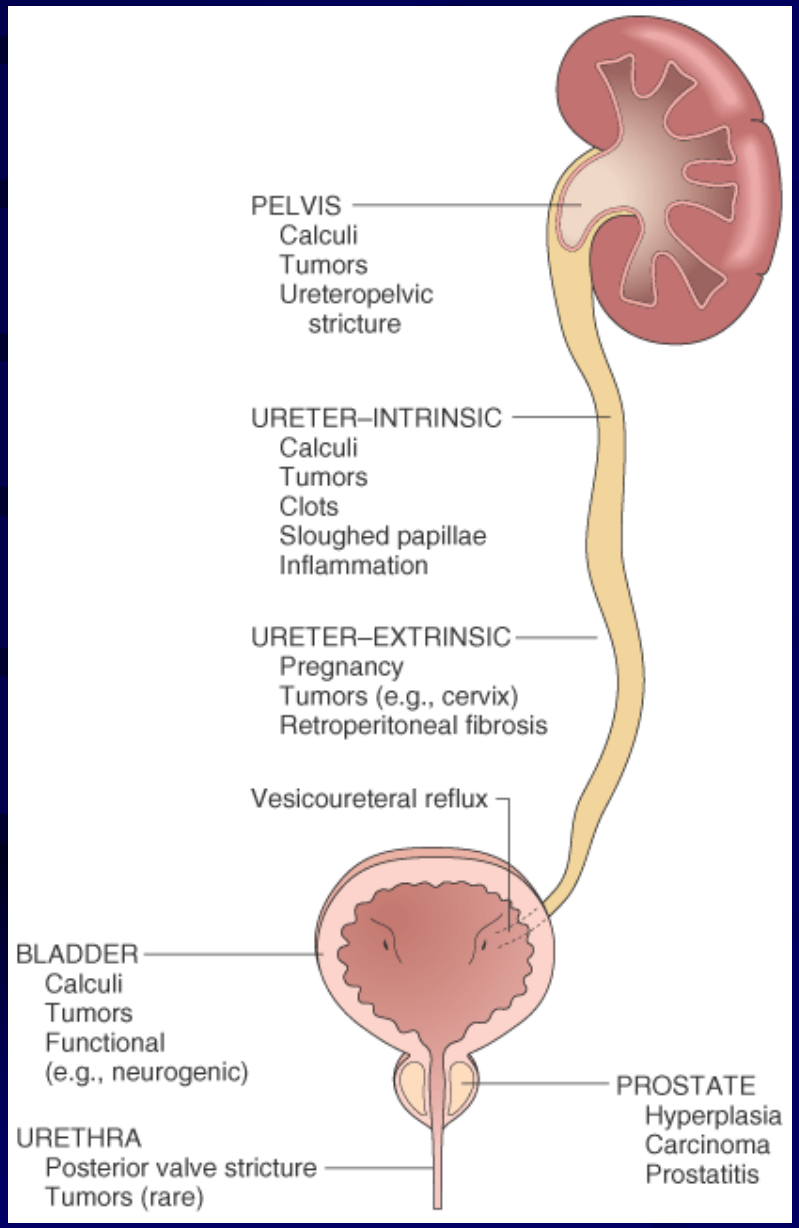
- Signs, symptoms and prognosis depends on level of obstruction
- Renal calculus → Renal colic
- BPH or Bladder tumor → bladder symptoms, anuria and pain

# Urinary obstruction

- Recognition is important because *obstruction increases susceptibility to infection and to stone formation, and*

- *Unrelieved obstruction almost always leads to permanent renal atrophy*, termed *hydronephrosis* or *obstructive uropathy*

# Obstructive lesions of the urinary tract

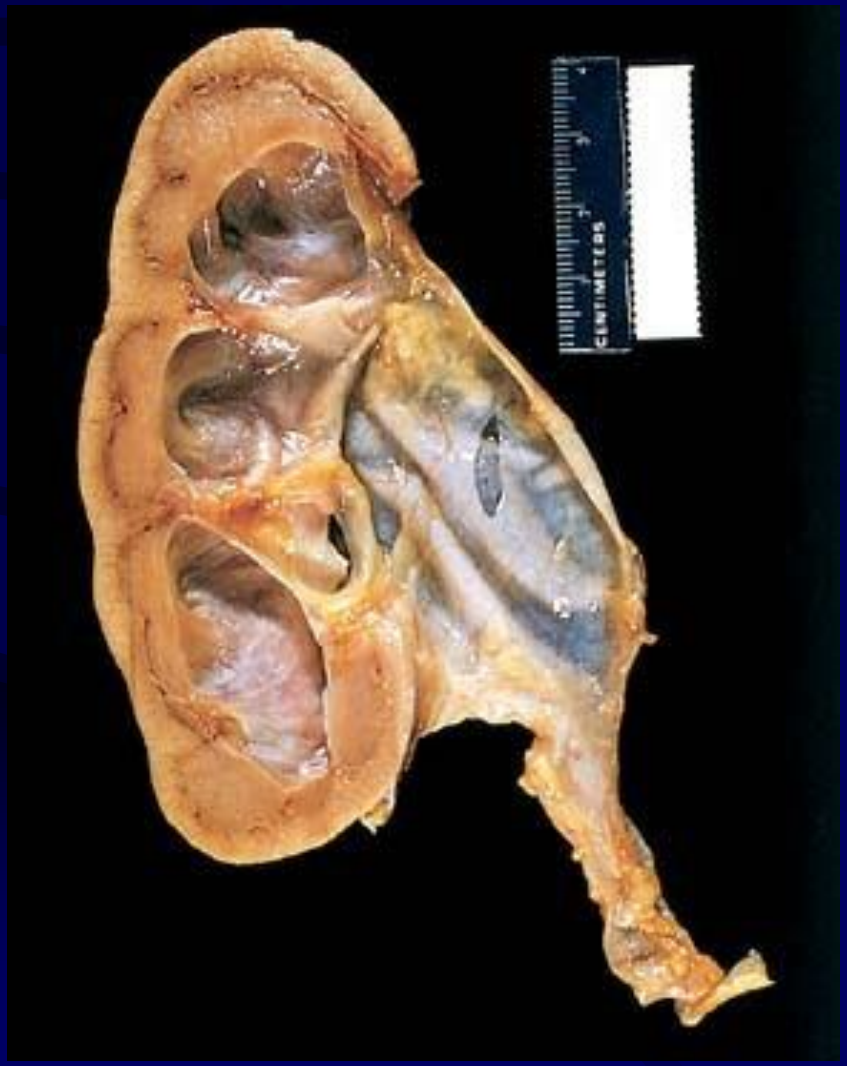


# *Hydronephrosis*

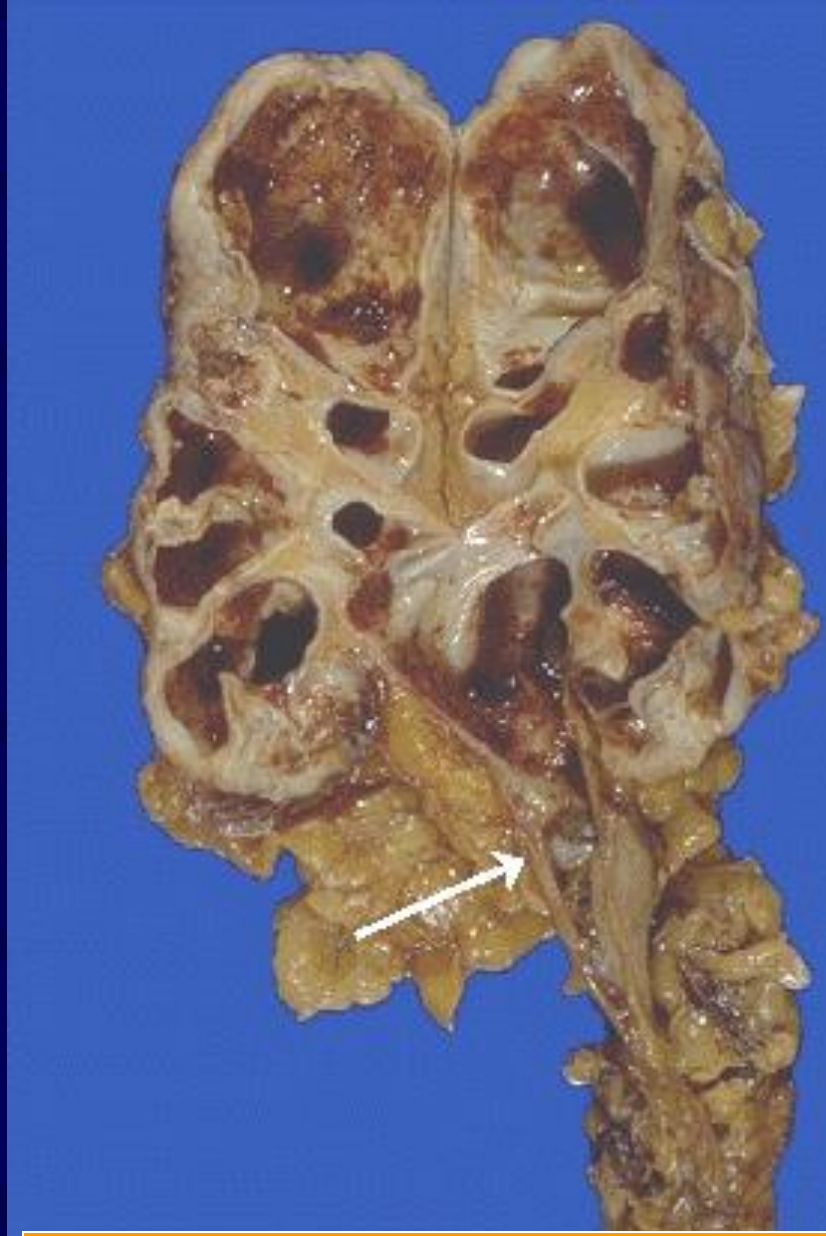
- *Used to describe dilation of the renal pelvis and calyces associated with progressive atrophy of the kidney due to obstruction to the outflow of urine*

- Renal stone (most common)
- Retroperitoneal fibrosis
- Cervical cancer, benign prostatic hyperplasia

Hydronephrosis of the kidney, with marked dilation of the pelvis and calyces and thinning of the renal parenchyma.



**Advanced hydronephrosis**

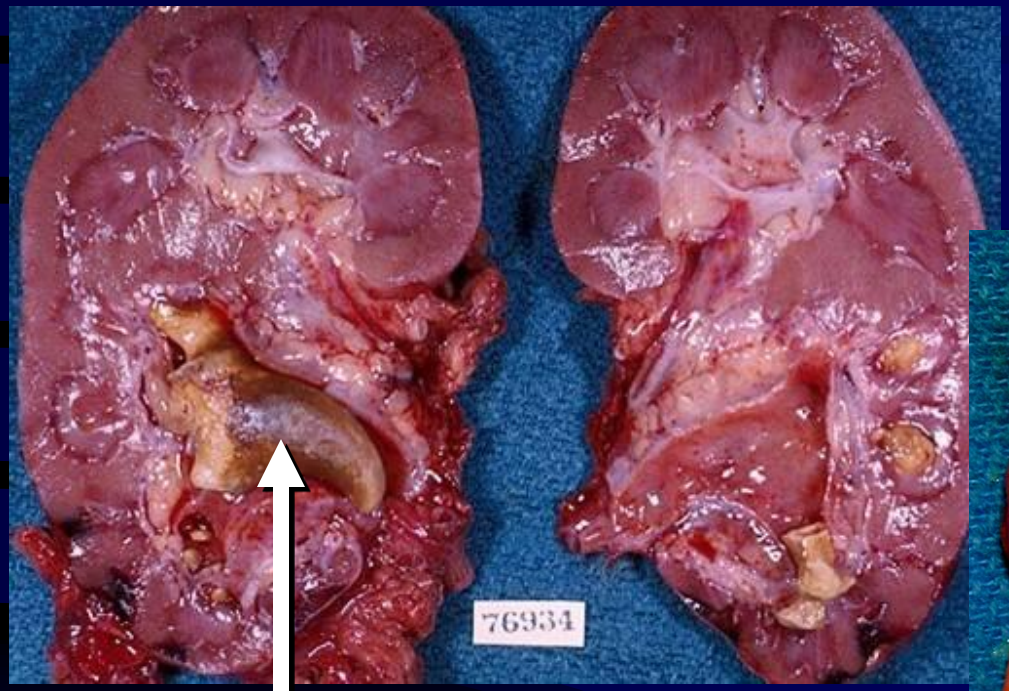


Hydronephrosis--a ureteral calculus caught at the ureteropelvic junction.

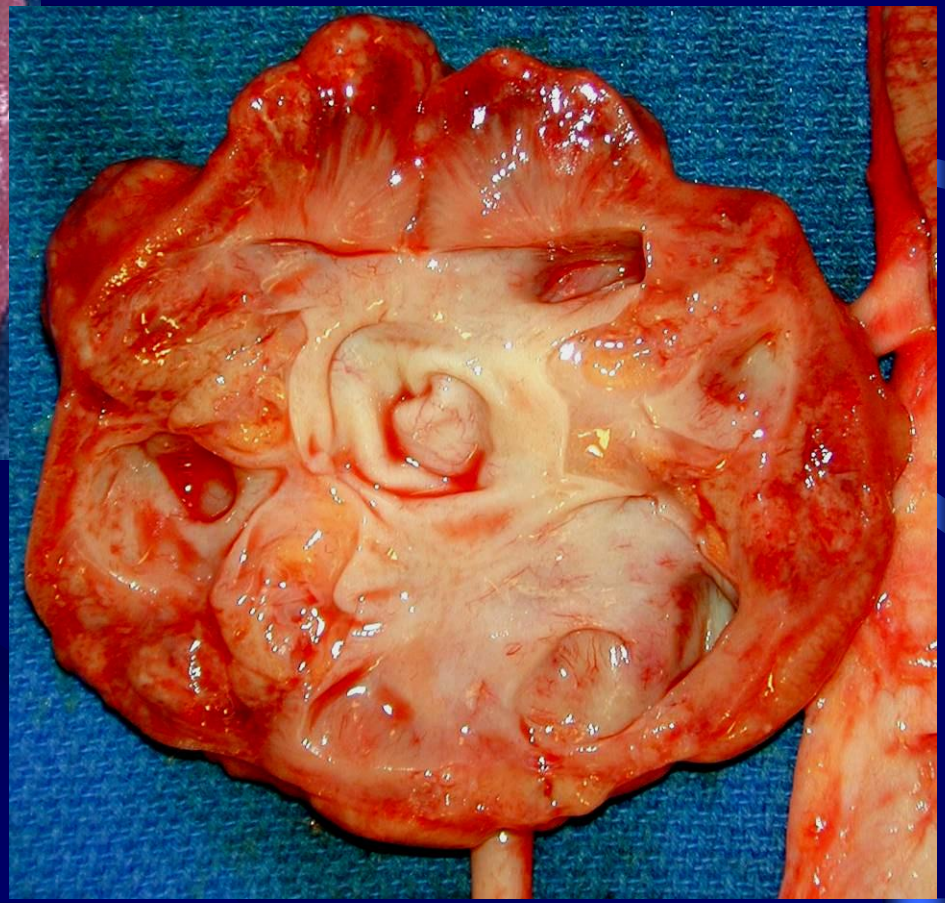
# Staghorn calculi

- Infections by urea-splitting bacteria (e.g., *Proteus* and some staphylococci) convert urea to ammonia.
- The resultant alkaline urine causes the precipitation of magnesium ammonium phosphate salts.
- *Staghorn calculi* occupy large portions of the renal pelvis are almost always a consequence of infection.

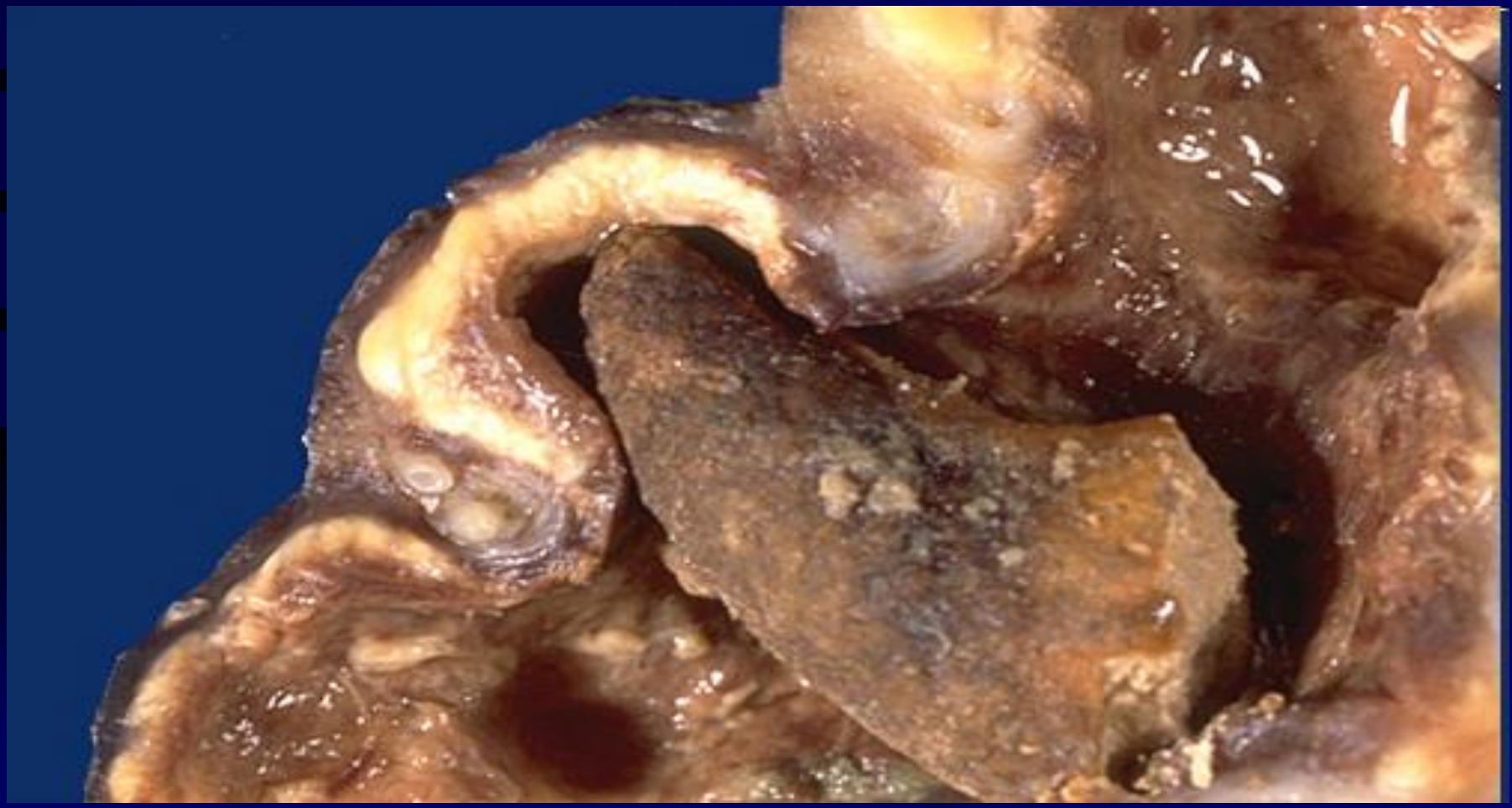
# Staghorn Stone and Hydronephrosis

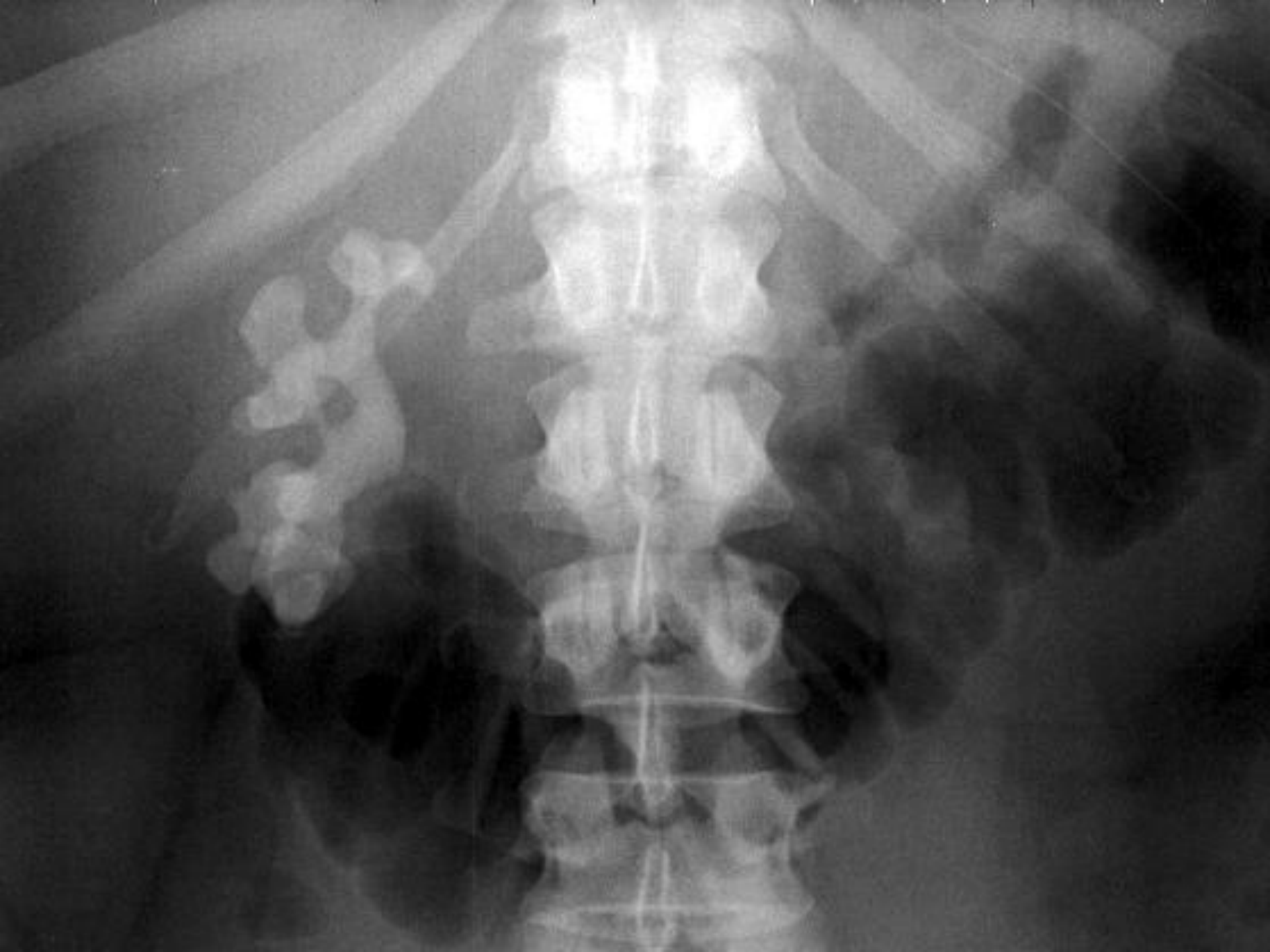


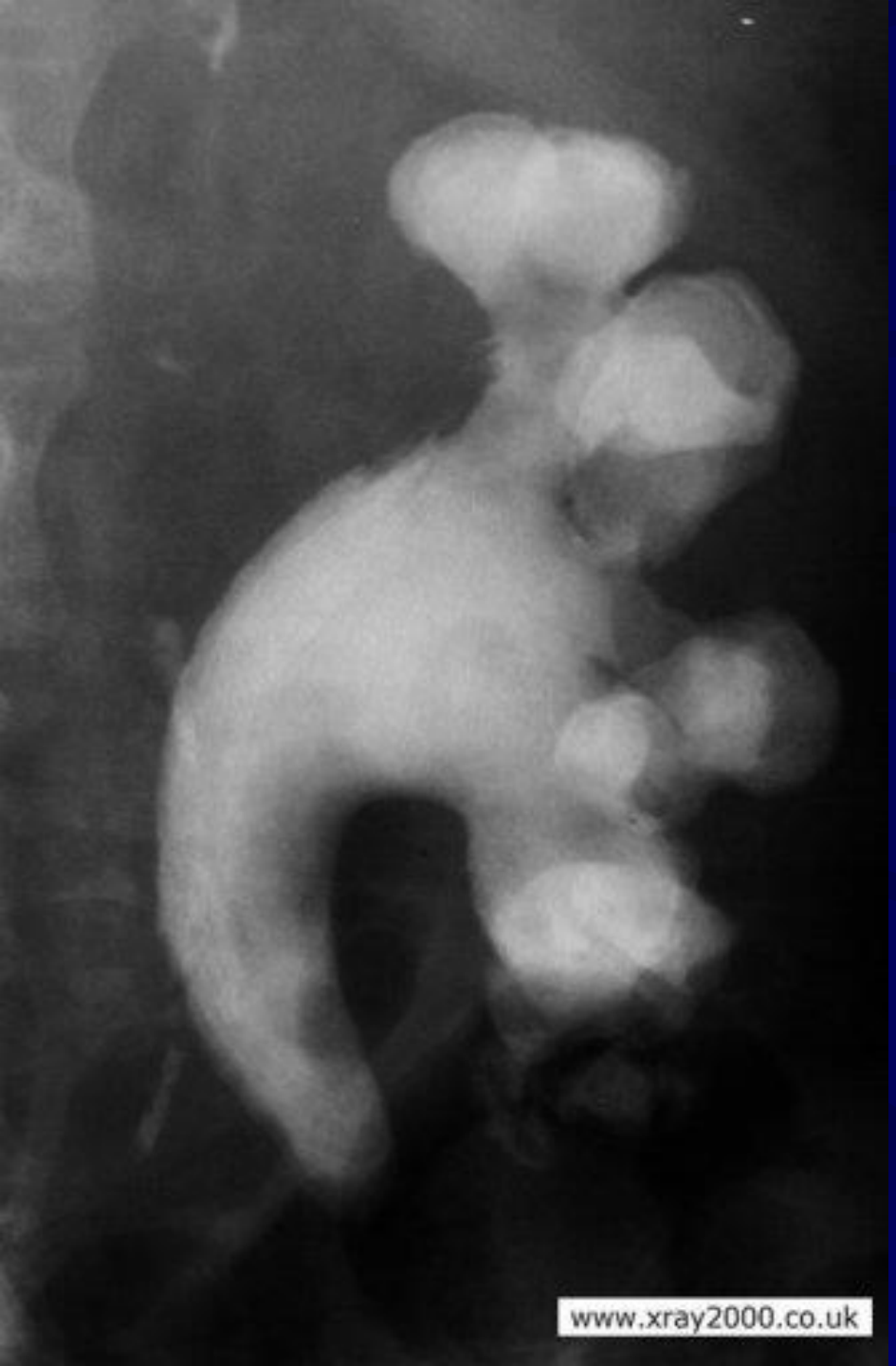

Stone



Sometimes a very large calculus nearly fills the calyceal system, with extensions into calyces that give the appearance of a stag's (deer) horns. Hence, the name "staghorn calculus". Seen here is a horn-like stone extending into a dilated calyx, with nearly unrecognizable overlying renal cortex from severe hydronephrosis and pyelonephritis. Nephrectomy may be performed because the kidney is non-functional and serves only as a source for infection.








# Staghorn stones

# Clinical course

- Many are asymptomatic
- Smaller stones - Their passage is marked by intense abdominal or back or flank pain. This pain can be paroxysmal, known as renal or ureteral "colic".
- Hematuria may also be present.
- Larger stones that cannot pass may produce hydronephrosis or hydroureter.
- UTI

# Urolithiasis

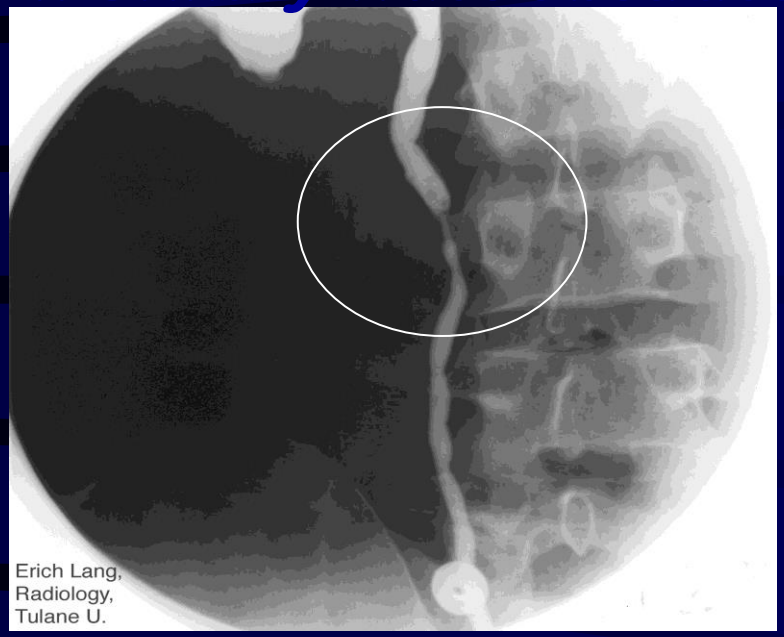
## Clinical Manifestations

- M » F; >20 – 30 yrs
- Small stones are usually silent
- Symptoms:
- May be asymptomatic
- Ureteral obstruction → renal colic
  - Paroxysm of flank pain, radiating to groin
  - Gross hematuria (“fresh” RBCs, no casts)
- Recurrent and intractable UTI
- Complications: obstruction and/or infection
  - Renal pelvis stones may predispose to Pyelonephritis and Hydronephrosis

# Urolithiasis

## Diagnosis

- X-Ray and Spiral CT
  - Calcium-containing stones: Radiopaque
  - Struvite, uric acid and cystine stones are radiolucent, unless they contain calcium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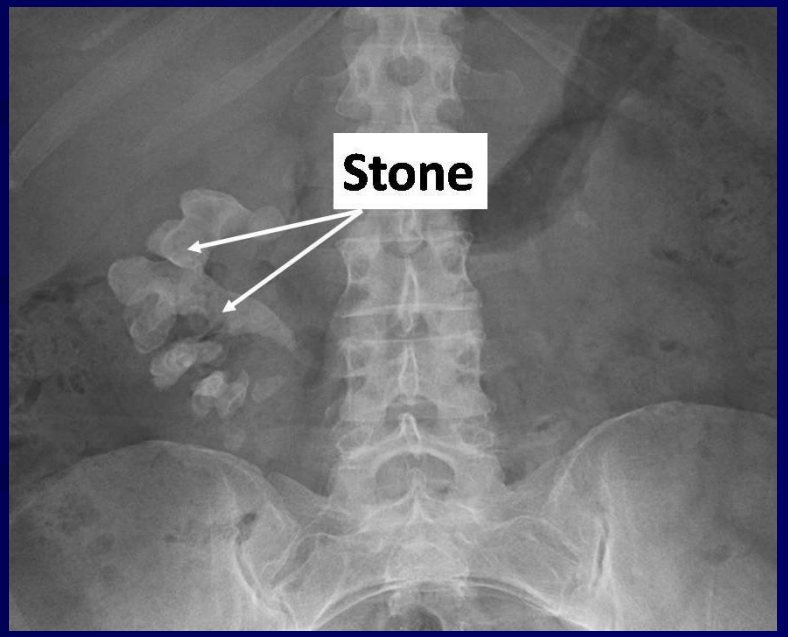
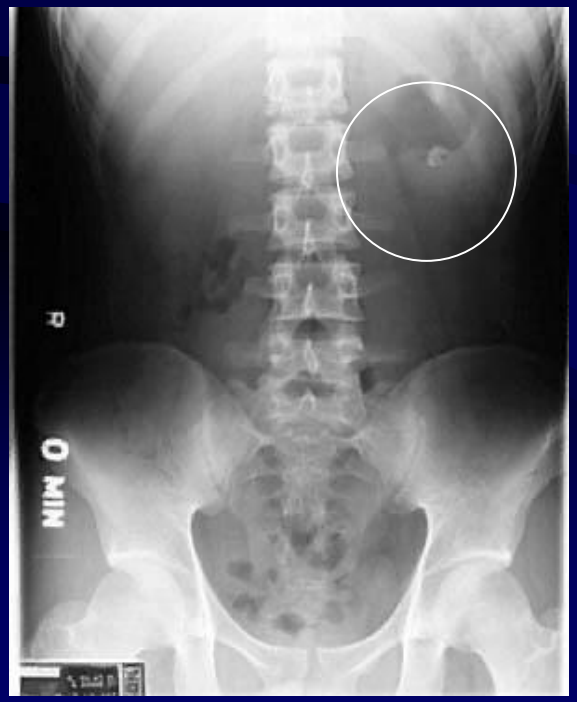

Stone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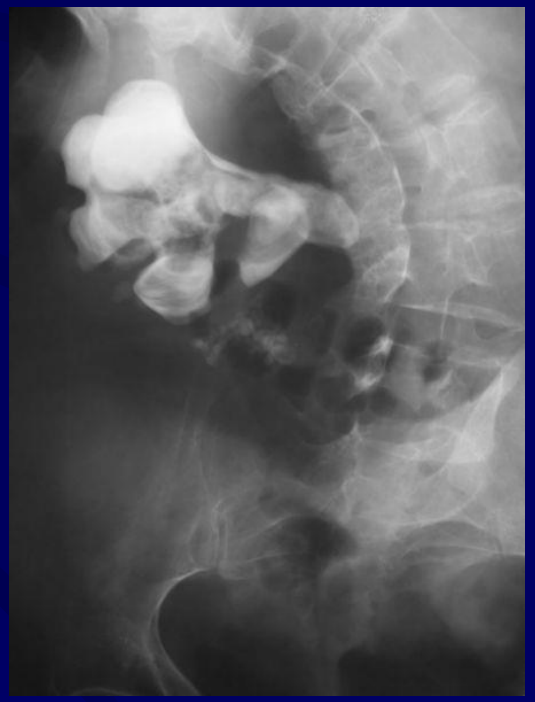

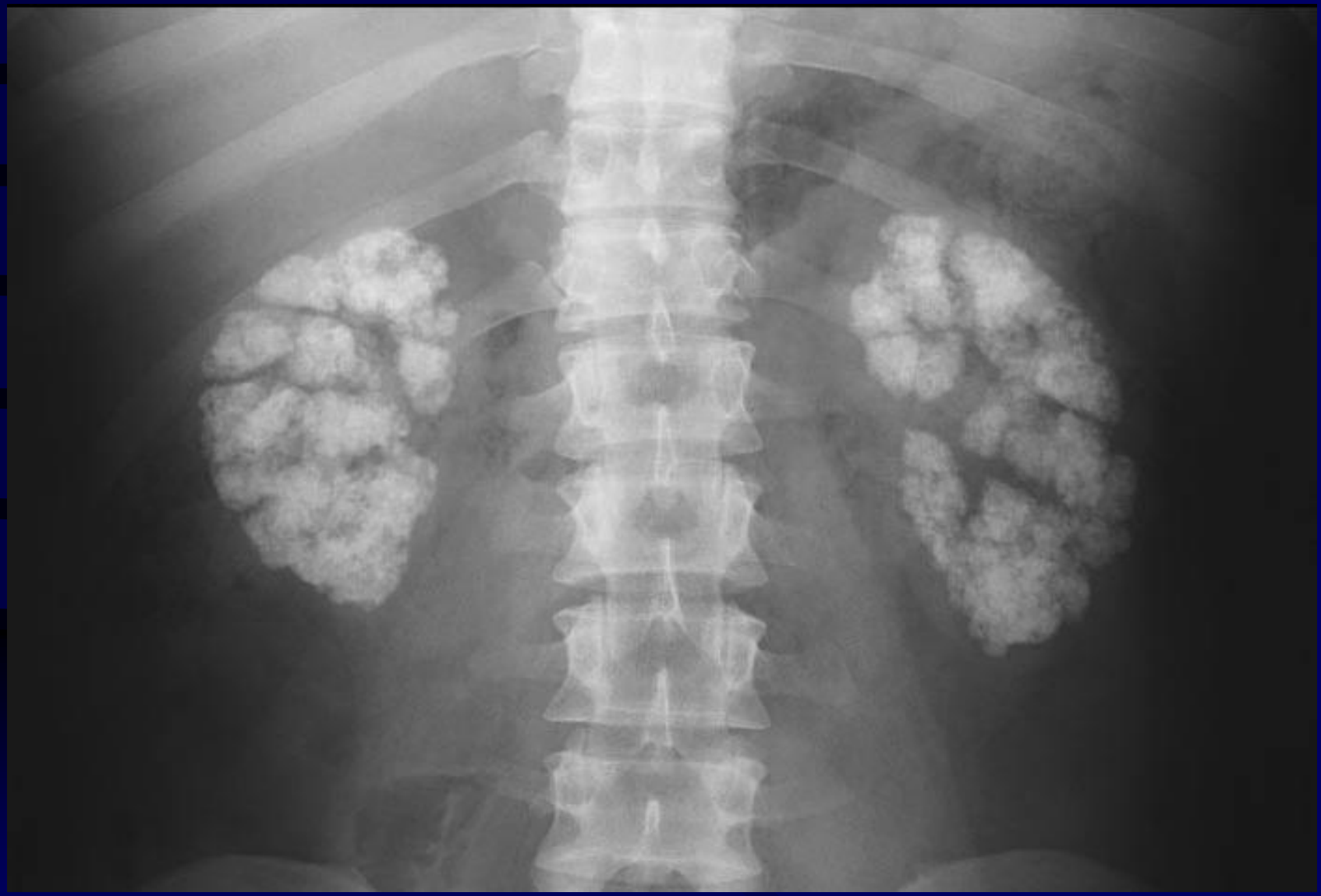

- Treatment: by lithotripsy, a non-invasive removal or surgical