Acute Kidney Injury (AKI)

- Characterized by decrease in kidney function within hours to days resulting in retention of urea Nitrogenous waste (Ammonia & Uric acid) & Dysregulation of Extracellular Volume and Electrolytes.
- It was earlier said as Acute Renal failure.
- It is defined as rise in serum creatinine by 0.3mg/dl or 50% higher than baseline within 24-48 hrs or a Reduction in Urine output less than 0.5ml/Kg/hours for longer than 6 hours.

Criteria for Diagnosis:

Kidney Disease Improving Global outcomes (KDIGO)

Stages	↑ Serum Creatinine	Decrease in Urine output	Time
1	1.5 times or ≥ 0.3mg/dl	≤ 0.5ml/Kg/hour	6 hrs
2.	2 times or	≤ 0.5ml/Kg/hr	12 hrs
3.	3 times or Value of ≥ 4mg/dl	≤ 0.3ml/Kg/hr Anuria	24 hrs. 12 hrs; or Renal Replacement therapy

Causes:

i) Pre Renal
ii) Renal
iii) Post Renal

i) Pre Renal AKI

a) Hypovolumeic state

- Acute haemorrhage
- GI loss (vomiting + Diarrhea)
- Renal loss (Hyperglycemia)
- Dermal loss (Burns)
- "Third spacing" - Sequestrating fluid (Pancreatitis or Sepsis)

b) Hypervolumic state

- Pump failure (Cardio renal syndrome)
- Hypoalbuminemia - Decompensated liver Disease (Hepato renal syndrome)

c) Medications - NSAIDs, ACE inhibitors, ARBs.

ii) Intra Renal AKI

a) Tubular

① Acute Tubular Necrosis by:
- Ischemia

② Nephrotoxins
- Heavy metals
- Myoglobin
- Ethylene Glycol
- Radiocontrast Dyes

b) Glomerular

① Isolated Cases Haematuria
- IgA Nephropathy

② Nephrotic Syndrome
- Membranous Nephropathy
- Membranoproliferative
- FSGS, Minimal change disease

③ Nephritis Syndrome
- PSGN, IgA, SLE, Infective Endocarditis
- Shunt nephritis

c) Interstitium

- NSAIDS, drugs
- Penicillin
- Diuretics

d) Vascular

① Renal Artery stenosis
- Atherosclerosis
- Fibromuscular dysplasia

iii) Postrenal:

- Outside → Tumour from abdomen, Cervical Cancer.
  Benign Prostatic Hyperplasia & Cancer. Rectal mass
  Inside → Kidney stones, strictures, Neurogenic Bladder.

Pathophysiology

- In Pre-Renal → Hypoperfusion → Acute Tubular Necrosis → Inflammation, Basement membrane break and interstitial odema.
→ Renal → Nephrotoxin by Type I & IV hypersensitivity. Damage to Glomerulus.
→ Post Renal → Due to Build up of Urine and Pressure → ↓ GFR → Chronic Renal Injury.

Clinical Features:

① Pre Renal Kidney Injury

→ Hypovolemic state → Tachycardia, Hypotension. Cold extremities, ~~Reduced~~ ↑ Skin Turgor
→ Hypervolemic state → Cardiorenal syndrome; ↓ Hypotension, Fatigue, Dyspnea, Peripheral odema
Hepatorenal → Ascites, Caput medusa, Peripheral odema, Splenomegaly, Hypotension.

② Renal

→ Tubular → Ethylene glycol → Confusion, dilated pupils.
Myoglobin → Muscle injury recently.

→ Glomerular → Nephrotic syndrome → Proteinuria, Hypoalbuminia, Odema, Hyperlidemia, lipiduria.

Nephritic syndrome → oliguria, Hypertension, Hematuria, odema, Proteinuria. SLE, and Infective Endocarditis symptoms.

→ Interstitium → Fever and rash.

→ Vascular → Arterial hypertension despite taking medications,

3) Post Renal

outside → Frequency, Urgency, Heritancy, weightloss, Fatigue.

Inside → Renal colic, Anuria.

Laboratory Investigation

1) Pre-Renal.

- Renin Angiotensin system is activated → H_2O & Sodium retention.

BUN : Creatinine	Urine Osmolality	Urine Sodium Excretion	FENa
20 : 1	>500 mosm/kg	<20 m Eq/L	<1%.

2) Renal - I

- Filtration, Secretion, Reabsorption affected.

BUN : Creatinine	Urine Osmolality	Urine Sodium Excretion	FENa
Below 20 : 1	<500 mosm/kg	>40 m Eq/L	>1%.

* Tubular → Microscopy

Glomerular → Haematuria & Proteinuria, 24 hr urine collection, Serology, Kidney Biopsy.

Interstitium → Eosinophilia, Urine dipstick → Haematuria, proteinuria, pyuria.

Microscopy → WBC, Casts, RBC.

3) Post renal AKI:

If obstruction present:

BUN : Creatine	Urine Osmolality	Urine Sodium Excretion	FENa.
Below 20:1	< 500 mOsm/kg	740 mEq/L	> 1%.

If the Tubes are functioning normally.

BUN : Creatinine	Urine Osmolality	Urine Sodium Excretion	FENa
20:1	> 500 Osm/kg	< 20 mEq/L	< 1%.

- Serum Prostate Specific antigen.
- PAP Smear.
- USG Abdomen, Non-Contrast CT Scan of abdomen.

Complications:

① Volume overload ② Metabolic acidosis ③ Hyperkalemia.
④ Uremic pericarditis (Pericardial Effusion) ⑤ Infection.

New Marker of AKI:

→ Cystatin present in Urine is a Marker.

Management:

- Treatment of underlying cause.
- Maintenance of fluid and electrolyte balance.
- Nutritional status, Monitoring life threatening complication.
- Renal Replacement therapy.

Diet:

Avoid Potassium, Protein, Sodium, phosphorus rich diet.
Consume → High caloric diet (Carbohydrate + Fat),