ΟΞΕΙΑ ΝΕΦΡΙΚΗ ΒΛΑΒΗ ΣΤΗΝ ΤΡΙΤΗ ΗΛΙΚΙΑ

Ιωάννης Γ. Γριβέας
Νεφρολογικό Τμήμα 417 ΝΙΜΤΣ
Μ.Χ.Α. "Νεφροιατρική"

athens-NEPHROLOGY

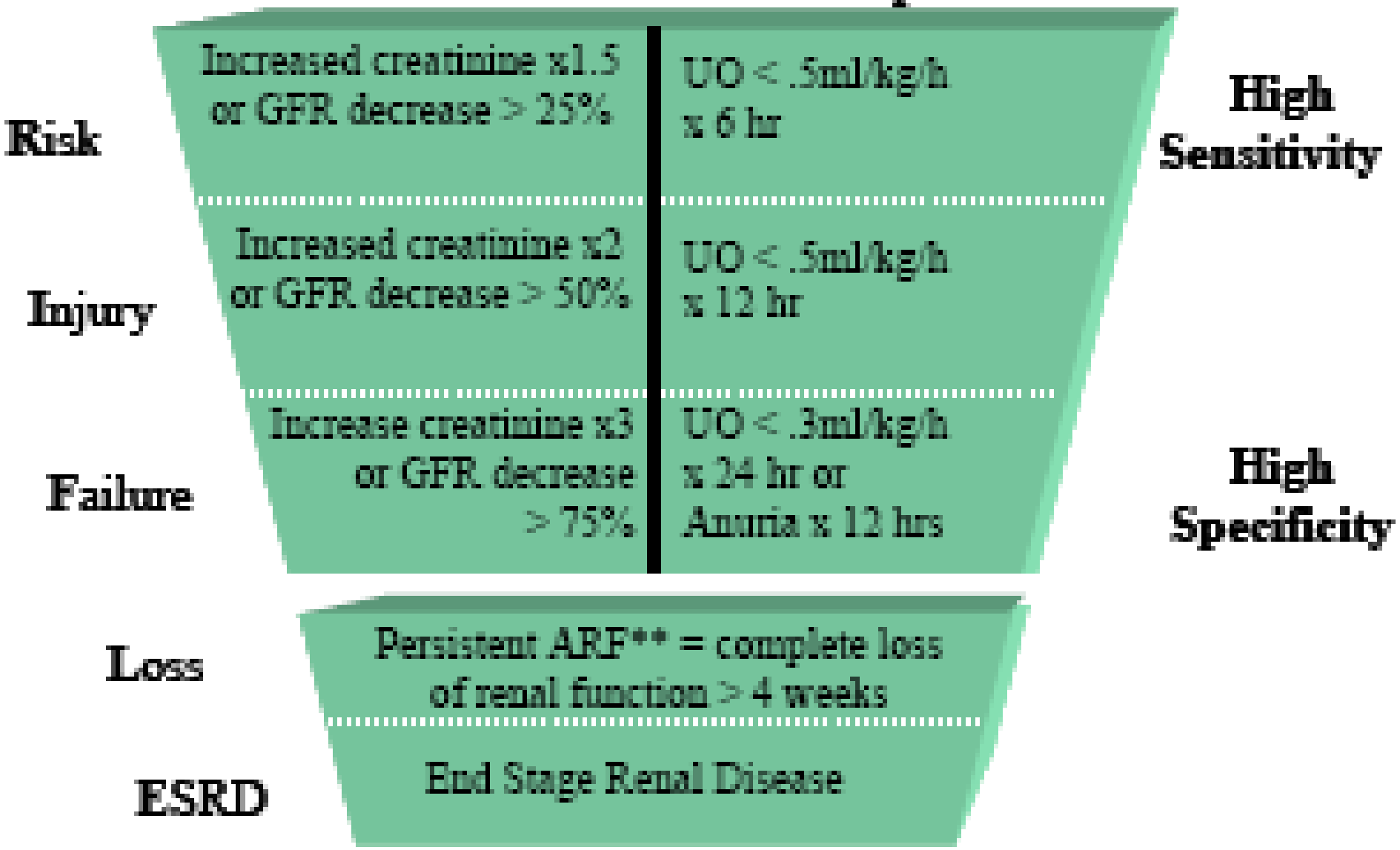
Definition Acute Renal failure (ARF)-Acute Kidney Injury (AKI)

- Inability of kidney to maintain homeostasis leading to a build up of nitrogenous wastes
- Different to renal insufficiency where kidney function is deranged but can still support life
- Exact biochemical/clinical definition not clear – 26 studies – no 2 used the same definition

AKI

- Occurs over hours/days
- Lab definition
  - Increase in baseline creatinine of more than 50%
  - Decrease in creatinine clearance of more than 50%
  - Deterioration in renal function requiring dialysis

RIFLE Criteria for Acute Renal Dysfunction



Acute kidney injury stages

AKI STAGE	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of 26 micromol/litre or more within 48 hours OR 1.5 to 2-fold increase from baseline	Less than 0.5 ml/kg/hour for more than 6 hours*
2	Increase in serum creatinine to more than 2 to 3-fold from baseline	Less than 0.5 ml/kg/hour for more than 12 hours
3	Increase in serum creatinine to more than 3-fold from baseline OR Serum creatinine more than 354 micromol/litre with an acute increase of at least 44 micromol/ litre	Less than 0.3 ml/kg/hour for 24 hours or anuria for 12 hours

* Urine output of less than 0.5 ml/kg/hour more than 8 hours in children and young people

AKI definitions

- Anuria – no urine output or less than 100mls/24 hours.
- Oliguria - <500mls urine output/24 hours or <20mls/hour.
- Polyuria - >2.5L/24 hours.

Del Giudice et al., J Nephrol Therapeut 2012, 2:6
http://dx.doi.org/10.4172/2161-0959.1000129

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Review Article Open Access

Acute Kidney Injury in the Elderly: Epidemiology, Risk Factors and Outcomes

Antonio Del Giudice[1*], Matteo Piemontese[1], Giuseppe Valente[1], Michele Prencipe[1], Chiara Di Giorgio[2] and Filippo Aucella[1]

[1]*Division of Nephrology and Dialysis, IRCCS Casa Sollievo della Sofferenza, San Giovanni Rotondo, Italy*
[2]*Proof-reader, Department of Emergency and Organ Transplantation, University Hospital of Bari, Bari, Italy*

Parenchyma: reduced total mass

Glomeruli: decreased epithelial cells, increased mesangial cells, sclerosis

Afferent arterioles: hyalinosis

Glomerular capillaries: atrophy

Proximal tubules: decreased number and length

Distal tubules: diverticula and cysts

Interstitium: fibrosis

Arterial vessels: atherosclerosis and constriction

Box 1: Structural changes occurring in aging kidney [10].

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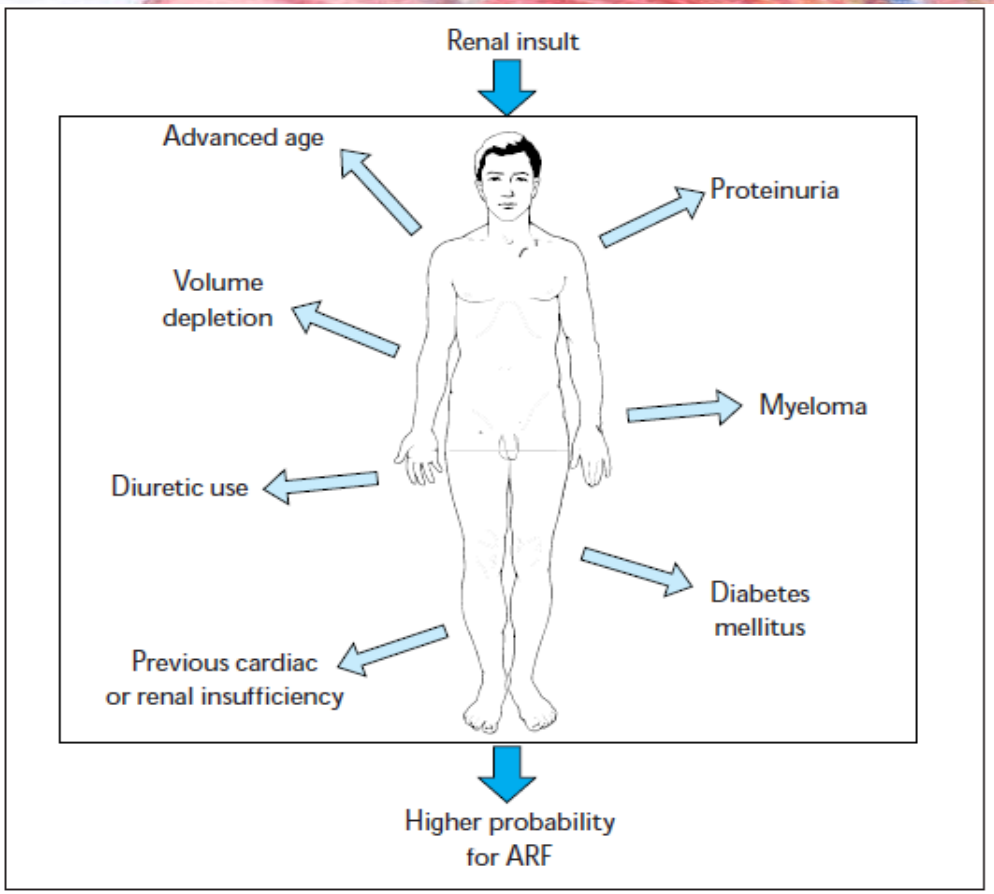
Authors	Reference	Incidence
Wen et al., China	[2]	2.76% (aged 65-80) 14.8% (aged >80)
Xue et al., USA	[7]	1.85% (aged <65) 2.86% (aged >85)
Ishani et al., USA	[22]	3.1%
Hsu et al., USA	[23]	78/100,000 person-yrs (aged <50) 3,545/100,000 person-yrs (aged ≥ 80)
Ali et al., Scotland	[24]	1,811 cases per million population
Liano and Pascual, Spain	[25]	209 cases per million population
Pasqual et al., Spain	[26]	3.5-times higher (aged >70) 5-times higher (aged >80)
Baraldi et al., Italy	[27]	10 times higher in aged ≥ 65 vs. ≤ 65
Fang et al., China	[28]	4.10% (aged 60-80) 6.17% (aged >80)
Uchino et al., multinational	[29]	5.7%
Garzotto et al., Italy	[30]	65,8%

Table 1: Incidence of acute kidney injury (AKI) in the elderly.



Acute Renal Failure: Causes and Prognosis

Fernando Liaño
Julio Pascual

Risk factors in adults having surgery or iodinated contrast agents

Risk factor	Surgery	Iodinated contrast agents
Age	Age 65 years or over	Age 75 years or over
CKD	CKD with eGFR <60	CKD with eGFR <40
Diabetes	Yes	Yes, if also has CKD
Heart failure	Yes	Yes
Hypovolaemia	Yes, especially if acutely unwell	Yes
Other conditions	Liver disease	Renal transplant
Clinical treatments or drugs	a) Emergency surgery, especially if patient has sepsis or hypovolaemia b) Nephrotoxic drugs in the perioperative period c) Intraperitoneal surgery	Increased volumes of contrast agent Intra-arterial route

AKI and Mortality

Brigham and Womens, 9210 adults Multivariable Odds Ratio for Death

•**AKI (Δ in S_{Cr} >0.5)**	**6.5**	**<0.0001**
•Age (per 10 yr)	1.7	<0.0001
•CKD	2.5	<0.0001
•CV dis.	1.5	<0.04
•Respiratory dis	3	<0.0001
•GI dis.	2.4	<0.001
•Cancer	2.9	<0.0001
•Infection	7.5	<0.0001

Chertow et al, JASN 16:3365-70 2005

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Baraldi et al., Italy	[27]	33.3% (aged ≥ 65) 2.5% (aged ≤ 64)
Ukino et al., multinational	[29]	60.3%
Garzotto et al., Italy	[30]	21.7% (intensive care unit and in-hospital)
James et al., Canada	[40]	3% at 35 months
Lo et al., USA	[42]	41.9% (dialysis-requiring acute renal failure) 1.14% (non-dialysis-requiring acute renal failure)
Gong et al., China	[56]	42%
Sesso et al., Brazil	[59]	41% (in-hospital) 59% (community)
Kohli et al., India	[60]	60.9%

Table 2: Mortality from acute kidney injury (AKI) in the elderly.

AKI

- Pre renal (functional)
- Renal-intrinsic (structural)
- Post renal (obstruction)

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JIACM 2011; 12(4): 290-6

Acute renal failure in the elderly

SC Dash*, D Bhowmik**

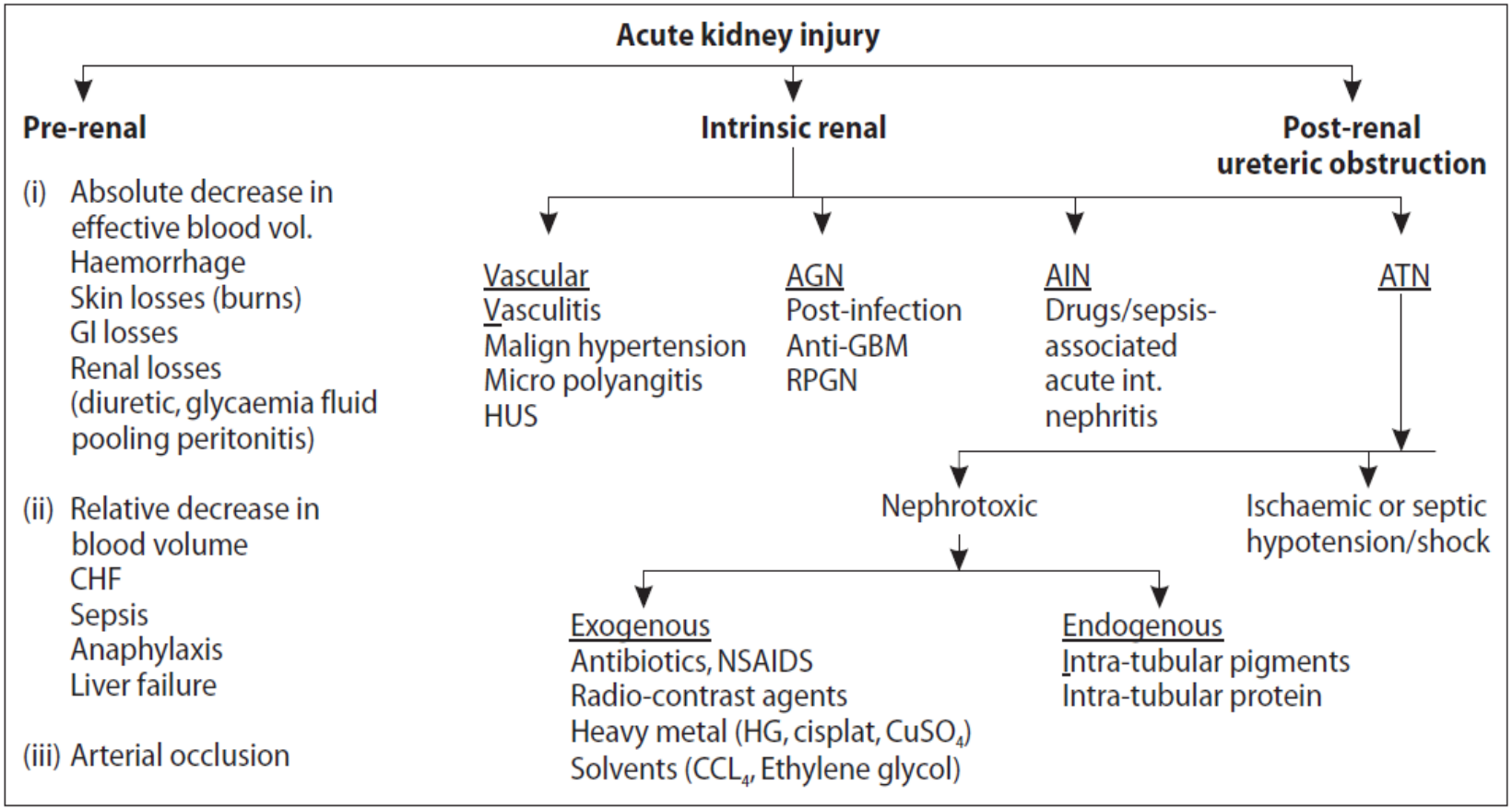

Fig. 1: *Acute kidney injury.*

REVIEW ARTICLE

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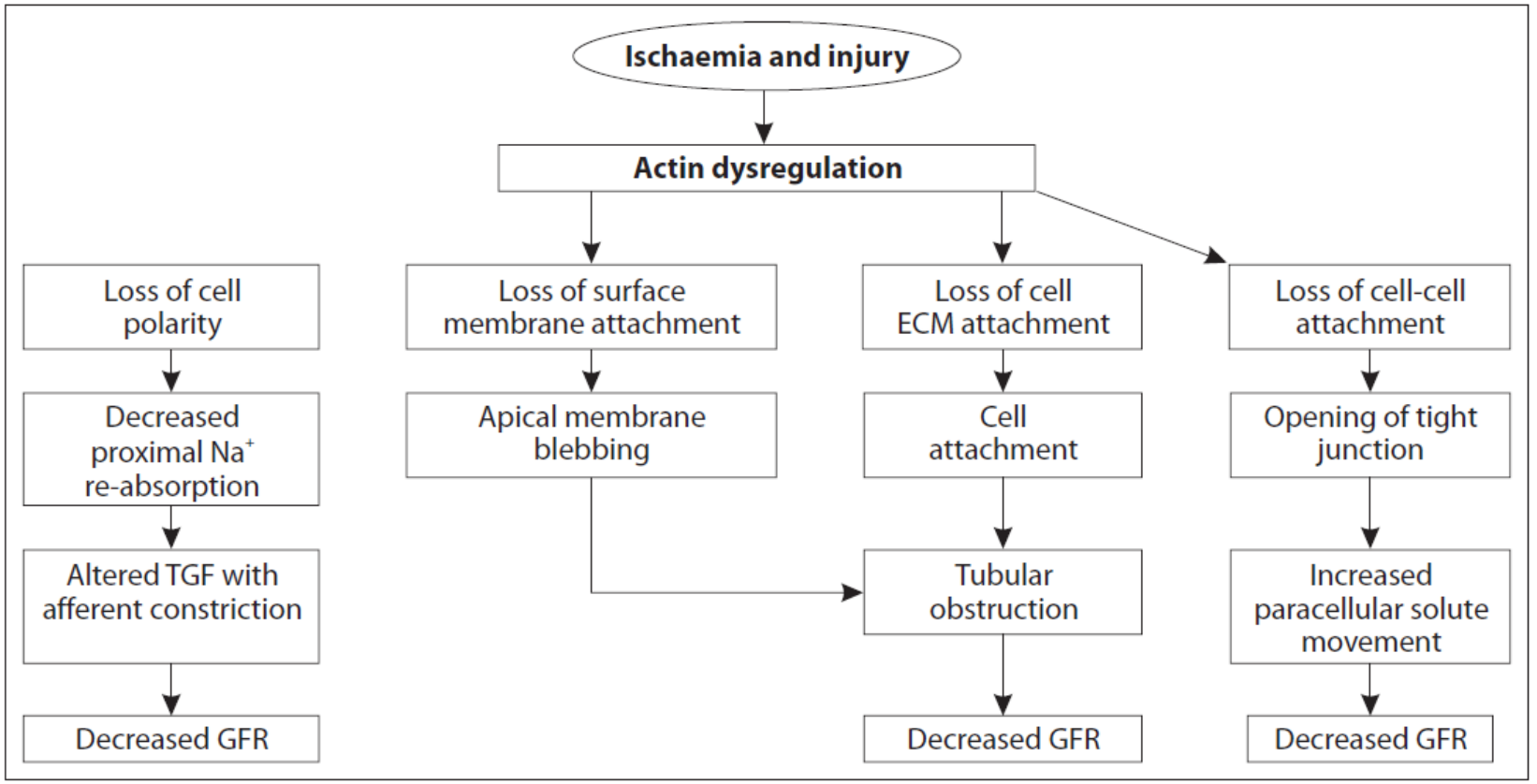
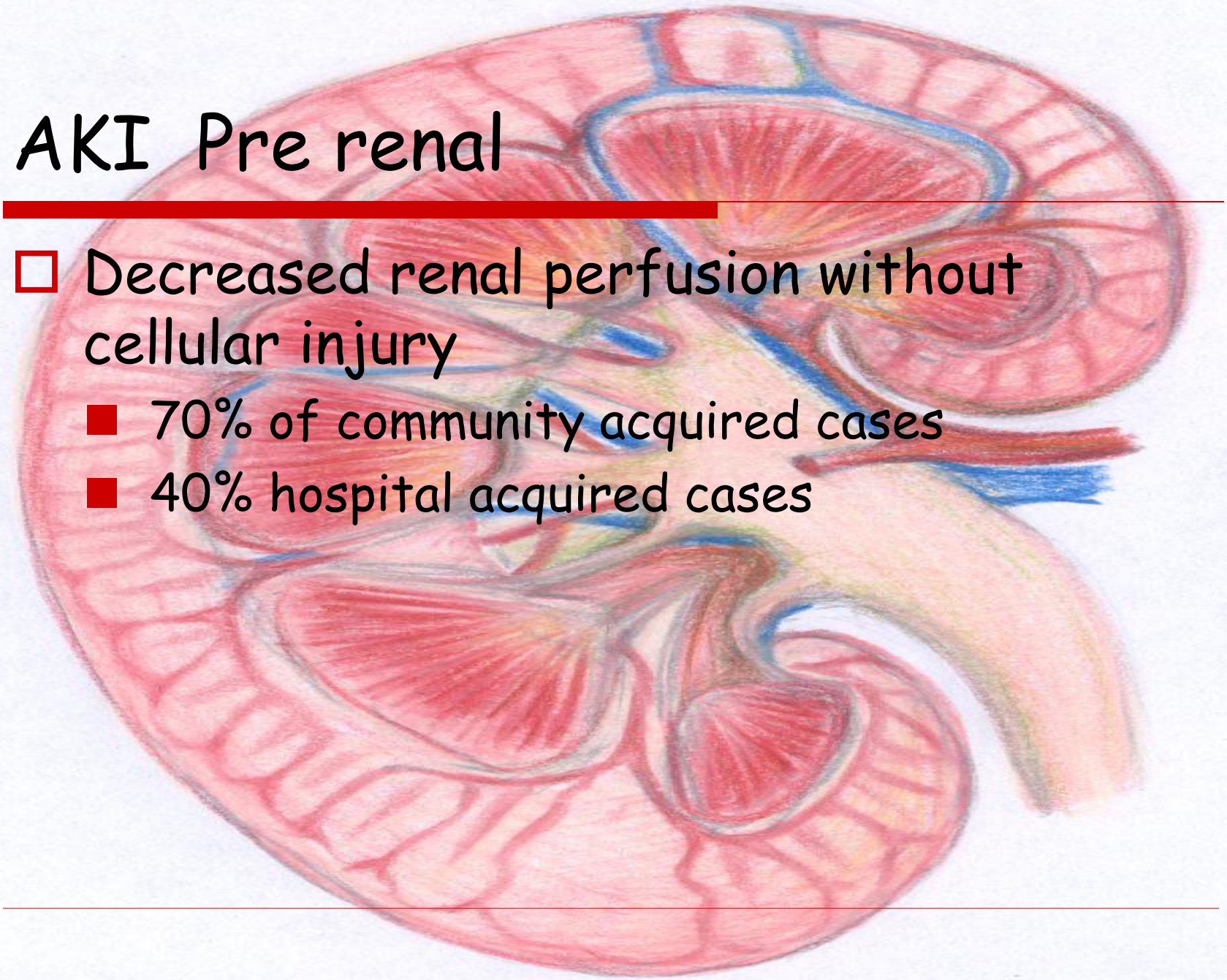


Fig. 2: *Renal ischaemia and injury.*

AKI Pre renal

- Decreased renal perfusion without cellular injury
  - 70% of community acquired cases
  - 40% hospital acquired cases

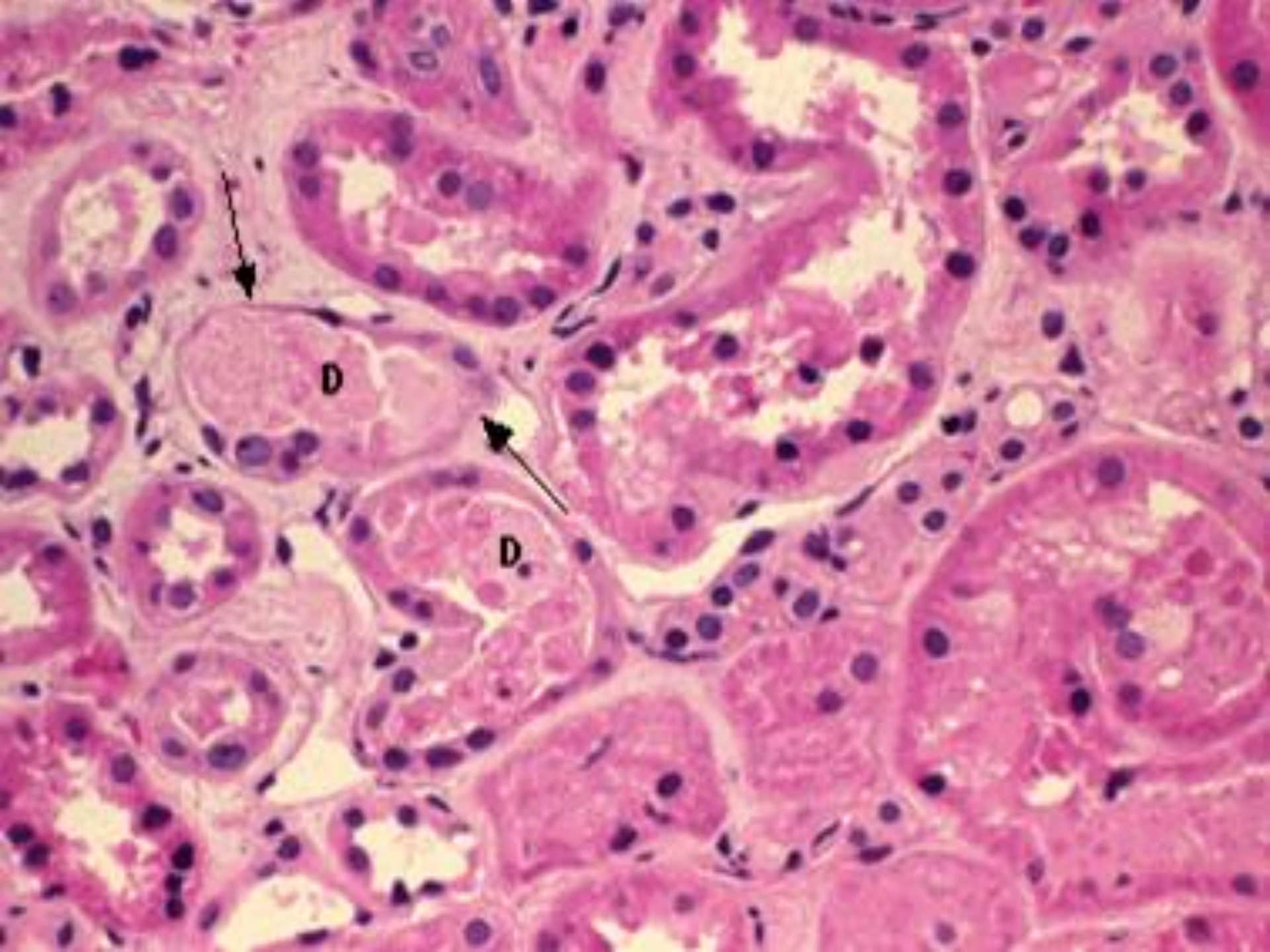


Pre-Renal Azotemia Pathophysiology

- Renal hypoperfusion
  - Decreased renal blood flow and GFR
  - Increased filtration fraction (GFR/RBF)
- Increased Na and H_2O reabsorption
  - Oliguria, high U_{osm}, low U_{Na}
  - Elevated BUN/Cr ratio

Prerenal

- Often rapidly reversible if we can identify this early
- The elderly at high risk because of their predisposition to hypovolemia and renal atherosclerotic disease
- This is by definition rapidly reversible upon the restoration of renal blood flow and glomerular perfusion pressure
- THE KIDNEYS ARE NORMAL
- This will accompany any disease that involves hypovolemia, low cardiac output, systemic dilation, or selective intrarenal vasoconstriction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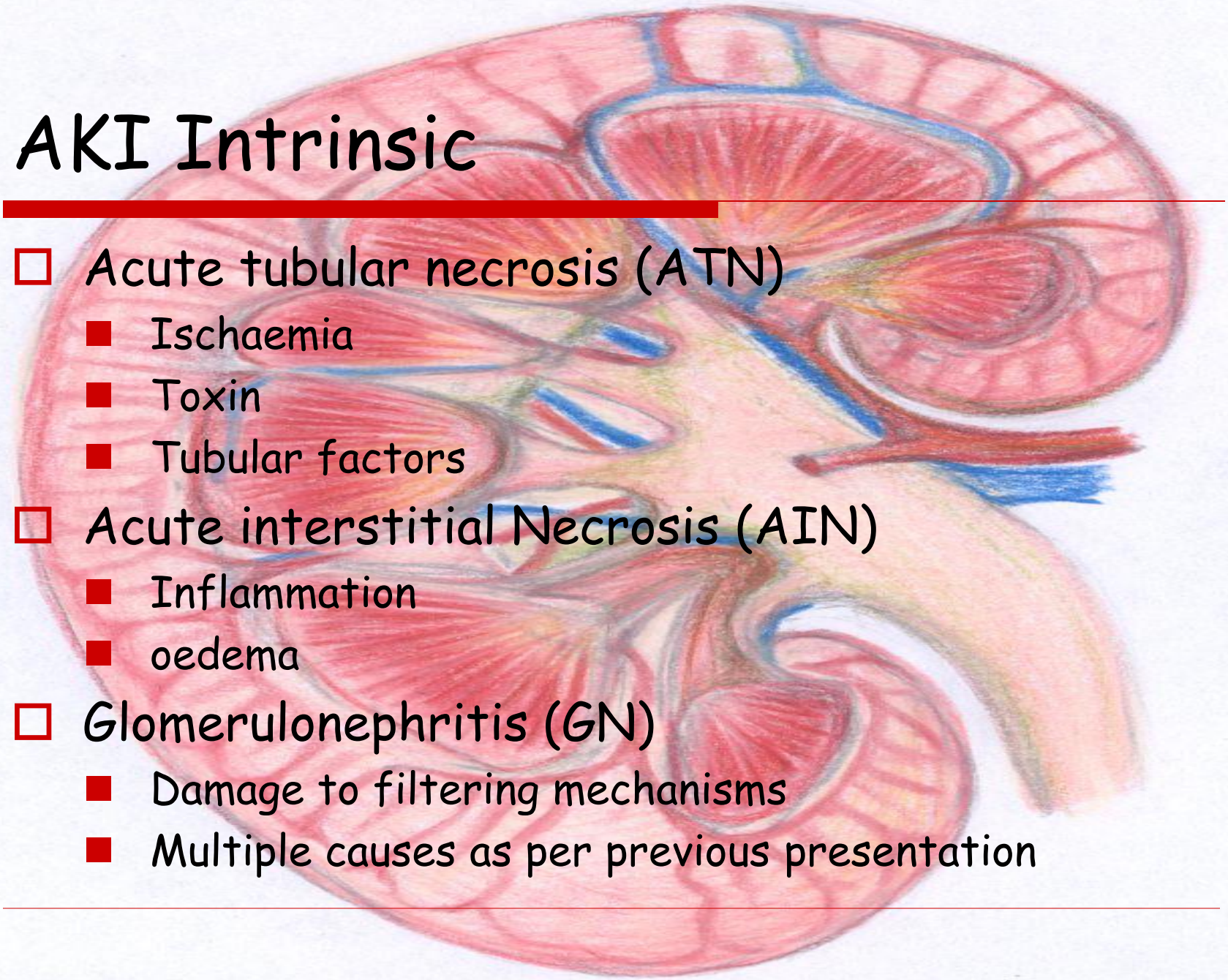
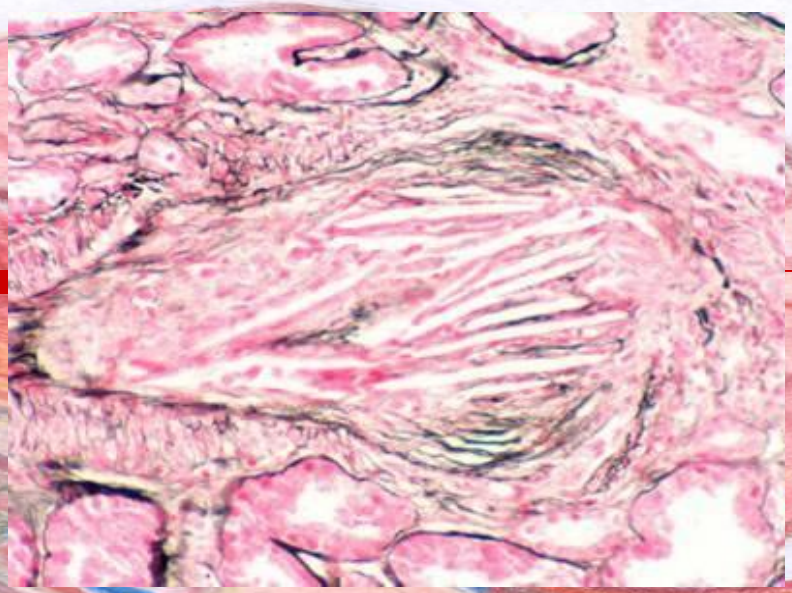
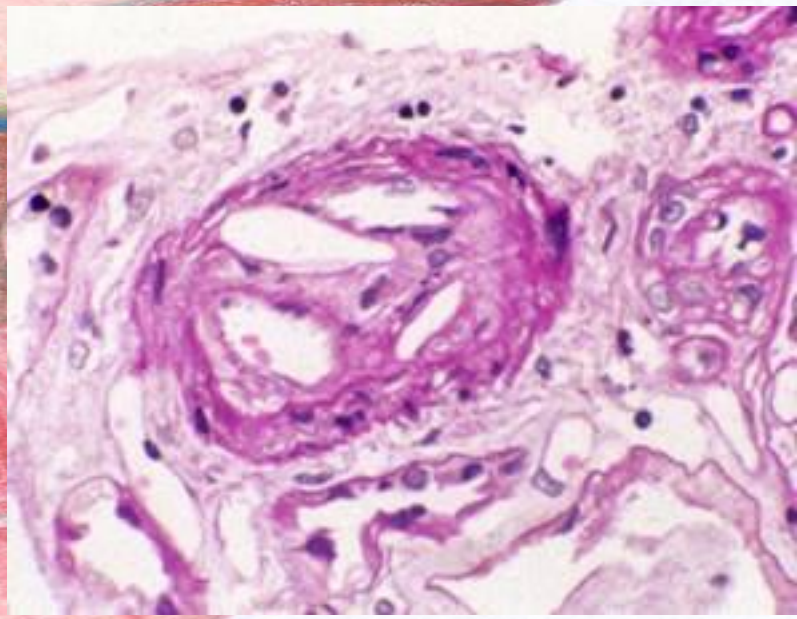
AKI Intrinsic

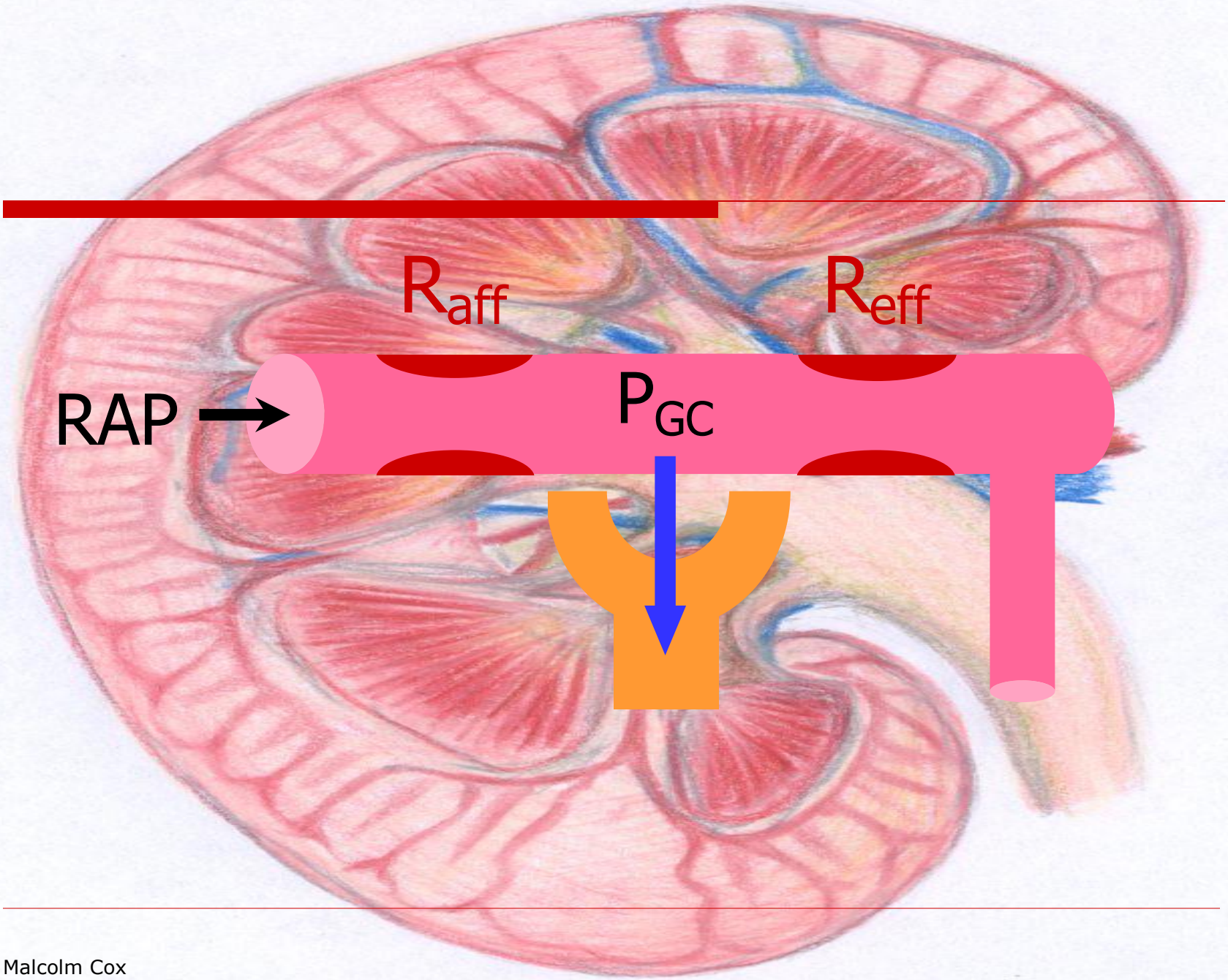


- Acute tubular necrosis (ATN)
  - Ischaemia
  - Toxin
  - Tubular factors
- Acute interstitial Necrosis (AIN)
  - Inflammation
  - oedema
- Glomerulonephritis (GN)
  - Damage to filtering mechanisms
  - Multiple causes as per previous presentation

Cholesterol Embolization

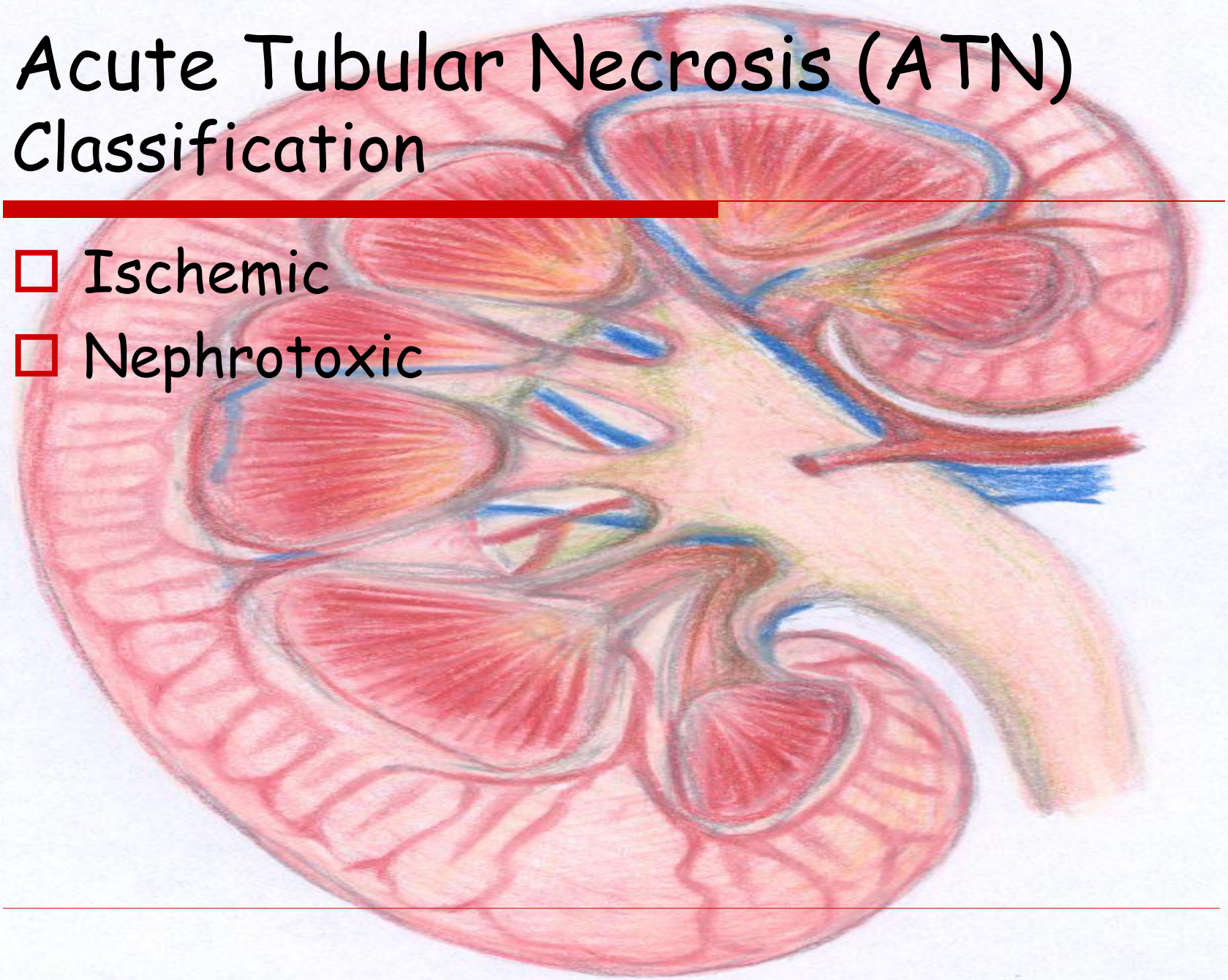





R_{aff}
R_{eff}
RAP
P_{GC}

Acute Tubular Necrosis (ATN)
Classification

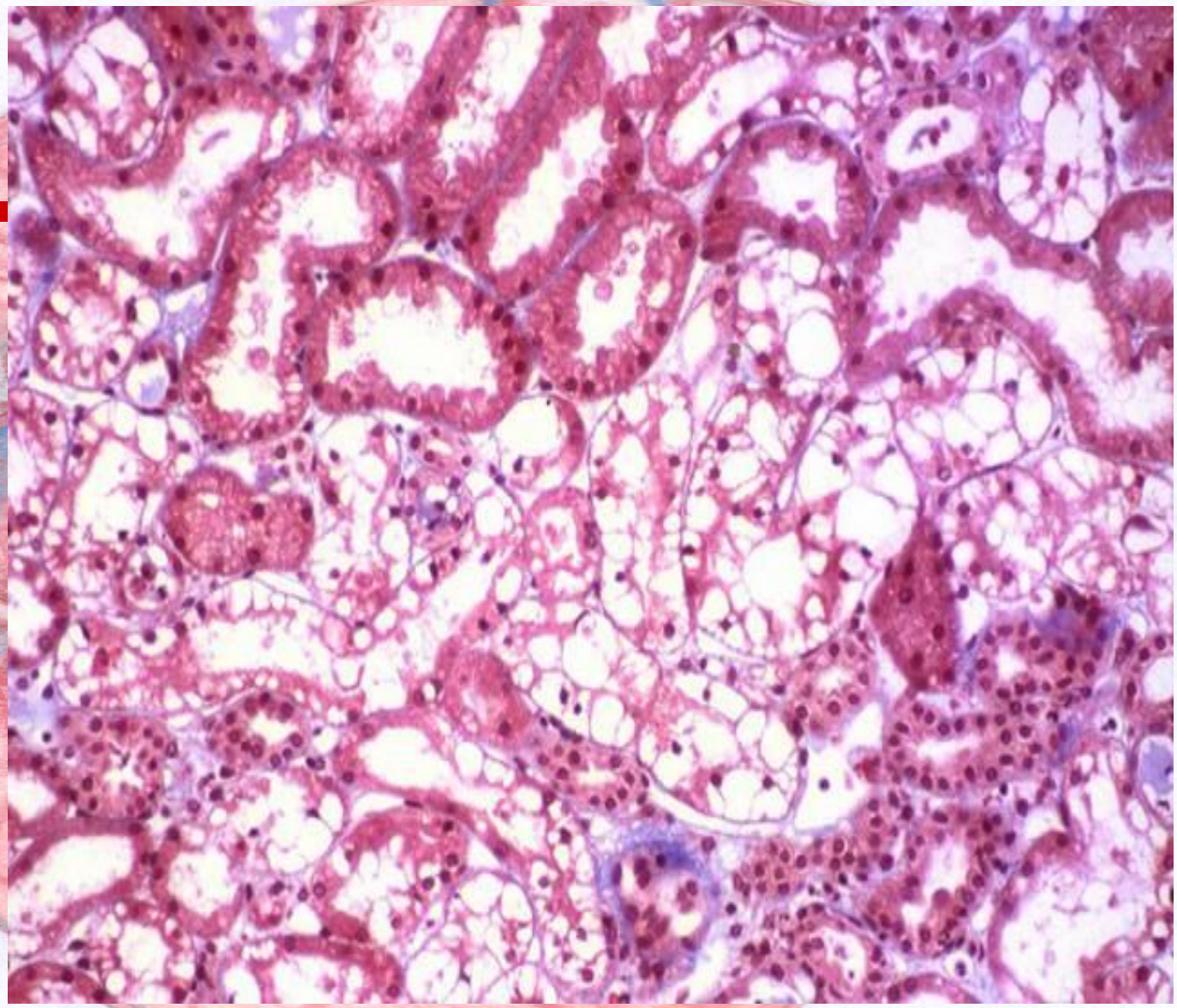
- Ischemic
- Nephrotoxic



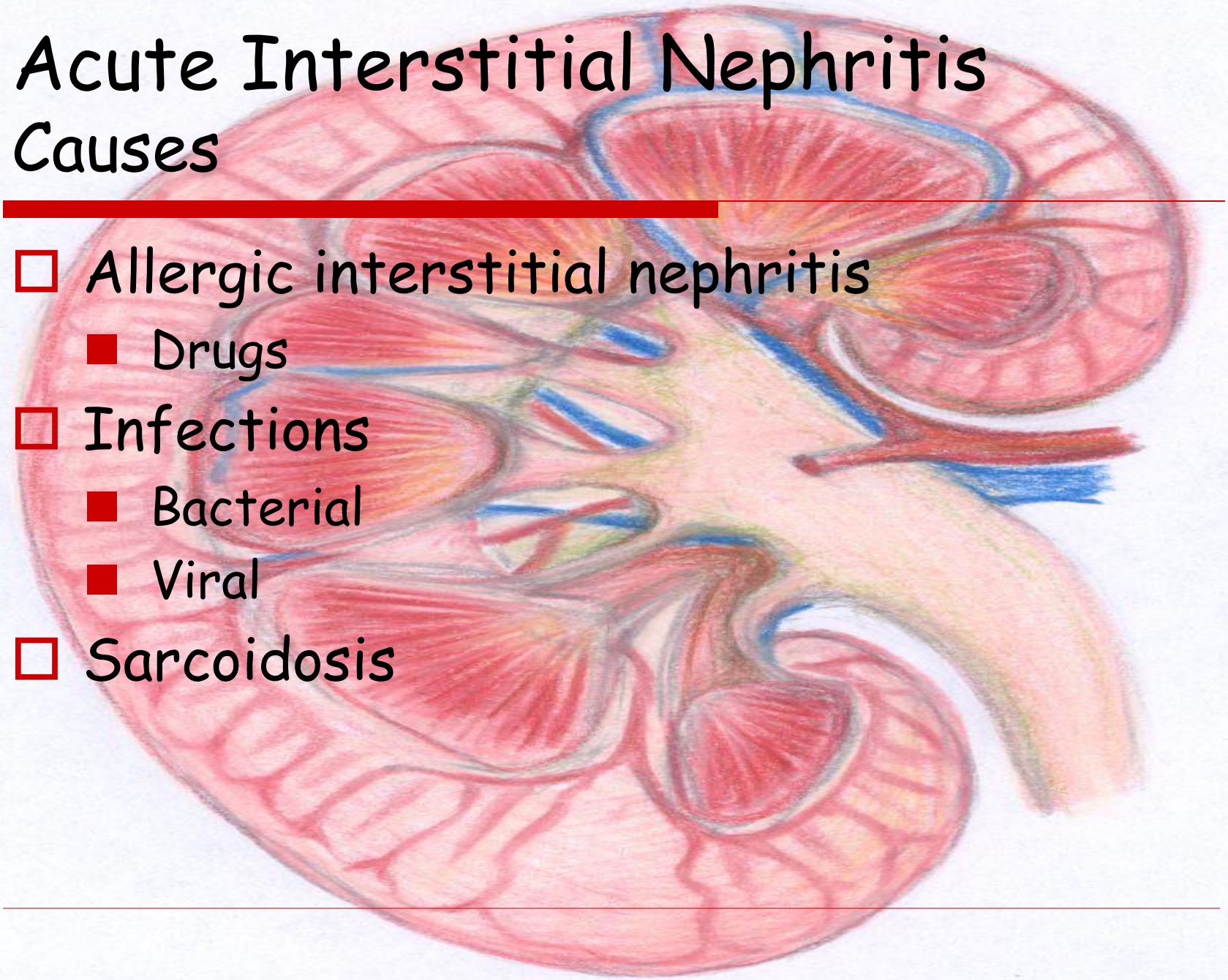
AKI
Nephrotoxic ATN

- Endogenous Toxins
  - Heme pigments (myoglobin, hemoglobin)
  - Myeloma light chains
- Exogenous Toxins
  - Antibiotics (e.g., aminoglycosides, amphotericin B)
  - Radiocontrast agents
  - Heavy metals (e.g., cis-platinum, mercury)
  - Poisons (e.g., ethylene glycol)

ATN

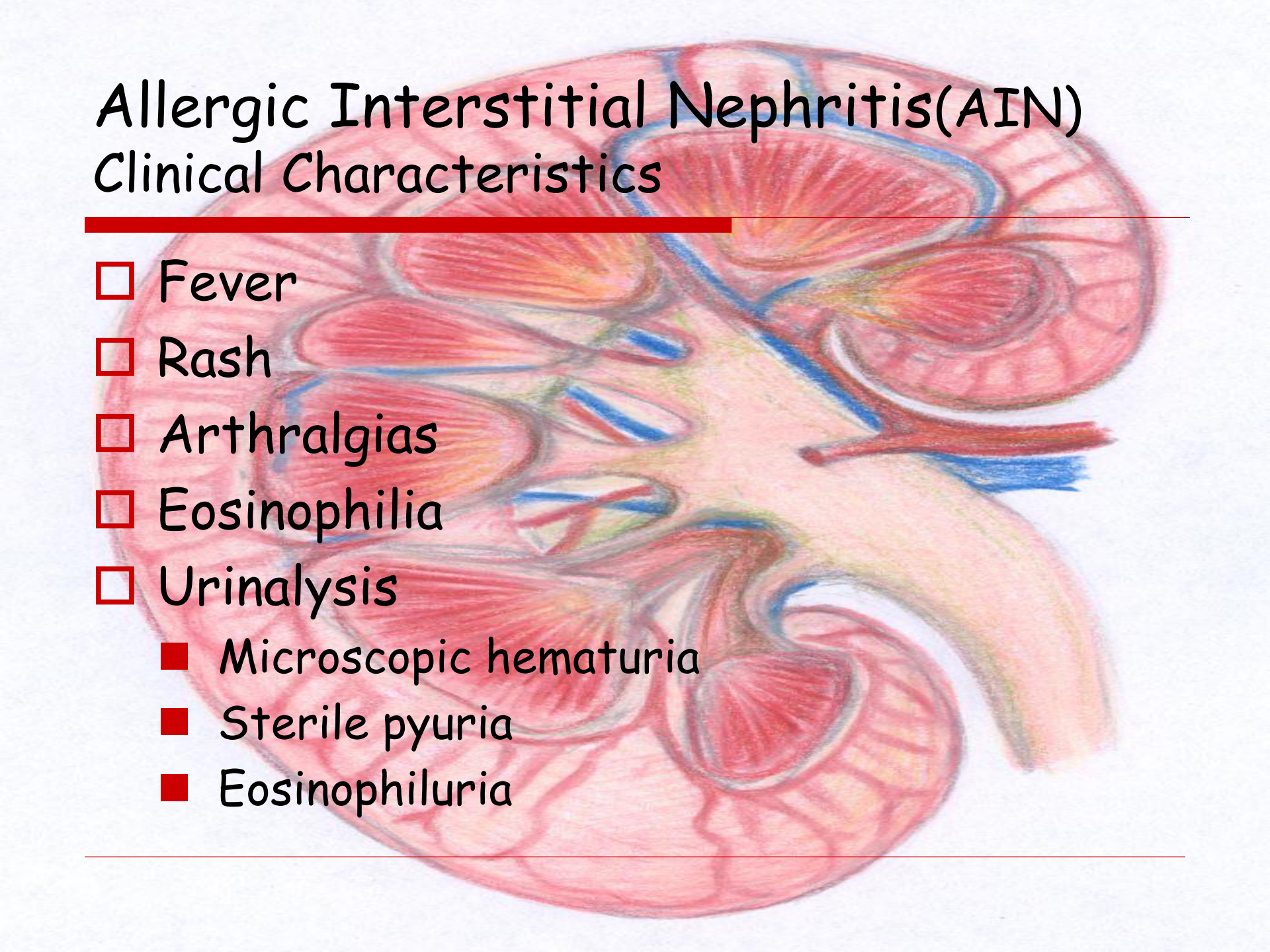


Acute Interstitial Nephritis Causes



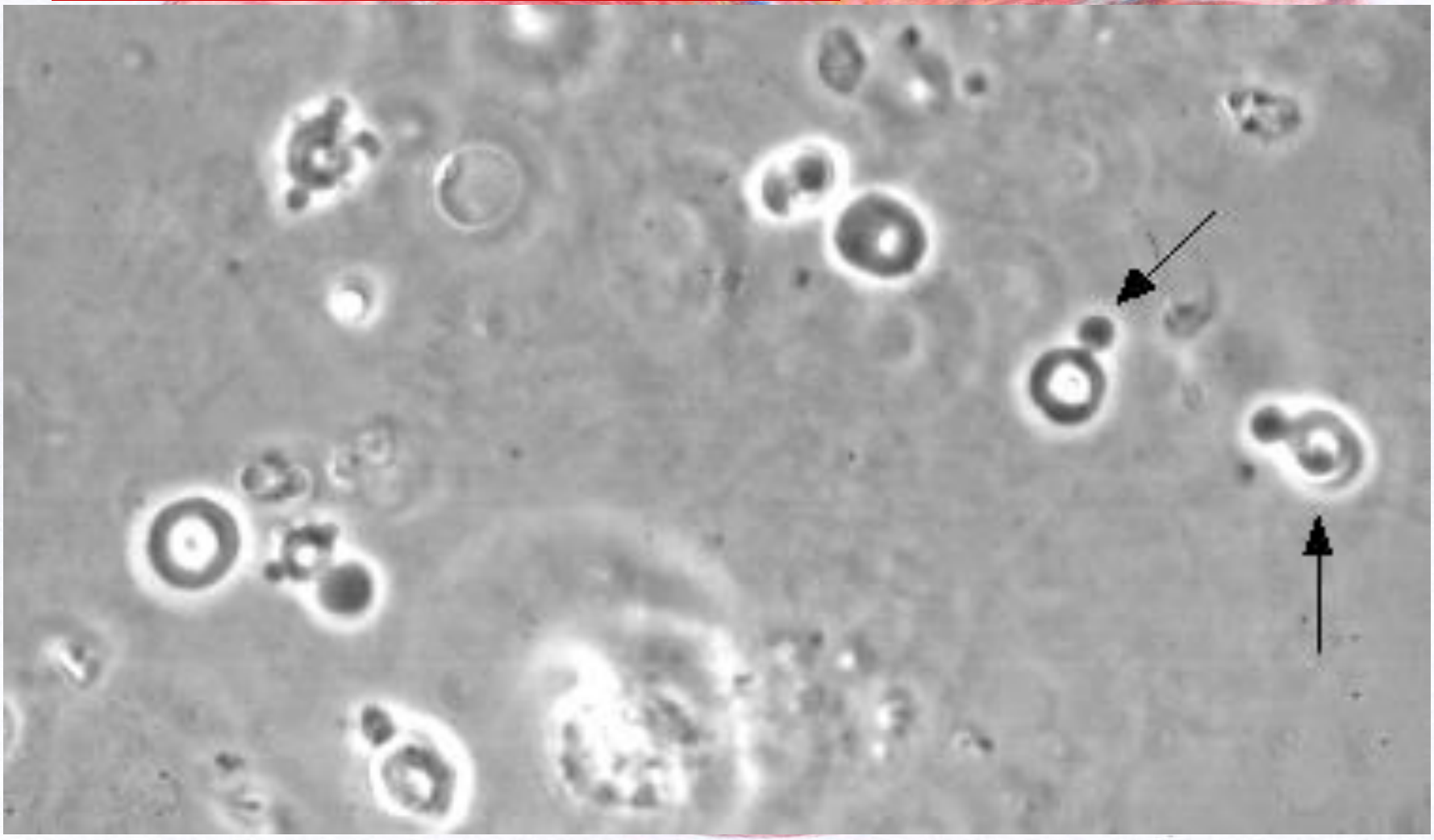
- Allergic interstitial nephritis
  - Drugs
- Infections
  - Bacterial
  - Viral
- Sarcoidosis

Allergic Interstitial Nephritis(AIN)
Clinical Characteristics

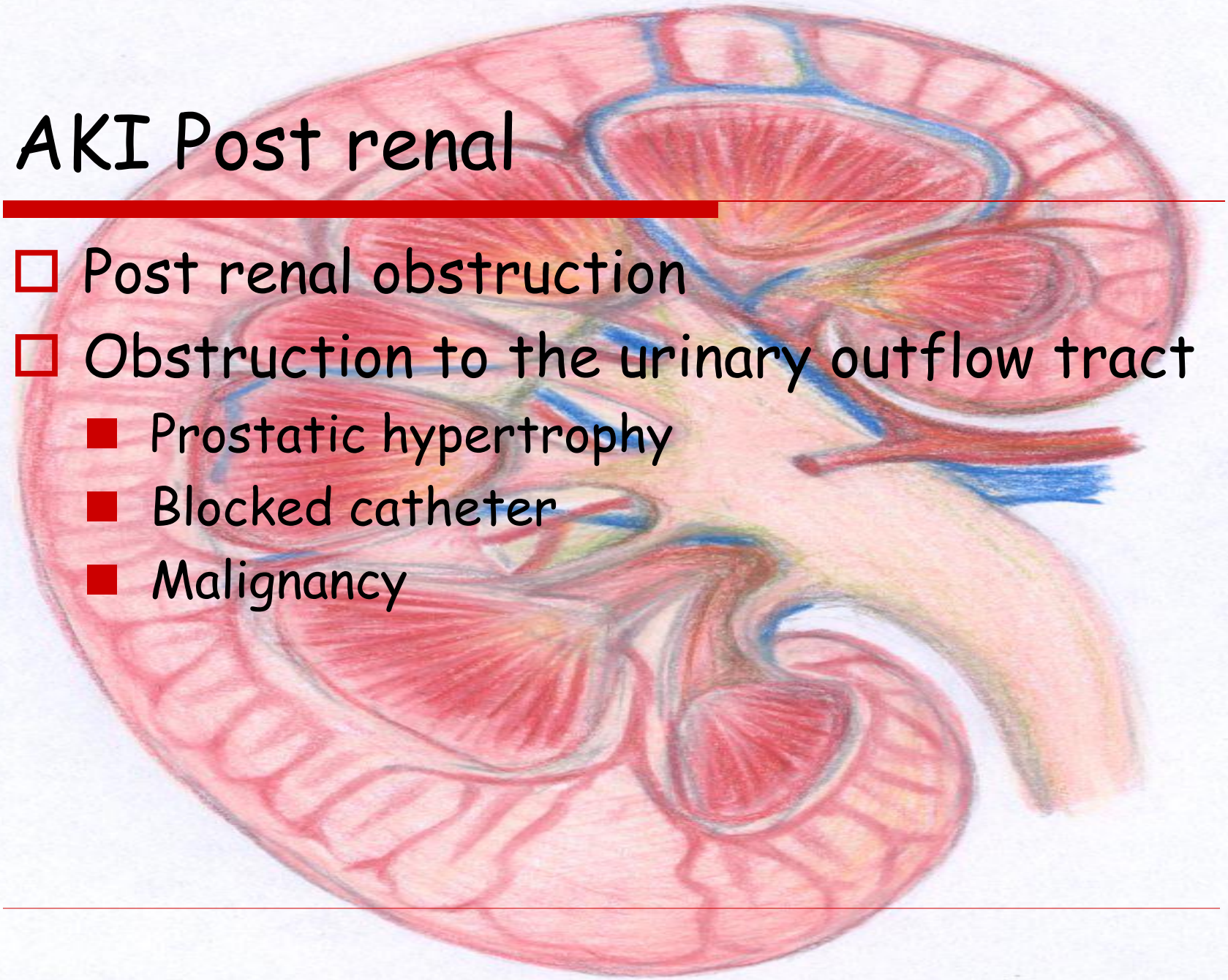


- Fever
- Rash
- Arthralgias
- Eosinophilia
- Urinalysis
  - Microscopic hematuria
  - Sterile pyuria
  - Eosinophiluria

Dysmorphic Red Blood Cells



AKI Post renal



- Post renal obstruction
- Obstruction to the urinary outflow tract
  - Prostatic hypertrophy
  - Blocked catheter
  - Malignancy

AKI Post-renal Causes

- Intra-renal Obstruction
  - Acute uric acid nephropathy
  - Drugs (e.g., acyclovir)
- Extra-renal Obstruction
  - Renal pelvis or ureter (e.g., stones, clots, tumors, papillary necrosis, retroperitoneal fibrosis)
  - Bladder (e.g., BPH, neuropathic bladder)
  - Urethra (e.g., stricture)

Differential Diagnosis

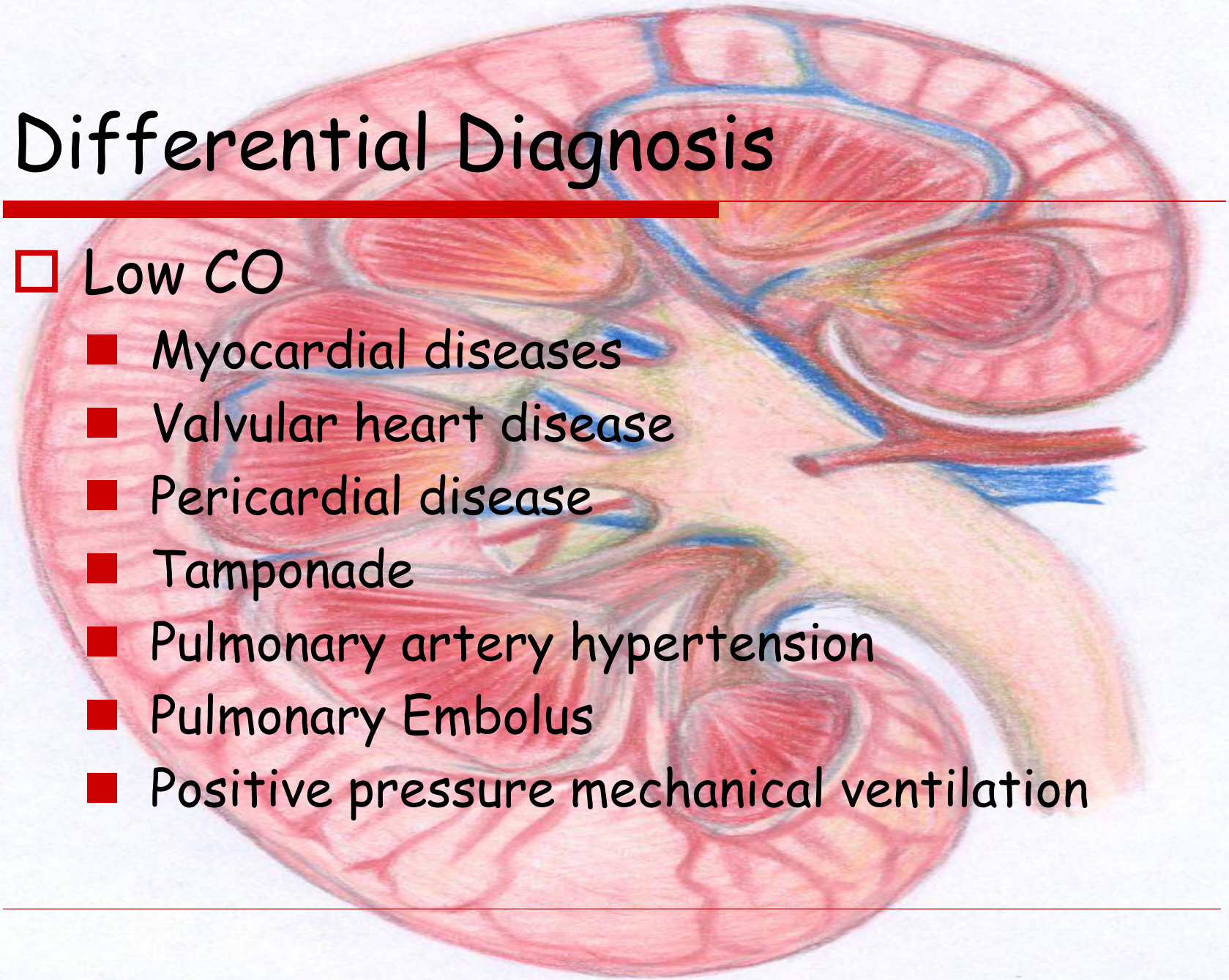
- Hypovolemia
  - GI loss: Nausea, vomiting, diarrhea (hyponatraemia)
  - Renal loss: diuresis, hypo adrenalism, osmotic diuresis (DM)
  - Sequestration: pancreatitis, peritonitis,trauma, low albumin (third spacing).
  - Hemorrhage, burns, dehydration (intravascular loss).

Differential Diagnosis

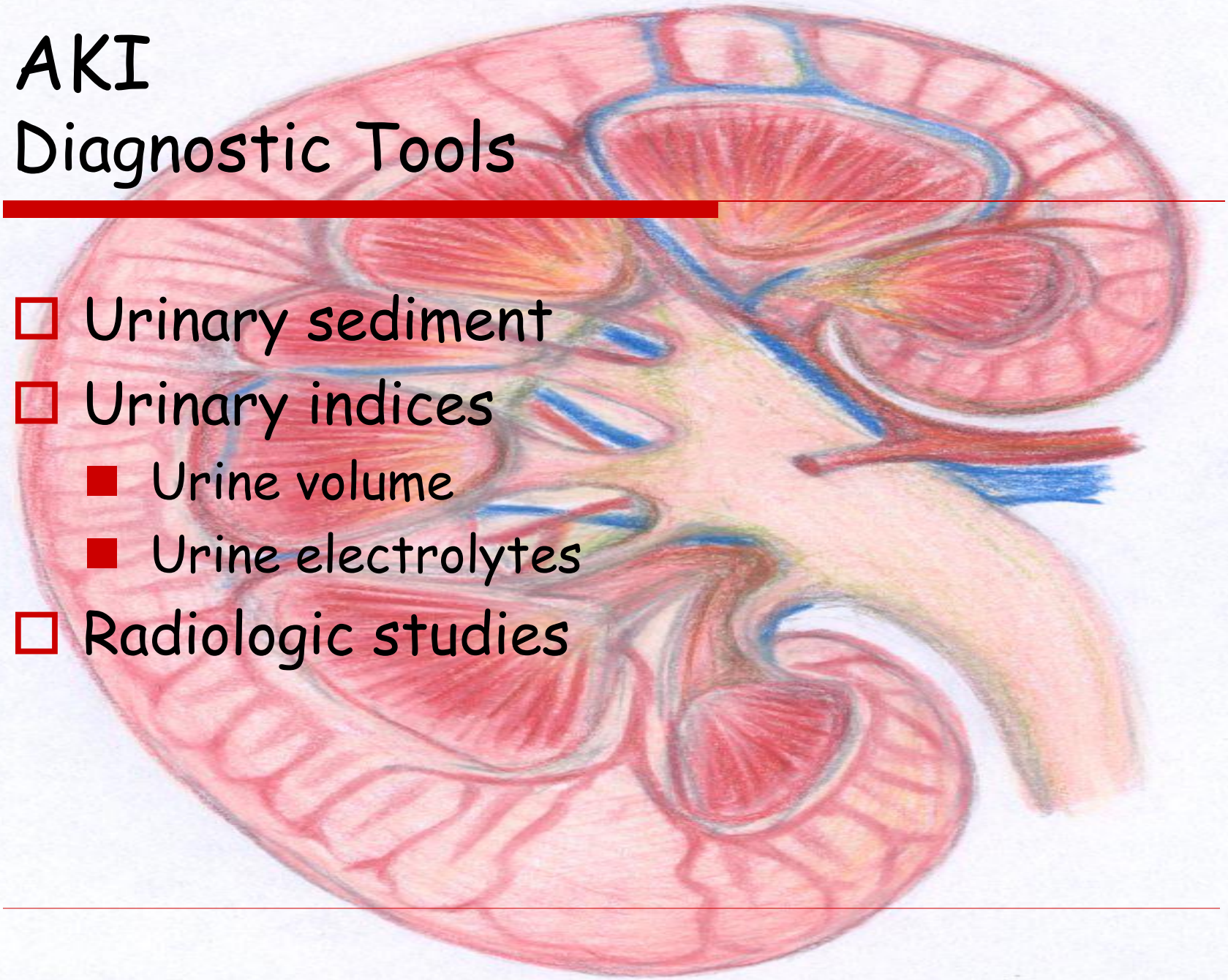
- Renal vasoconstriction: hypercalcaemia, adrenaline/noradrenaline, cyclosporin, tacrolimus, amphotericin B.
- Systemic vasodilation: sepsis, medications, anesthesia, anaphylaxis.
- Cirrhosis with ascites
- Hepato-renal syndrome
- Impairment of autoregulation: NSAIDs, ACE, ARBs.
- Hyperviscosity syndromes: Multiple Myeloma, Polycyaemia rubra vera

Differential Diagnosis

- Low CO
  - Myocardial diseases
  - Valvular heart disease
  - Pericardial disease
  - Tamponade
  - Pulmonary artery hypertension
  - Pulmonary Embolus
  - Positive pressure mechanical ventilation



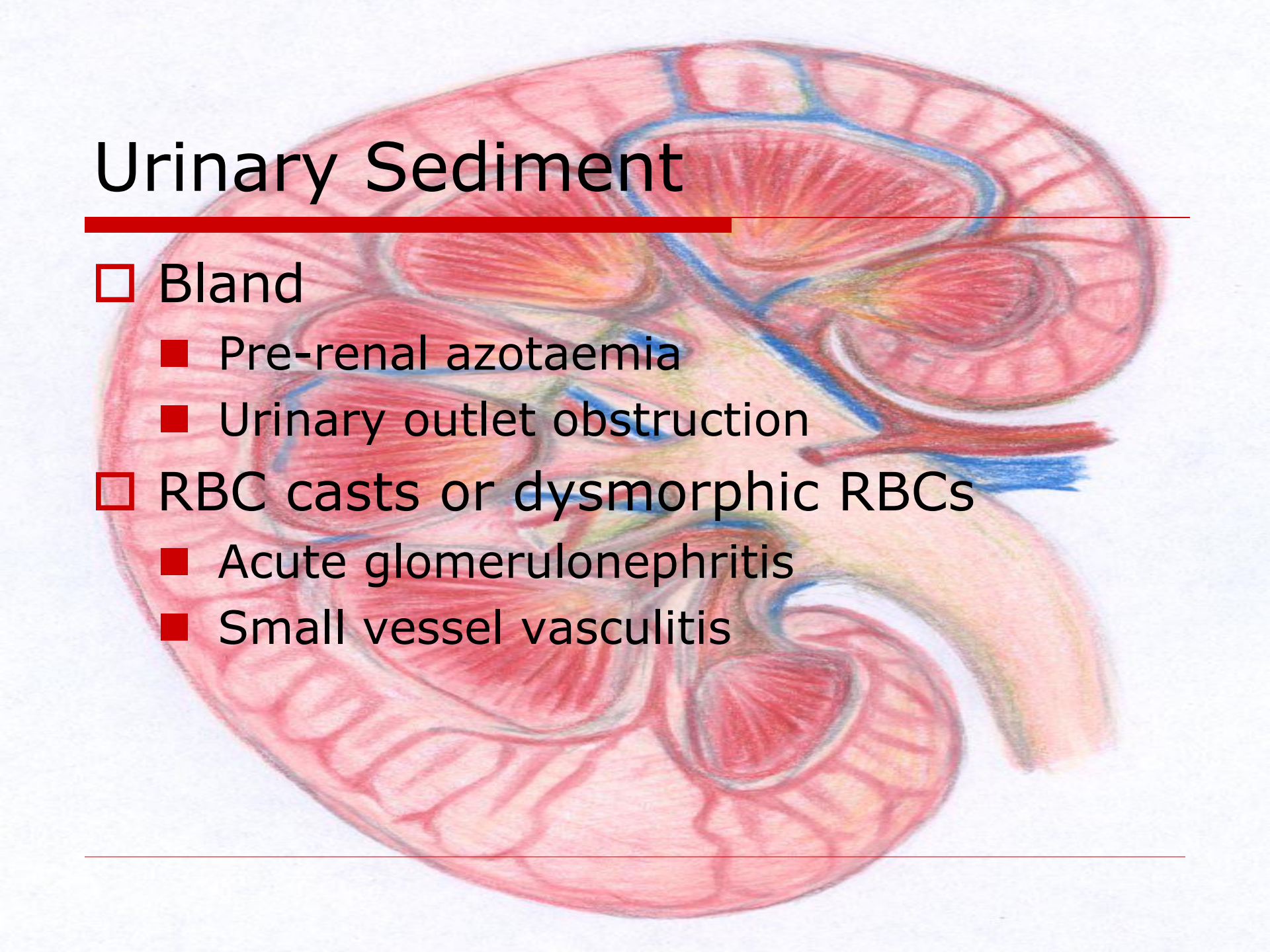
AKI
Diagnostic Tools



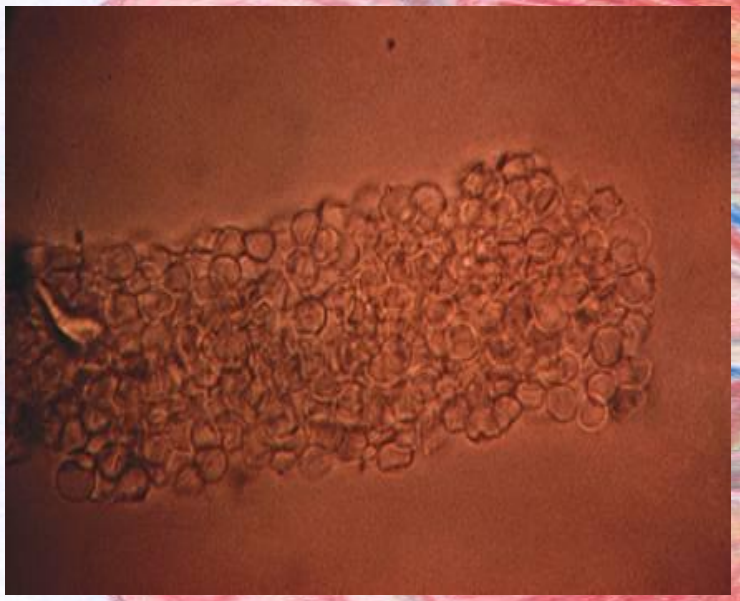
- Urinary sediment
- Urinary indices
  - Urine volume
  - Urine electrolytes
- Radiologic studies

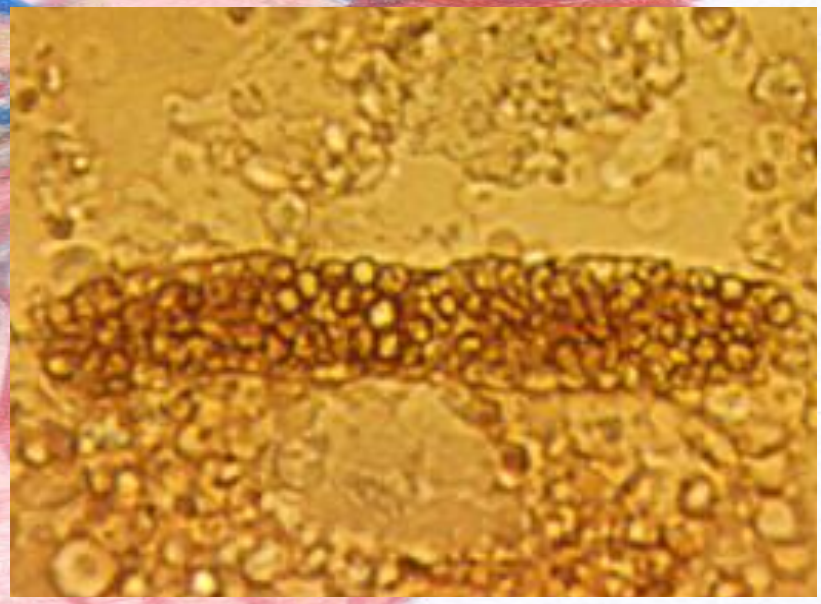
Urinary Sediment

- Bland
  - Pre-renal azotaemia
  - Urinary outlet obstruction
- RBC casts or dysmorphic RBCs
  - Acute glomerulonephritis
  - Small vessel vasculitis



Red Blood Cell Cast





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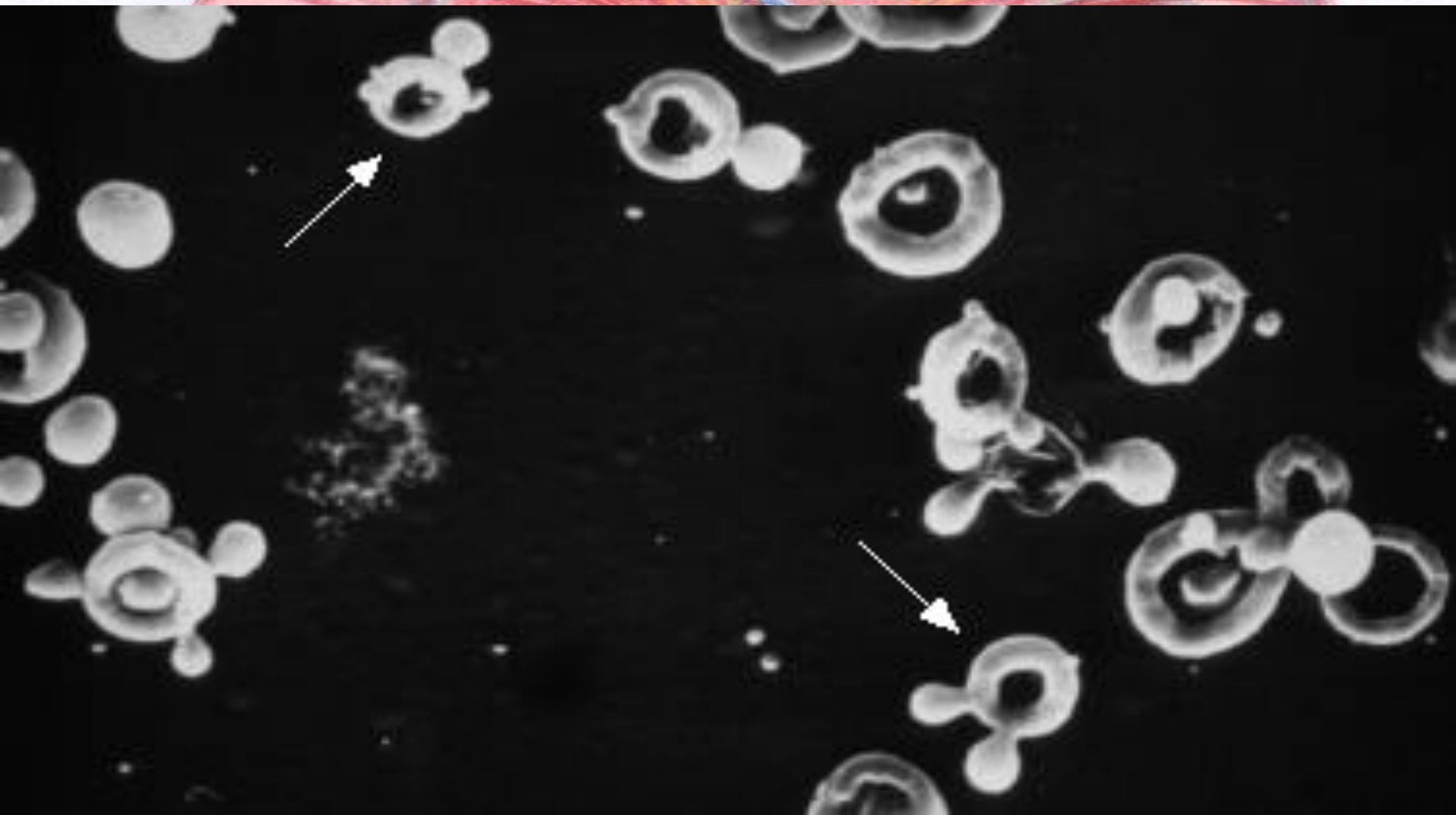
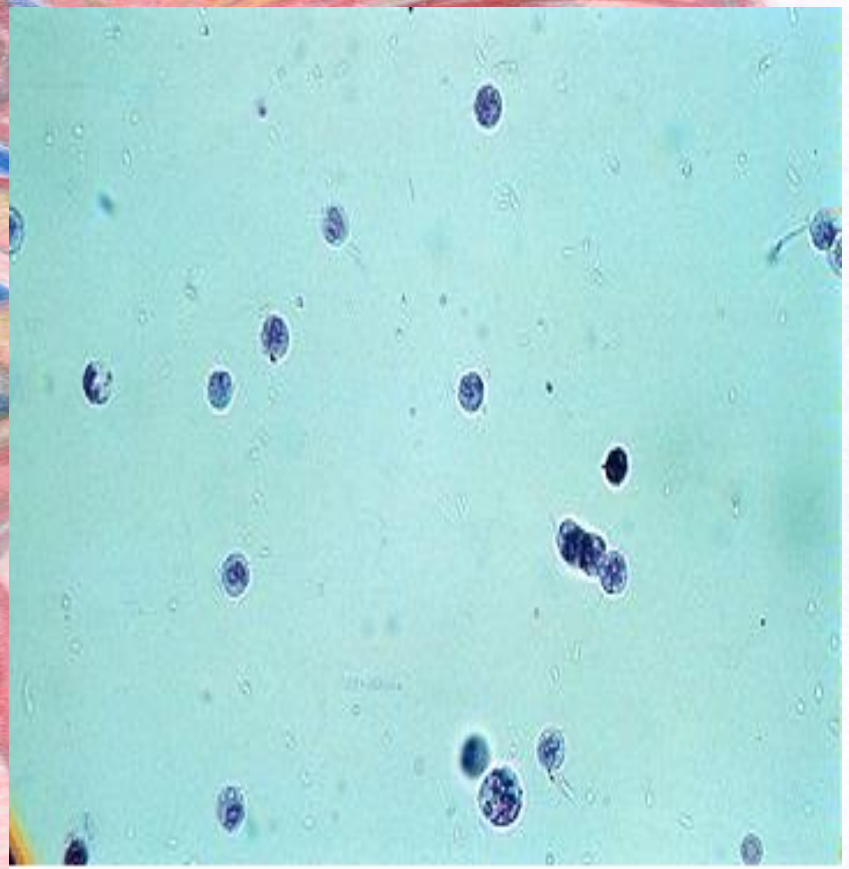
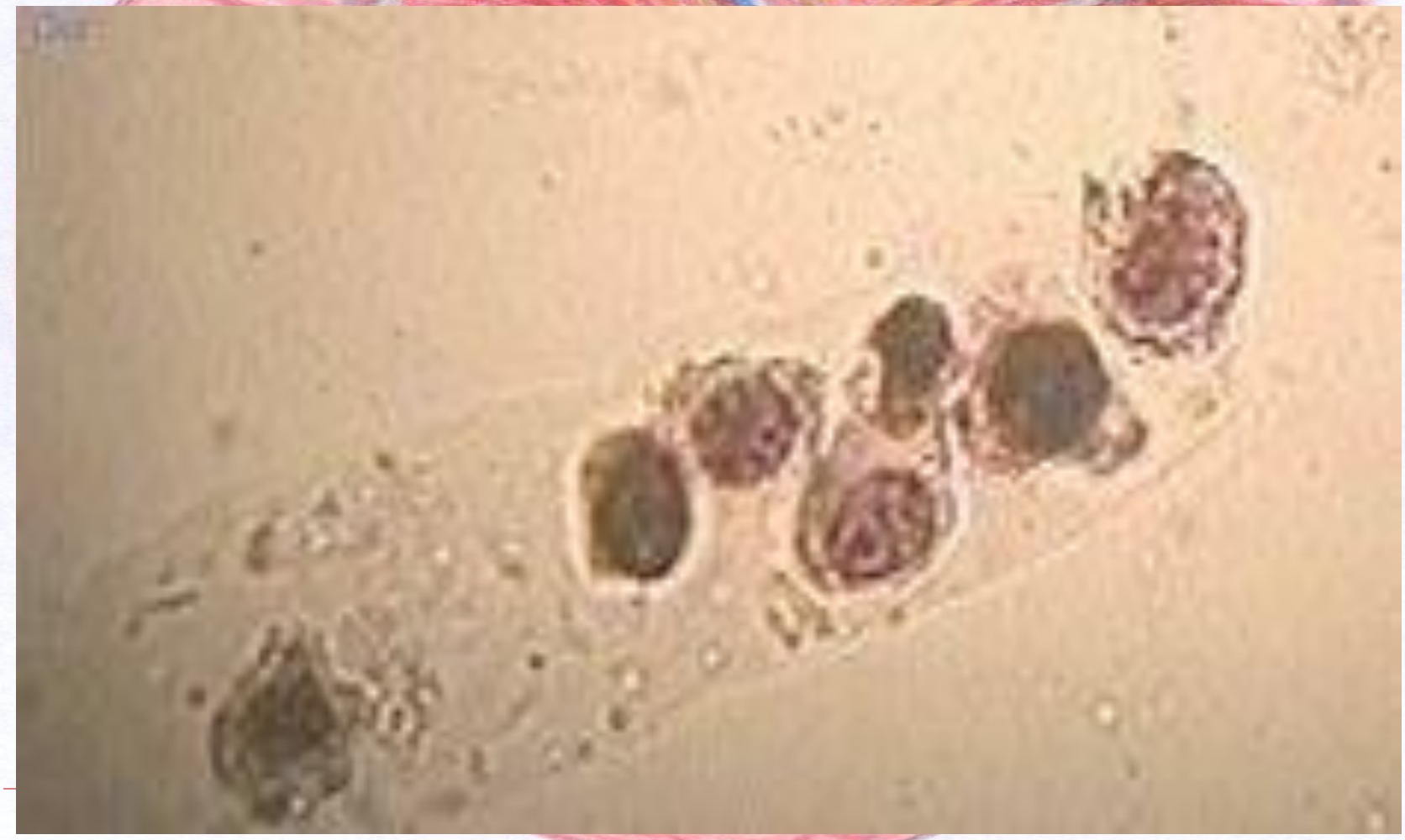


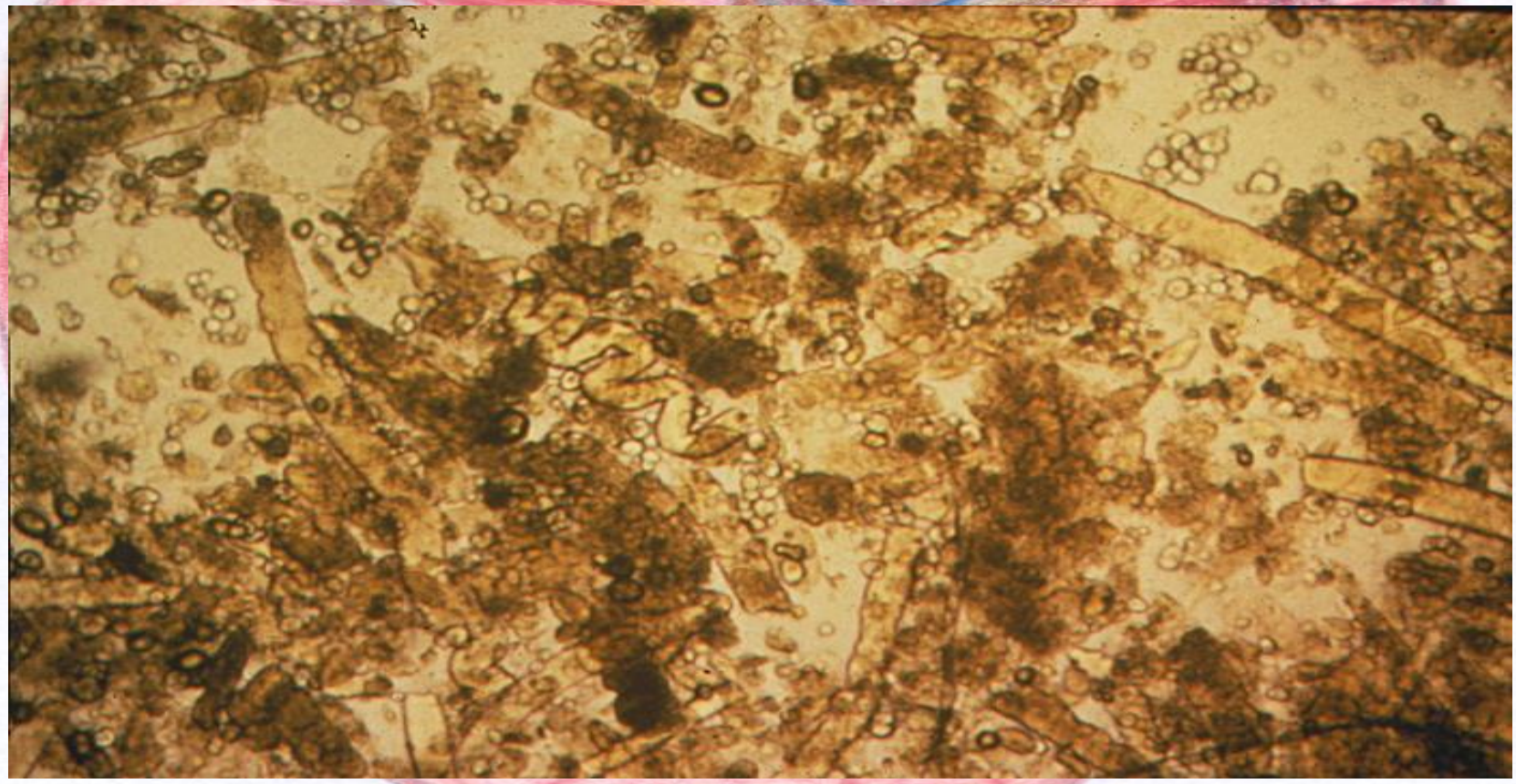
White Blood Cells



Renal Tubular Epithelial Cell Cast



Pigmented Granular Casts



Chapter 18: Acute Kidney Injury in the Elderly

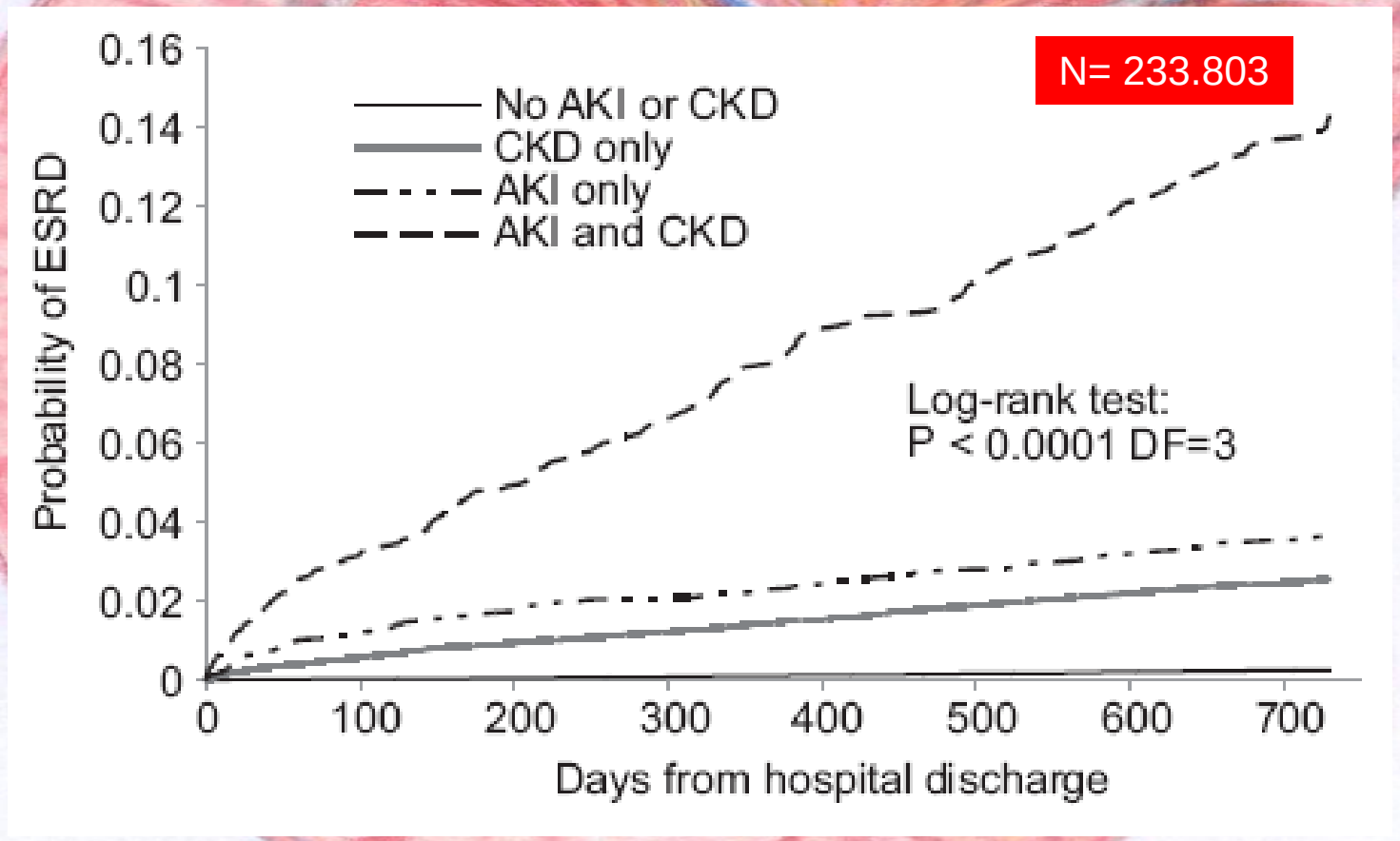
Mitchell H. Rosner

Division of Nephrology, University of Virginia Health System, Charlottesville, Virginia

TREATMENT OF THE ELDERLY WITH AKI

In general, the treatment of AKI in the elderly follows the same principles as for the general population. However, the decision to initiate dialytic support in the very elderly with multiple comorbidities and a very poor prognosis may be difficult. This is especially true for those individuals with significant baseline renal impairment where the likelihood of renal recovery may be low. The decision to initiate dialysis in these patients requires a coordinated discussion with family members, consulting physicians and other care providers.

Acute kidney injury increases risk of ESRD among elderly



Ishani A et al. *J Am Soc Nephrol* 2009

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Chapter 18: Acute Kidney Injury in the Elderly

Mitchell H. Rosner

Division of Nephrology, University of Virginia Health System, Charlottesville, Virginia

Table 1. General approaches for the prevention of AKI

- Avoidance of nephrotoxins
  - Recognition of potential nephrotoxic agents
  - Recognition of high risk patients and clinical settings
  - Avoidance of concomitant use of multiple nephrotoxins
  - Use of lowest dose and for shortest time possible
  - If applicable, monitoring of drug dose
  - Frequent monitoring of renal function
  - Maintain euvolemia
- Minimization of nosocomial infection
- Extracellular fluid expansion
  - (maintain good urine output, stable hemodynamics)
- Avoid agents that impair renal blood flow autoregulation
  - (NSAIDS, ACE inhibitors, ARBs)
- Pharmacologic interventions if applicable
- Use of computer surveillance systems
  - Identify high risk patients and medications
  - Determine correct dose for GFR

Chapter 18: Acute Kidney Injury in the Elderly

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Division of Nephrology, University of Virginia Health System, Charlottesville, Virginia

Table 2. Examples of specific renal protective strategies

Exposure	Strategy
Radiocontrast agents	Intravenous hydration (normal saline) Intravenous sodium bicarbonate (?) N-acetylcysteine Vitamin C Iso-osmolar contrast
Aminoglycoside antibiotics	Once-daily dosing Monitoring of drug levels
Tumor lysis (uric acid)	Allopurinol/rasburicase Intravenous hydration/urine alkalinization
Ethylene glycol ingestion	Ethanol/fomepizole Hemodialysis
Rhabdomyolysis	Intravenous hydration/urine alkalinization ± mannitol
Methotrexate	Intravenous hydration/urine alkalinization
Acyclovir	Intravenous hydration
Calcineurin inhibitors	Monitor drug levels ± calcium-channel blockers
Amphotericin B	Use of lipid formulation

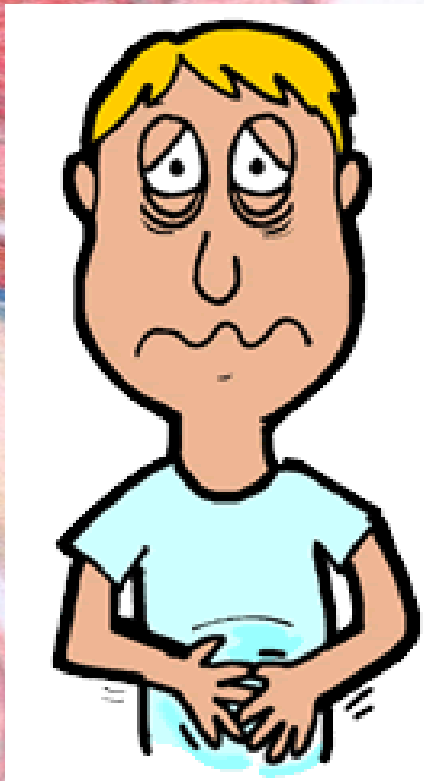
RECOVERY OF RENAL FUNCTION AND PROGNOSIS OF AKI IN THE ELDERLY

A recent systematic review and meta-analysis of recovery of kidney function after AKI in the elderly has shown that recovery after AKI is approximately 28% less likely to occur when the patient is older than 65 yr.[24] Whether these results are caused by the effects of advanced age on the kidney itself or the increased number of comorbidities (including baseline CKD) in the elderly is not certain. Long-term recovery is also less likely and it is believed that AKI in elderly more often results in CKD.[24] The lower likelihood of renal recovery in the elderly may be due to the effects of aging to impair the capacity for kidney repair.[25] The capacity for renal epithelial cell proliferation declines with aging as does the function of progenitor and stem cells that are critical for tubular repair.[25]

Several other individual studies have not been able to show that age is specifically associated with impaired renal recovery.[18] Thus, in the individual patient, it may not be clear if age is an independent predictor of a poor prognosis and other comorbid conditions may play a more important role in driving the risk for poor outcomes.[26]

Conclusion

For numerous reasons, elderly patients are at higher risk for the development of AKI, and certain causes of AKI are more commonly seen in this group. In fact, there are more often multiple etiologies of AKI in this age group. Diagnostic approaches to AKI should focus on the most likely etiologies. Because of multiple factors, the likelihood of complete renal recovery is impaired in this group.



Chapter 18: Acute Kidney Injury in the Elderly

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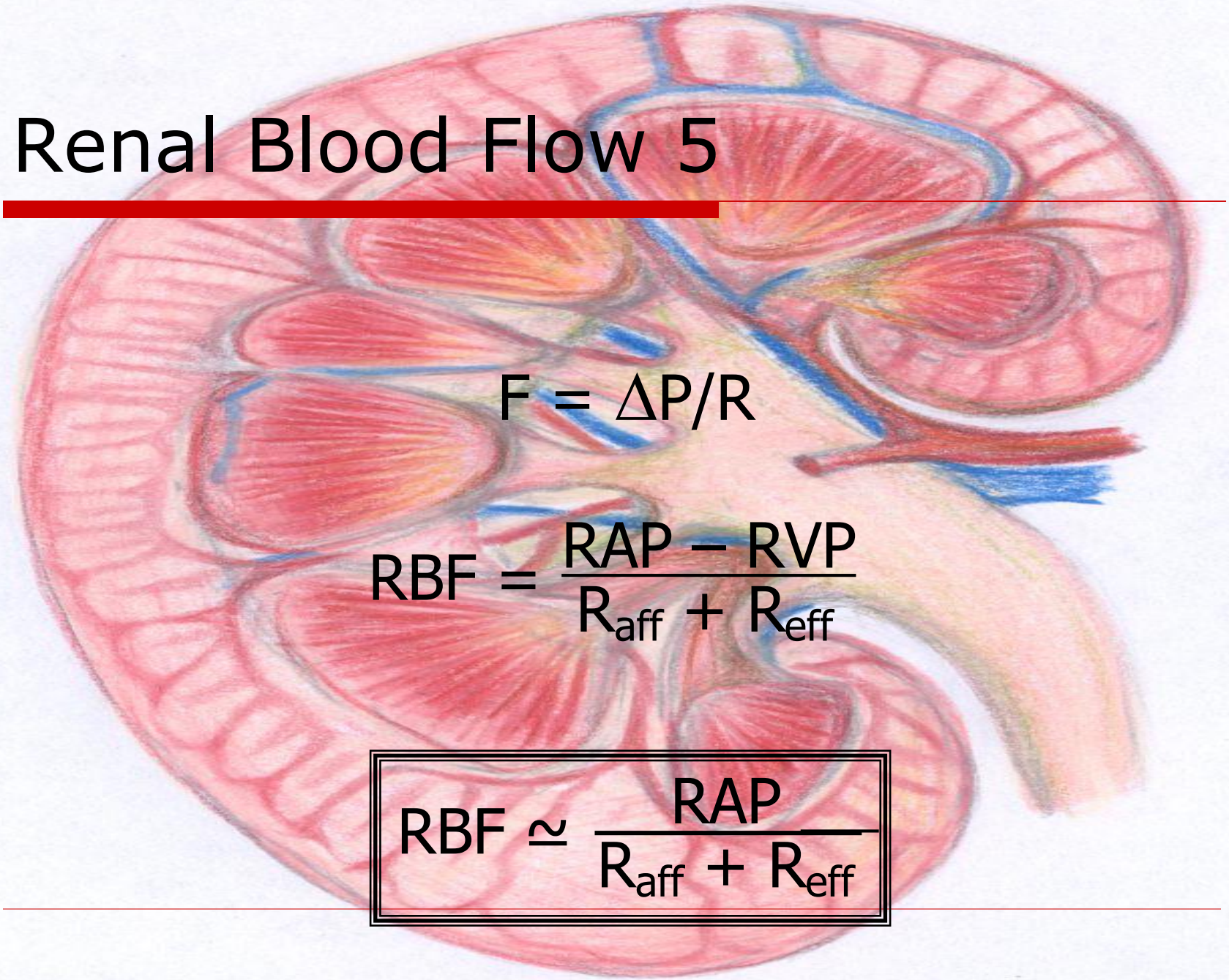
The only organ with entry and exit arteries

Renal Blood Flow 5

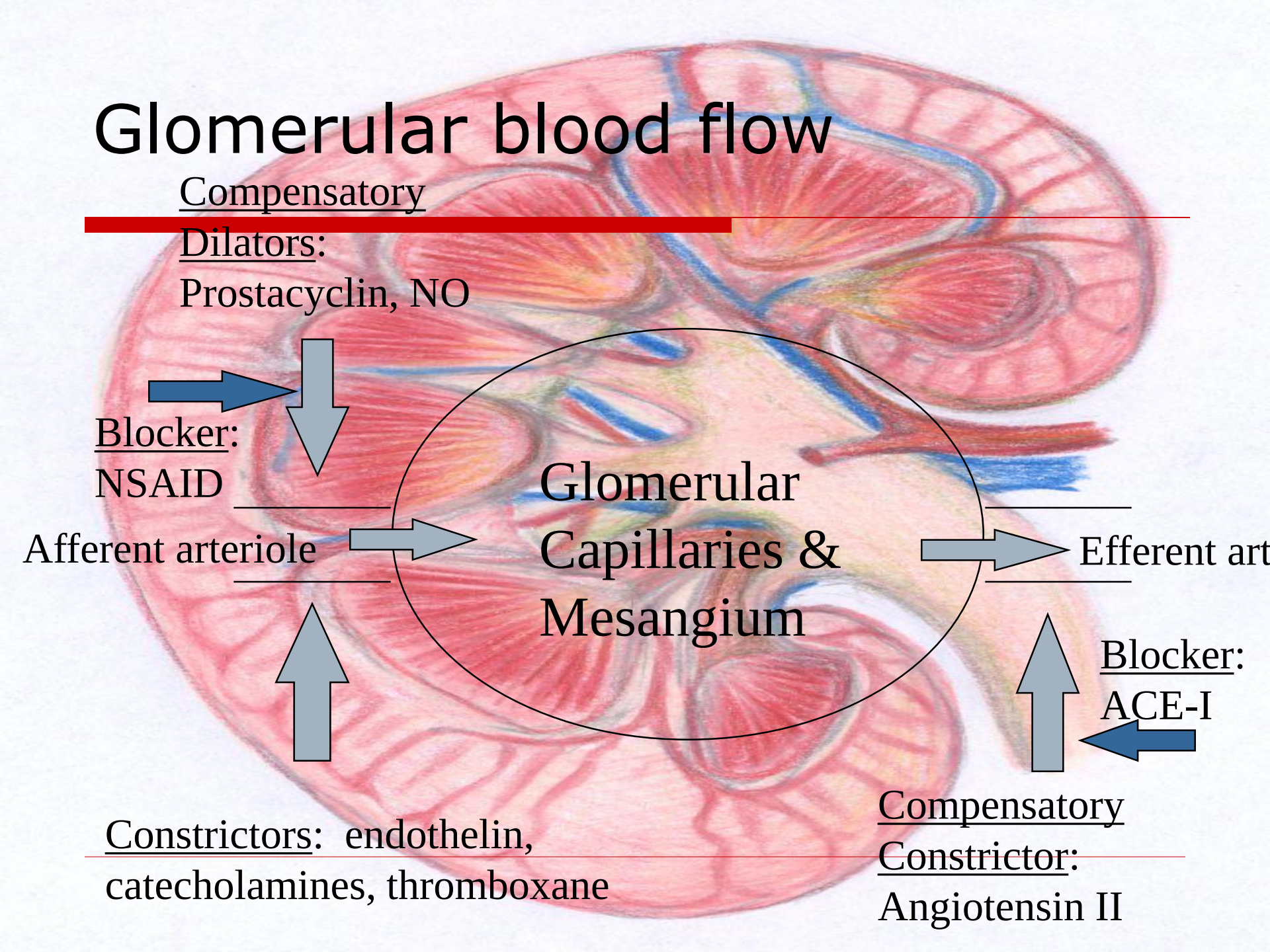


$$F = \Delta P / R$$

$$RBF = \frac{RAP - RVP}{R_{aff} + R_{eff}}$$

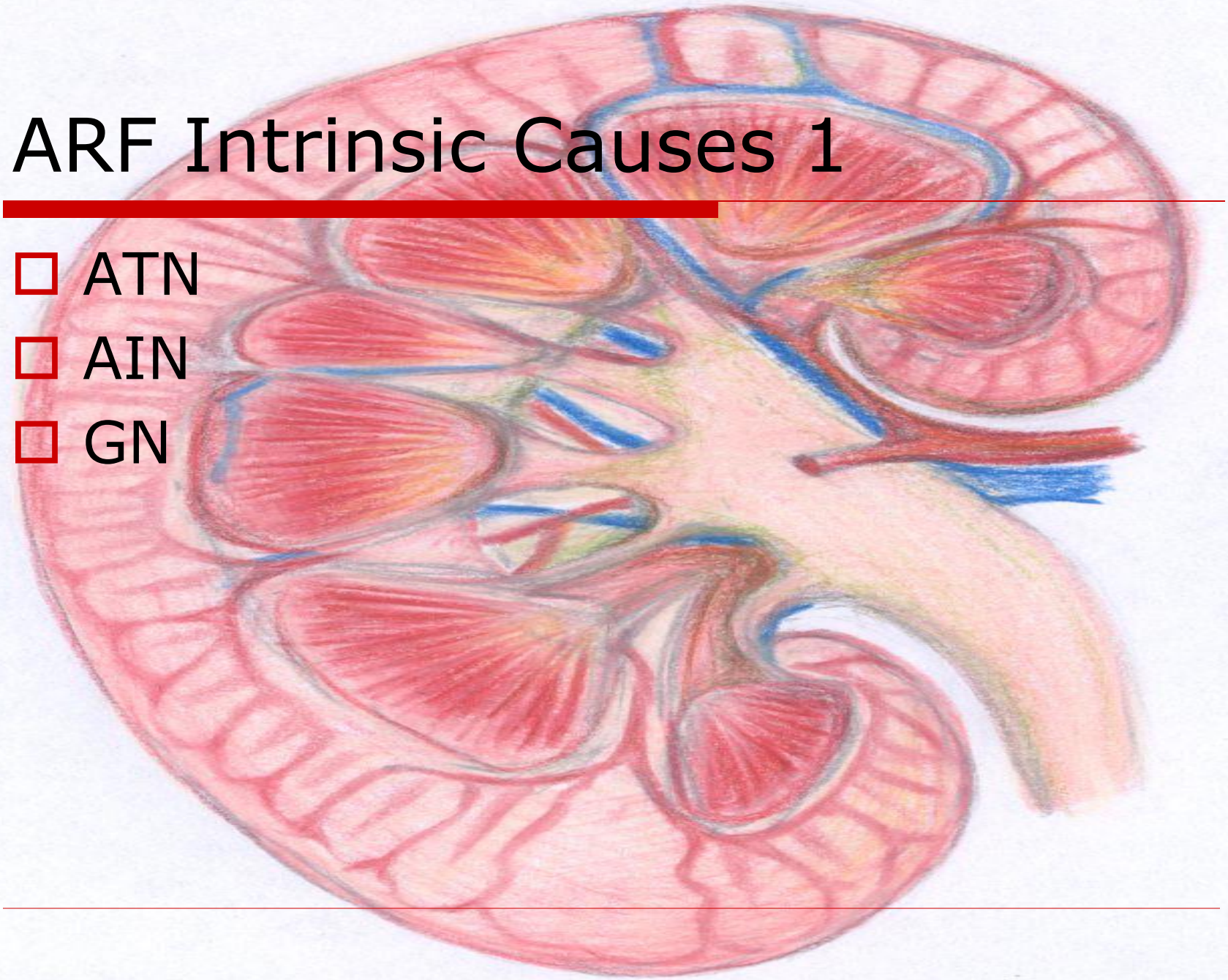
$$RBF \simeq \frac{RAP}{R_{aff} + R_{eff}}$$

Glomerular blood flow

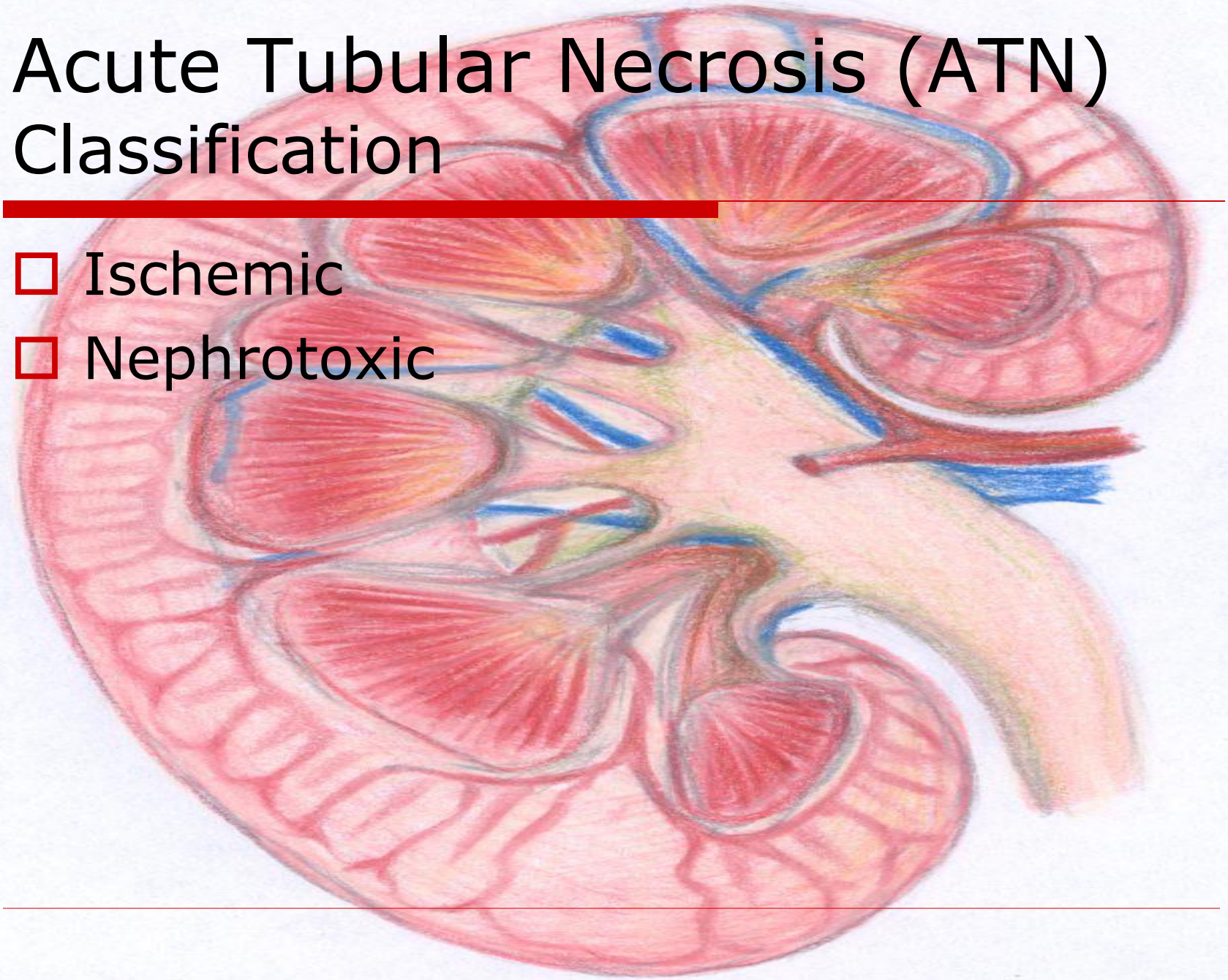


ARF Intrinsic Causes 1

- ATN
- AIN
- GN

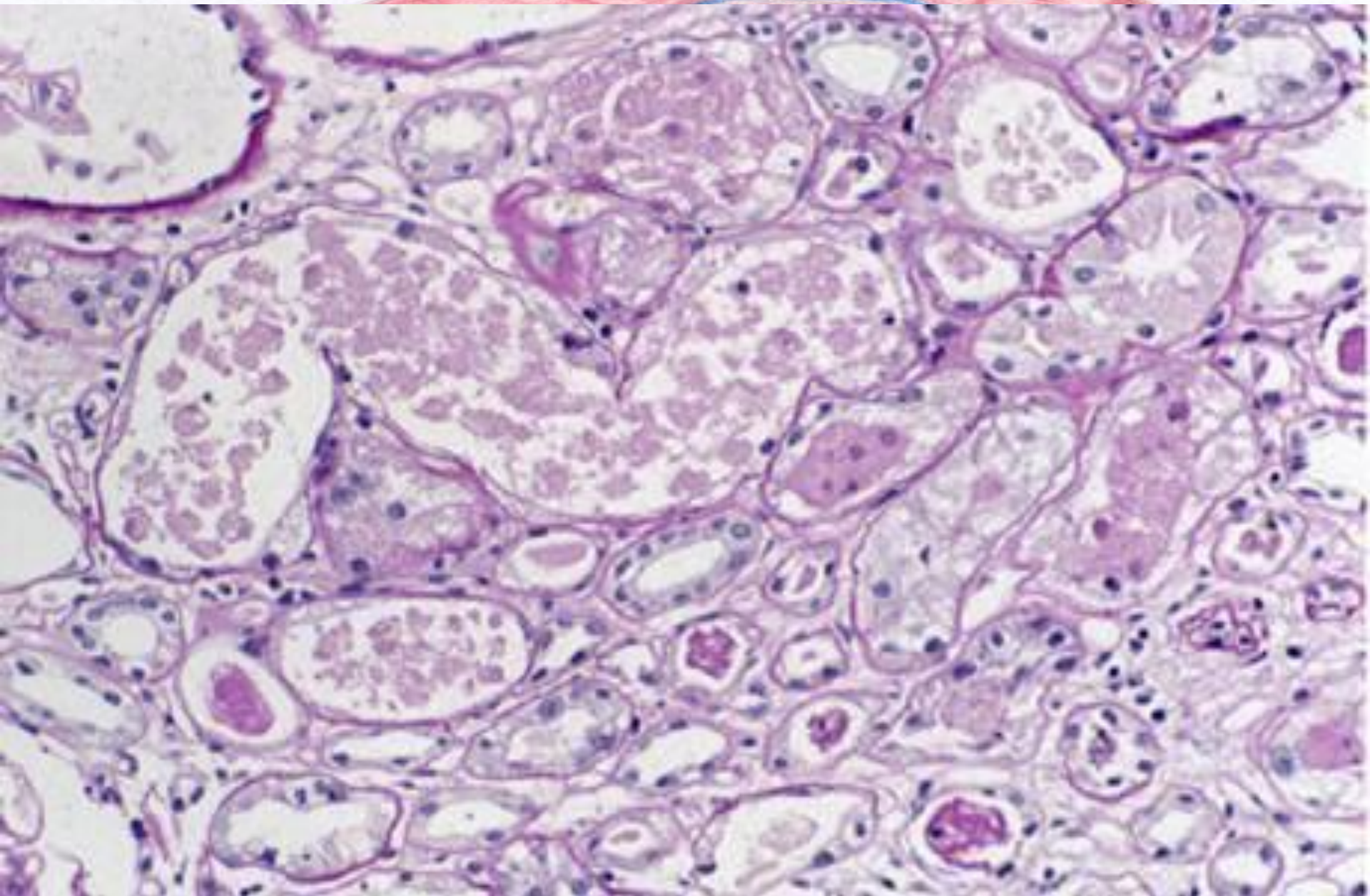


Acute Tubular Necrosis (ATN)
Classification



- Ischemic
- Nephrotoxic

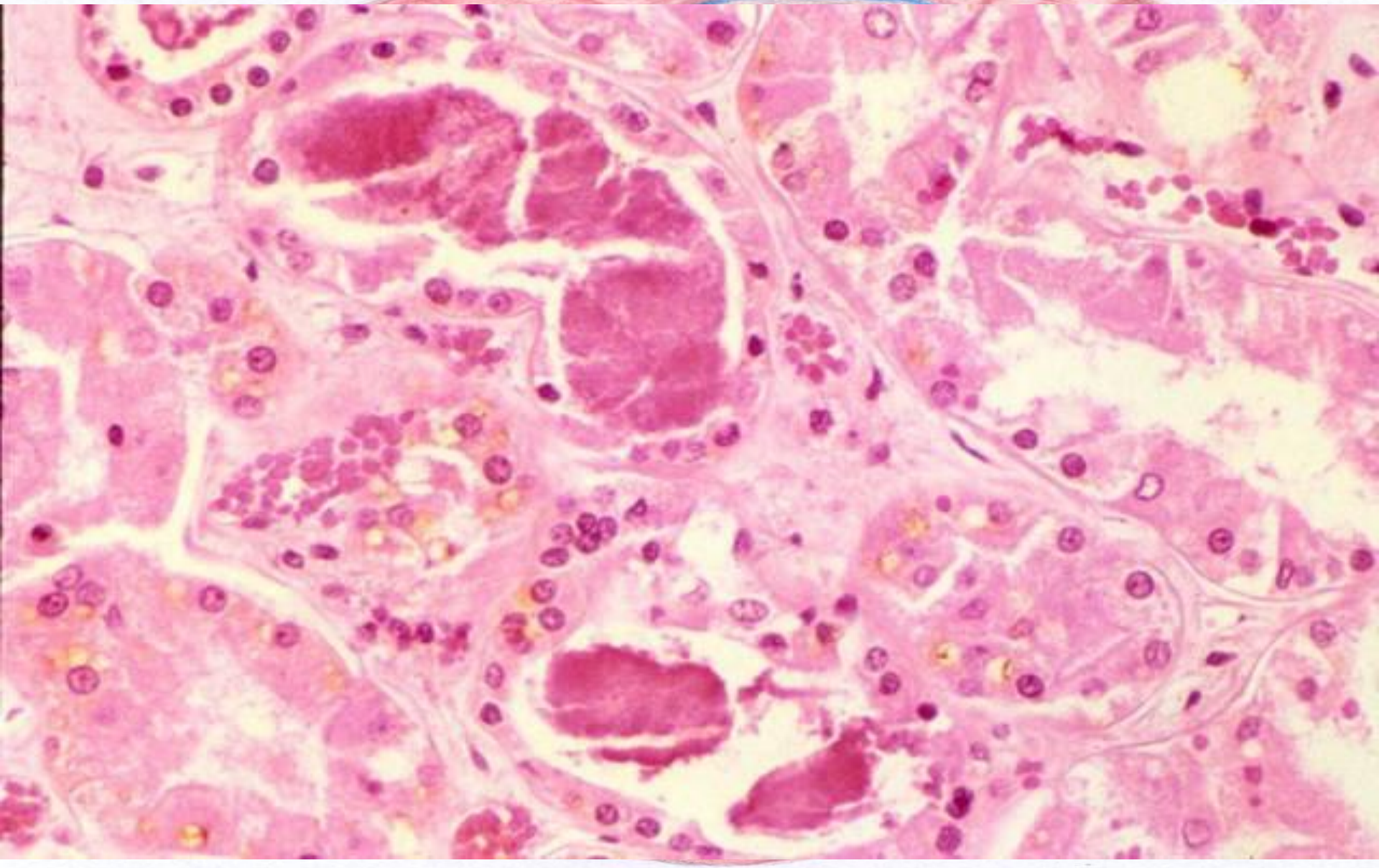
ATN



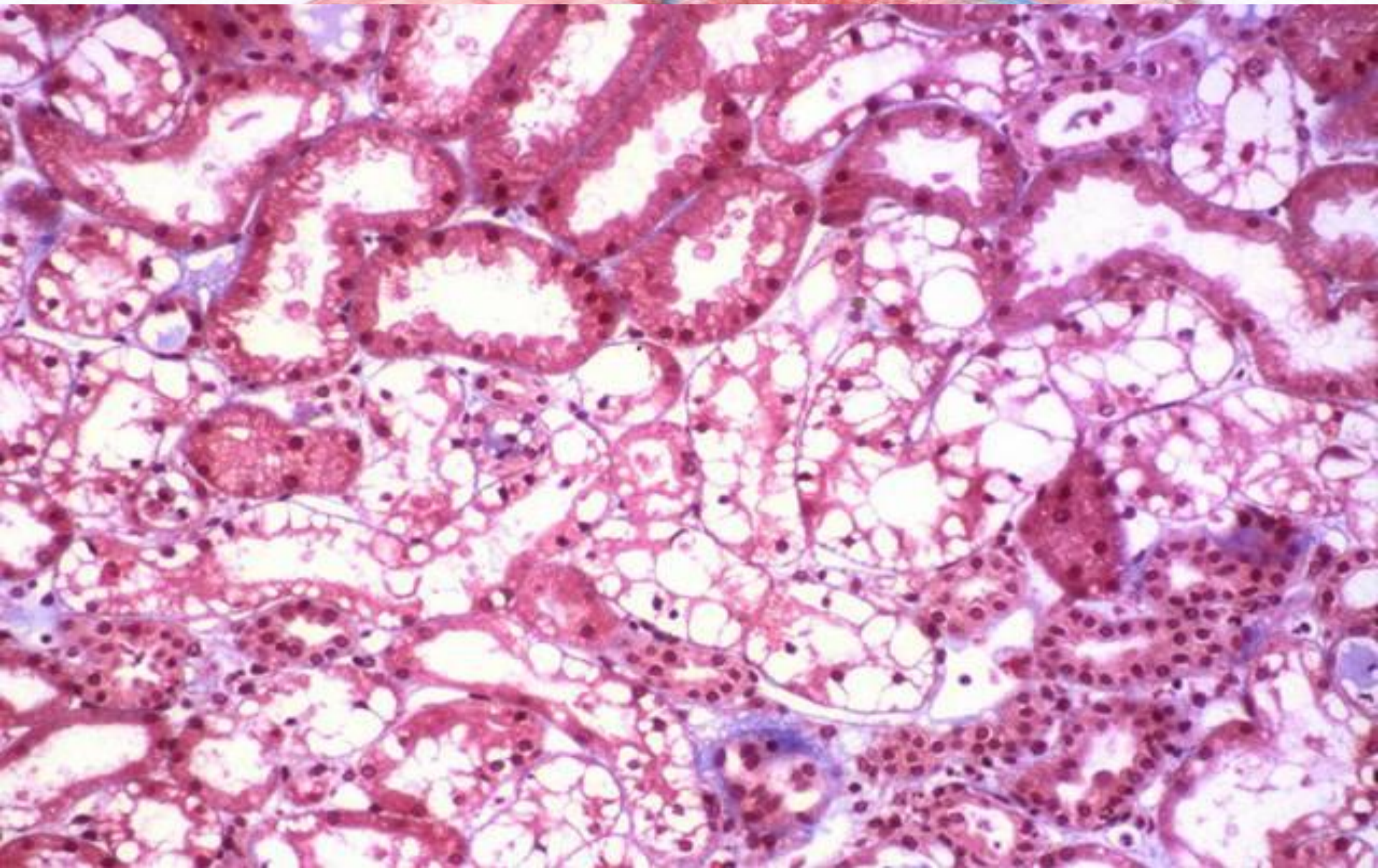
Acute Renal Failure
Nephrotoxic ATN

- Endogenous Toxins
  - Heme pigments (myoglobin, hemoglobin)
  - Myeloma light chains
- Exogenous Toxins
  - Antibiotics (e.g., aminoglycosides, amphotericin B)
  - Radiocontrast agents
  - Heavy metals (e.g., cis-platinum, mercury)
  - Poisons (e.g., ethylene glycol)

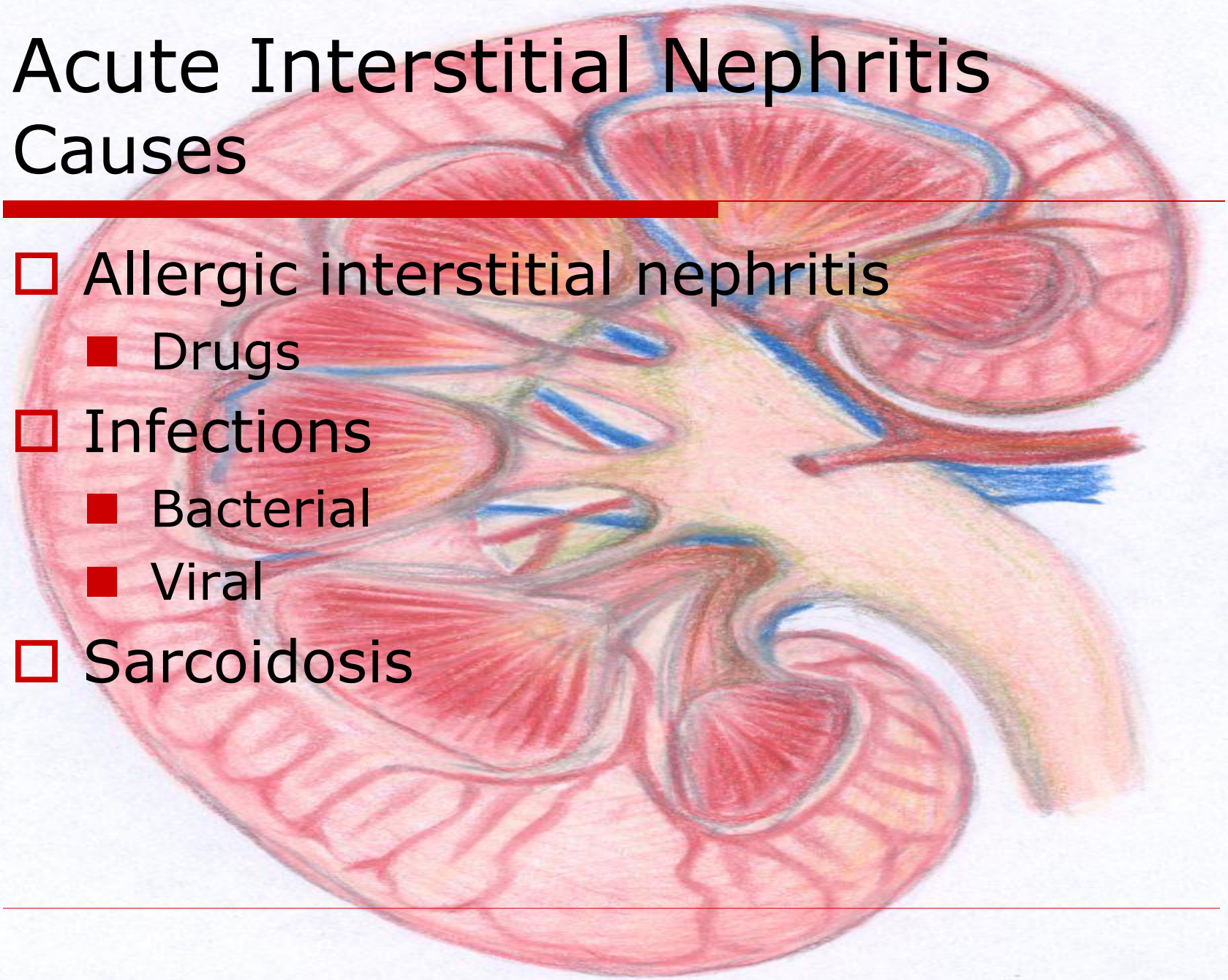
ATN



ATN

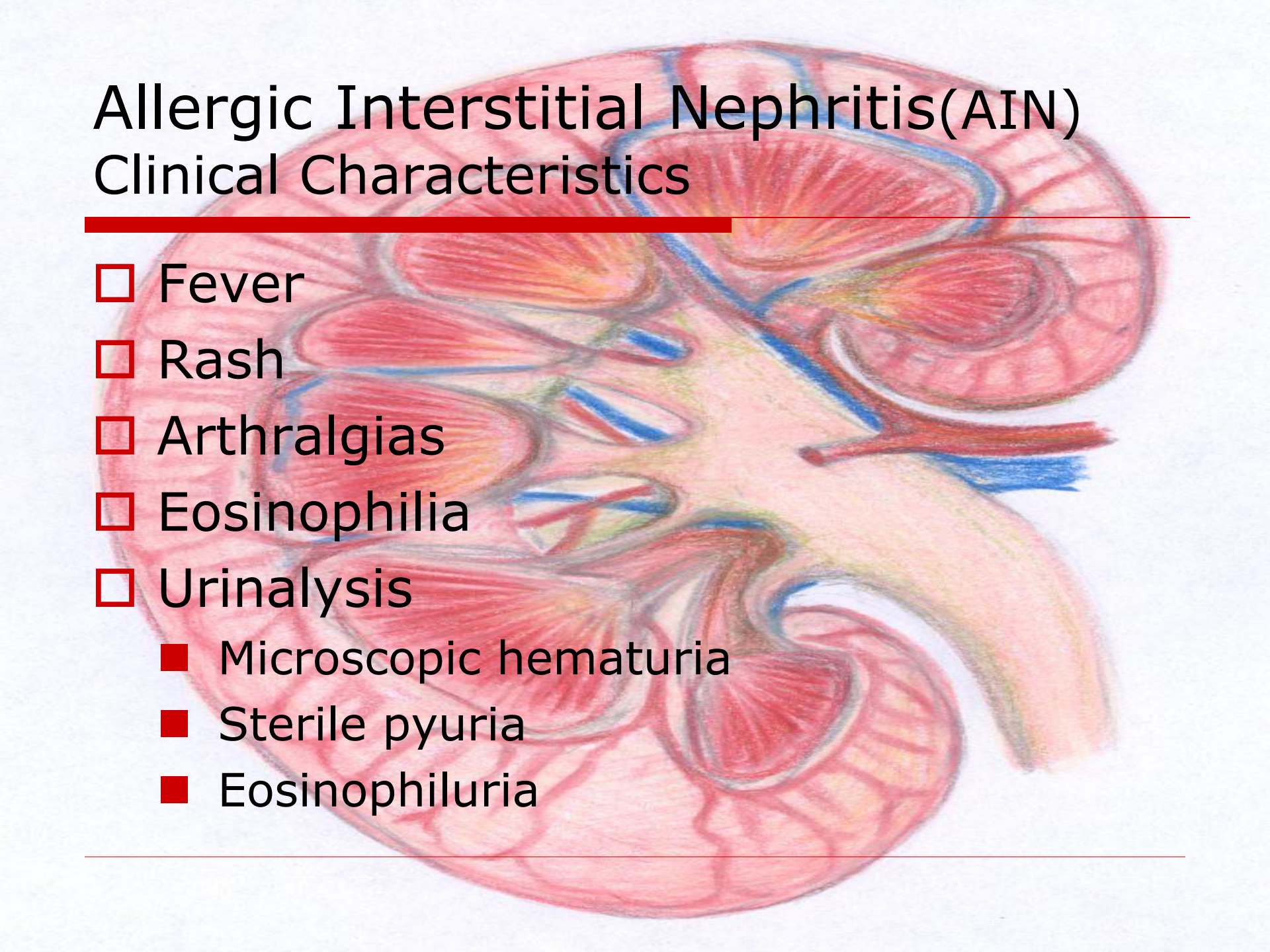


Acute Interstitial Nephritis Causes



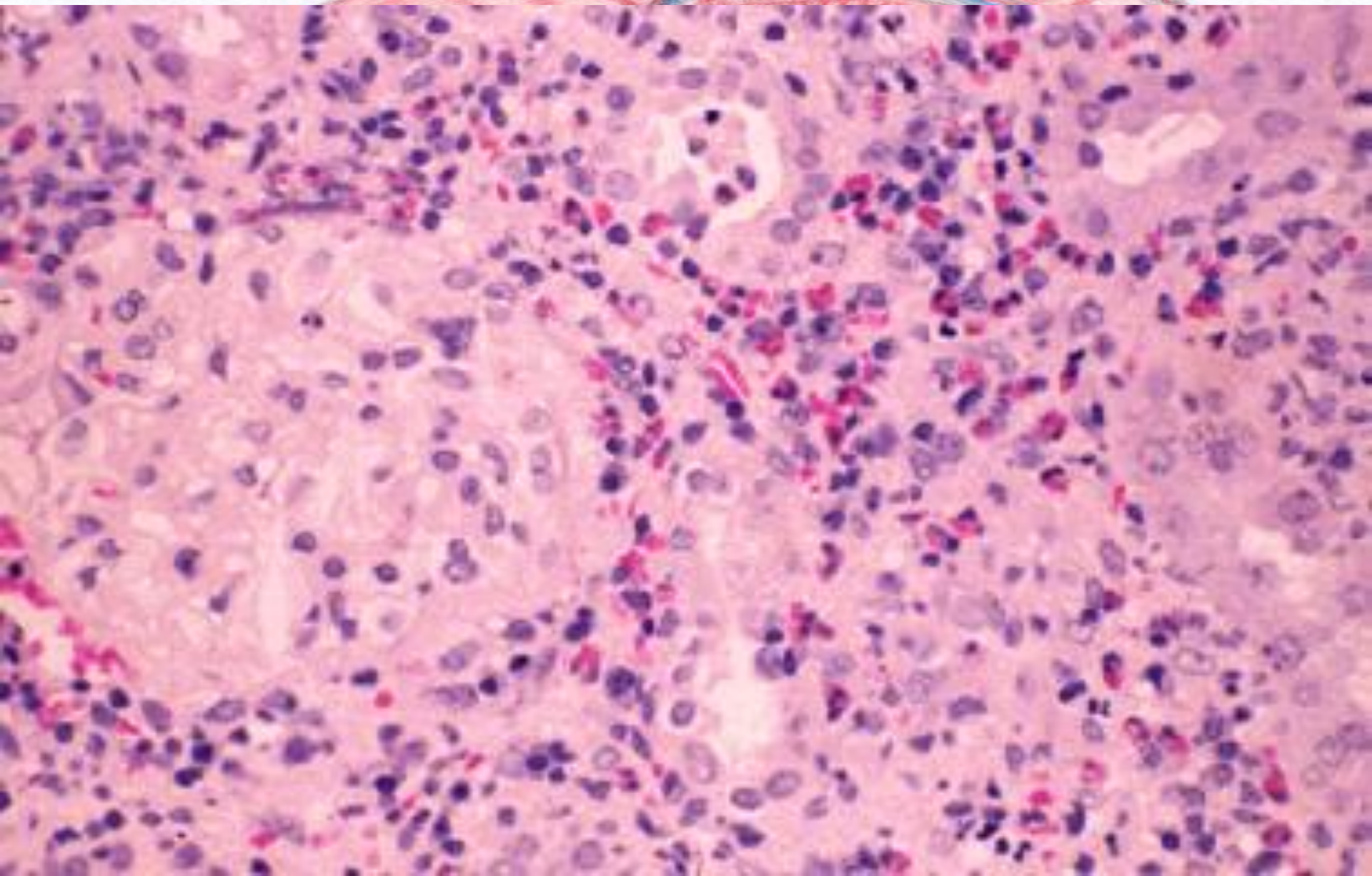
- Allergic interstitial nephritis
  - Drugs
- Infections
  - Bacterial
  - Viral
- Sarcoidosis

Allergic Interstitial Nephritis(AIN)
Clinical Characteristics

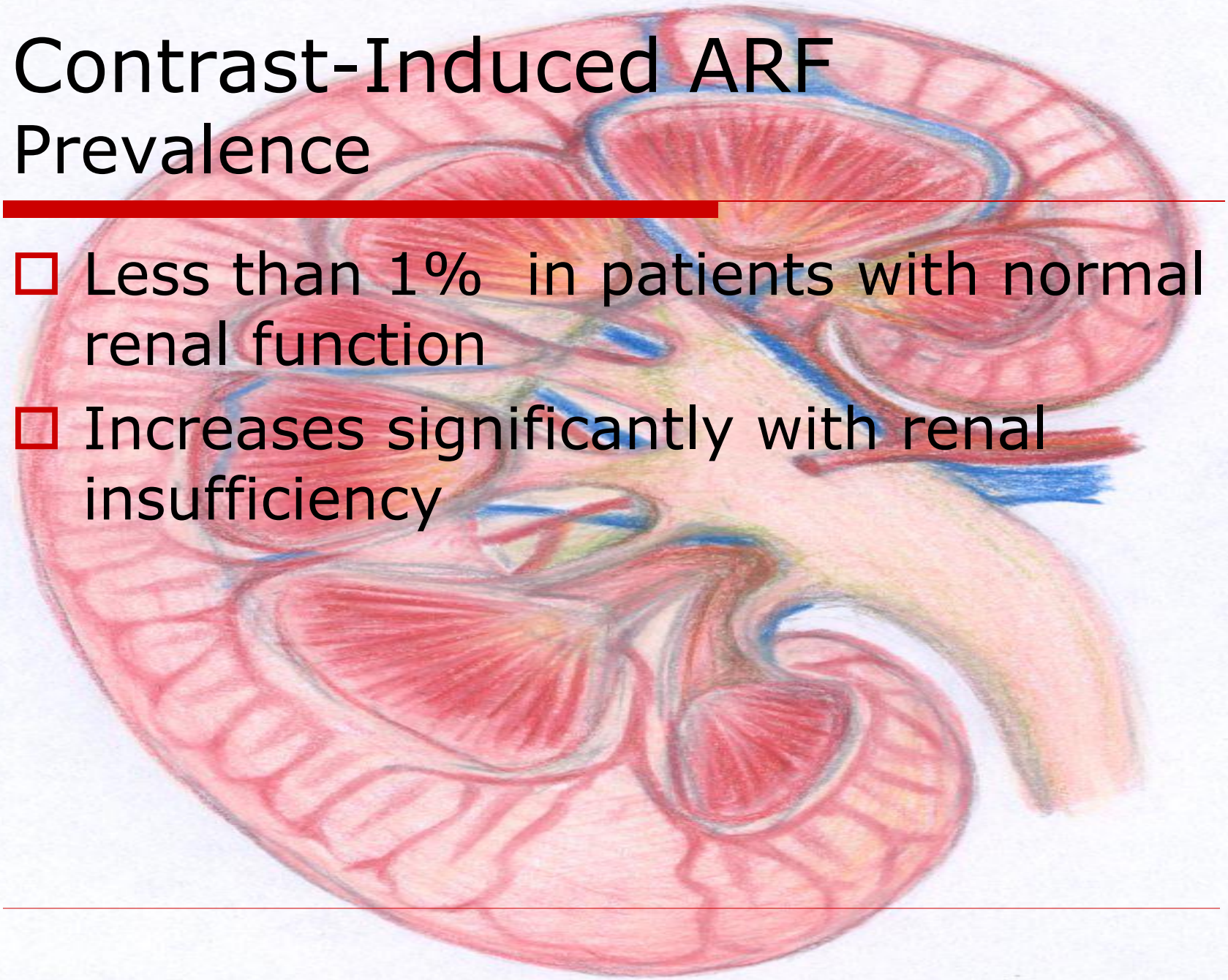


- Fever
- Rash
- Arthralgias
- Eosinophilia
- Urinalysis
  - Microscopic hematuria
  - Sterile pyuria
  - Eosinophiluria

AIN

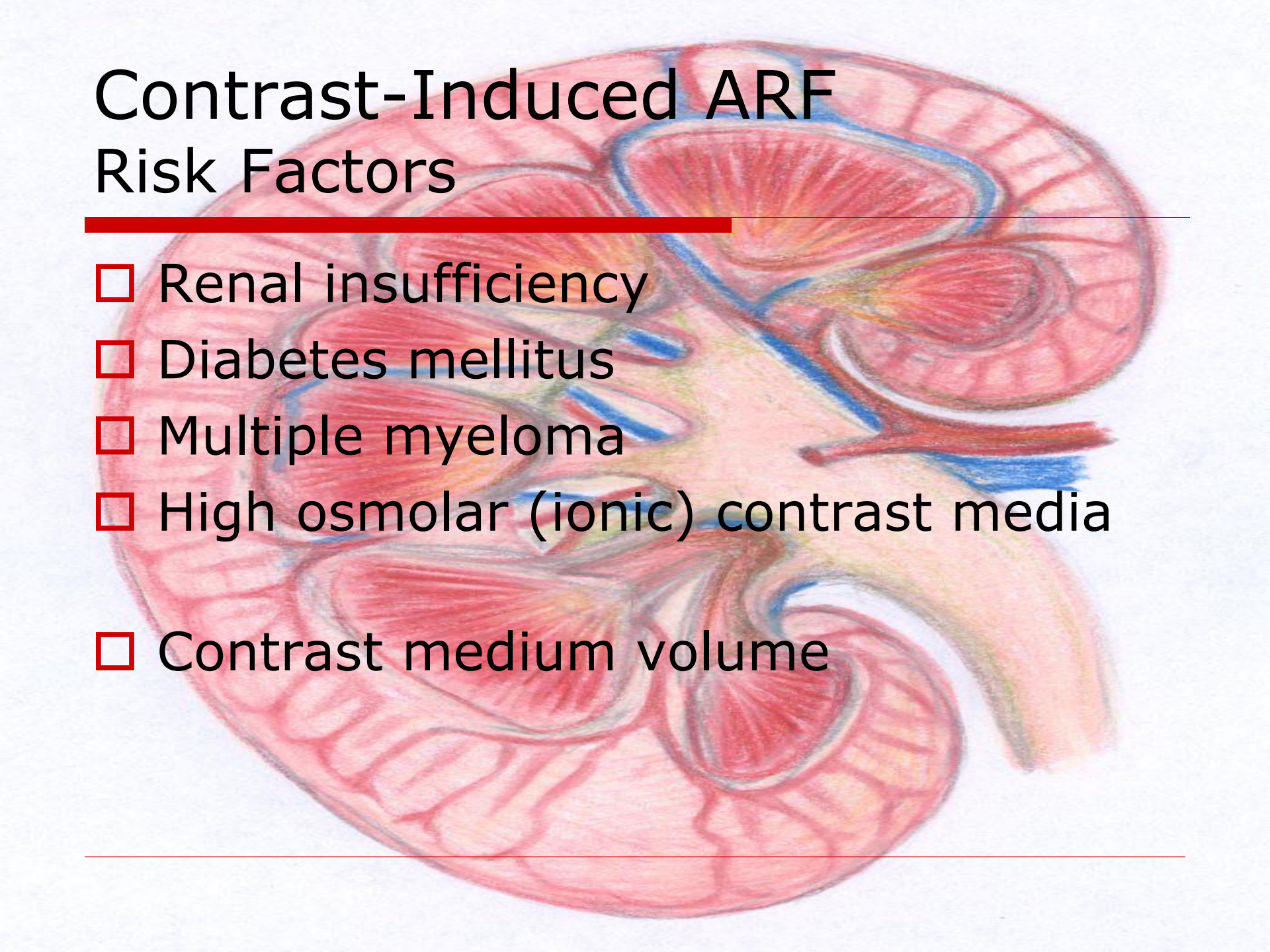


Contrast-Induced ARF
Prevalence



- Less than 1% in patients with normal renal function
- Increases significantly with renal insufficiency

Contrast-Induced ARF
Risk Factors



- Renal insufficiency
- Diabetes mellitus
- Multiple myeloma
- High osmolar (ionic) contrast media

- Contrast medium volume

Contrast-induced ARF
Clinical Characteristics

- Onset - 24 to 48 hrs after exposure
- Duration - 5 to 7 days
- Non-oliguric (majority)
- Dialysis - rarely needed
- Urinary sediment - variable
- Low fractional excretion of Na

Pre-Procedure Prophylaxis

1. IV Fluid (N/S)

1-1.5 ml/kg/hour x12 hours prior to procedure and 6-12 hours after

2. Mucomyst (N-acetylcysteine)

Free radical scavenger; prevents oxidative tissue damage 600mg po bd x 4 doses (2 before procedure, 2 after)

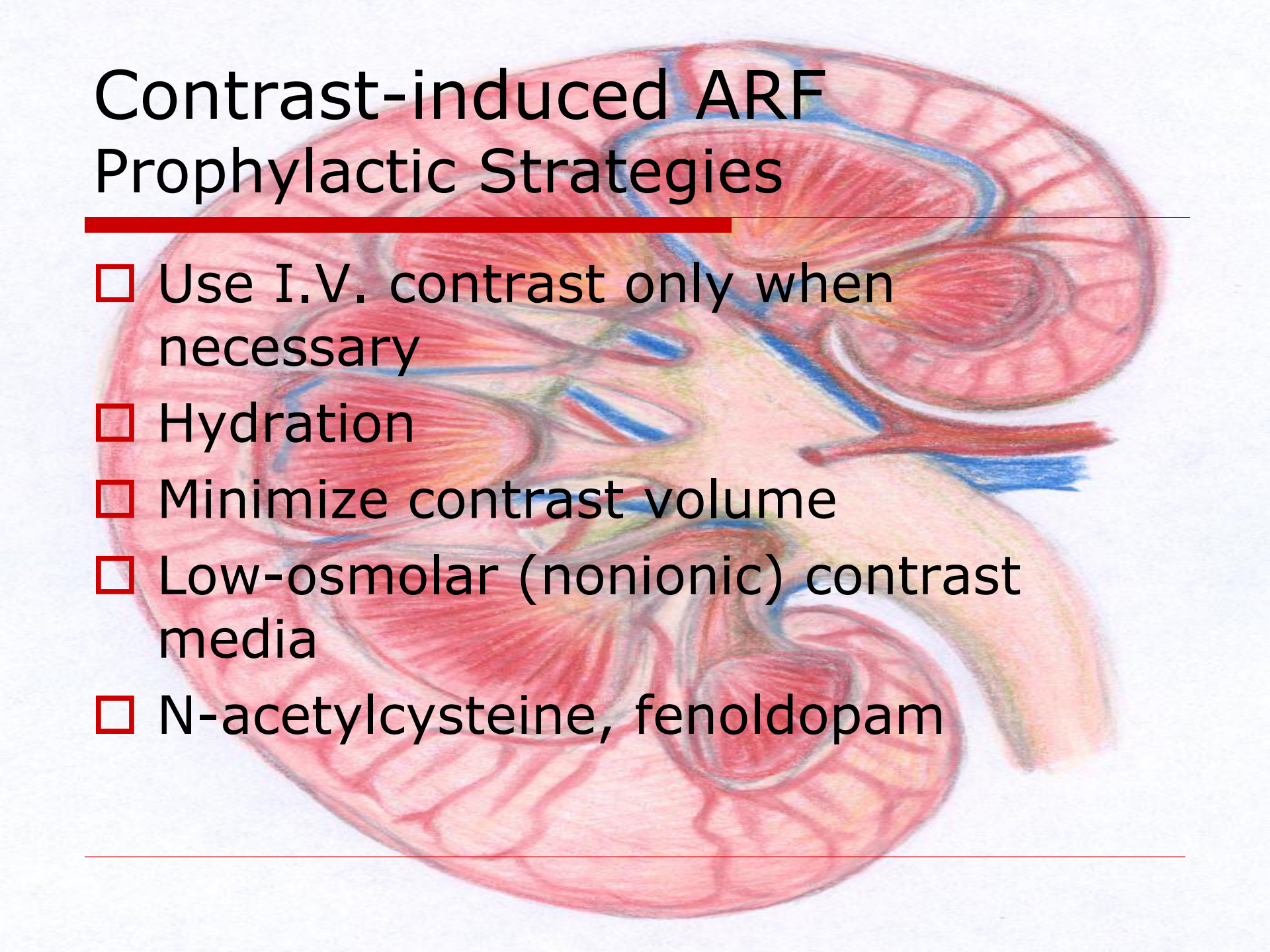
3. Bicarbonate (JAMA 2004)

Alkalinizing urine should reduce renal medullary damage

5% dextrose with 3 amps HCO3; bolus 3.5 mL/kg 1 hour preprocedure, then 1mL/kg/hour for 6 hours postprocedure

4. Possibly helpful? Fenoldopam, Dopamine
5. Not helpful! Diuretics, Mannitol

Contrast-induced ARF
Prophylactic Strategies



- Use I.V. contrast only when necessary
- Hydration
- Minimize contrast volume
- Low-osmolar (nonionic) contrast media
- N-acetylcysteine, fenoldopam

Classification of Glomerular Disease According to Clinical Features

Focal glomerulonephritis – Active urine sediment without renal insufficiency or nephrotic syndrome

- Less than 15 years – Mild postinfectious glomerulonephritis, IgA nephropathy, thin basement membrane disease, hereditary nephritis, Henoch-Schönlein purpura, mesangial proliferative glomerulonephritis
- 15 to 40 years – IgA nephropathy, thin basement membrane disease, lupus, hereditary nephritis, mesangial proliferative glomerulonephritis
- Greater than 40 years – IgA nephropathy

Diffuse glomerulonephritis – Active urine sediment with renal insufficiency and variable proteinuria, which can include nephrotic syndrome

- Less than 15 years – Postinfectious glomerulonephritis, membranoproliferative glomerulonephritis
- 15 to 40 years – Postinfectious glomerulonephritis, lupus, rapidly progressive glomerulonephritis, fibrillary glomerulonephritis, membranoproliferative glomerulonephritis
- Greater than 40 years – Rapidly progressive glomerulonephritis, vasculitis (including mixed cryoglobulinemia), fibrillary glomerulonephritis, postinfectious glomerulonephritis

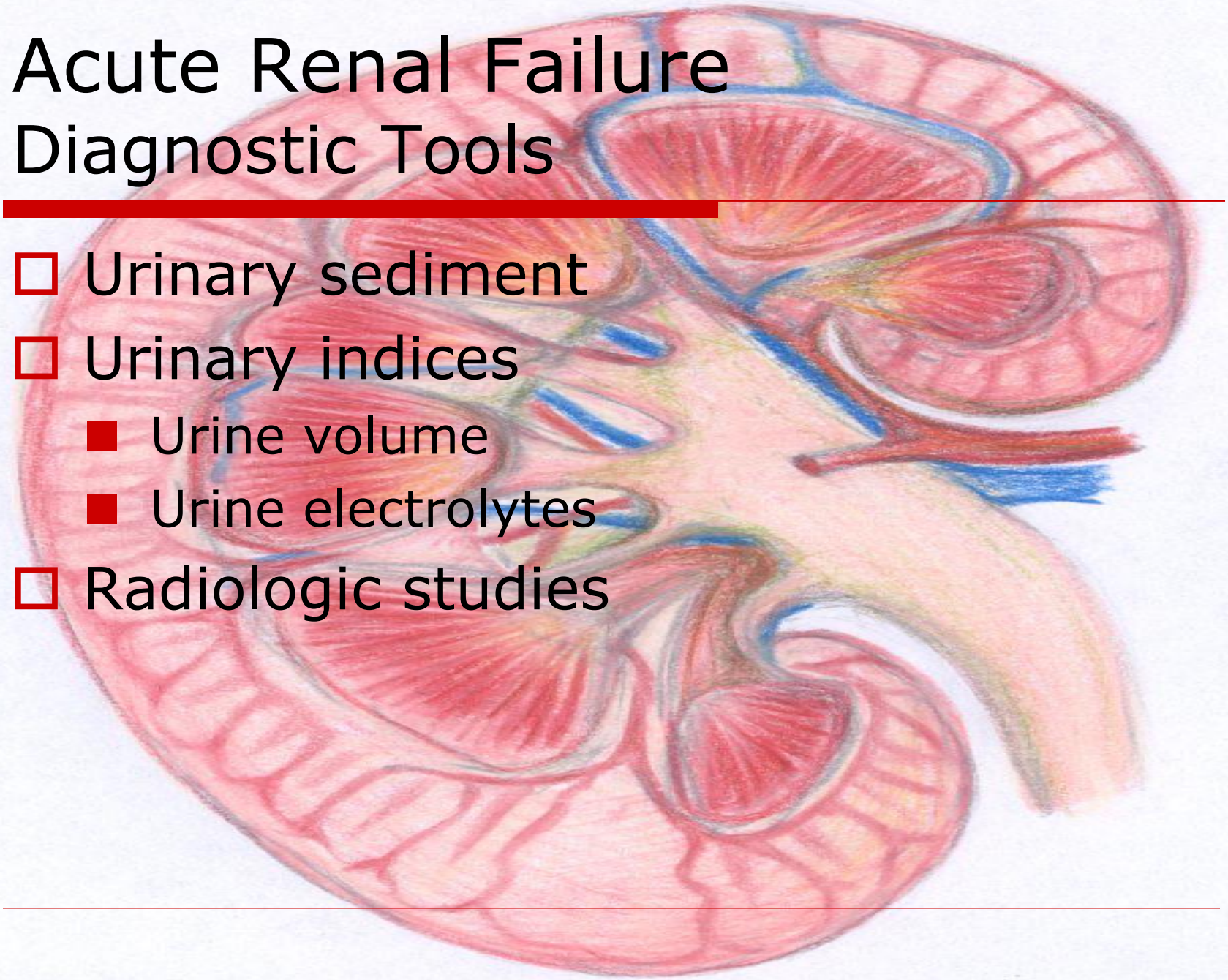
Nephrotic syndrome – Heavy proteinuria, bland sediment although some hematuria allowed

- Less than 15 years – Minimal change disease, focal glomerulosclerosis, mesangial proliferative glomerulonephritis
- 15 to 40 years – Focal glomerulosclerosis, minimal change disease, membranous nephropathy (including lupus), diabetic nephropathy, preeclampsia, postinfectious glomerulonephritis (later stage)
- Greater than 40 years – Focal glomerulosclerosis, membranous nephropathy, diabetic nephropathy, minimal change disease, IgA nephropathy, primary amyloidosis or the related disorder light chain deposition disease (which can account for 15 to 20 percent of cases in patients over the age of 60), benign nephrosclerosis, postinfectious glomerulonephritis (later stage)

ARF Post-renal Causes 1

- Intra-renal Obstruction
  - Acute uric acid nephropathy
  - Drugs (e.g., acyclovir)
- Extra-renal Obstruction
  - Renal pelvis or ureter (e.g., stones, clots, tumors, papillary necrosis, retroperitoneal fibrosis)
  - Bladder (e.g., BPH, neuropathic bladder)
  - Urethra (e.g., stricture)

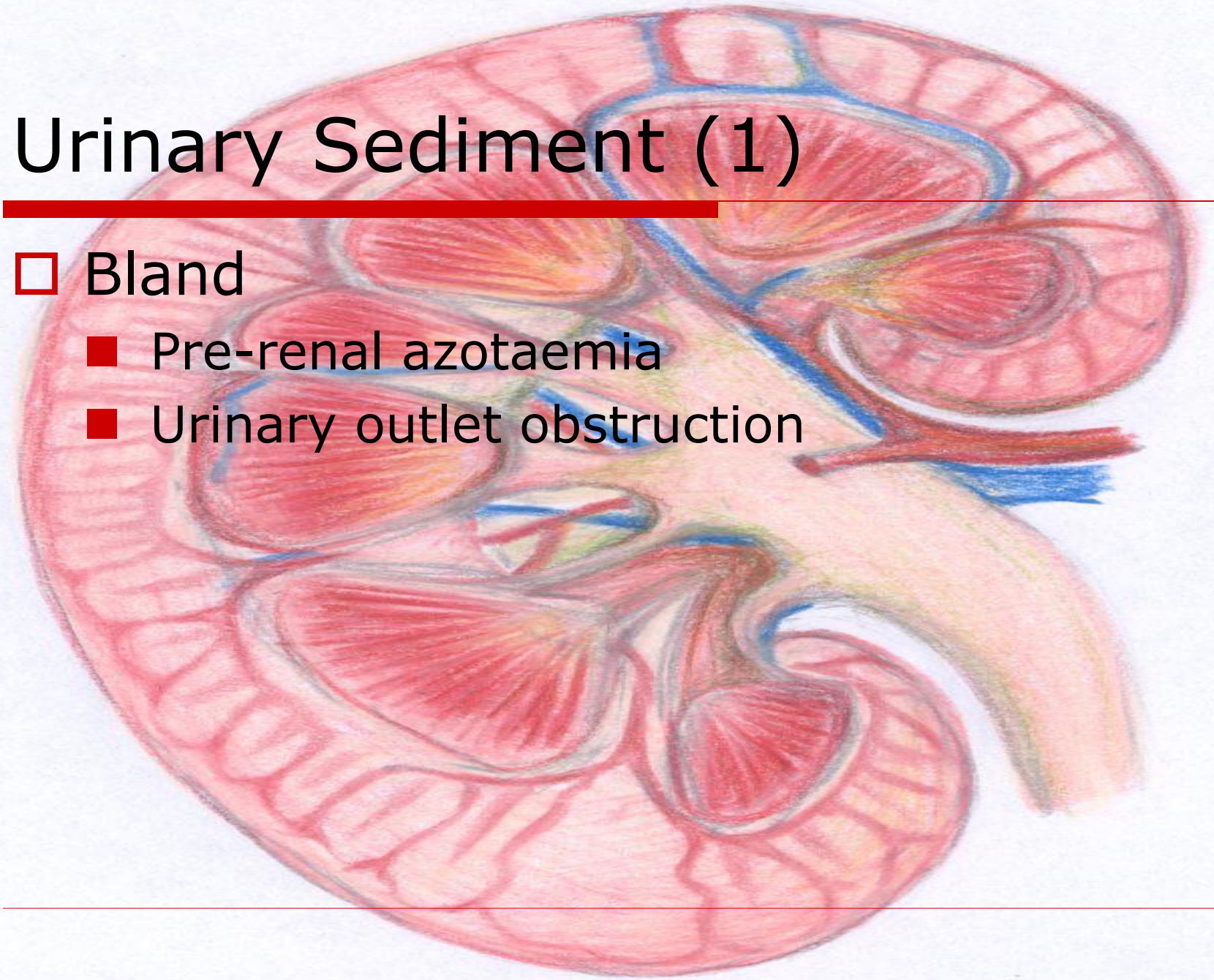
Acute Renal Failure
Diagnostic Tools



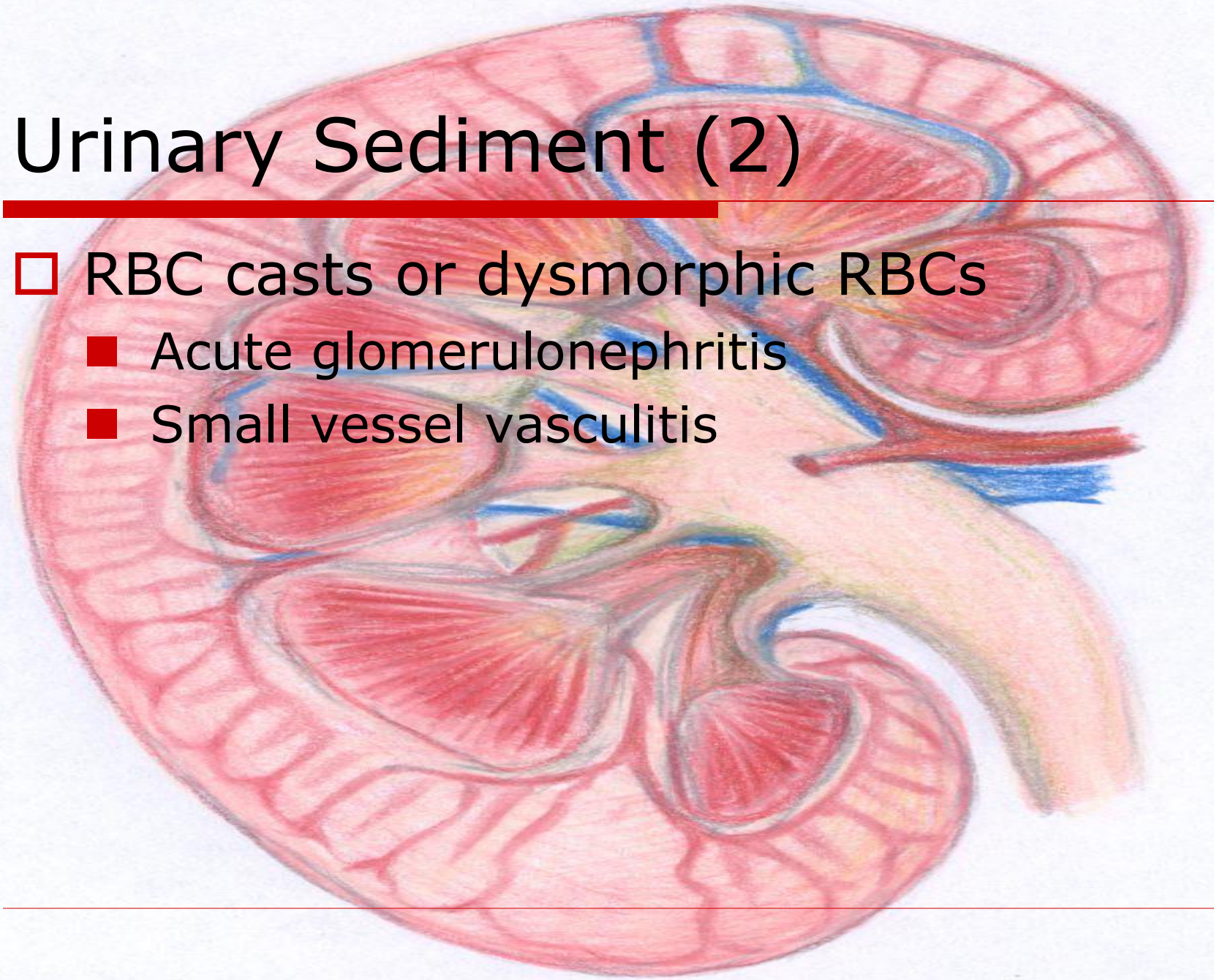
- Urinary sediment
- Urinary indices
  - Urine volume
  - Urine electrolytes
- Radiologic studies

Urinary Sediment (1)

- Bland
  - Pre-renal azotaemia
  - Urinary outlet obstruction

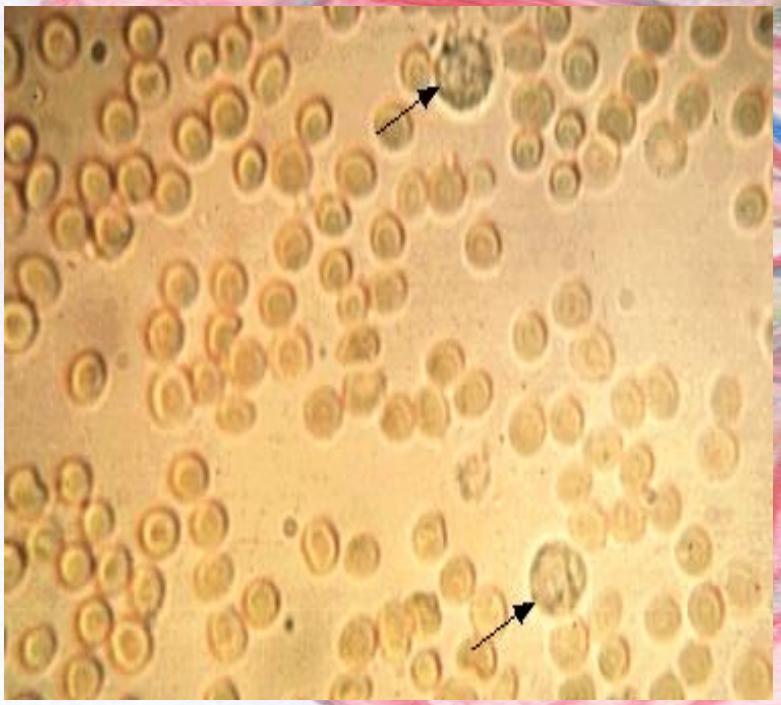


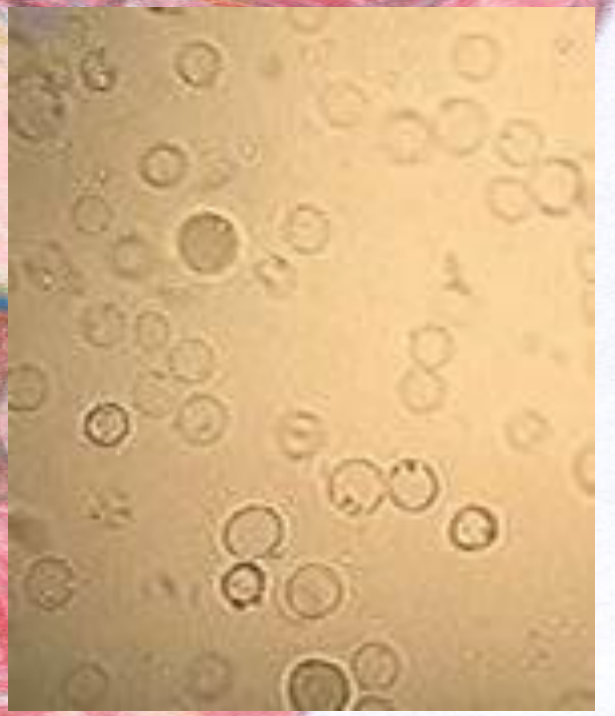
Urinary Sediment (2)



- RBC casts or dysmorphic RBCs
  - Acute glomerulonephritis
  - Small vessel vasculitis

Red Blood Cells

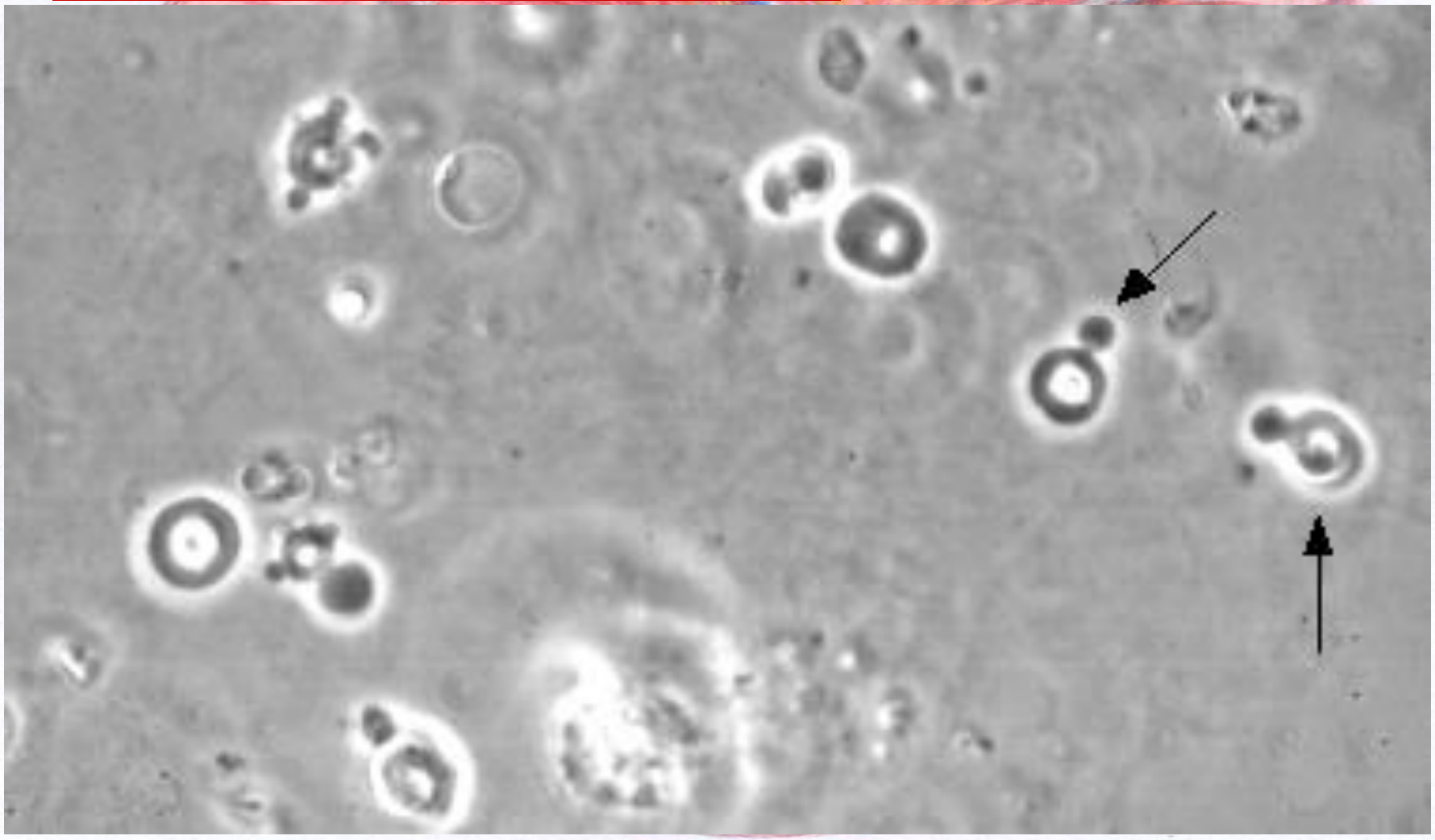




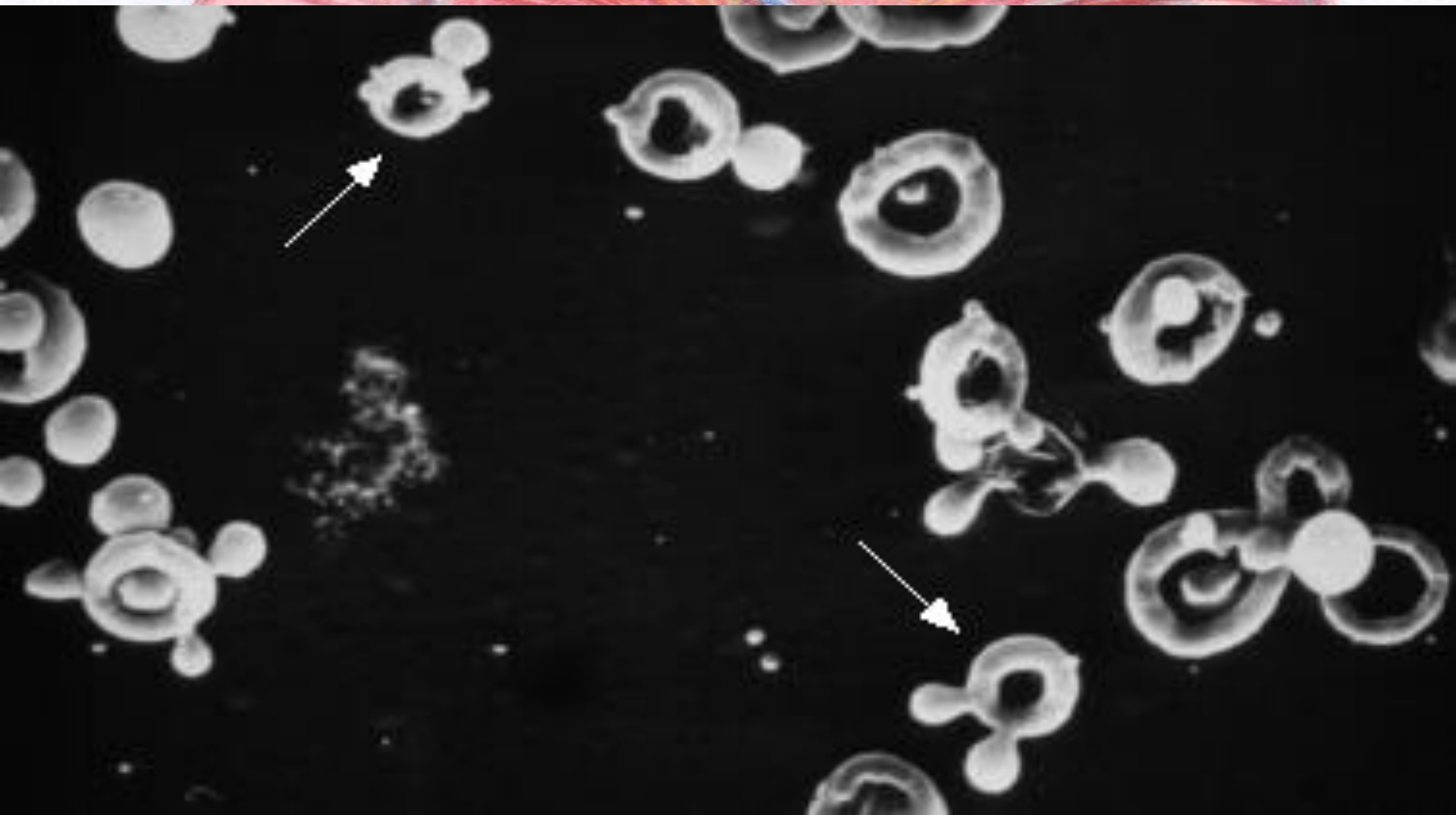
Monomorphic

Dysmorphic

Dysmorphic Red Blood Cells

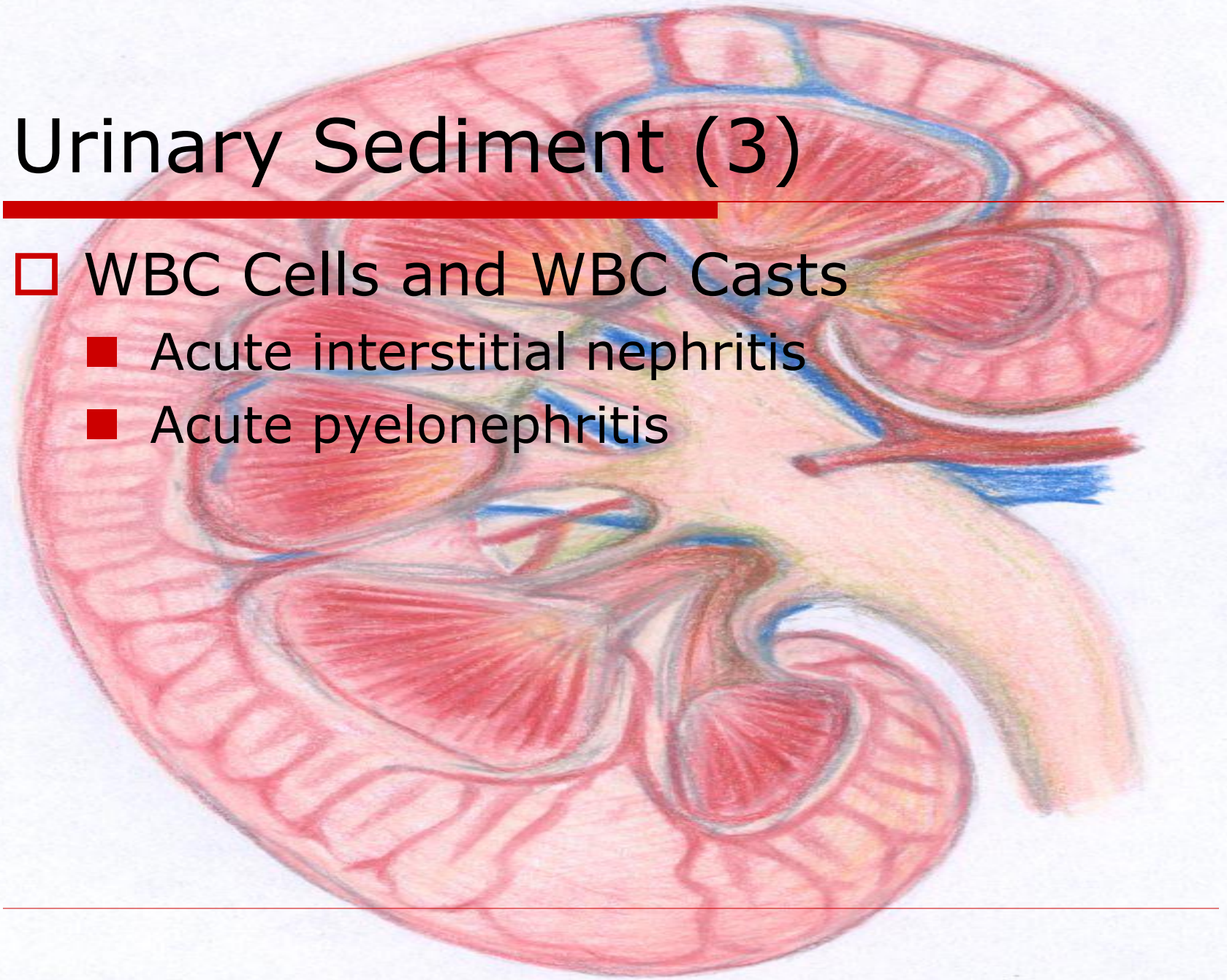


Dysmorphic Red Blood Cells



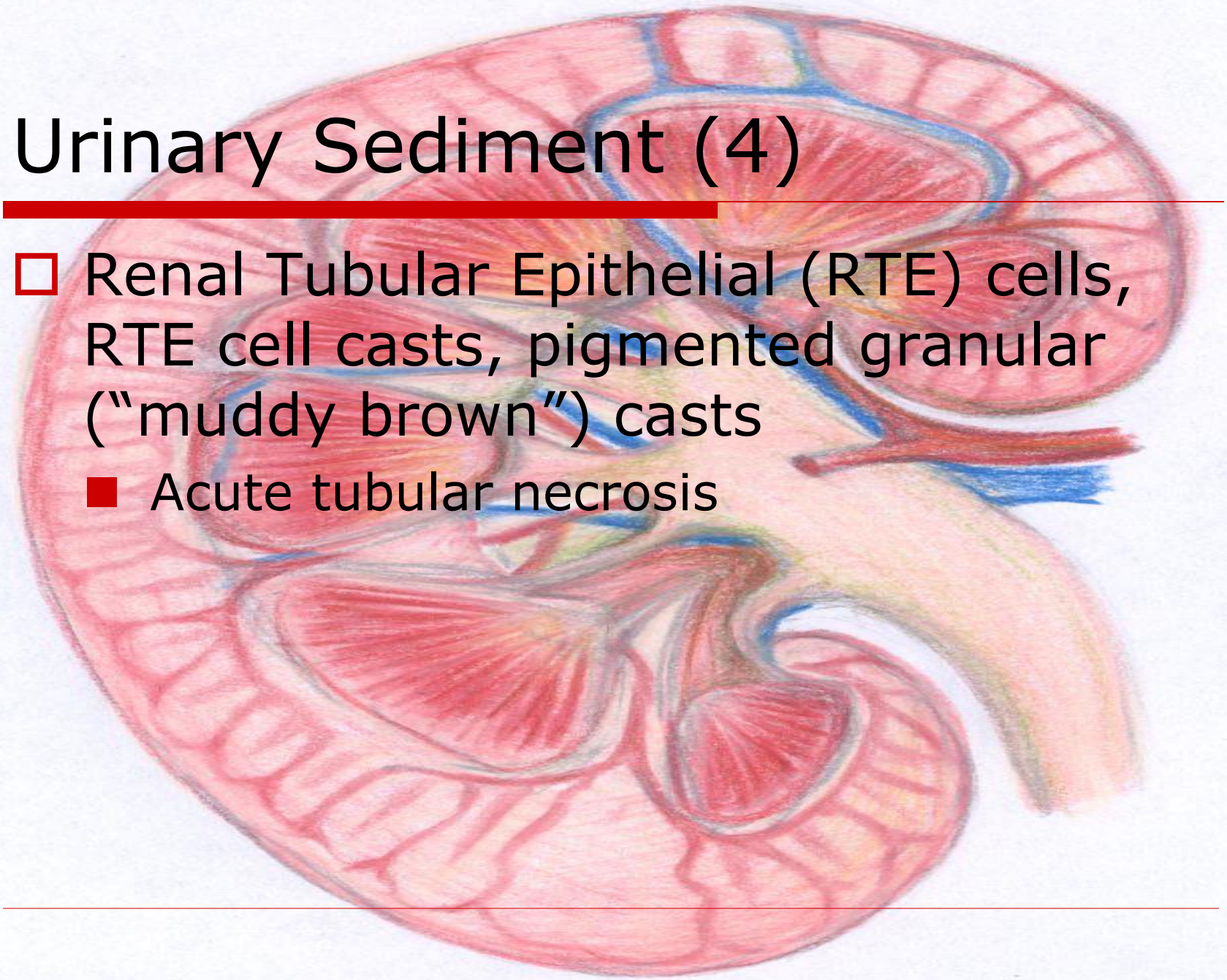
Urinary Sediment (3)

- WBC Cells and WBC Casts
  - Acute interstitial nephritis
  - Acute pyelonephritis

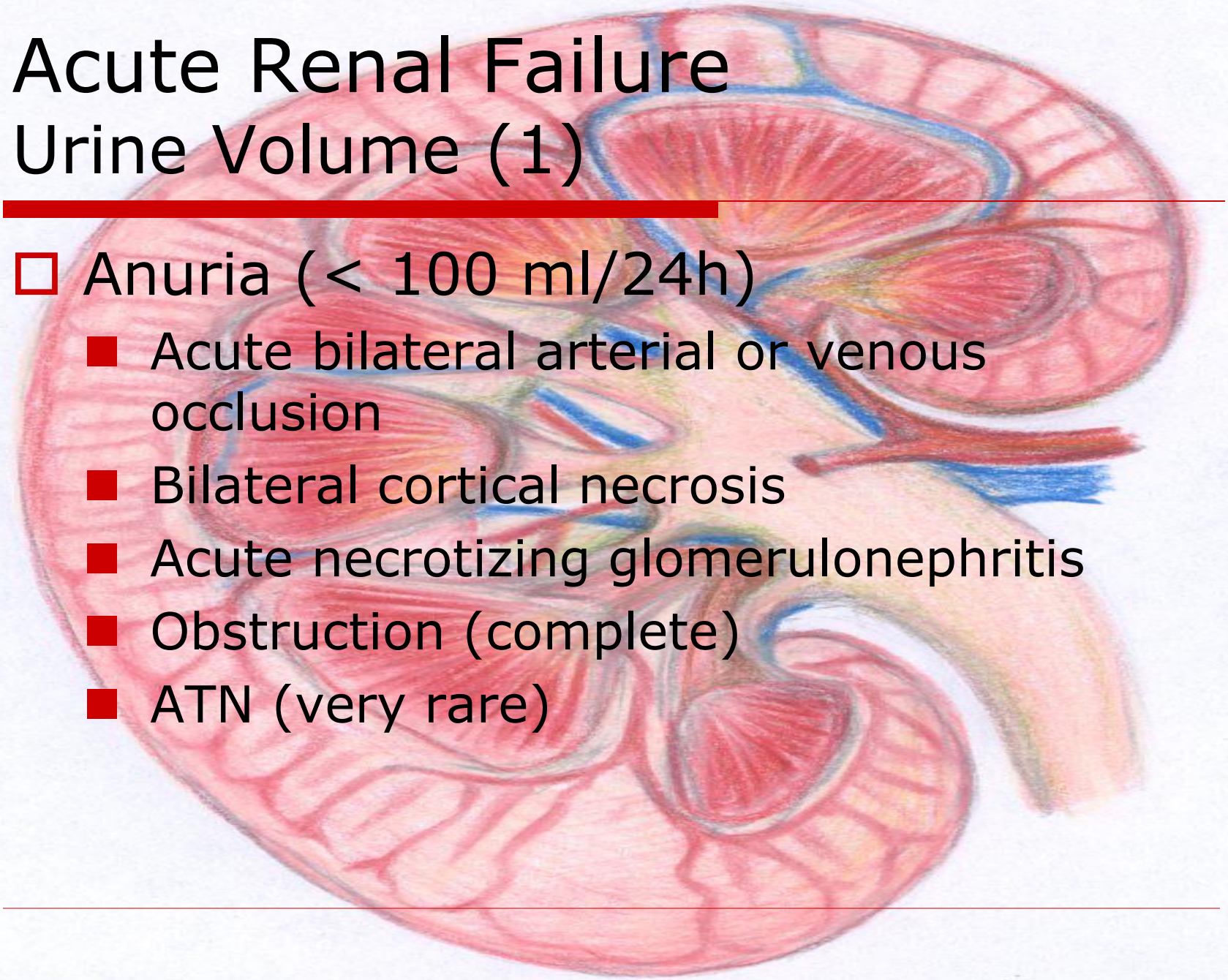


Urinary Sediment (4)

- Renal Tubular Epithelial (RTE) cells, RTE cell casts, pigmented granular ("muddy brown") casts
  - Acute tubular necrosis

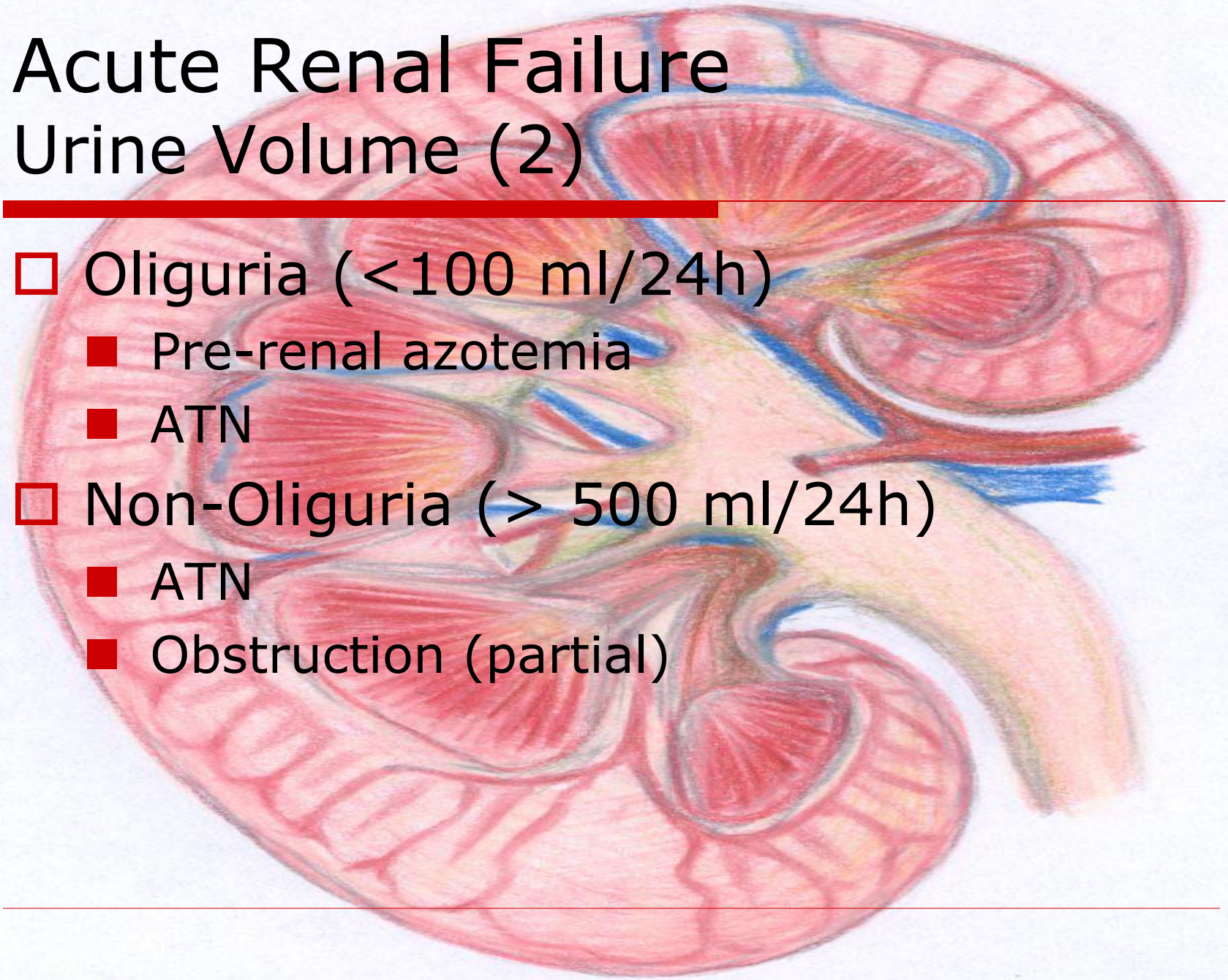


Acute Renal Failure
Urine Volume (1)



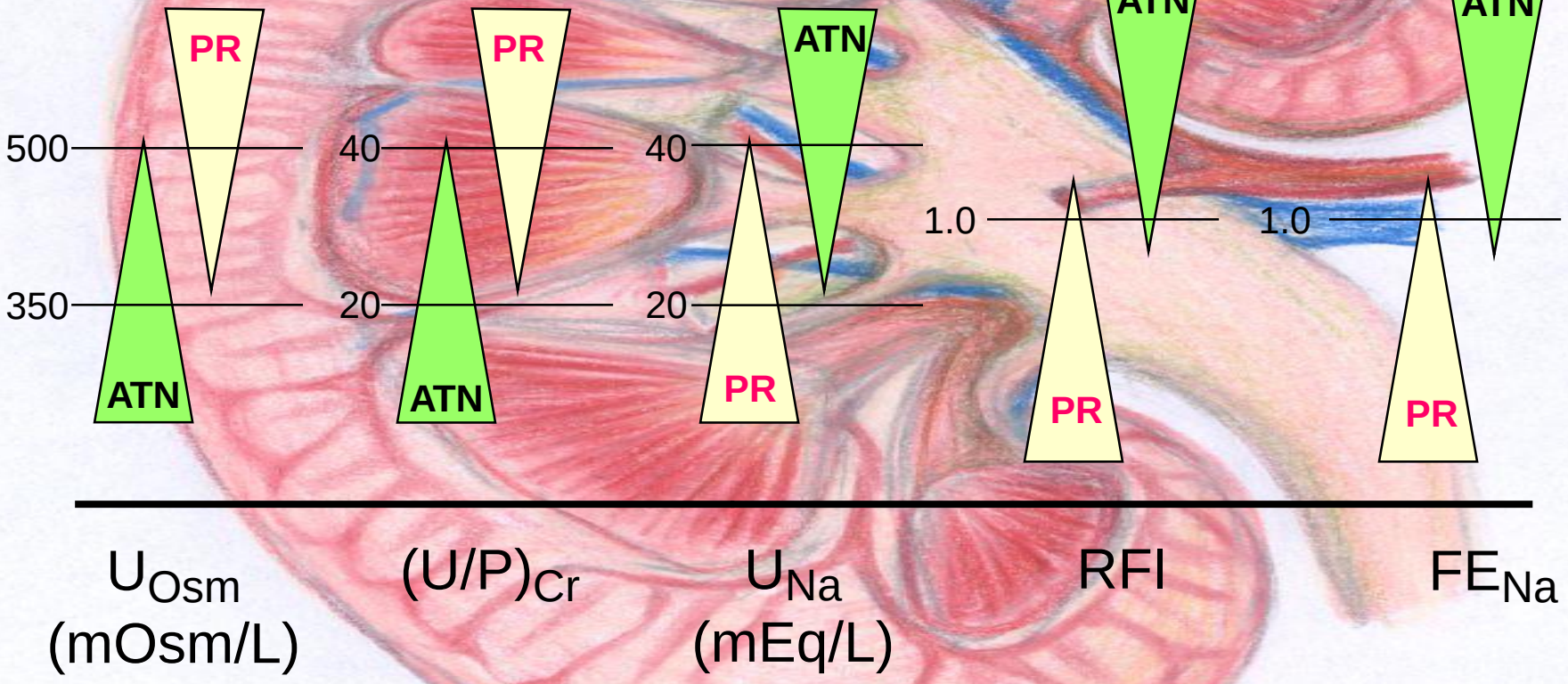
- Anuria (< 100 ml/24h)
  - Acute bilateral arterial or venous occlusion
  - Bilateral cortical necrosis
  - Acute necrotizing glomerulonephritis
  - Obstruction (complete)
  - ATN (very rare)

Acute Renal Failure
Urine Volume (2)



- Oliguria (<100 ml/24h)
  - Pre-renal azotemia
  - ATN
- Non-Oliguria (> 500 ml/24h)
  - ATN
  - Obstruction (partial)

Acute Renal Failure
Urinary Indices



ARF Urine indices

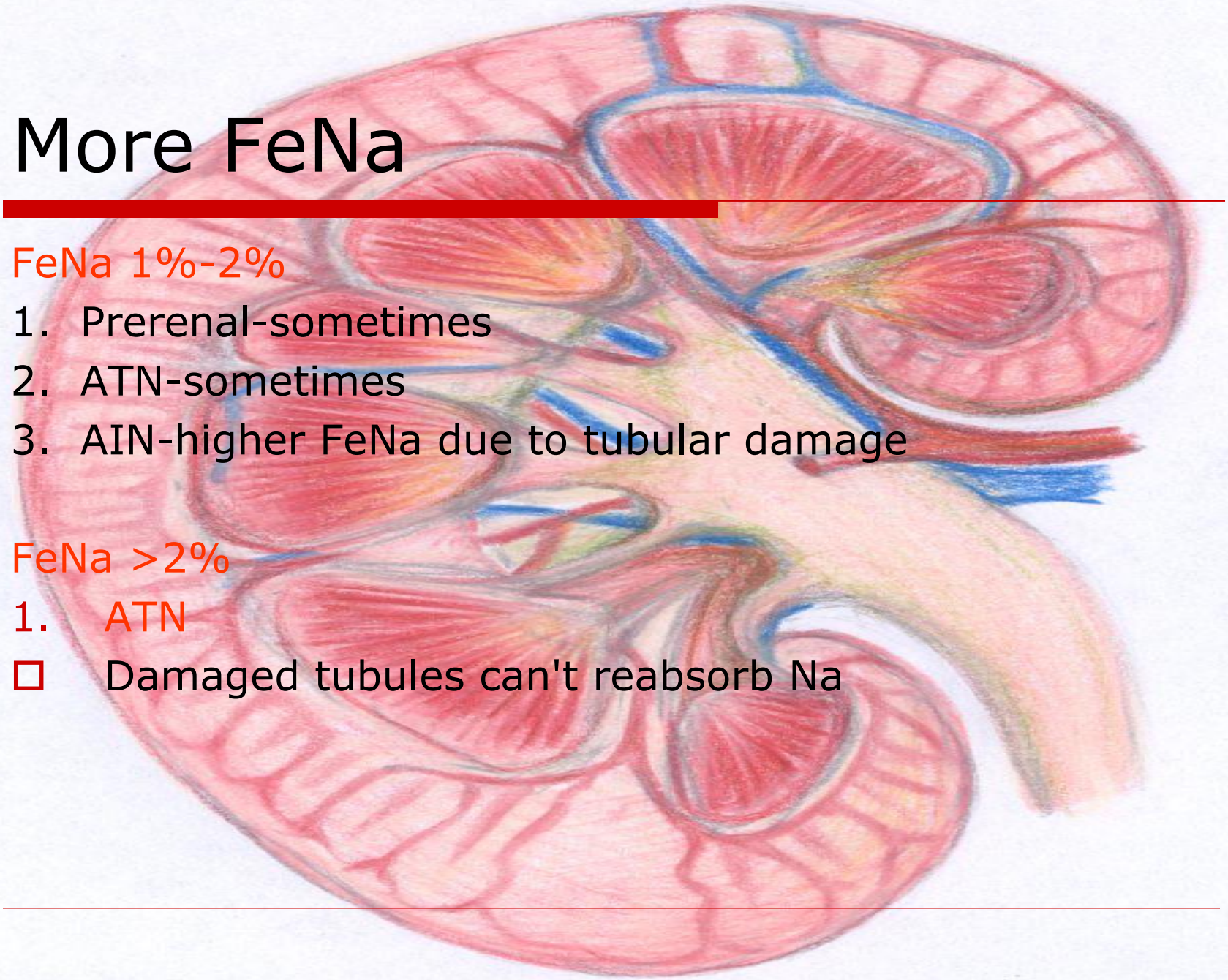
- Urinary Indices;
  - FE Na = $(U/P)_{Na} \times (P/U)_{Cr} \times 100$
    - FENa < 1% C/W Pre-renal state
      - May be low in selected intrinsic cause
        - Contrast nephropathy
        - Acute GN
        - Myoglobin induced ATN
    - FENa> 1% C/W intrinsic cause of ARF

FeNa = $\frac{\text{(urine Na x plasma Cr)}}{\text{(plasma Na x urine Cr)}}$

FeNa <1%

1. PRERENAL
   - Urine Na < 20. Functioning tubules reabsorb lots of filtered Na
2. ATN (unusual)
   - Postischemic dz: most of UOP comes from few normal nephrons, which handle Na appropriately
   - ATN + chronic prerenal dz (cirrhosis, CHF)
3. Glomerular or vascular injury
   - Despite glomerular or vascular injury, pt may still have well-preserved tubular function and be able to concentrate Na

More FeNa



FeNa 1%-2%

1. Prerenal-sometimes
2. ATN-sometimes
3. AIN-higher FeNa due to tubular damage

FeNa >2%

1. ATN

- Damaged tubules can't reabsorb Na

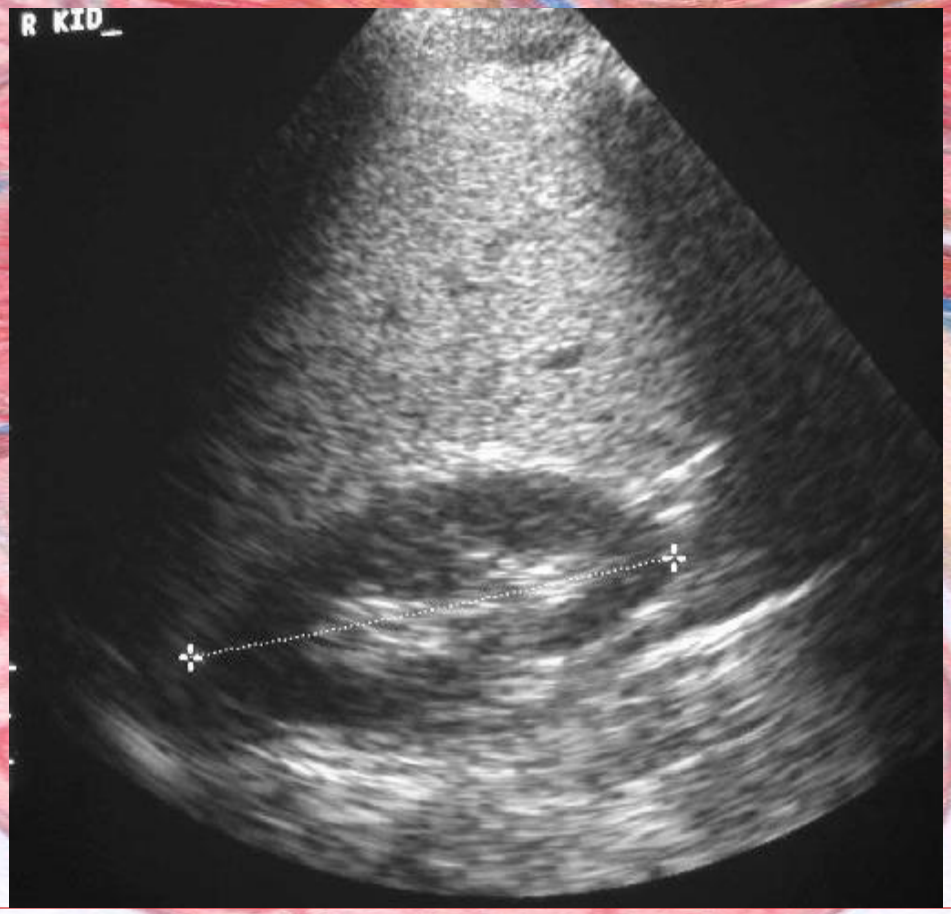
Calculating FeNa after pt has gotten Lasix…

- Caution with calculating FeNa if pt has had Loop Diuretics in past 24-48 h
- Loop diuretics cause natriuresis (incr urinary Na excretion) that raises U Na-even if pt is prerenal
- So if FeNa>1%, you don't know if this is because pt is euvolemic or because Lasix increased the U Na
- **So helpful if FeNa still <1%, but not if FeNa >1%**

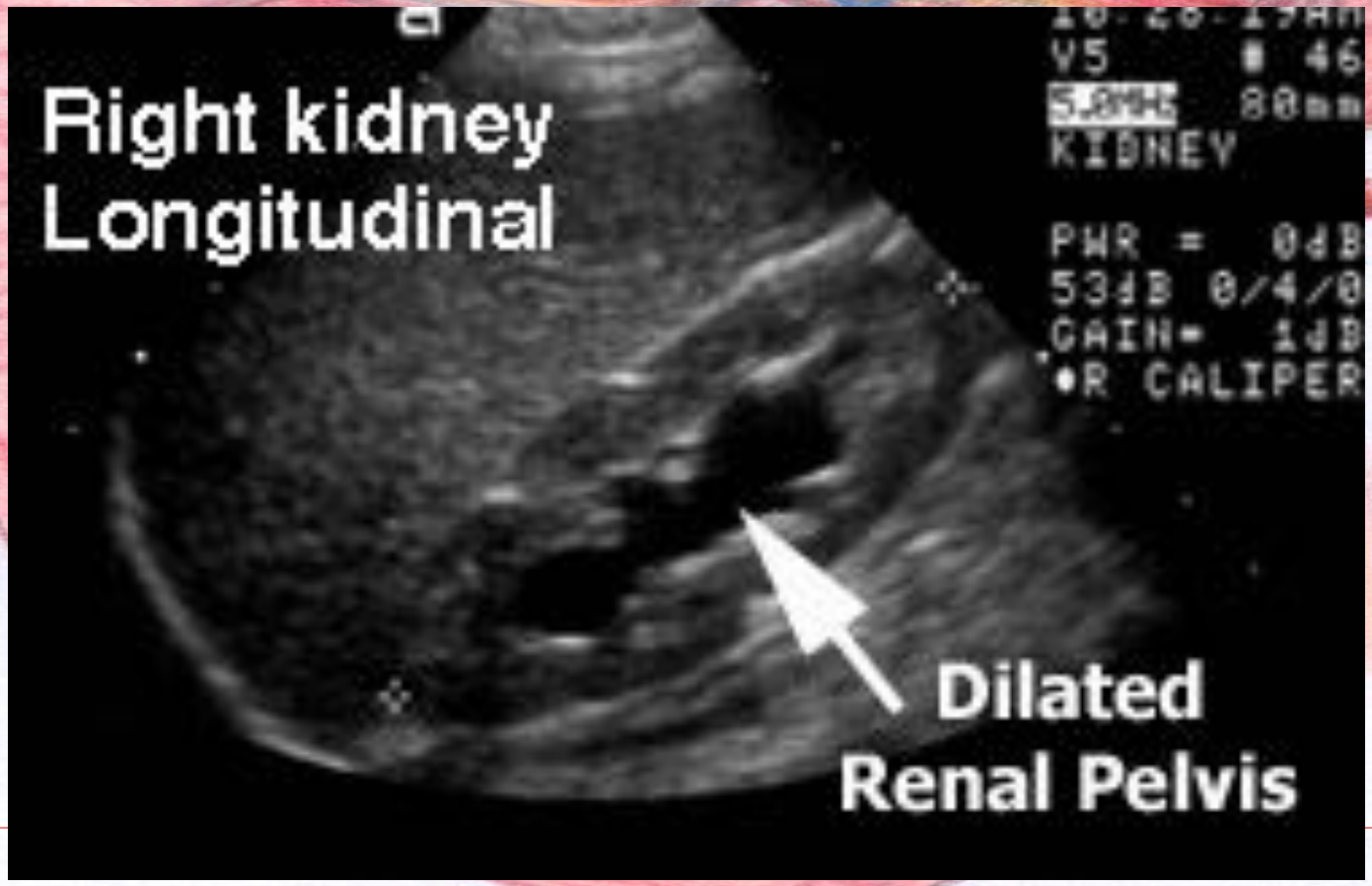
1. Fractional Excretion of Lithium (endogenous)
2. Fractional Excretion of Uric Acid
3. Fractional Excretion of Urea

Hydronephrosis

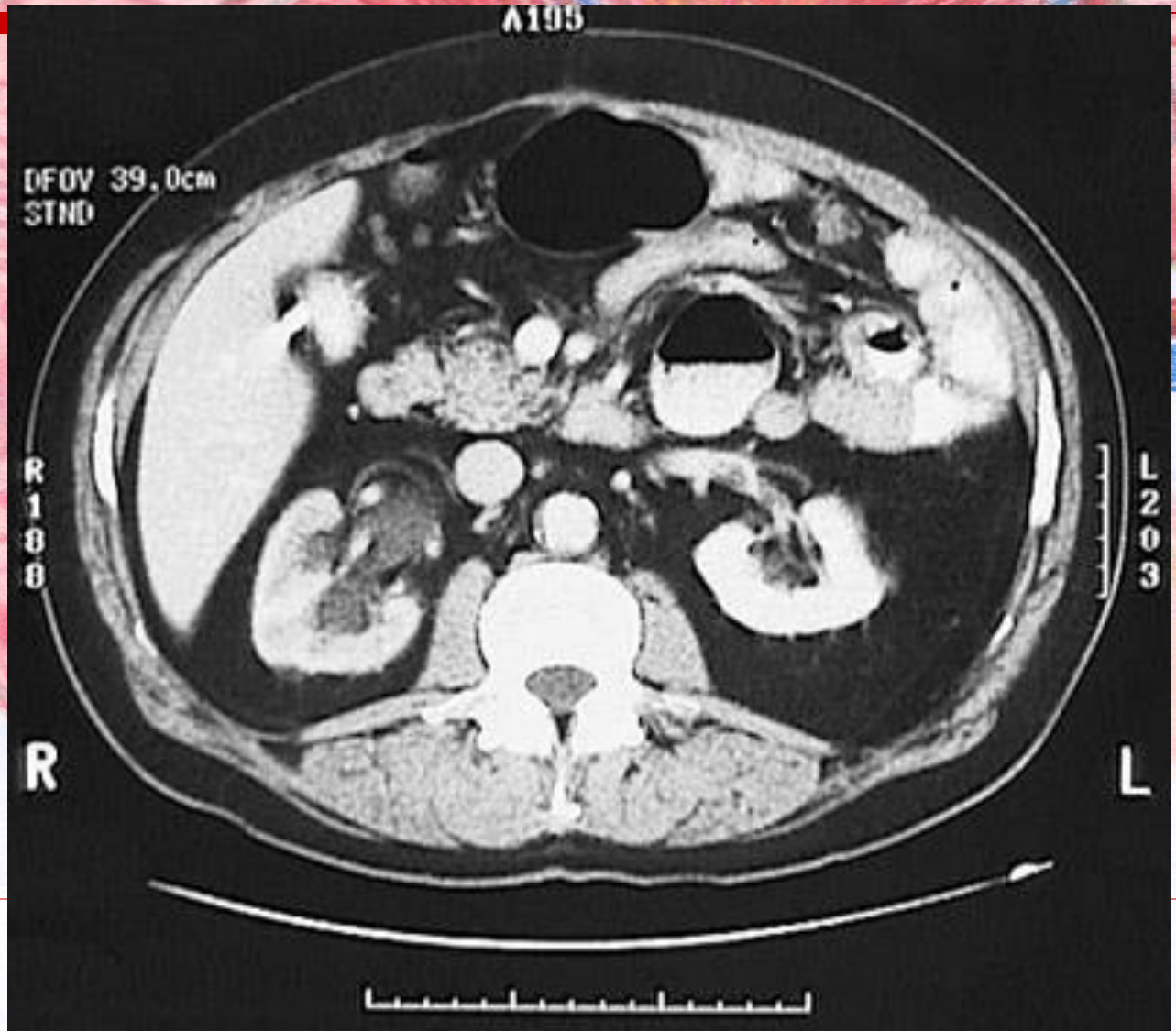
Normal Renal Ultrasound



Hydronephrosis

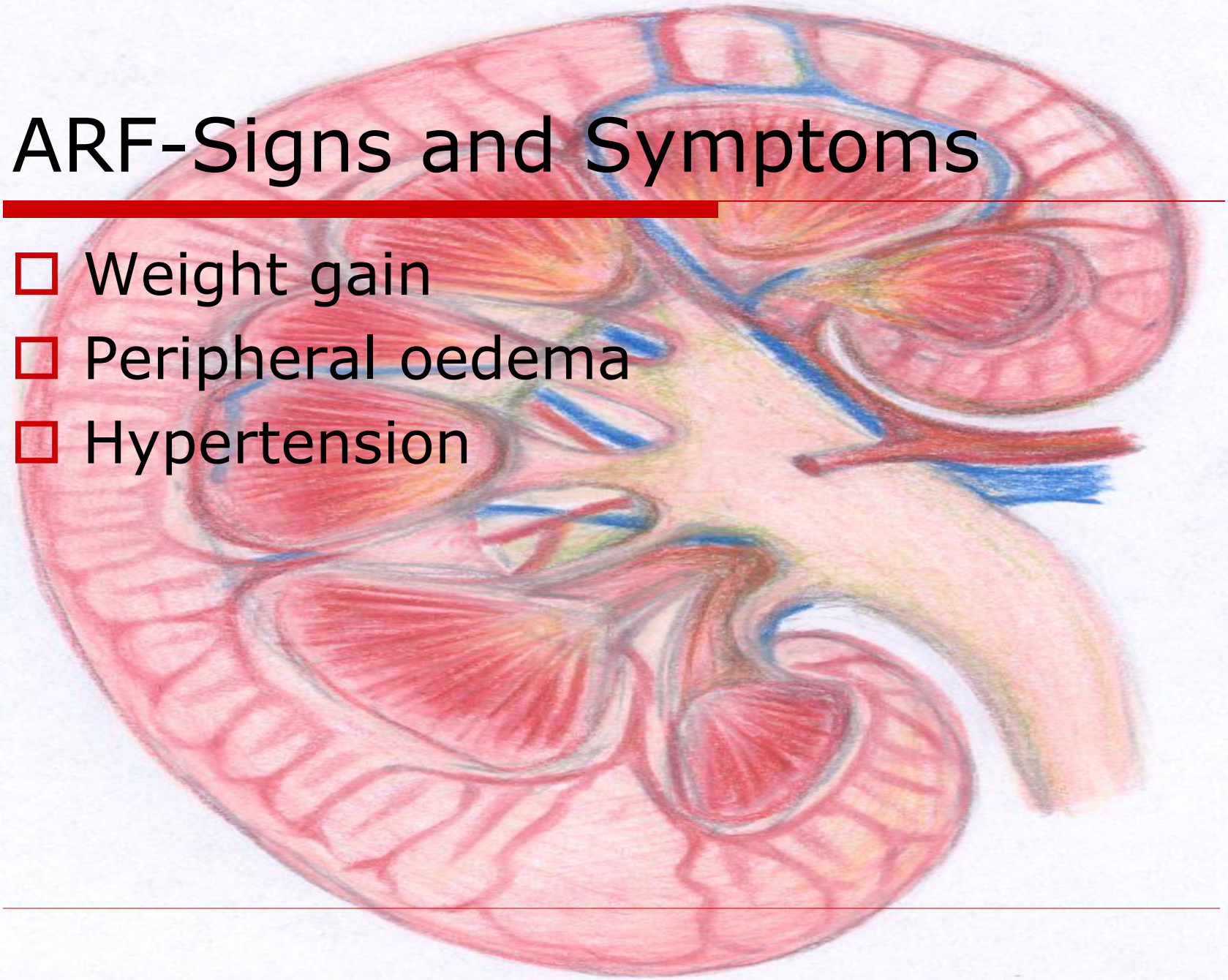


Hydronephrosis



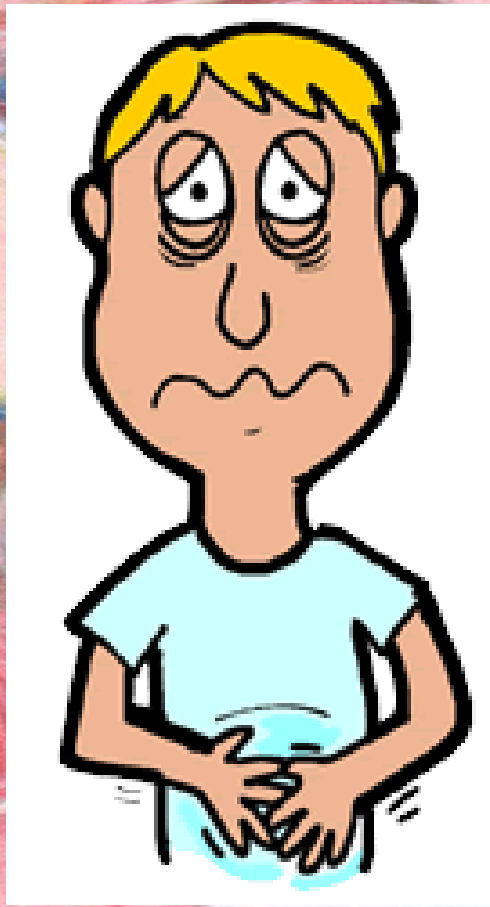
ARF-Signs and Symptoms

- Weight gain
- Peripheral oedema
- Hypertension



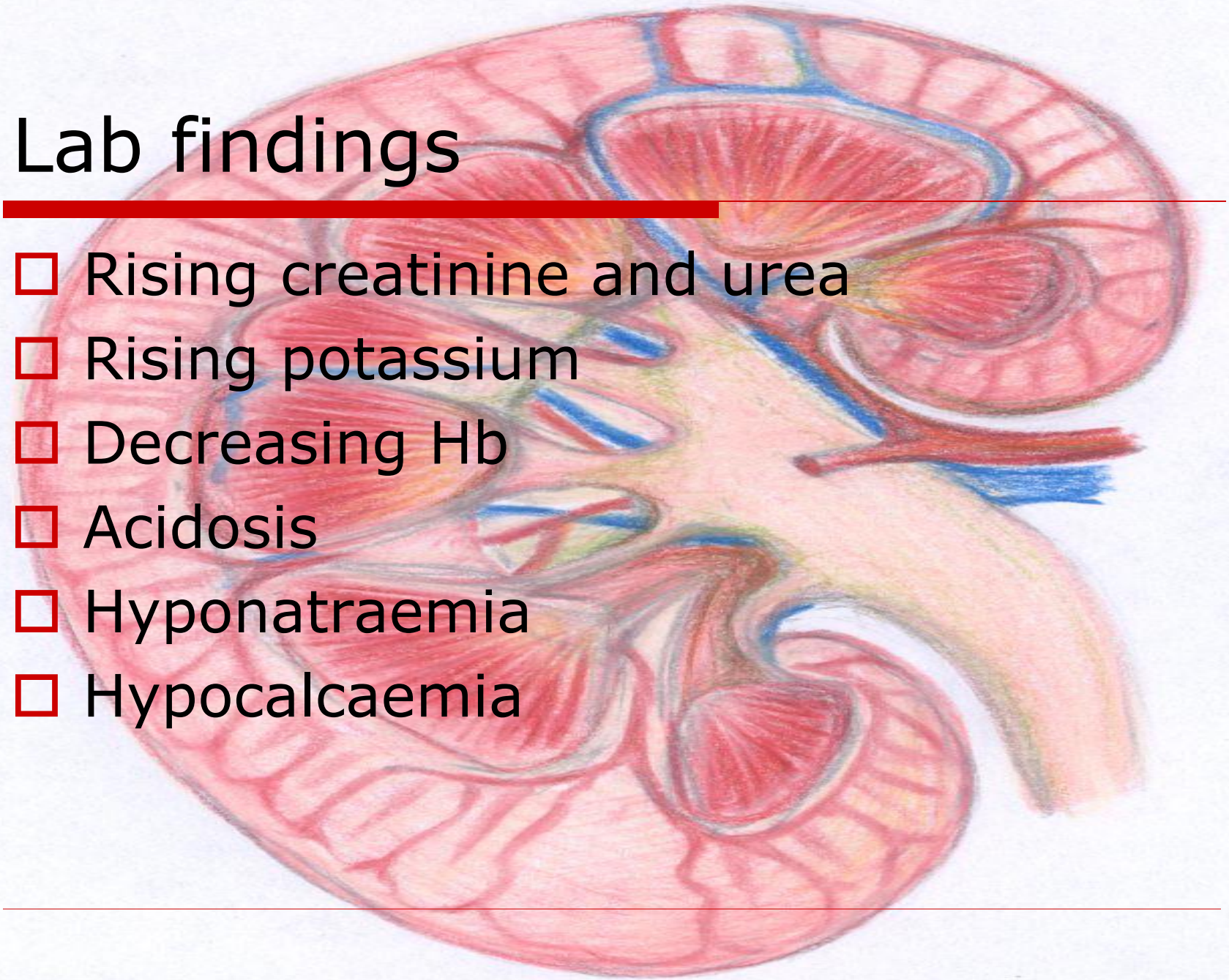
ARF Signs and Symptoms

- Hyperkalemia
- Nausea/Vomiting
- Pulmonary edema
- Ascites
- Asterixis
- Encephalopathy



Lab findings

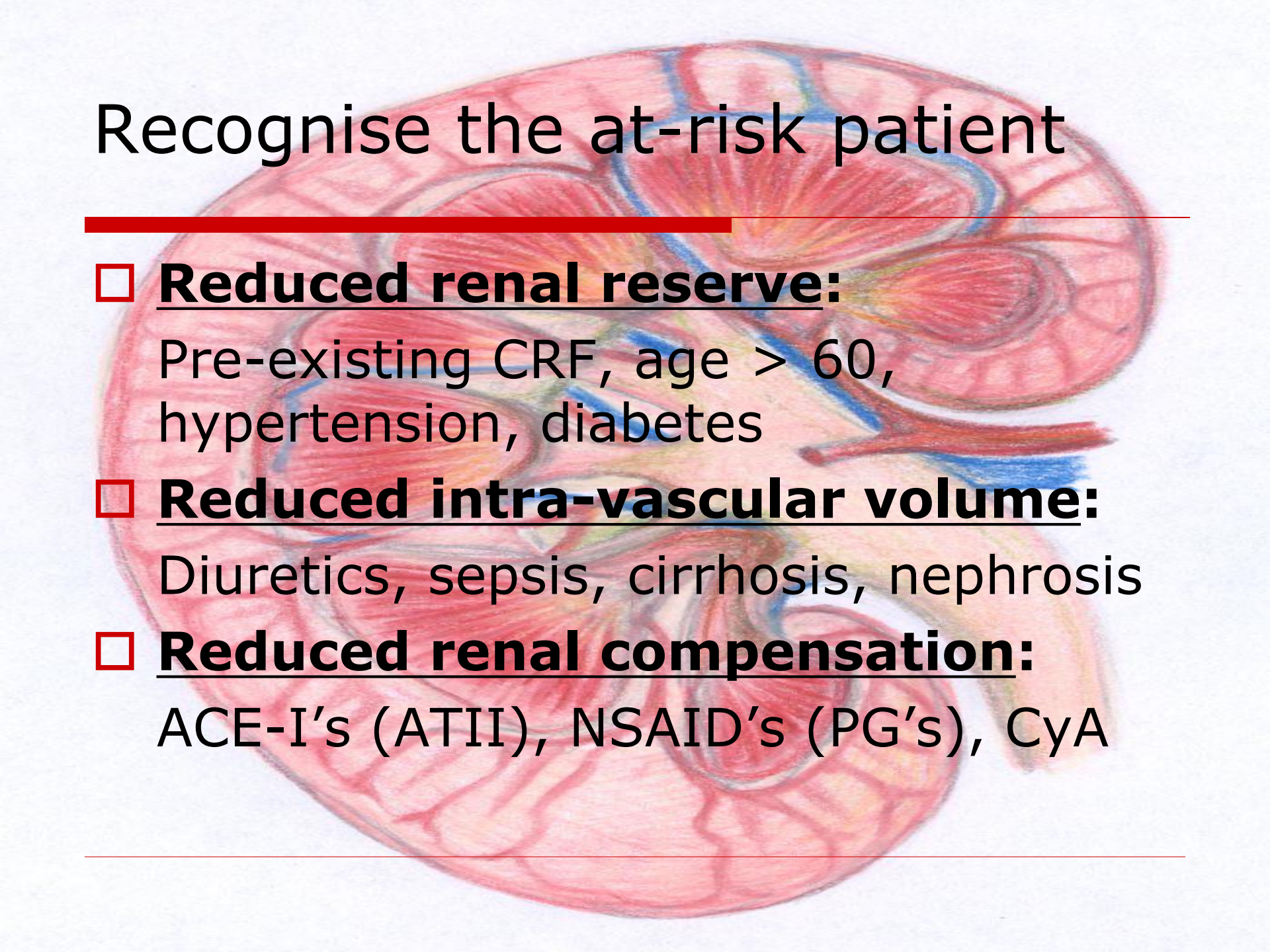
- Rising creatinine and urea
- Rising potassium
- Decreasing Hb
- Acidosis
- Hyponatraemia
- Hypocalcaemia



Mx ARF

- Immediate treatment of pulmonary edema and hyperkalaemia
- Remove offending cause or treat offending cause
- Dialysis as needed to control hyperkalaemia, pulmonary edema, metabolic acidosis, and uremic symptoms
- Adjustment of drug regimen
- Usually restriction of water, Na, and K intake, but provision of adequate protein
- Possibly phosphate binders and Na polystyrene sulfonate

Recognise the at-risk patient



- **Reduced renal reserve:**

  Pre-existing CRF, age > 60, hypertension, diabetes

- **Reduced intra-vascular volume:**

  Diuretics, sepsis, cirrhosis, nephrosis

- **Reduced renal compensation:**

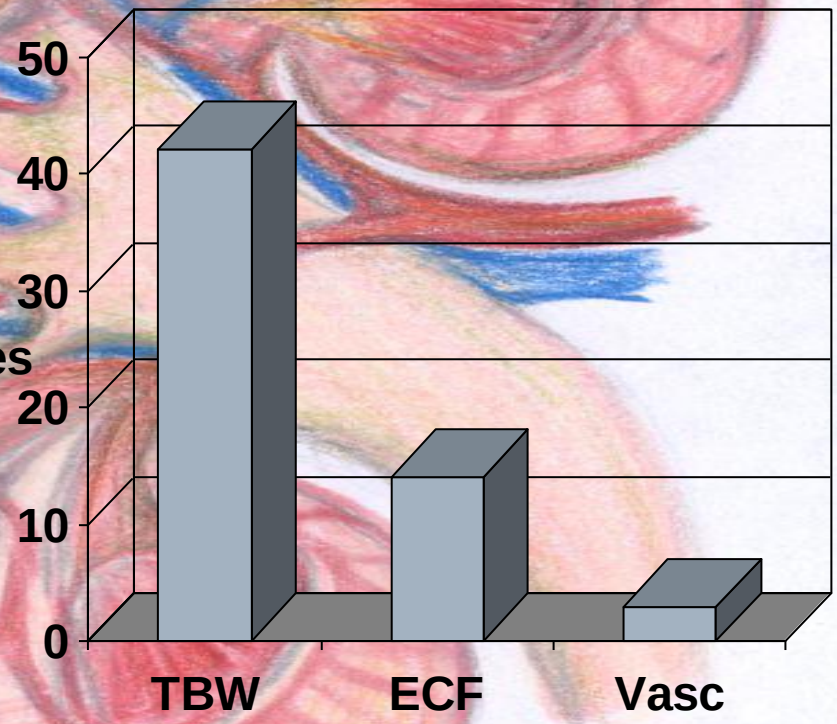
  ACE-I's (ATII), NSAID's (PG's), CyA

Acute Tubular Necrosis
Clinical Characteristics

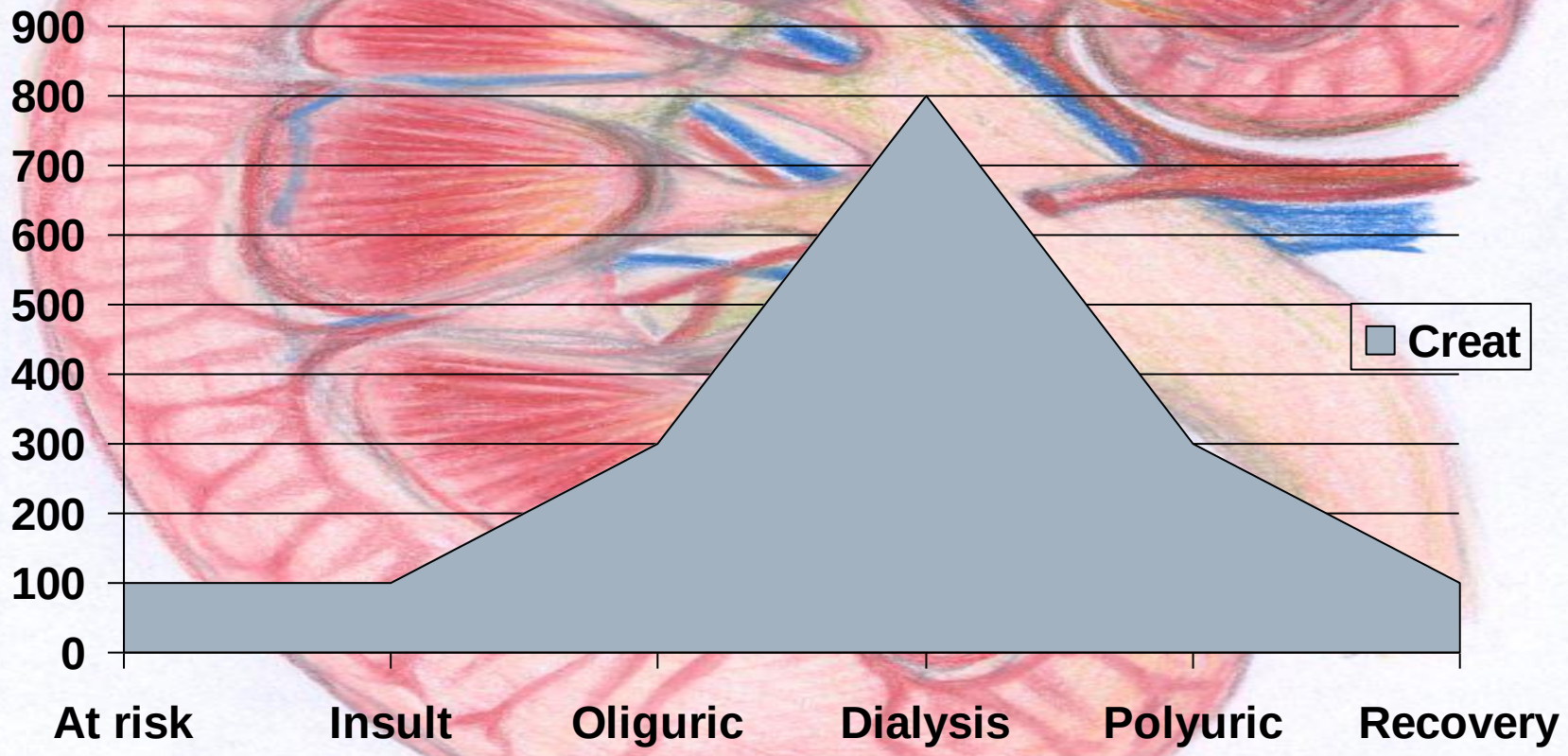
Characteristic	Oliguric ATN	Non-Oliguric ATN
Incidence	41%	59%
Toxin-induced	8%	30%
UV (ml/24h)	< 400	1,280 $\pm$ 75
U_{Na} (mEq/L)	68 $\pm$ 6	50 $\pm$ 5
FE_{Na} (%)	6.8 $\pm$ 1.4	3.1 $\pm$ 0.5
Dialysis required	84%	26%
Mortality	50%	25%

Assessment of Volume Status

- **Total Body Water:**
  - weight, serum Na
- **ECF** (= Total Body Na):
  - oedema, skin turgor
- **Intravascular:**
  - Venous: JVP/CVP/PCWP
  - Arterial: BP (lying/sitting)
  - Peripheral perfusion: fingers, toes, nose



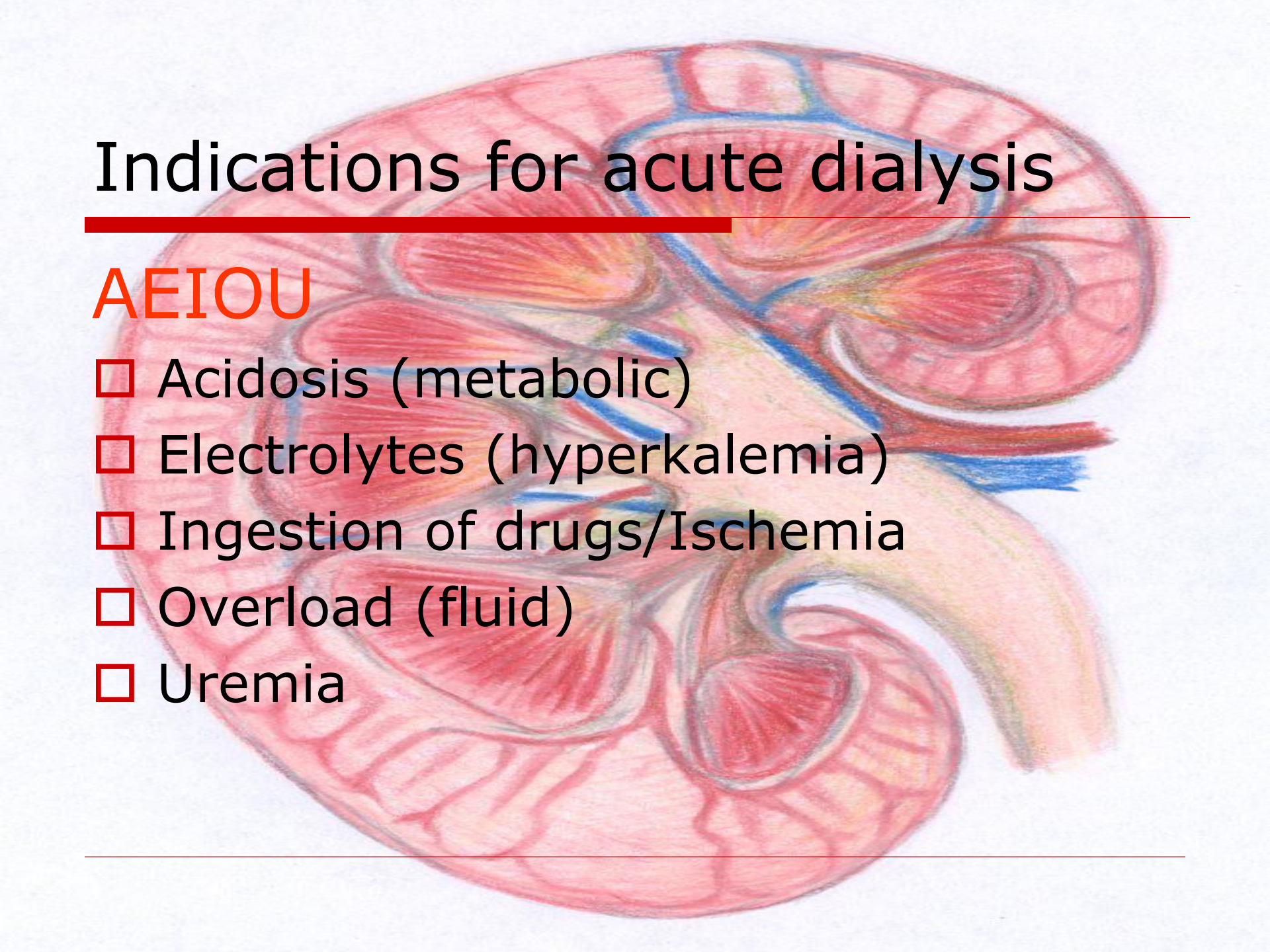
Phases of ATN



Indications for acute dialysis

AEIOU

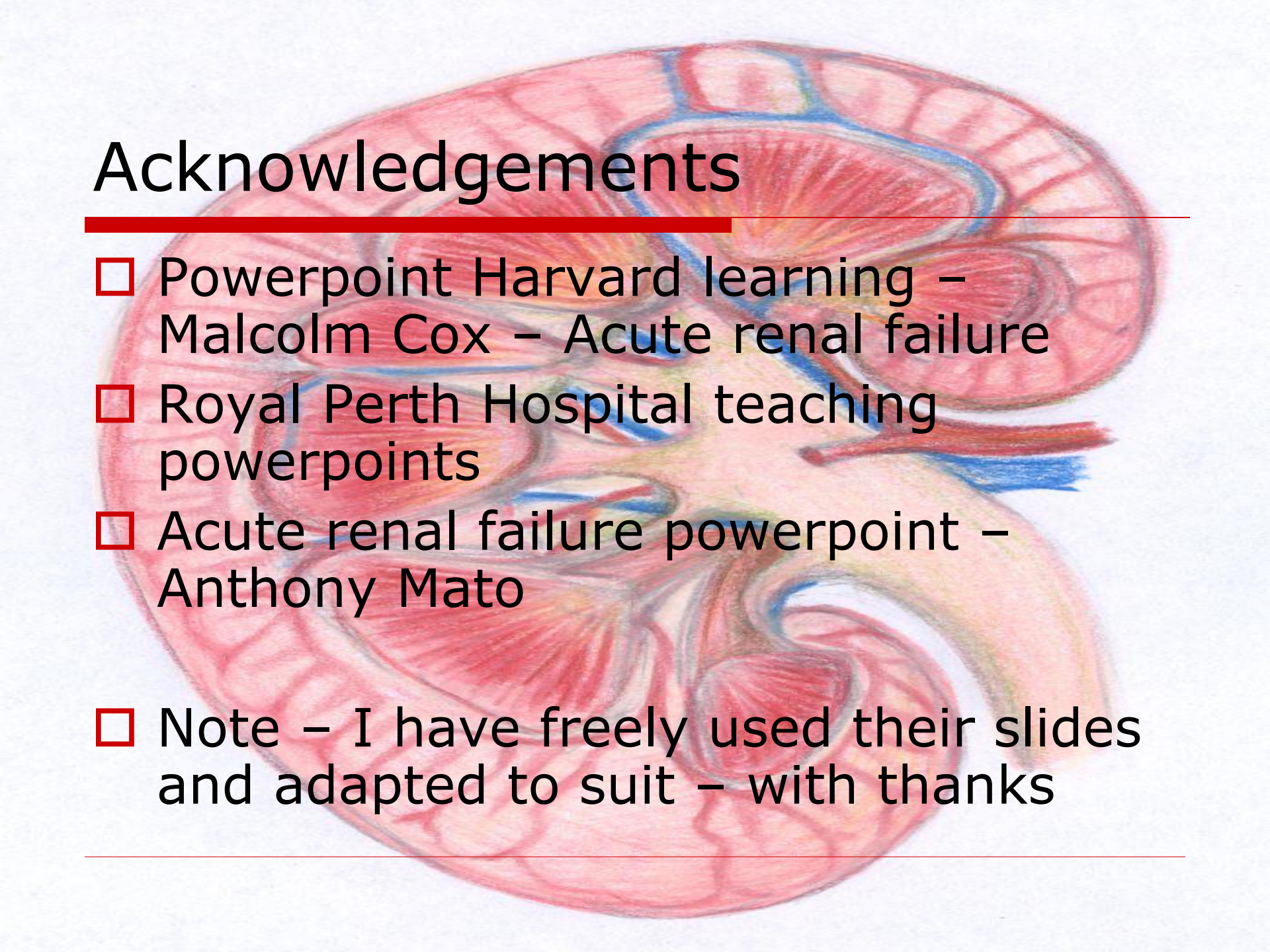
- Acidosis (metabolic)
- Electrolytes (hyperkalemia)
- Ingestion of drugs/Ischemia
- Overload (fluid)
- Uremia



Conclusion

- Think about who might be vulnerable to acute renal failure
- Think twice before initiating therapy that may cause ARF
- Think about it as a diagnosis – don't look/won't find

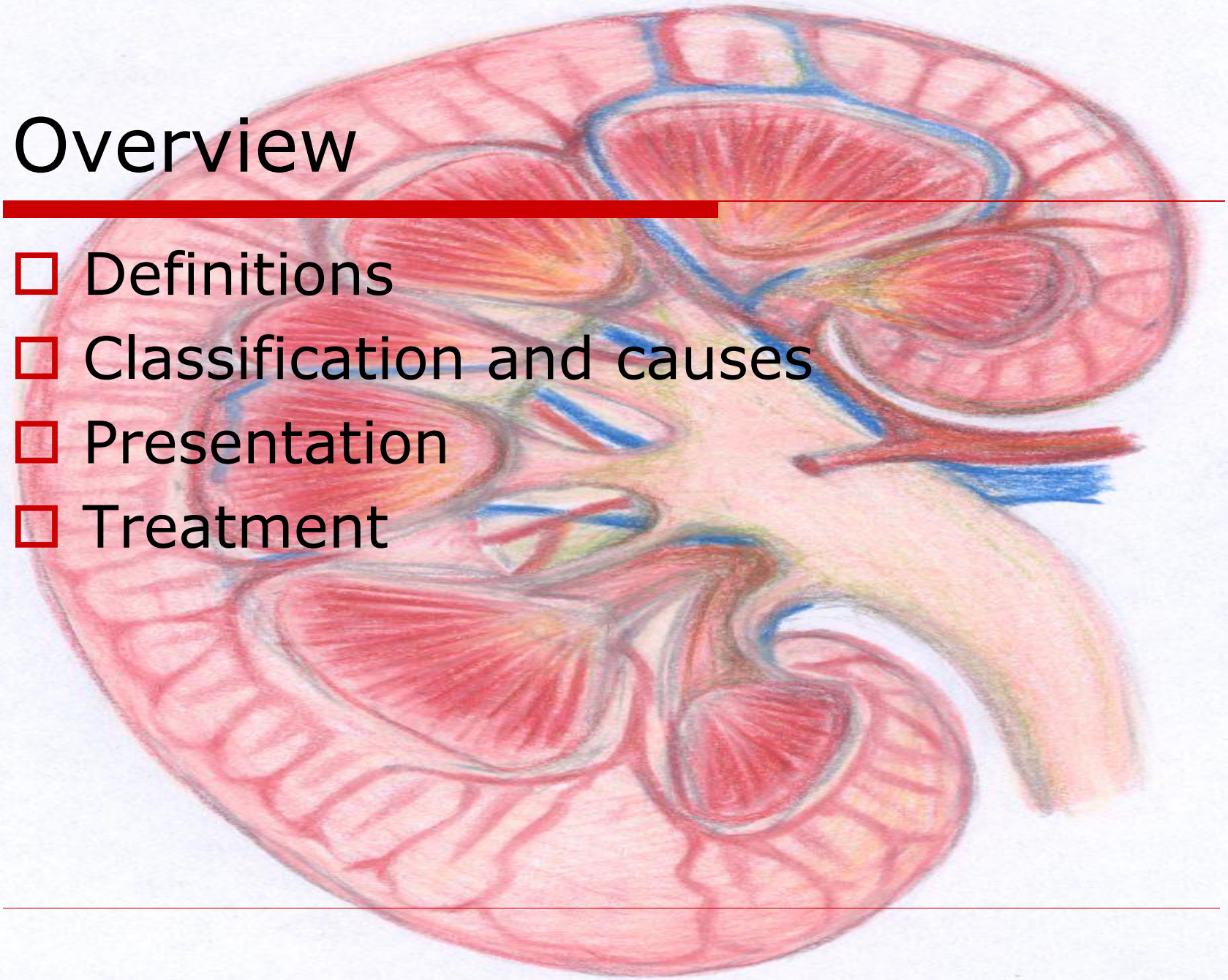
Acknowledgements



- Powerpoint Harvard learning – Malcolm Cox – Acute renal failure
- Royal Perth Hospital teaching powerpoints
- Acute renal failure powerpoint – Anthony Mato

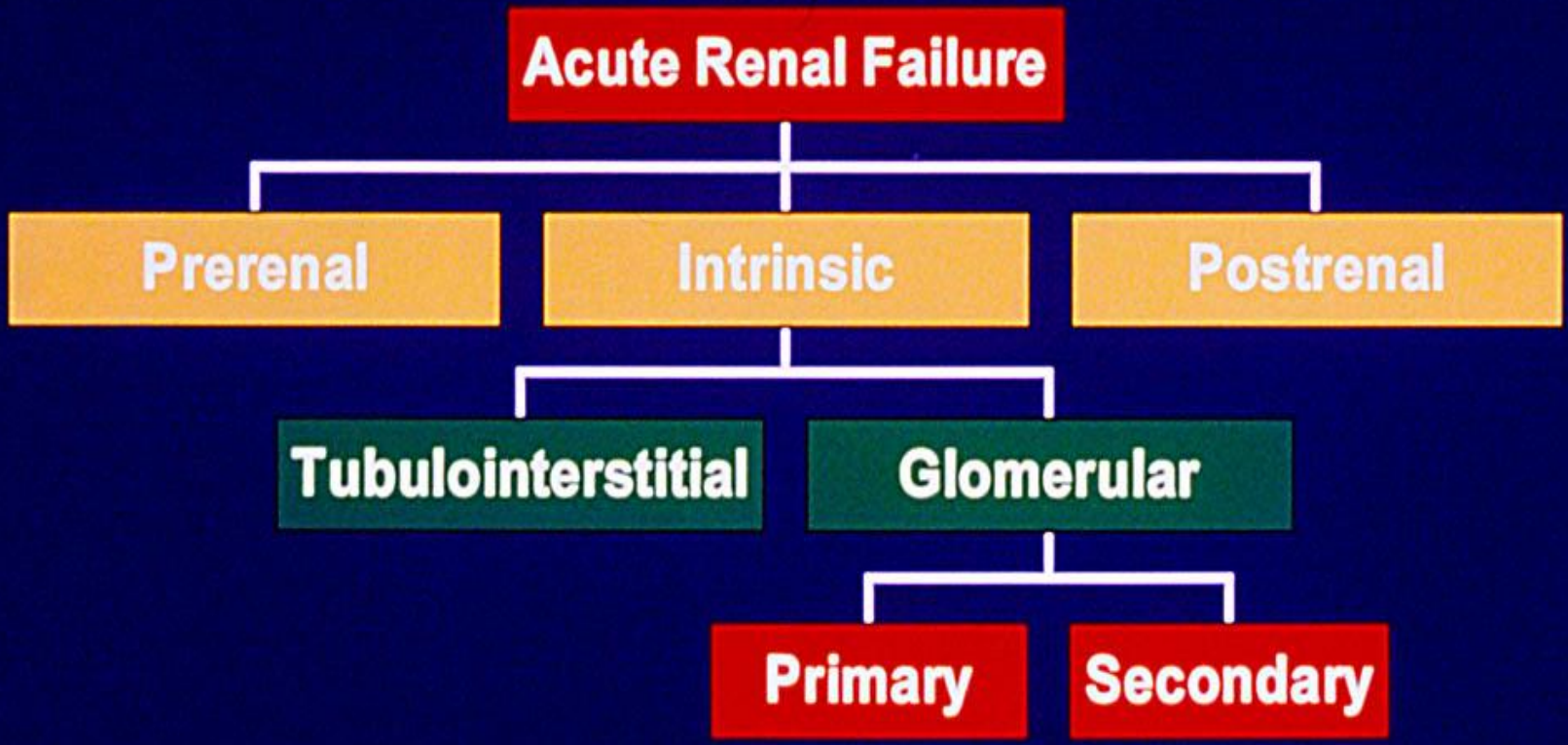
- Note – I have freely used their slides and adapted to suit – with thanks

Overview

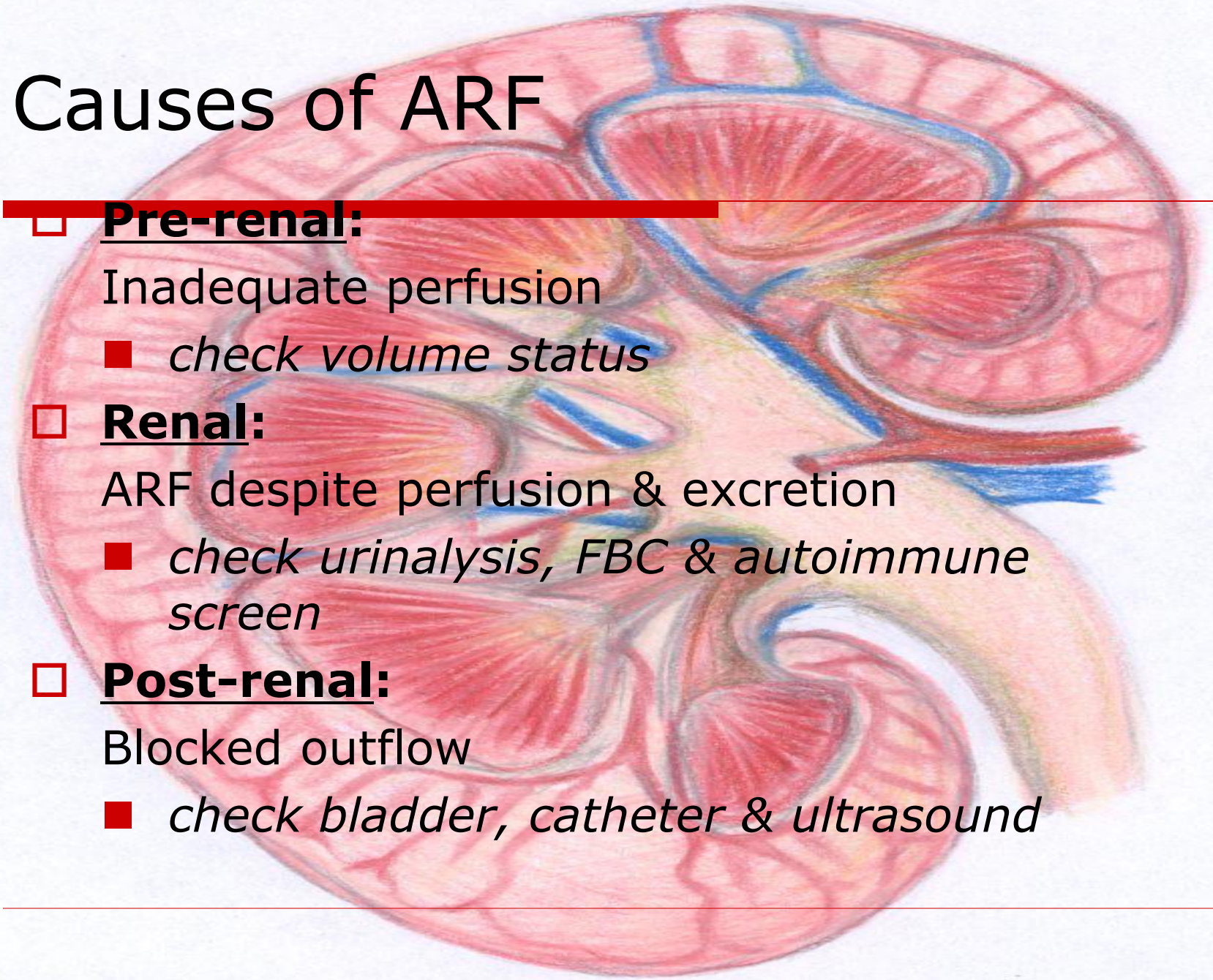


- Definitions
- Classification and causes
- Presentation
- Treatment

Work-up of Acute Renal Failure



Causes of ARF



- **Pre-renal:**

  Inadequate perfusion

  - *check volume status*

- **Renal:**

  ARF despite perfusion & excretion

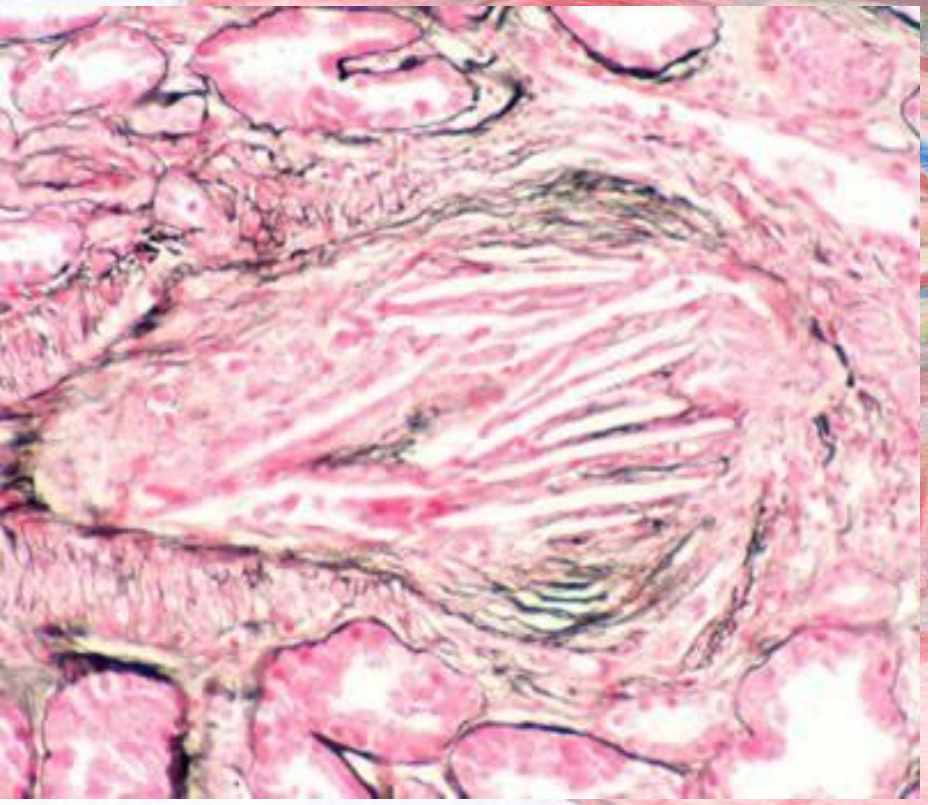
  - *check urinalysis, FBC & autoimmune screen*

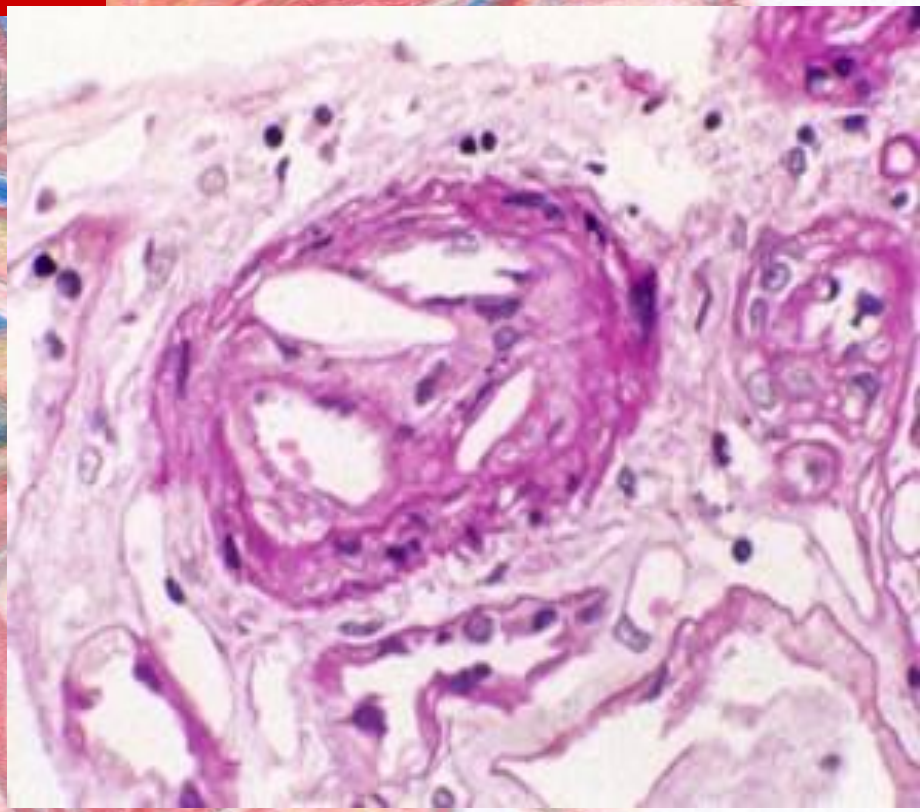
- **Post-renal:**

  Blocked outflow

  - *check bladder, catheter & ultrasound*

Cholesterol Embolization





Causes of ARF

Pre-renal	**Renal**	**Post-renal**
Absolute hypovolaemia	Glomerular (RPGN)	Pelvi-calyceal
Relative hypovolaemia	Tubular (ATN)	Ureteric
Reduced cardiac output	Interstitial (AIN)	VUJ-bladder
Reno-vascular occlusion	Vascular (atheroemboli)	Bladder neck-urethra