Pediatric AKI & CKD: Final Review

1. Acute Kidney Injury (AKI) Basics

Definition: Abrupt loss of kidney function resulting in a decline in GFR, retention of urea/nitrogenous waste, and dysregulation of volume and electrolytes. Classified via pRIFLE or KDIGO criteria.

Biomarkers & Limitations

- **Serum Creatinine (SCr):** Poor early biomarker. Insensitive to small GFR changes (doesn't rise until ~50% function is lost). Delayed rise (up to 72 hours post-insult). Affected by age, sex, muscle mass, and volume.
- **Normal SCr ranges by age:** Newborn (0.3-1.0), Infant (0.2-0.4), Child (0.3-0.7), Adolescent (0.5-1.0).
- **Novel Biomarkers:** NGAL, KIM-1, IL-18, Cystatin C. (Promising for early detection, but costly and not yet standard everywhere).

Urine Output (UOP) Definitions

- **Oliguria (Infants):** < 1 mL/kg/hr.
- **Oliguria (Children/Adults):** < 0.5 mL/kg/hr for > 6 hours.
- **Anuria:** Zero urine output.
- *Note:* The majority of neonates with AKI actually have *nonoliguric* AKI.

2. Classifications of AKI

Prerenal AKI (Most Common)

Caused by reduced renal perfusion (e.g., hemorrhage, gastroenteritis, burns) or reduced effective volume (heart failure, shock). Renal tubular function remains intact, aggressively reabsorbing sodium and water to compensate.

> **MCQ PEARL: RENAL COMPENSATORY MECHANISMS**
>
> To maintain GFR during hypoperfusion, the kidney uses two main tools. **Examiners love testing the drugs that block these!**
>
> - **Prostaglandins** dilate the afferent arteriole to bring more blood IN. *Blocked by NSAIDs (Ibuprofen, Indomethacin).*
> - **Angiotensin II** constricts the efferent arteriole to block blood from leaving, increasing pressure. *Blocked by ACE inhibitors.*

> **MEMORY AID: PDA / ACE**
>
> **P**rostaglandins **D**ilate **A**fferent.
> **A**ngiotensin **C**onstricts **E**fferent.

Intrinsic AKI

Structural damage to the parenchyma. Most commonly Acute Tubular Necrosis (ATN) from prolonged hypoperfusion or sepsis. Also caused by glomerulonephritis (PSGN, HUS), interstitial nephritis, rhabdomyolysis, or nephrotoxins.

Postrenal AKI

Obstructive causes (e.g., Posterior Urethral Valves (PUV) in males, stones, strictures).

3. AKI Management & Emergencies

Treatment is primarily supportive and focused on maintaining perfusion and fixing electrolytes. There are no direct medications to "cure" established AKI.

Fluid Management

- **Hypovolemic:** 10-20 mL/kg Normal Saline bolus (over 30 mins, repeat x3 max). If no UOP, insert bladder catheter.
- **Euvolemic:** Furosemide challenge. Replace insensible losses (300-400 mL/m²/day) + urine/GI losses with 0.45% NS.
- **Hypervolemic:** Restrict fluids. Furosemide trial. Volume overload >20% is a critical threshold for poor prognosis and usually requires dialysis.

Hyperkalemia Management

Most pronounced in massive tissue breakdown (TLS, rhabdo). ECG changes progress predictably.

> **MEMORY AID: HYPERKALEMIA ECG CHANGES**
>
> Think of pulling a tent string straight UP from the middle of the ECG:
> 1. **T**all, peaked T waves (tent goes up).
> 2. Prolonged **P**R interval.
> 3. Flattened **P** waves (edges pull in).
> 4. Widened **Q**RS (base of tent widens).
> 5. Ventricular fibrillation.

Goal	Medication	Mechanism/Onset
Stabilize Myocardium	Calcium Gluconate	Immediate (Does NOT lower K+ level)

Shift K+ into Cells	Insulin + Glucose Sodium Bicarbonate Salbutamol (Albuterol)	30 minutes
Remove K+ from Body	Kayexalate (Polystyrene) Furosemide Hemodialysis	1 to 2 hours (Definitive treatment)

4. Indications for Renal Replacement Therapy (RRT)

> **MEMORY AID: AEIOU FOR DIALYSIS**
>
> - **A**cidosis: Severe, unresponsive to bicarb.
> - **E**lectrolytes: Refractory Hyperkalemia.
> - **I**ntoxication: Dialyzable toxins/nephrotoxins.
> - **O**verload: Fluid overload (>20%) with pulmonary edema/HTN unresponsive to diuretics.
> - **U**remia: BUN > 80-100 mg/dL, causing encephalopathy or pericarditis.

Dialysis Modalities

- **Peritoneal Dialysis (PD):** Therapy of choice in neonates and small infants (no vascular access needed, no systemic anticoagulation).
- **Hemodialysis (HD):** Hard for small kids due to large extracorporeal blood volume required.
- **CRRT:** Best for hemodynamically unstable PICU patients (prevents rapid fluid shifts).

5. Chronic Kidney Disease (CKD)

Definition: Structural/functional abnormality > 3 months (Cannot diagnose in children < 2 years). Incidence is higher in males due to Congenital Anomalies of the Kidney and Urinary Tract (CAKUT). Most common cause of death is Cardiovascular Disease.

Pathophysiology

Adaptive hyperfiltration occurs in remaining healthy nephrons -> This maintains normal SCr and electrolytes initially, but the high pressure eventually destroys the remaining glomeruli, leading to proteinuria and ESRD.

Stages of CKD (By GFR)

- **Stage 1:** ≥ 90 (Kidney damage, normal GFR)
- **Stage 2:** 60-89 (Mild)
- **Stage 3:** 30-59 (Moderate) - *Acidosis usually begins here (eGFR <30)*
- **Stage 4:** 15-29 (Severe)
- **Stage 5:** < 15 (Kidney Failure / ESRD)

6. CKD Complications & Management

Anemia of CKD

Universally seen in Stage 4 & 5. Due to primary deficiency of **Erythropoietin (EPO)** production by renal interstitial fibroblasts. Target Hb is 11-12 g/dL. Treat with synthetic EPO and iron.

Hypertension & Proteinuria

Treat with ACE inhibitors or ARBs. These are renoprotective as they lower intraglomerular pressure and slow disease progression.

CKD-Mineral and Bone Disorder (CKD-MBD)

Failure to excrete phosphorus initiates a vicious cycle leading to bone disease, fractures, and poor growth.

> **MCQ PEARL: THE CKD-MBD PATHOPHYSIOLOGY CASCADE**
>
> 1. Kidneys fail to excrete **Phosphorus** -> Hyperphosphatemia.
> 2. High Phosphorus triggers **FGF23** release.
> 3. FGF23 directly inhibits 1-alpha-hydroxylase in the kidney -> **Low Active Vitamin D (1,25-OH)**.
> 4. Low Vitamin D -> Decreased intestinal **Calcium** absorption -> Hypocalcemia.
> 5. Low Calcium and High Phosphorus directly stimulate the parathyroid gland -> **Secondary Hyperparathyroidism** (Bone resorption to free up calcium).
>
> **Treatment Order:** 1) Restrict dietary phosphate. 2) Calcium-based phosphate binders. 3) THEN give active Vitamin D (Calcitriol) to suppress PTH. *Check PTH every 3 months.*