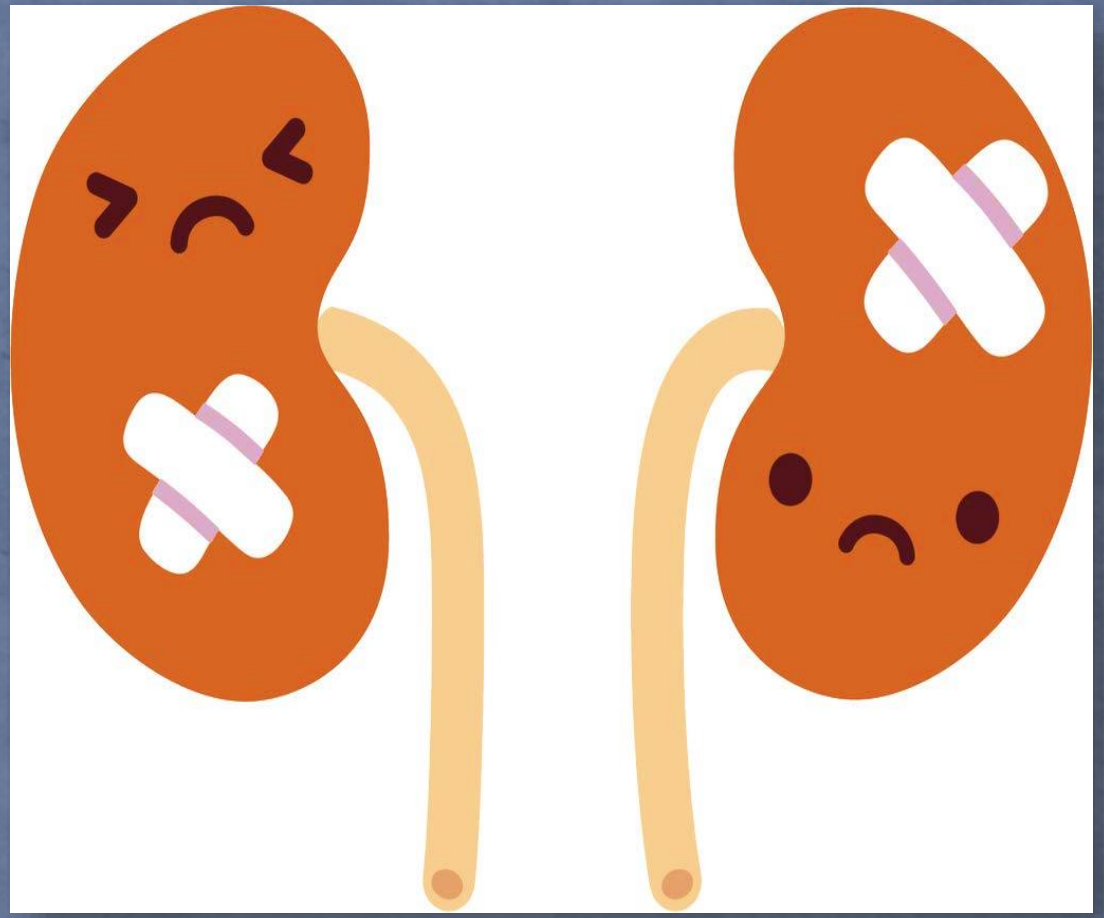
Acute Kidney Injury in children



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SUMS

Outlines

- Definition and classification
- Pathogenesis
- Case presentations
- General Management

Definition:

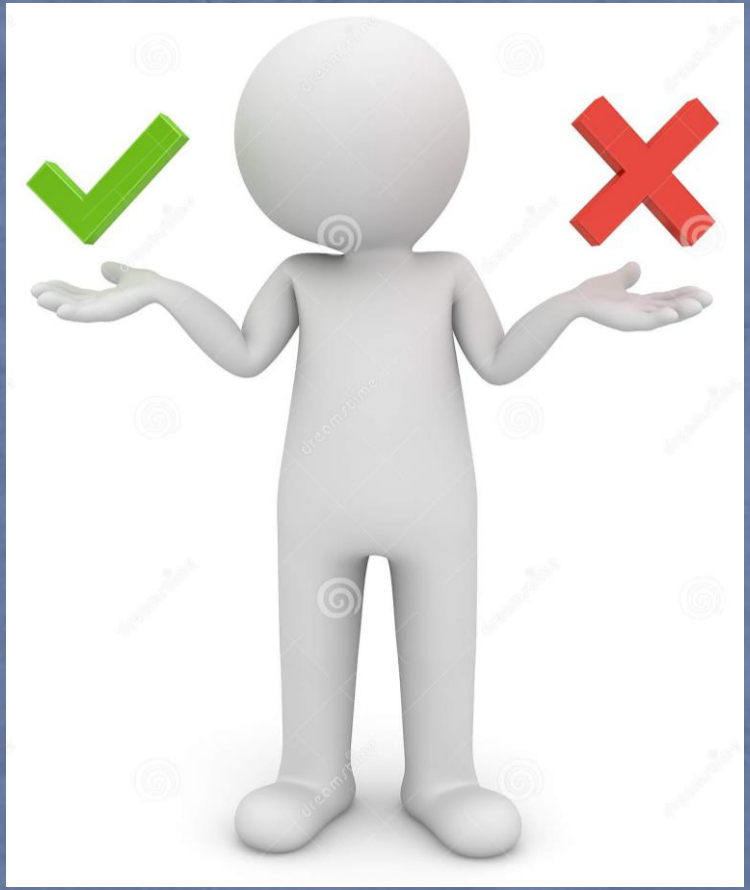
Abrupt loss of kidney function leading to a rapid decline in the GFR

- Accumulation of waste products
- Dysregulation of extracellular volume
- Electrolyte and acid base disturbance

AKI or ARF?

AKI:

Renal dysfunction that ranges from a small increase in serum creatinine to complete anuric renal failure



KDIGO classification:

Stage	Serum Cr	Urine output
1	1.5-1.9 times baseline, OR ≥0.3 mg/dL increase	<0.5 mL/kg/hr for 6-12 hr
2	2.0-2.9 times baseline	<0.5 mL/kg/hr for ≥ 12 hr
3	3.0 times baseline, OR SCr ≥ 4.0 mg/dL, OR Initiation of renal replacement therapy, OR eGFR < 35 mL/min per 1.73 m2 (< 18 yr)	<0.3 mL/kg/hr for ≥ 24 hr, OR Anuria for ≥ 12 hr

Normal ranges of serum creatinine values by age :

- Newborn – 0.3 to 1 mg/dL (27 to 88 micromol/L)
- Infant – 0.2 to 0.4 mg/dL (18 to 35 micromol/L)
- Child – 0.3 to 0.7 mg/dL (27 to 62 micromol/L)
- Adolescent – 0.5 to 1 mg/dL (44 to 88 micromol/L)

Limitations of serum Creatinine

- Creatinine is not a sensitive biomarker for tubular injury.
- Significant elevations are not apparent until 24-48 hours after the insult.
- Creatinine values is dependent to age, sex, muscle mass, nutrition.
- Creatinine can double or triple and remain within the laboratory normal range.

Despite these limitations, a relative change in serum creatinine remains the principal method of diagnosing AKI.

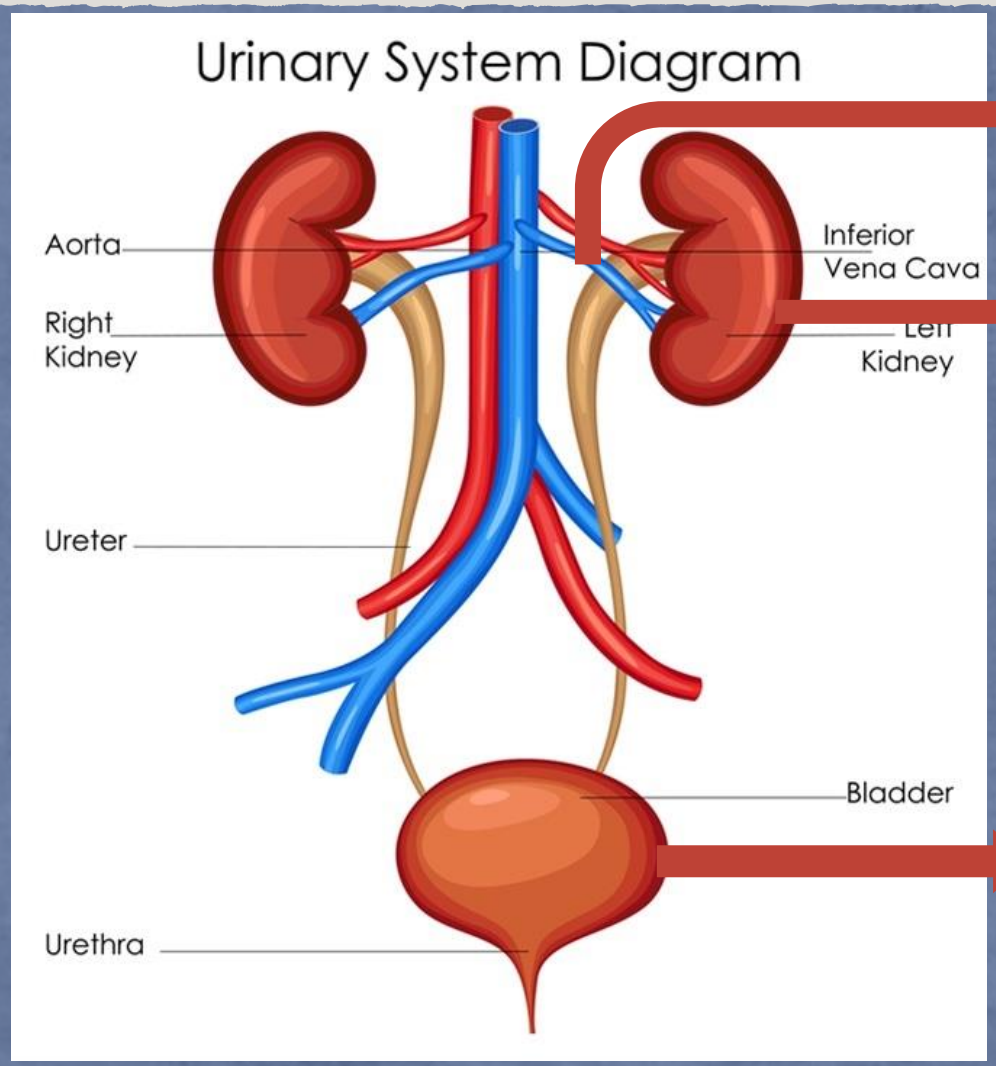
Newer biomarker

- Cystatin C
- Neutrophil gelatinase–associated lipocalin (NGAL)
- Interleukin 18
- Kidney injury molecule-1
- fibroblast growth factor 23 (FGF23)
- Insulin growth factor binding protein 7 (IGFBP-7)
- Tissue inhibitor of metalloproteinases 2 (TIMP-2)

Risk factors of AKI in children:

- **Critically ill patients in PICU**
- **Nephrotoxin Use:**
  - Antibiotics: aminoglycosides, vancomycin
  - Antiviral agents
  - Radiocontrast agents
  - Angiotensin-converting enzyme inhibitors
  - Calcineurin inhibitors
  - Non-steroidal anti-inflammatory drugs (NSAIDs)

Pathogenesis:



Pre renal (Dehydration, heart failure, Sepsis, hypoalbuminemia)

Renal

- **Glomerular**
- **vascular**
- **Tubular**
- **Interstitial**

Post renal

- Extraluminal
- Intra luminal

Diagnosis:

- History
- Physical examination
- Para clinics:
  - **Urine analysis**
  - **Fractional excretion of sodium**
  - **Kidney sonography (**kidney size, renal parenchymal survey, obstruction, vessels)
  - **Kidney biopsy (** in most cases of acute GN and unknown causes of AKI)

Table 550.4 Urinalysis, Urine Chemistries, and Osmolality in Acute Kidney Injury

	HYPOVOLEMIA	ACUTE TUBULAR NECROSIS	ACUTE INTERSTITIAL NEPHRITIS	GLOMERULONEPHRITIS	OBSTRUCTION
Sediment	Bland, may have hyaline casts	Broad, brownish granular casts	White blood cells, eosinophils, cellular casts	Red blood cells, red blood cell casts	Bland or bloody
Protein	None or low	None or low	Minimal but may be increased with NSAIDs	Increased, > 100 mg/dL	Low
Urine sodium (mEq/L)*	<20	>40	>30	<20	<20 (acute) >40 (few days)
Urine osmolality (mOsm/kg)	>400	<350	<350	>400	<350
Fractional excretion of sodium %†	<1	>2‡	Varies	<1	<1 (acute) >1 (few days)

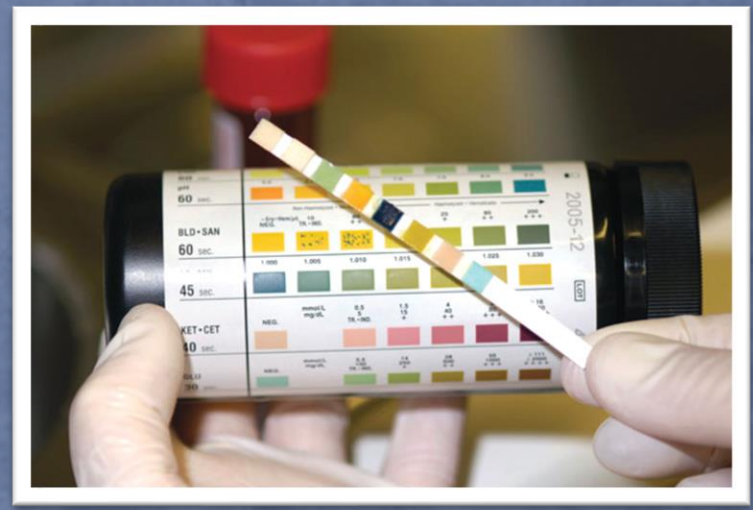
Case 1:

2 years old boy with history of gastroenteritis and bloody diarrhea since 2 days before admission. Patient has decreased urine output. In Physical examination patient has severe dehydration and lab data shows : BUN: 45 mg/dl , Cr:1.8 mg/dl

How can you differentiate a prerenal vs renal cause (HUS)?

The urinalysis is normal prerenal AKI (bland U/a)

So normal urinalysis does not R/o AKI



Case 2:

- An 8 years old boy has been admitted due to headache, flank pain and tea color urine since 3 days ago. He has history of sore throat 2 weeks before.
- In PE the pt had mild periorbital edema, BP: 140/80
- lab data :
- WBC:7500, Hb:10.4, Plt: 290,000
- BUN:35 mg/dl, Cr:2 mg/dl, Na:136 mEq/L, K:6.1 mEq/L
- U/a : 3+ blood, 1+ Pro, RBC: many

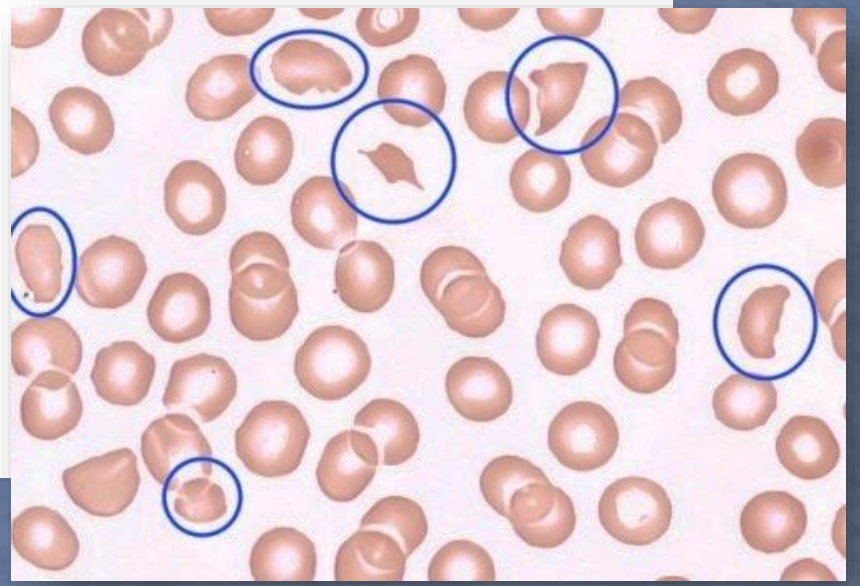
What is your diagnosis and management?

Dx: PSGN
Treatment plan:

- ✓ *Salt and fluid restriction*
- ✓ *Controlling hypertension: Loop diuretic*
- ✓ *Correction of hyperkalemia*

Case 3:

- 1.5 years old boy with edema and irritability following an episode of gastroenteritis. The patient has become anuric since last night .
- P/E: Periorbital edema, BP: 130/85
- Lab data : Bun : 60 mg/dl, Cr:4 mg/dl , Na: 135mEq/L, K: 7.3 mEq/L
- Hb: 6.5, Plt : 60,000
- U/a: 2+ blood, 2+ pro, RBC: 20-25
- What is your probable diagnosis?



Case 4:

- A 8 years old girl with nausea, vomiting and abdominal pain. Recently she had a dentist visit and has been taking Ibuprofen to control tooth pain.
- P/E: Nothing significant except flank tenderness
- Labdata:BUN:45mg/dl, Cr:2.5 mg/dl;
- U/a: SG:1007, 1+ pro, WBC:10-12 , leukocyte esterase :1+

Proteinuria is minimal in most cases of Acute TIN but may be increased with the use of NSAIDs

Case 5:

- 13 years old boy has been admitted due to fever, flu like symptoms and generalized body pain.
- P/E: tachypnea, tachycardia , normal blood pressure
- O2 sat: 87% at room air
- Lab data: BUN: 60 mg/dl, Cr: 2.3 mg/dl, Na:139 mg/dl, K: 5.6 mg/dl
- CXR: bilateral infiltration
- Nasopharyngeal swab for SARS COV2: +ve

Both COVID 19 infection and MIS-C are risk factors for acute kidney injury

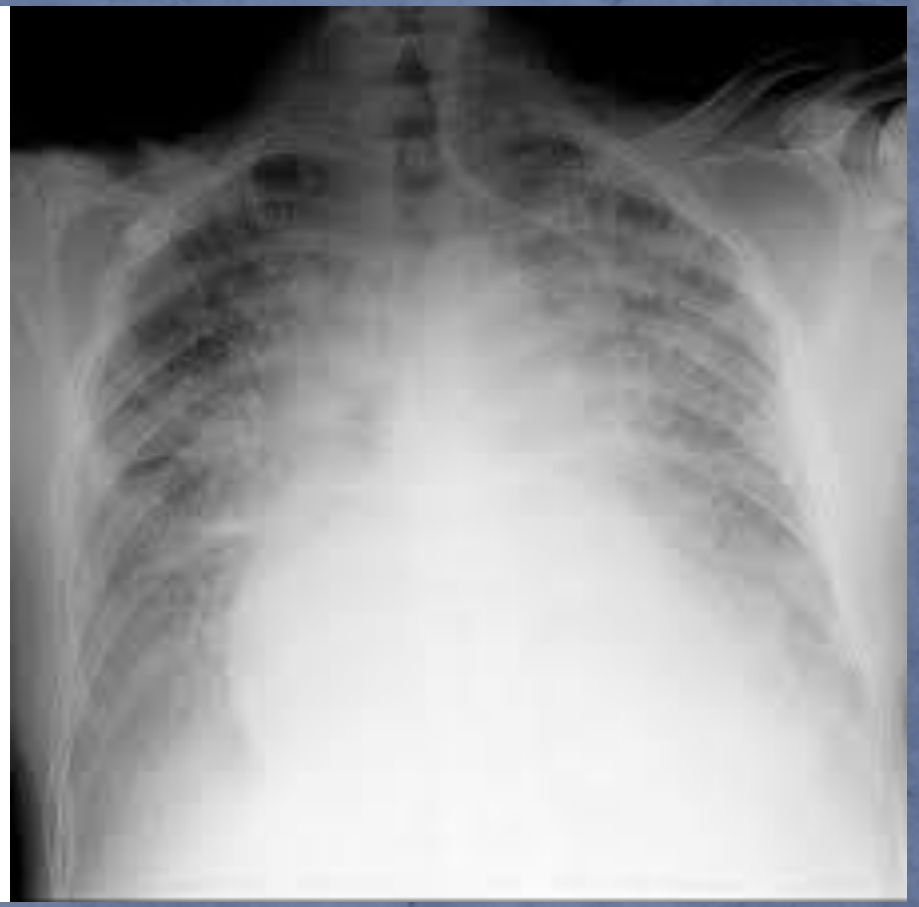
Case 6:

- A 7 years old girl who has been diagnosed as T cell ALL has developed oliguria following chemotherapy.
- Lab data: BUN: 40 mg/dl, Cr: 2.6mg/dl , K: 6.8 mmg/dl, Ca: 6.5 mg/dl, Phos: 9.1 mg/dl, uric acid: 8.5 mg/dl
- What is your diagnosis?

Tumor lysis syndrome

Case 7:

- A 6 years old girl was admitted in pediatric surgery ward due to abdominal pain and irritability. She was consulted due to increased BUN& Cr.
- In P/E the patient had tachypnea, tachycardia, hepatomegaly and cold extremities.
- Lab data: AST:1300, ALT: 20, BUN: 35 mg/dl ;Cr: 1.9 mg/dl; Na: 132 mEq/L; K: 5 mEq/L.
- u/a: Normal , SG: 1018
- Echocardiography: Ejection fraction 10% , dilated chambers



Prevention of AKI:

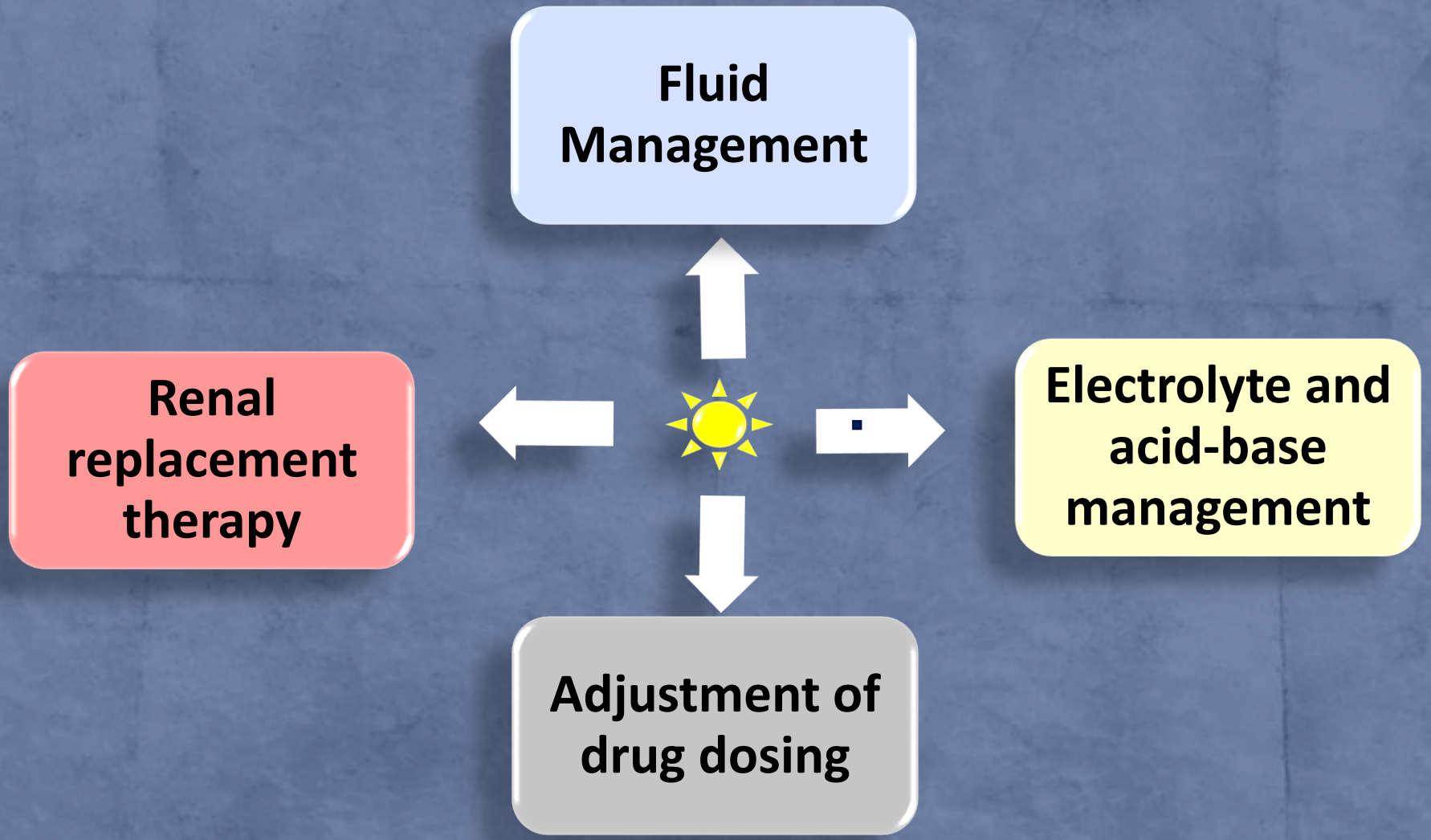
Proven measures:

- **Fluid administration** in settings, such as hypovolemia
- **Avoidance of hypotension** (inotropic support in critically-ill children following adequate volume repletion)
- **Readjustment and substitution of nephrotoxic medications** (close monitoring of kidney function and drug levels)

Unproven measure:

- Mannitol?
- Loop Diuretics ?
- Low-dose Dopamine?
- Fenoldopam?
- Atrial Natriuretic Peptide?
- N-acetylcysteine?

General Management:



Fluid Management

Hypovolemia:

- Adequate hydration with 10-20 cc/kg bolus of isotonic saline
- Consider vasopressin in hypotensive pts.
- Diuretic therapy only after adequate hydration; single high dose bolus (2-5 mg/kg Lasix, max 200 mg)
- If diuretic bolus was effective, →Lasix infusion (0.1-0.3 mg/kg/hr)
- Low dose Dopamine?

Euvolemia

- 10-20 cc/kg of isotonic saline
- Diuretic only after adequate hydration (2-5 mg/kg Lasix)
- Lasix infusion
- Low dose dopamine?
- IV: IWL+ replace output

Volume overload

- Fluid restriction and fluid removal

Electrolyte and acid-base management

Hyperkalemia:

K:6-7 mEq /L

- **None emergent management:**
  - ECG: peaked T wave, widening of the QRS intervals, ST segment depression, ventricular arrhythmias, and cardiac arrest
  - DC dietary sources and K+ in IV fluid
  - DC medications causing hyperkalemia
  - Start Sodium polystyrene sulfonate resin (**Kayexalate**), 1 g/kg (orally/ retention enema)
    - Each 1 gr/kg decrease 1mEq of serum potassium
    - Can be repeated every 2 hours

Electrolyte and acid-base management

Hyperkalemia:

- Symptomatic hyperkalemia (muscle weakness)
- ECG changes (other than tall T wave)
- K>7 mEq/L
- K :6-7 mEq/L but at risk of hyperkalemia (TLS)

- **Non emergent management**

+

- **Emergency management:**
  - **Calcium gluconate** 10% solution, 100 mg/kg/dose (maximum 3000 mg/ dose)
  - **Sodium bicarbonate**, 1-2 mEq/kg intravenously, over 5-10 min
  - **Regular insulin**, 0.1 units/kg, with glucose 50% solution, 1 mL/kg, over 1 hr
  - **Beta adrenergic agonists**

Metabolic acidosis:

- **Indication of bicarbonate therapy :**
  - **PH < 7.15; Bicarbonate < 8 mEq/L**
  - **Goal :arterial PH :7.20 ; Bicarbonate to 12 mEq/L**
- The remainder of the correction may be accomplished by oral administration of sodium bicarbonate
- Rapid correction of acidosis with IV bicarbonate → ↓ Ionized Ca→ tetany

Electrolyte and acid-base management

Hyponatremia:

- Most commonly dilutional → Corrected by fluid restriction rather than sodium chloride administration.

- Administration of hypertonic (3%) saline in symptomatic patients (seizures, lethargy) or those with a serum sodium level < 120 mEq/L.

Anemia

- Causes:
  - Hemodilution
  - HUS
  - SLE
  - Active bleeding

➢ **Considerations for PC transfusion:**

- ✓ **Hb<7 mg/dl**
- ✓ **Slow** 4-6 hr transfusion to prevent further volume expansion, hypertension, heart failure, and pulmonary edema.
- ✓ **Fresh** RBC minimizes the acute risk of hyperkalemia
- ✓ **Washed RBC** minimizes the chronic risk of sensitization for future renal replacement therapy.
- ✓ In severe hypervolemia or hyperkalemia→ **under dialysis or UF**

Indications of renal replacement therapy RRT:

- Anuria/oliguria
- Volume overload with evidence of hypertension and/or pulmonary edema refractory to diuretic therapy
- Persistent hyperkalemia
- Severe metabolic acidosis unresponsive to medical management
- Uremia (encephalopathy, pericarditis, neuropathy)

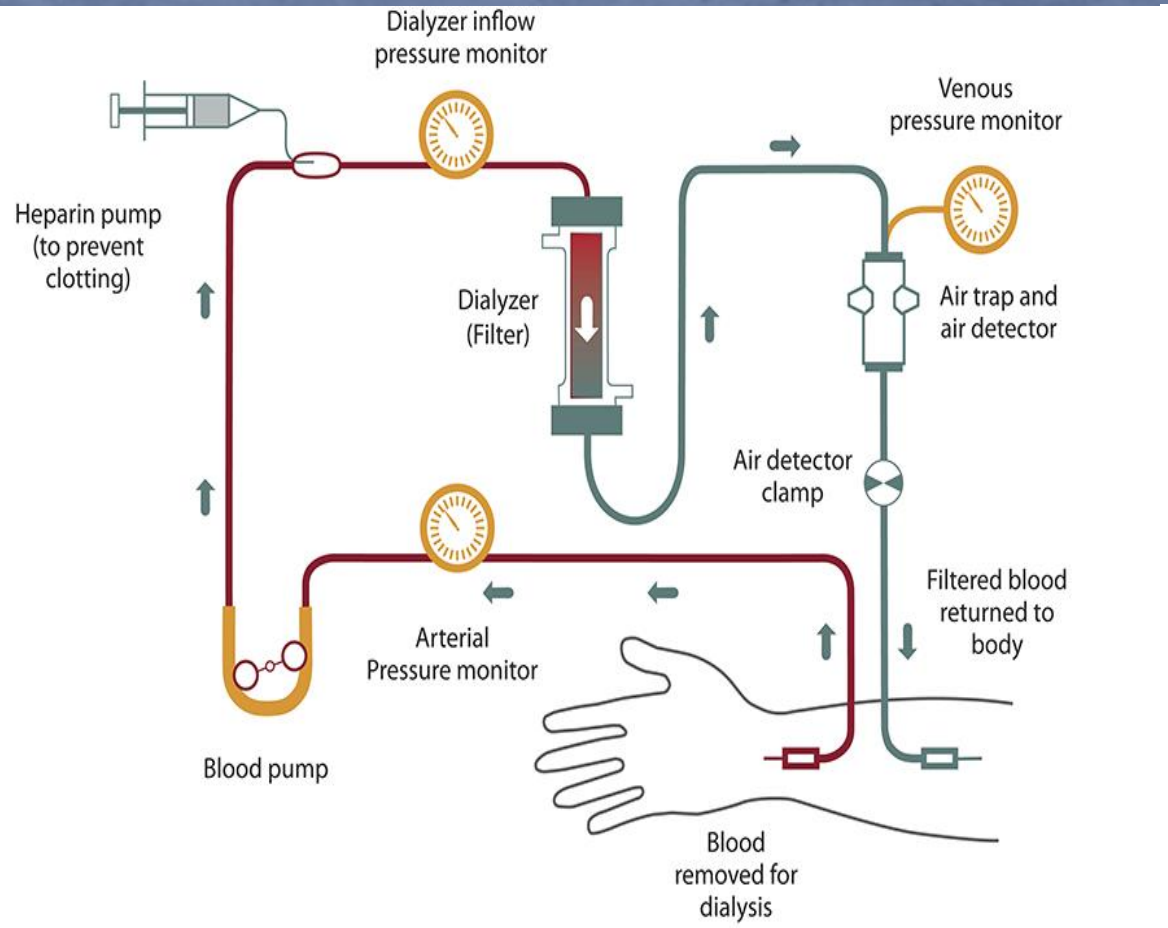
❖Dialysis support may be necessary for days or for up to 12 wk.

RRT modalities

- **RRT choice depends on** :

  - ❖Clinical status and age of the patient
  - ❖The expertise of the clinician
  - ❖The availability of appropriate resources

Hemodialysis

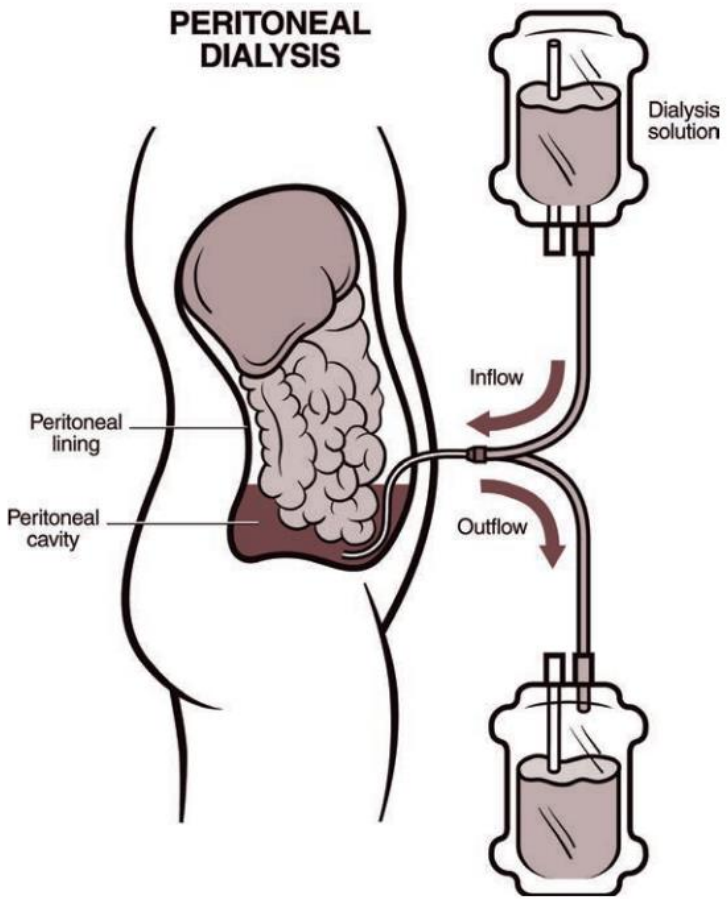


Pros:

Ability to rapidly correct imbalances in fluid, electrolyte, and acid-base status.

- **Cons:**
  - Requires central vascular access
  - Specialized equipment and technical personnel
  - Anticoagulation
  - The ability to tolerate a large extracorporeal volume.

Peritoneal dialysis



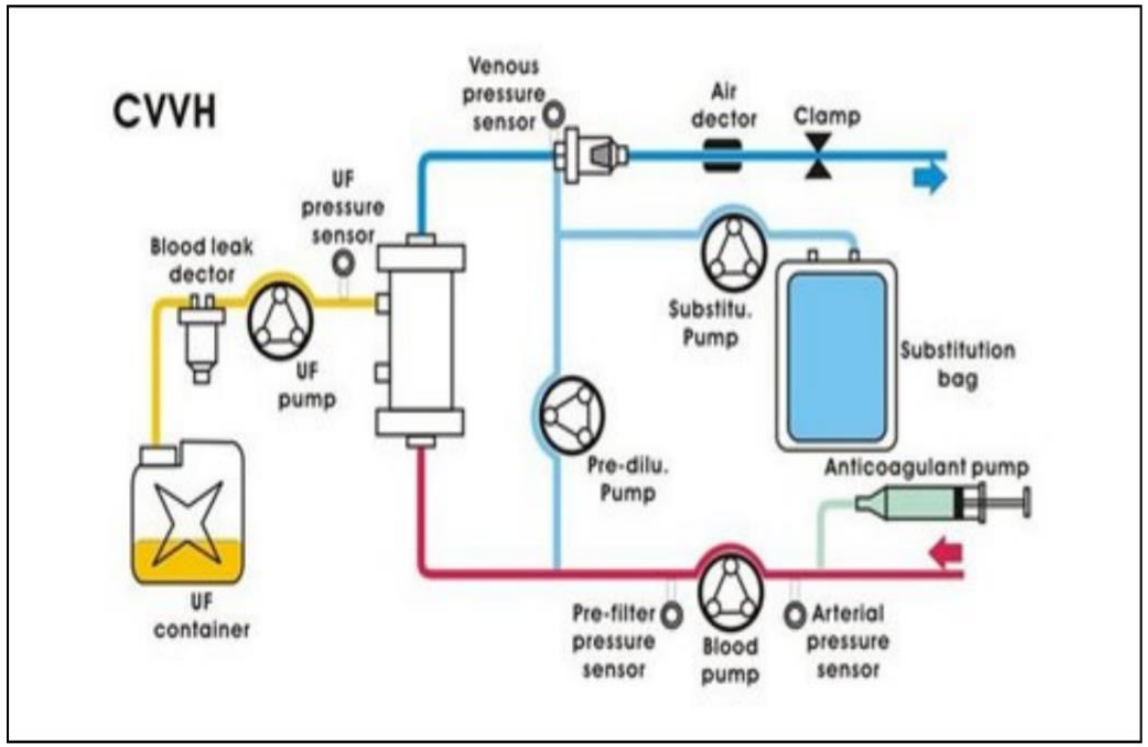
Pros:

- ✓ Ease of performance
- ✓ No requirement for specialized equipment , personnel, or systemic anticoagulation.
- ✓ Therapy of choice in neonates and small infants.

Cons:

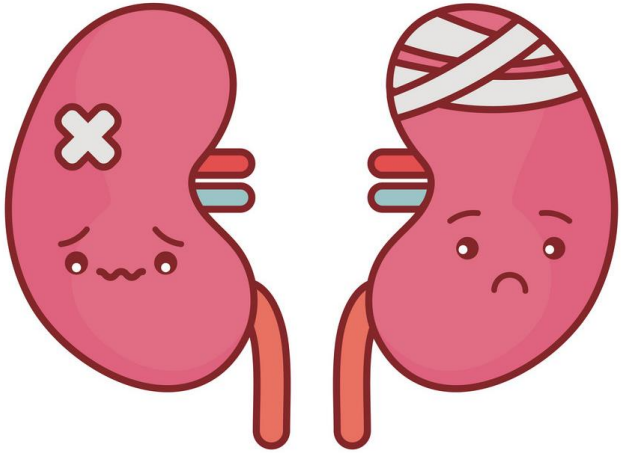
- Slower than HD in correction of fluid overload

CRRT



- **Pros:**
  - Useful in unstable hemodynamic status, concomitant sepsis, or multiorgan failure
  - Fluid, electrolytes, and small- and medium-size solutes are continuously removed from the blood (24 hr/day)
- **Cons:**
  - Requires central vascular access
  - Specialized equipment and technical personnel
  - Anticoagulation

The Pediatric nephrologists most often becomes involved in patients with AKI well after the injury has occurred.



More close monitoring of at -risk patients increases the likelihood of earlier diagnosis and preventing or at least reducing the severity of AKI.