Acute Kidney Injury:

Definition

It is a clinical syndrome in which sudden deterioration in the renal function result in inability of the kidneys to maintain fluid& electrolytes homeostasis.

AKI is characterized by:

Increase in serum creatinine by ≥ 0.3 mg/dL from baseline within 48 hr.

Or Increase in serum creatinine to ≥ 1.5 times baseline within the prior 7 days

Or Urine volume ≤ 0.5 mL/kg/hr for 6 hr.

Normal S.creatinine 62-124 umol/L
Normal B.urea 3.3-7.5 mmol/L

Pathogenesis:

According to the cause, AKI classified into 3 categories:

▲ ***prerenal AKI (55-60% of AKI)***

- **dehydration**
- **hemorrhage**
- **sepsis**
- **hypoalbuminemia**
- **cardiac failure**

▲ ***Intrinsic renal AKI (35-40%)***

- **Glomerulonephritis:**

  -APSGN -SLE
  -HSP
  -membranoprolifrative GN

- **HUS**
- **ATN**
- **RVT**
- **Acute interstitial nephritis**
- **Tumor infiltration**
- **Tumor lysis syndrome**

▲*post renal AKI: just 5% AKI*

- **posterior urethral valves**
- **uretro-pelvic junction obstruction**
- **uretro-vesicular junction obstruction**
- **uretrocele**
- **urolithiasis**
- **tumor**

<u>Clinical manifestation:</u>

Carefully taken history is critical in defining the causes of ARF.
diarrhoea & Vomiting→ dehydration →prerenal AKI

recent pharyngitis, periorbital edema, HT, gross hematuria→ intrinsic AKI related to APSGN.

Critically ill child with hypotension & exposure to nephrotoxic drug→ acute tubular necrosis.

Male neonate with history of hydronephrosis on prenatal U/S & apalpable bladder→ posterior urethral valve.

<u>physical Examination:</u>

Tachycardia, dry mucous membranes &poor peripheral perfusion suggest inadequate circulation & prerenal AKI.

Periphral edema, rales & cardiac gallop→ volume over load & intrinsic AKI.

Rash &arithritis may suggest SLE or HSP.

Palpable flank mass→ RVT, tumor, or UT obstruction

Lab. Finding:

- **CBP may reveal:**

Anemia usually dilutional or hemolytic as in SLE, HUS, RVT.

Leukopnea→ as in SLE
Thrombocytopnea: SLE, RVT, HUS

- **Hyponatremia (dilutional).**

- **metobolic acidosis**

- **↑blood urea & serum creatinine**

●↑S.uric acid, potassium, phosphate due to diminished renal function.

- **hypocalcemia.**

- **C3 level: ↑ in SLE, post streptococcal GN.**

- **Antibodies to streptococcal Ag, nuclear Ag.**

- **GUE:**

hematuria, proteinuria &RBC cast→ usually seen intrinsic AKI.

Treatment:

▲Medical Treatment
Fluid Management:

1. **Dehydration or Shock:**

If the patient has dehydration or presented in shock state, intravascular volume expansion by I.V isotonic saline 20cc/kg over 30 min is recommended.

Severe hypovolemia may require additional fluid boluses.

Diuretic therapy should be considered only after the adequacy of the circulating blood volume has been established.
Furosemide (2-4 mg/kg) may be administered as a single intravenous dose. If urine output is not improved, then a continuous diuretic infusion may be considered.
To increase renal cortical blood flow, many clinicians administer dopamine(2-3 µg/kg/min) in conjunction with diuretic therapy.

If there is no response to a diuretic challenge, diuretics should be discontinued and fluid restriction is essential.

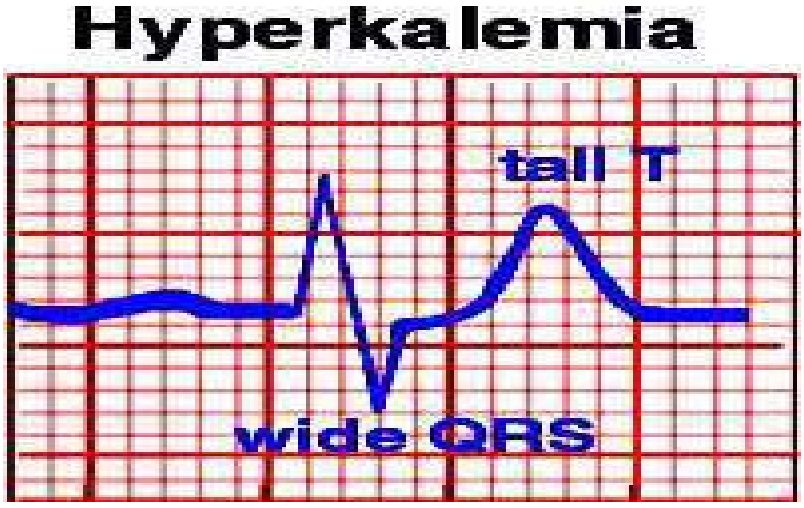
2.<u>Established AKI with fluid over load:</u>

The fluid should restrict to insensible water loss (400 cc/m2/day) + urine output. Type of fluid is GW 10-30%

<u>Complications of AKI:</u>

<u>Hyperkalemia:</u>

S.K+> 6mEq/L presented with muscles weakness lethargy, paresthesia, hyporeflexia, and cardiac arrythemia.



ECG finding in hyperkalemia.

Treatment:

- **Eliminate exogenous source of k+.**
- **Continuous ECG monitoring.**
- **Sodium bicarbonate 1-2 mmol/kg iv over 5-10 min**
- **Na-polystyrene resin 1g/kg given orally or rectally, May repeated every 2hr.**
- **Calcium gluconate 100 mg/kg/dose (maximum 3000 mg/dose) over 5-10 min.**
- **Regular insulin 0.1u/kg with 50%glucose 1g/kg i.v over 1 hr.**
- **Persistent hyperkalemia should be managed by dialysis.**

<u>Hypocalcemia:</u>

Treated by lowering the s. phosphorus level by:
♦ low phosphate diet
♦ Oral phosphate binders (Ca-carbonate, Ca-acetate).

<u>Hyponatremia:</u>

***fluid restriction**
***administration of hypertonic 3%saline for symptomatic hyponatremia (seizure, lethargy) or if serum sodium level<120Mq/L.**

<u>Hypertension:</u>
Treated by:
♦salt-water restriction ♦ use of diuretics
♦Antihypertensive agent: like

Calcium channel blockers (amlodipine, 0.1-0.6 mg/kg/24 hr divided bid)
Or β-blockers (propranolol, 0.5-8 mg/kg/24 hr divided bid, labetalol, 4-40 mg/kg/24 hr divided bid) may be helpful in maintaining control of blood pressure.

<u>Anaemia:</u>

Blood transfusion if Hb<7g/dl, usually use packed RBC, given slowly over 4-6hr. 10 cc/kg to avoid risk of hypervolemia.

Dialysis:

Indications:

1. **Volume over load with HT &/or pulmonary edema refractory to diuretic therapy.**
2. **Persistent hyperkalemia.**
3. **Severe metabolic acidosis unresponsive to medical treatment.**
4. **Neurological symptom (seizures, altered mental status).**
5. **Anuria/oliguria**
6. **Calcium/ phosphorus imbalance with hypocalcemic tetany.**

Hemolytic Uremic Syndrome (HUS)

Definition:

This syndrome is the most common cause of acute kidney

injury in young children, &characterized by:

♦ Microangiopathic hemolytic anemia.

♦ Thrombocytopenia.

♦ Renal cortical injury.

Etiology:

This disease frequently follows an episode of gastroenteritis caused by enterohemorrhagic strains of *E.coli (0157:H7)*.

These organisms elaborate shiga-toxin which absorbed from the intestine & initiate endothelial injury.

Other infections less often associated with HUS :

***Bacterial* : shigella, salmonella, campylobacter.**

***Viral*: influenza, HIV, Parvovirus B19.**

***Drugs*: contraceptives, cyclosporin, mithramycin.**

***Systemic diseases*: SLE, malignant HT, preclampsia,**

radiation nephritis.

Clinical Manifestation:

This syndrome is most common in children <4years old.
The onset usually preceded by gastroenteritis often bloody, or less commonly by upper respiratory tract infection. This followed in 5-10days by sudden onset of:

•Pallor
•Irritability
•Weakness
•Lethargy
•Oliguria
Physical examination reveals
•Edema.
•Dehydration.
•Petechiae.
•Hepatosplenomegaly and irritability.

<u>Diagnosis:</u>

CBP:
reveal microangiopathic hemolytic anemia with Hb level in a range of 5-9g/dl.

Blood film reveals helmet cells, burr cells, schistocytes & fragmented RBC.

Elevated reticulocytes count, thrombocytopenia.

Urinalysis: microscopical hematuria, proteinuria.

PT , PTT normal.

Coombs test –ve.

Renal function tests usually elevated indicating renal involvement.

<u>Differential Diagnosis:</u>

1. Lupus nephritis

2. Malignant HT

3. Bilateral renal vein thrombosis.

Complications :

♦Anemia ♦ Acidosis ♦ Uremia

♦Hyrekalemia ♦ Hypertension ♦ Heart failure

♦D.M ♦ Colitis ♦ Intestinal perforation

♦CNS manifestation: irritability, seizure

Treatment :

***Supportive care includes careful management of fluid and electrolytes, control of hypertension, and early institution of dialysis if the patient becomes anuric or significantly oliguric.**

***washed Red cell transfusions are usually required because hemolysis can be brisk and recurrent until the active phase of the disease has resolved.**

***Anticoagulation, antiplatelet, and fibrinolytic therapy is specifically contraindicated because they increase the risk of serious hemorrhage.**

***Plasma infusion or plasmapheresis has been proposed for patients suffering severe manifestations of HUS, primarily serious CNS involvement.**