Renal pathology

Urologic diseases of the kidney

I. Developmental abnormalities

II. Cystic diseases

III. Renal stones

IV. Obstructive uropathy

V. Pyelonephritis

Congenital abnormalities

1. ***Agenesis of the kidney***

In utero detected bilateral agenesis indicates abortion

2. ***Hypoplasia of the kidney***

Smaller kidney with contralateral compensatory hypertrophy

3. ***Oligomeganephronia***

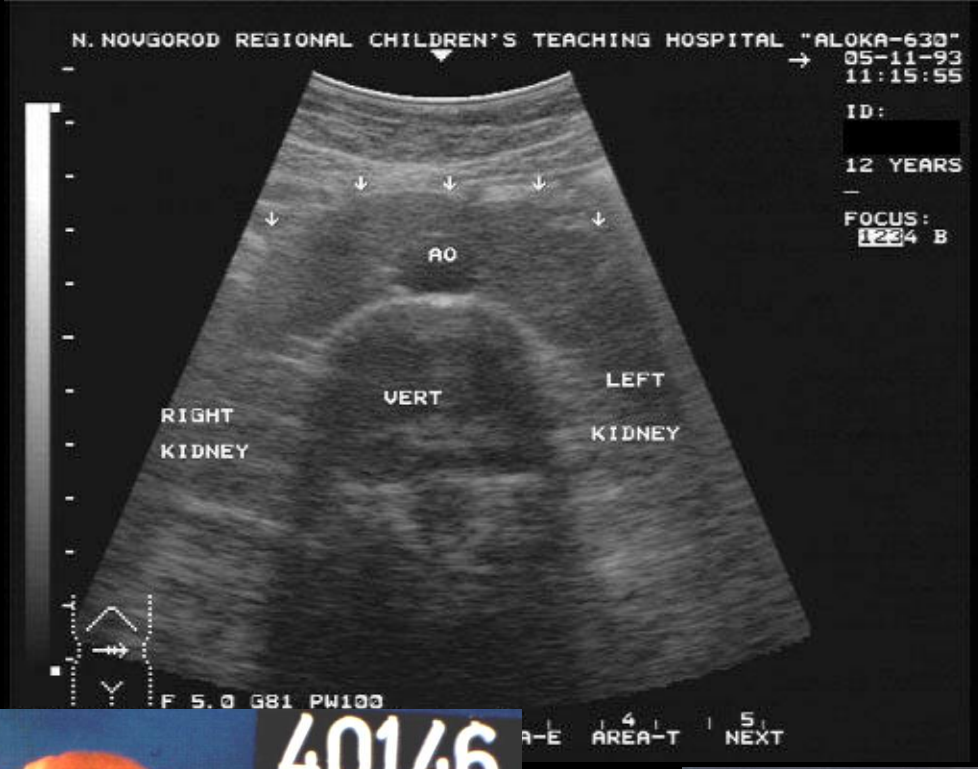
Reduced number of nephrons leading to end stage renal disease (ESRD) by the time of adolescence

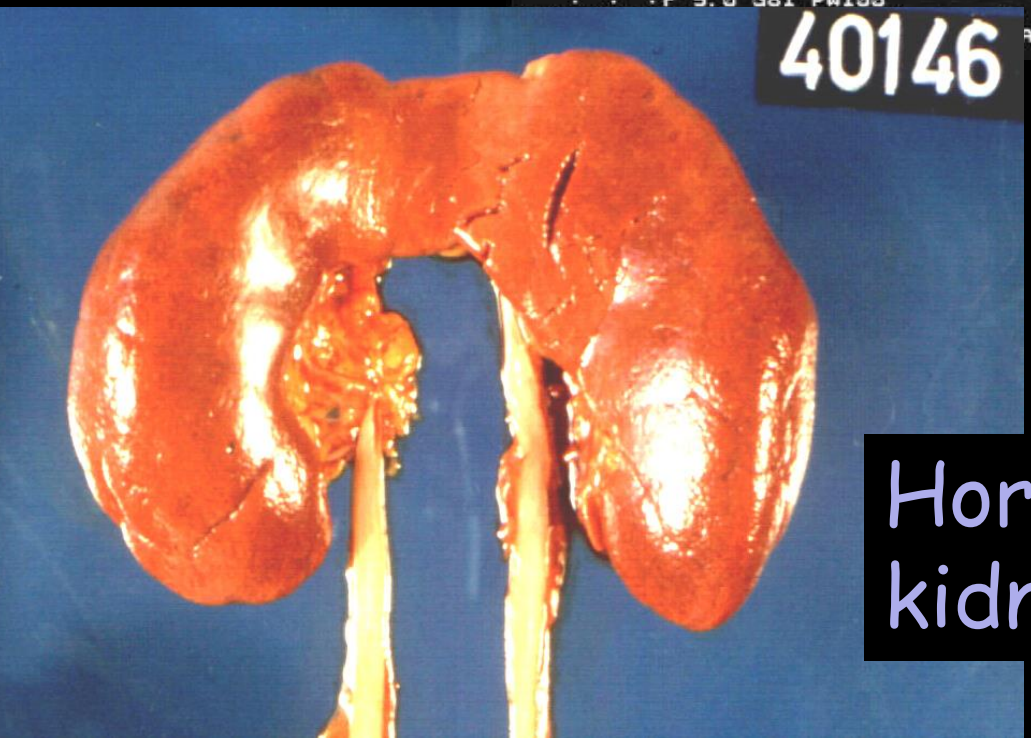
4. ***Horseshoe kidney***

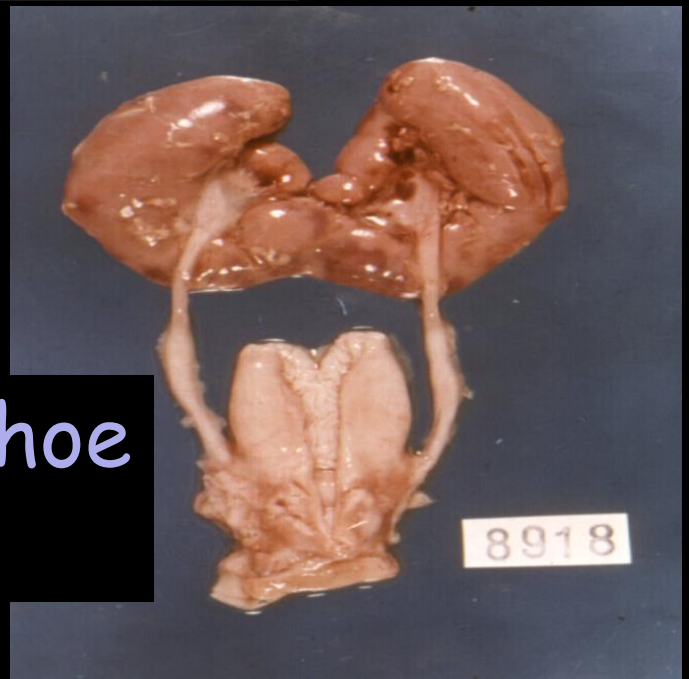
The most common congenital anomaly (1/500-1000) resulting from the fusion of lower (90%) or upper (10%) poles

5. ***Ectopic kidneys***

The kidney lies at the pelvic brim or within the pelvis

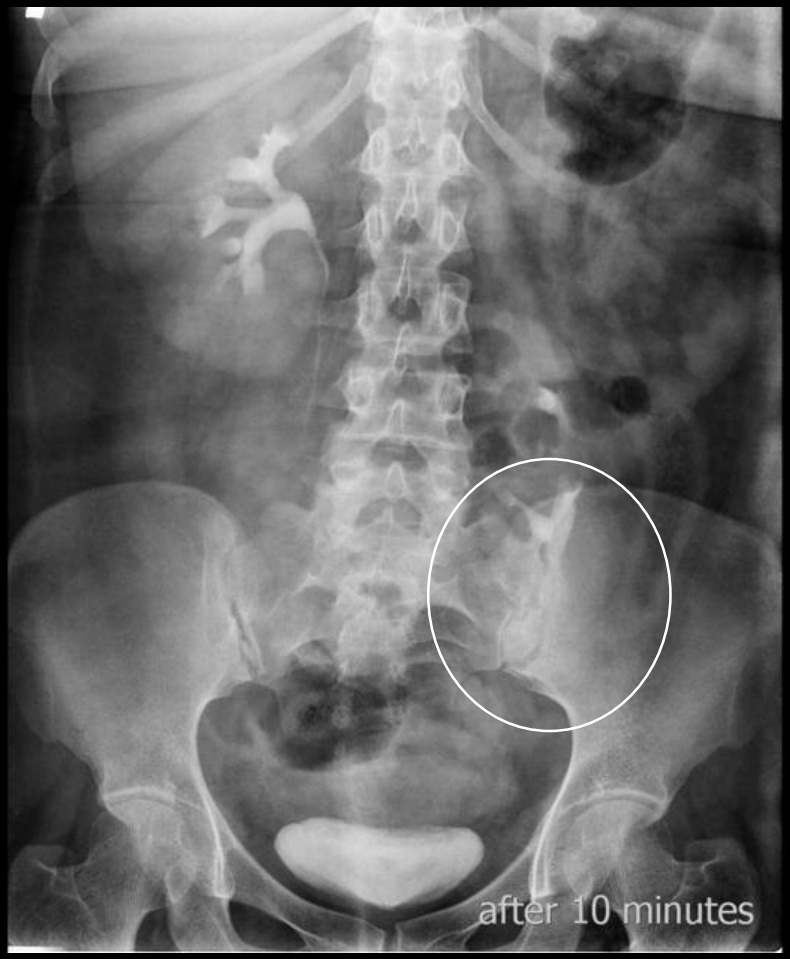


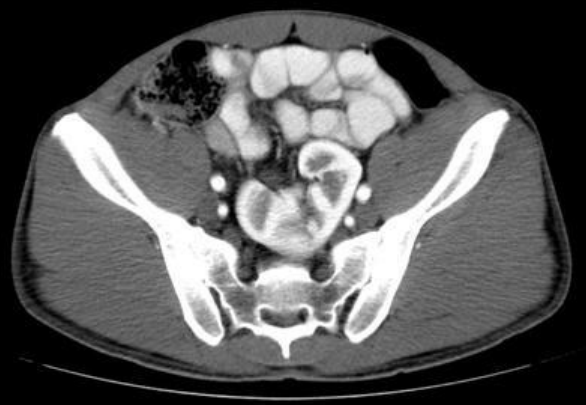




Horseshoe kidney

Renal dystopy



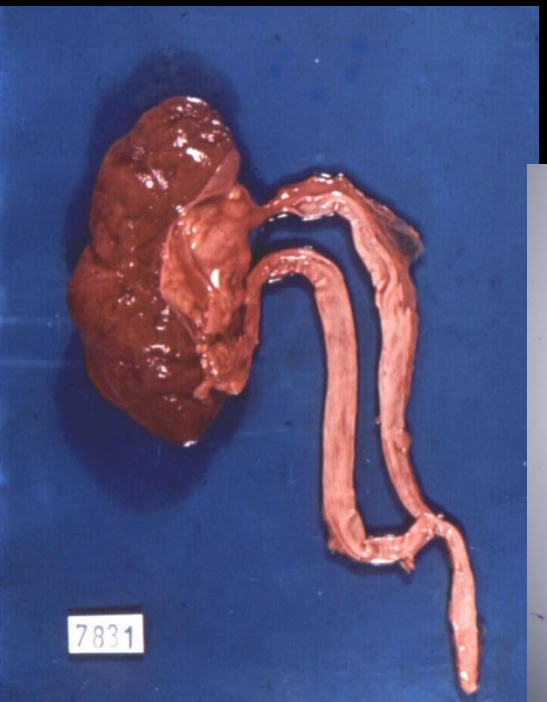


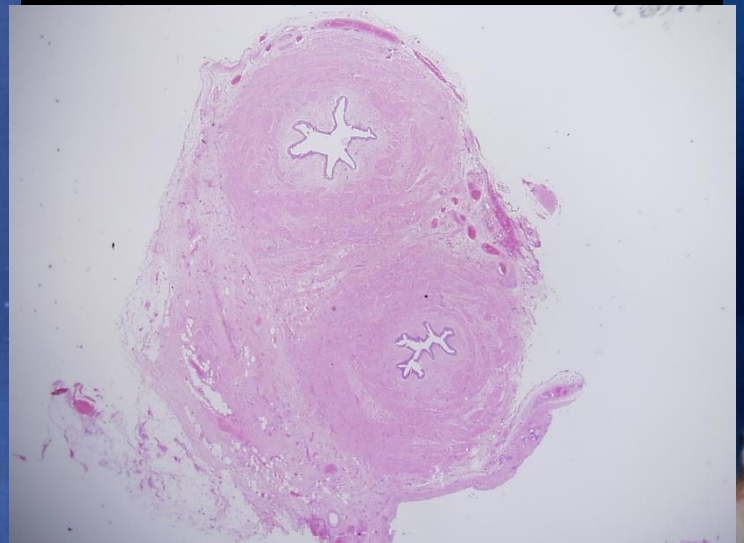
Congenital abnormalities

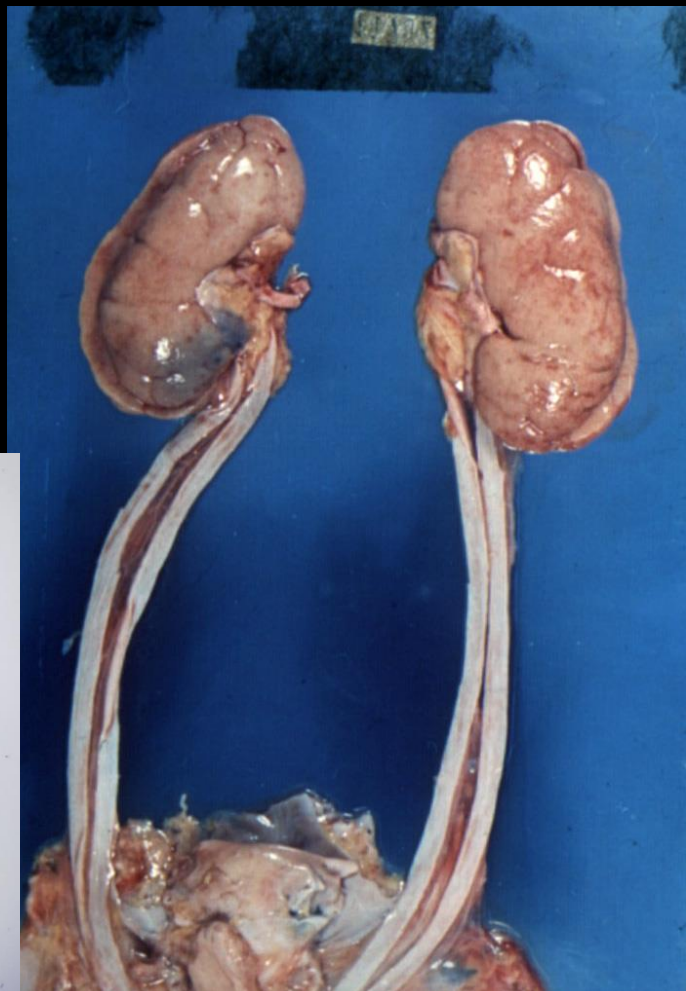
6. *Duplication of the renal pelvis and the ureter*
7. *Ureteropelvic junction stenosis*

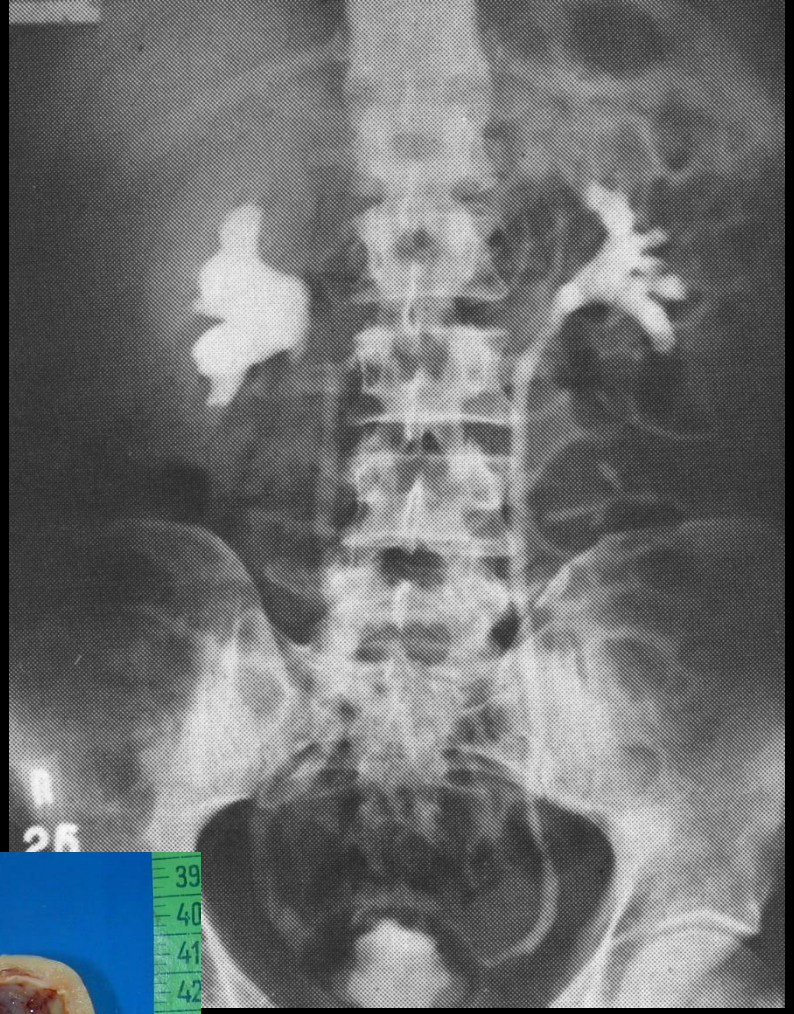
Usually unilateral, leads to hydronephrosis, early operation can save the kidney

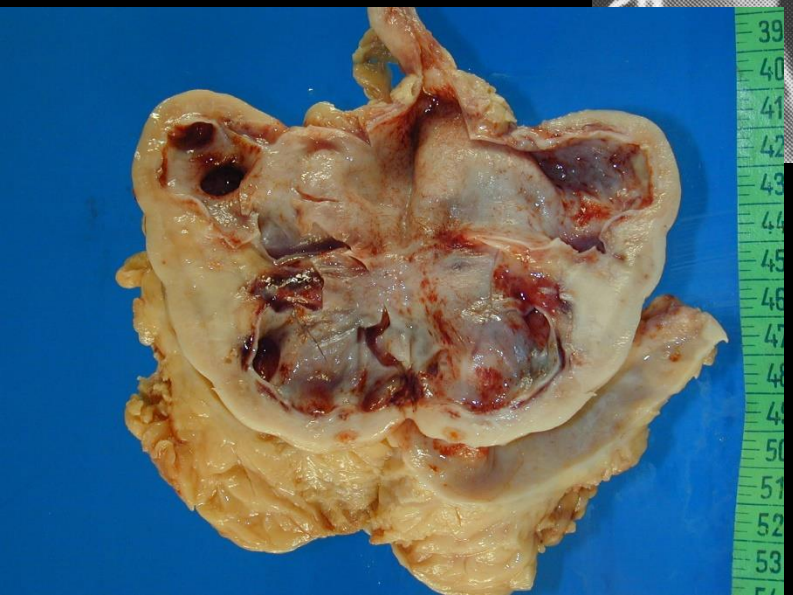
8. *Accessory renal artery*
9. *Multicystic renal dysplasia*







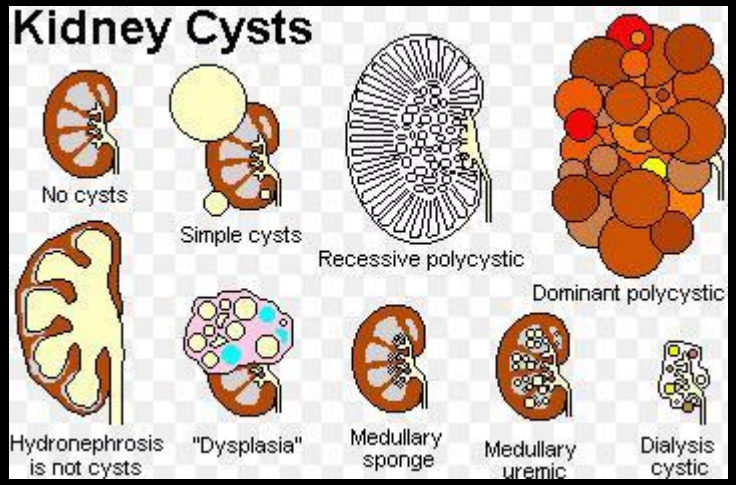




Stricture of the ureteropelvic junction

Cystic diseases of the kidney

Cystic renal dysplasia
Polycystic kidney
Adult (AD)
Infantile (AR)
Medullary cystic („sponge") kidney
Dialysis associated cystic kidney
Simplex cysts
Renal cysts associated with inherited diseases (ST)
Glomerulocystic diseases
Extraparenchymal renal cysts



Renal dysplasia
(Cystic renal dysplasia)

Developmental anomaly, NOT neoplastic process!

Sporadic

Unilateral, (or bi~) complete or segmental

Metanephric differentiation abnormality

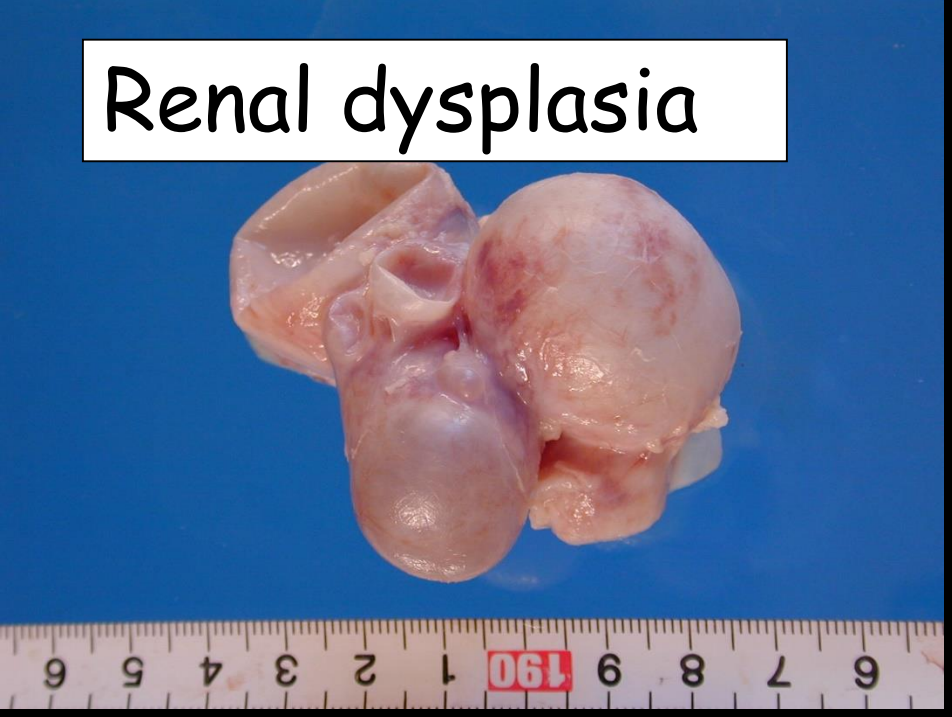
Deficient development of the collecting ducts combined with abnormalities of the lower urinary tract development

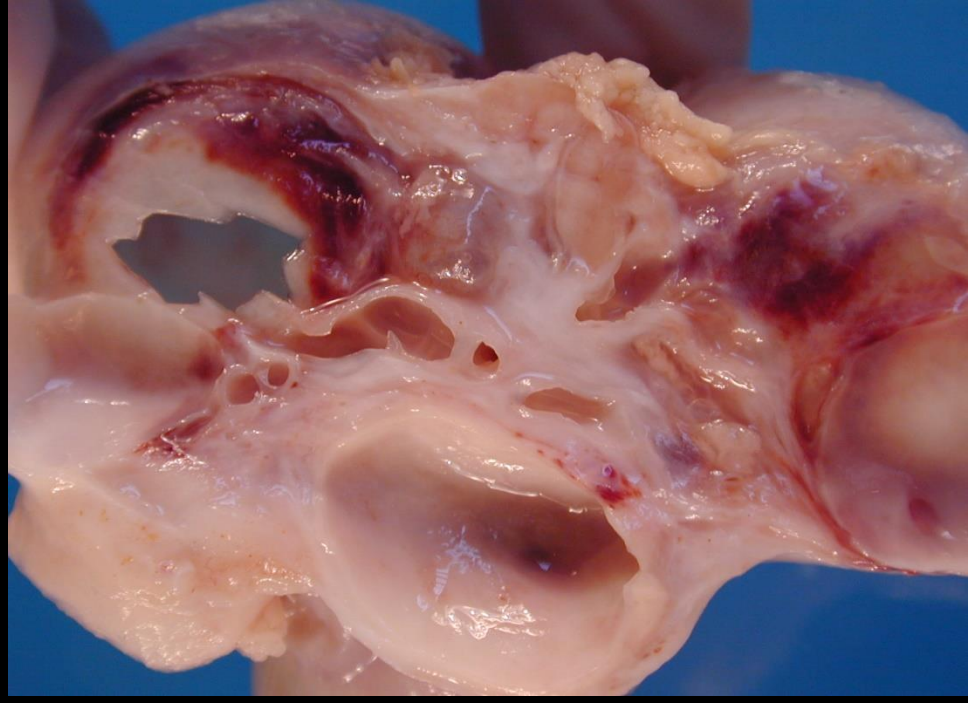
(Stricture of the vesicoureteralis junction, ureter agenesia)

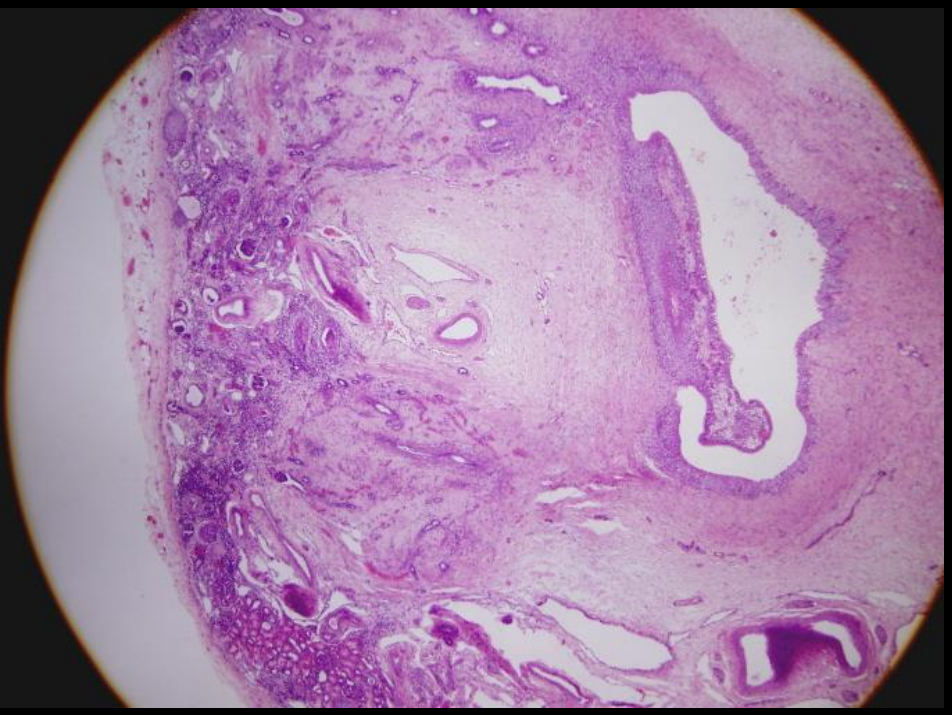
Dg.: palpable abdominal mass

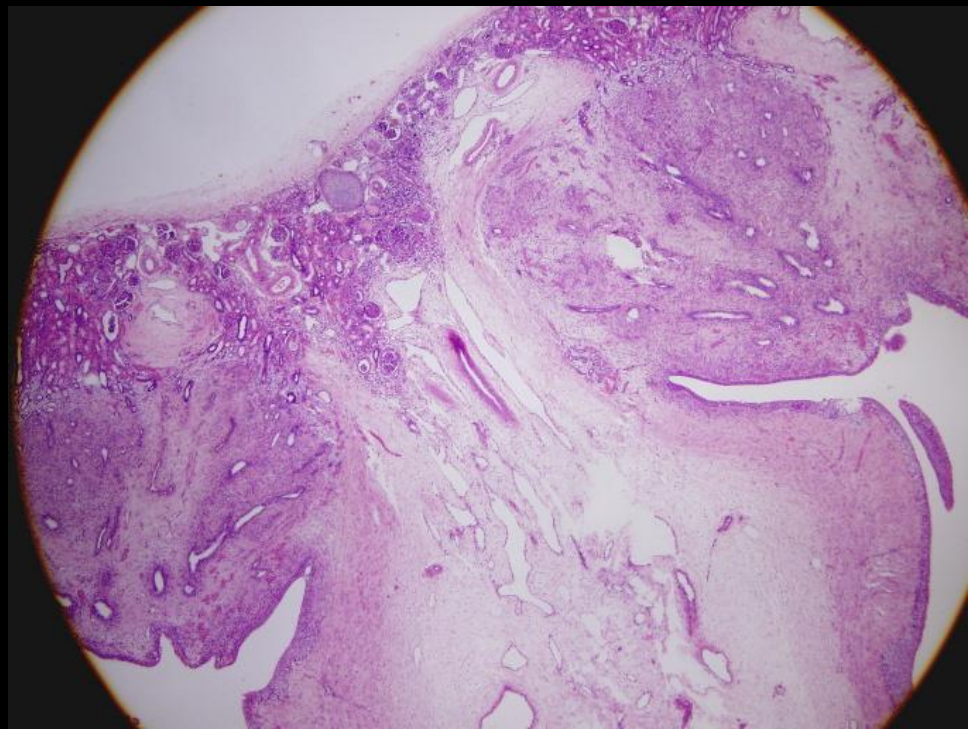
- Abnormal tissue elements (cartilage, undifferentiated mesenchyme)
- Cysts
- Enlarged kidney with insufficent function

Renal dysplasia

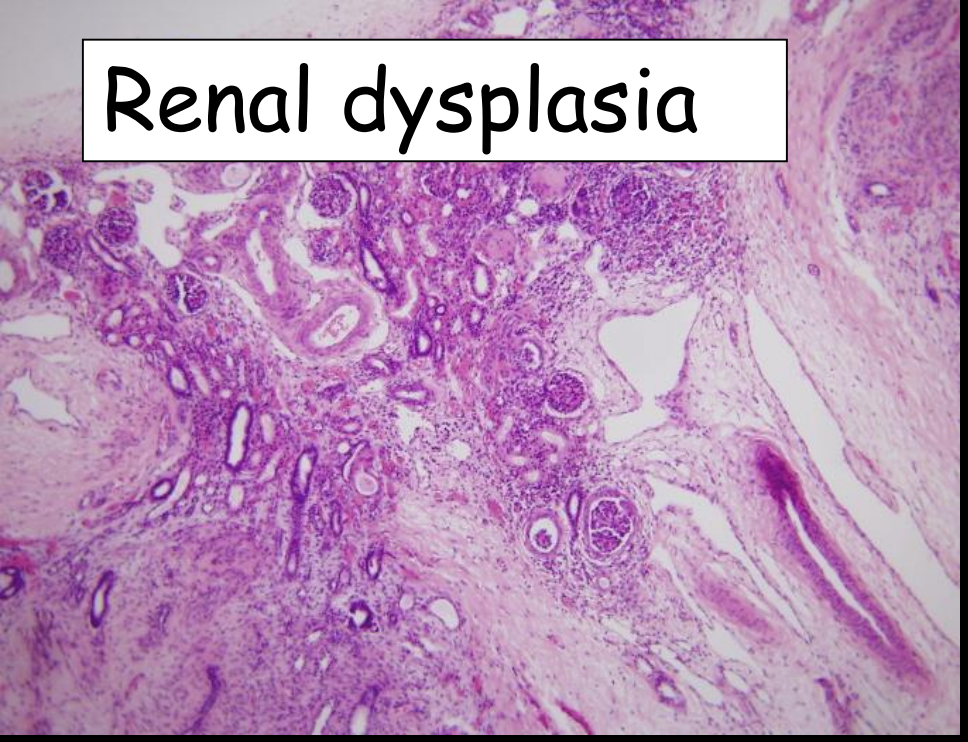


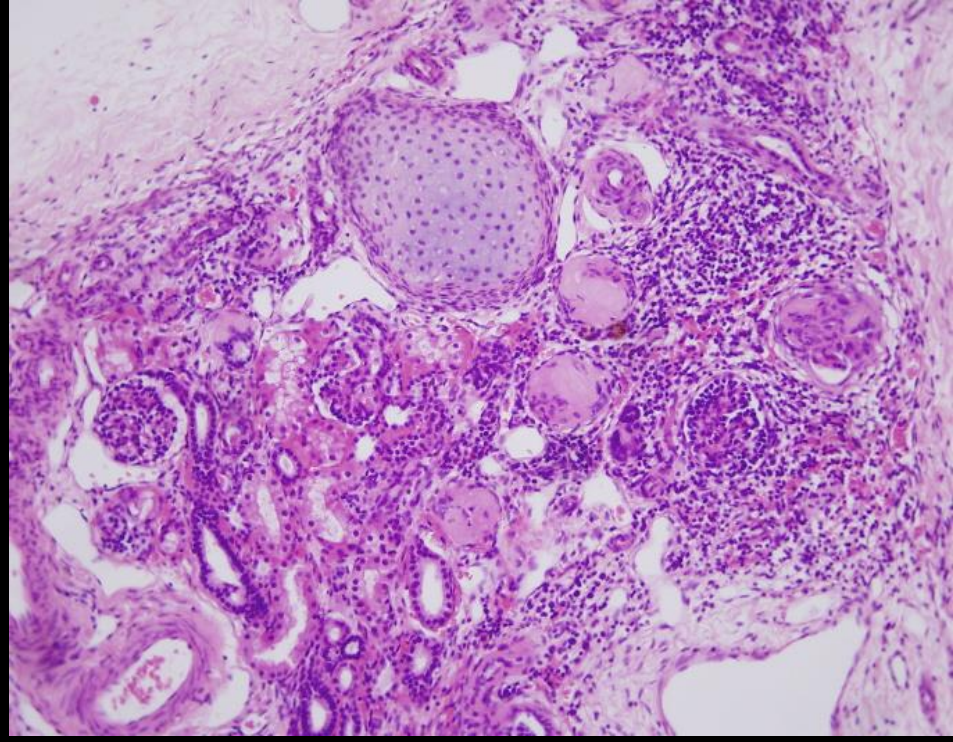


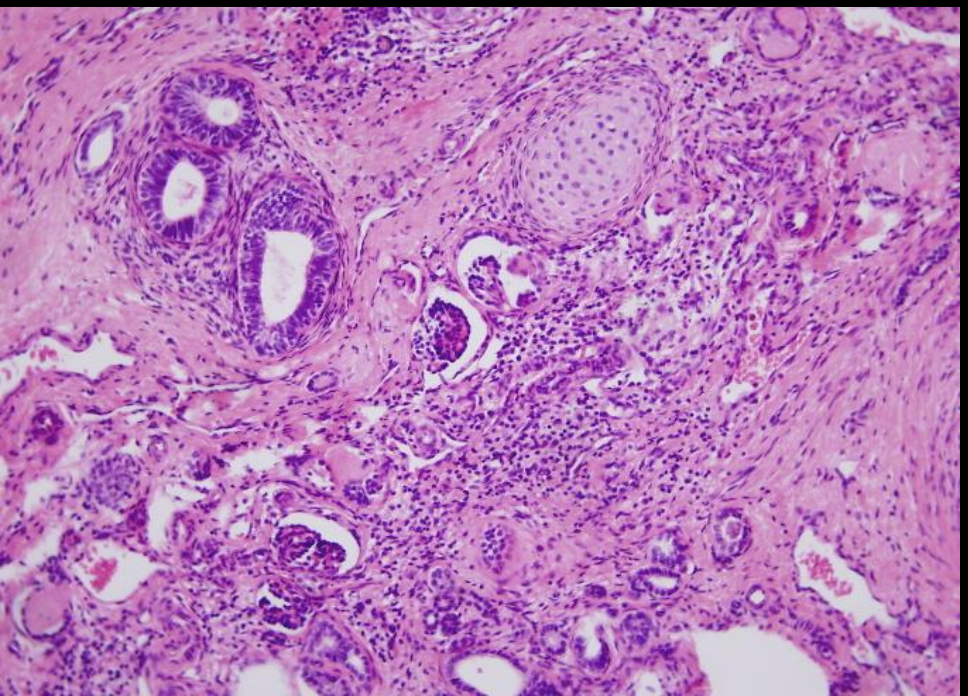


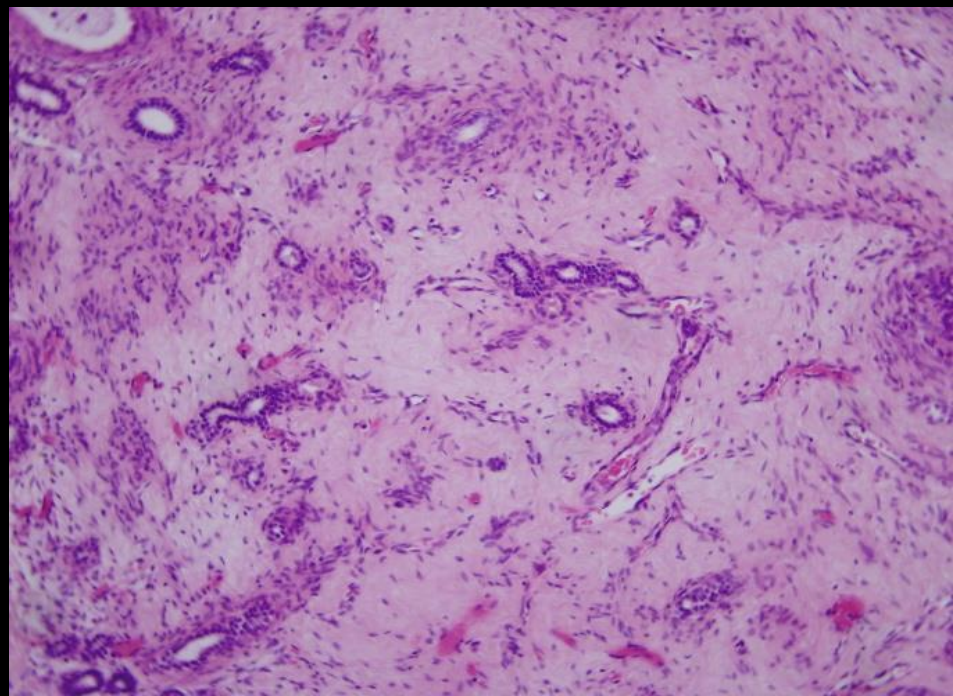


Renal dysplasia









Polycystic kidney (ADPKD)

Occurrence: 1: 400-1000

CRF 5-10%

AD

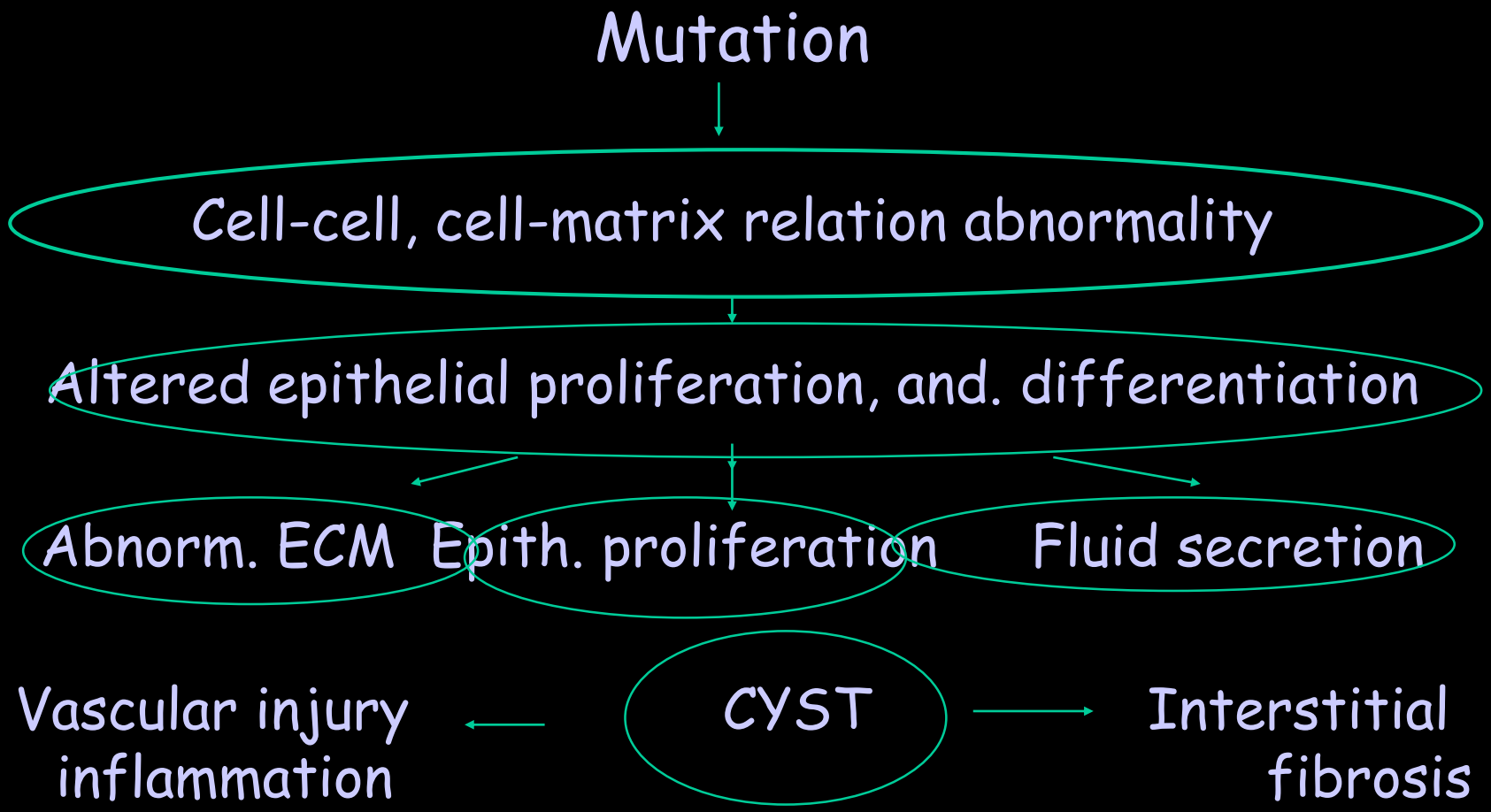
PKD1 : 16p13.3 (85%) RF: 40 y.: 5%, 50 y: 35%, 60 y.: 70%, 70y:95%

polycystin-1

PKD2 : 4q21 (15%) RF: 0, 5, 15, 45

polycystin-2

Scenario of the development of ADPKD



Polycystic kidney – other accompaniing congenital anomalies

Hepatic cysts

Berry anerysms

Mitralis prolapse – other valvular anomalities

Th.: Dialysis, renal transplatation

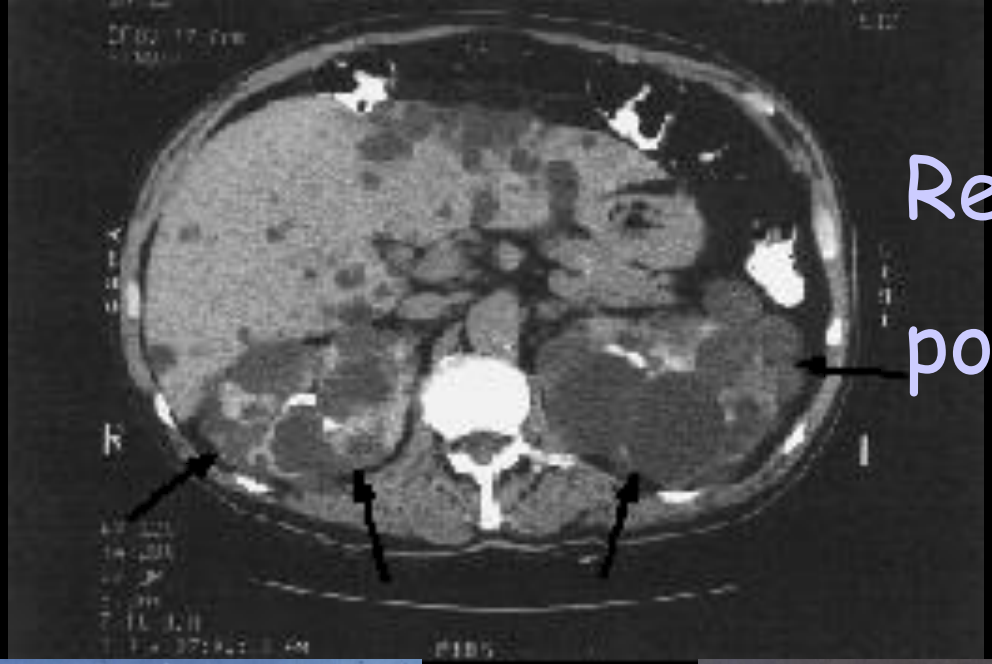
† :

40 % : coronary / hypertensive heart disease

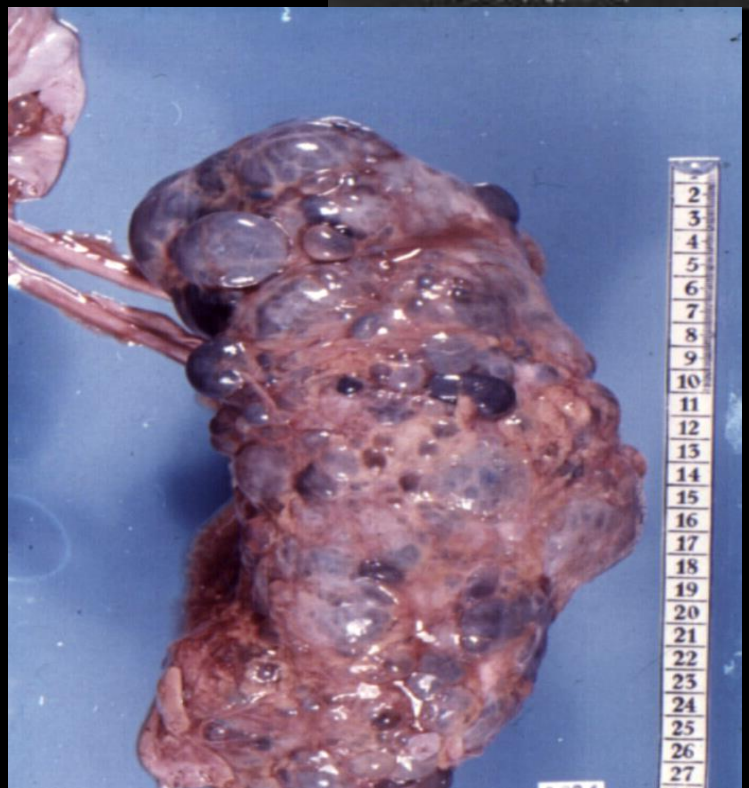
25 % : infection

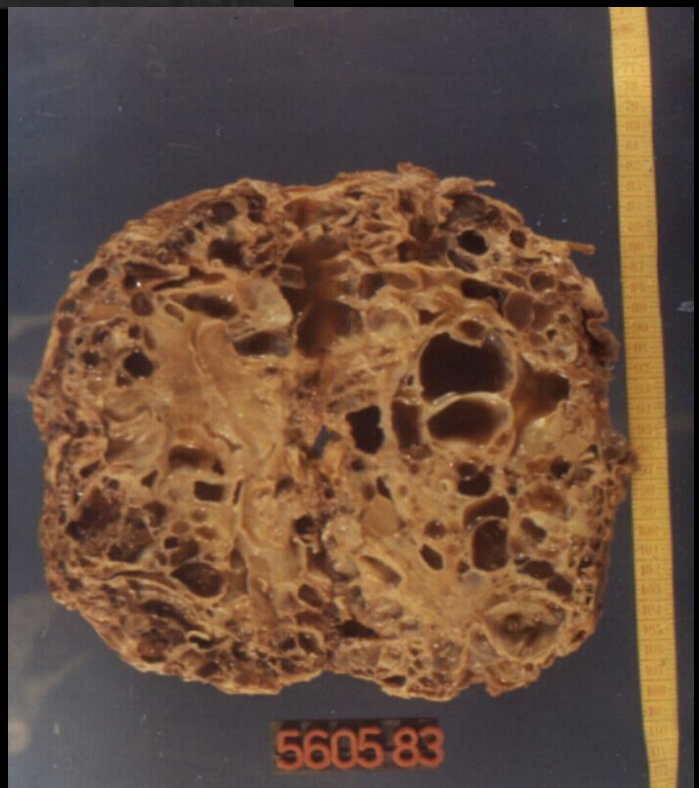
15 % : aneurysm rupture, hypertensive intracerebral haemorrhage

20% : other causes



Ren (hepar) policysticum





Polycystic kidney – clincal signs

Pain

Haematuria

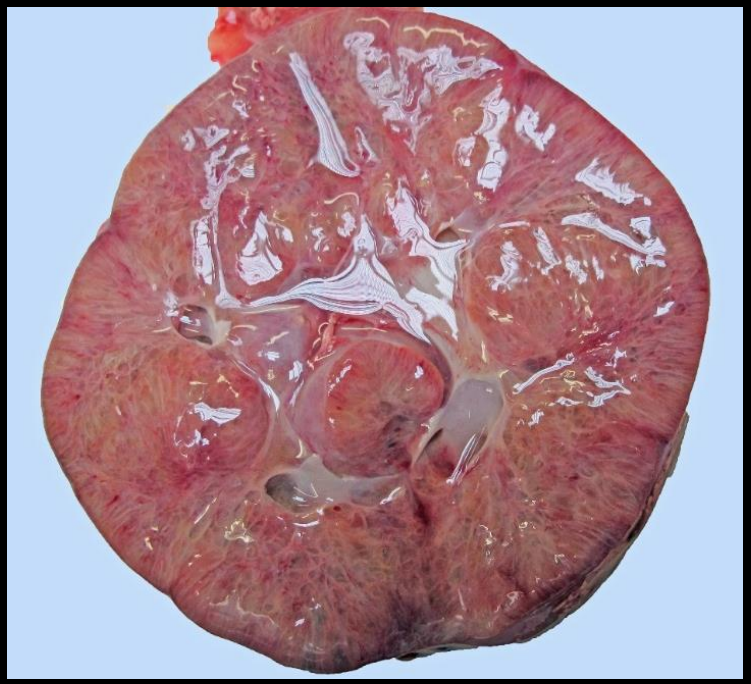
Progressive renal insufficiency

Proteinuria (not more than 2 g / day)

Polyuria

Hypertension

Autosomal-recessive (ARPKD)



Perinatal, neonatal, infantile, juvenile subcategories

PKDH1 gene (fibrocystin)

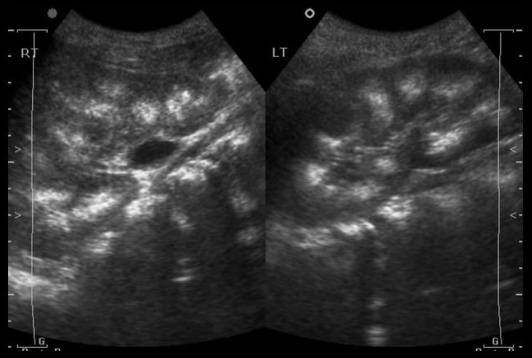
Markedly enlarged kidneys

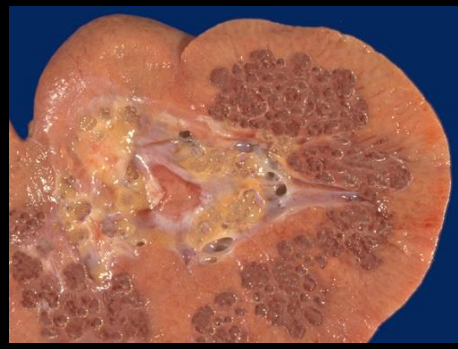
Lung hypoplasia, oligohydramnion

Medullary and cortical elongated cysts, sponge-like appearance

Usually leads to death within the first month of life

Cystic diseases of the renal medulla



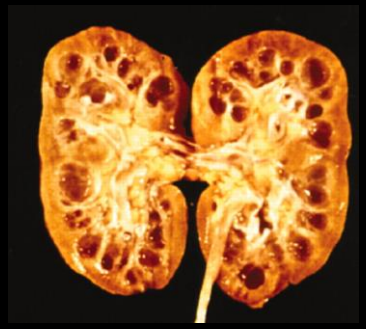


A) Medullary sponge kidney

1-3 mm medullary cysts of the collecting ducts, leads to infections

Does not lead to renal failure

B) Nephronophtysis



Sporadic or familial

Atrophic kidneys, cysts at the corticomedullary junction

Leads to ESRD

Simple cysts

Common finding, single or multiple, do not influence the renal function

Cystic diseases of the kidney

Acquired cystic disease of the kidney

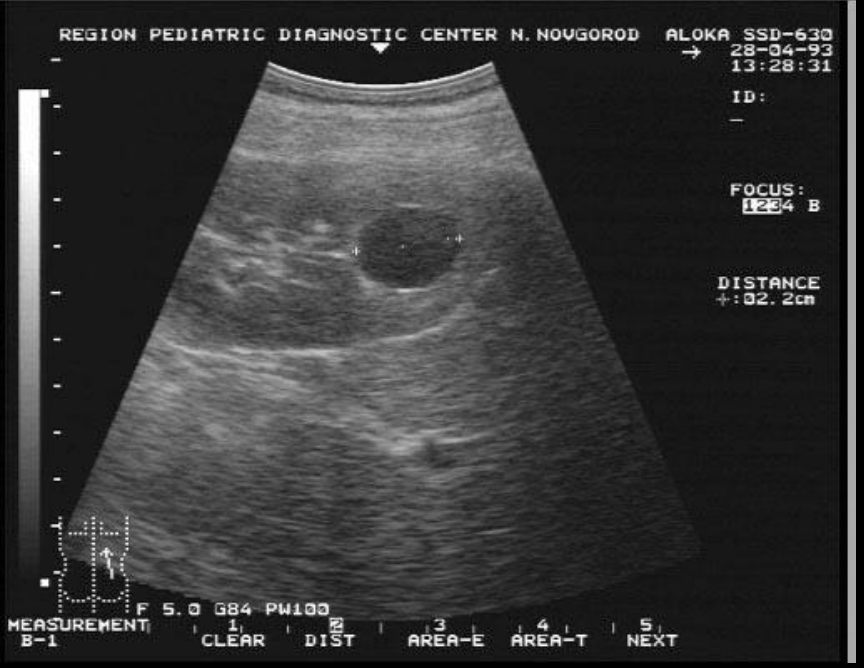
In case of long-standing dialysis

Glomerulocystic kidney disease

Cysts in hereditary syndromes

von Hippel-Lindau syndrome

Tuberous sclerosis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ALOKA SSD-630
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F 5.0 G84 PW100
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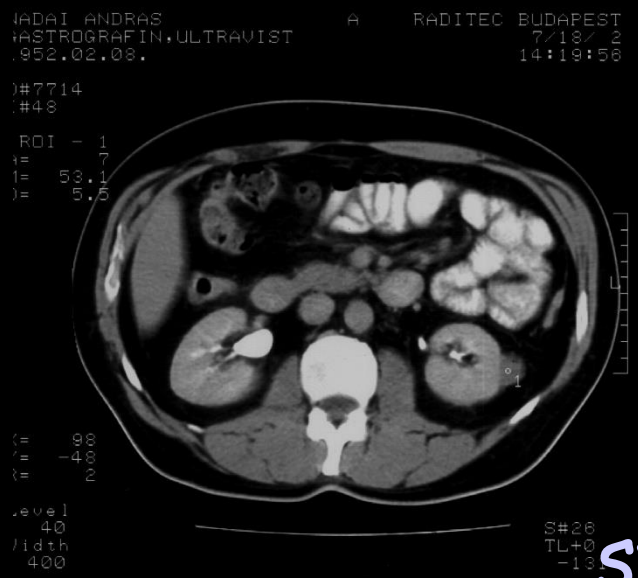
B-1
1 CLEAR
2 DIST
3 AREA-E
4 AREA-T
5 NEXT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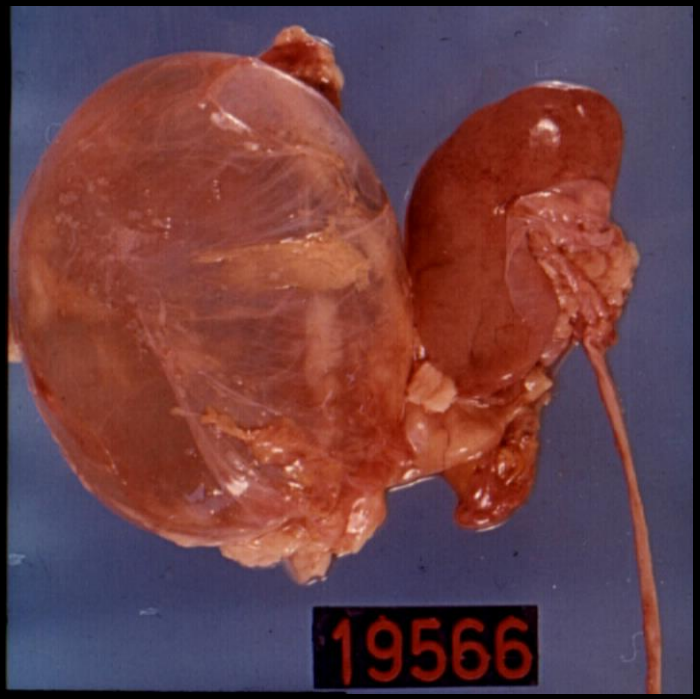
symple cyst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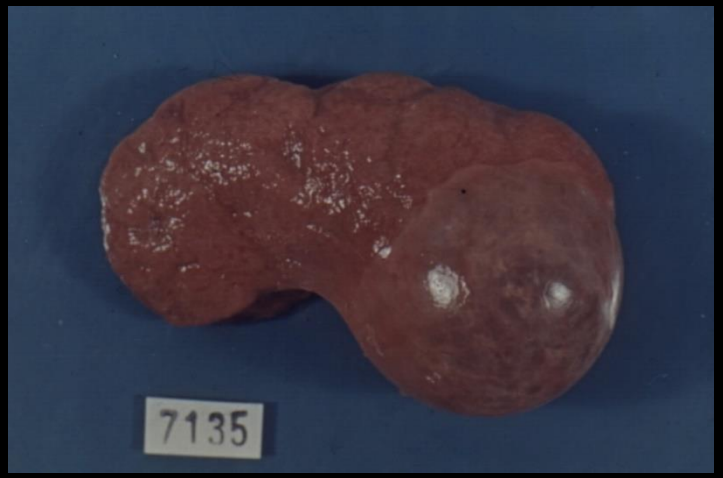
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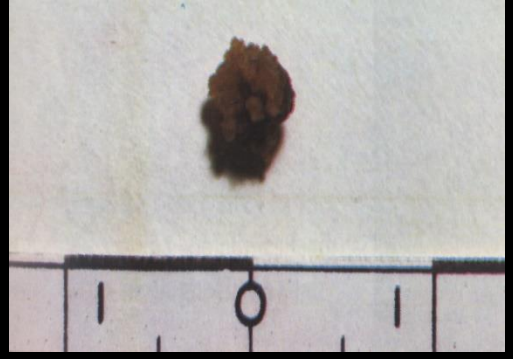
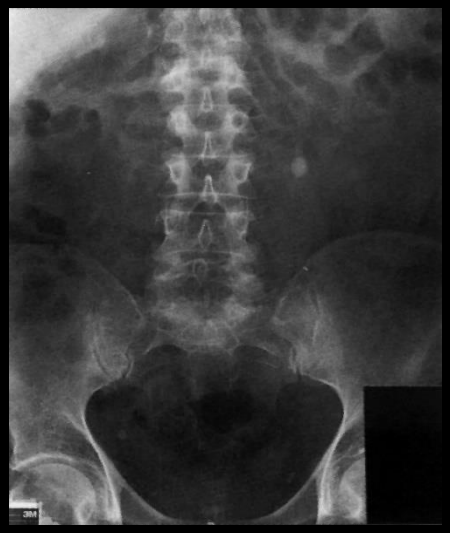
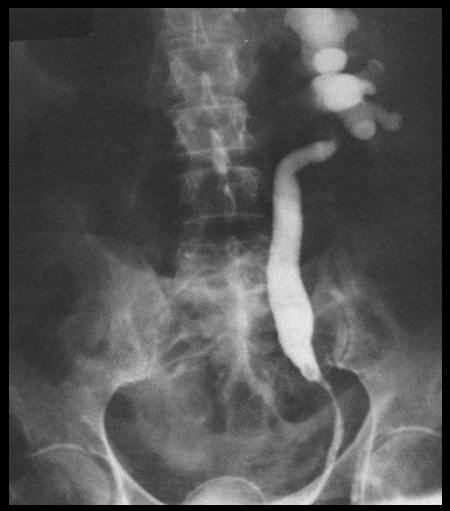
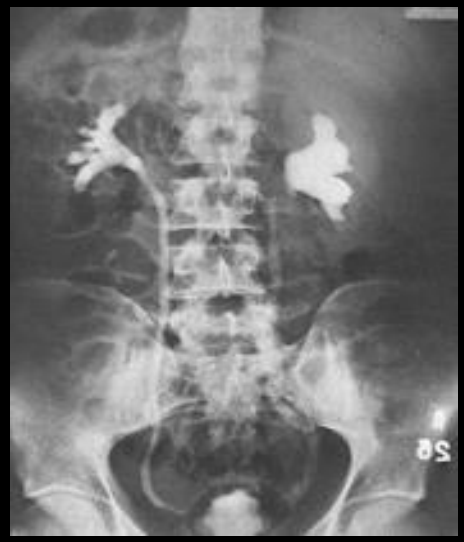


Urolithiasis

Three factors:

Salts that are capable of crystallization

Core that triggers crystallizaton (cell debris, urinary cast)

Lack of inhibitors of crystallization

1. Calcium stones 60-70%

Calcium oxalate/calcium phosphate

Hypercalciuria (with or without hypercalcemia), hyperoxaluria

Brown-black, 1-2 cm, visible by X-ray

2. Struvite stones 15%

Magnesium ammonium phosphate

After infection (e.g., Proteus)

Grey-yellow, staghorn calculi

3. Uric acid stones 15%

Hyperuricemia (gout, rapid cell turnover e.g., leukemias)

White or orange, radiolucent

4. Cystine stones 1-2%

Cystinuria

Clinical features

Uni- (80%) or bilateral

Kidney stone attack:

intense pain in the lower back (lumbal area)

extending into the groin

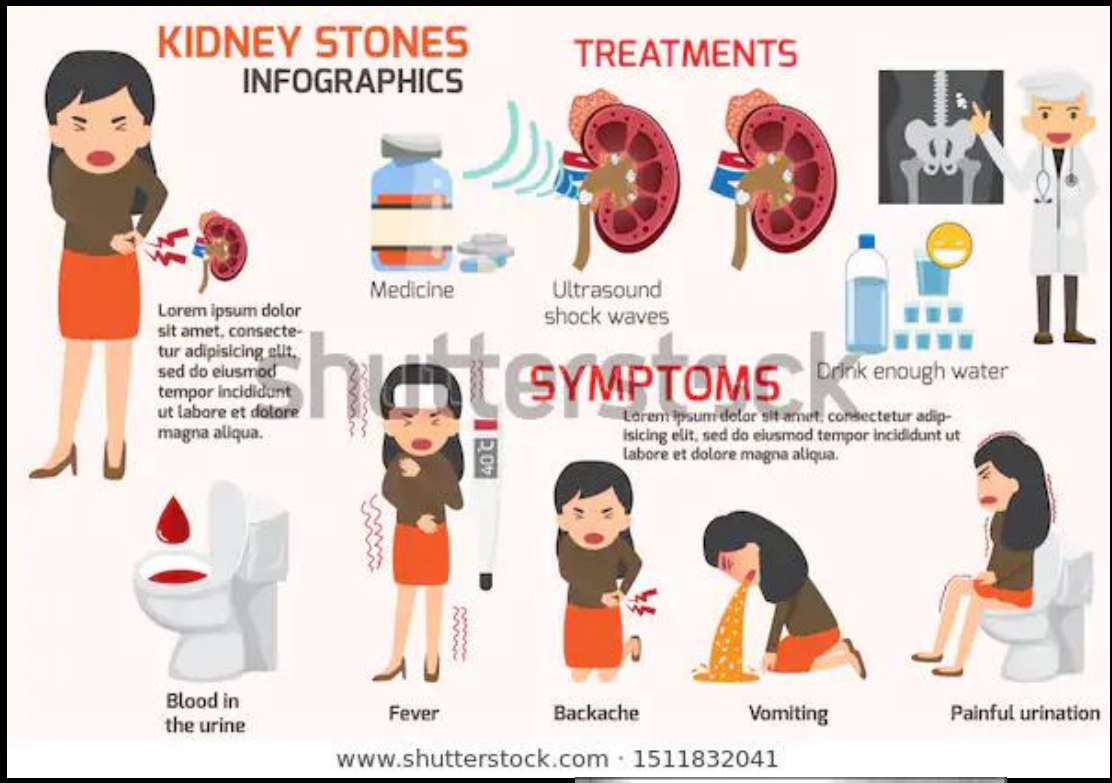
Nausea, vomiting

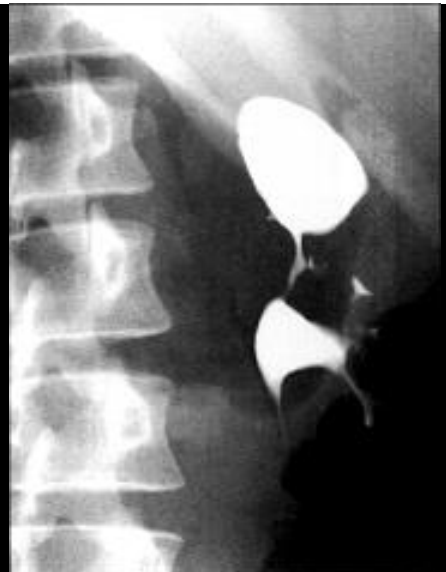
Smaller stones are more hazardous

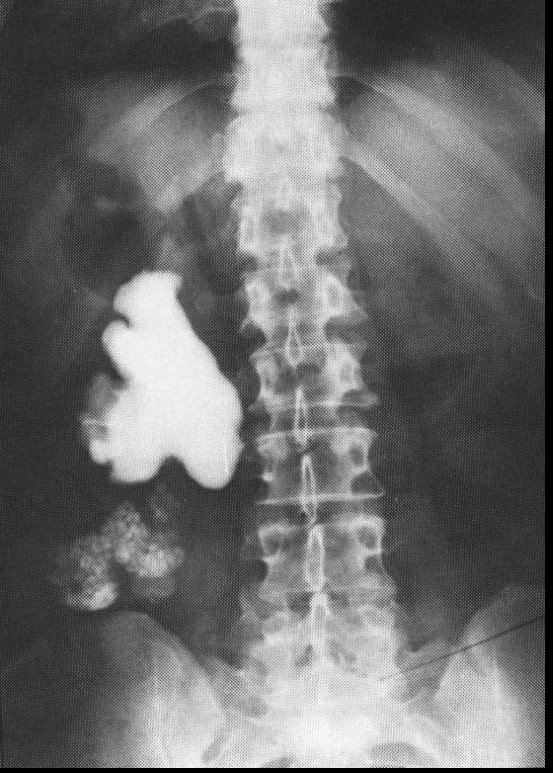
Hematuria

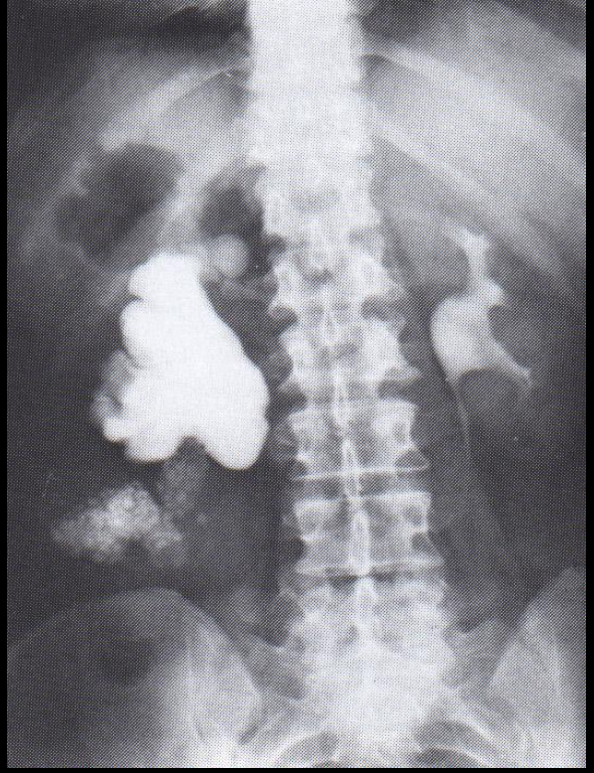
Might be without symptomes

Predisposes for infections







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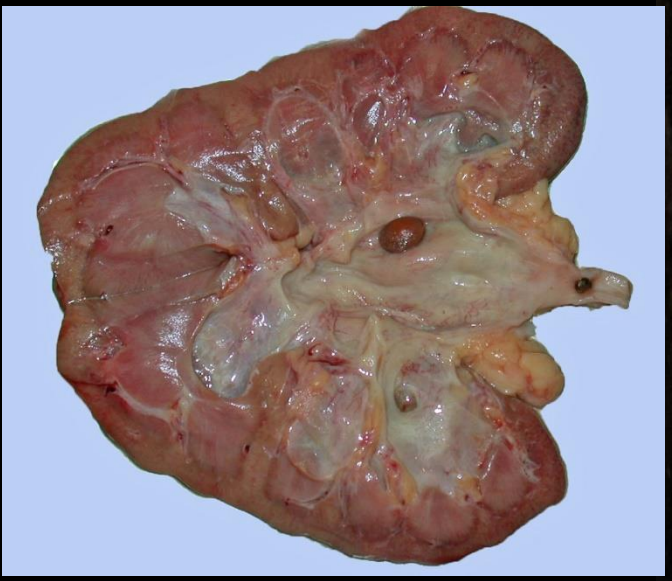
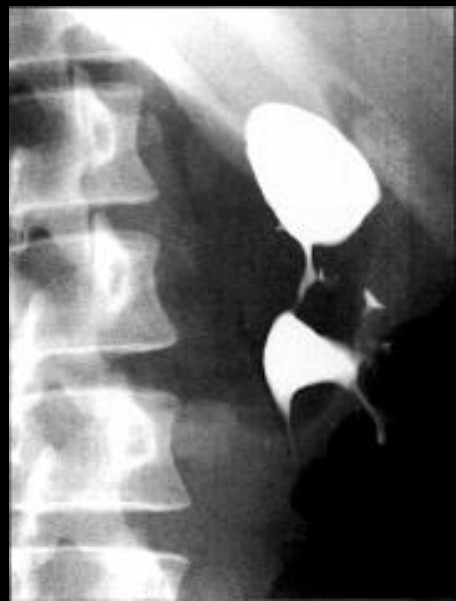
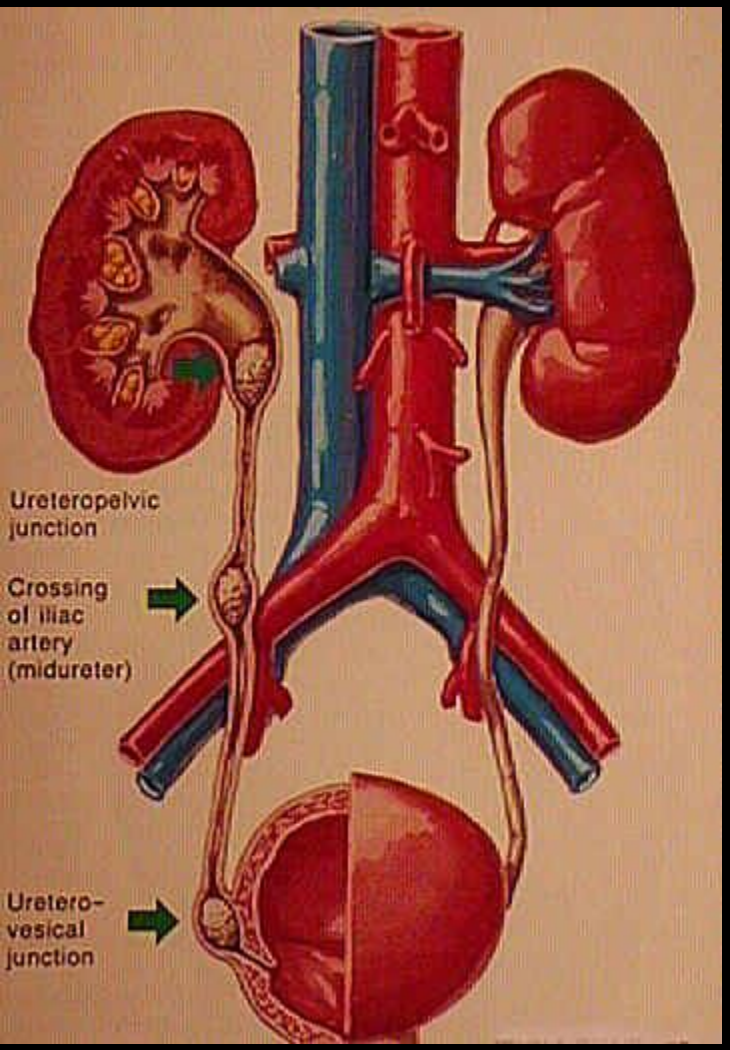
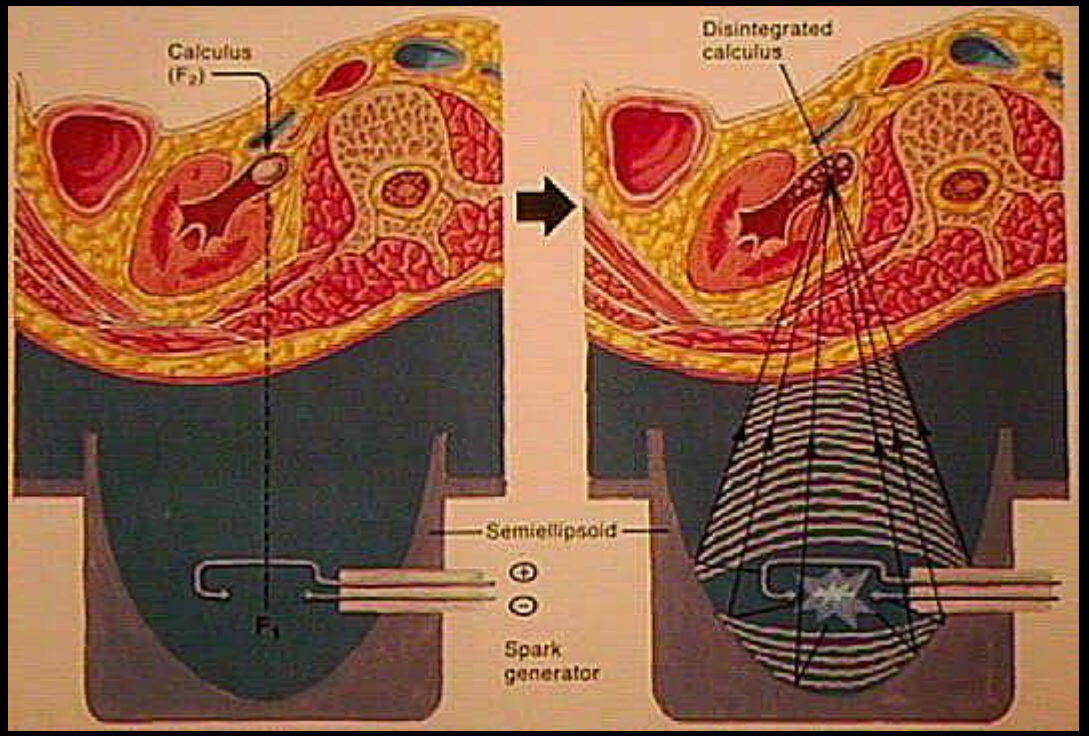






Shock-wave therapy



Causes of Tubulointerstitial Nephritis

Infection

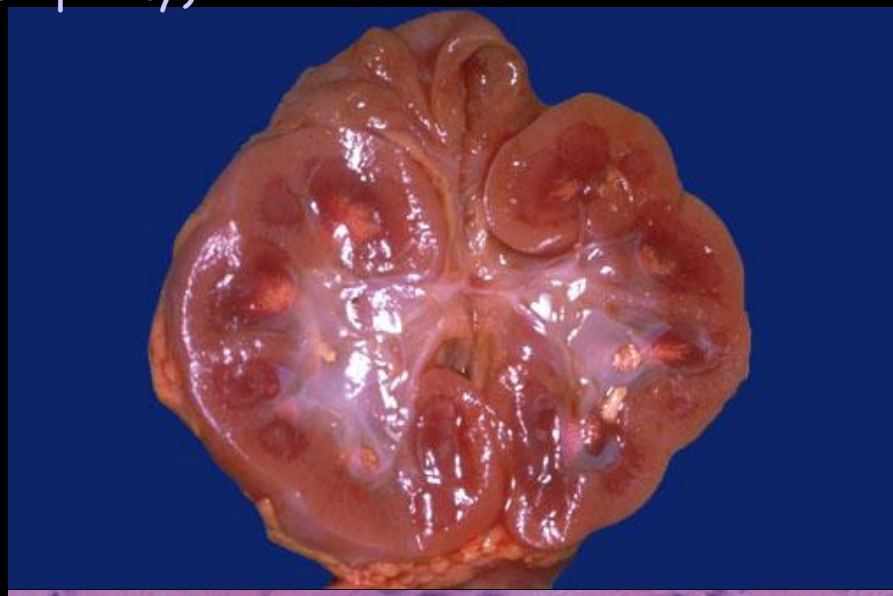
Acut bacterial pyelonephritis
Chronic pyelonephritis (reflux nephropathy)
Other infections (viral, parasitic)

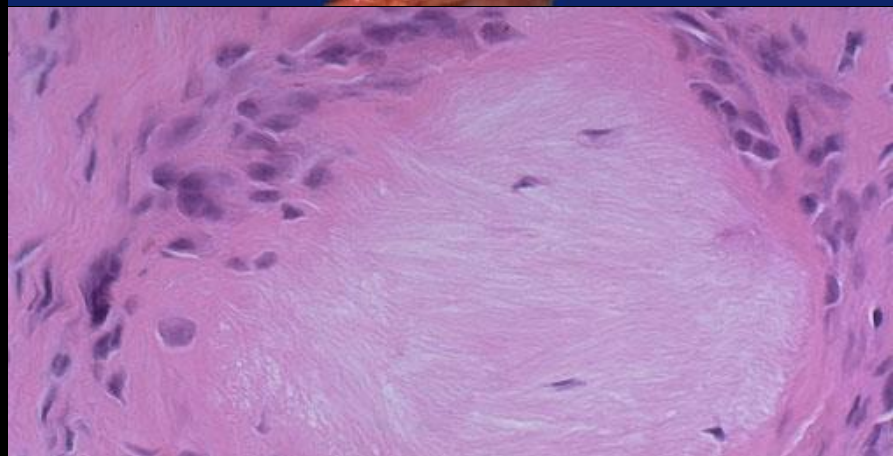
Toxins

Drugs
Acute hypersensitive nephritis
Analgesic nephropathy
Heavy metals
Lead, Cadmium

Metabolic diseases

Urate nephropathy
Hypercalcaemic nephropathy
Hypokalaemic nephropathy
Oxalate nephropathy





Tubulointerstitial nephritis

Physical factors

Chronic obstruction
Radiation nephropathy

Neoplasms

Myeloma multiplex

Immunological diseases

Transplant rejection
Sjögren sy
Sarcoidosis

Vascular disorders

Other

Balkan nephropathy
Nephronophthisis – medullary cystic disease
„Idiopathic" interstitial nephritis

Obstructive uropathy

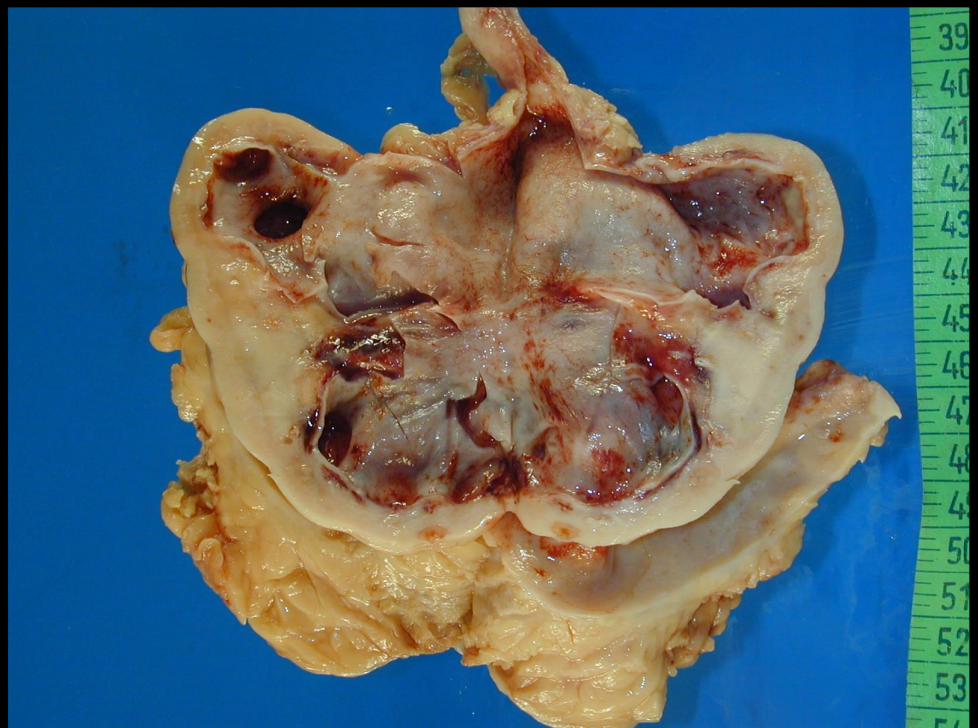
Obstruction predisposes for infections and stone formation

Unrelieved obstruction leads to hydronephrosis

Hydronephrosis:

Dilation of the renal pelvis and calyces

Progressive atrophy of the kidney



Obstructive uropathy

Causes:

1. Congenital anomalies
2. Calculi
3. Prostatic hyperplasia
4. Tumors
5. Lower urinary tract inflammations
6. Pregnancy
7. Uterine prolapse
8. Functional disorders

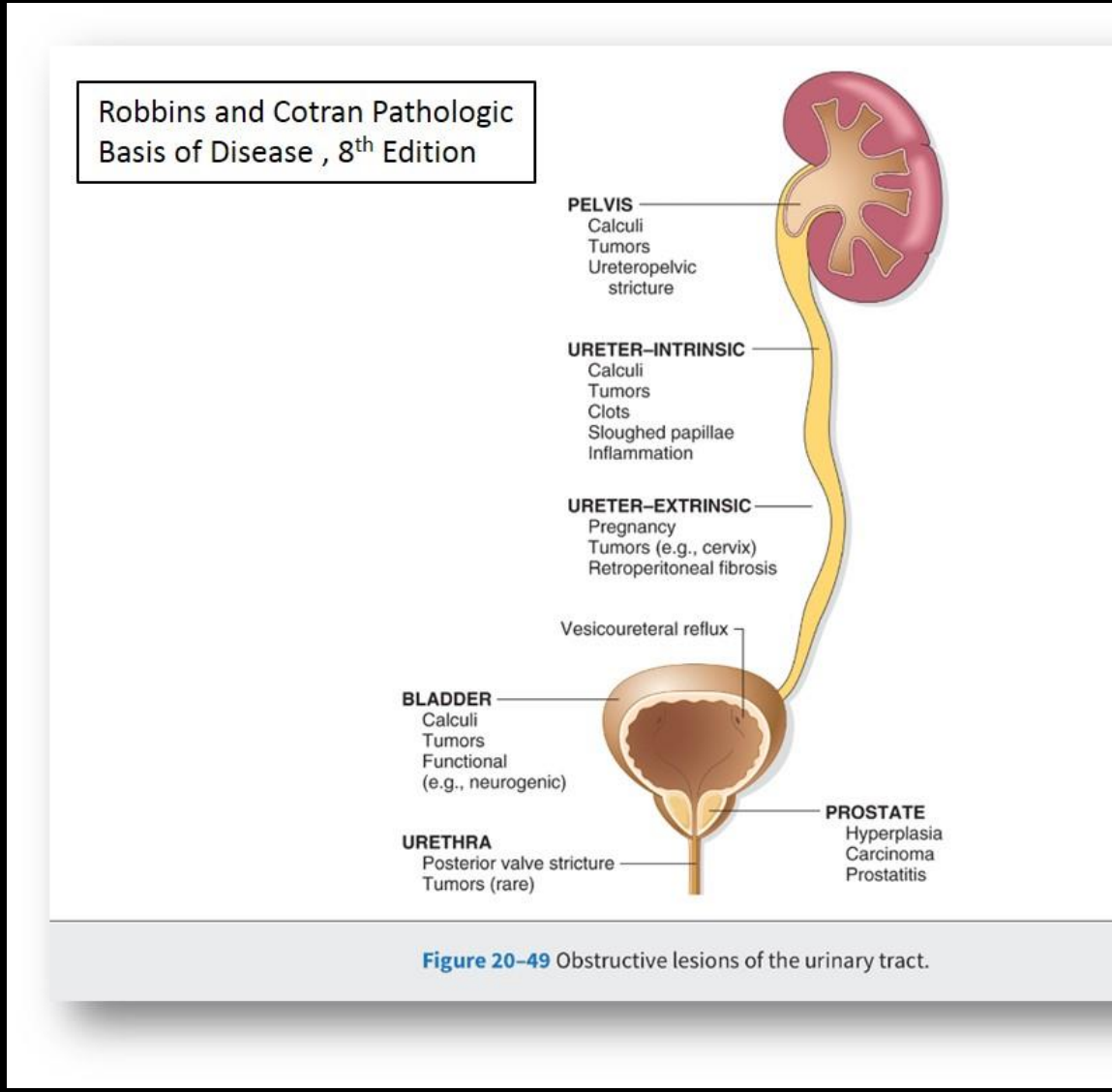


Figure 20–49 Obstructive lesions of the urinary tract.

Obstructive uropathy

Clinical features:

Acute obstruction usually provokes pain

Partial obstruction may remain silent

Partial bilateral obstruction leads to inabilty to concentrate the urine resulting in polyuria followed by chronic tubulointerstitial nephritis

Complete bilateral obstruction leads to oliguria or anuria

Pyelonephritis

1. Acute pyelonephritis

Inflammation of the tubules, the interstitium, the calyces and the renal pelvis

Caused by bacterias (**E. coli**, Proteus mirabilis, Klebsiella, Enterococcus)

Usually consequence of an ascending urinary tract infection

Less commonly result of a hematogenous spread

In normal kidneys, or as a complication of urinary tract disorders (e.g. VUR)

Predisposing factors: catheter, diabetes, pregnancy, lower urinary tract obstruction, immunsuppression

Acute pyelonephritis

Morphology:

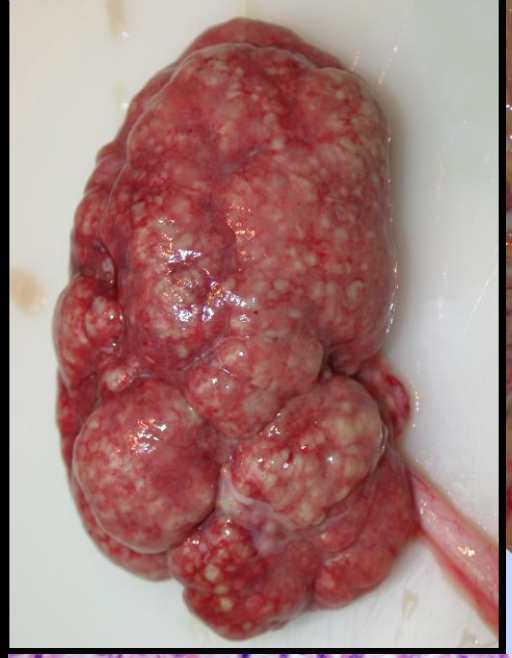
Sligthly enlarged kidney(s)

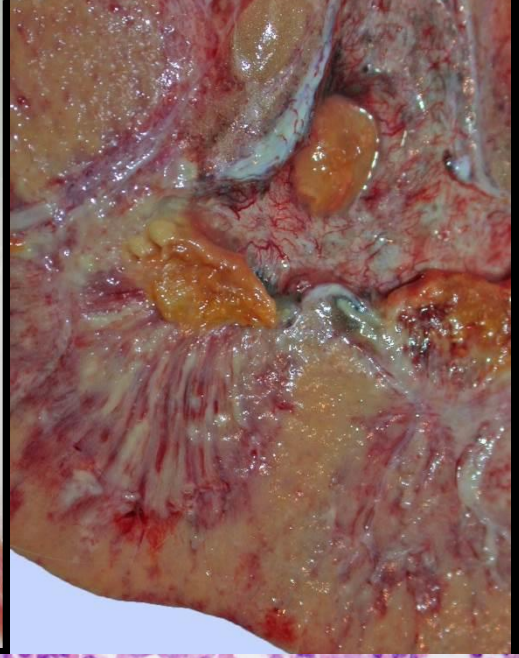
1-3 mm yellowish abscesses on the surface and in the parenchyme (pyelonpehritis apostematosa)

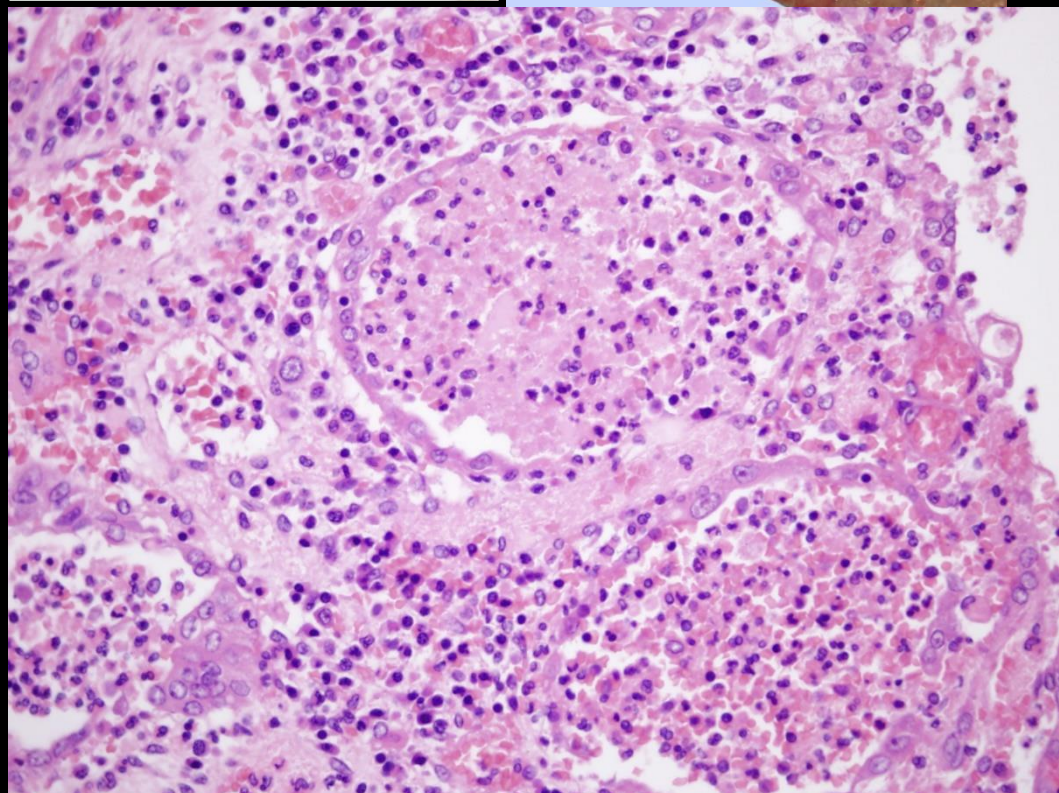
The calyces and the renal pelvis are reddish

Patchy interstitial suppurative inflammation, aggregates of neutrophils in the tubules, tubular necrosis

Glomeruli are also affected in case of hematogenous origin







Acute pyelonephritis

Clinical features:

Uni- or bilateral

Sudden onset with high fever

Pain at the costovertebral angle

Leukocytosis, high sedimentation rate

Pyuria, bacteruria

Usually follows a benign course (with appropriate antibiotic therapy)

Complicated cases can be fatal

Chronic pyelonephritis

Chronic injury of the interstitium and the tubules resulting in scar formation

The renal pelvis and the calyces are also affected

Causes: reflux nephropathy, chronic obstruction

Recurrent infections

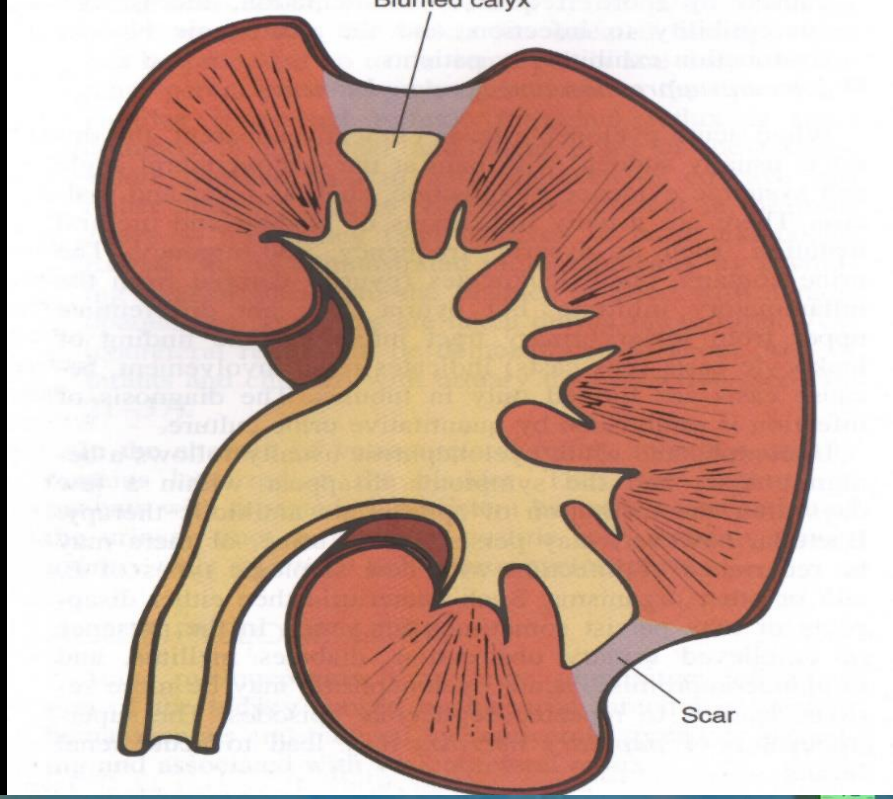
Morphology:

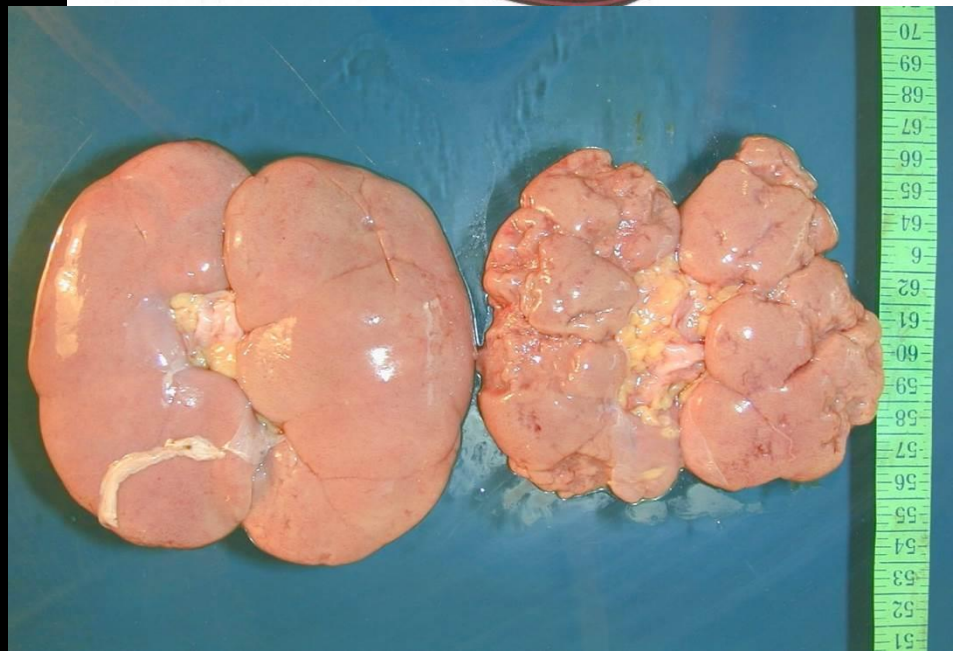
Kidneys are irregularly scarred, corticomedullary scars overlying dilated calyces, flattening of the papillae

The calyces are dilated and their mucosa is thickened

Micr.: Focal interstitial fibrosis, atrophic tubules, tubular casts (thyroidization), lymphoid infiltration

The mucosa of the calyces and pelvis is fibrotic and contains cronic inflammation





Histology:

Lymphoid cell infiltration

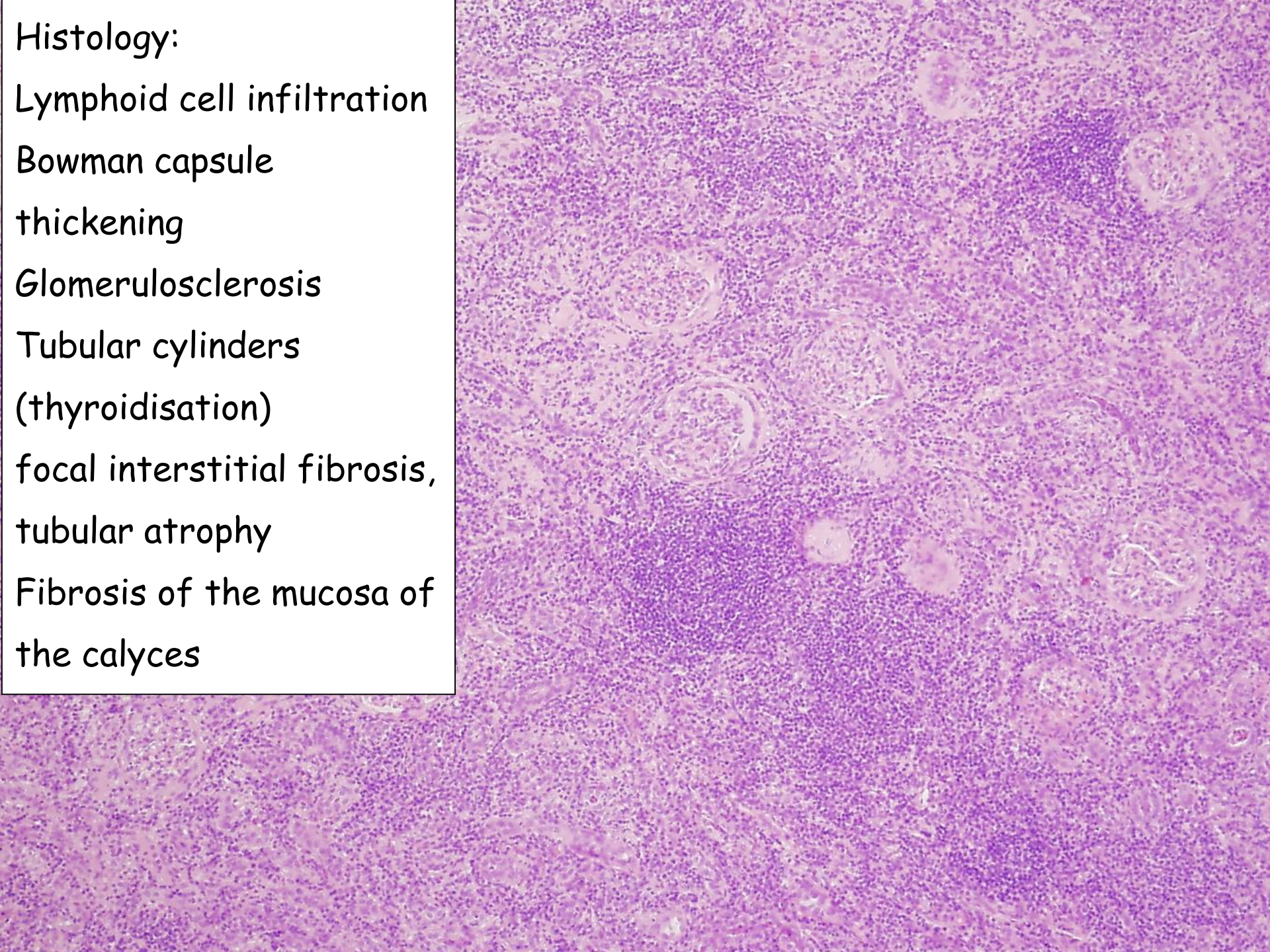
Bowman capsule thickening

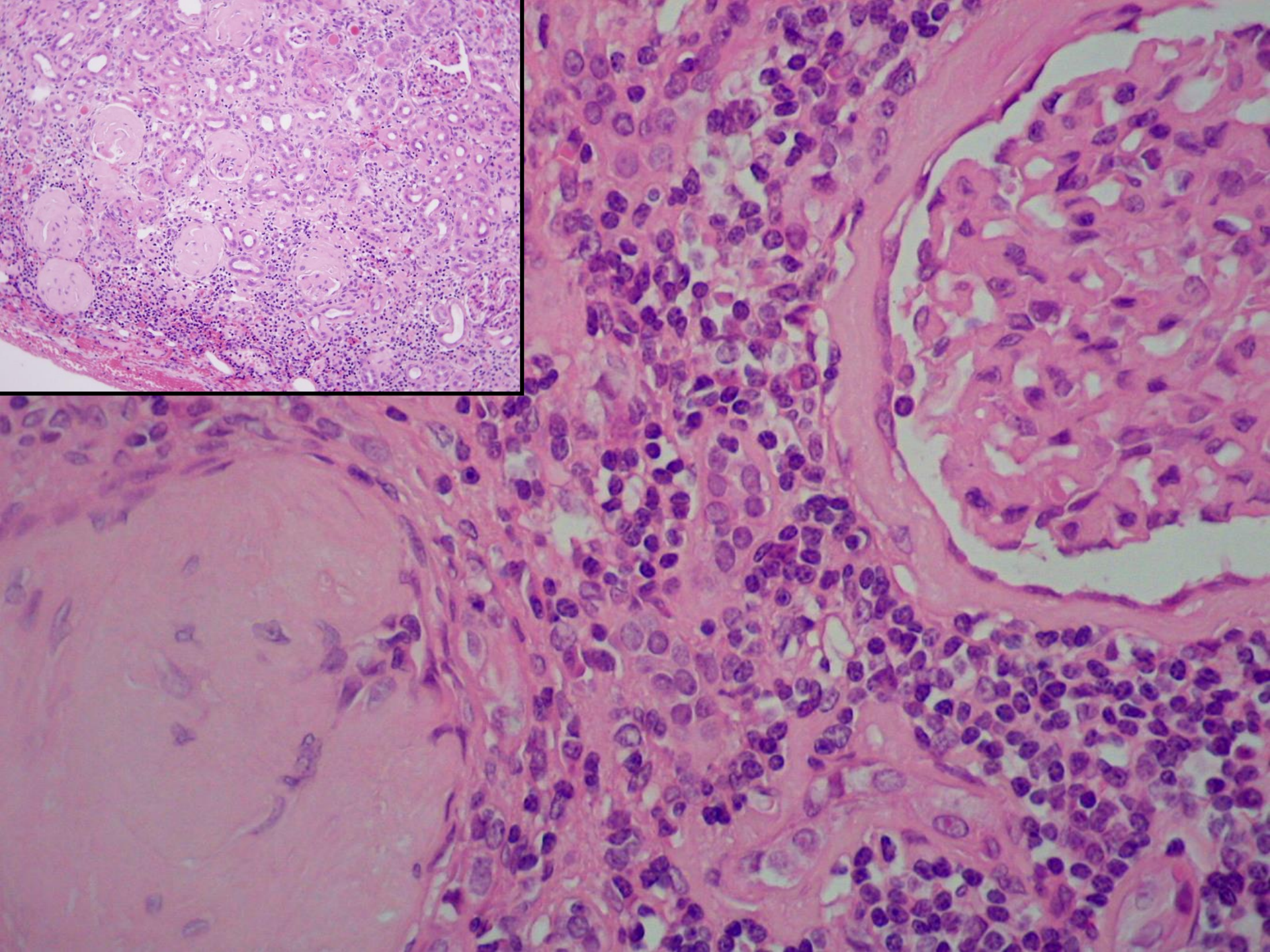
Glomerulosclerosis

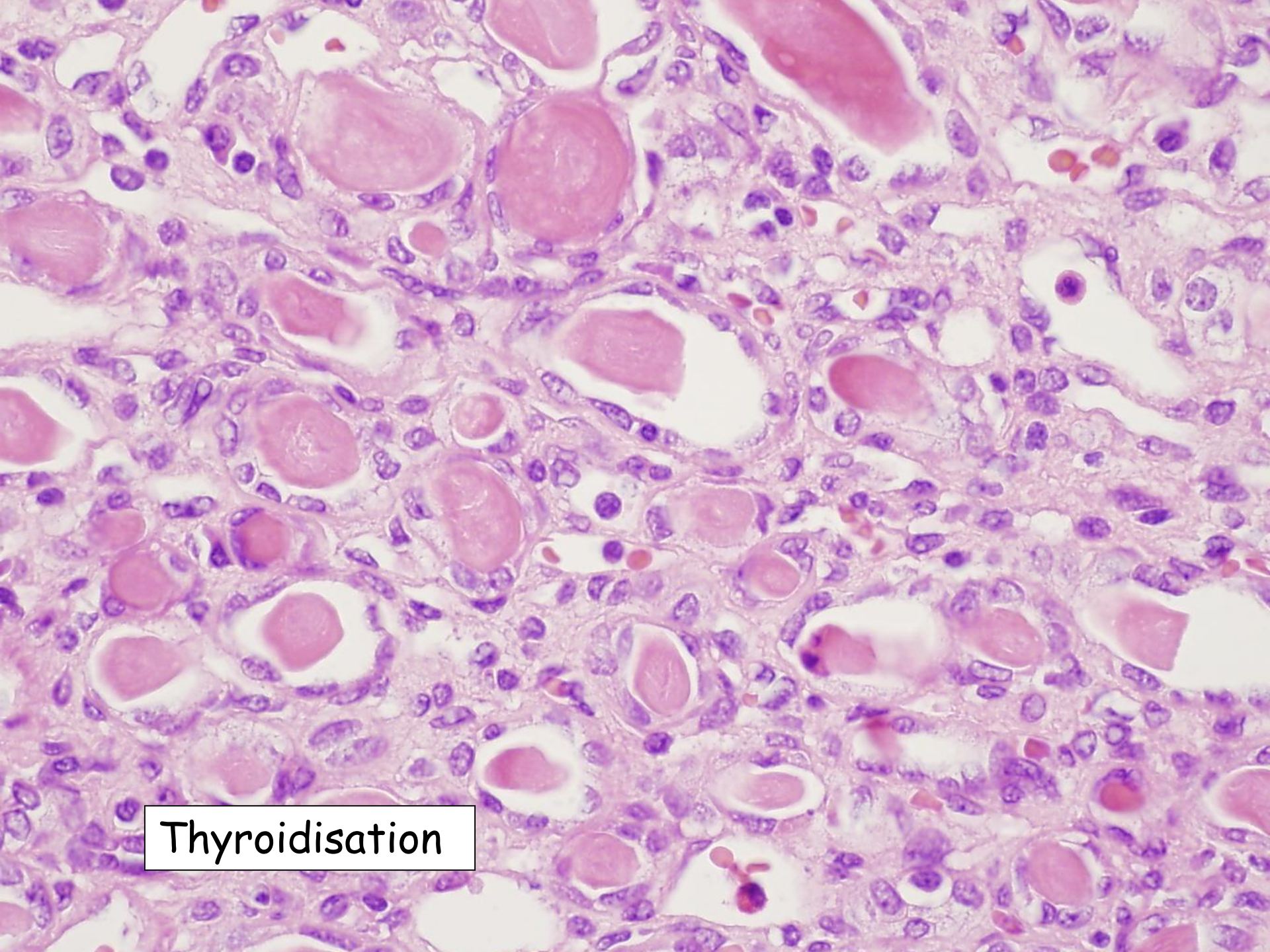
Tubular cylinders (thyroidisation)

focal interstitial fibrosis, tubular atrophy

Fibrosis of the mucosa of the calyces

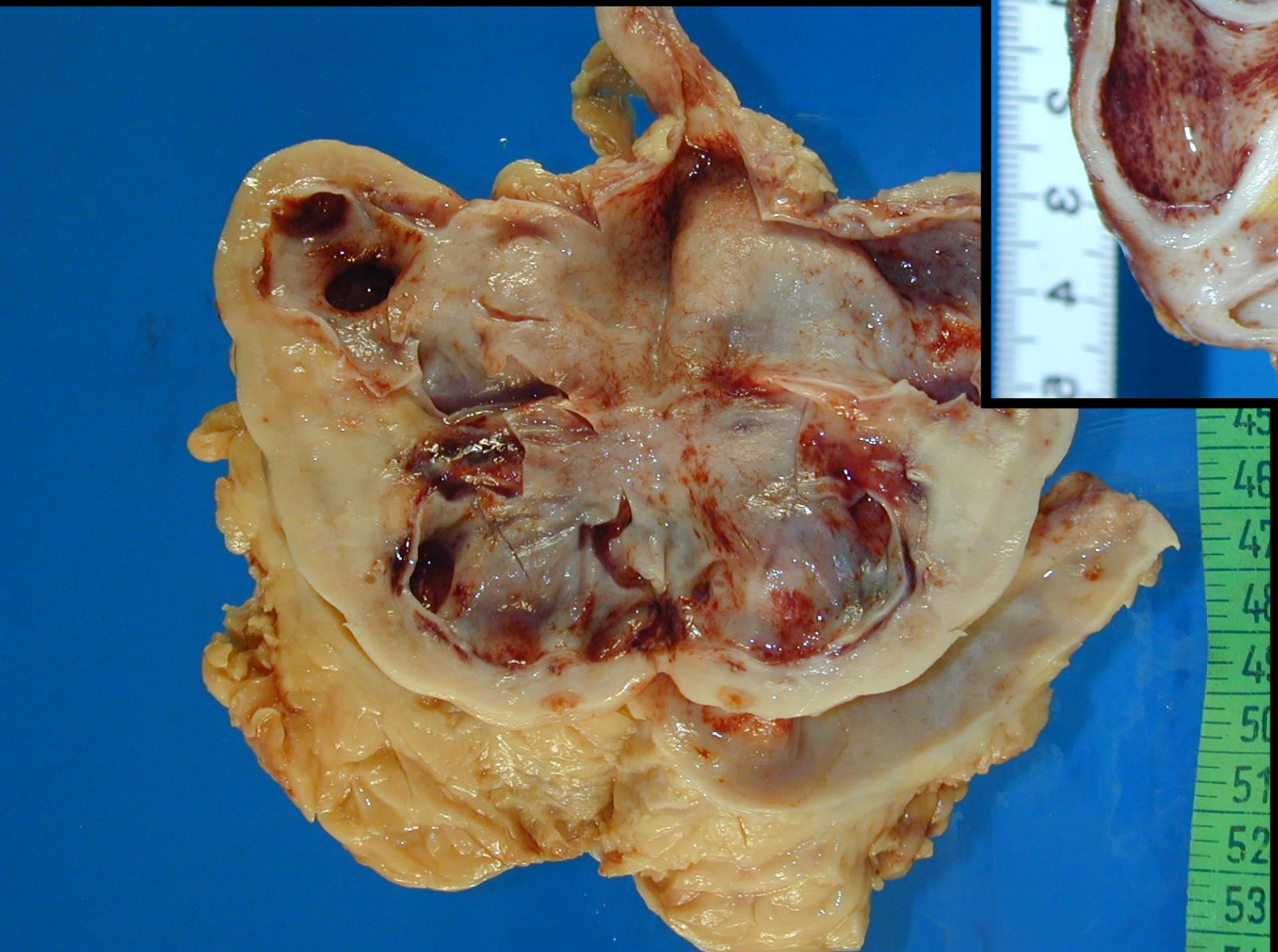


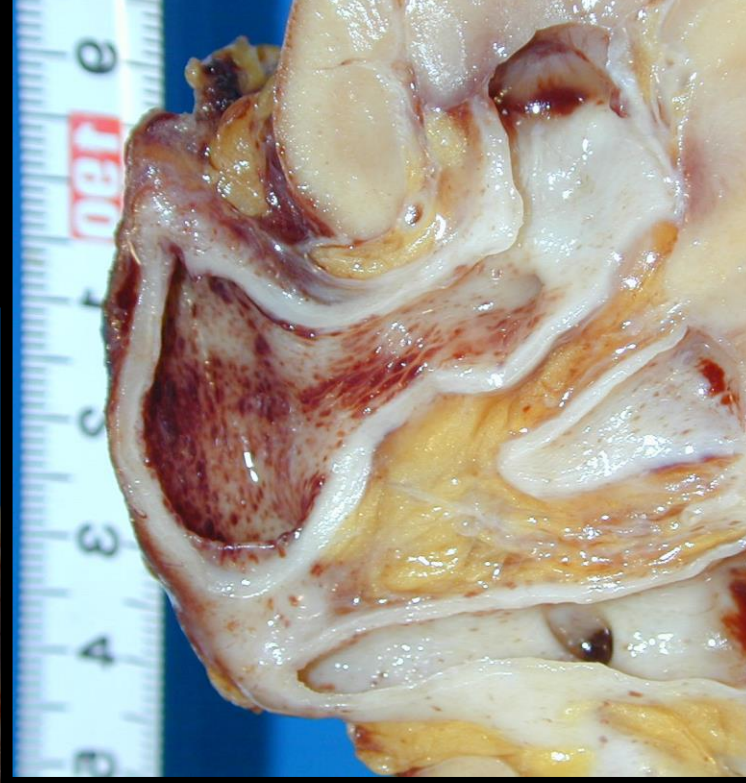


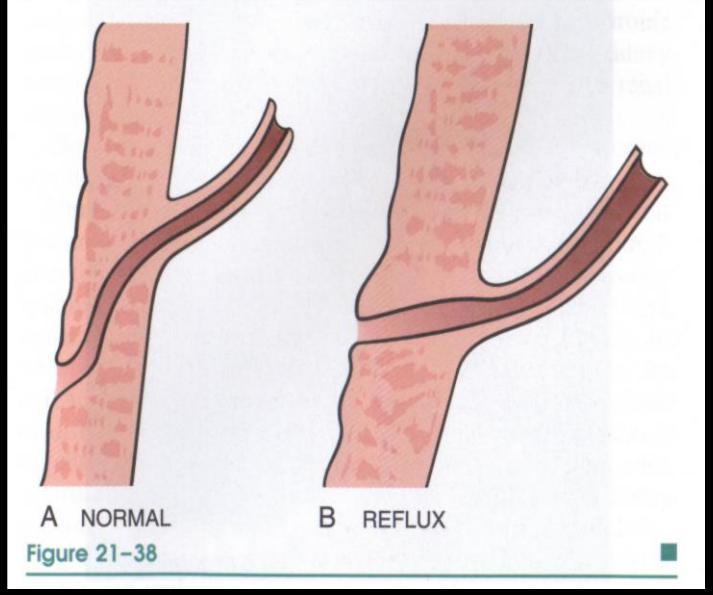

Thyroidisation

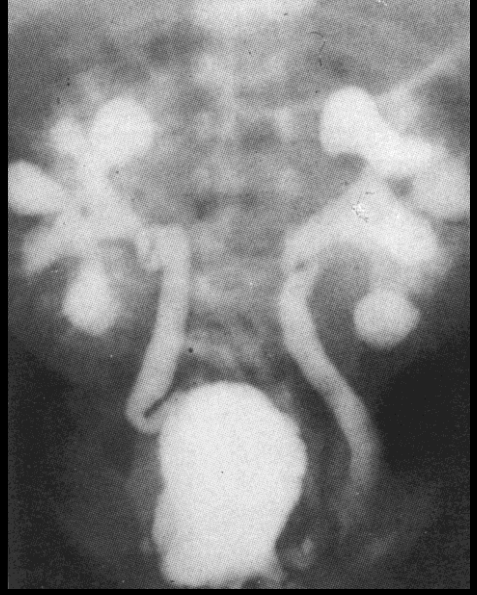
Pyelitis, pyelectasia

+ pressurenatrophy

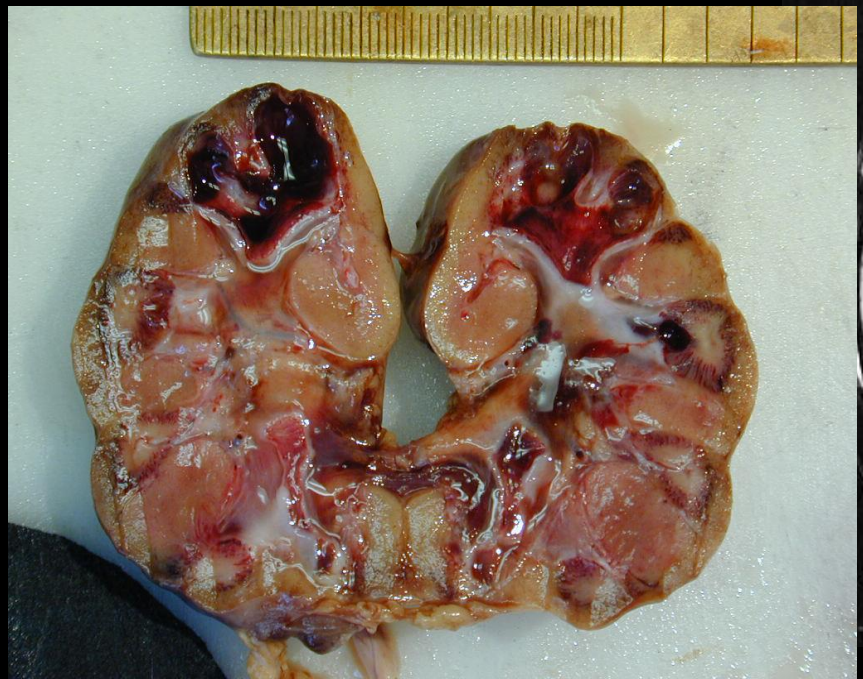


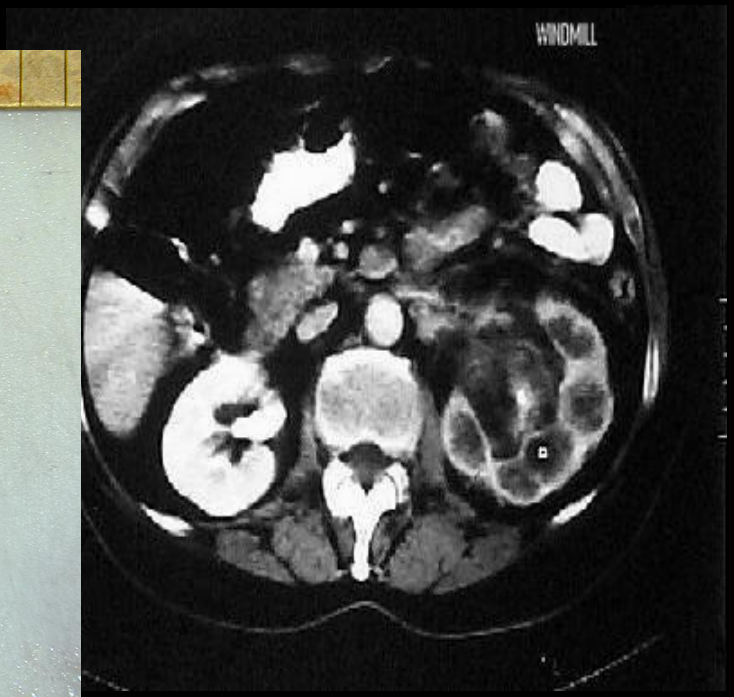






Vesicoureteral reflux (VUR)

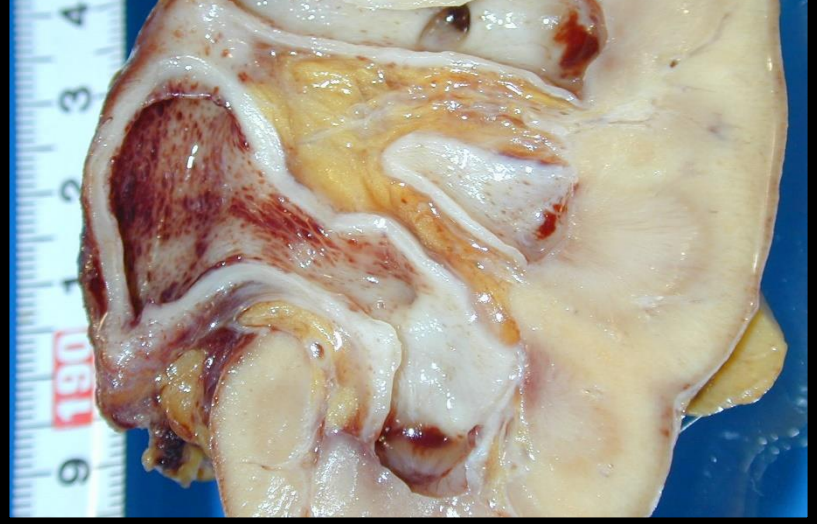




Chronic pyelonephritis

Clinical features:

Uni- or bilateral



Episodes of acute pyelonephritis, or
silent clinical course leading to destruction

Bilateral chronic pyelonephritis can result in hypertension and renal insufficiency

10% of patients on dialysis therapy have chronic pyelonephritis

Xanthogranulomatous pyelonephritis

Middle-aged women with diabetes

Usually unilateral

Proteus mirabilis infection

Tumor-like lesion

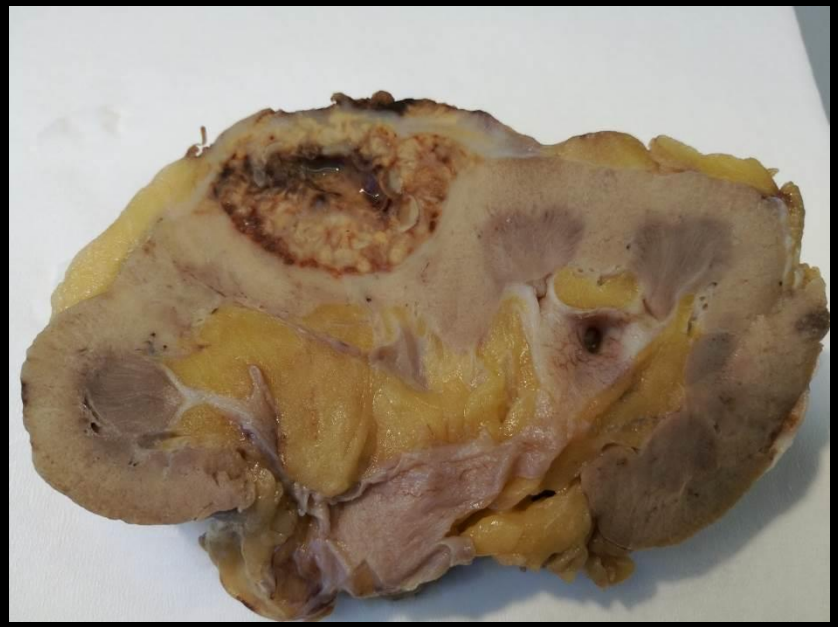
Yellowish areas, extracapsular spread,

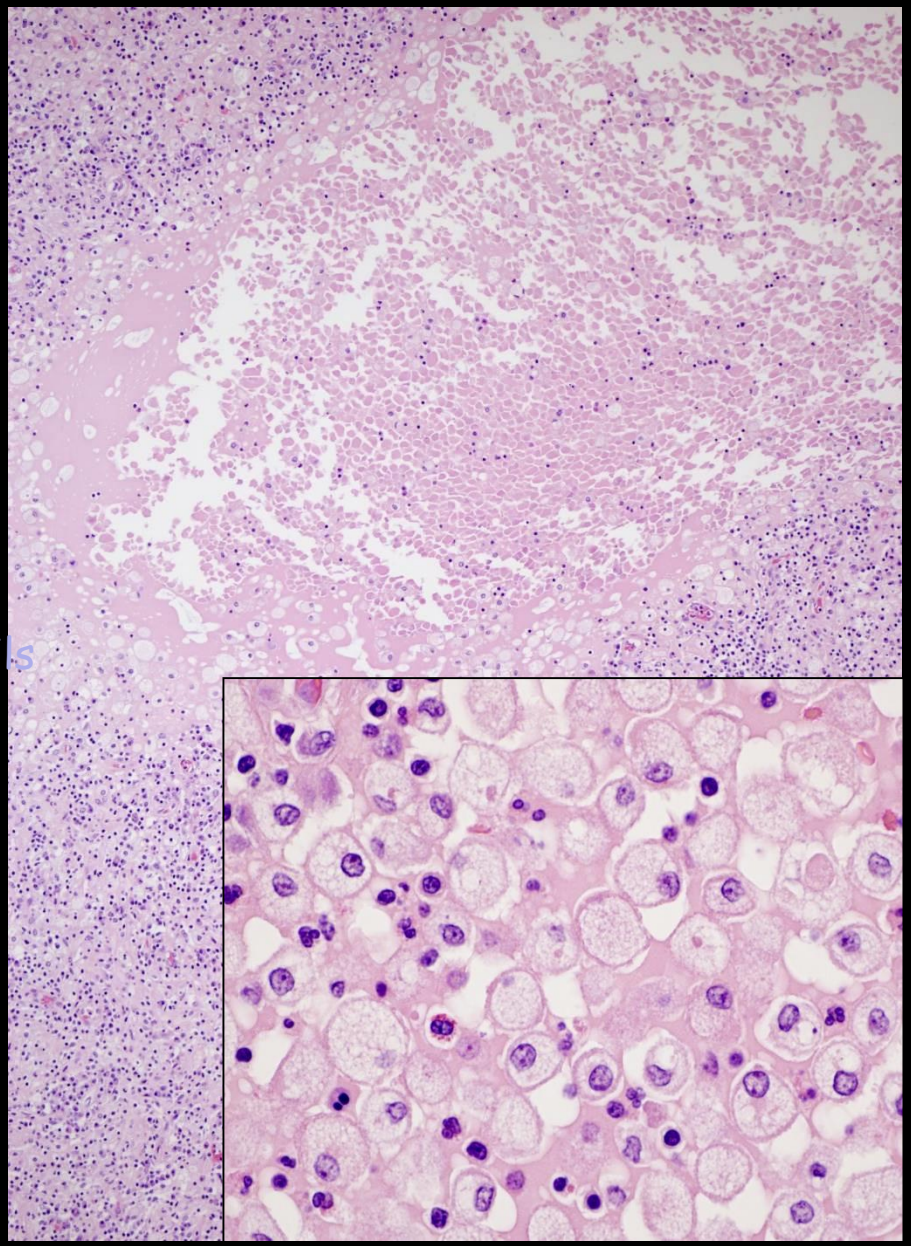
infiltrative pattern

Foamy histiocytes,

giant cells, lymphocytes, plasma cells, neutrophils

Indicates nephrectomy





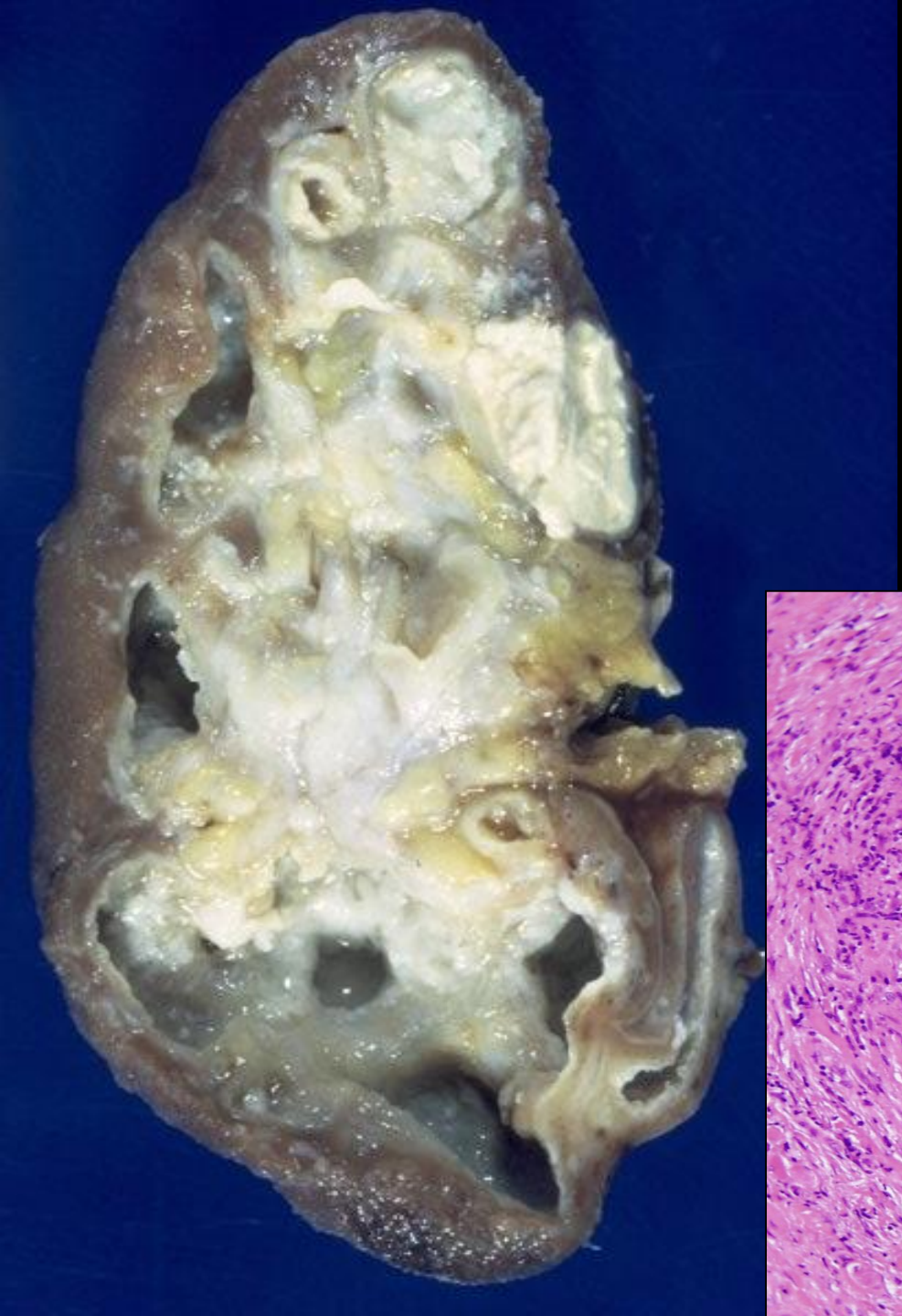
Female 73

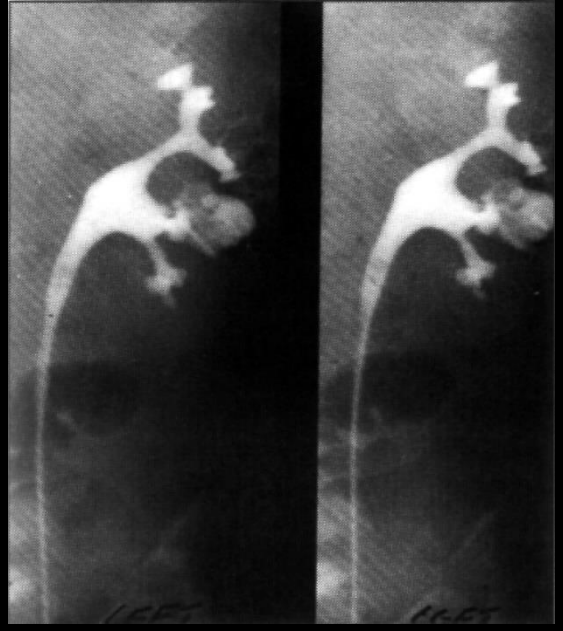
Hypertension

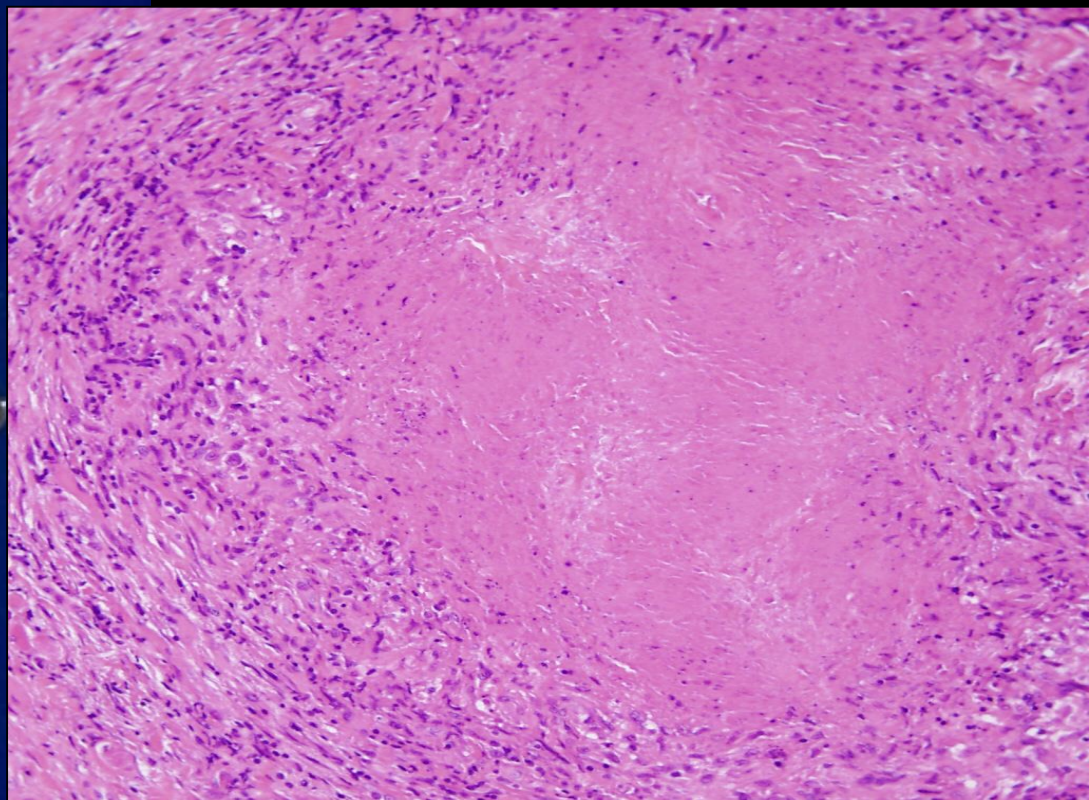
Diabetes

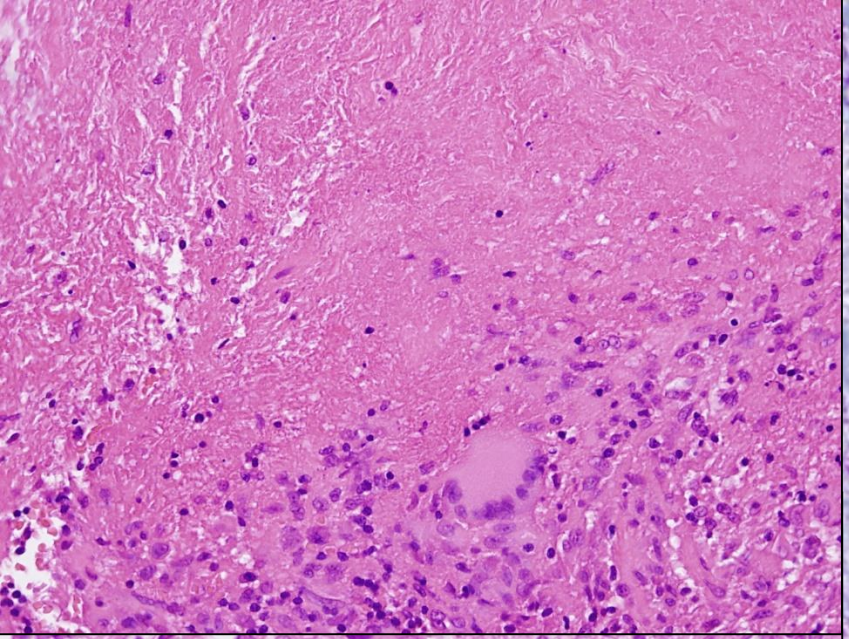
Decreased renal reserve

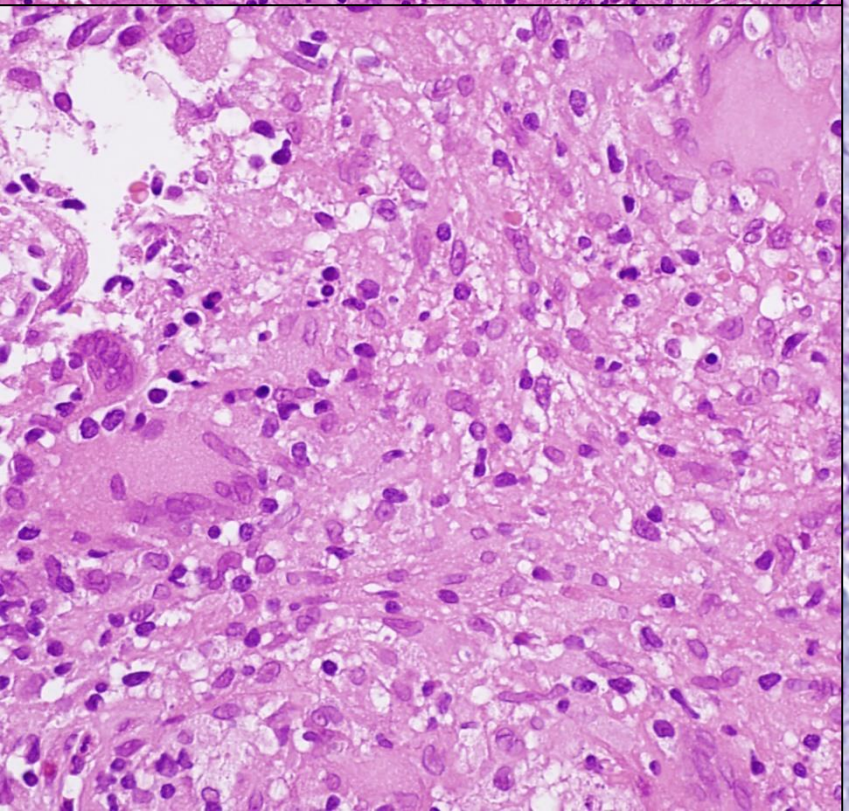
Process destroying the renal calyces

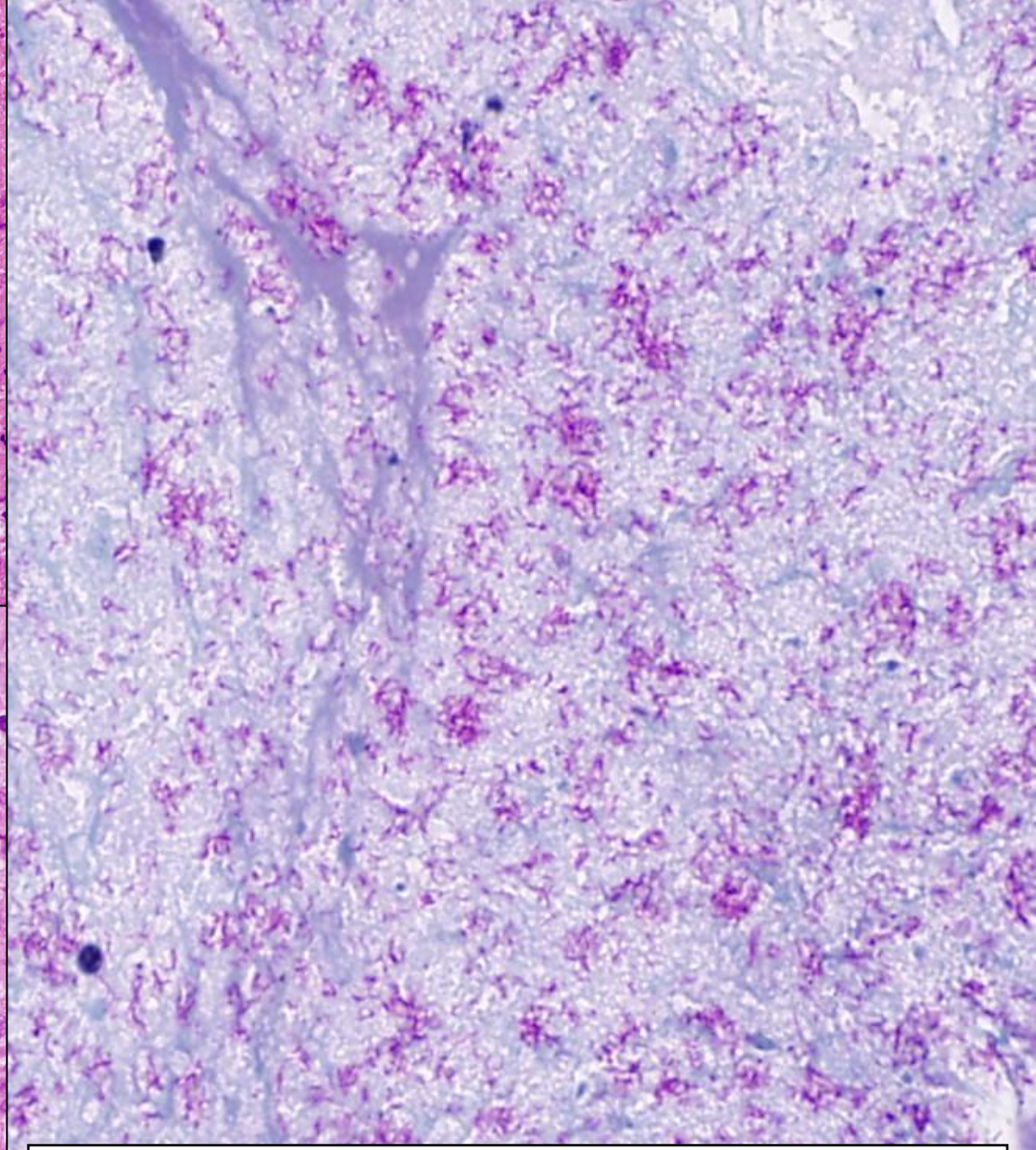












ZN stainig:

acid fast bacilli

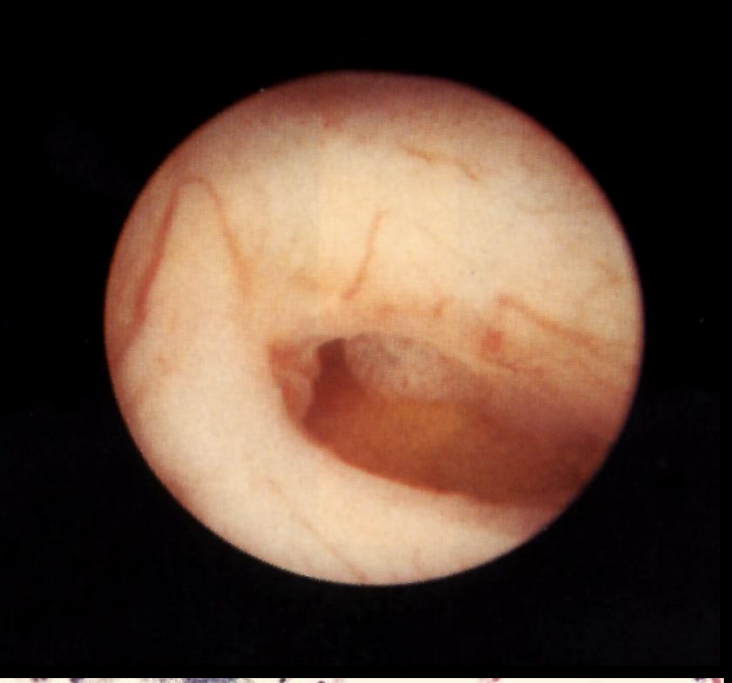
DG.: TBC

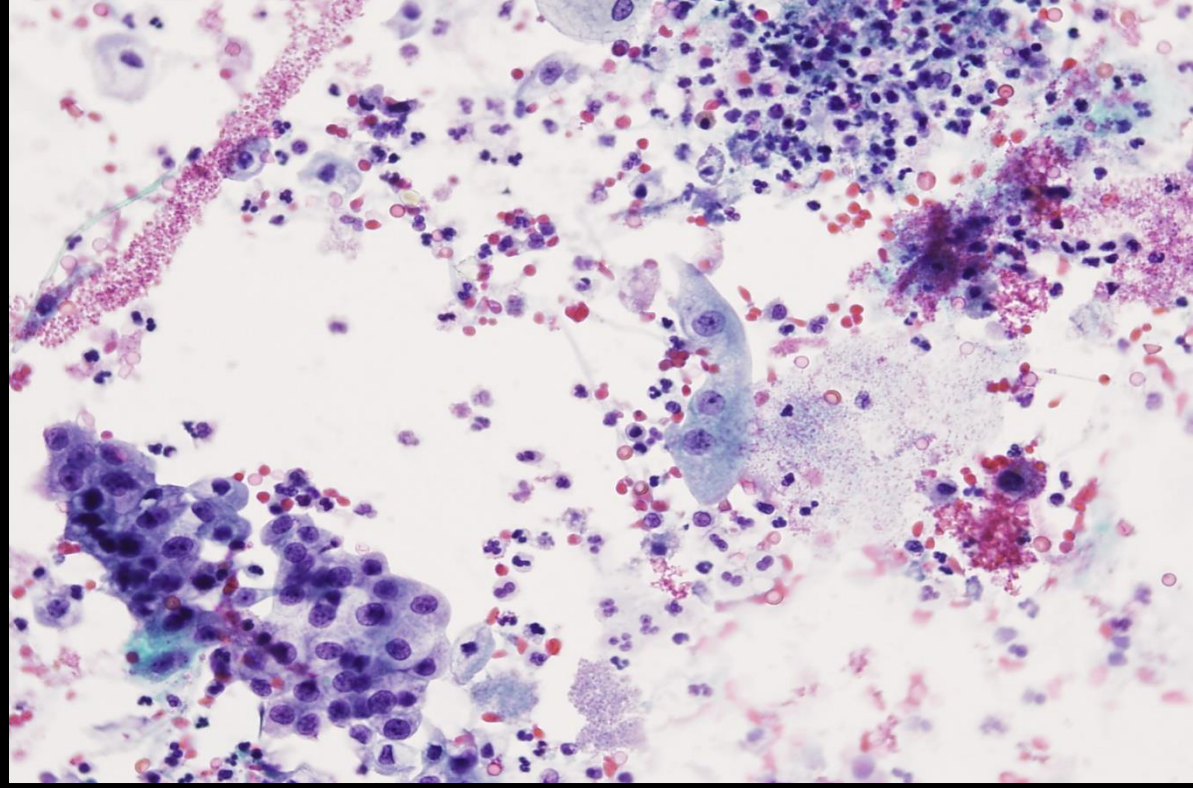
Female, 45

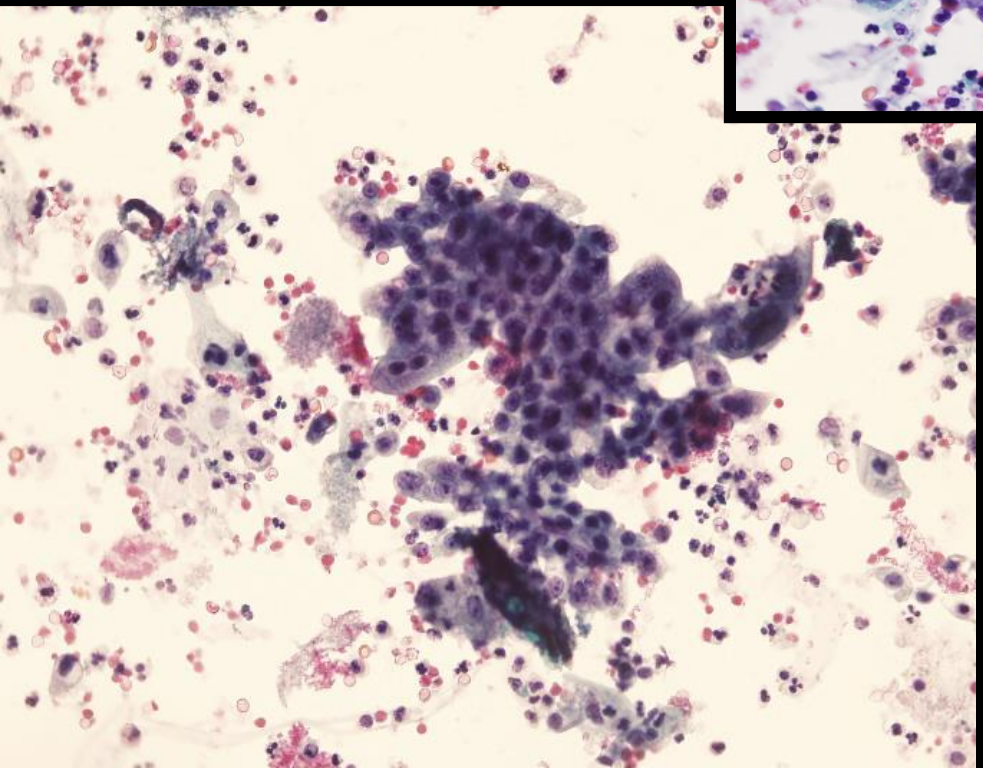
Hematuria, costovertebral pain

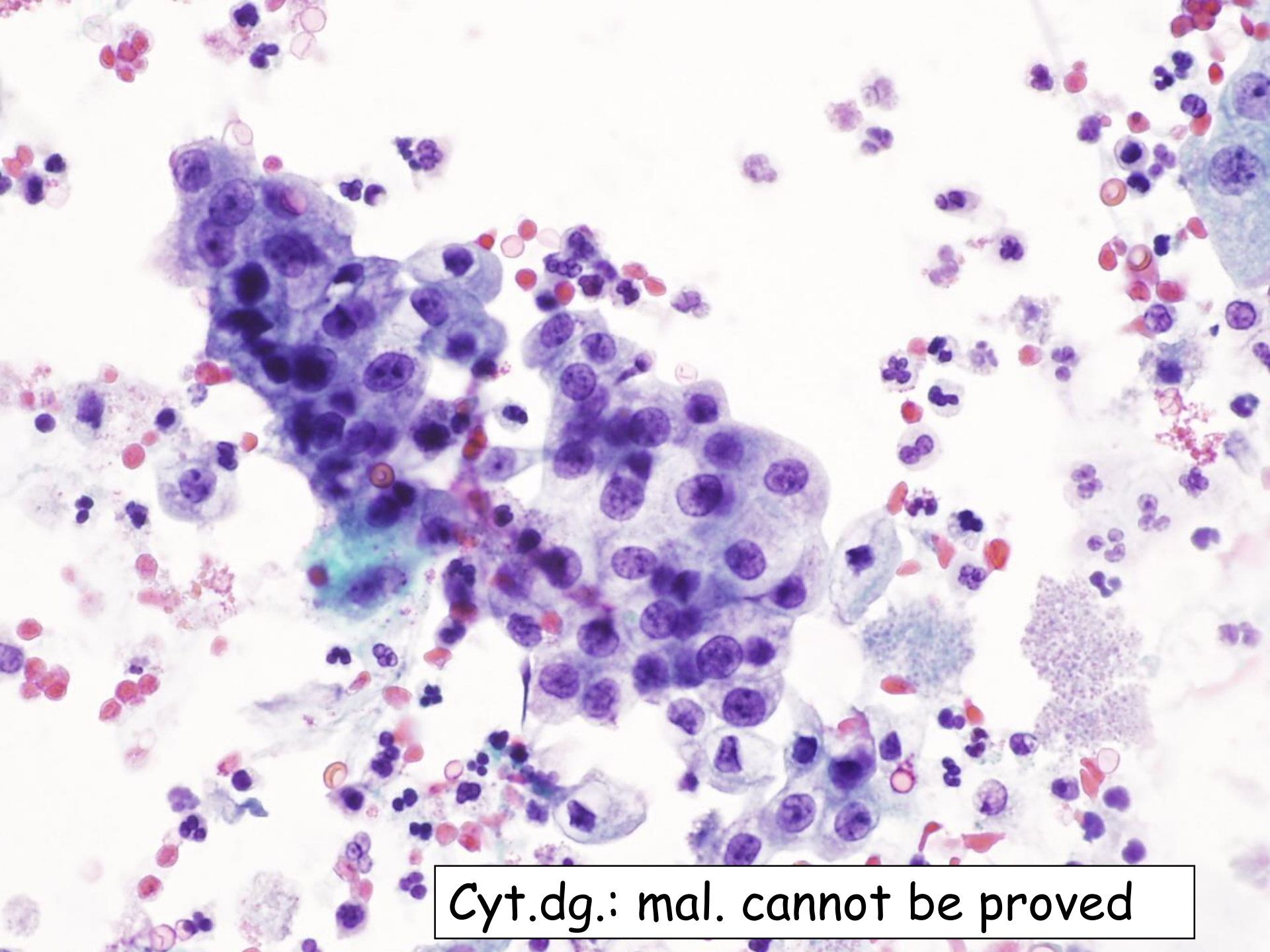
Vascularised lesion protruding into the pyelon by ureteroscope

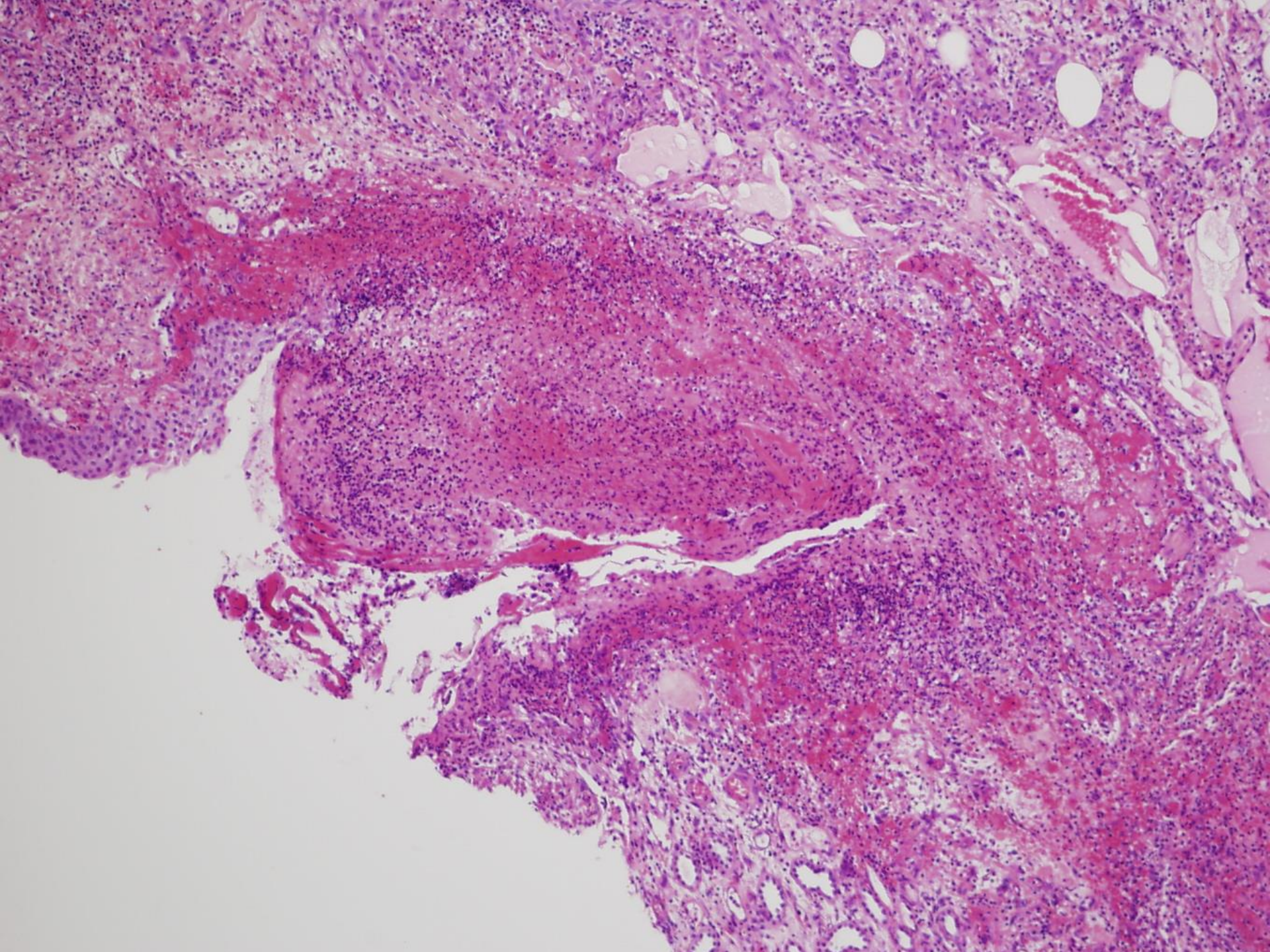
Malignant?

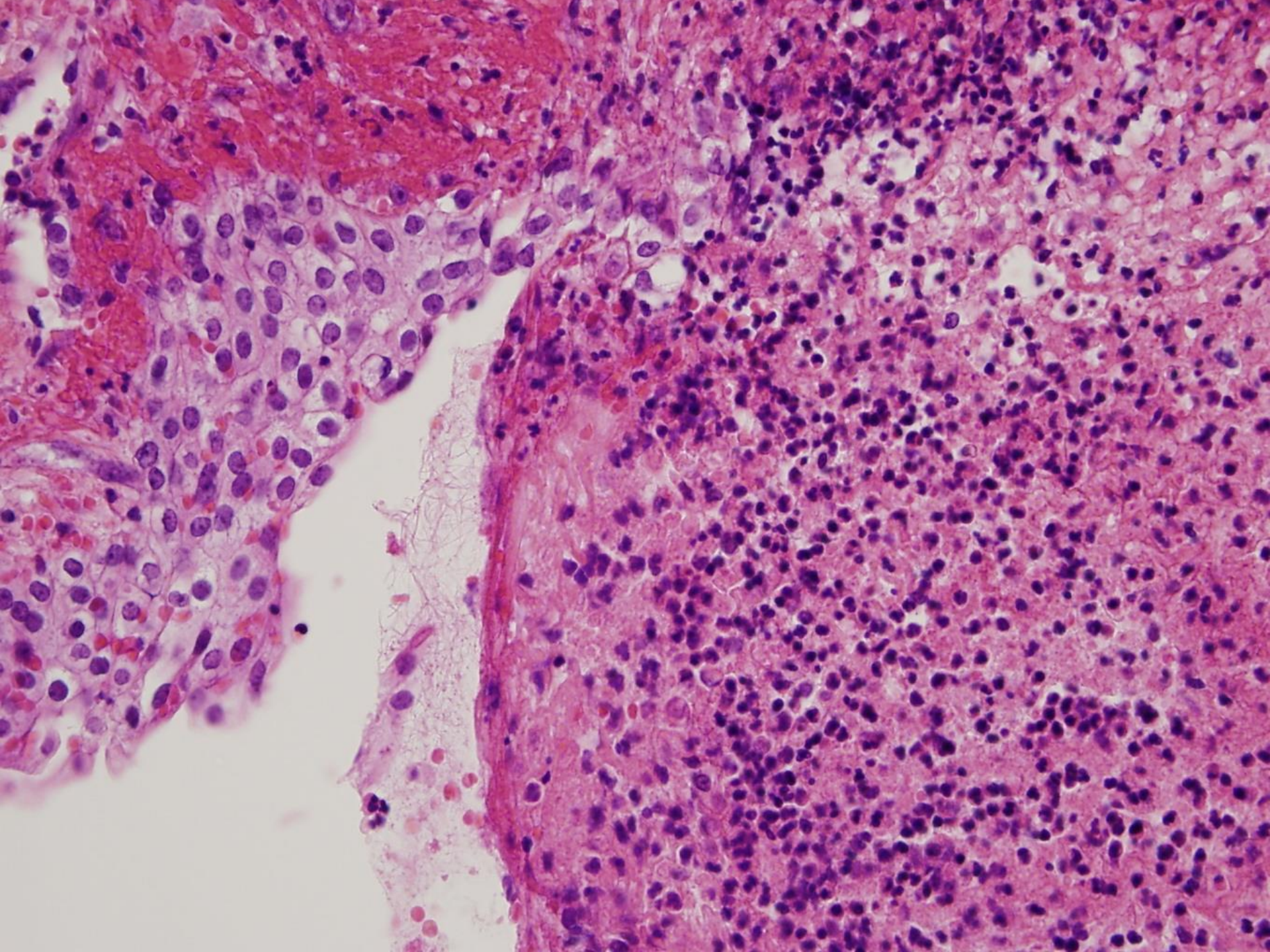


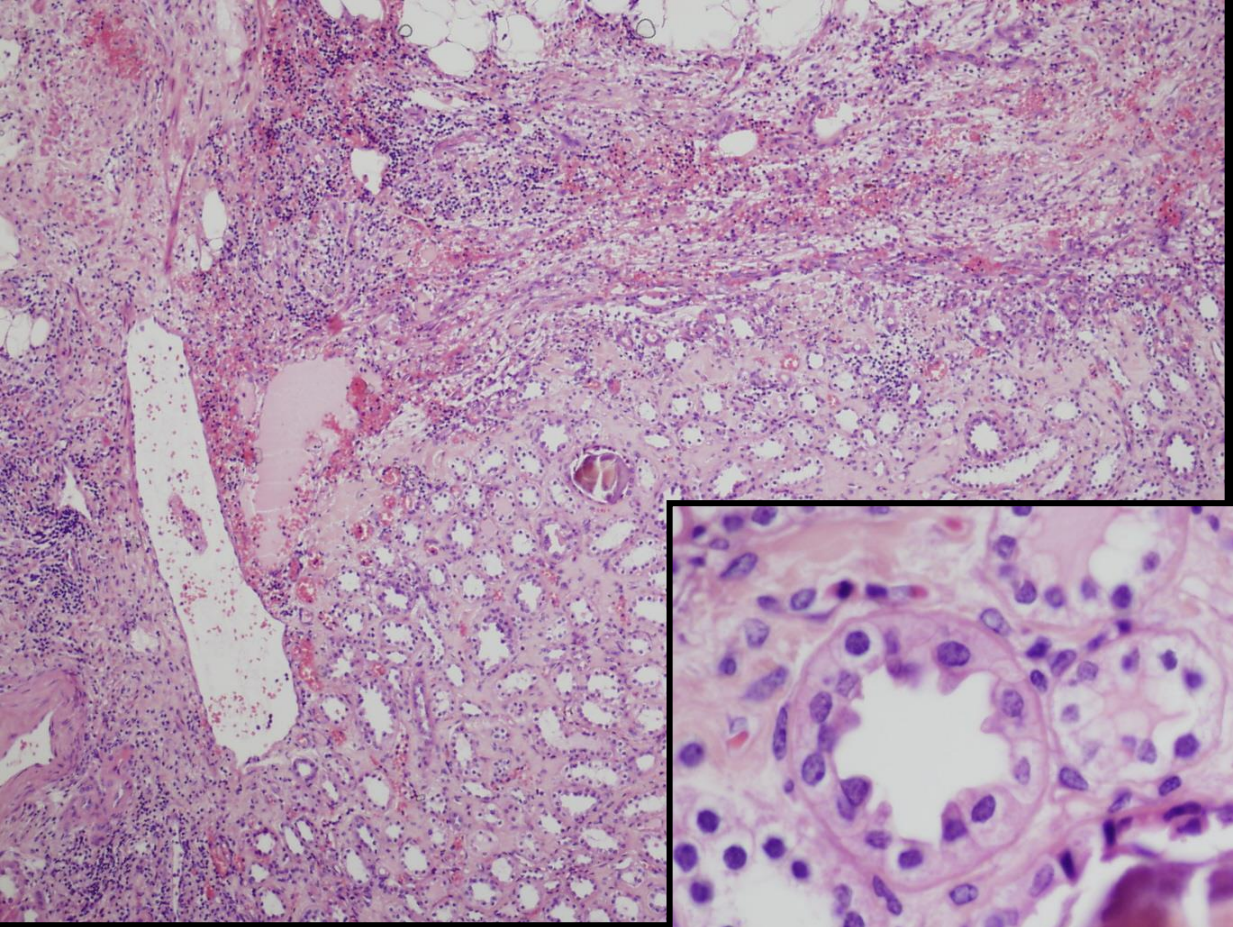





Cyt.dg.: mal. cannot be proved

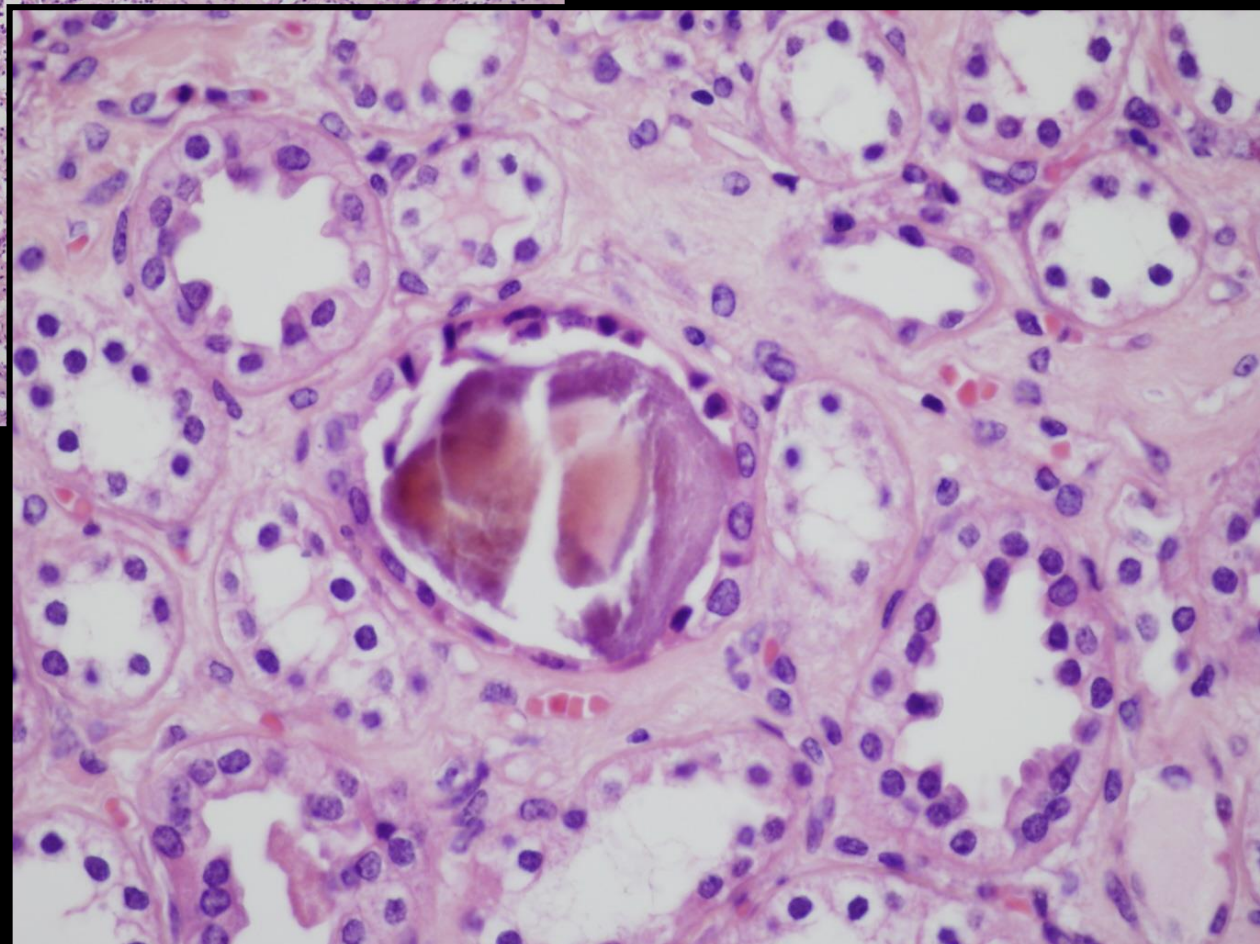






Tu. neg.

VS. nephrocalcinosis



Pyelitis