

KIDNEY (TUBULES, INTERSTITIUM & BLOOD VESSELS)

TUBULAR DISEASES

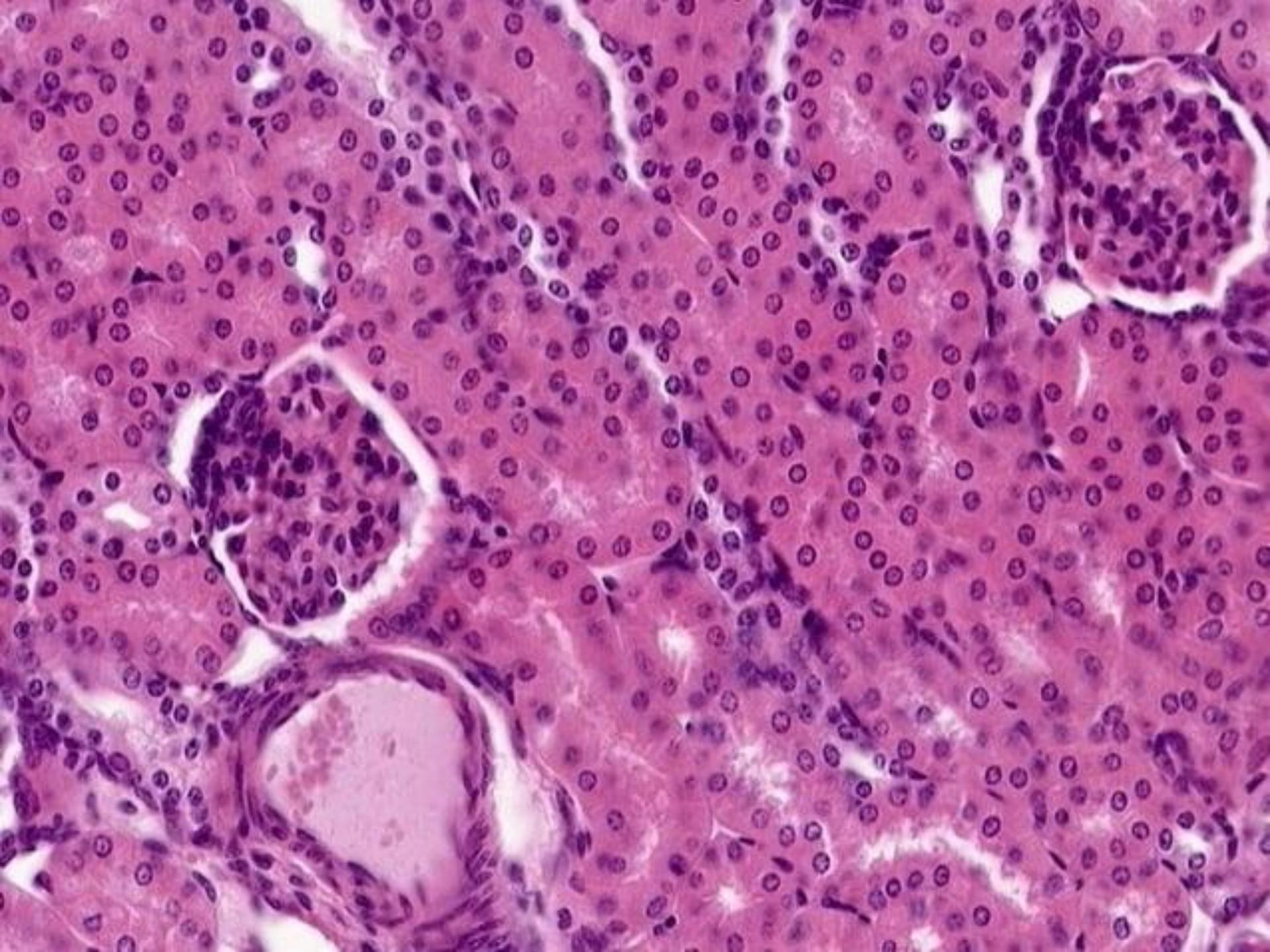
- ACUTE TUBULAR NECROSIS
- TUBULOINTERSTITIAL NEPHRITIS
 - PYELONEPHRITIS
 - ACUTE
 - CHRONIC
 - DRUGS
 - TOXINS
- URATE NEPHROPATHY
- HYPERCALCEMIA/NEPHROCALCINOSIS
- MULTIPLE MYELOMA

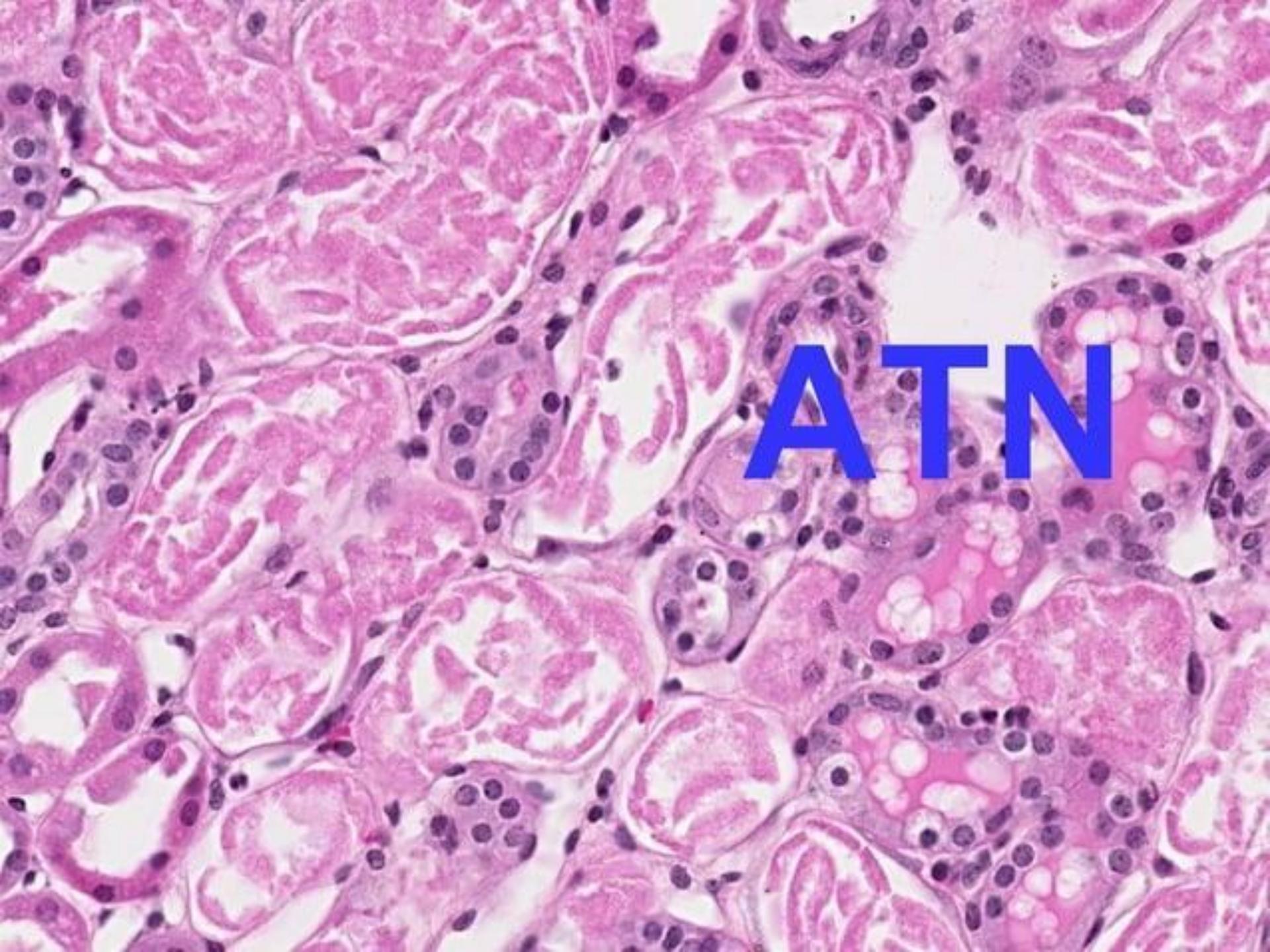
ACUTE TUBULAR NECROSIS

- Destruction of renal **TUBULAR** epithelium
- Loss of renal function
- 50% of **ACUTE** renal failure
- Two types:

ISCHEMIC NEPHROTOXIC

- AMINOGLYCOSIDES**
- AMPHOTERICIN B**
- CONTRAST AGENTS**

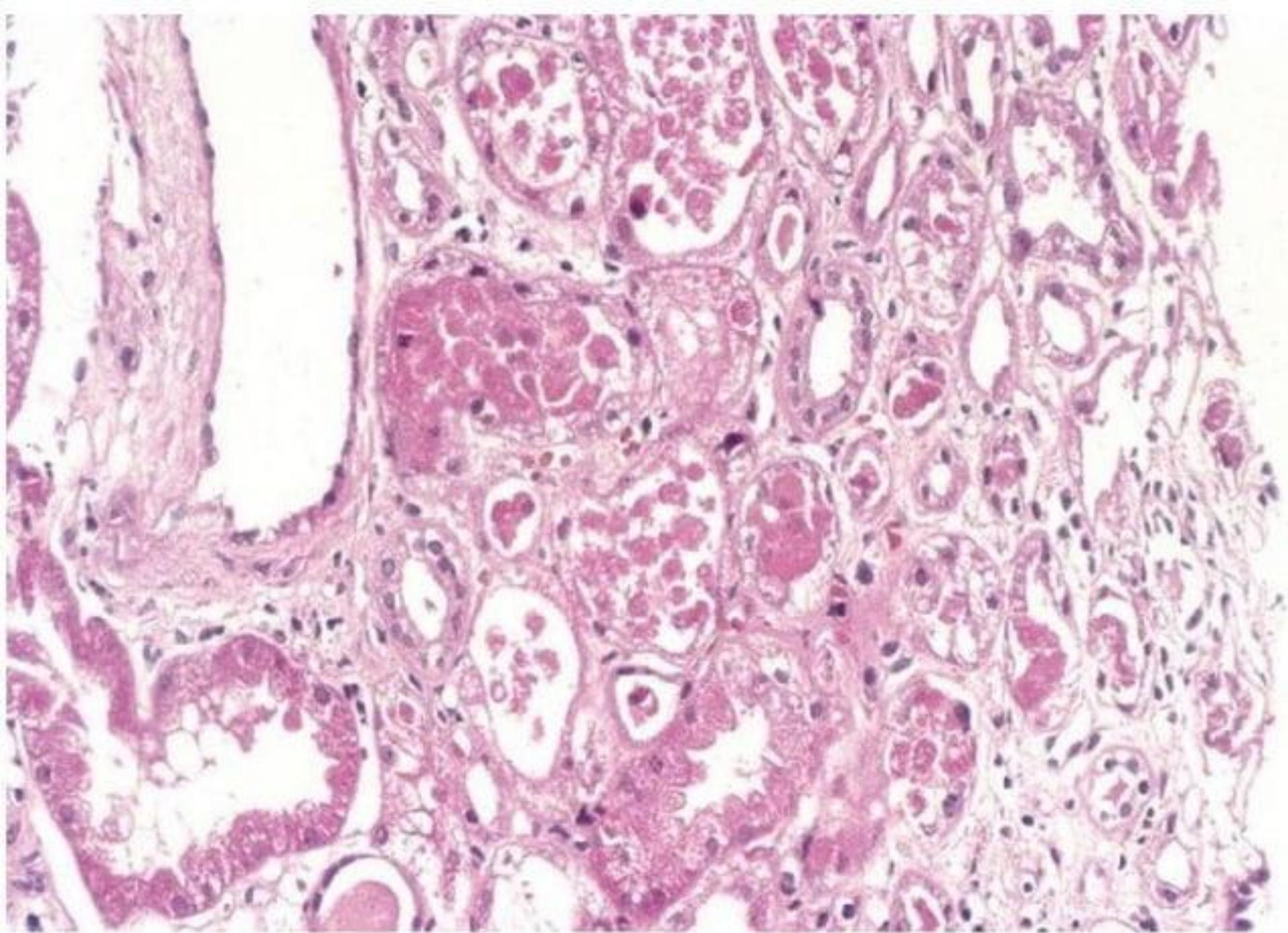


A high-magnification light micrograph of kidney tissue, likely a renal cortex. The image shows various tubular structures and cellular components. Superimposed on the right side of the image is the text "ATN" in a large, bold, blue sans-serif font.

ATN

ATN PATHOGENESIS

- BLOOD FLOW DISTURBANCES (ISCHEMIC)
- TUBULAR INJURY (NEPHROTOXIC)



CLINICAL COURSE

- INITIATION (36 hours)
 - Mild OLIGURIA
 - Mild AZOTEMIA
- MAINTENANCE
 - More OLIGURIA
 - More AZOTEMIA
 - DIALYSIS NEEDED
- RECOVERY
 - HYPOKALEMIA main problem
 - BUN, CREATININE return to normal

TUBULO/INTERSTITIAL NEPHRITIS

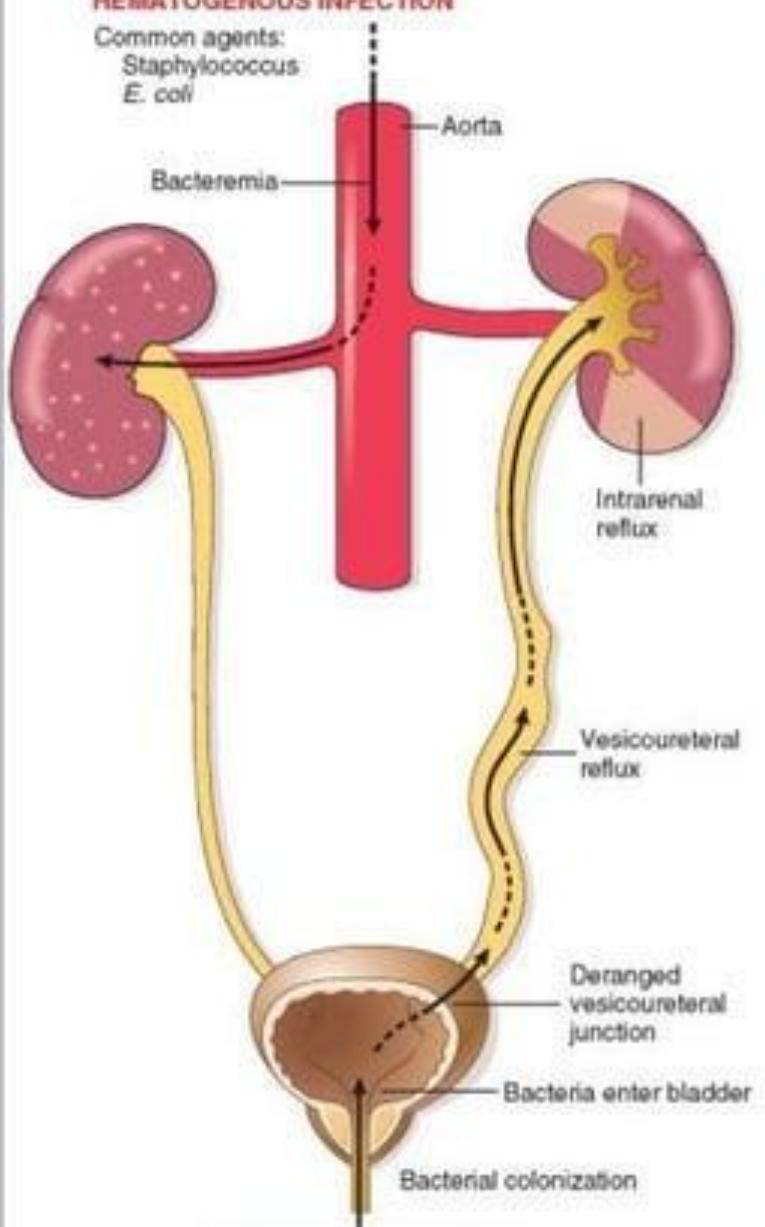
- **INFECTIONS**, i.e., pyelonephritis
- **TOXINS**, heavy metals, chemo, NSAIDS
- **METABOLIC**, urates, Ca++, Oxalates
- **PHYSICAL**, obstruction, radiation
- **IMMUNOLOGIC**, esp. transplant rejection

PYELONEPHRITIS

- GI Gram NEGATIVES: *E. COLI*, *Proteus*, *Klebsiella*, *Enterobacter*, *Strep. faecalis*, usually “NORMAL” flora
- ASCENDING, by FAR, the most common, i.e., reflux, obstruction
- HEMATOGENOUS too
- ACUTE PYELONEPHRITIS, neutrophils
- CHRONIC PYELONEPHRITIS, lymphocytes, scars

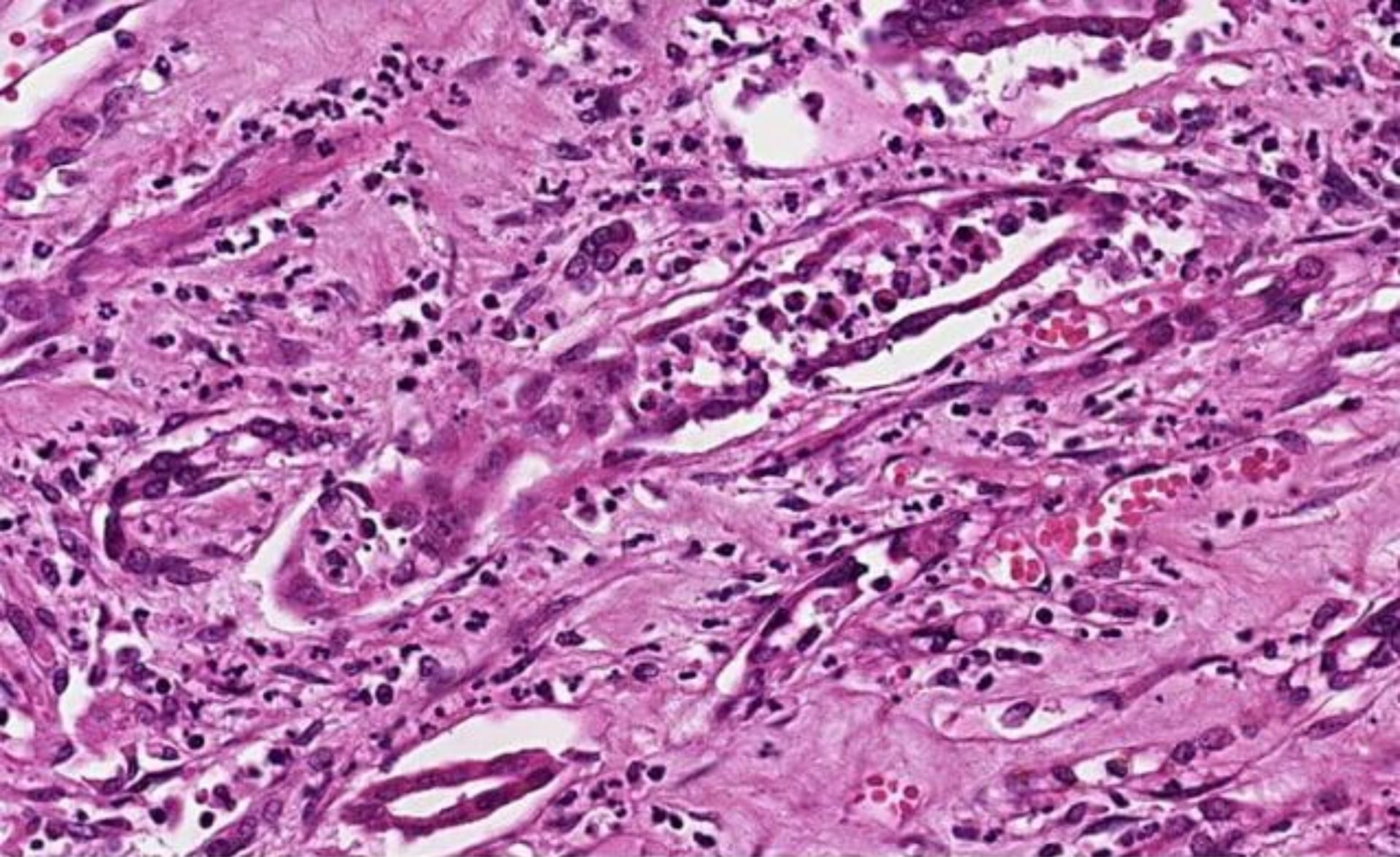
HEMATOGENOUS INFECTION

Common agents:
Staphylococcus
E. coli



ASCENDING INFECTION

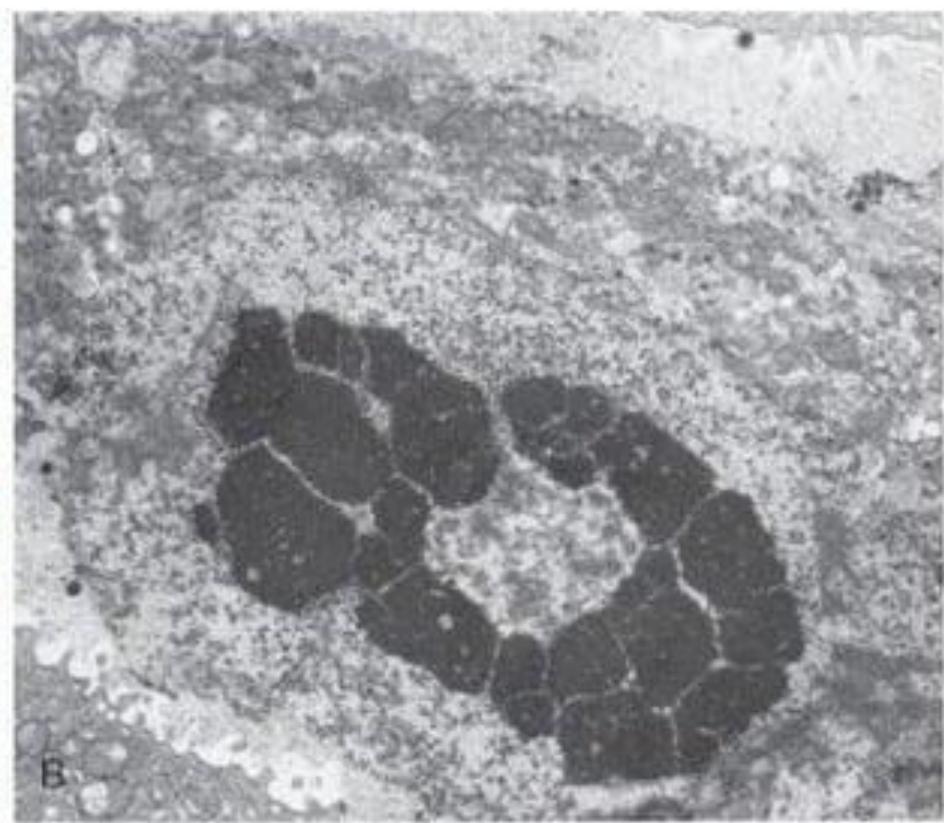
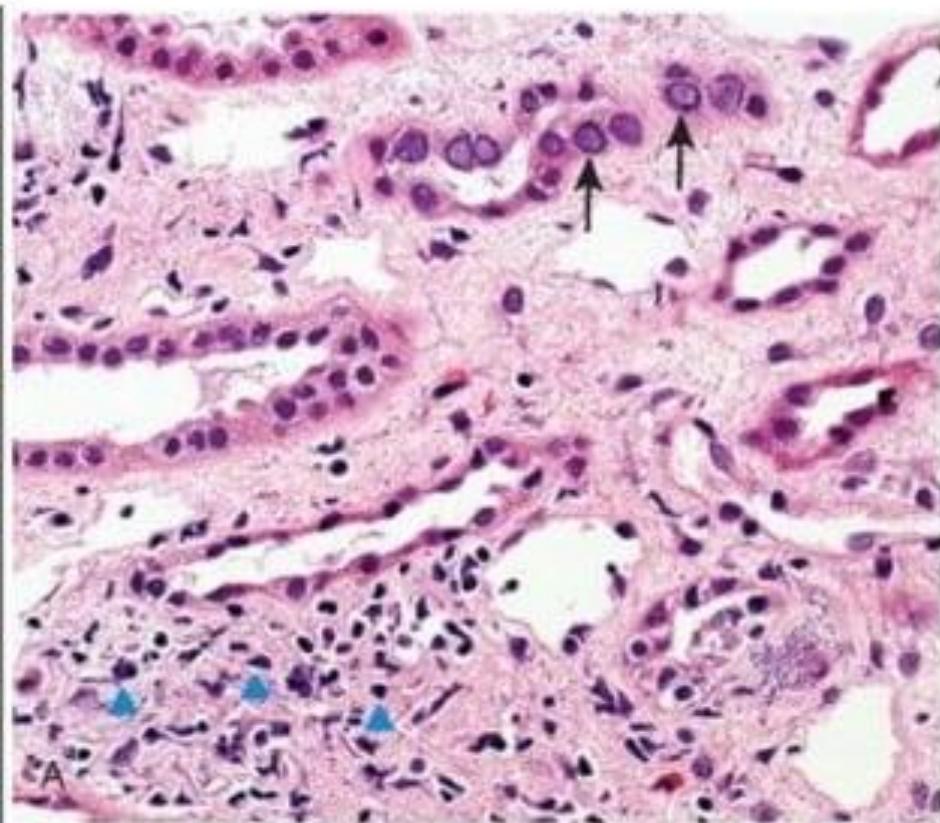
Common agents:
E. coli
Proteus
Enterobacter



ACUTE or CHRONIC PYELONEPHRITIS?

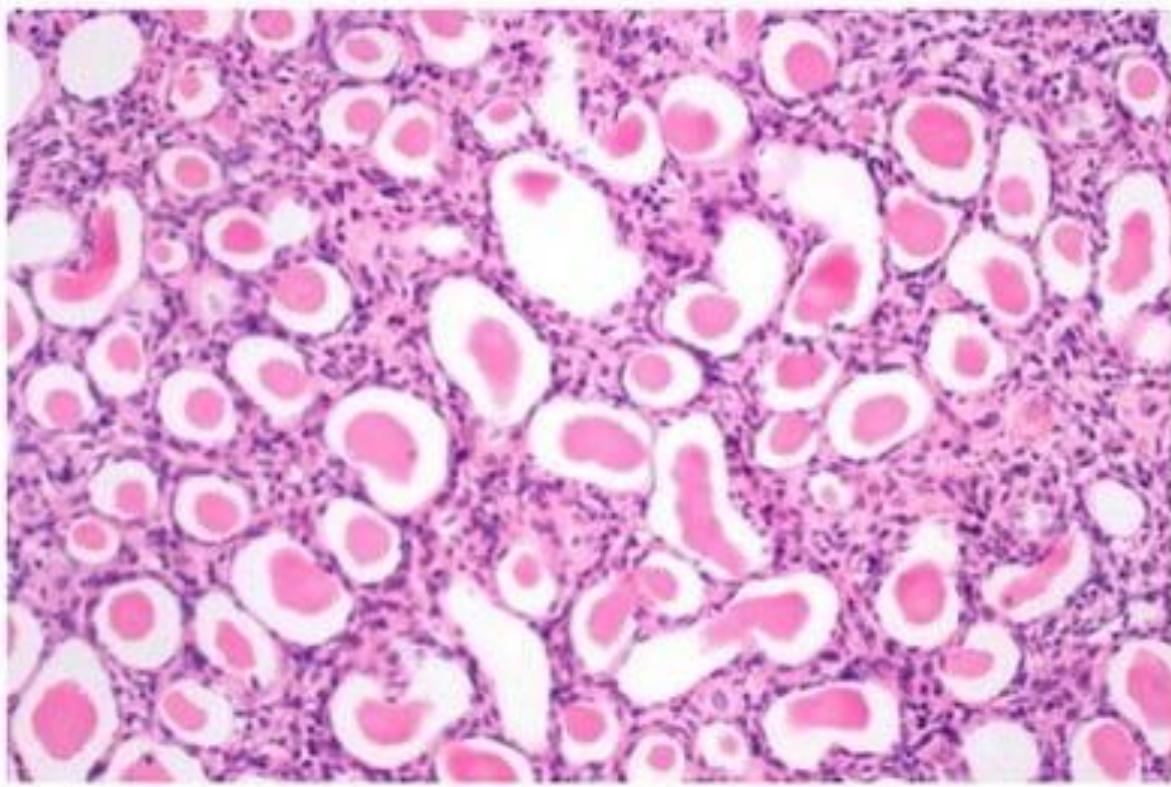
MORPHOLOGY

- **patchy interstitial suppurative inflammation, intratubular aggregates of neutrophils, and tubular necrosis.**
- Complication:
 - Papillary necrosis
 - Pyonephrosis
 - Perinephric abscess





ACUTE or CHRONIC PYELONEPHRITIS?



ACUTE or CHRONIC PYELONEPHRITIS?

FORMS OF PYELOPNEPHRITIS

- Chronic pyelonephritis can be divided into two forms:

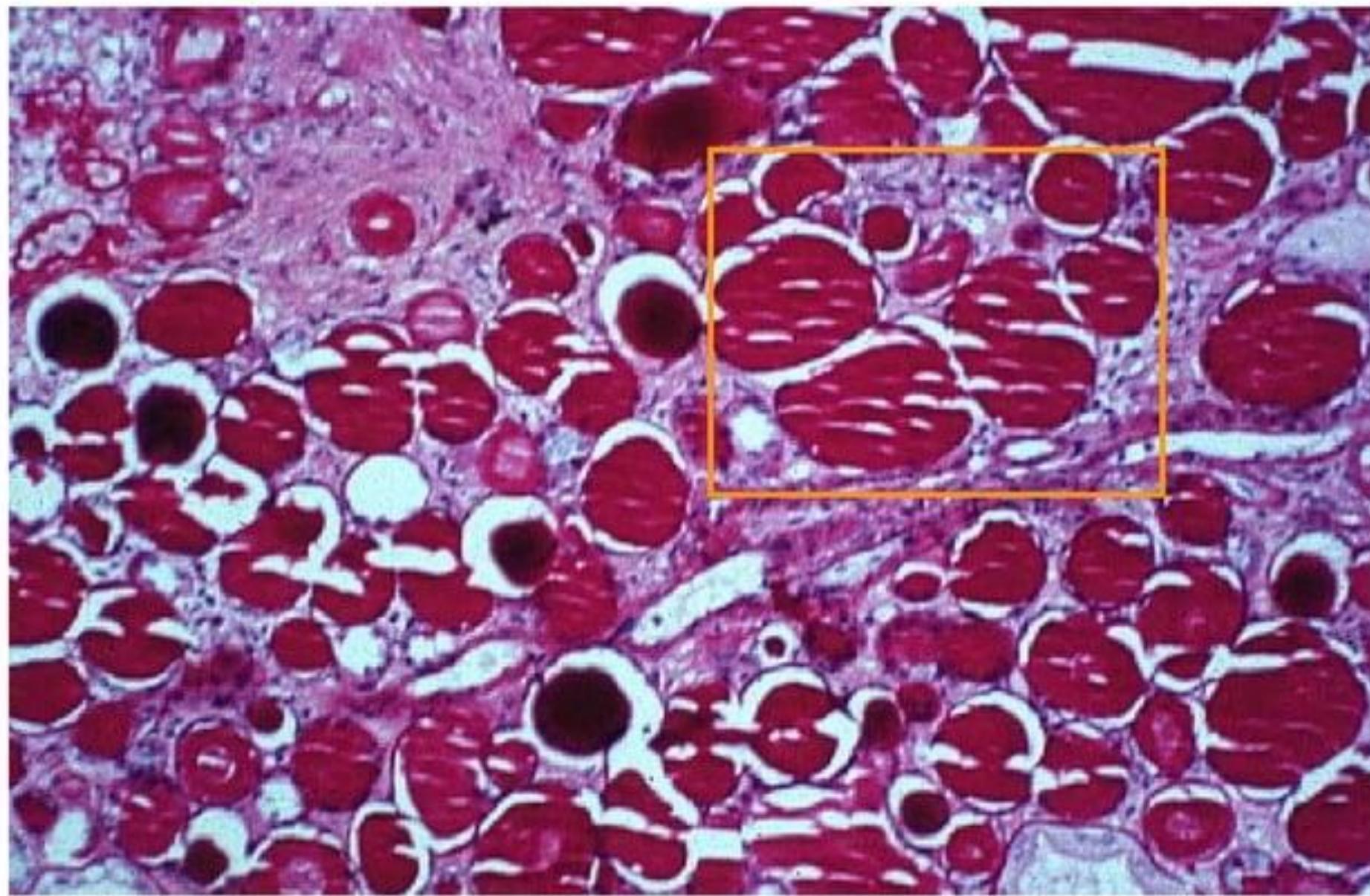
→ REFLUX NEUROPATHY

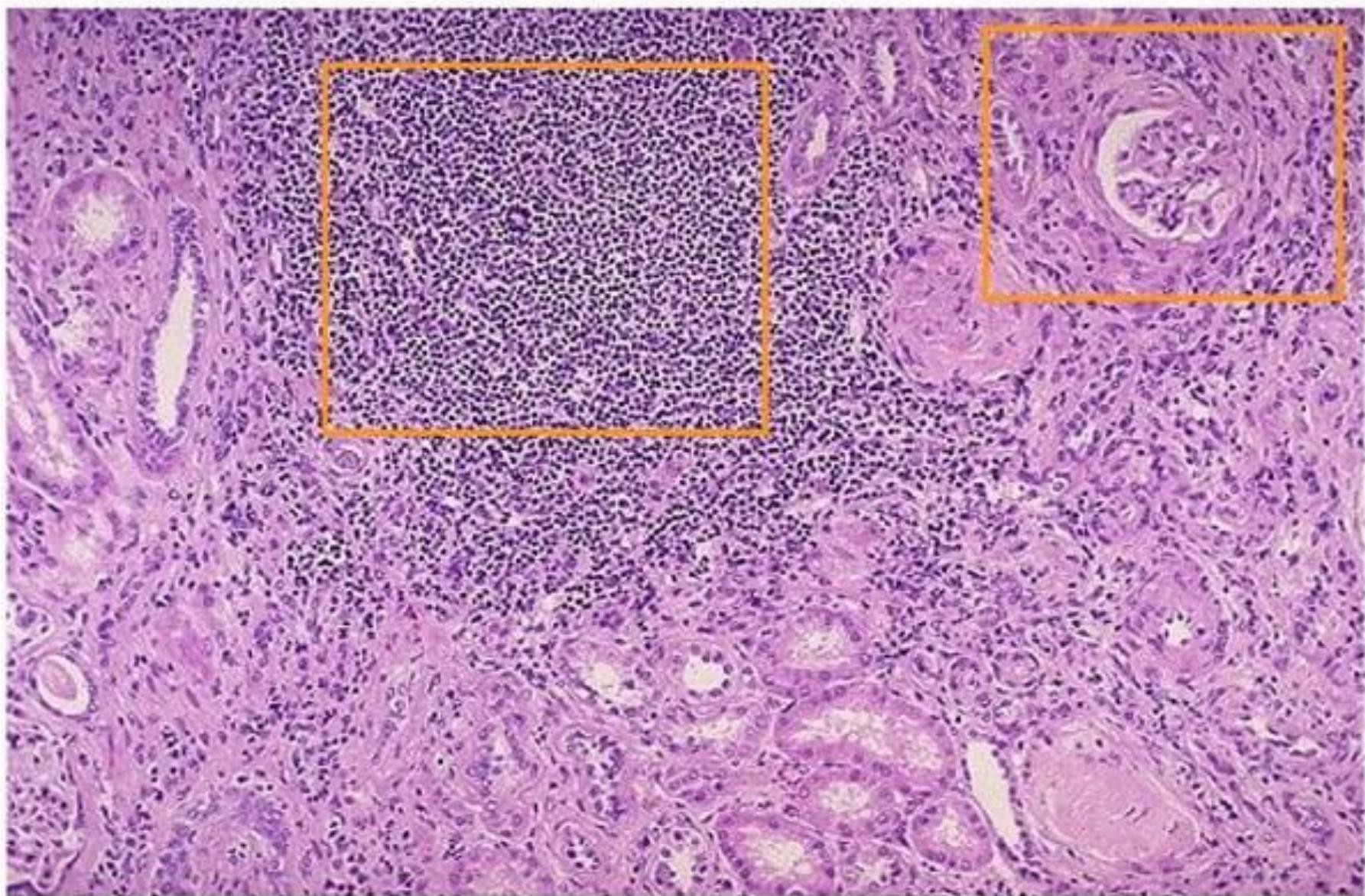
→ CHRONIC OBSTRUCTIVE PYELONEPHRITIS

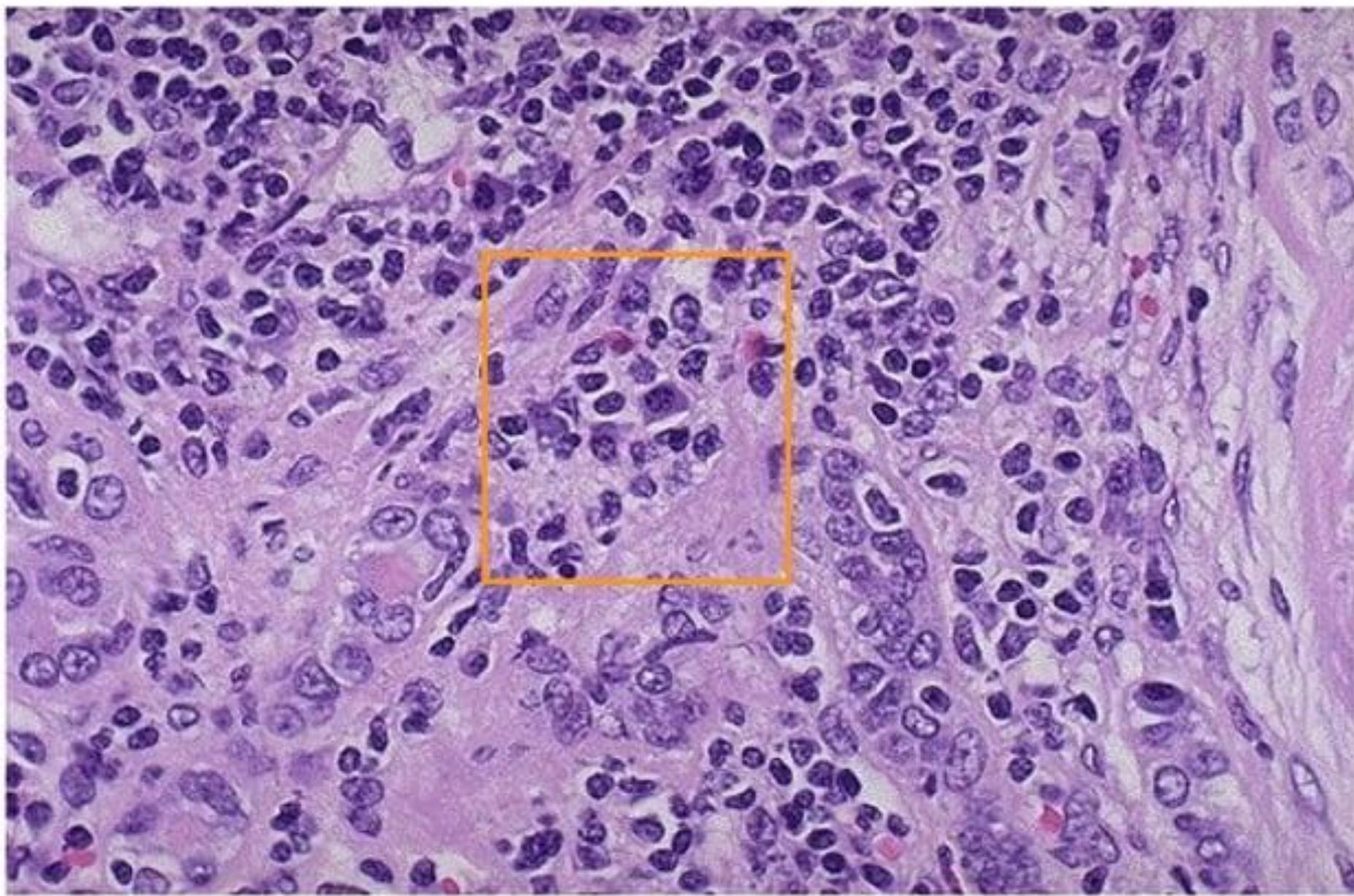
GROSS

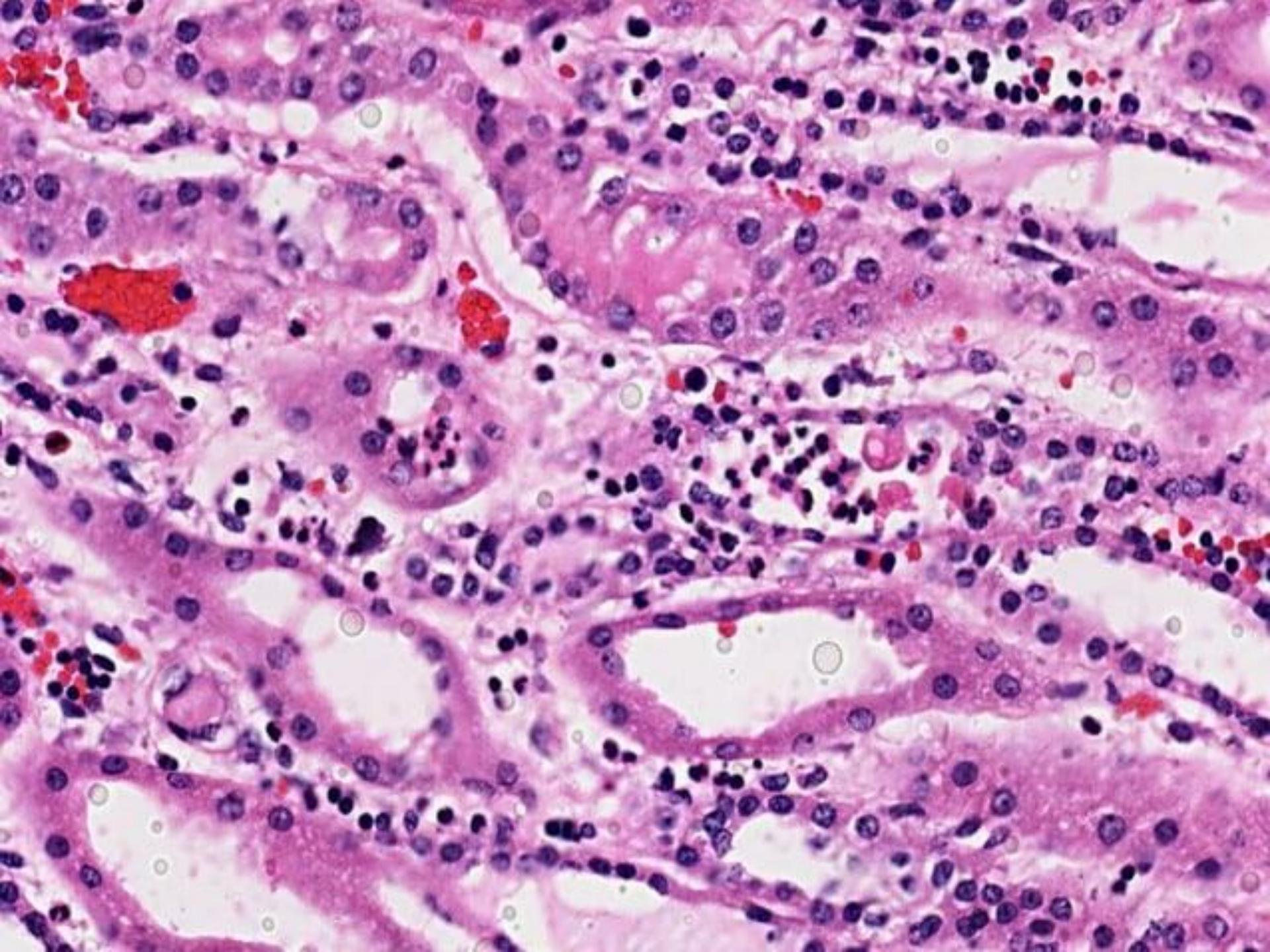
- Shrunken , scarred kidneys.
- If both kidneys are involved - involvement is asymmetrical (in contrast with chronic glomerulonephritis in which the kidneys are symmetrically involved).
- Coarse , discrete cortico - medullary scarring overlying blunted deformed calyces.

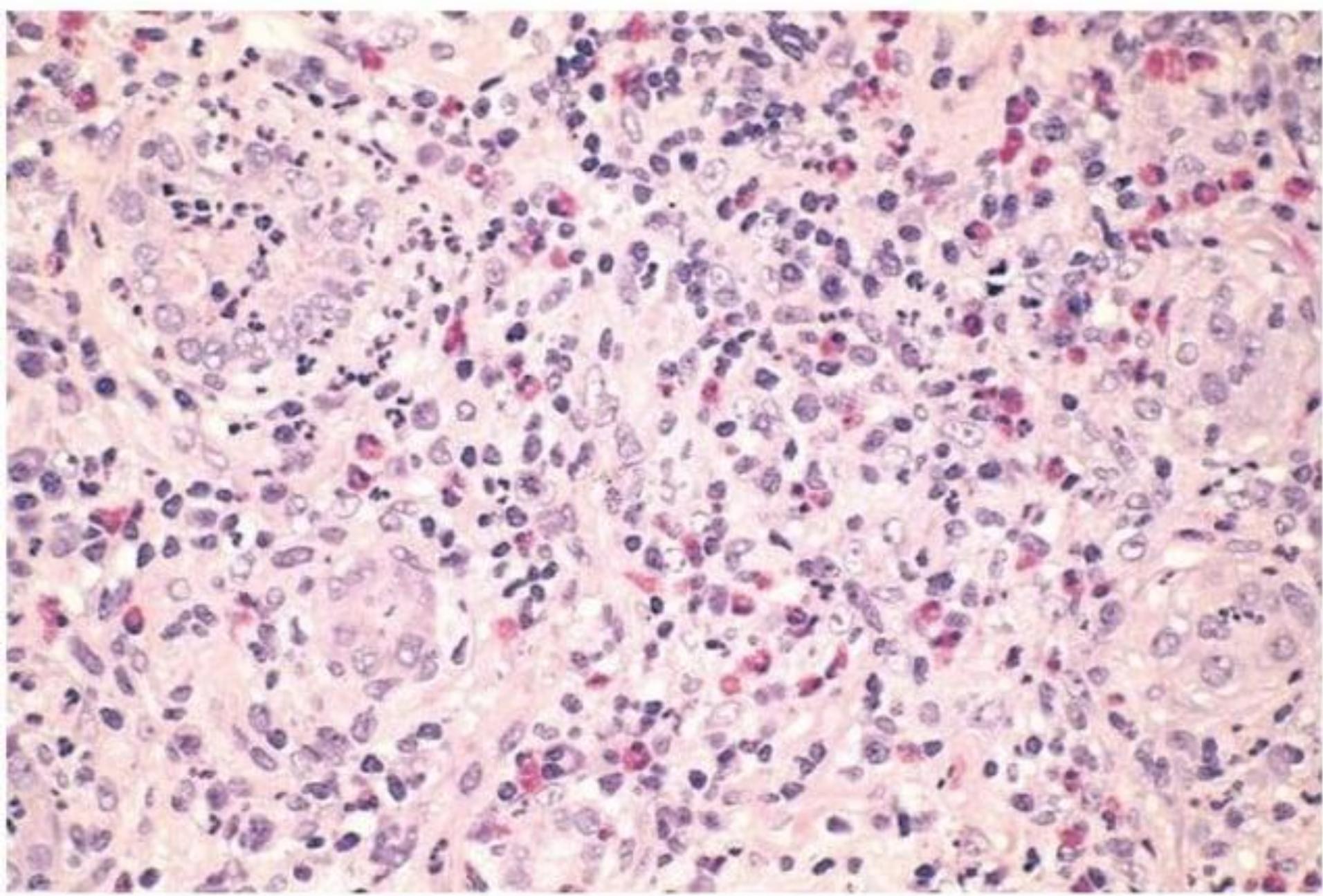












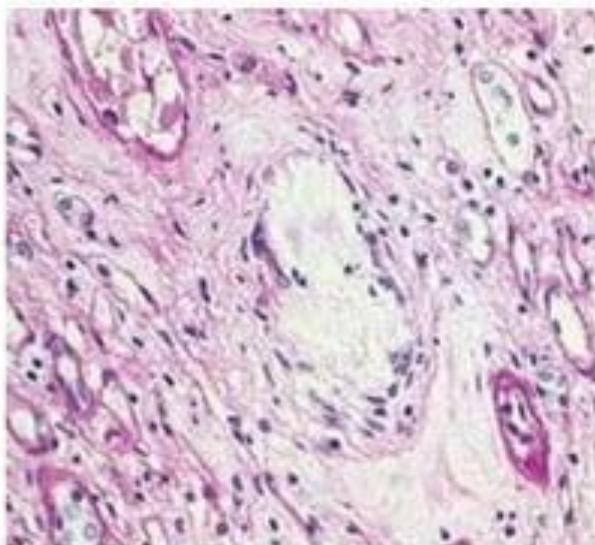
ANALGESIC NEPHROPATHY

- ASPIRIN, TYLENOL, NSAIDS
 - TUBULOINTERSTITIAL NEPHRITIS
 - PAPILLARY NECROSIS (also Dm & HbS)

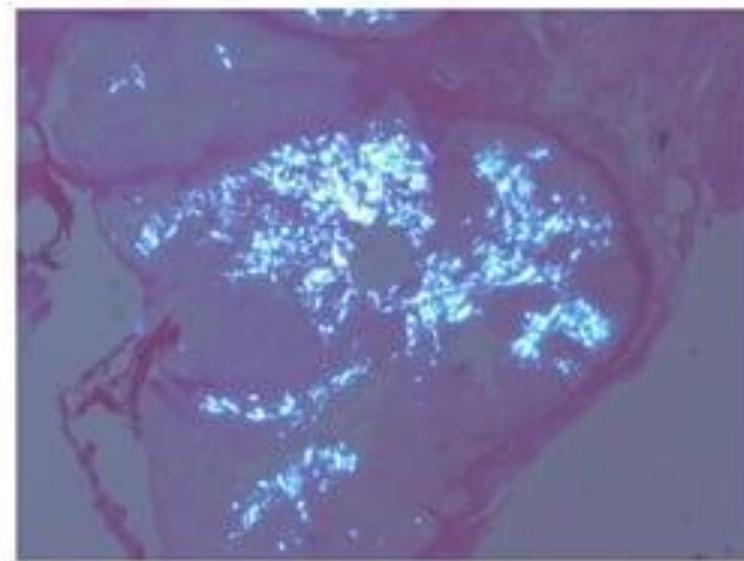


URATE NEPHROPATHY

- Precipitation of Uric Acid Crystals in the TUBULES, especially in a LOWER than usual PH situation (mini-TOPHUS)



H & E alcohol fixed



POLARIZED LIGHT MICROSCOPY

- Three forms:
 - Acute uric acid nephropathy ... chemotherapy related
 - Chronic urate nephropathy ... gouty, tophi
 - Nephrolithiasis ... stones

HYPERCALCEMIA NEPHROCALCINOSIS

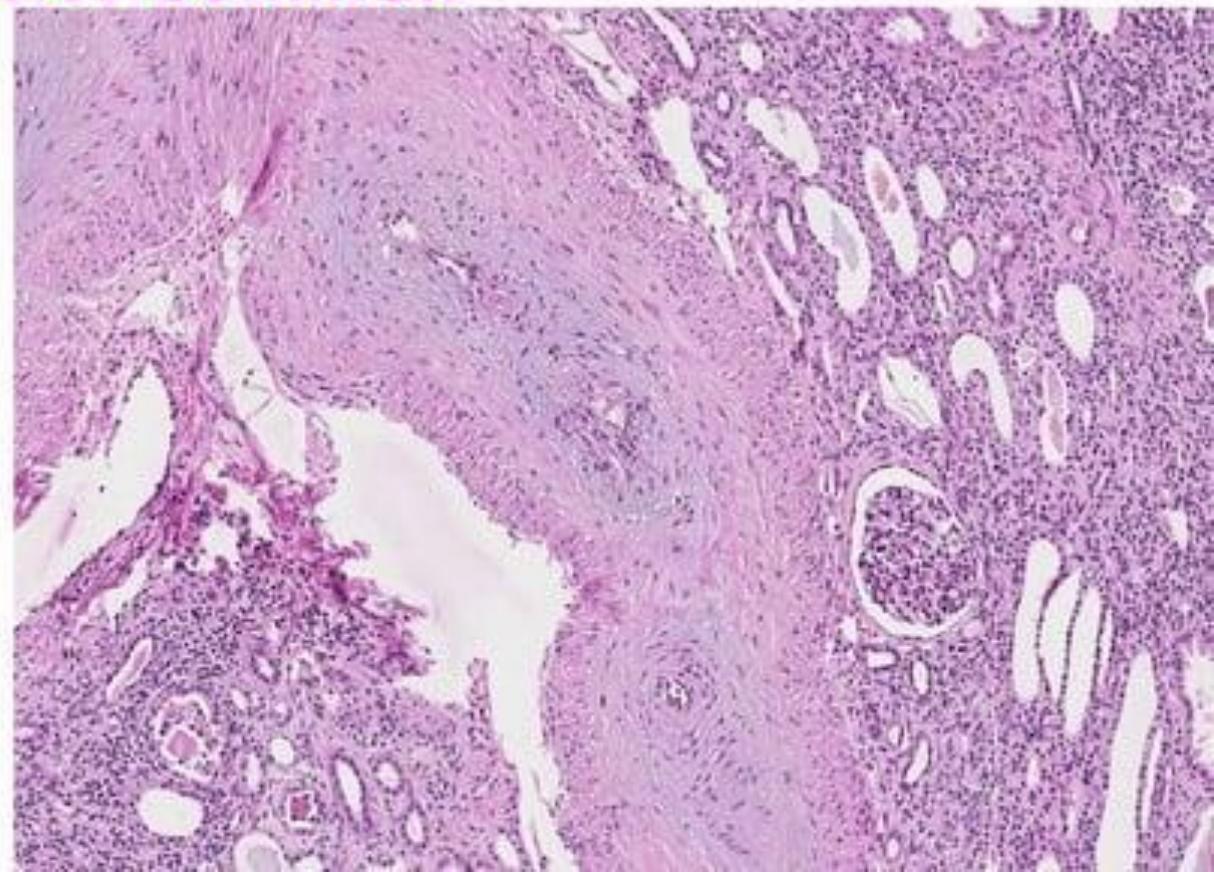
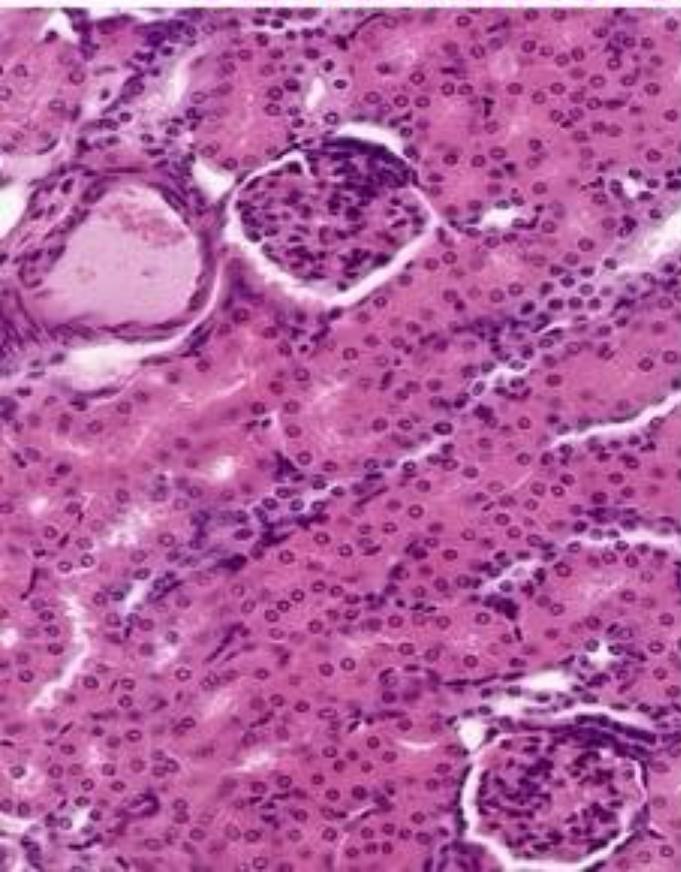
PRINCIPLE: In extreme or uncontrolled or chronic HYPERCALCEMIA, calcium stones form in the tubulo-interstitium of the kidney, which can eventually lead to tubular obstruction and loss of function

VASCULAR DISEASES

- BENIGN NEPHROSCLEROSIS
- MALIGNANT NEPHROSCLEROSIS (i.e., malignant hypertension)
- RENAL ARTERY STENOSIS
- THROMBOTIC MICROANGIOPATHIES
 - Hemolytic-Uremic Syndromes, Child, Adult, TTP
- THROMBI, EMBOLI, INFARCTS
 - SICKLE CELL
 - DIFFUSE CORTICAL NECROSIS

BENIGN NEPHROSCLEROSIS

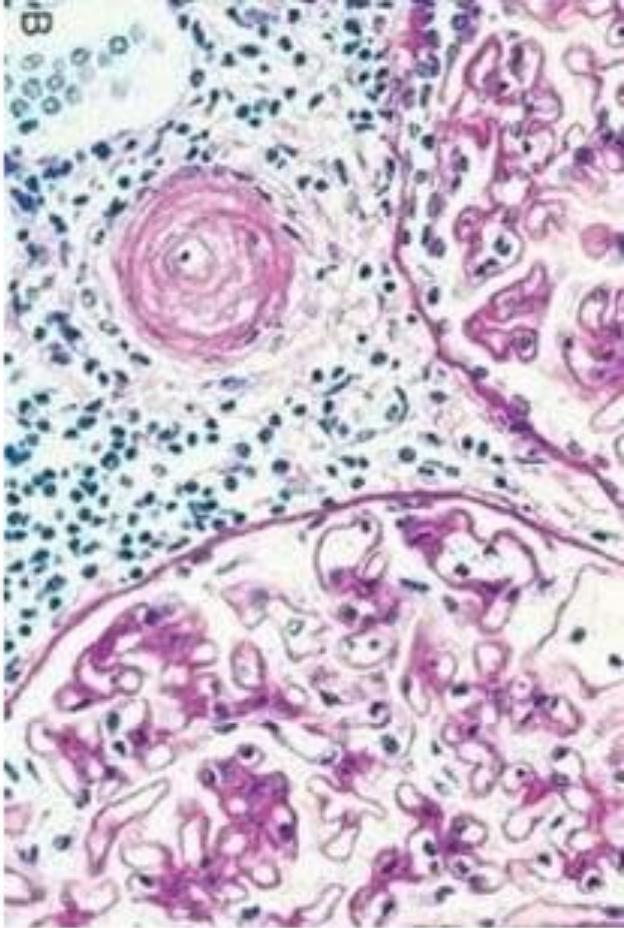
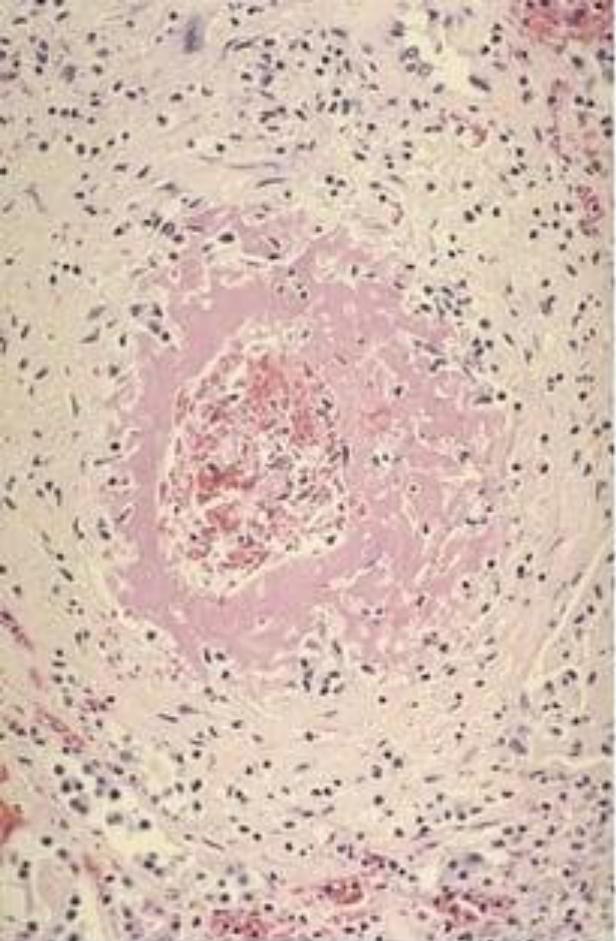
- Sclerosis, i.e., “hyalinization” of arterioles and small arteries, i.e., arterio-, arteriolo-
- Is this part of “routine” atherosclerosis????
- **VERY VERY VERY** common



MALIGNANT NEPHROSCLEROSIS

(i.e., malignant hypertension)

- NOT a part of “routine” atherosclerosis
- By definition, associated with rapidly progressive hypertension (1-2% of HTN)
- **VASCULAR DAMAGE**
- **FIBRINOID NECROSIS**
- **“ONION SKINNING”**
- **SIGNIFICANT LUMENAL NARROWING**



What is “onion-skinning”?

What is an onion?

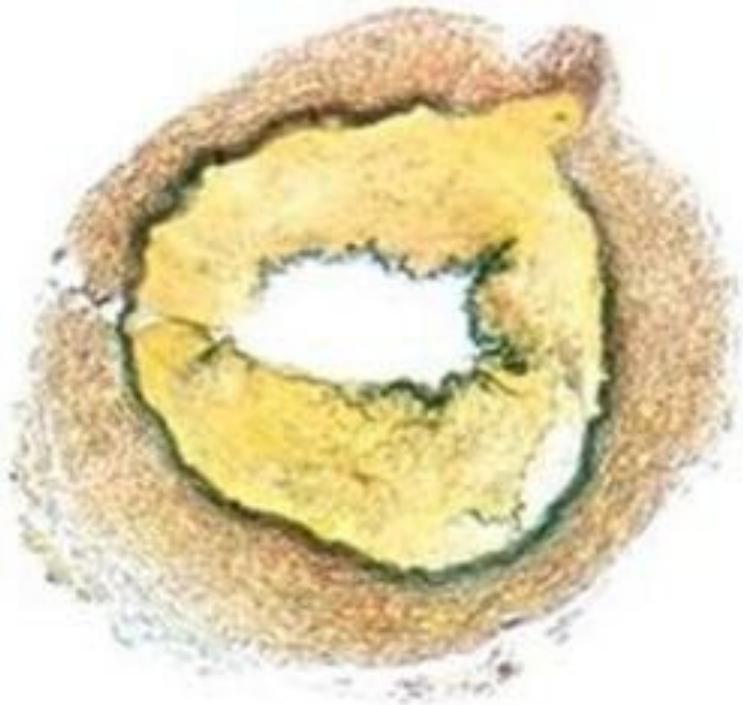
What is “fibrinoid” necrosis?

Renal Artery Stenosis

- Rare cause of HTN
- **SMALL** Kidney
- 1) Plaque type is usual cause, yes regular old atherosclerosis
- 2) Fibromuscular “dysplasia” type:
 - INTIMAL HYPERPLASIA
 - MEDIAL HYPERPLASIA
 - ADVENTITIAL HYPERPLASIA
 - In younger women



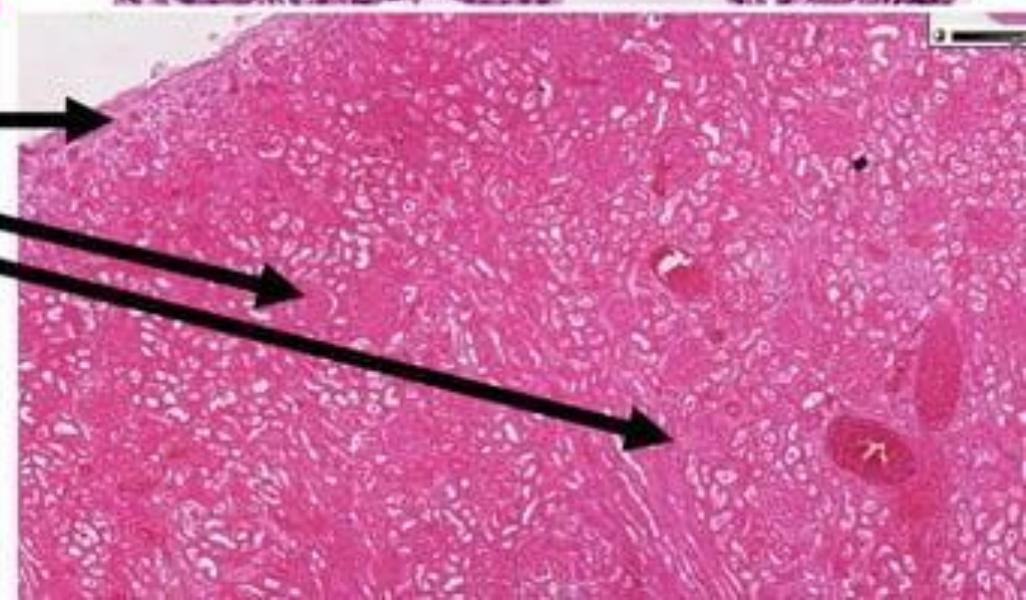
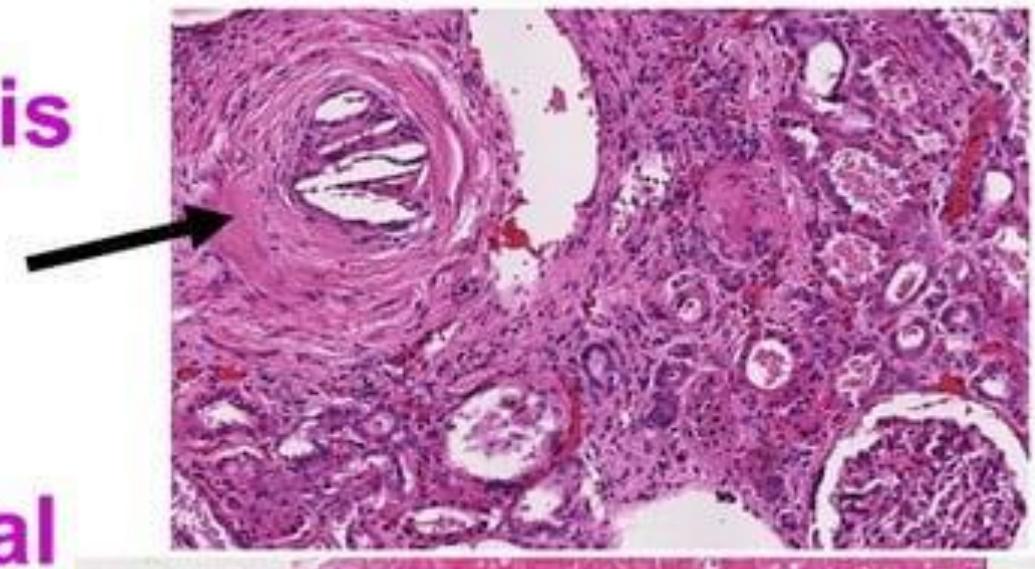
PLAQUE, i.e.,
ATHEROSCLEROSIS



**FIBROMUSCULAR
DYSPLASIA**

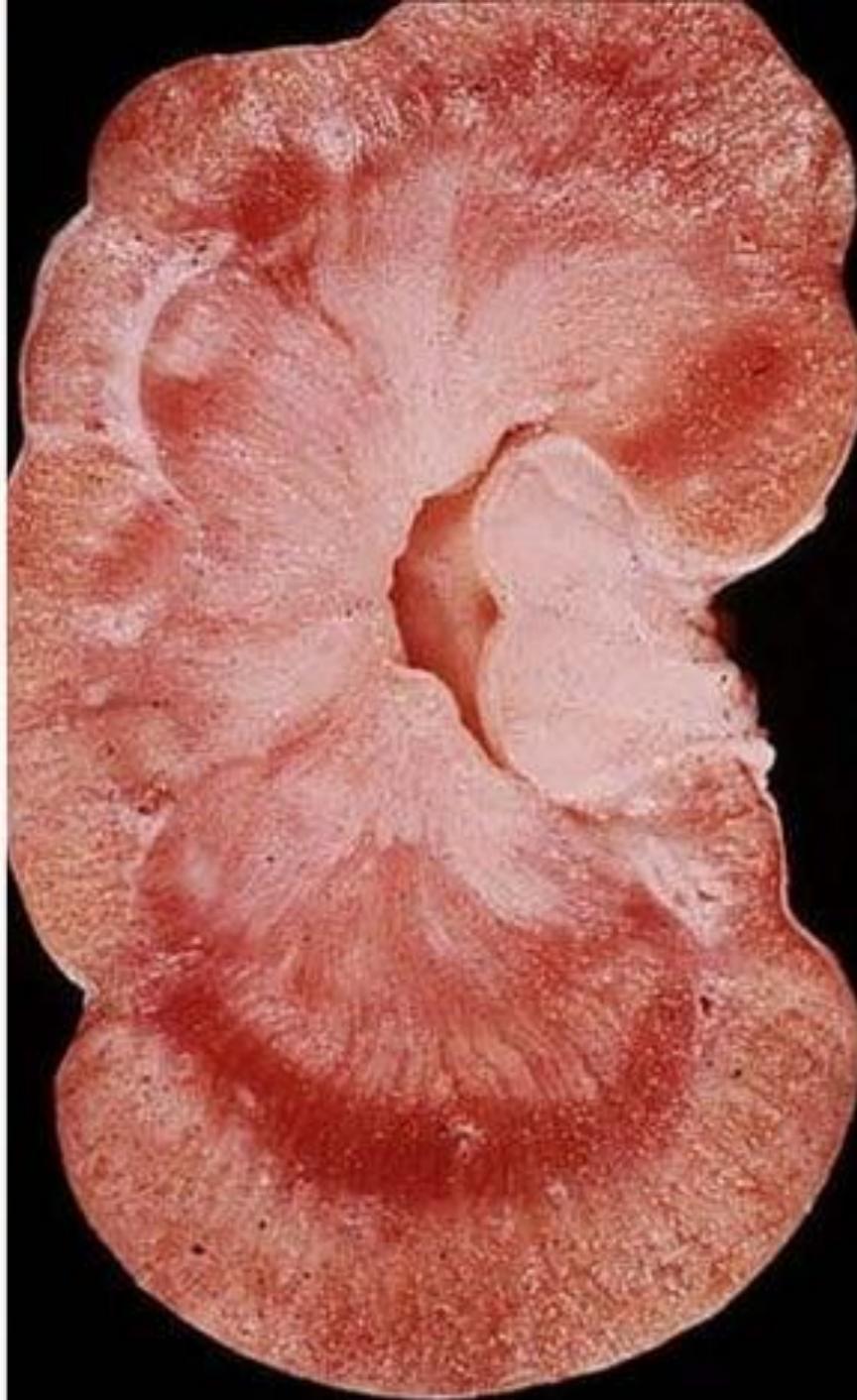
OTHER VASCULAR

- Atherosclerosis
- Atheroemboli
- Sickle Cell
- Diffuse Cortical Necrosis



RENAL INFARCTS

- WEDGE SHAPED
- WELL DELINEATED
- “WHITE” (anemic) INFARCT
- Perhaps a little “YELLOW”
- HEAL WITH A SCAR





Kidney is precious.