ACUTE KIDNEY INJURY (AKI)

Acute kidney injury (AKI) previously called as acute renal failure is a clinical syndrome in which a sudden deterioration in renal function results in the inability of the kidneys to maintain fluid and electrolyte homeostasis.

PEDIATRIC MODIFIED RIFLE CLASSIFICATION FOR AKI

CRITERIA	ESTIMATED CREATININE CLEARANCE (eCCl)
RISK	eCCl decreases by ≥ 25%
INJURY	eCCl decreases by ≥ 50%
FAILURE	eCCl decreases by ≥ 75%
LOSS	Persistent failure > 4 weeks
END-STAGE	Persistent failure > 3 months

URINARY INDICES FOR PRERENAL VS INTRINSIC RENAL ETIOLOGY

	Pre-renal cause	Intrinsic renal cause
Urine specific gravity	> 1.020	< 1.010
Urine osmolality (mosm/kg)	> 500	< 350
Urinary sodium (mEq/L)	< 20	> 40
Fractional excretion of sodium (UNaxSCr)/(SNaxUCR)x100	< 1 %	> 2 %
BUN / creatinine	> 20	< 20
Urine	bland	Leucocytes,RBC,cast,proteinuria

Causes of AKI maybe;

A) Pre-renal

B) Renal
C) Post-renal

A) Pre-renal

Elicit a history of ;

- diarrhea,
- vomiting,
- fluid loss,
- burns

Look for;

- signs of dehydration,
- tachycardia,
- hypotension,
- dry mucous membranes,
- decreased urine output

- Rmember that the kidney function is intact in patients with prerenal azotemia.

B) Renal (intrinsic kidney disease)

Common causes are;

- ATN (Acute Tubular Necrosis)
  - Usually occurs after an ischemic event or exposure to nephrotoxic agents.
- AIN (Acute Interstitial Nephritis)
  - Classic presentation is fever, rash, eosinophilia and Creatinine bump 7-10 days after drug exposure.
- CIN (Contrast Induced Nephropathy)
  - Increased Creatinine of 0.5mg/dl or >25% 48hrs after contrast administration.
- Others – Glomerular Disease, Pigmented Nephropathy, Thrombotic microangiopathy

C) Post-renal

Caused by obstruction anywhere in the urinary tract;

- Bladder outlet obstruction

- Ureteral obstruction and hydronephrosis
- Patients often have a history of poor urinary stream, dribbling of urine, repeated urinary tract infections, retention, kidney stones, irradiation, congenital abnormalities, kidney procedures or surgeries.

MANAGEMENT

1. **General measures:**

Identify patients at risk of AKI. They include patients with the following:

Prematurity, asphyxia, trauma, burns, post-surgical states, other organ failures (e.g., heart, liver), pre-existing renal disease, malignancy (leukemia, B-cell lymphoma).

Monitor patients-at-risk actively with regards to renal function and urine output.

Try to ensure effective non-dialytic measures, which include:

- Restoring adequate renal blood flow.
- Avoiding nephrotoxic agents if possible, or at least maximizing renal perfusion before exposure to nephrotoxic agents
- Catheterizing the patient
- Maintaining strict intake output record

2. Fluid balance:

1. *In Hypovolaemia(PRE RENAL ETIOLOGY)*

- Fluid resuscitation regardless of oliguric / anuric state
- Give crystalloids e.g. isotonic 0.9% saline / Ringer's lactate 20 ml/kg fast (in < 20 minutes) after obtaining vascular access. Can repeat 3 times
- Transfuse blood if hemorrhage is the cause of shock.
- Hydrate to normal volume status.
- If urine output increases, continue fluid replacement.
- If there is no urine output after 4 hours (confirm with urinary catheterization), monitor central venous pressure to assess fluid status restrict fluids.

2. *In Hypervolaemia / Fluid overload*

Features of volume overload include;

- hypertension,
- raised JVP,
- displaced apexbeat,
- basal crepitations,
- hepatomegaly
- increasing ventilatory requirements

- If necessary to give fluids, restrict to insensible loss (400 ml/m^2/day or 30ml/kg in neonates depending on ambient conditions) plus losses
- IV Frusemide 2 mg/kg/dose (over 10-15 minutes),maximum of 5 mg/kg/dose *or* IV Frusemide infusion 0.5 mg/kg/hour.
- Dialysis if no response or if volume overload is life-threatening

3. Electrolyte abnormalities

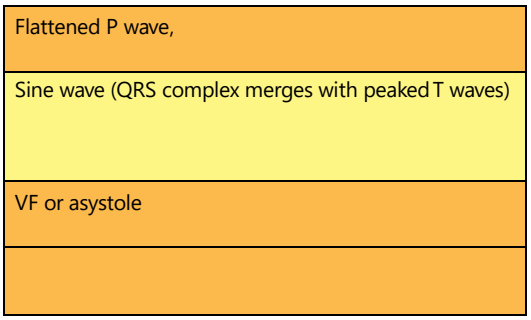
Hyperkalaemia

Definition: Serum K^+ > 6.0 mmol/l (neonates) and > 5.5 mmol/l (children).

Cardiac toxicity generally develops when plasma potassium > 7 mmol/l

Regardless of degree of hyperkalemia, treatment should be initiated in patients with ECG abnormalities from hyperkalemia.

ECG changes in Hyperkalemia
Tall, tented T waves
Prolonged PR interval
Widened QRS complex



Management of hyperkalemia :

- Calcium gluconate 10% solution = 1.0 mL/kg IV over 5–10 min
- Sodium bicarbonate = 1–2 mEq/kg IV over 5–10 min
- Regular insulin = 0.1 U/kg with glucose 50% solution
- Nebulise with salbutamol
- Kayaxelate (if available) 1gm/kg PO or PR in sorbitol
- Restrict potassium in fluids and by mouth
- Dialysis if potassium still high despite these measures

Insulin

Best to give 50 Gm of Glucose +1 iu of Insulin at the rate of 1m/kg/hr
Practically; 10Gm of Glucose + 0.2 iu of Insulin rate 5ml/kg/hr
5Gm of Glucose + 0.1 iu of insulin rate 10ml/kg/hr
(Prep : Add 10 iu of insulin to 10 ml of saline (0.1ml of this solution will contain 0.1 iu of insulin)

Metabolic acidosis

- Treat only if pH < 7.2 or symptomatic , Hco3 LEVEL<12 (must correct dehydration BEFORE giving Hco3)
- Bicarbonate deficit = 0.3 x body weight (kg) x base excess (BE)
- Intravenous bicarbonate followed by oral bicarbonate when pH >7.2 and HCO3 >12

Hyponatremia

- Usually dilutional from fluid overload

- If asymptomatic, fluid restriction
- Dialyse if symptomatic or the above measures fail

Hypocalcemia

- Treat if symptomatic (usually serum Ca^{2+} < 1.8 mmol/ or 7.2 mg/dl).
- If Sodium bicarbonate is required for hyperkalaemia, give IV 10% Calcium gluconate
- 0.5 ml/kg, given over 10 – 20 minutes, preferably with cardiac monitoring

Hyperphosphatemia

- May need treatment if level high
- • Phosphate binders e.g. calcium carbonate orally with main meals.

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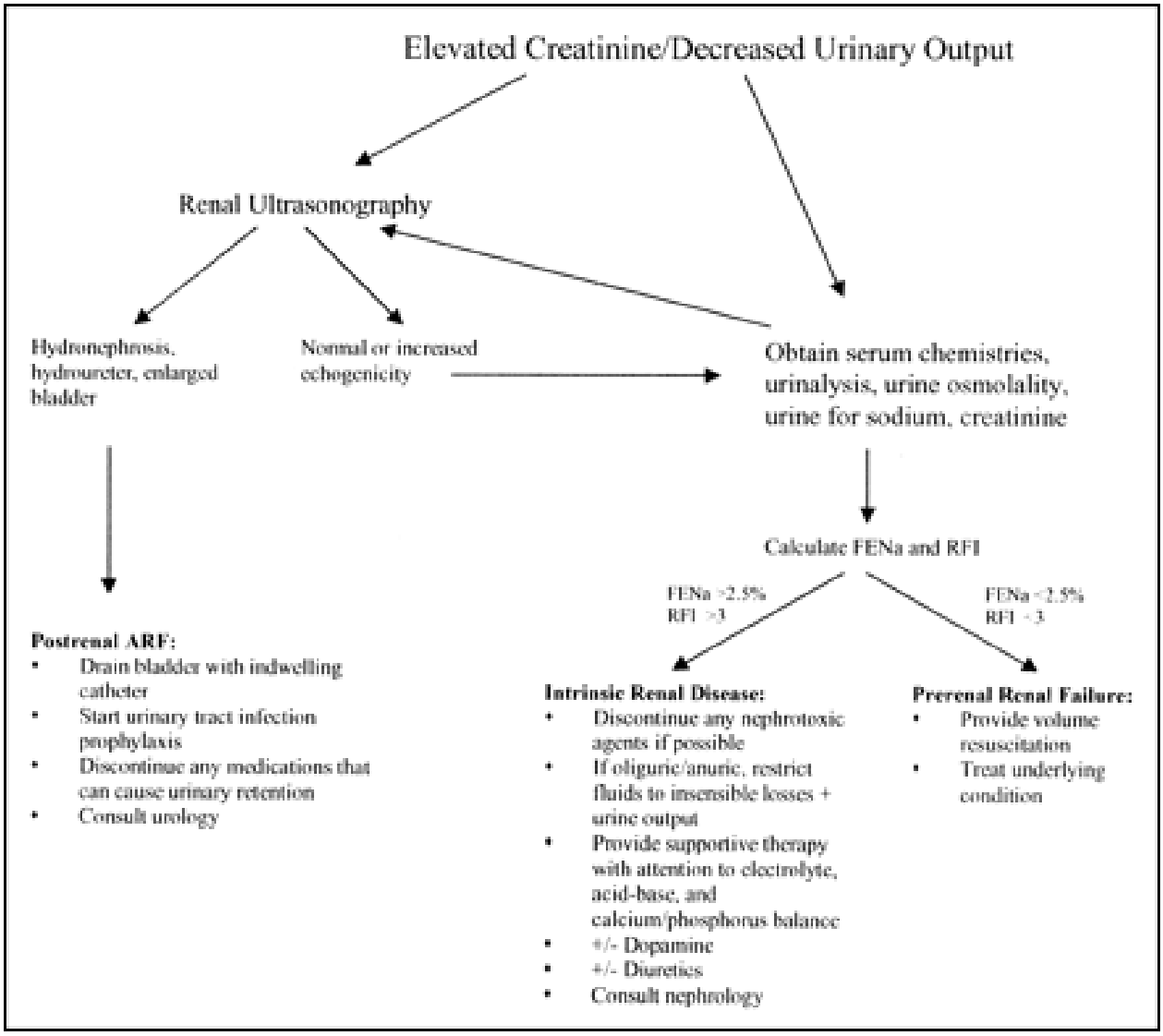
4. Hypertension : Follow protocol of hypertension

5. Renal replacement therapy

Indications for dialysis in AKI include the following:

- Volume overload with evidence of hypertension and/or pulmonary edema refractory to diuretic therapy.
- Persistent hyperkalemia
- Severe metabolic acidosis unresponsive to medical management
- Neurologic symptoms (altered mental status, seizures)
- Blood urea nitrogen greater than 100–150 mg/dL (or lower if rapidly rising)
- Oliguria following recent cardiac surgery.
- Poisoning (hemodialysis)

Algorithm for management of acute renal failure



NUTRITION

Optimal intake in AKI is influenced by nature of disease causing it, extent of catabolism, modality and frequency of renal replacement therapy.

Special consideration is given to:

- Avoiding excessive protein intake
- Minimizing phosphorus and potassium intake
- Avoiding excessive fluid intake (if applicable)
- If the gastro-intestinal tract is intact and functional, start enteral feeds as soon as possible.
- Total parenteral nutrition via central line. If enteral feeding is not possible; use concentrated dextrose (25%), lipids (10-20%), protein (1.0-2.0g/kg/day)
- If oliguric and caloric intake is insufficient because of fluid restriction, start dialysis earlier.

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