Acute Renal failure (Acute kidney injury)

By

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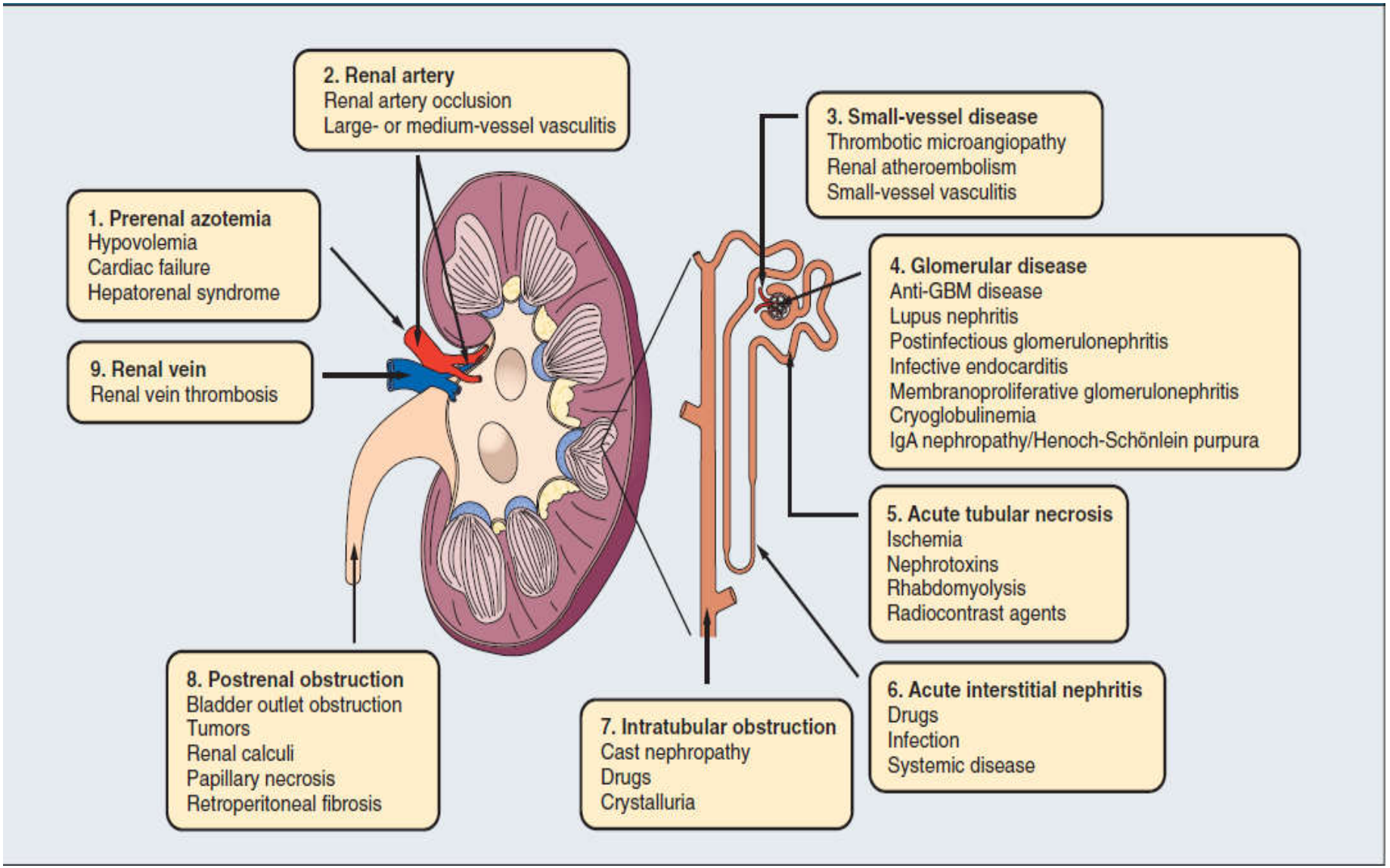
Acute Renal failure

- **Definition :**
  - **Acute kidney injury (AKI) or acute renal failure (ARF),** was previously defined as a rapid deterioration of renal functions resulting in the accumulation of nitrogenous wastes such as urea and creatinine. However, immediately after a kidney injury, BUN or creatinine levels may be normal, and the only sign of a kidney injury may be decreased urine production.

Causes Of AKI



Causes of AKI can be categorized into:

Pre-renal (functional)

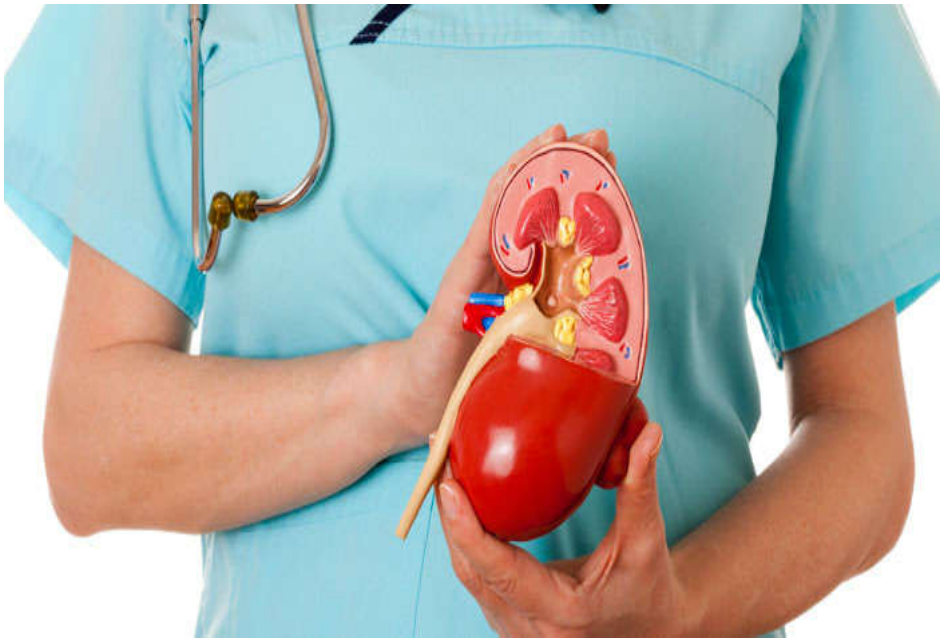
- Hypovolemia, e.g. bleeding, gastrointestinal losses
- Sepsis
- Cardiac arrhythmias
- Myocardial infarction
- Renal artery stenosis

Intrinsic renal (damage)

- Prolonged hypoperfusion causing tubular injury
- Infiltrative disease, e.g. myeloma
- Nephrotoxins
- Glomerulonephritis
- Interstitial nephritis
- Rhabdomyolysis

Post-renal (functional)

- Renal stone disease
- Pelvic masses, e.g. cervical cancer
- Prostatic hypertrophy / cancer
- Urethral stricture



Acute tubular necrosis

- **Causes :**
  1. ***Ischemic causes (Prerenal factors*** if not corrected rapidly)

     Prolonged pre-renal cause for ARF → Tubular ischemia & necrosis
  2. ***Toxic causes :***

Endogenous :

- Hemoglobin (Hemolytic anemia).
- Myoglobin (Rhabdomyolysis).
- Bilirubin Nephropathy in obstructive jaundice**.**

Exogenous :

- Antibiotics: Aminoglycosides, Amphotercin B
- NSAIDs
- Radio-contrast dye
- Heavy metals: **A**rsenic, **M**ercury.

Acute tubular necrosis

- **Pathophysiology : 3 stages**
  1. **Oliguric phase :** *Lasts few hours to weeks:*
     - Oliguria (↓ urine output < 400 cc/d) due to :
       - a. Obstruction of tubules by epithelial casts.
       - b. Afferent vasoconstriction due to excess renin activation.
     - **Na** : decreased (dilutional hyponatremia)
     - **K** : Hyperkalemia.
     - **Phosphorous** : increased
     - **Ca** : ↓ or may still normal.
     - **H+ ions, urea & creatinine** : ↑↑

Acute tubular necrosis

Pathophysiology

2. Diuretic phase

- Polyuria in this phase is due to **partial** recovery of the tubules (incomplete power of the tubules to reabsorb the filtered water)
- The kidney tubules still unable to concentrate urine).
- oThe urine output increases (up to 10 L/d) but it may take several days before the creatinine starts to drop.
- Dehydration & hypokalemia may occur

Acute tubular necrosis

Pathophysiology

3. Post diuretic phase:

- Convalescent phase
- may takes weeks or months
- there is complete recovery with improvement of the general health
- normalization of the chemistry occur

Clinical picture

1. Oliguric phase : (2-4 weeks)

A. **Oliguria : ↓ urine output < 400 cc/d.**

B. **Manifestations of hyporvolemia : in cases of acute renal failure due to pre renal causes** e.g. marked reduction of blood pressure with oligurea, decreased skin turger, reduced jugular venous pressure and dry mucous membranes.

C. **Manifestations of hypervolemia : in cases of acute renal failure due to intrinsic renal disease**

- **Headache.**
- **Congested neck vein.**
- **Pulmonary edema.**
- **Hypertension.**
- **Edema lower limb.**

Clinical picture

1. **Oliguric phase :**
   - D. **Manifestations of hyponatremia :** Convulsion, Confusion, Coma.
   - E. **Manifestations of hyperkalemia :**
     - Skeletal muscle : weakness, flaccid paralysis, areflexia.
     - Smooth muscle : Intestinal colic, nausea, vomiting.
     - Cardiac muscle : Arrhythmias & can progress to cardiac arrest.
   - F. **Manifestations of uremia** :
     - Anorexia, nausea, vomiting.
     - Pericarditis, Bleeding.
     - Convulsion, Confusion, Coma.

Clinical picture

2. Diuretic phase :

- Improvement of general condition.
- Urine out put 3-5 L /d
- Dehydration & hypokalemia may occur

3. Post diuretic phase : *Convalescent phase*

- Improvement of the general health & normalization of the chemistry
- It may takes weeks or months.
- Urine output becomes normal volume, concentrated.

Investigations

1. To diagnose ARF

A. Urine examination:

- Oliguria with low fixed specific gravity

B. Biochemical changes :

- Azotemia : ↑ Urea & creatinine.
- Creatinine clearance :↓
- Na : ↓ (dilutional hyponatremia)
- K : ↑
- PO4 : ↑ & ↓ $\boldsymbol{Ca}$
- ↓ Hco3

C. Ultrasound : Normal diameter.

Investigations

1. **To Diagnose the cause :**
   A. Urinary Indices

Laboratory test	Prerenal azotemia	ARF
Urine osmolality (mOsm/kg)	>500	<400
Urine sodium level (mEq/l)	<20	>40
Urine/plasma creatinine ratio	>40	<20
Fractional excretion of sodium (%)	<1	>2
Fractional excretion of urea (%)	<35	>35

Fractional Excretion of Sodium

$$FE_{Na} = 100 \times \frac{\text{sodium}_{\text{urinary}} \times \text{creatinine}_{\text{plasma}}}{\text{sodium}_{\text{plasma}} \times \text{creatinine}_{\text{urinary}}}$$

- **FENa < 1 indicates an intact tubular function.**
- **FENa > 1 indicates euvolemia or tubular dysfunction.**

Investigations

B. In renal causes :

- Muddy-brown granular cast : in ATN.
- Pigmented cast in myoglobinuria.
- White cells cast in AIN.
- Red cell casts : in AGN
- Pus cells : in acute pyelonephritis

C. In post renal causes : to detect the cause of obstruction

- Rerograde pyelography & angiography.
- Plain film of the abdomen : to detect the stones.

Indications of Renal Biopsy in AKI

1. **Clinical and laboratory findings suggesting Glomerular or Interstitial nephritis**

2. **Unknown cause**

3. **Prolonged ATN**

Treatment

1. Oliguric phase :

- The aim of management is to keep the patient alive until spontaneous recovery of renal function occurs.
- Urinary catheter : to assess urine volume & to exclude post-renal cause.
- Central venous catheter : to assess the blood volume.
- Immediate volume infusion to prevent ATN.
- Fluid balance l.e, daily fluid intake should equal urine output plus losses from fistulae and from vomiting plus an allowance of 500 ml daily for insensible loss.
- Acidosis treated with I.V sodium bicarbonate.

Treatment

1. Oliguric phase : (cont.)

- Control blood pressure.
- **Diet**
  - Sodium and potassium restriction with rare exception.
  - Protein restriction to 40 gm/day if it is hoped to avoid dialysis.
  - Patients treated by dialysis must receive 70 gm/day protein.
  - Hypercatabolic patients will require an even higher nitrogen intake to prevent negative nitrogen balance.
- **Hyperkalemia** :
  - Acidosis correction : $NaHCO_3$.
  - Ca gluconate (slowly infusion)
  - Insulin + glucose ↑intracellular shift of K.
  - Dialysis.

Treatment

1. **Oliguric phase :**

- Indication of dialysis in patients with ARF
  - Bad general condition (severe symptoms)
  - Marked acidosis
  - Serum K > 7 or if not controlled by the conservative measures
  - Pulmonary edema
  - pericarditis

Treatment

2. **Diuretic phase**
   - Adequate fluid to avoid dehydration.
   - Na and K supplements.

THANKS

AKI Definition and staging:

- Most recently the international guideline group, Kidney Disease: Improving Global Outcomes **(KDIGO)** has made a definition and staging system
- **KDIGO Definition of AKI**
  - Increase in SCr by ≥ **0.3** mg/dl within **48** hours; or
  - Increase in SCr to ≥ **1.5** times baseline, which is known or presumed to have occurred within the prior **7** days; or
  - Urine volume **< 0.5** ml/kg/h for **6** hours.

Staging of AKI

- Patients that meet the definition for AKI can be staged according to whichever criteria (serum creatinine or urine output) gives them the highest stage.

Stage	Serum creatinine	Urine output
1	1.5–1.9 times baseline OR ⩾0.3 mg/dl (⩾26.5 μmol/l) increase	<0.5 ml/kg/h for 6–12 hours
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ⩾12 hours
3	3.0 times baseline OR Increase in serum creatinine to ⩾4.0 mg/dl (⩾353.6 μmol/l) OR Initiation of renal replacement therapy OR, In patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m^2	<0.3 ml/kg/h for ⩾24 hours OR Anuria for ⩾12 hours