4. Pathology Anatomy of Urinary Tract

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KIDNEY

I. Congenital / developmental disorders

II. Renal calculi

III. Urinary obstruction

IV. Glomerular disease

V. Pyelonephritis (tubulointerstitial nephritis)

VI. Hypertension

VII. Acute renal failure (ARF)

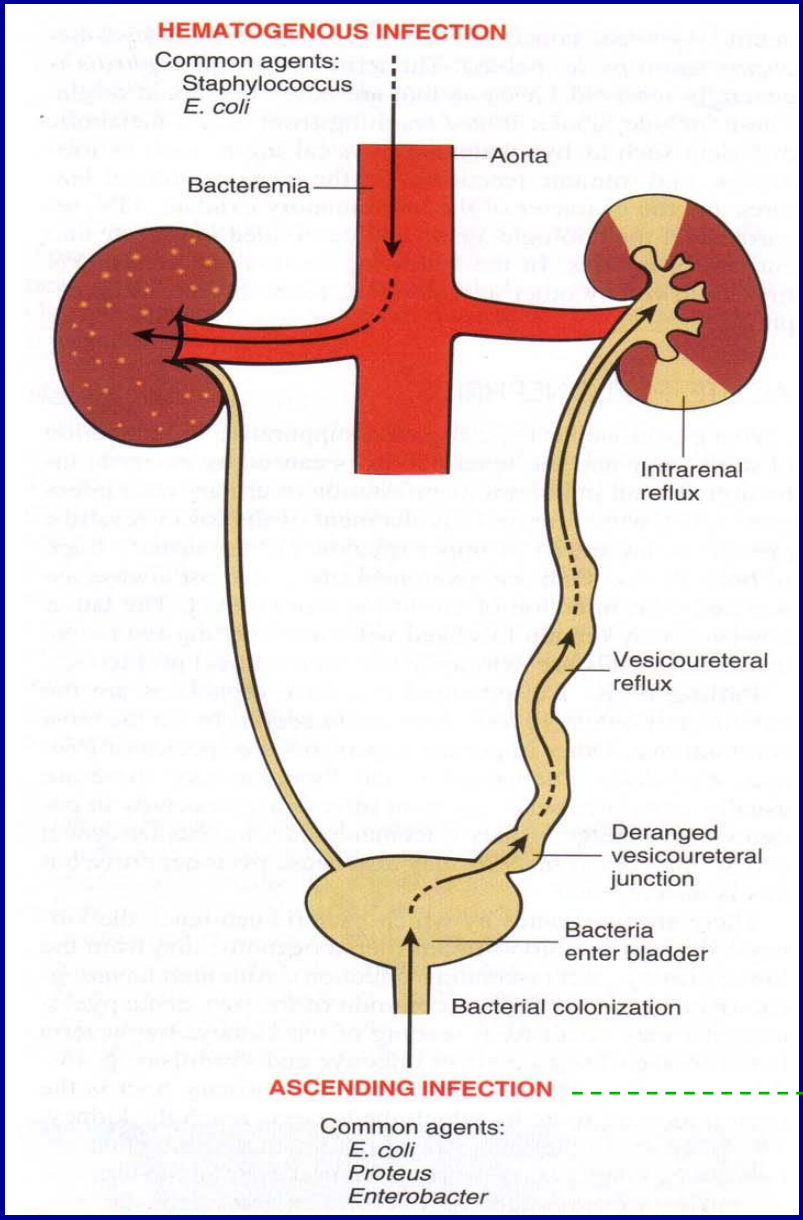
VIII. Chronic renal failure (CRF)

IX. Neoplasms

X. Renal manifestations of systemic disease

V. Pyelonephritis (TUBULOINTERSTTIAL NEPHRITIS)

- Most commonly infectious in origin: → gram negative bacteria (usually E. Coli) from patient's intestinal tract
- Normal kidney is resistant to blood borne infection → Most common route of infection: bacterial ascension from the urinary bladder



Schematic representation of pathways of renal infection

More common

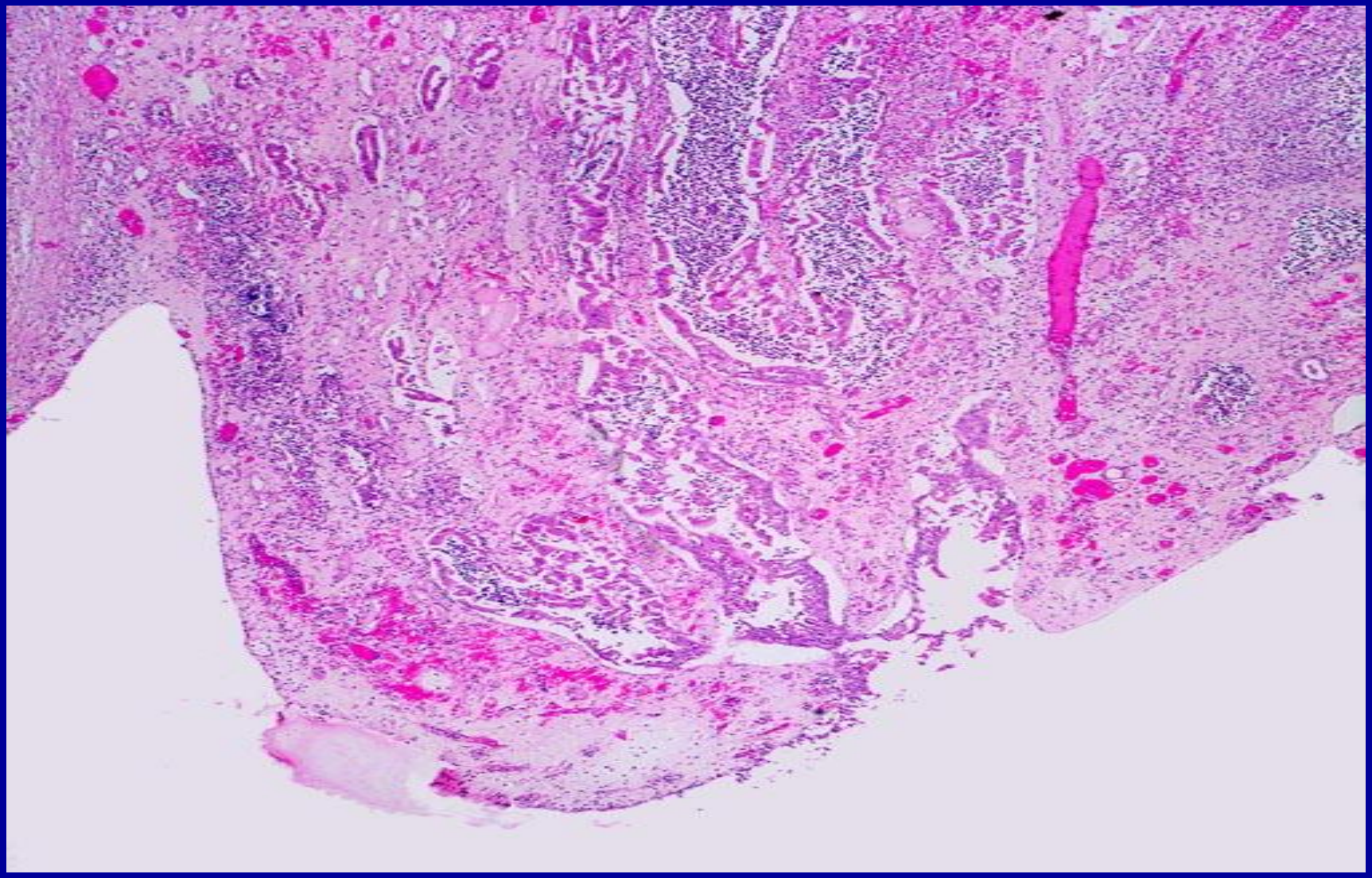
V. A. Acute pyelonephritis

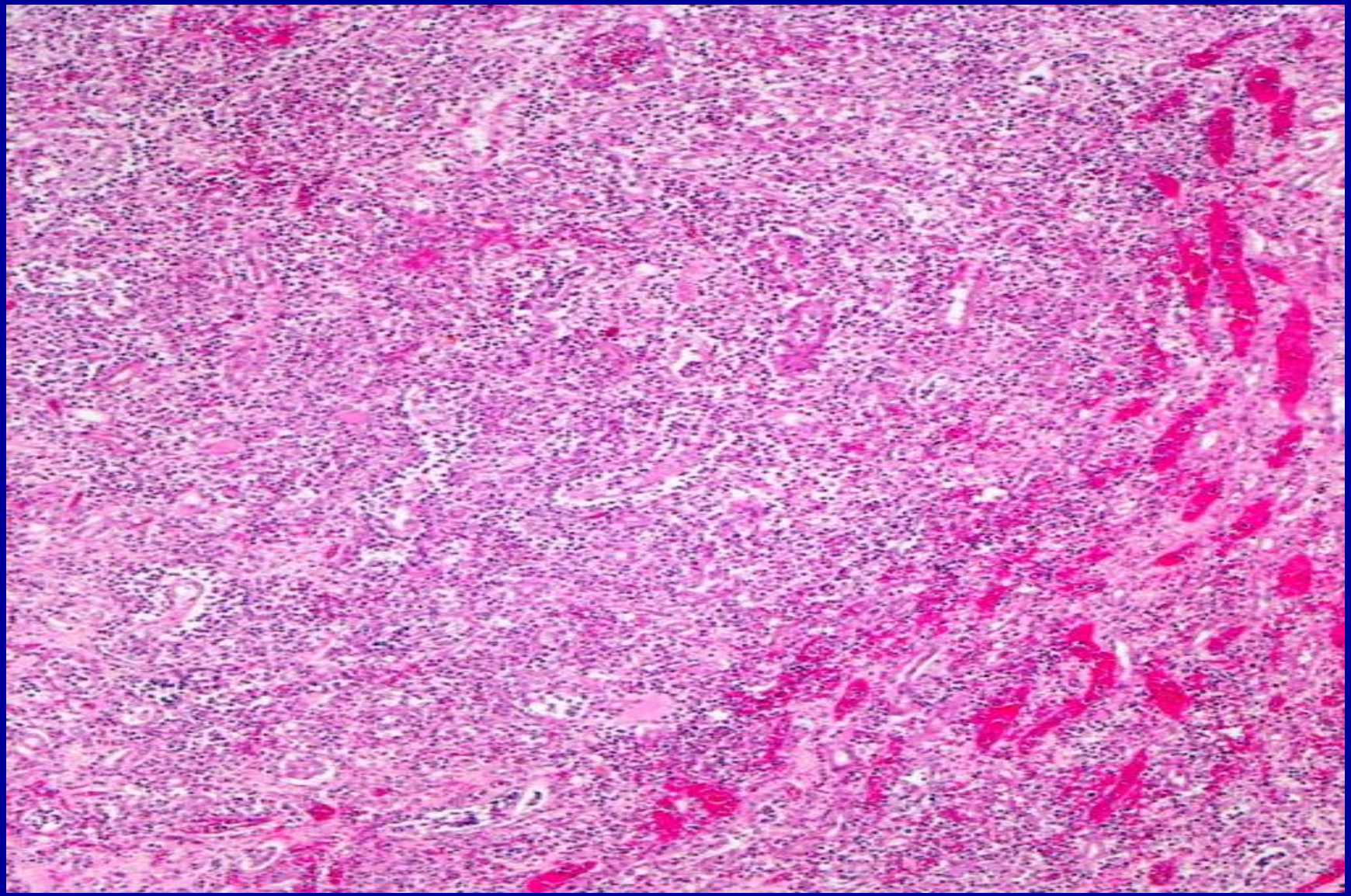
Acute disease often associated with:

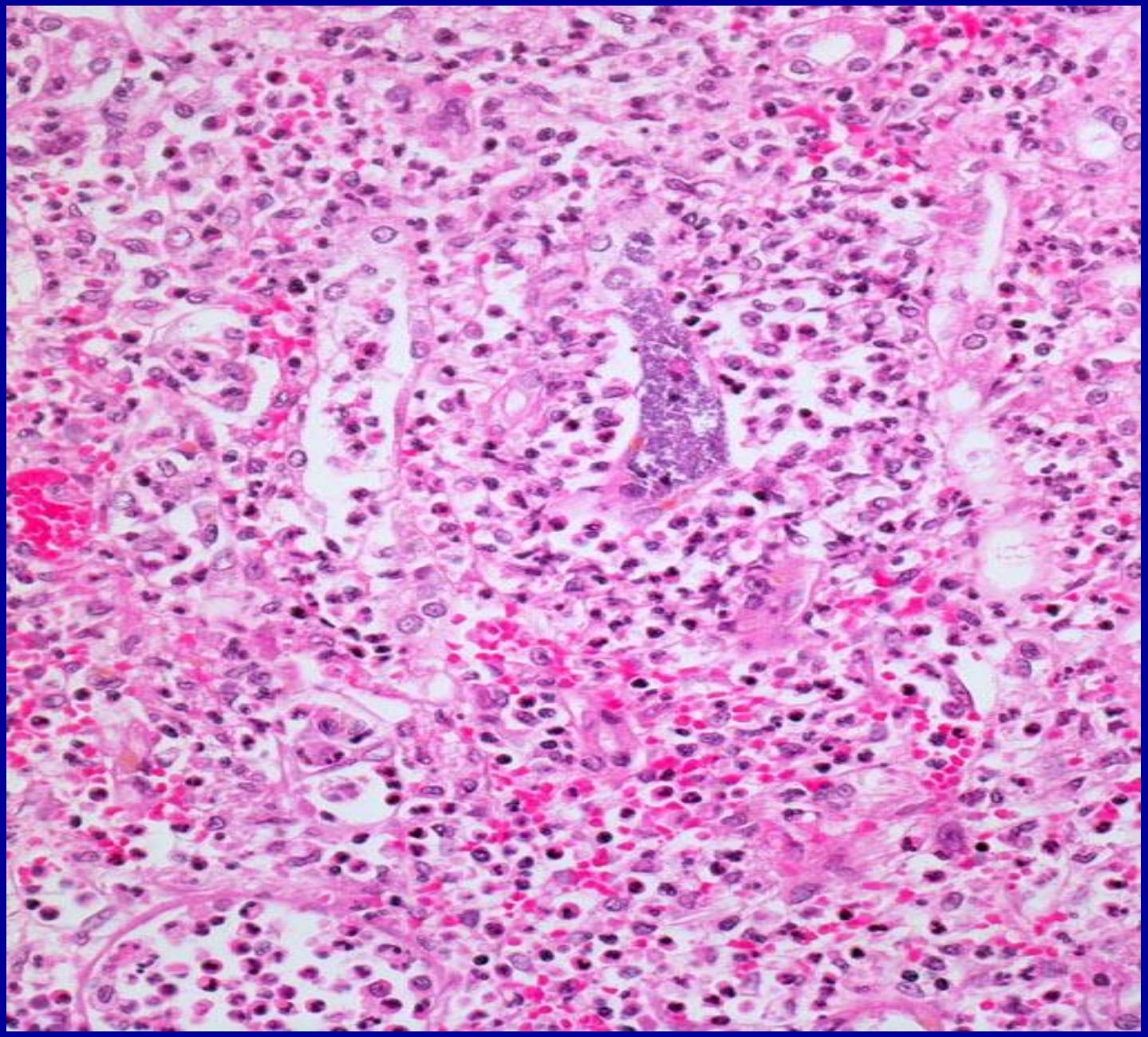
- Urinary obstruction
- Instrumentation of the urinary tract
- Vesicoureteral reflux
- Pregnancy
- Prior renal disease
- diabetes

Histology:

- Interstitial suppurative inflammation and tubular necrosis
- Abscesse often multiple → rupture to tubules → collecting ducts → complications: necrotizing papillitis, pyonephrosis, perinephric abscess





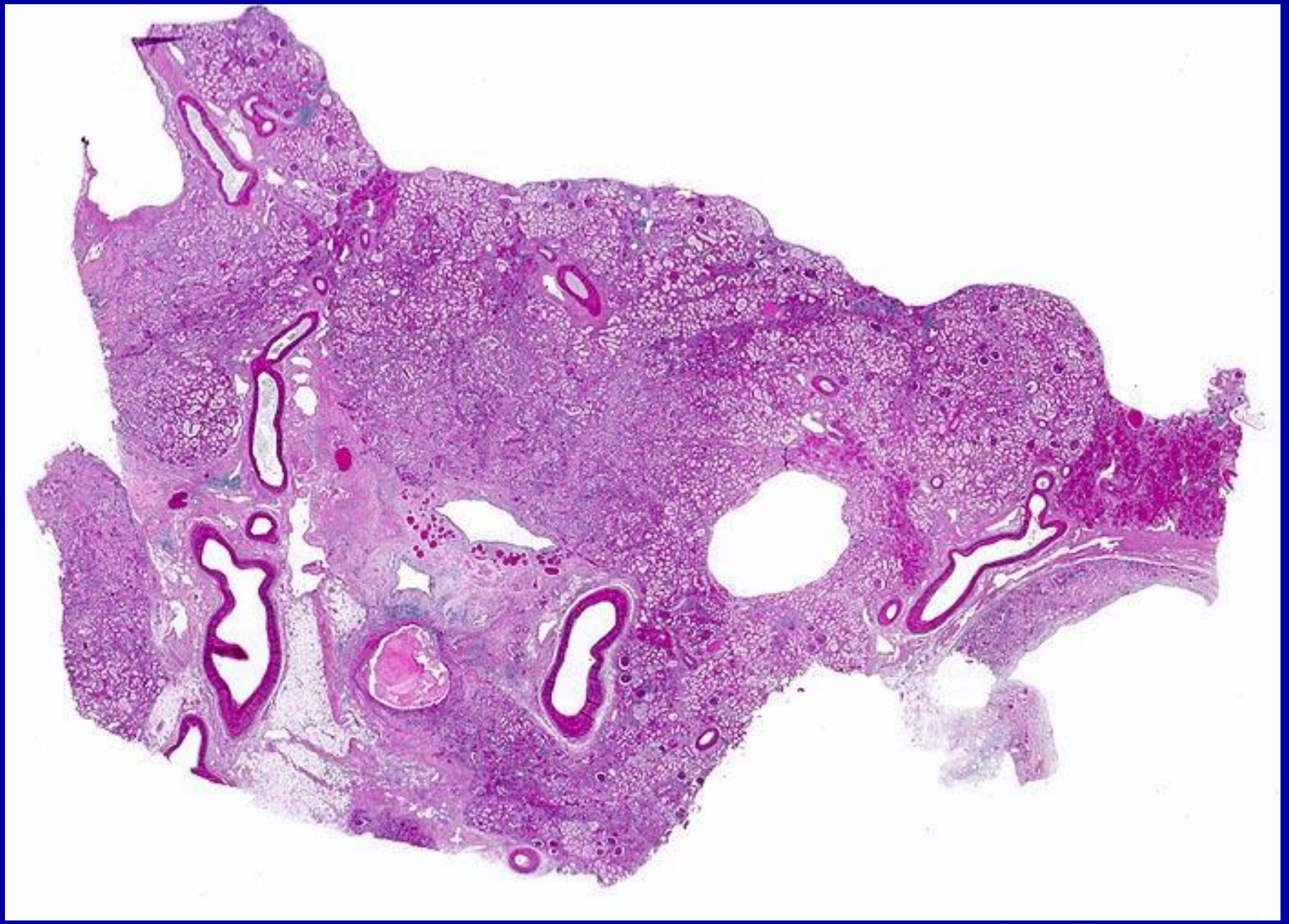


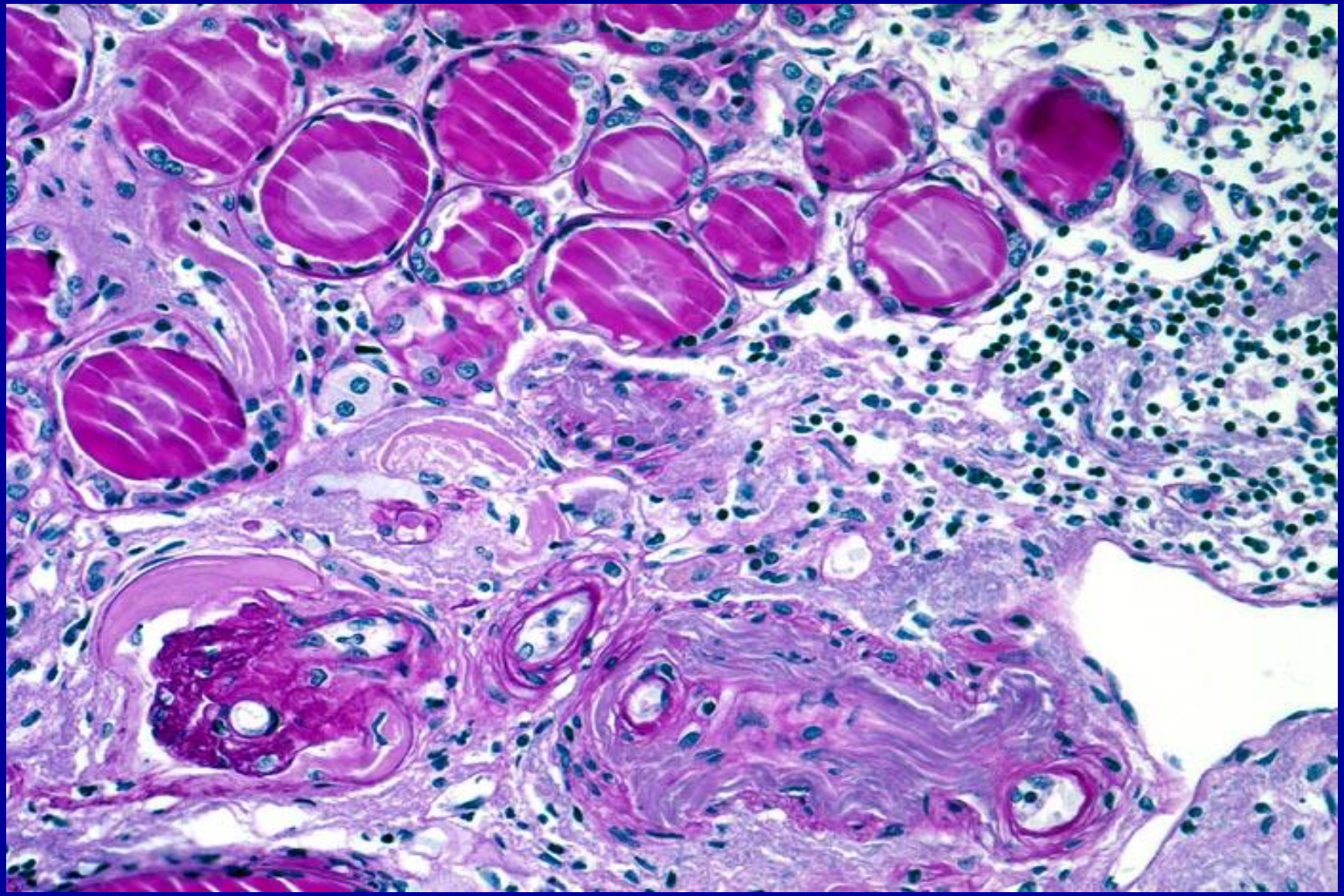
Acute Pyelonephritis

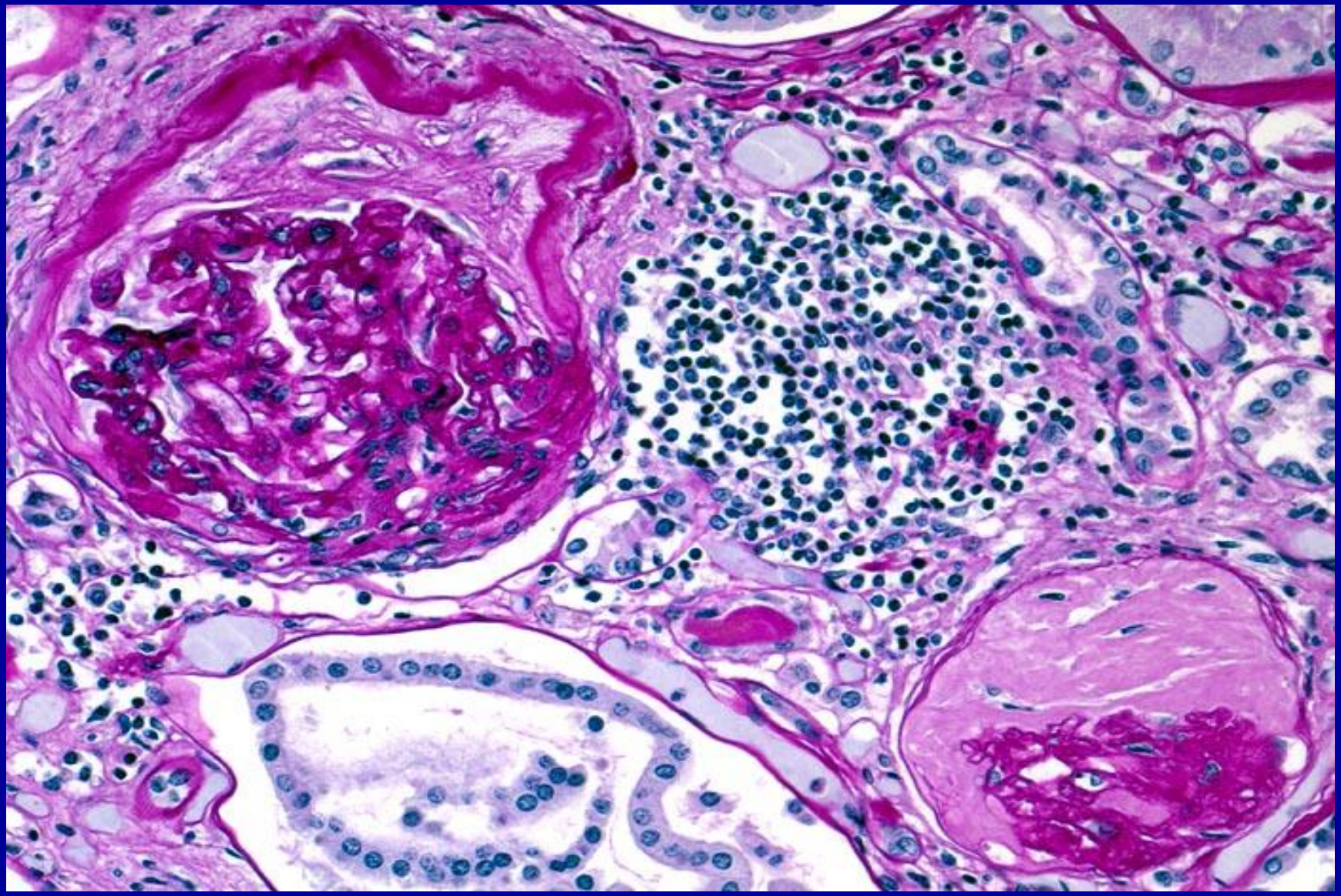
- The cortical surface is studded with focal pale abscesses
- The lower pole is relatively unaffected
- Between the abscess there is dark congestion of the renal surface

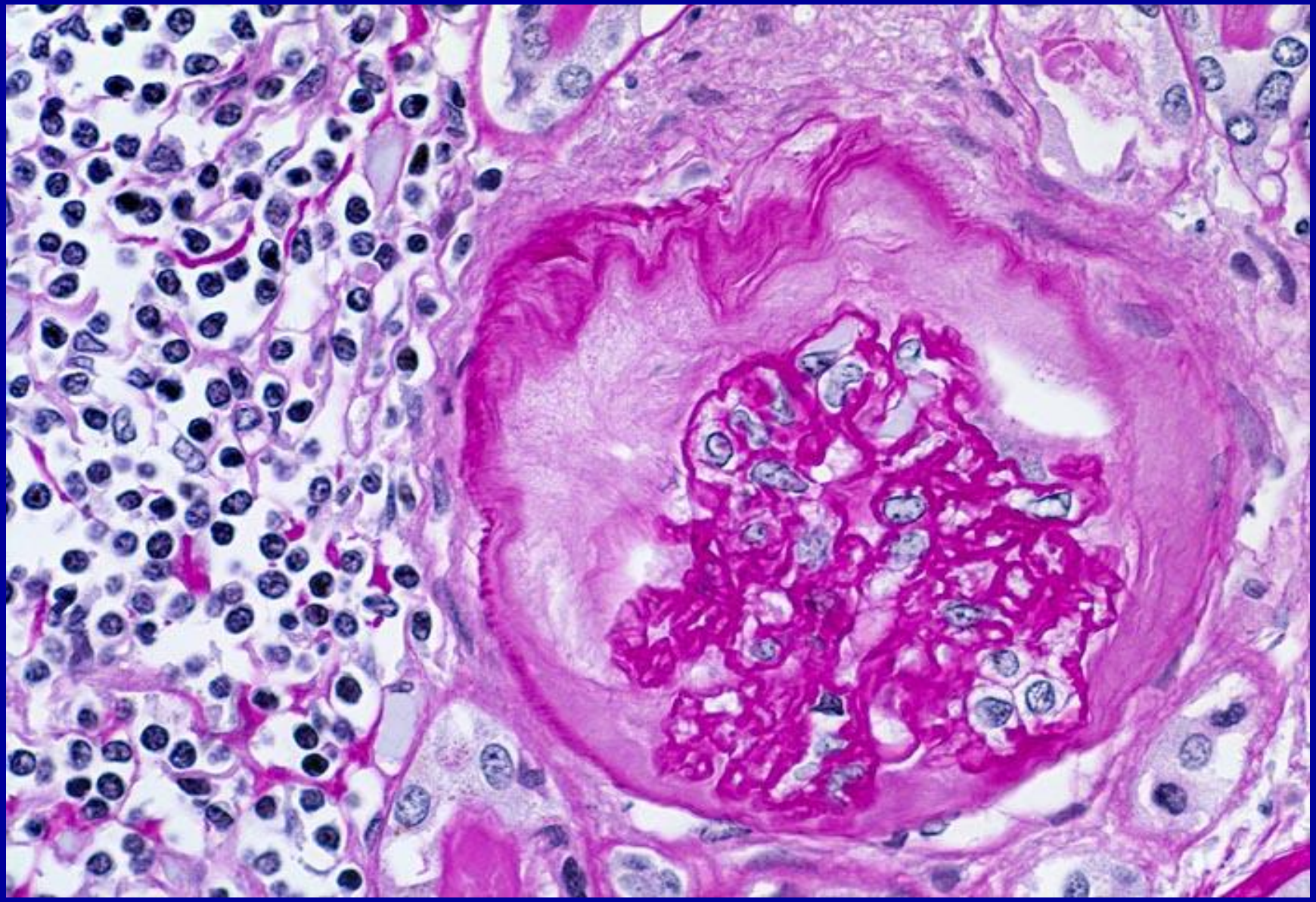
V. B. Chronic pyelonephritis

- Major cause of CRF
- Common etiology reflux nephropathy, and less common by chronic obstruction leading to recurrent infections
- Clinically may be insidious presenting as renal insufficiency and hypertension, or as repeated acute pyelonephritis
- Tubular damage → polyuria and nocturia
- Grossly: kidney are small with broad irregular scar and deformed blunted calyces
- Mic: tubular atrophy and/or thyroidization with chronic inflammation → periglomerular fibrosis → glomerular sclerosis (late state)

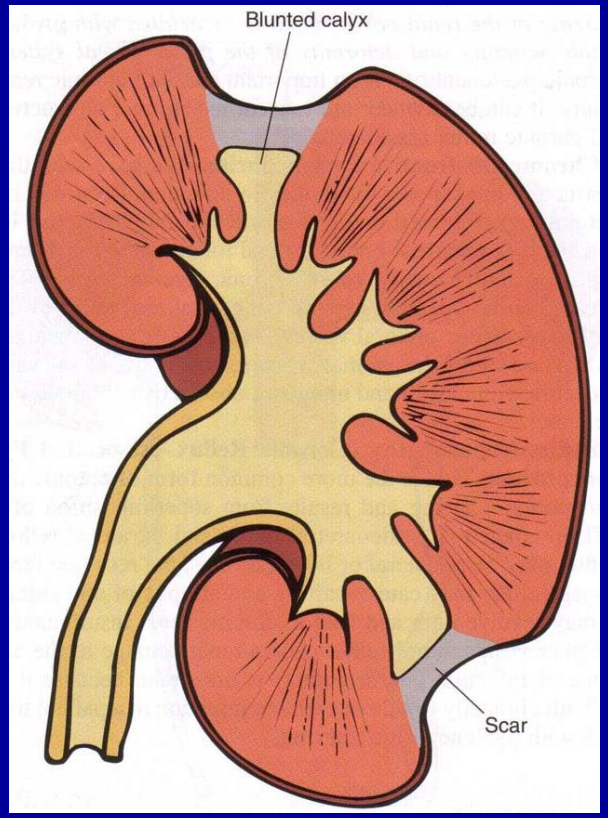






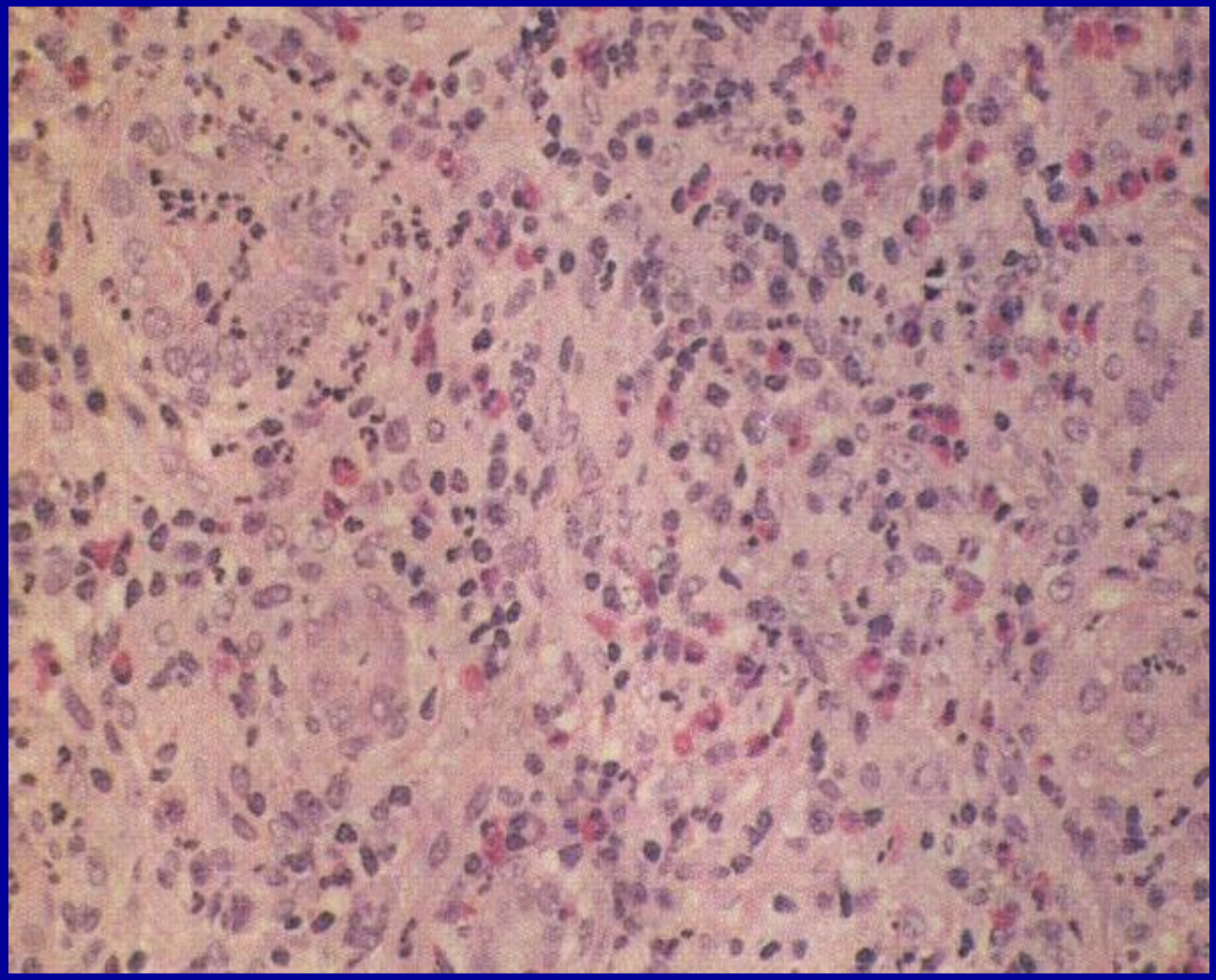


Chronic Reflux-Associated Pyelonephritis



- Typical coarse scars → the scars are usually polar and are associated with with underlying blunted calices

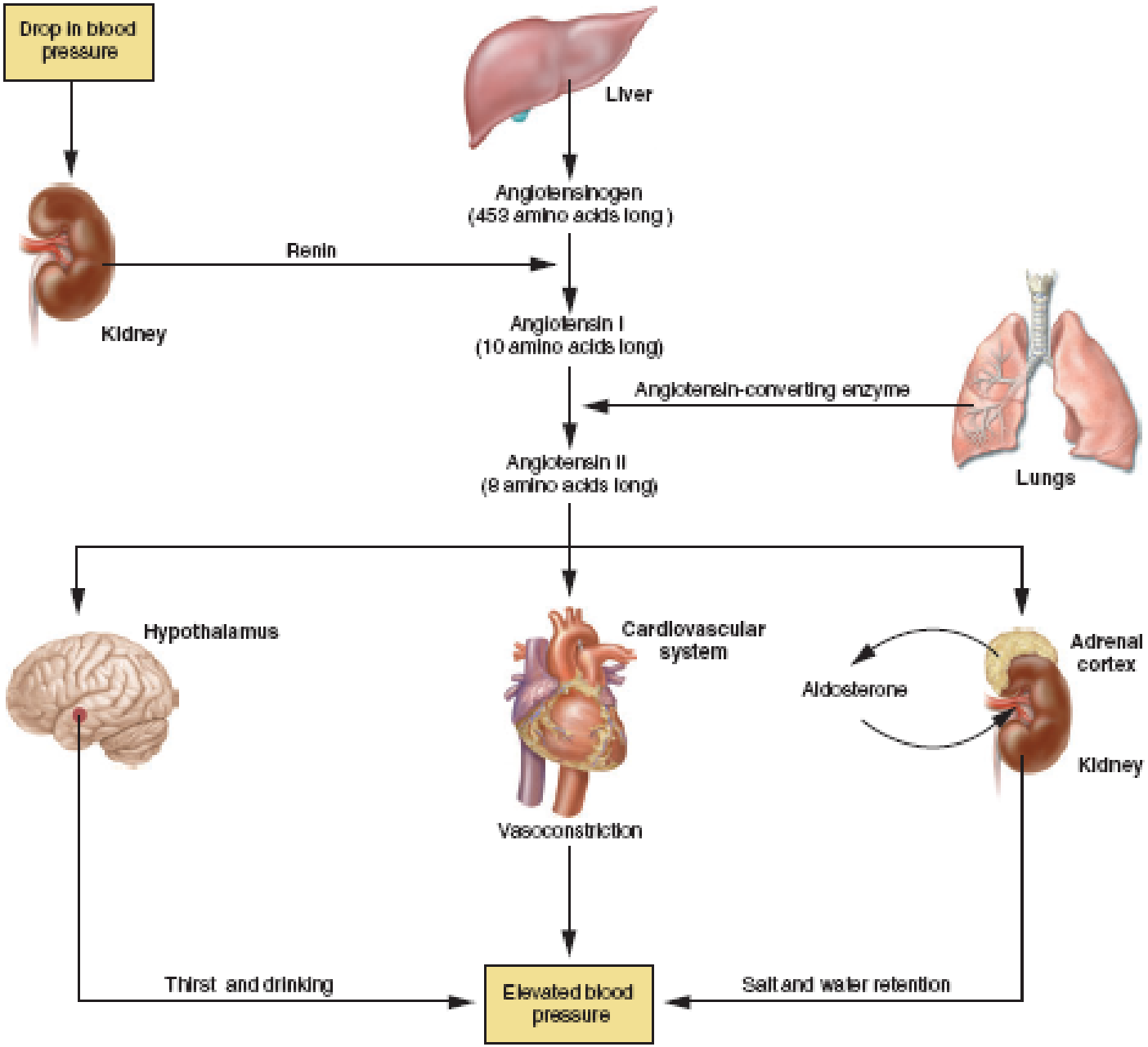
Drug-induced interstitial nephritis



- Prominent eosinophilic and mononuclear infiltrate

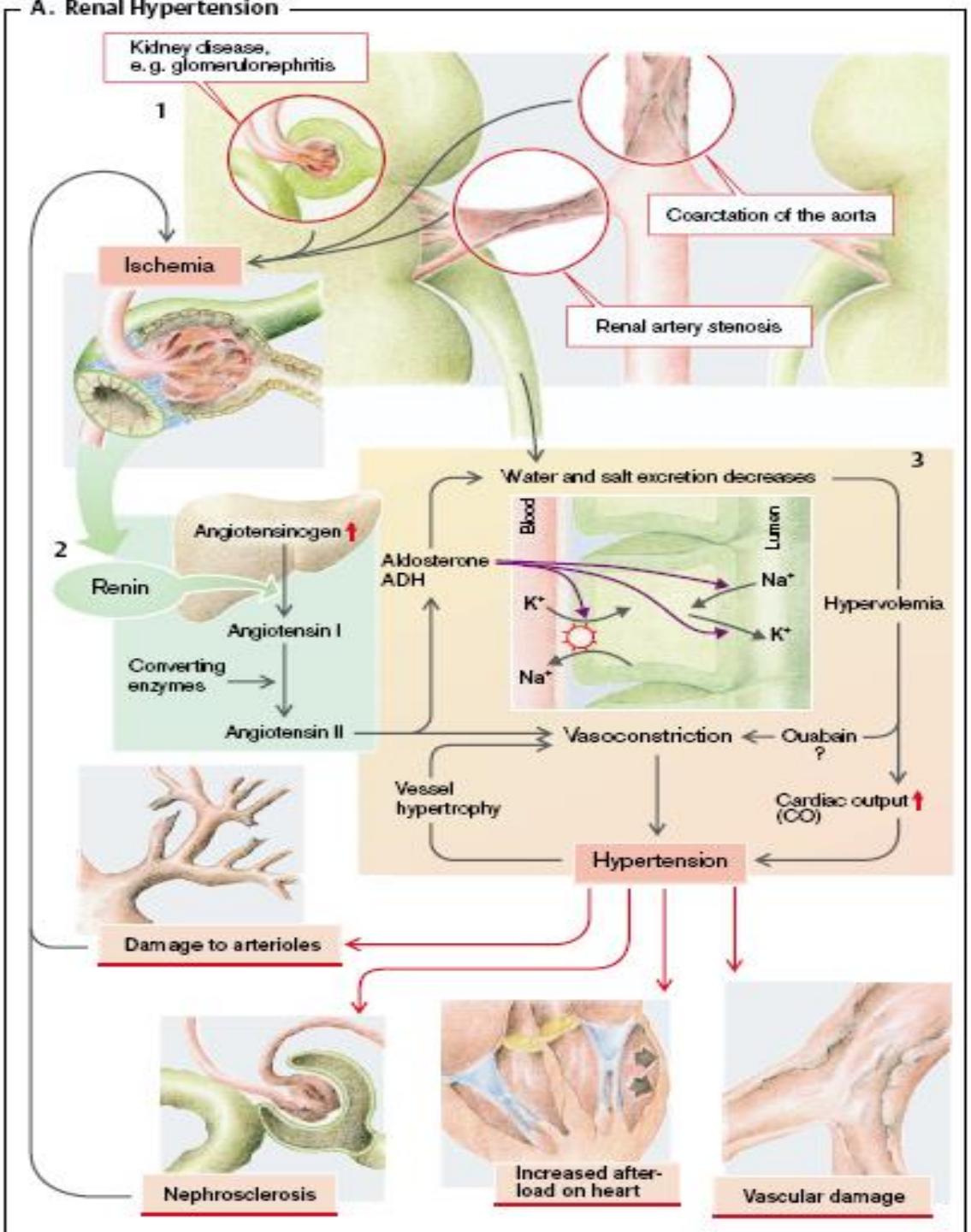
VI. Hypertension

- Blood pressure is determined by cardiac output and peripheral resistance of arterioles
- Peripheral resistance is regulated by vaso-constrictors (angiotensin II, leukotrienes, thromboxane, catecholamines) and vasodilators (kinin and prostaglandin)



23.13 The Renin-Angiotensin-Aldosterone Mechanism. This chain of events is activated by a drop in blood pressure and acts to raise

A. Renal Hypertension



VI. Hypertension: A. Renal Control

- Sekresi substansi pressor: renin: circulating liver derived globulin (angiotensinogen) → angiotensin I → angiotensin II → increased peripheral resistance
- Maintenance of fluid and electrolit balance (mechanism → increased and H2O retension → increased blood-pressure)

  Sodium is controled by:
  - GFR (decreased GFR → less filtered NA^+ & increased proximal tubular reabsorption)
  - aldosterone (increases NA^+ reabsorption in distal tubules)
  - natriuretic factor (increased of this → loss of NA^+
- Renal antihypertensive: prostaglandin, kallikrein-kinin, neutral lipid factor

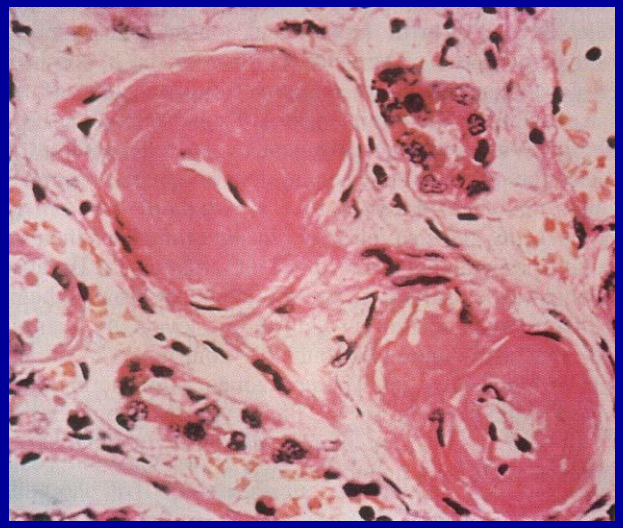
VI. Hypertension: B. Renal manifestations

- Benign (essential) hypertension
  - Bilateral fine cortical granularity
  - Arteriolonephrosclerosis → ischemic damage → glomerular damage, tubular atrophy & interstitial fibrosis.
  - Collagen is deposited inside Bowman's capsule
- Malignant hypertension
  - onion skin
  - fibrinoid necrosis

Table 23.1 Hormones Affecting Renal Function

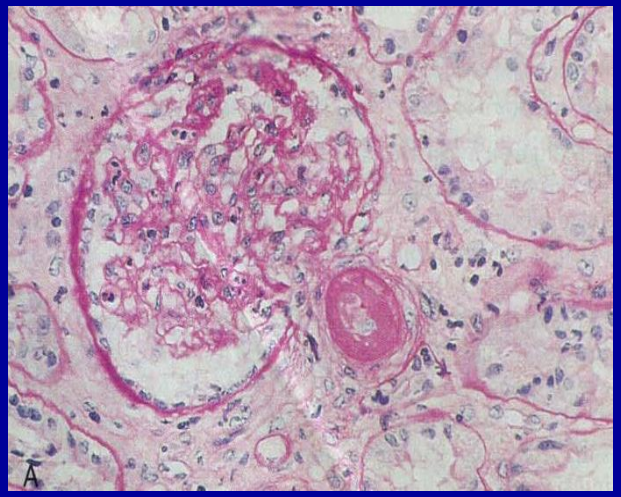
Hormone	Target	Effects
Aldosterone	Distal tubule, collecting duct	Promotes Na^+ reabsorption, K^+ secretion; reduces urine volume
Angiotensin II	Afferent and efferent arterioles	Constricts arterioles, reduces GFR; stimulates ADH and aldosterone secretion; stimulates thirst; promotes water intake and reduces urine volume
Antidiuretic hormone	Collecting duct	Promotes H_2O reabsorption; reduces urine volume, increases concentration
Atrial natriuretic peptide	Afferent and efferent arterioles, collecting duct	Dilates afferent arteriole, constricts efferent arteriole, increases GFR; inhibits secretion of renin, ADH, and aldosterone; inhibits NaCl reabsorption by collecting duct; increases urine volume
Epinephrine and norepinephrine	Juxtaglomerular apparatus, afferent arteriole	Induces renin secretion; constricts afferent arteriole; reduces GFR and urine volume
Parathyroid hormone	Proximal and distal tubules, nephron loop	Promotes Ca^{2+} reabsorption by loop and distal tubule and Mg^{2+} reabsorption by proximal tubule; inhibits phosphate reabsorption by proximal tubule; promotes calcitriol synthesis

Benign Nephrosclerosis

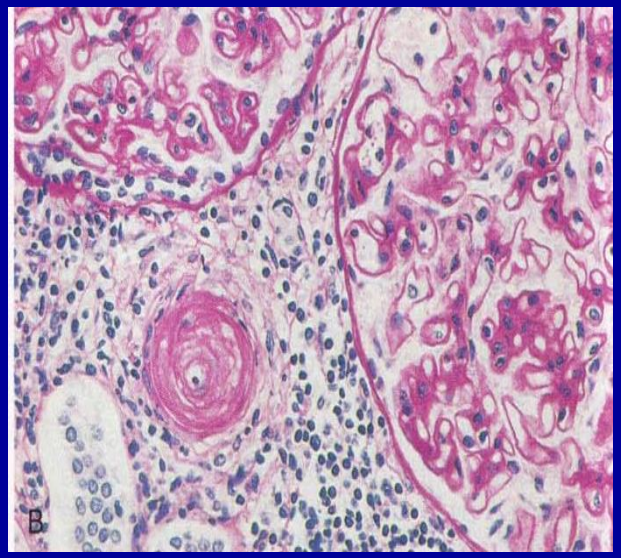


- Arterioles with hyaline deposition, marked thickening of the walls, and a narrowed lumen

Malignant Hypertension



A. Fibrinoid necrosis of afferent arteriole



B. Hyperplastic arteriole (onion-skin lesion)

(PAS stain)

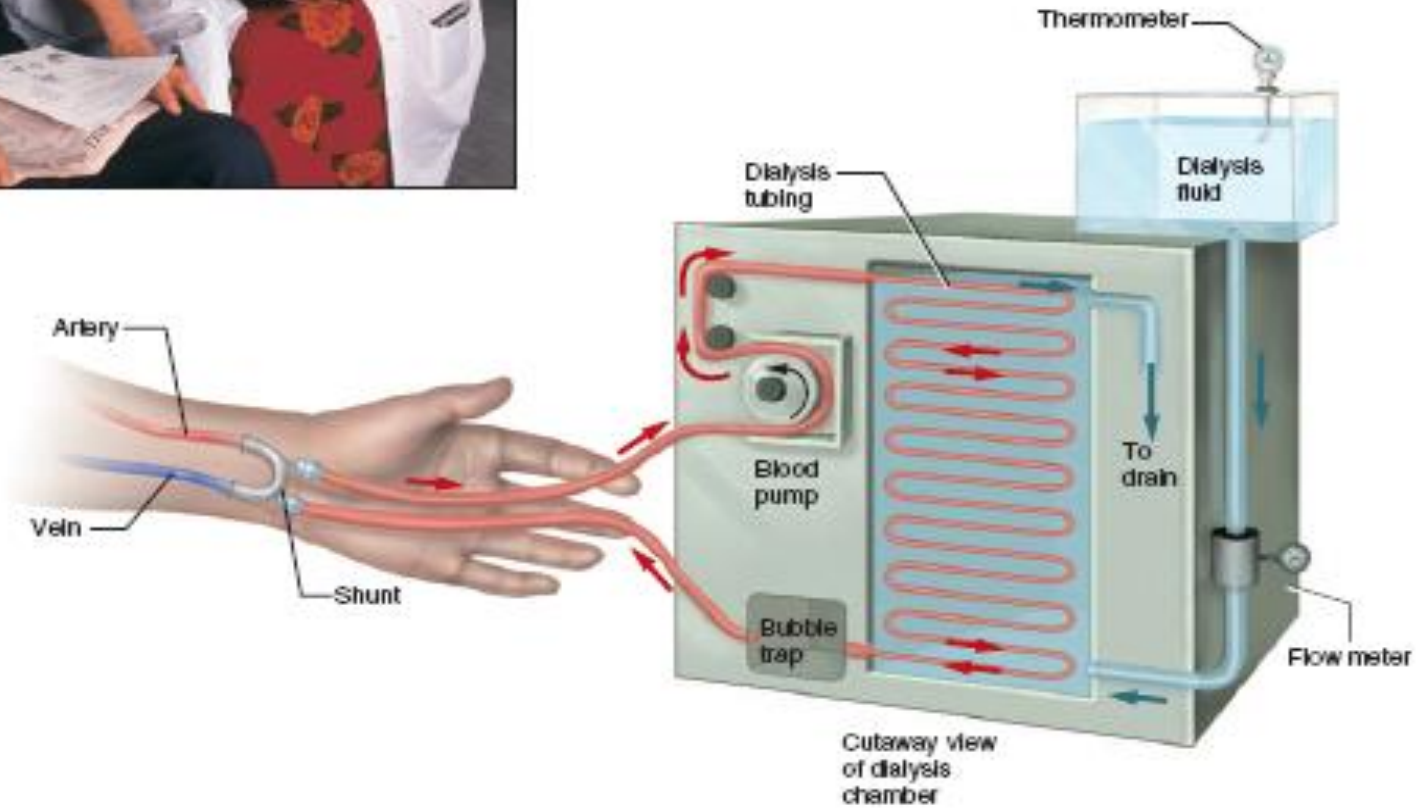


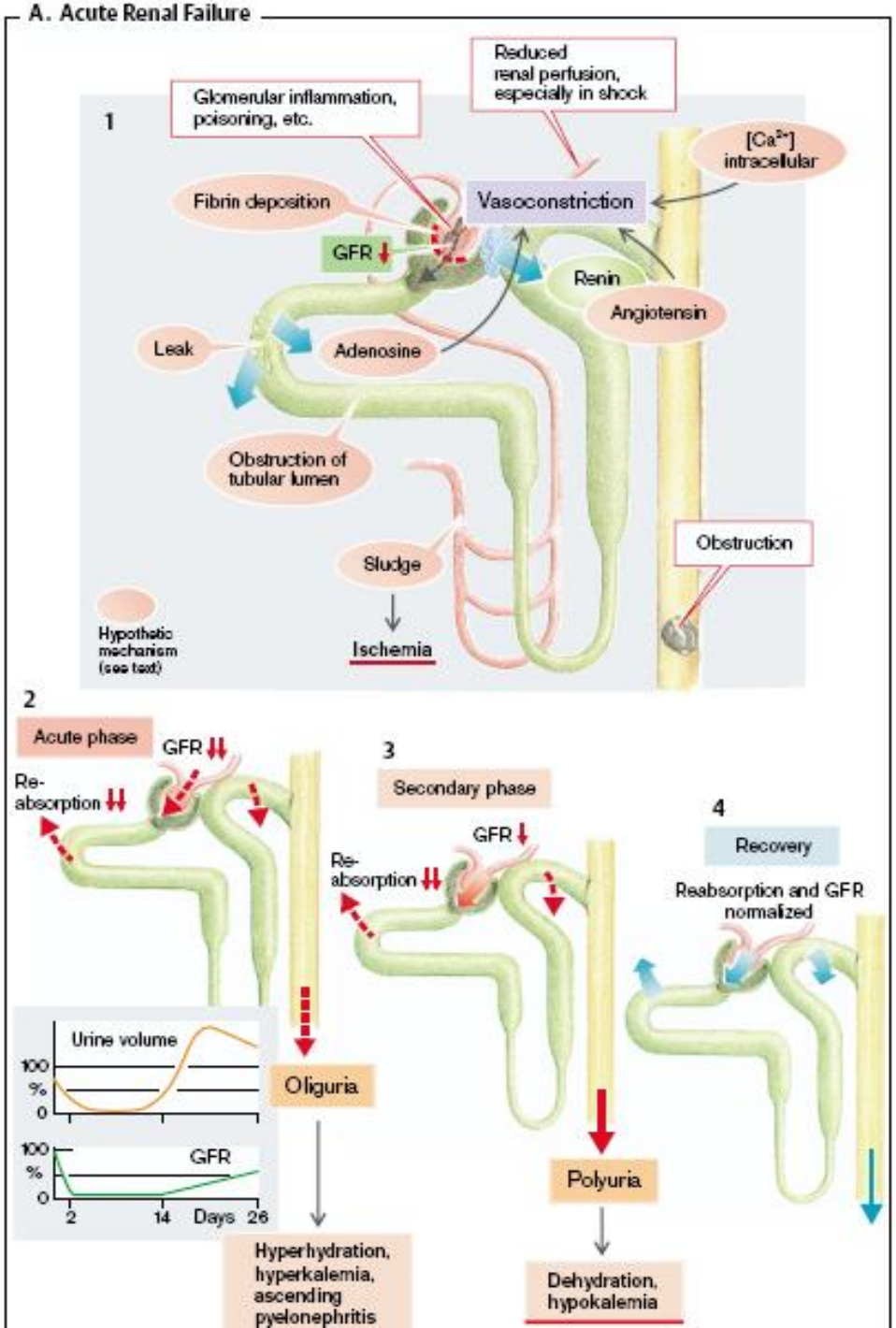
Figure 23.22 Hemodialysis. Blood is pumped into a dialysis chamber, where it flows through selectively permeable dialysis tubing surrounded by dialysis fluid. Blood leaving the chamber passes through a bubble trap to remove air before it is returned to the patient's body. The dialysis fluid picks up excess water and metabolic wastes from the patient's blood and may contain medications that diffuse into the blood.

VII. Acute renal failure

Etiologi:

- Diffuse vascular disease: polyarteritis, malignant hypertension
- Severe glomerular disease: RPGN
- Acute interstitial disease: hypersensitivity to drug
- Massive pyelonephritis
- Cortical necrosis
- Urinary obstruction
- Acute tubular necrosis

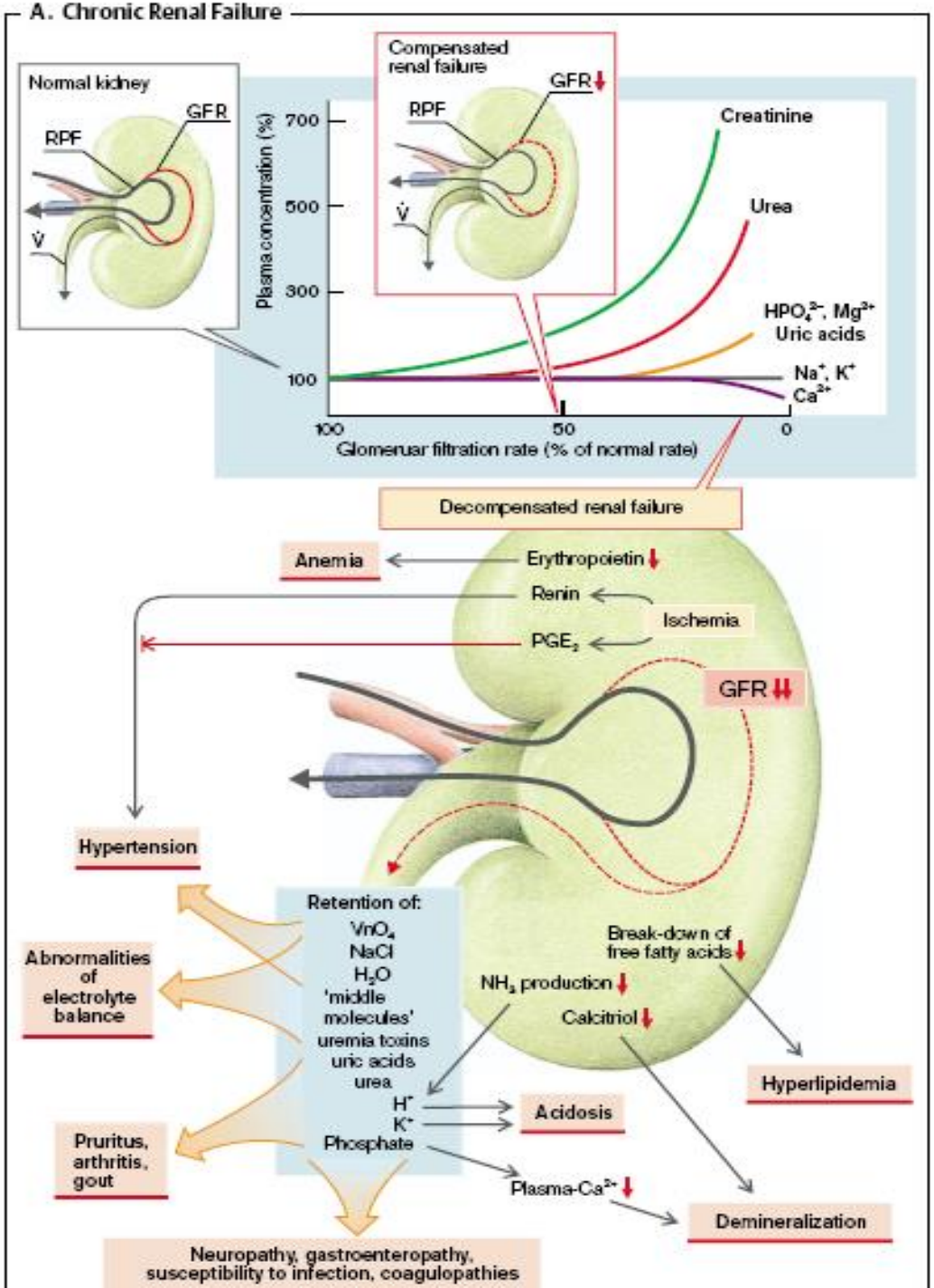
A. Acute Renal Failure



VIII. Chronic renal failure

- GFR 20-30% of normal → azotemia (increased BUN & creatinine) → associated with hypertension
- GFR 15-20% of normal → azotemia is complicated by uremic syndrome
- Clinical manifestations are systemic

A. Chronic Renal Failure

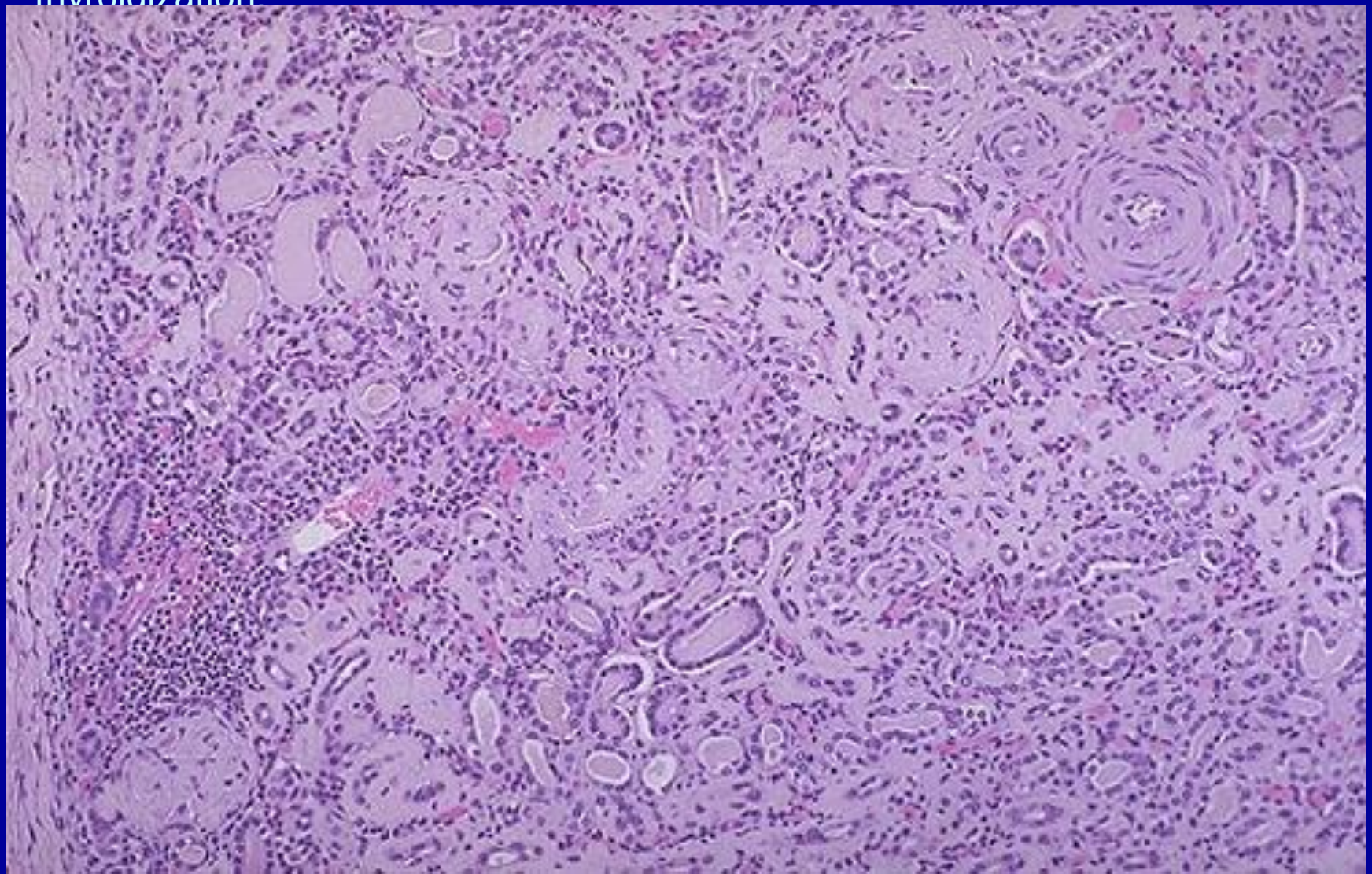


Clinical manifestations

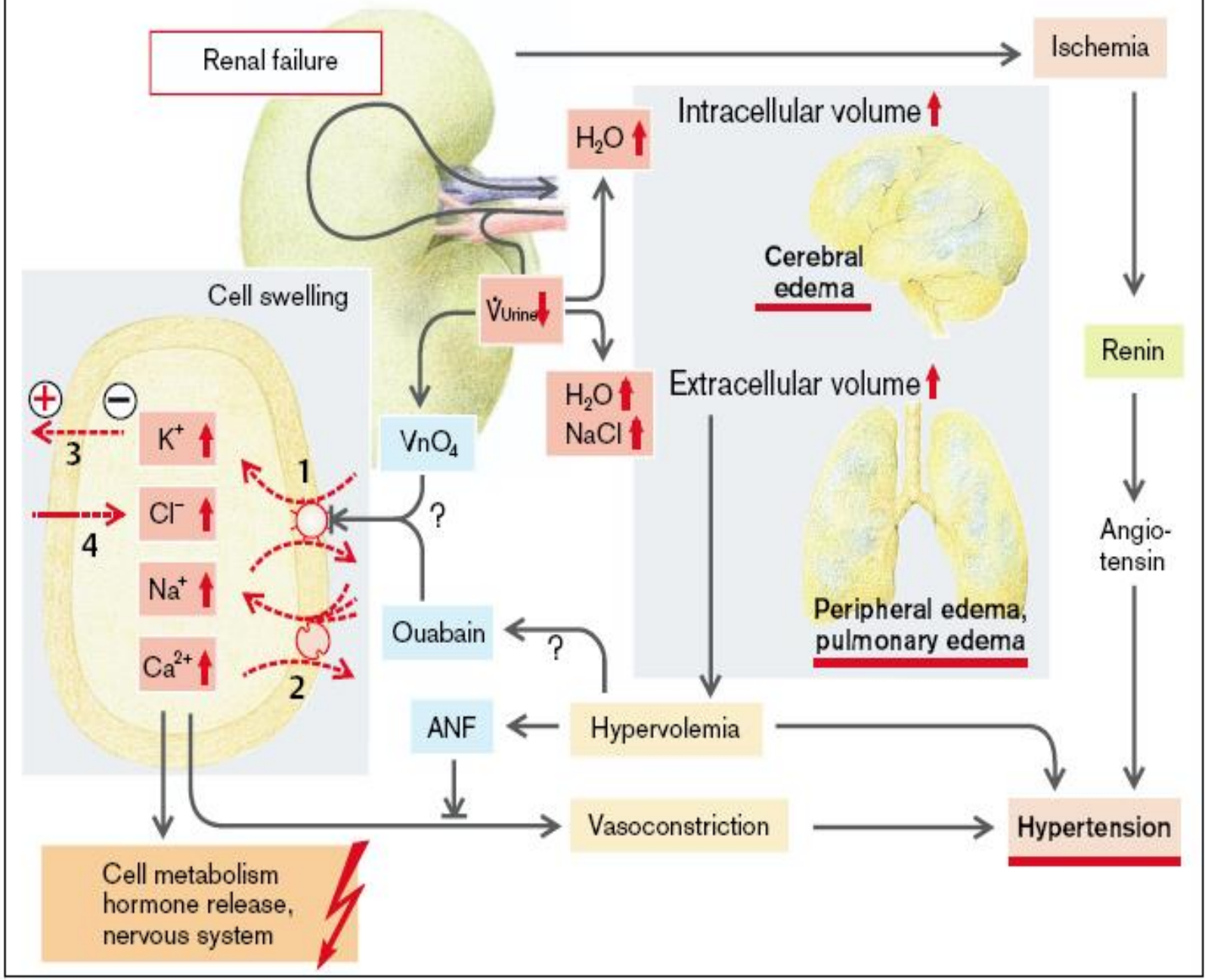
- Fluid, electrolyte, and acid/base manifestation
- Cardiopulmonary manifestation
- Hematologic manifestation
- Gastrointestinal manifestation
- Dermatologic manifestation
- Neurologic manifestation

The end result of many renal diseases -- whether they are renal vascular diseases, glomerulonephritis, or chronic pyelonephritis--is end stage renal disease (ESRD). In ESRD, the kidneys are small bilaterally, as shown here. This condition is associated with chronic renal failure, and the patient's blood urea nitrogen (BUN) and serum creatinine continue to increase. Chronic renal failure can be treated by dialysis or by kidney transplantation, as shown here.

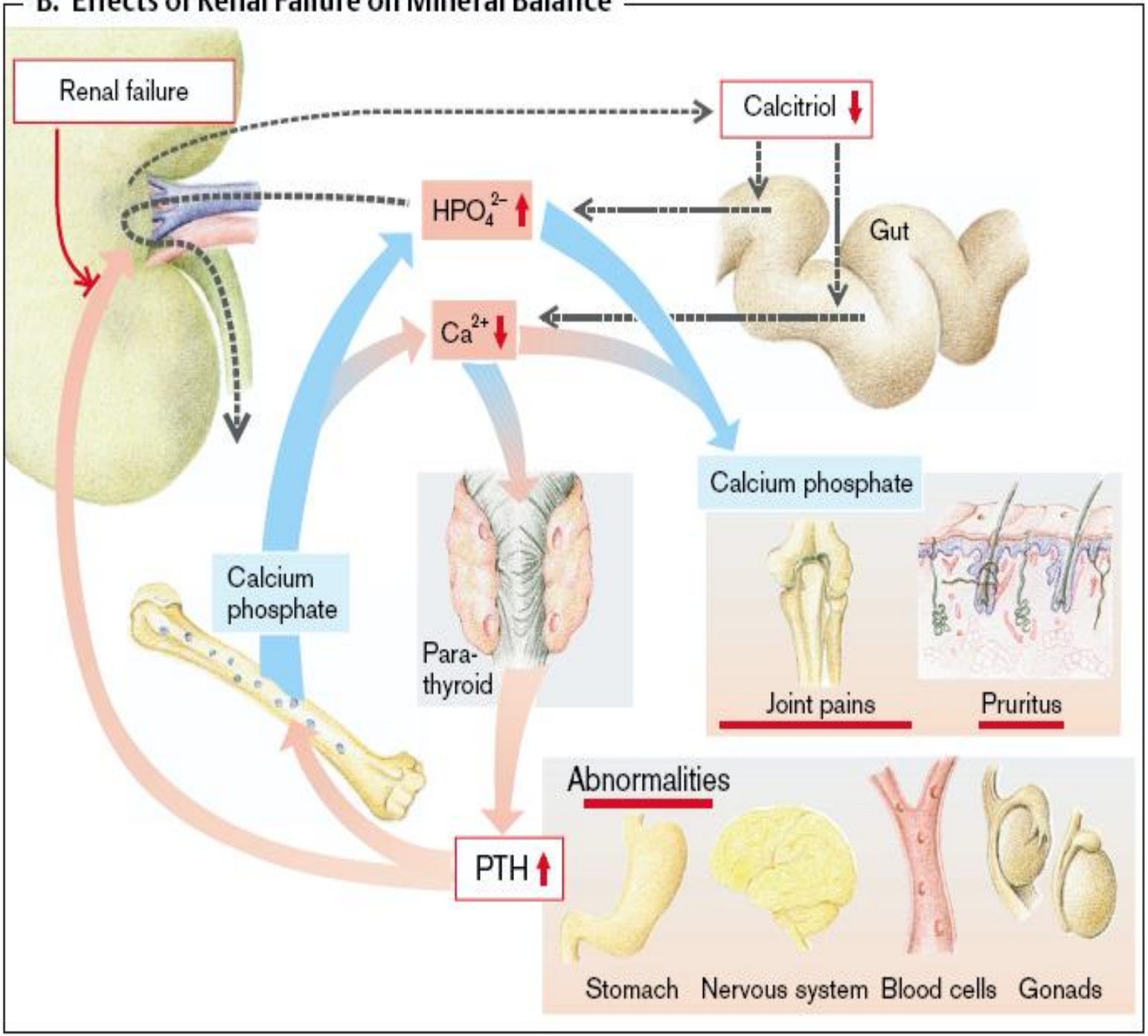
The microscopic appearance of the "end stage kidney" is similar regardless of cause, which is why a biopsy in a patient with chronic renal failure yields little useful information. The cortex is fibrotic, the glomeruli are sclerotic, there are scattered chronic inflammatory cell infiltrates, and the arteries are thickened. Tubules are often dilated and filled with pink casts and give an appearance of "thyroidization."

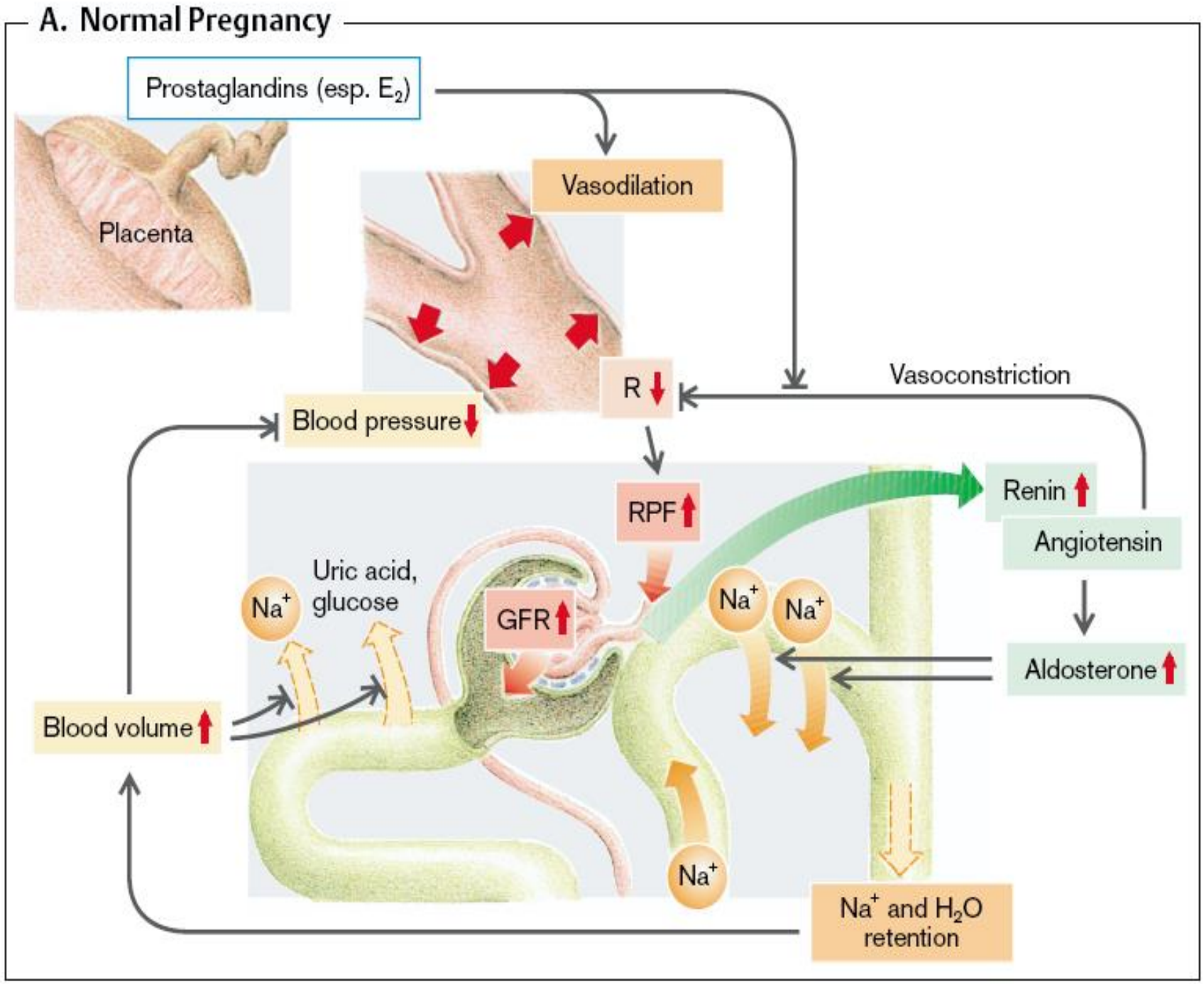


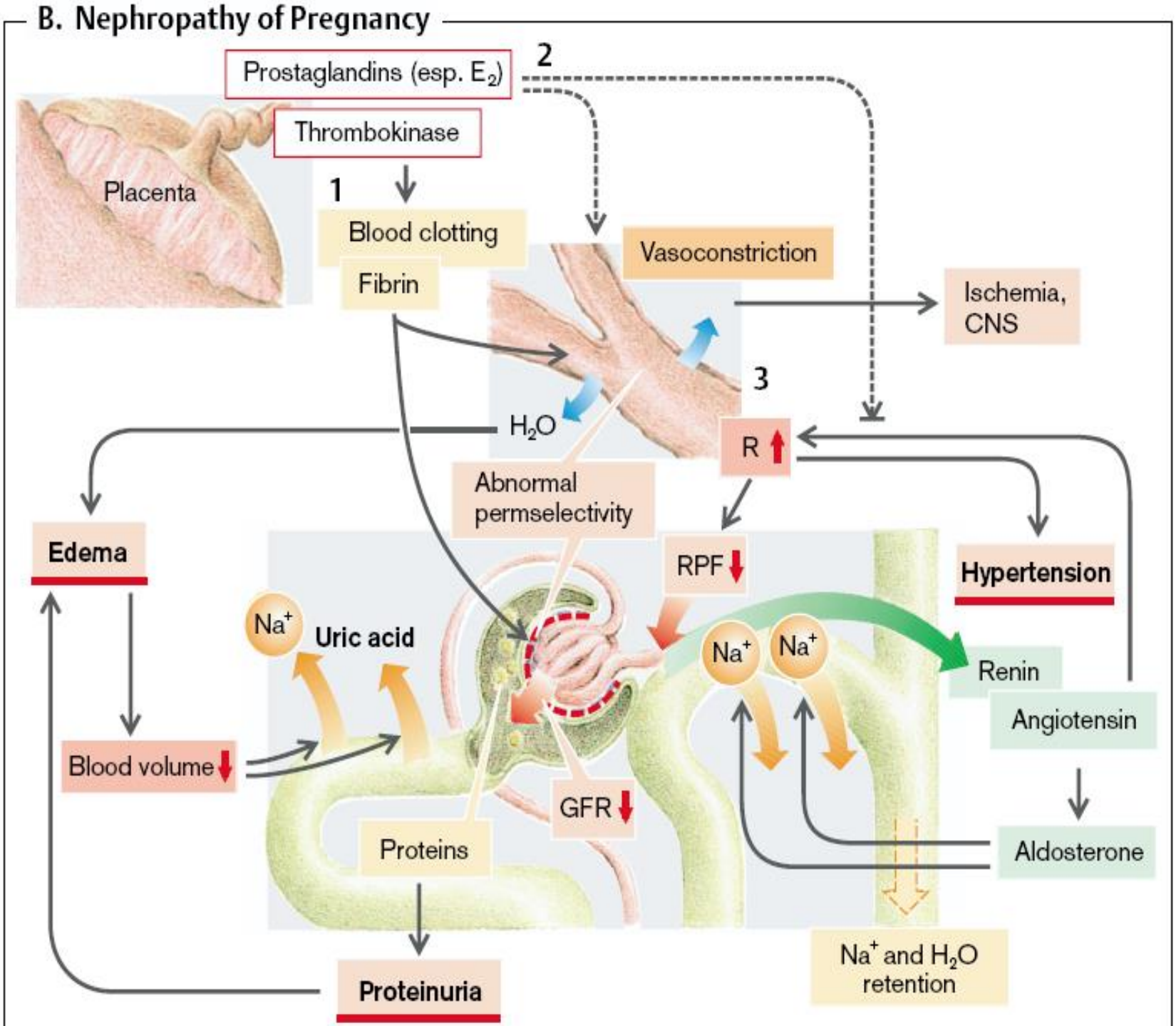
A. Disorders of Salt and Water Balance in Renal Failure



B. Effects of Renal Failure on Mineral Balance




A. Normal Pregnancy
Prostaglandins (esp. E_2)
Placenta
Vasodilation
R
Vasoconstriction
Blood pressure
RPF
Renin
Angiotensin
Uric acid, glucose
Na^+
GFR
Na^+
Na^+
Aldosterone
Blood volume
Na^+
Na^+ and H_2O retention


B. Nephropathy of Pregnancy
Prostaglandins (esp. E_2)
2
Thrombokinase
Placenta
1
Blood clotting
Fibrin
Vasoconstriction
Ischemia, CNS
3
H_2O
R
Abnormal permselectivity
Edema
RPF
Hypertension
Na^+
Uric acid
Na^+
Na^+
Renin
Angiotensin
Blood volume
GFR
Proteins
Aldosterone
Proteinuria
Na^+ and H_2O retention

IX. Neoplasms

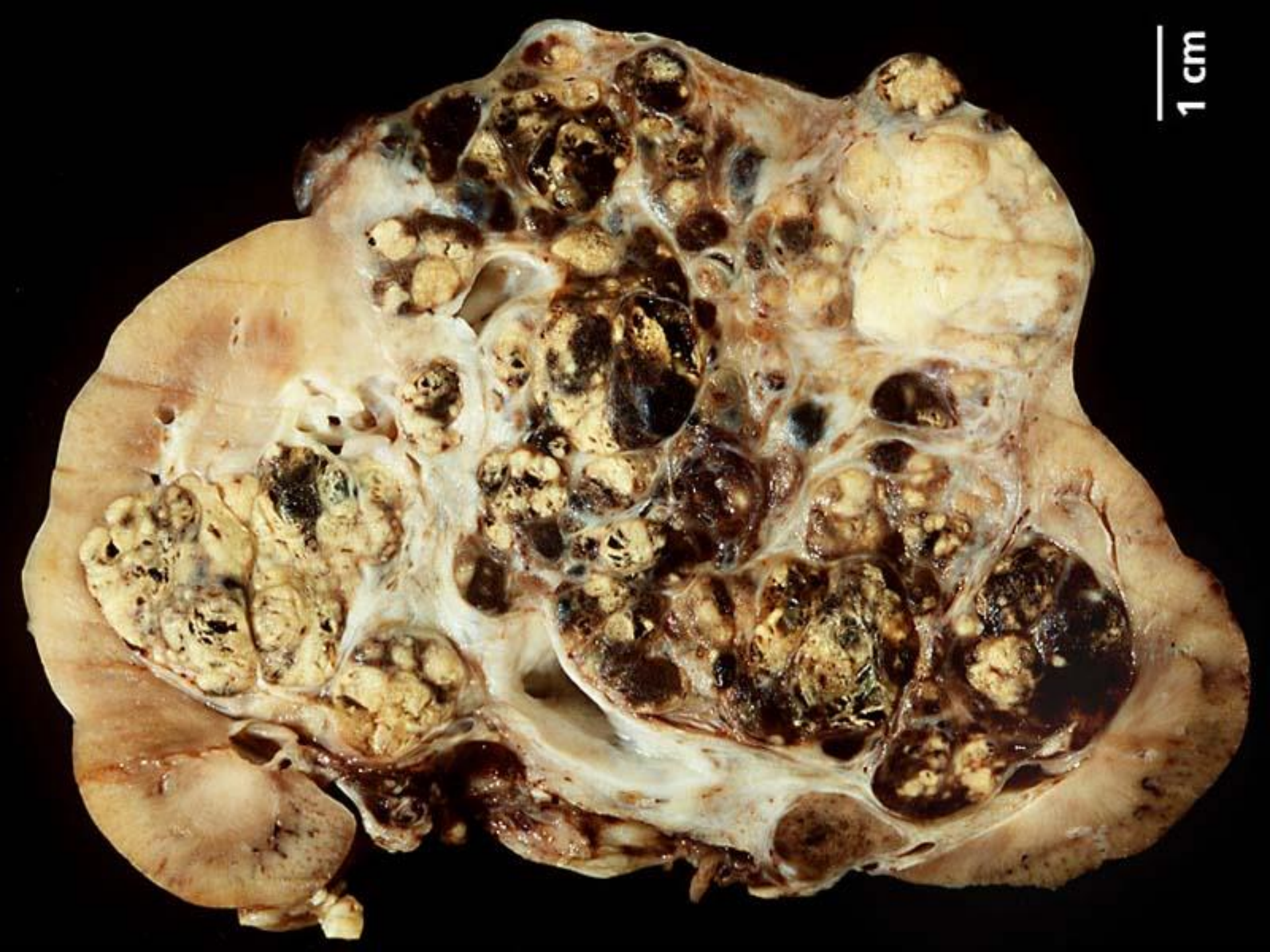
- Renal cell carcinoma (RCC)
  - The most frequent renal cancer in adult
  - It may have endocrine-like effect → procuce polycytemia, hypercalcemia, hypertension (renin) Cushing's syndrome, etc.
- Wilms' tumor

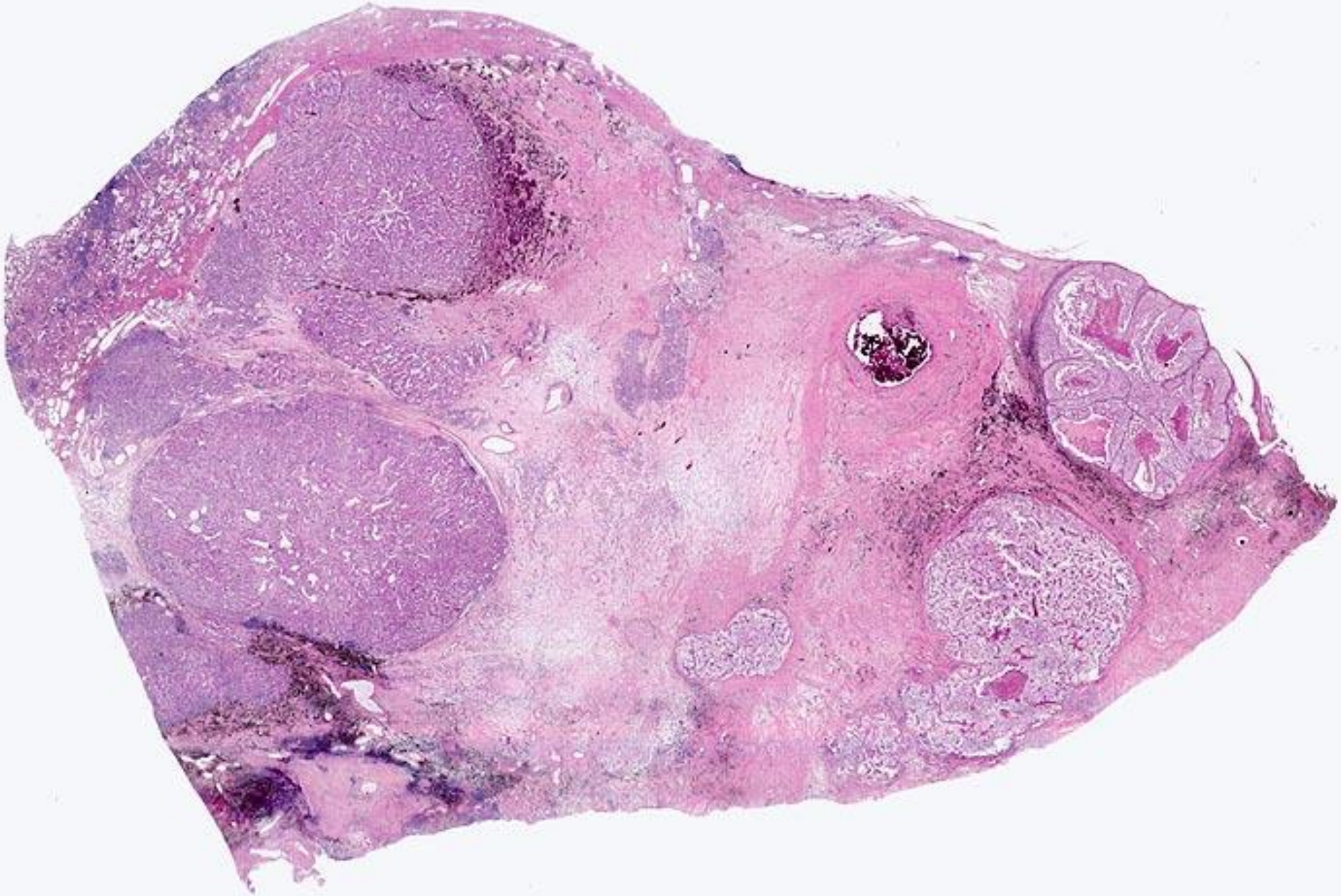
  - More common tumor in childhood
- Tansitional cell carcinoma

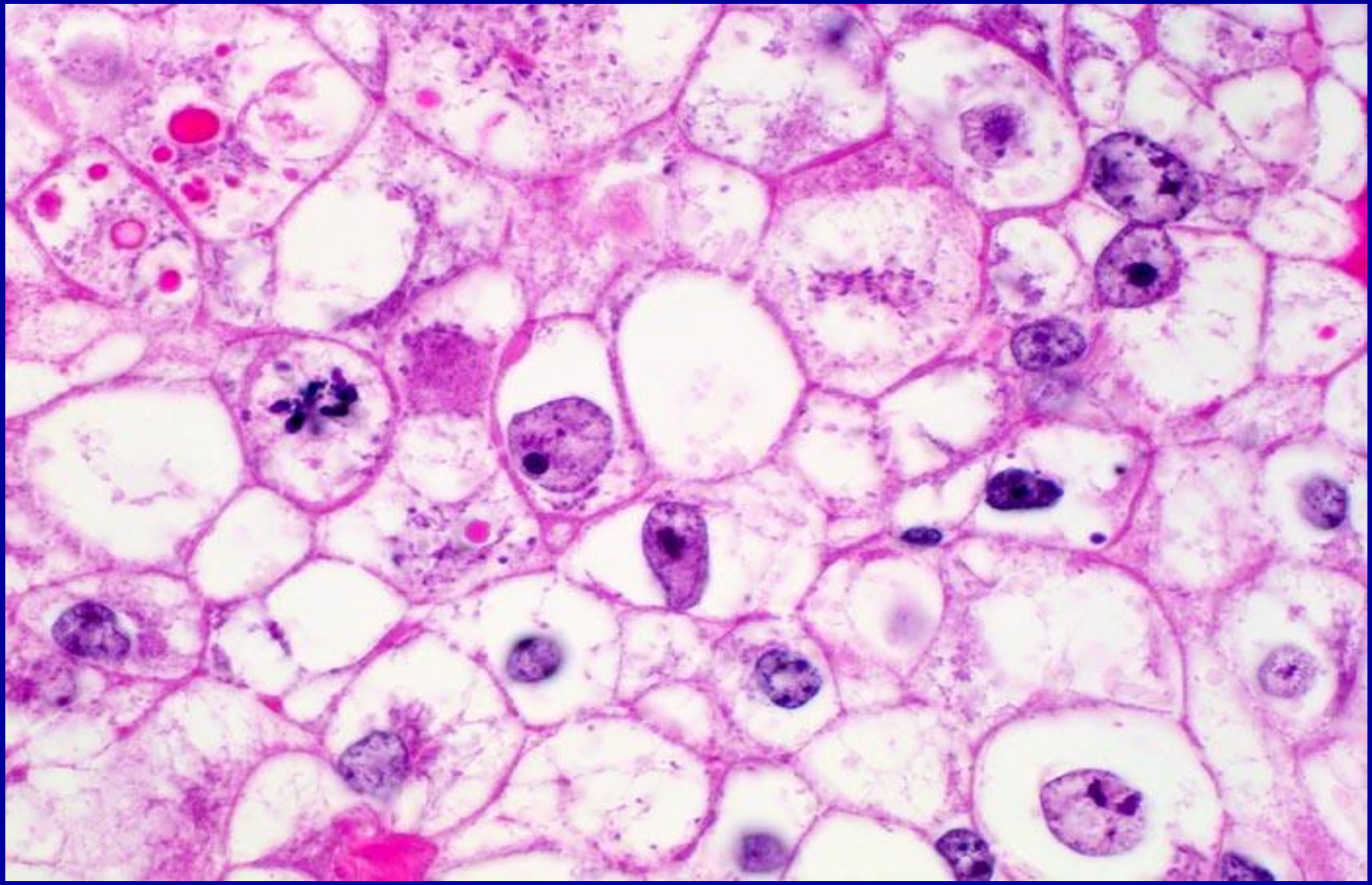
  - 5-10% of adult cancer

Renal cell carcinoma

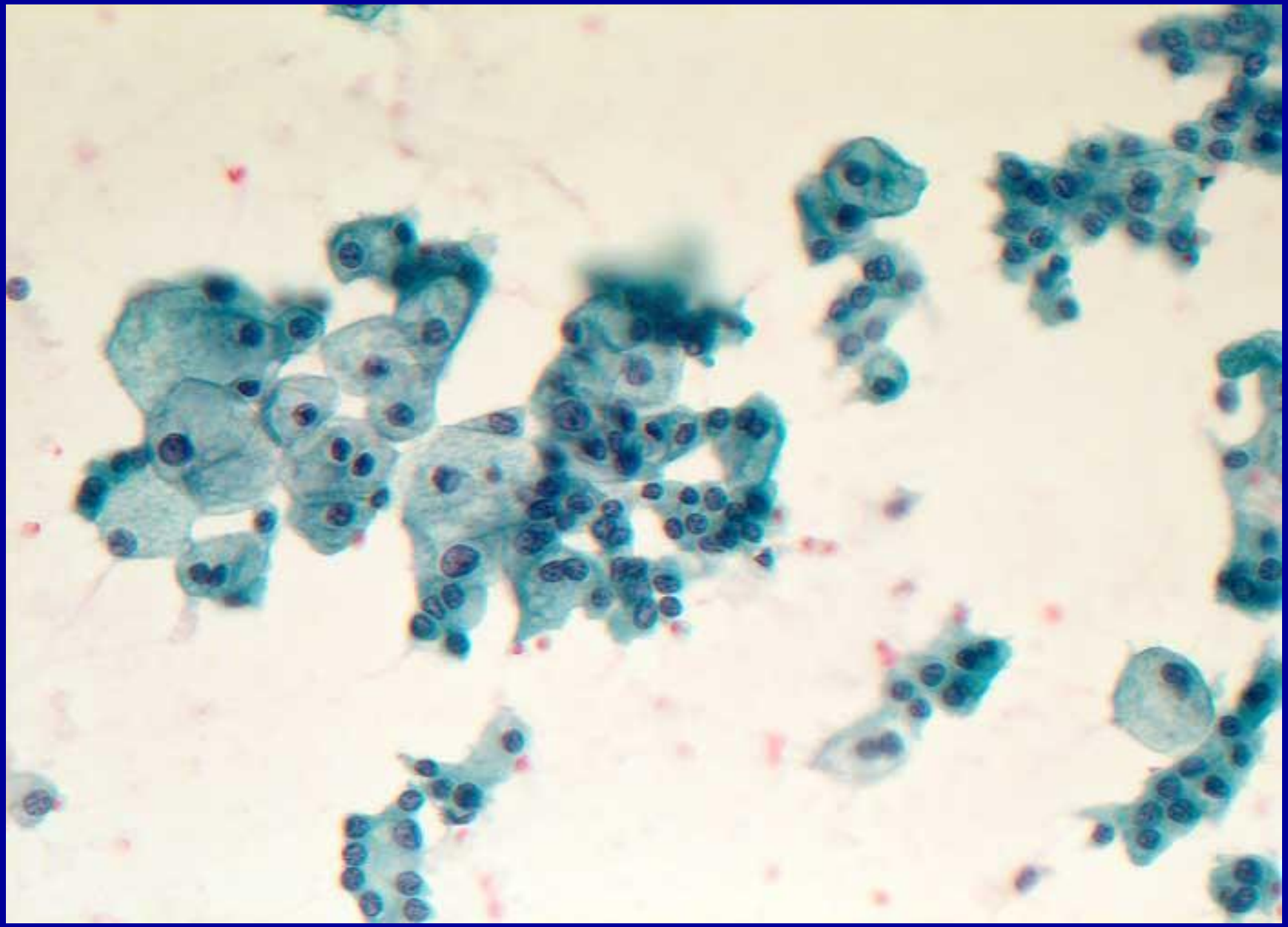
- Typical cross-section of yellowish, spherical neoplasm in one pole of the kidney
- Note the tumor in the dilated, thrombosed renal vain


1 cm






1 cm

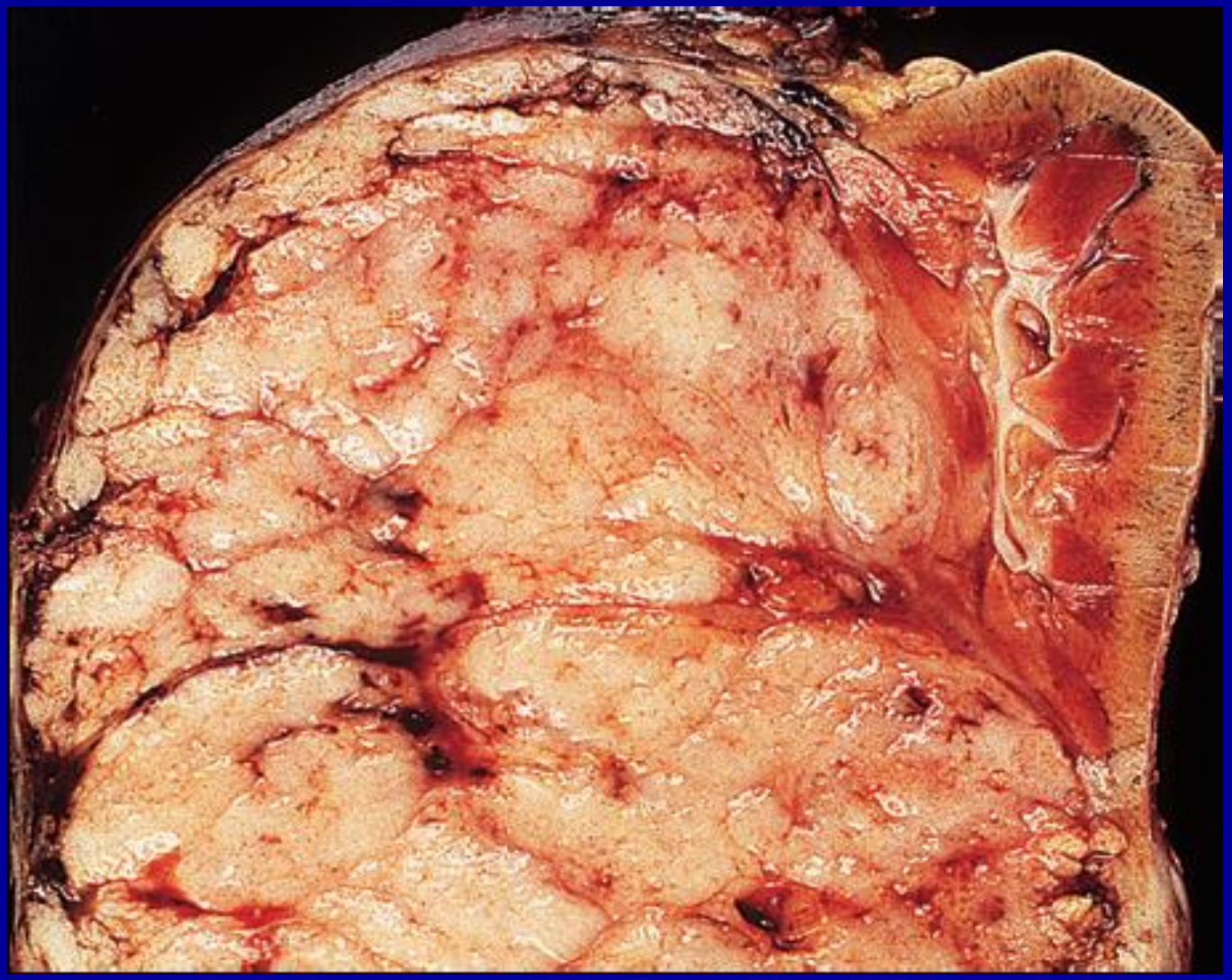



1 cm

TUMOR WILMS (NEPHRO BLASTOMA)

- Neoplasma ganas ini termasuk tumor embrional, yang mengandung bermacam komponen sel dan jaringan, semua berasal dari mesoderm.
- Nama lainnya adalah adenomyosarcoma, embrional carcinoma, embryonal mixed tumor.

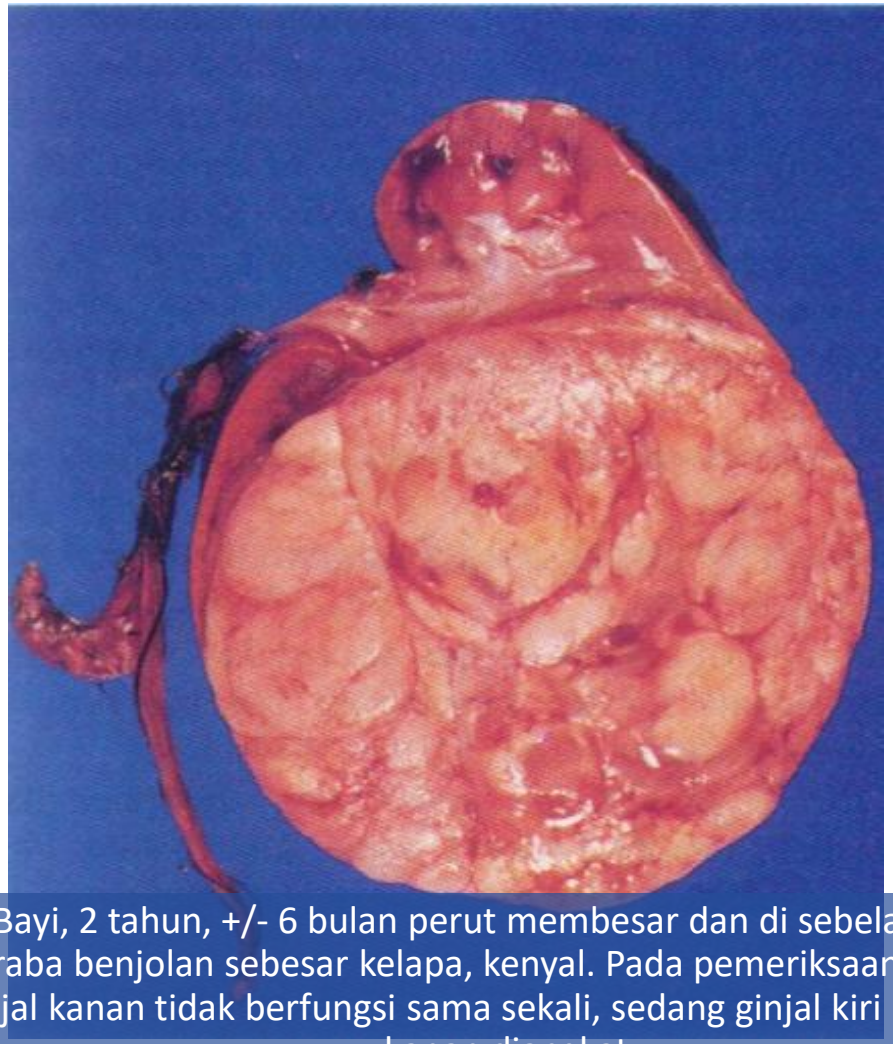
Wilms' tumor (kidney)



TUMOR WILMS (NEPHRO BLASTOMA)

Makroskopis:

Tampak tumor menyatu dengan ginjal, warna abu-abu lunak meluas dari cortex ke medulla, banyak didapat daerah perdarahan dan nekrose

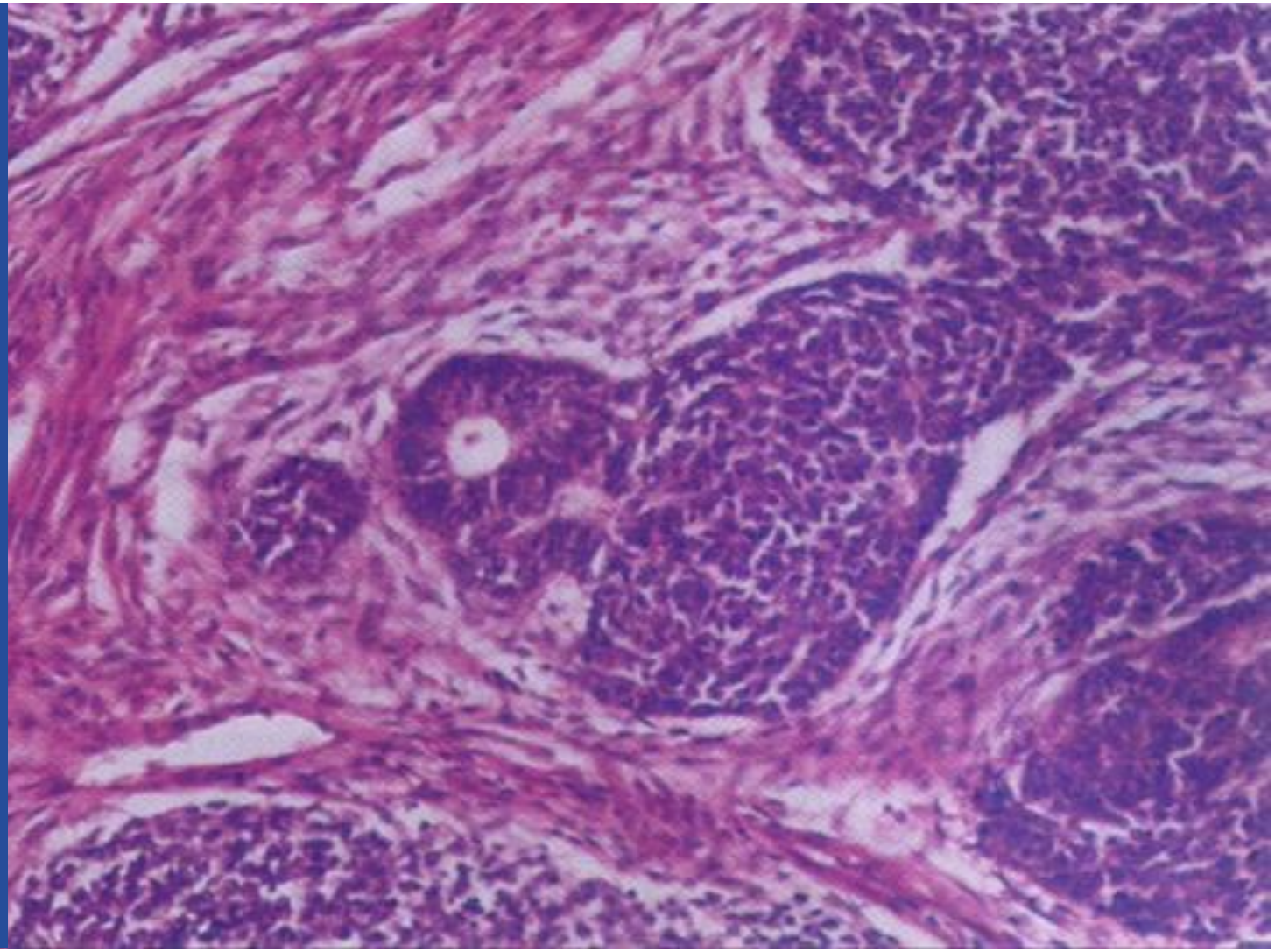


Bayi, 2 tahun, +/- 6 bulan perut membesar dan di sebelah kanan teraba benjolan sebesar kelapa, kenyal. Pada pemeriksaan pielografi ginjal kanan tidak berfungsi sama sekali, sedang ginjal kiri baik. Ginjal kanan diangkat.

TUMOR WILMS (NEPHRO BLASTOMA)

Perbesaran lemah
Terlihat gambaran tumor yang terdiri atas 2 macam elemen:

- bagian sarcomatous, nampak sebagian jaringan ikat dengan sel-sel atipi umumnya spindel & bulat
- bagian epitelial dengan bagian-bagian tubuler dan bagian yang padat/solid dengan sel-sel atipi, polimorfi dan mitosis banyak



TUMOR WILMS (NEPHRO BLASTOMA)

Perbesaran lemah

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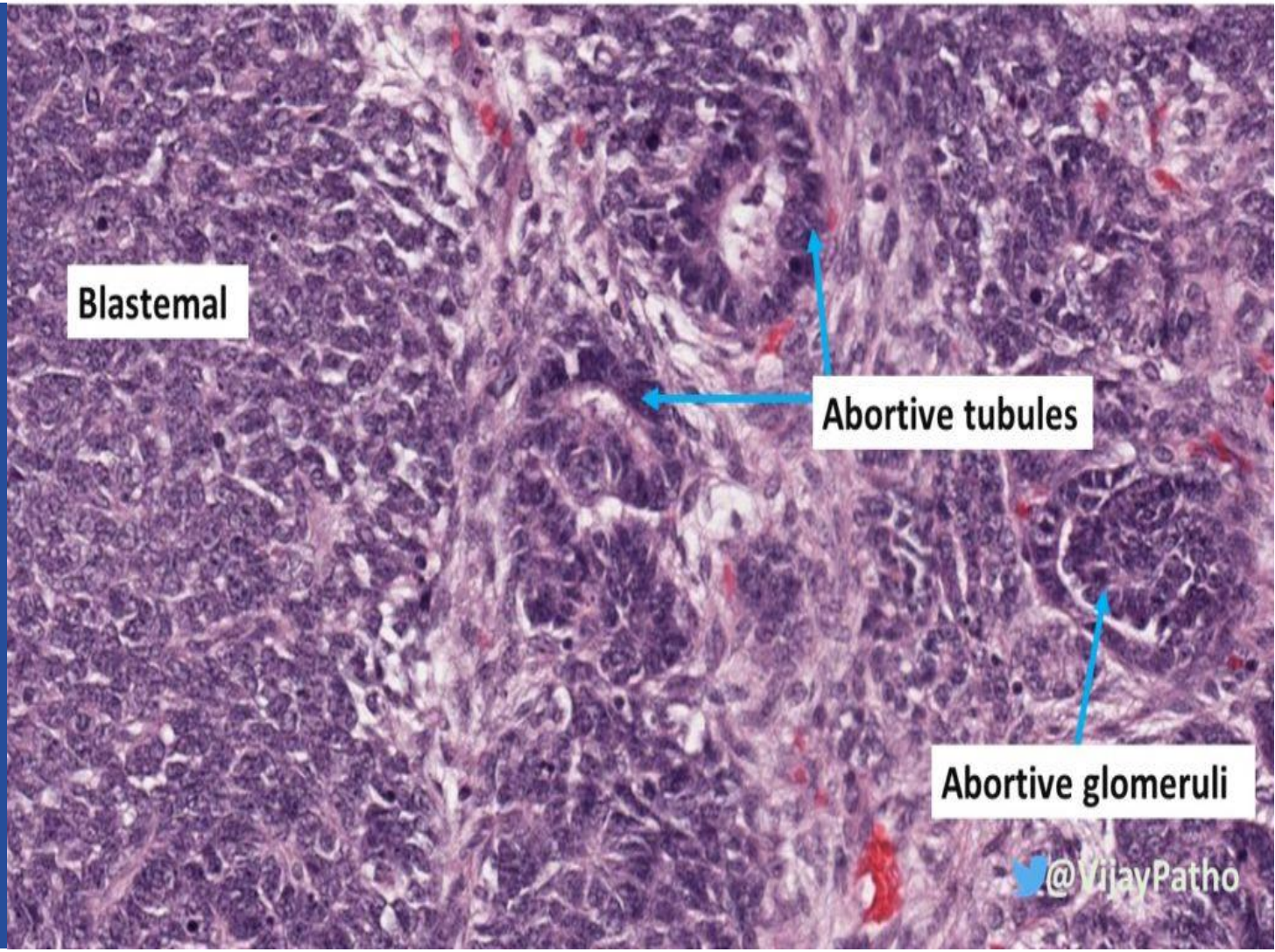
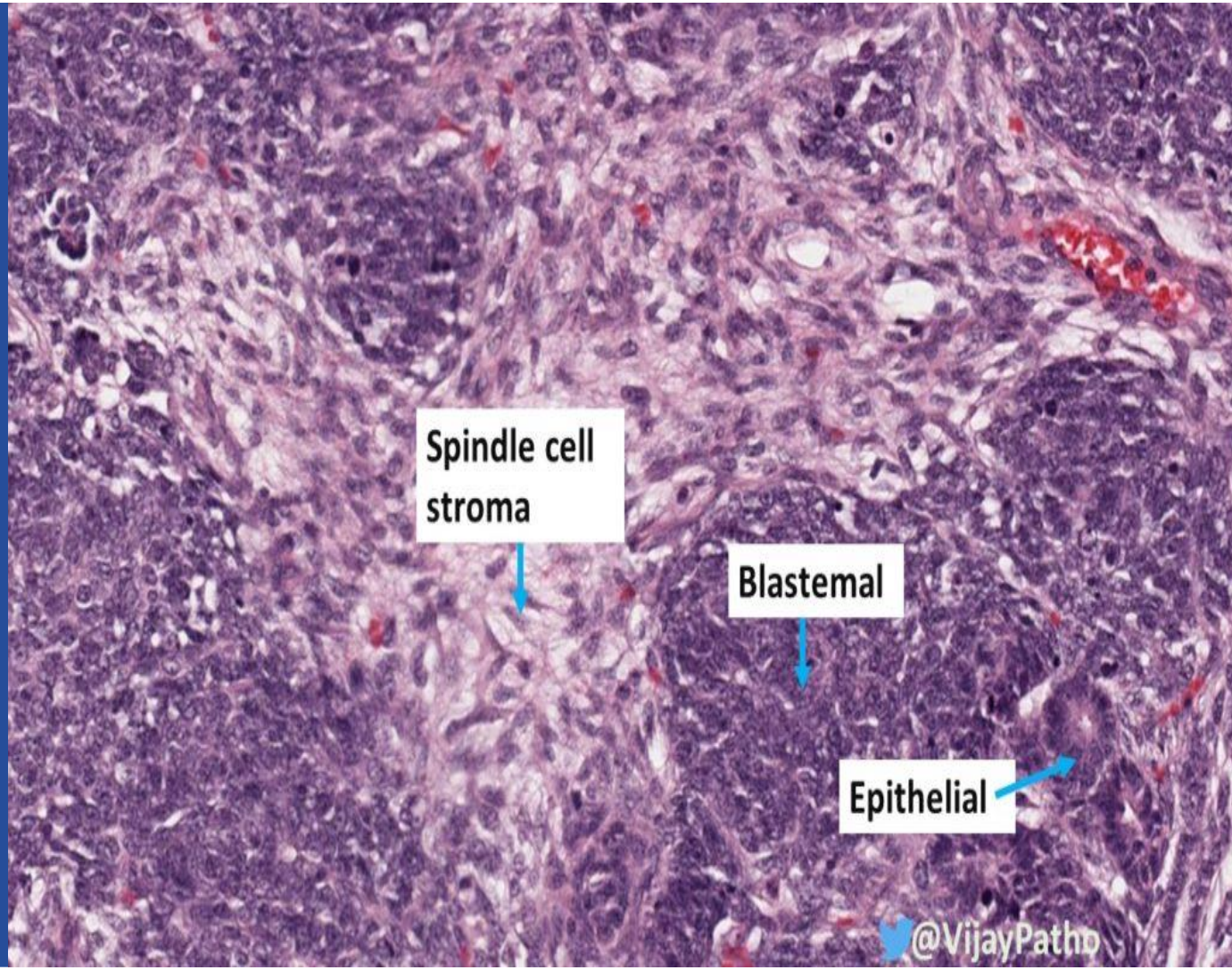


TUMOR WILMS (NEPHRO BLASTOMA)

Perbesaran kuat

- Suatu jaringan sarkoma yang telah mengalami deferensiasi dan sel-sel terdiri atas sel-sel yang spindel & bulat dan pada bagian ini ditemukan:
- banyak pembuluh darah dan bagian-bagian perdarahan
- mitosis banyak ditemukan.
- Bagian-bagian yang degenerasi sampai nekrotis



Wilms' tumor (kidney)