# AKI

# AKI

- AKI is a clinical manifestation of underlying diseases
- Acute impairment of kidney function, resulting in the retention of urea and other nitrogenous waste products and metabolic derangement (dysregulation of extracellular volume and electrolytes)
- In patients <18 years, stage 3 AKI is also defined by KDIGO as a decrease in estimated glomerular filtration rate (eGFR) to <35 mL/min/1.73 m2.

| | RIFLE[1] | AKIN[2] | KDIGO[3] |
|---|---|---|---|
| **Diagnostic criteria*** | | | |
| | | Increase in serum creatinine of ≥0.3 mg/dL or ≥50% within 48 hours<br><br>OR<br><br>Urine output of <0.5 mL/kg/hour for >6 hours | Increase in serum creatinine of ≥0.3 mg/dL within 48 hours or ≥50% within 7 days<br><br>OR<br><br>Urine output of <0.5 mL/kg/hour for >6 hours |

| | | | |
|---|---|---|---|
| **Staging criteria** | | | |
| Risk (RIFLE) or stage 1 (AKIN/KDIGO) | Increase in serum creatinine to 1.5 times baseline<br>OR<br>Urine output of <0.5 mL/kg/hour for 6 to 12 hours | Increase in serum creatinine of ≥0.3 mg/dL or to 150 to 200% baseline<br>OR<br>Urine output of <0.5 mL/kg/hour for 6 to 12 hours | Increase in serum creatinine of ≥0.3 mg/dL or 1.5 to 1.9 times baseline<br>OR<br>Urine output of <0.5 mL/kg/hour for 6 to 12 hours |
| Injury (RIFLE) or stage 2 (AKIN/KDIGO) | Increase in serum creatinine to 2 times baseline<br>OR<br>Urine output of <0.5 mL/kg/hour for 12 to 24 hours | Increase in serum creatinine to 200 to 300% baseline<br>OR<br>Urine output of <0.5 mL/kg/hour for 12 to 24 hours | Increase in serum creatinine to 2 to 2.9 times baseline<br>OR<br>Urine output of <0.5 mL/kg/hour for 12 to 24 hours |
| Failure (RIFLE) or stage 3 (AKIN/KDIGO) | Increase in serum creatinine to 3 times baseline<br>OR | Increase in serum creatinine to >300% baseline<br>OR | Increase in serum creatinine to ≥3 times baseline<br>OR |
| | Increase in serum creatinine by >0.5 mg/dL to >4 mg/dL<br>OR<br>Urine output of <0.3 mL/kg/hour for >24 hours or anuria for >12 hours<br>OR<br>Initiation of kidney replacement therapy | Increase in serum creatinine by >0.5 mg/dL to ≥4 mg/dL<br>OR<br>Urine output of <0.3 mL/kg/hour for >24 hours or anuria for >12 hours<br>OR<br>Initiation of kidney replacement therapy | Increase in serum creatinine of ≥0.3 mg/dL to ≥4 mg/dL¶<br>OR<br>Urine output of <0.3 mL/kg/hour for ≥24 hours or anuria for ≥12 hours<br>OR<br>Initiation of kidney replacement therapy |
| Loss (RIFLE) | Need for kidney replacement therapy for >4 weeks | | |
| End stage (RIFLE) | Need for kidney replacement therapy for >3 months | | |

# AKI

- The KDIGO define AKI as follows:
  - Increase in serum creatinine by **≥0.3 mg/dL within 48 hours**, or
  - Increase in serum creatinine **to ≥1.5 times baseline** occurred within the prior **seven days**, or
  - Reduction in urine volume <0.5 mL/kg/hour for six hours

# KDIGO Staging

| Stage | S creatinine | Urine output |
|---|---|---|
| 1 | 1.5-1.9 times baseline<br>or ≥ 0.3 mg/dl increase | <0.5 ml/kg/hour<br>for 6-12 hours |
| 2 | 2.0-2.9 times baseline | <0.5 ml/kg/hour<br>for ≥12 hours |
| 3 | 3 times baseline or<br>S creatinine ≥ 4 mg/dl | ◆ <0.3 ml/kg/hr for ≥ 24 hours or<br>◆ Anuria for ≥12 hours |

# KDIGO STAGE

| Stage | Serum Creatinine | Urine output |
|---|---|---|
| Stage I | Increase in serum creatinine of ≥0.3 mg/dL or 1.5 to 1.9 times baseline | Urine output of <0.5 mL/kg/hour for 6 to 12 hours |
| Stage II | Increase in serum creatinine to 2 to 2.9 times baseline | Urine output of <0.5 mL/kg/hour for 12 to 24 hours |
| Stage III | Increase in serum creatinine to ≥3 times baseline<br><br>OR,<br><br>Increase in serum creatinine of ≥0.3 mg/dL to ≥4 mg/Dl | Urine output of <0.3 mL/kg/hour for ≥24 hours or anuria for ≥12 hours<br><br>OR,<br><br>Initiation of kidney replacement therapy |

# Causes of AKI

Pre-renal

Renal/Intrinsic

Postrenal

# Prerenal causes

- Hypovolemia: Acute diarrhoea, vomiting, burn, sepsis, haemorrhage, diabetic ketoacidosis
- Congestive heart failure
- Perinatal asphyxia
- Third space loss: septicemia, nephrotic syndrome
- Drugs: Diuretics, ACE inhibitors

# Renal/Intrinsic causes

- Vascular: HUS, Vasculitis, renal vein thrombosis
- Tubular
  - Acute tubular necrosis (ATN)
  - Wasp sting, snake venom
  - Nephrotoxic drug e.g. diethyl glycol, methanol
  - Tumor lysis syndrome (uric acid crystals tubular obstruction)
- Glomerulonephritis
  - Post infectious GN
  - Membranous proliferative GN
  - Systemic disorder: SLE, Henoch-Schonlein syndrome, Microscopic polyangiitis
- Interstitial: Interstitial nephritis, pyelonephritis
- Medications: aminoglycosides, radiocontrast, amphotericin B, ACE inhibitor, Indomethacin, NSAIDs

# Post renal causes

- Urinary obstruction:
  - Posterior urethral valves, Urethral stricture
  - Bilateral UPJ obstruction
  - Blood clot in the urinary tract
  - **Neurogenic bladder**

# Pathogenesis

- Rapid decline in GFR which results in
  - Accumulation of nitrogenous wastes in the body
    - Elevation of blood urea, creatinine, blood urea nitrogen (BUN)
  - Impairment of water, electrolytes and acid-base balances
    - Dyselectrolytaemia e.g. hyperkalaemia
    - Acid-base imbalance e.g. metabolic acidosis
    - Fluid overload, hypertension

# Clinical features

## Hallmark of AKI

- Scanty urine (oliguria)
- Complete cessation of urine (anuria)

## Other manifestations

- Vomiting
- Convulsions

# Approach of AKI

- History
  - H/o anuria, oliguria, vomiting, or blood loss
  - Assessment of fluid intake in the previous 24 hours
  - History to find out the causes –
    - Fluid loss e.g. diarrhoea, severe vomiting
    - Pre-existing kidney disease e.g. AGN, NS
    - Ingestion of nephrotoxic drug e.g. diethyl glycol in paracetamol, aminoglycoside

# Examination

Features of fluid overload e.g.

- Facial puffiness, oedema, hypertension
- Heart failure (hepatomegaly, pulmonary oedema)

Features of severe dehydration e.g.

- Drowsiness
- Skin pinch not going back quickly

Toxic features of AKI e.g. unconsciousness, arrhythmia, vomiting, convulsion

Flank: Palpable renal mass (renal vein thrombosis)

Urinary bladder: Palpable ( PUV)

Haemodynamic status e.g. pulse, BP, capillary refill time

# Investigations

CBC: Hb (reduced), Leukopenia (sepsis), Thrombocytopenia (HUS)

Blood biochemistry

- S creatinine, urea, BUN: Raised
- S electrolytes: High K+, low $HCO_3^-$ Low Na+
- S calcium (low), Phosphate (high)
- ABG: Metabolic acidosis

Urine R/M/E: RBC, protein, crystal, granular cast

Blood: ASO titre, C3 , C4, ANA, Anti-ds DNA, Ab to GBM

# Investigations

Chest X-Ray: Cardiomegaly, pulmonary congestion, pleural effusion

Renal ultrasound scan:

To rule out UTI obstruction

Kidney morphology

Renal biopsy

ECG

# Treatment

01

Hospitalize the child

02

Counsel the parents about the illness

03

Discontinue nephrotoxic drug, if any

04

Introduce, a catheter when suspected PUV, and

05

Monitor urine output

# Treatment

FLUID RESUSCITATION

TREATMENT OF THE UNDERLYING CAUSE OF AKI

MANAGEMENT OF ASSOCIATED CONDITIONS

NUTRITION SUPPORT

DIALYSIS

RENAL REPLACEMENT THERAPY

# Fluid resuscitation

**No volume overload or CCF**

- NS, 20 mL/kg over 30 minutes (hypovolemic patient generally void within 2 hours)

**Hypotension due to sepsis**

- IV fluid along with continuous infusion of **vasopressor**

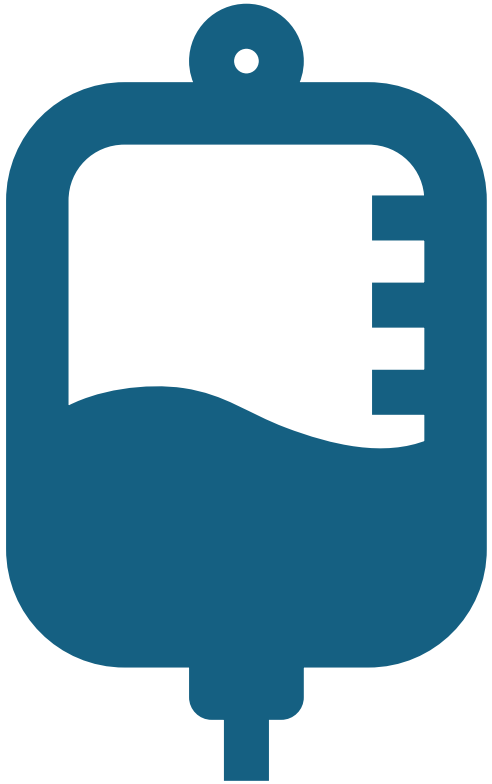
**Adequate circulatory volume is established/pulmonary oedema**

- O2 inhalation, propped-up position
- Furosemide (2-4 mg/kg)- single IV

**No urine output with this single dose of frusemide**

- Continuous diuretic infusion ± Injection Dopamine (2-3 g/kg/min) for renal cortical blood flow

# Fluid resuscitation



- If still no response to the diuretic challenge
  - Stop giving diuretics
  - Restrict fluid to 400 mL/m2 /day (insensible loss) + previous day urine output
  - Replace any external loss (blood, GIT) meticulously with appropriate fluid
  - Readjust fluid allocation, if volume overload
  - Monitor intake-output, body weight and S chemistries daily

# Treatment of underlying causes

- Treat underlying causes if any

# Management of associated conditions

- Hyperkalemia
  - Calcium gluconate 10% IV 0.5-1 ml/kg over 5-10 minutes
  - Salbutamol 5-10 mg nebulized
  - Sodium bicarbonate 7.5% 1-2 ml/kg over 15 minutes
  - Dextrose 10% 0.5-1 g/kg and insulin 0.1-0.2 U/kg IV
  - Calcium or sodium resonium (Kayexalate) 1 g/kg/day

# Management of associated conditions

- Metabolic acidosis:
  - Sodium bicarbonate (1ml/kg), IV if less than 18 mEq/l
- Hypertension
  - Asymptomatic cases: Isradipine (0.05-0.15 mg/kg/dose) QID, nifedipine
  - Symptomatic cases e.g. hypertensive encephalopathy, Na Nitroprusside (0.5-10 µg/kg/min) or Labetalol (0.25-3.0 mg/kg/hour) by infusion under the supervision
  - Others: Amlodipine ( 0.1-0.6 mg/kg/day) BID
  - Labetelol (4-40 mg/kg/day)

# Management of associated conditions

- Hyponatremia
  - Fluid restriction
  - Sensorial alteration or seizures: 3% saline  6-12 ml/kg over 30-90 minutes
- Severe anemia
  - Packed cells 3-5 ml/kg; consider exchange transfusion
- GIT bleeding: IV Omeprazole
- Hypocalcaemia:
  - Lowering s PO4 by phosphate binder will help to improve s calcium. Injection Calcium gluconate should not be given until tetany (1-2 ml/kg)
- Hyperphosphatemia: Dietary restriction

# Nutritional support

- Encourage a high-calorie diet, rich in carbohydrates and fat to reduce protein catabolism
- Protein: 1-1.2 g/kg in infant, 0.8-1.2 g/kg in others
- Calories: 60-80 cal/kg/day
- Vitamin, and micronutrient supplements
- Restrict extra salt intake
- Avoid foods rich in potassium e.g. citrus fruits, tomato paste, chocolates and potato chips

# Dialysis

## Indications

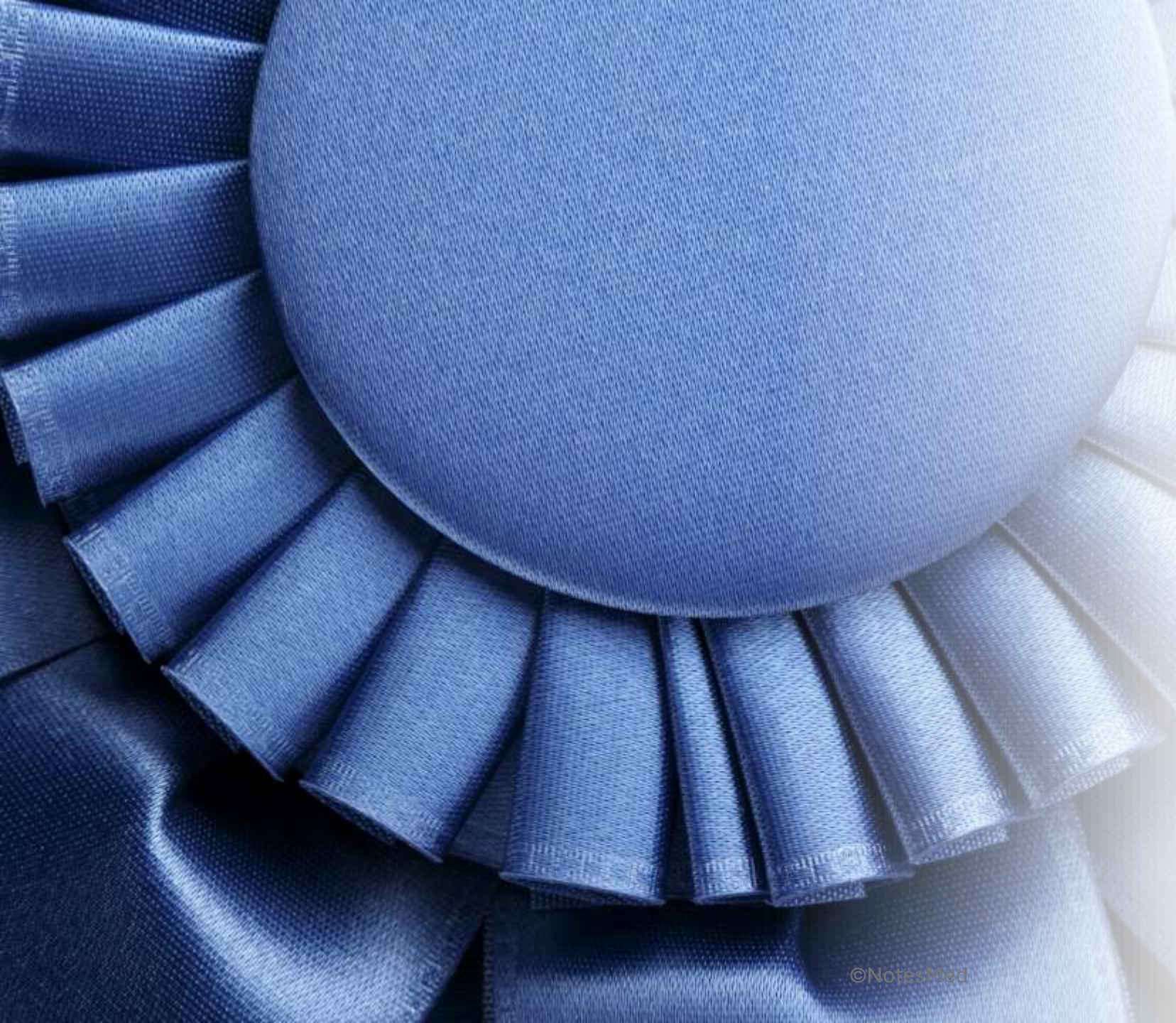
- Anuria/oliguria
- Volume overload with evidence of hypertension and/or pulmonary edema refractory to diuretic therapy
- Persistent hyperkalaemia
- Severe metabolic acidosis, unresponsive to medical management
- Uremia (encephalopathy, pericarditis, neuropathy)
- Calcium: Phosphorus imbalance, with hypocalcemic tetany that cannot be controlled by other measures
- Inability to provide adequate nutiritional intake because of the need for severe fluid restriction

# References


Ghai Essential Pediatrics-10th edition

Nelson Essential of Pediatrics-24th edition

UpToDate



# Thank You