

The background features a dark blue gradient with a starry space pattern. Overlaid on this are several technical diagrams in a lighter blue color. These include circular gauges with numerical scales (e.g., 140, 150, 160, 170, 180, 190, 200, 210, 220, 230, 240, 250, 260), concentric circles, and curved arrows indicating motion or flow. The diagrams are semi-transparent and scattered across the left and top portions of the frame.

RENAL BLOCK

HALA KFOURY, MD

OBJECTIVES

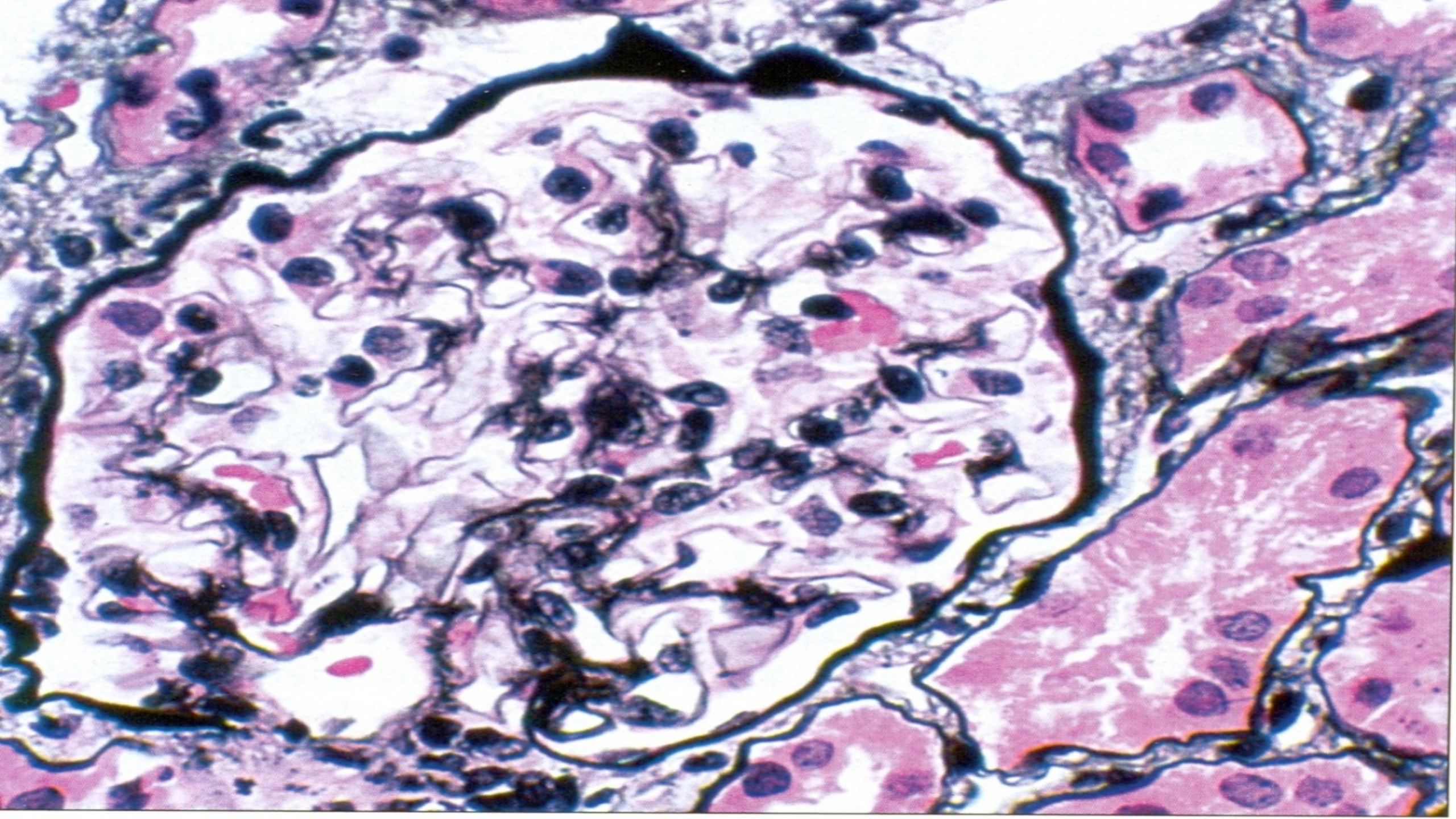
- Understand the relationship between the anatomical structures of different components of the Renal system and their functions.
- Discuss the pathology, microbiology, pathogenesis, and factors contributing to the development of most common diseases affecting the Renal system.
- Use basic sciences to explain patient's signs and symptoms, interpret investigation results, and provide justifications for their views.

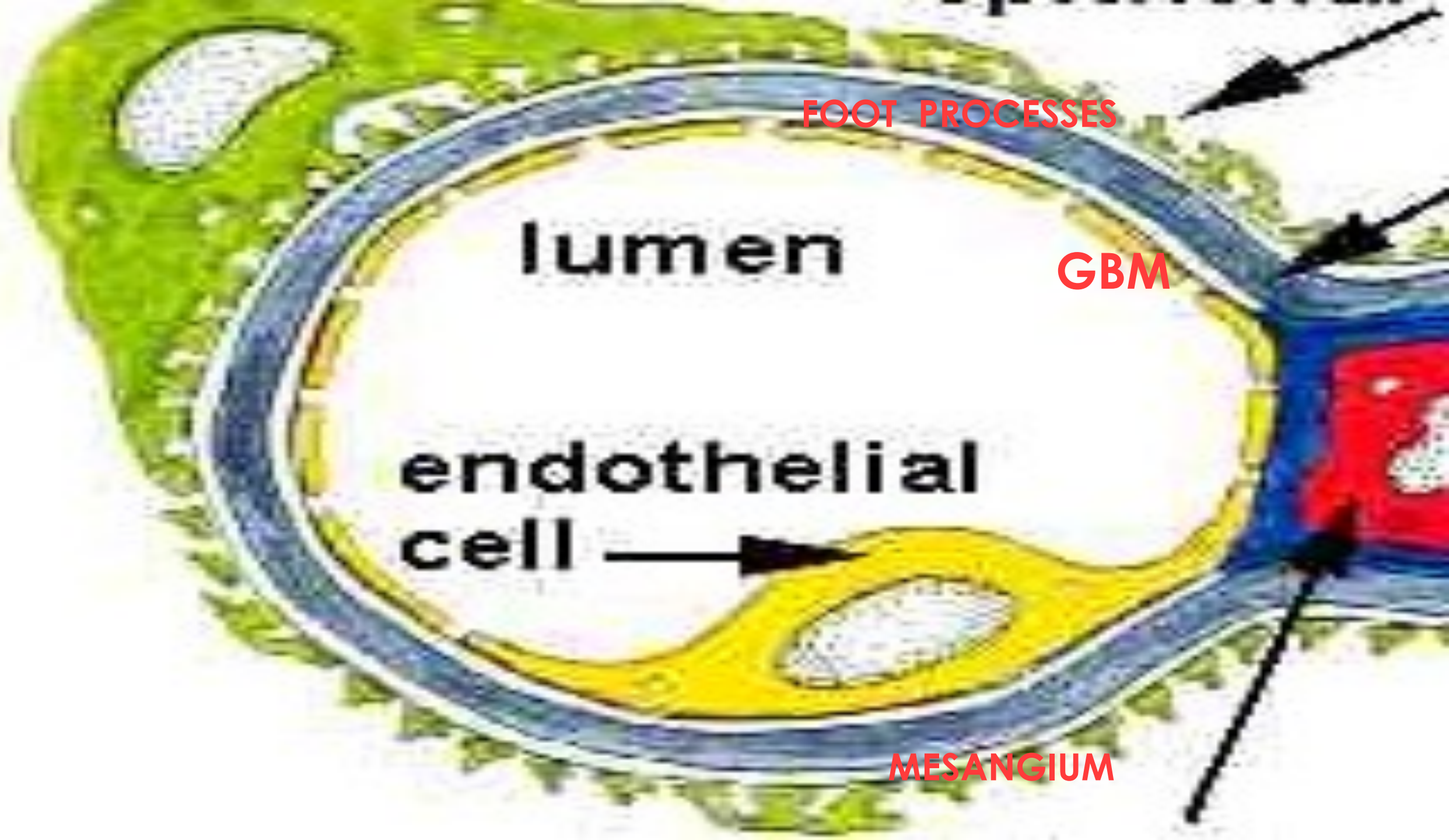
OBJECTIVES

- Develop communication skills and explore psychosocial, and ethical issues in their assessment.
- Use clinical cases to apply knowledge learnt, generate hypotheses, build an enquiry plan, and use evidence to refine their hypotheses, and justify their views.
- Design a management plan, and understand the pharmacological basis of drugs used in the management of common diseases affecting the Renal system.

ACUTE KIDNEY INJURY OBJECTIVES

- Introduction to the renal pathology
- Acute Kidney Injury
- Definition, Types, Clinical Overview, Causes
- Pathological findings
- Differential Diagnosis





FOOT PROCESSES

lumen

GBM

endothelial
cell

MESANGIUM



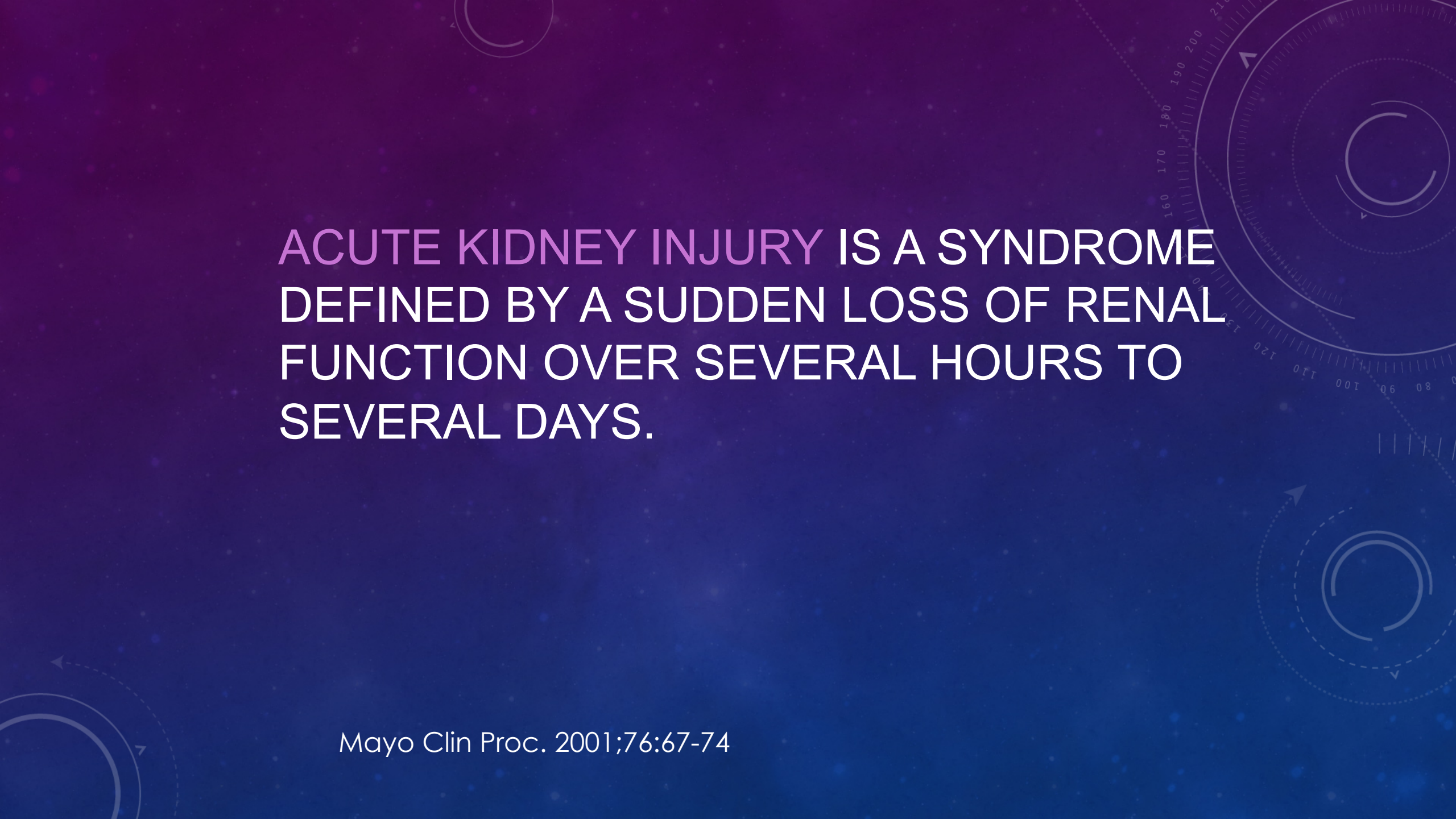
Pc

Ep

End

Ep

L

The background features a dark blue gradient with a starry space pattern. Overlaid on this are several technical diagrams, including circular gauges with numerical scales (e.g., 160, 170, 180, 190, 200, 210) and arrows, and concentric circles with dashed lines and arrows, suggesting a scientific or medical context.

**ACUTE KIDNEY INJURY IS A SYNDROME
DEFINED BY A SUDDEN LOSS OF RENAL
FUNCTION OVER SEVERAL HOURS TO
SEVERAL DAYS.**

Mayo Clin Proc. 2001;76:67-74

WHAT CONSTITUTES THE SYNDROME OF ARF?

- Accumulation of nitrogenous waste products.
- Increased Scr.
- Derangement of extracellular fluid balance.
- Acid-base disturbance.
- Electrolyte and mineral disorders.

WHAT CONSTITUTES UREMIA?

- Renal failure
- Lethargy
- Anorexia
- Dysgeusia
- Pericarditis
- Neuropathy
- Nausea and vomiting
- Pruritis
- Dyspnea

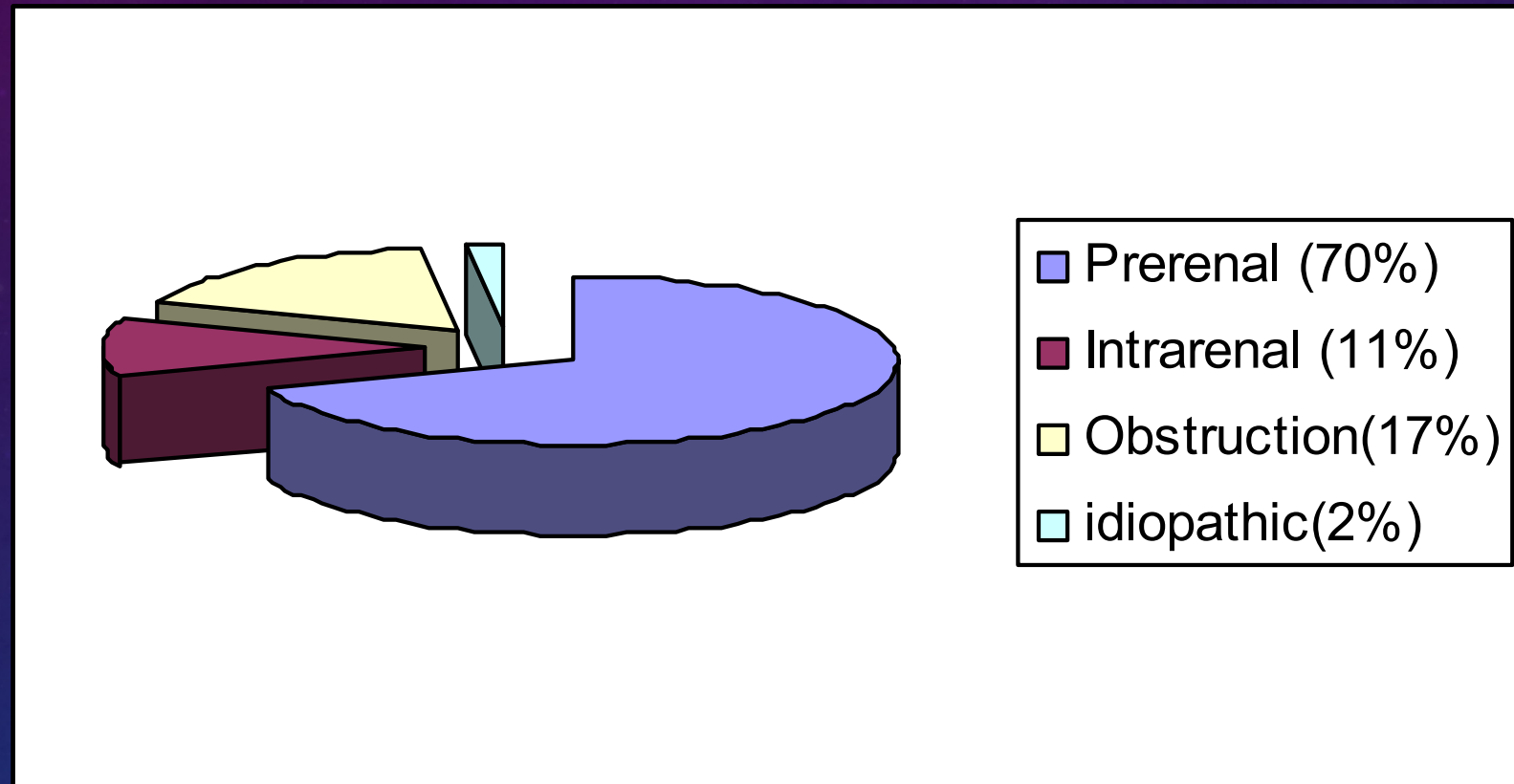
Azotemia: elevated blood urea nitrogen not from an intrinsic renal disease

Oliguria: urine output less than 500cc/24hr.

Nonoliguria: urine output greater than 500cc/24hr.

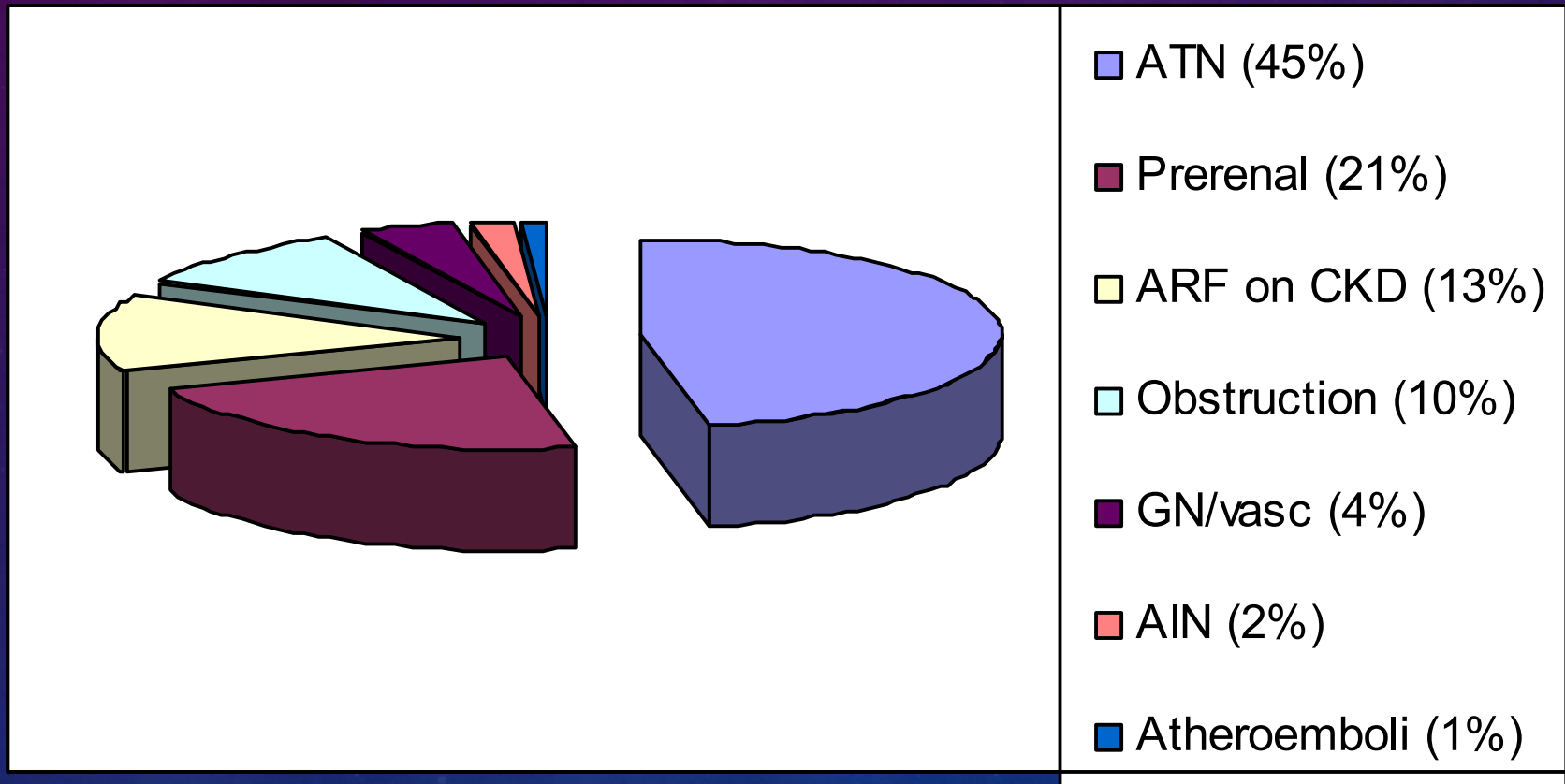
Anuria: urine output less than 50cc/24hr.

ETIOLOGY OF ARF AMONG OUTPATIENTS

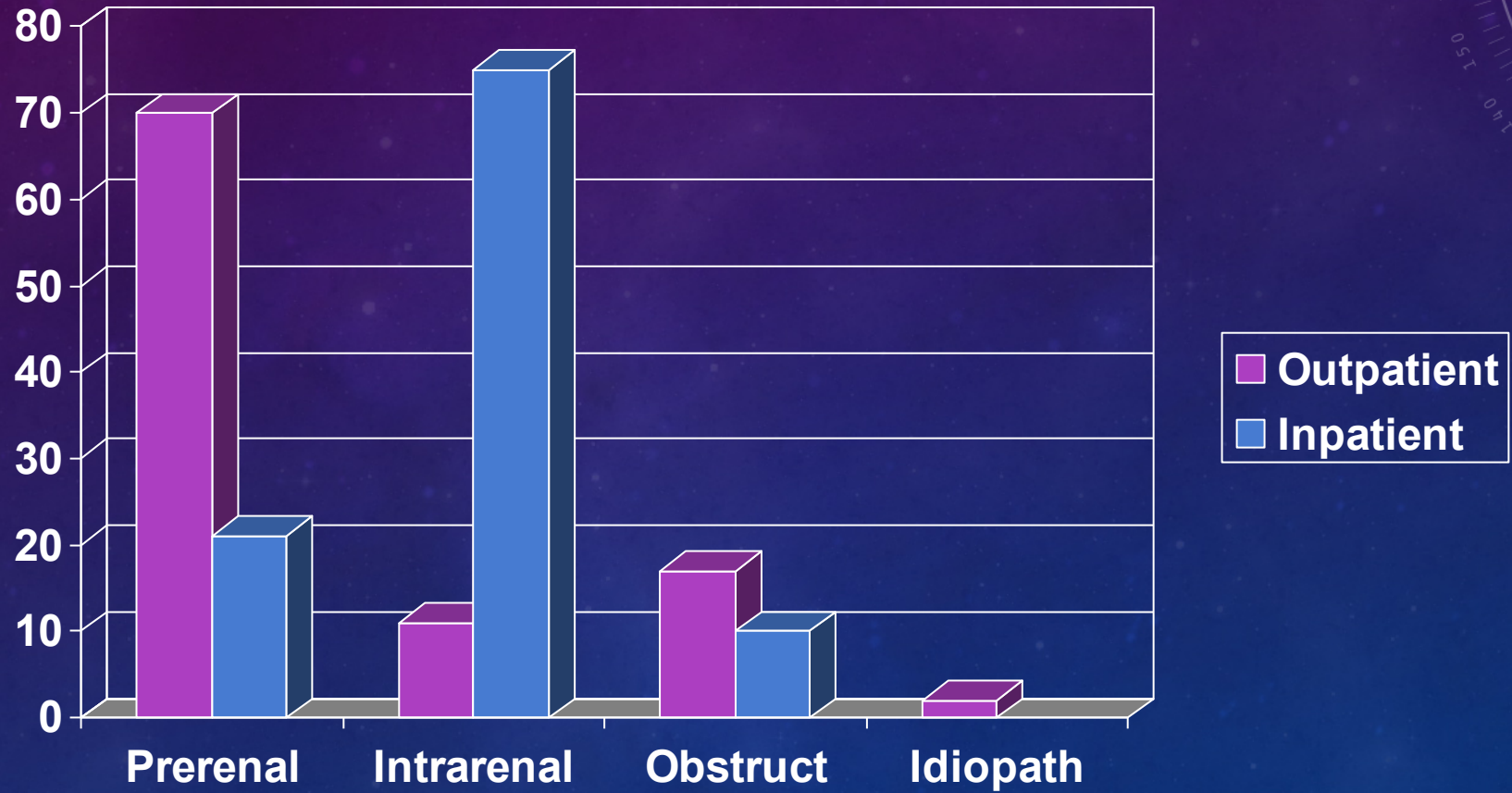


AJKD 17:191-198, 1991

ETIOLOGY OF ARF AMONG INPATIENTS



ETIOLOGY OF ARF



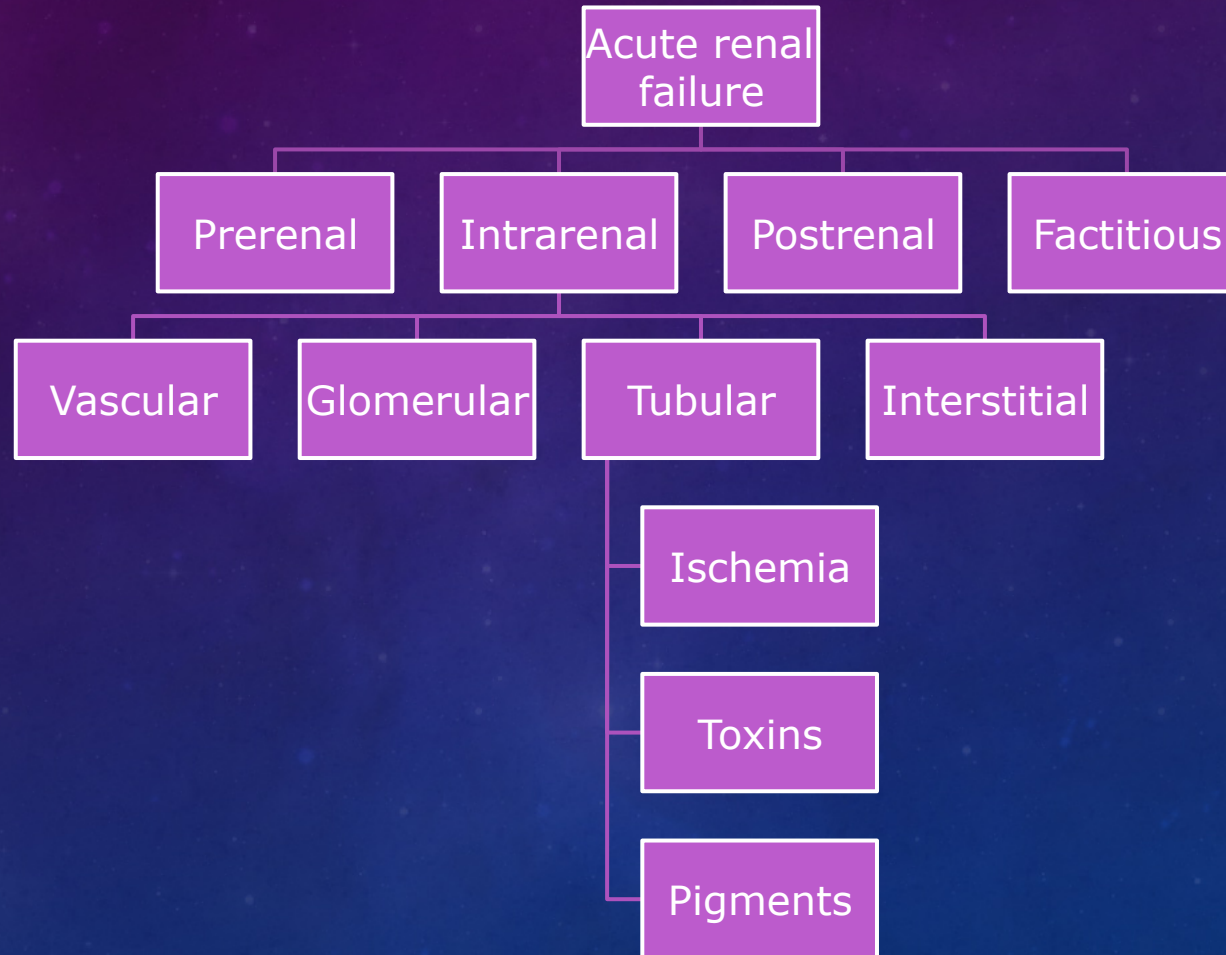
MORTALITY OF ARF

- “Despite technical progress in the management of acute renal failure over the last 50 years, mortality rates seem to have remained unchanged at around 50%.”

PREDICTORS OF DIALYSIS IN ARF

- Oliguria:
 - <400cc/24hr 85% will require dialysis
 - >400cc/24hr 30-40% will require dialysis
- Mechanical ventilation
- Acute myocardial infarction
- Arrhythmia
- Hypoalbuminemia
- ICU stay
- Multi-system organ failure

THE PATHOPHYSIOLOGY OF ARF



PRERENAL ARF (DECREASED RENAL BLOOD FLOW)

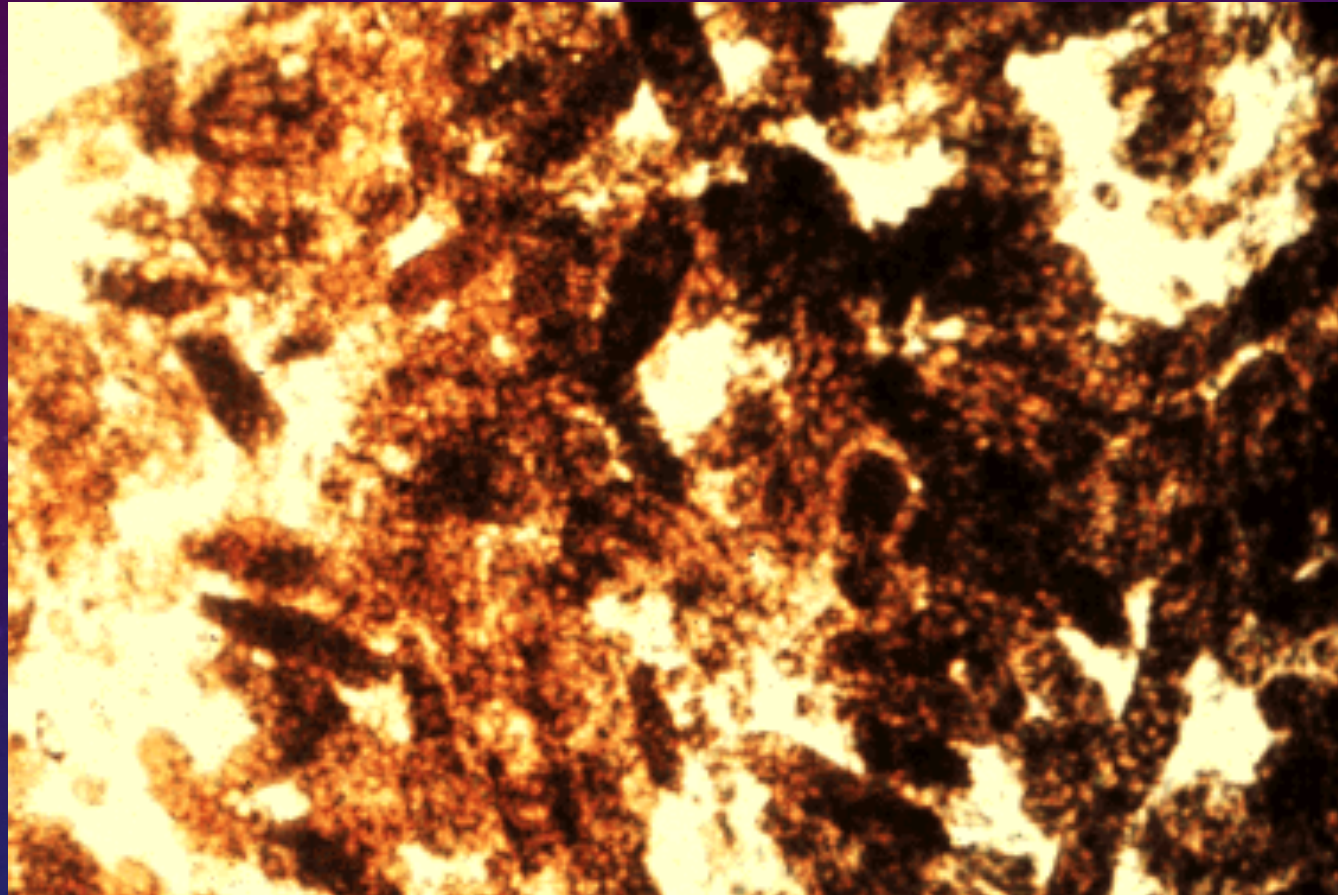
- Hypotension
 - Sepsis, cardiogenic, medication
- Cardiogenic
- Vascular
 - Vasculitis, renal artery compromise, AAA, atheroemboli
- Third Spacing
 - Bowel obstruction, cirrhosis, nephrotic syndrome, major surgery,
- Volume depletion
 - GI losses: vomiting, diarrhea
 - Skin losses: burns, sweat
 - Renal losses: DKA, DI, Addison's, Na wasting
- Drug-induced
 - NSAID, CsA, FK506, ACE, ARB

THERE IS A CONTINUUM FROM
PRERENAL PHYSIOLOGY TO
ISCHEMIC PATHOLOGY.



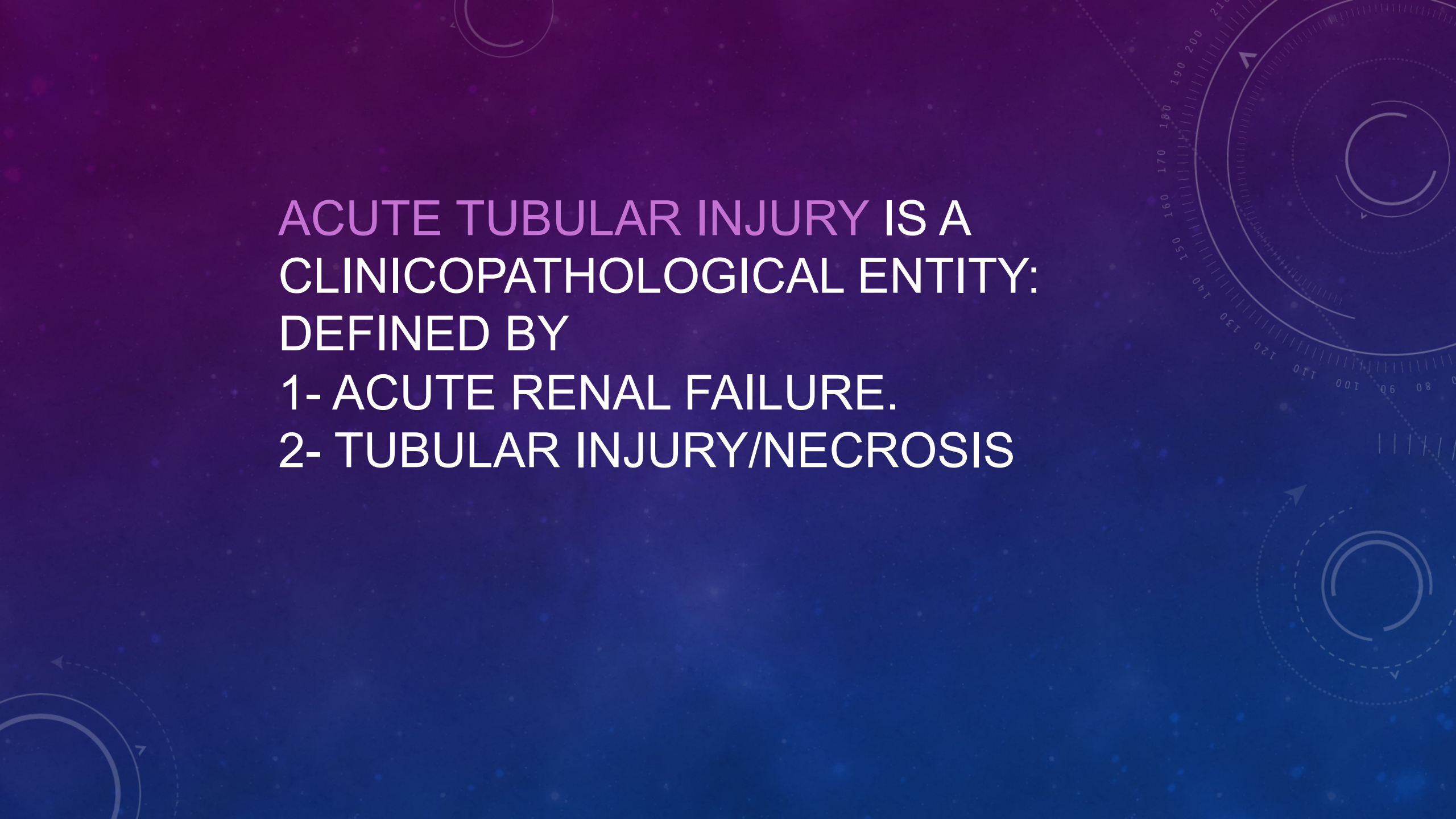
TUBULAR TOXINS

- Antimicrobials: aminoglycosides, vancomycin, foscarnet, pentamidine, amphotericin B
- Chemotherapeutics: cisplatin, mitomycin C, ifosfamide
- Immunotherapy: IVIG
- Complex Sugars: maltose, sucrose, mannitol
- Heavy metals
- Sepsis, hypoxia
- Radiocontrast agents



Sediment in ATN Urine sediment showing multiple, muddy brown granular casts. These findings are highly suggestive of acute tubular necrosis in a patient with acute renal failure. Courtesy of Harvard Medical School.

Uptodate Online 11.2, Rose BD, 2003



ACUTE TUBULAR INJURY IS A
CLINICOPATHOLOGICAL ENTITY:
DEFINED BY

- 1- ACUTE RENAL FAILURE.
- 2- TUBULAR INJURY/NECROSIS

ACUTE RENAL FAILURE

I. Acute tubular necrosis (ATN)

II. Ischemic

- 1. Shock***
- 2. Sepsis***
- 3. Incompatible blood transfusions***
- 4. thrombotic diseases***

ACUTE RENAL FAILURE

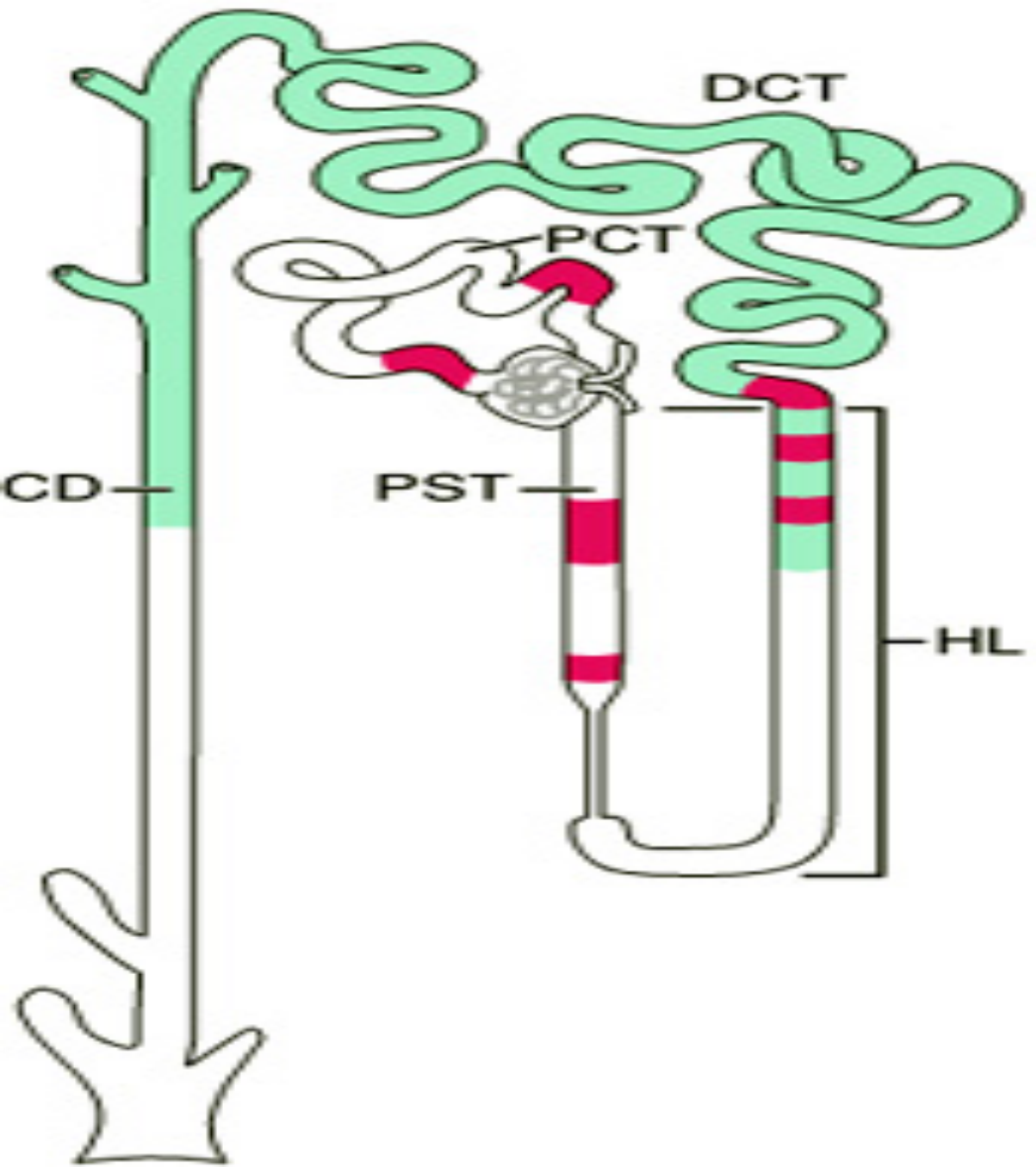
I. Acute tubular necrosis (ATN)

III. Toxic : A- Endogenous: Crush injury- Hemoglobinopathy.
B- Exogenous: Drugs- radiocontrast dye- metals..

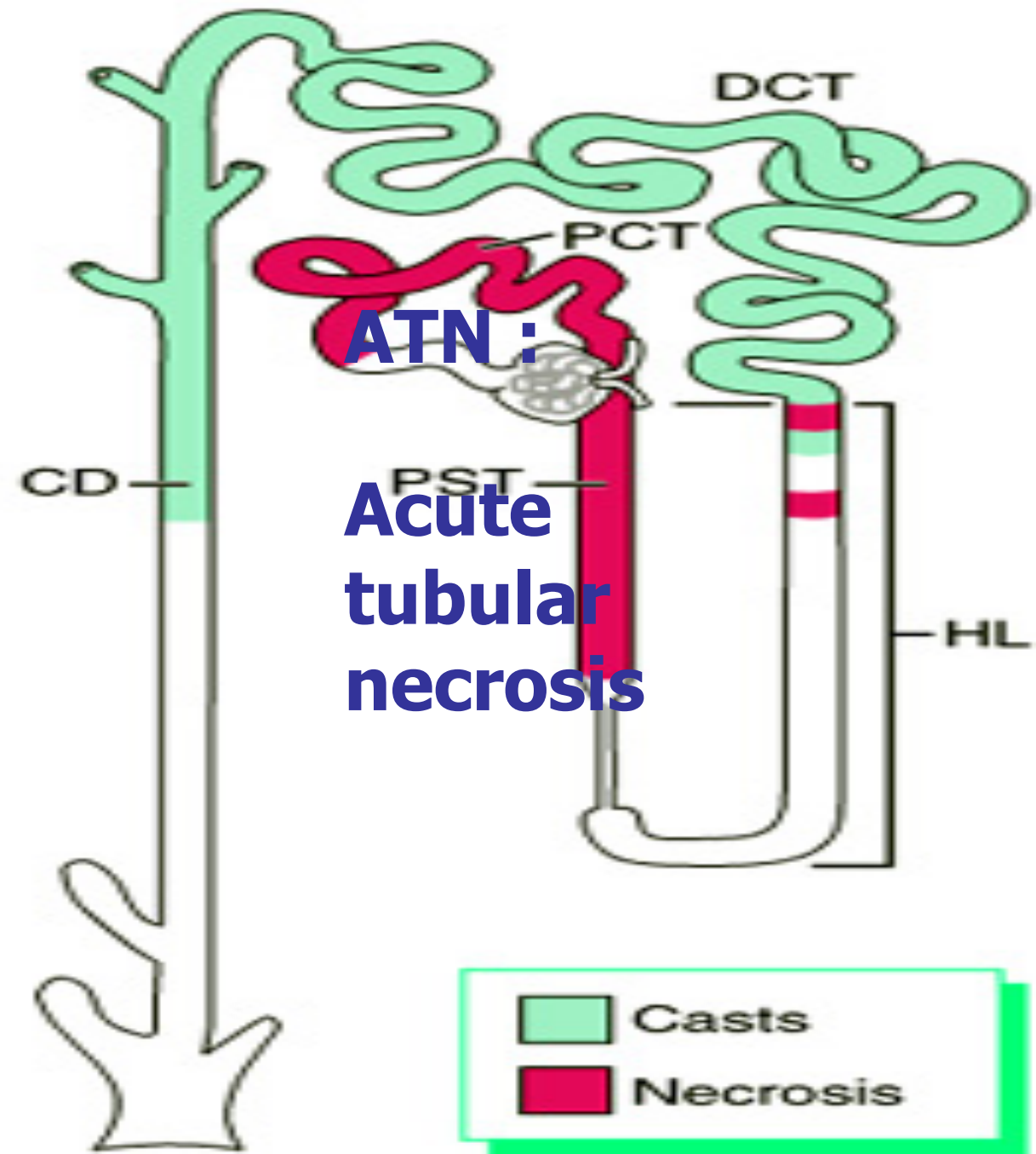
Acute tubular necrosis (ATN)

- ***Clinicopathological entity***
- ***Destruction of tubular epithelial cell***
- ***Clin. acute suppression of renal function
(no urine or below 400 ml/24h)***
- ***Most common cause of renal failure***

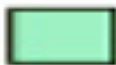
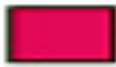
ISCHEMIC TYPE

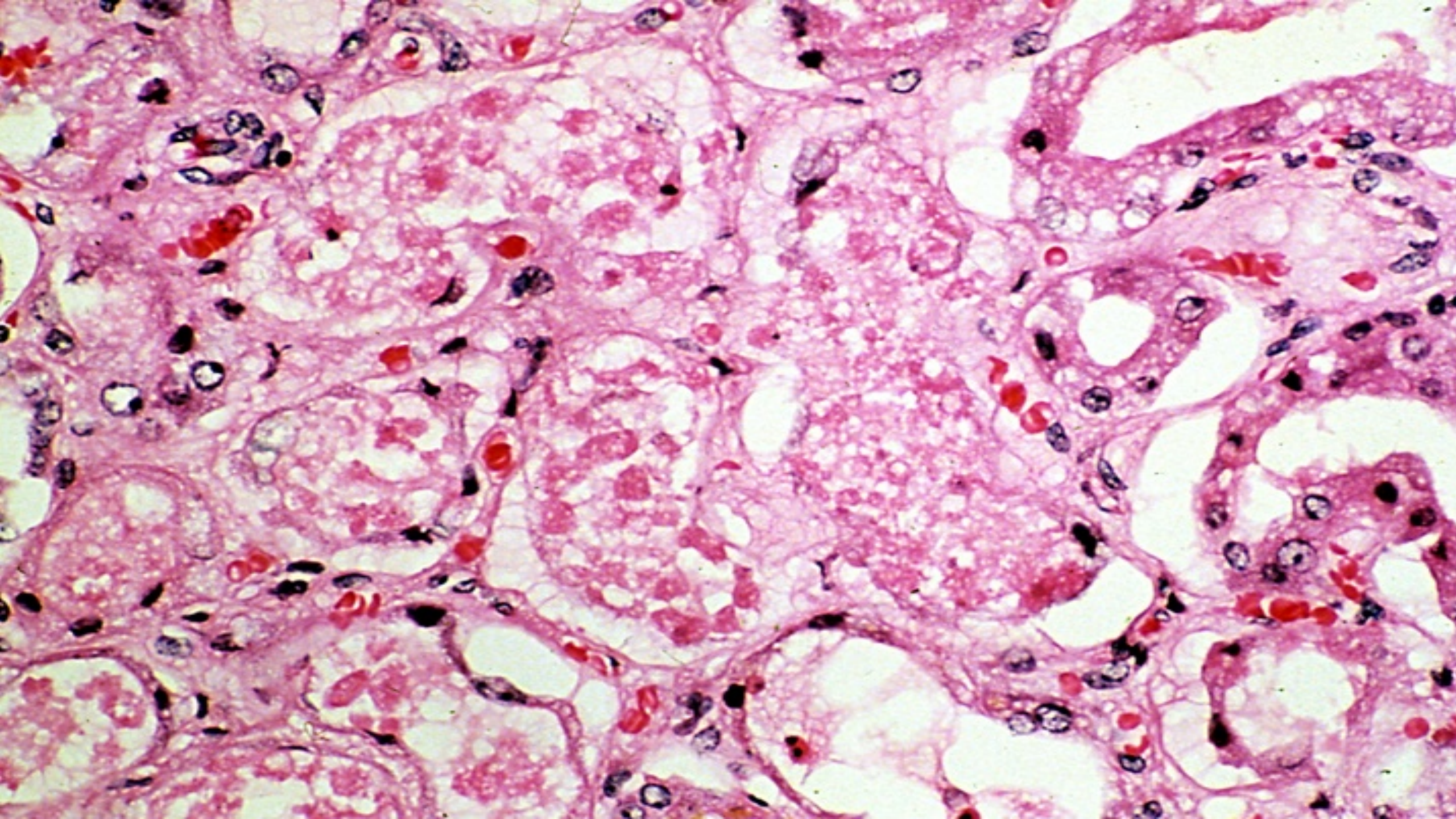


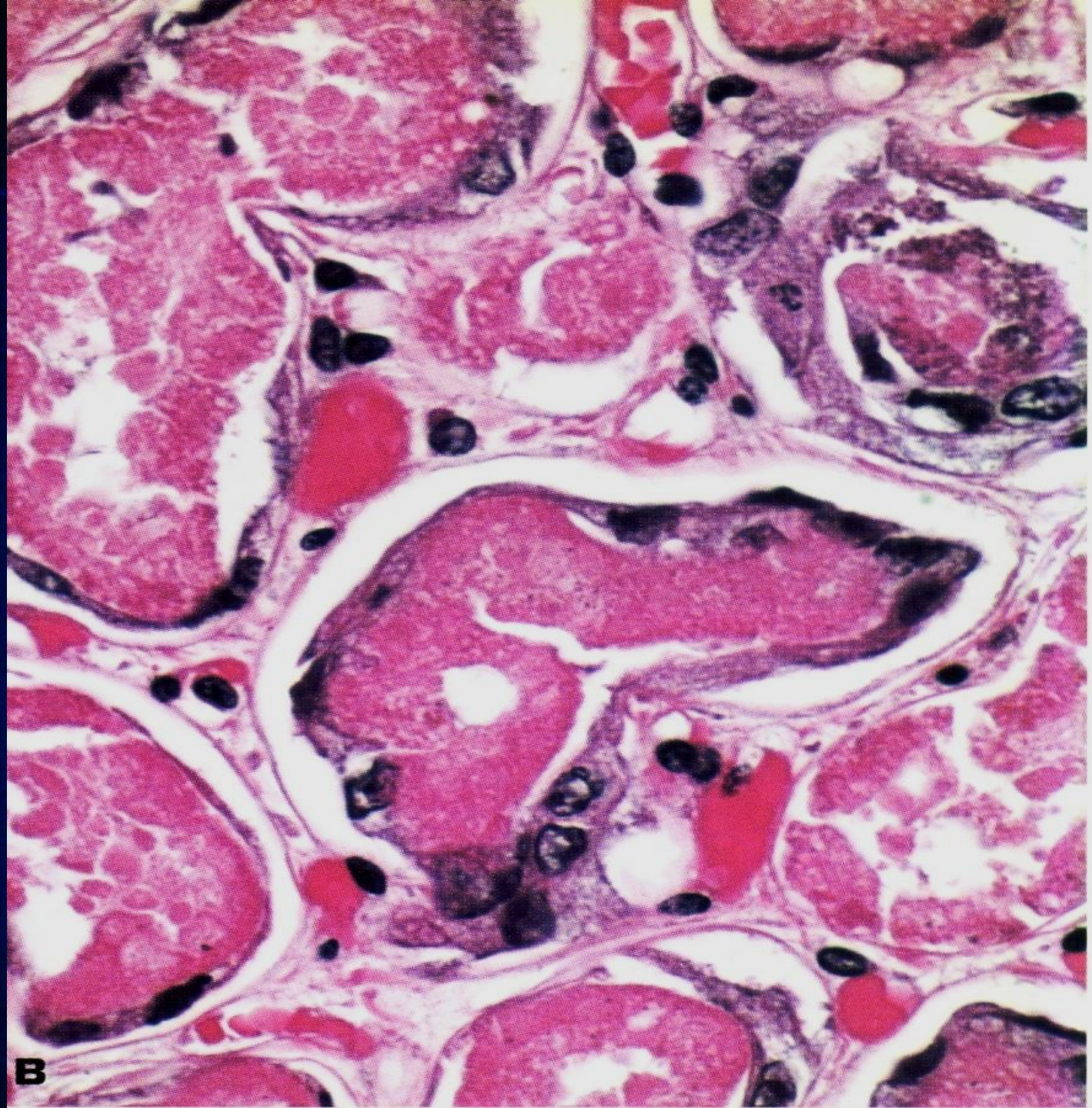
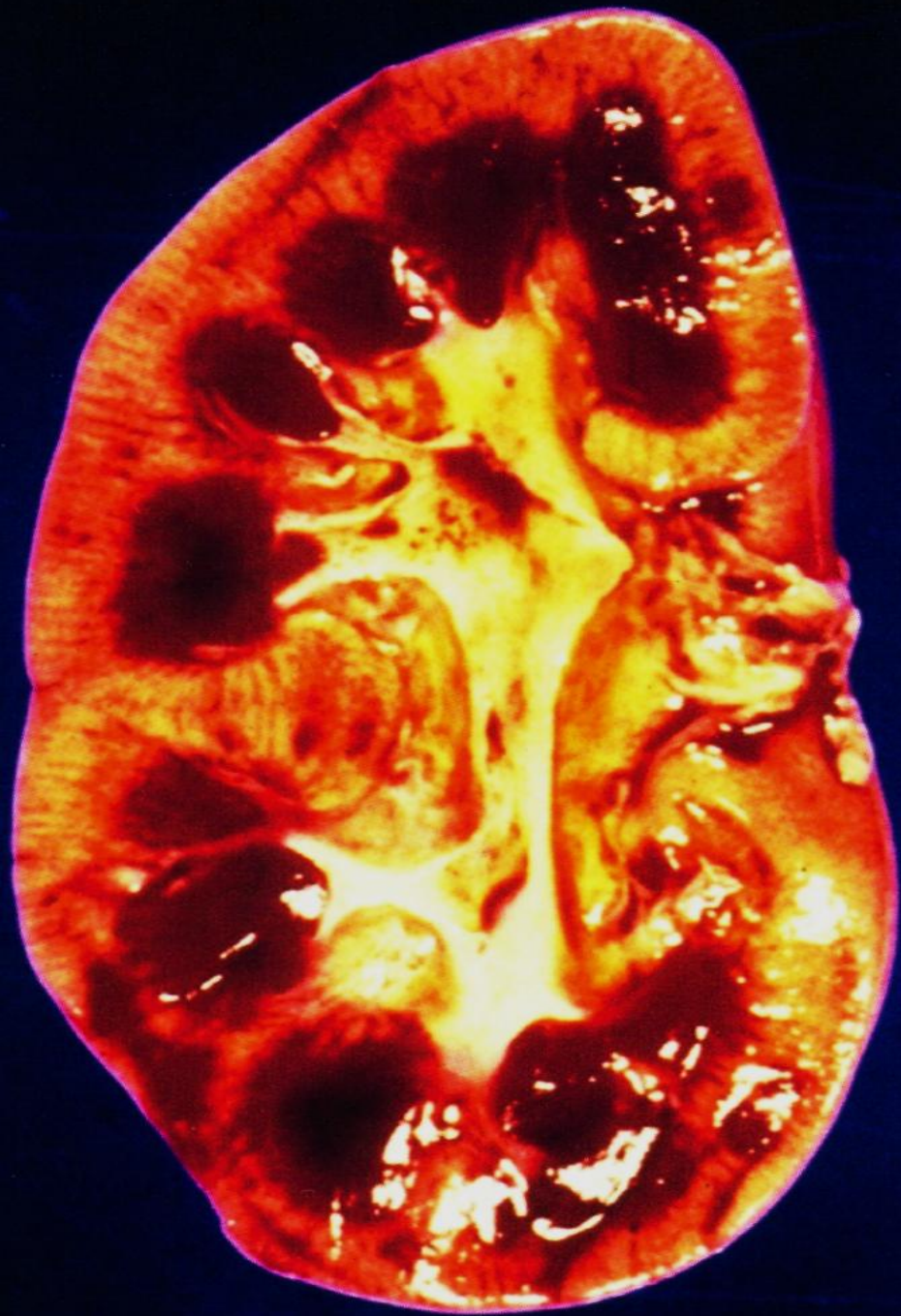
TOXIC TYPE



ATN :
Acute tubular necrosis

	Casts
	Necrosis

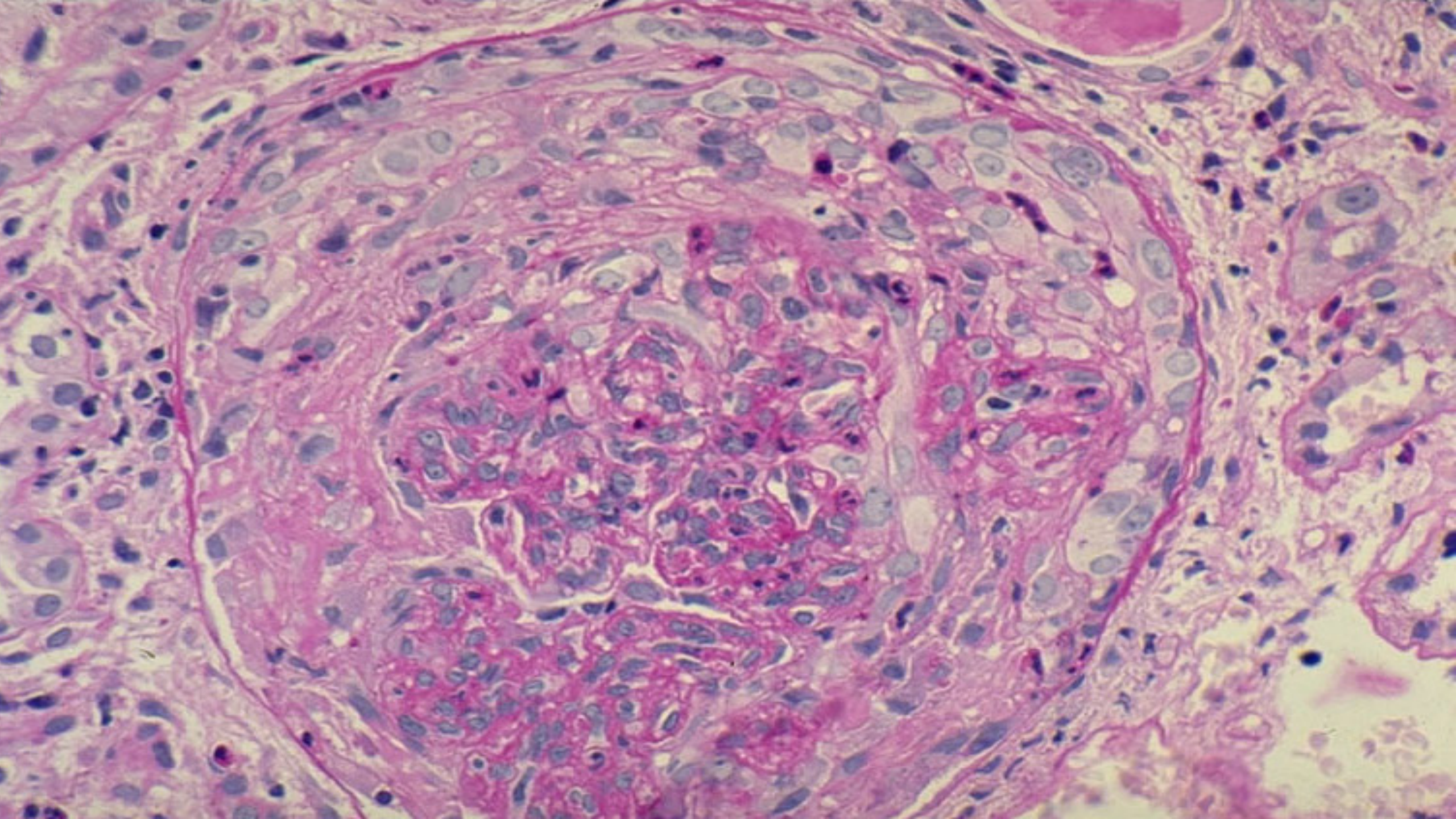




A

B

RPGN (RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS) IS A SYNDROME DEFINED BY THE RAPID LOSS OF RENAL FUNCTION OVER DAYS TO WEEKS DUE TO ACUTE GLOMERULONEPHRITIS.



ACUTE KIDNEY INJURY

- Homework :
- 1- Autosomal dominant polycystic kidney disease
- 2- Autosomal recessive polycystic kidney disease
- 3- Renal dysplasia