Management and Investigation of Acute Renal Failure

Dr. Nariman Fahmi 5th year lecture 2011-2012
Assistant professor / Baghdad Medical College

Introduction
Each kidney contains approximately 1 million nephrons (glomeruli and associated tubules). In humans, formation of nephrons is complete at birth, but functional maturation with tubular growth and elongation continues during the first decade of life. Because new nephrons cannot be formed after birth, progressive loss of nephrons may lead to renal insufficiency.

The Urinary System is a group of organs in the body concerned with filtering out excess fluid and other substances from the bloodstream. The Urinary organs include the kidneys, ureters, bladder, and urethra. The Urinary system works with the other systems of the body to help maintain homeostasis. The kidneys are the main organs of homeostasis because they maintain the acid base balance and the electrolytes balance of the blood.
Anatomy of the Glomerulus

The kidneys lie in the retroperitoneal space slightly above the level of the umbilicus. They range in length and weight, respectively, from approximately 6 cm and 24 g in a full-term newborn to 12 cm or more and 150 g in an adult. The kidney has an outer layer, the cortex, that contains the glomeruli, proximal and distal convoluted tubules, and collecting ducts and an inner layer, the medulla, that contains the straight portions of the tubules, the loops of Henle, the vasa recta, and the terminal collecting ducts.

Definition of Acute Renal Failure

Sudden loss of the ability of the kidneys to excrete wastes, concentrate urine and conserve electrolytes

ARF occurs in 2–3% of children admitted to pediatric tertiary care centers and in as many as 8% of infants in the neonatal intensive care unit.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

A carefully taken history is critical in defining the cause of ARF
The physical examination must be thorough, with careful attention to volume status.

On examination : note state of dehydration

Signs and symptoms
- Oligurea or anurea
- Fluid retention Ankle ,legs swelling
- Changes in mental status
- Drowsiness , lethargy, confusion ,coma
- Seizures
- Vomiting
- Hypertension

Lab. Values
- BL.urea , S. creatinin may increase suddenly
- S.electrolyte : S. K may increase
- Arterialblood gas: metabolic acidosis
- Ultrasonography can be helpful
Aetiology

Pre-renal
Decrease in the effective circulatory volume
Decline in renal cortical blood flow
Reduced GFR

As in (shock, hypovolemia) from Hemorrhage, diarrhea, burn, Hypotension as in sepsis, heart failure

Renal
• GN, developmental anomalies, HUS

Post renal
• Obstructive uropathy

Treatment
The goal is to
• 1-identify any reversible causes
• 2- preventing excess accumulation of fluids and wastes

Hospitalizations is required for treatment and monitoring

Management of Acute Renal Failure

- Fluid Management

• Fluid Overload:
  o Frusemide 2 - 10mg/kg bolus
  o Fluid restriction
  o Minimise drug infusion volumes

• Accurate input/output
• Daily weight
• Dialysis
• Beware Polyuria

As a rule fluid therapy should equal insensible fluid losses plus output (urine, vomiting, drain losses, diarrhoea etc)

□ Correction of Hypovolaemia

Acid–Base Disorders

Mild metabolic acidosis is common in ARF because of retention of hydrogen ions, phosphate, and sulfate, but it rarely requires treatment. If acidosis is severe (arterial pH <7.15; serum bicarbonate <8 mEq/L) or contributes to hyperkalemia, treatment is
required. The acidosis should be corrected partially by the intravenous route, generally giving enough bicarbonate to raise the arterial pH to 7.20 (which approximates a serum bicarbonate level of 12 mEq/L). The remainder of the correction may be accomplished by oral administration of sodium bicarbonate after normalization of the serum calcium and phosphorus levels. Correction of metabolic acidosis with intravenous bicarbonate may precipitate tetany in patients with renal failure as rapid correction of acidosis reduces the ionized calcium concentration.

hyperkalemia In ARF, rapid development of hyperkalemia (serum potassium level >6 mEq/L) may lead to cardiac arrhythmia, cardiac arrest, and death. The earliest electrocardiographic change seen in patients with developing hyperkalemia is the appearance of peaked T waves. This may be followed by widening of the QRS intervals, ST segment depression, ventricular arrhythmias, and cardiac arrest.

Procedures to deplete body potassium stores should be initiated when the serum potassium value rises above 6.0 mEq/L. Exogenous sources of potassium (dietary, intravenous fluids, total parenteral nutrition) should be eliminated.

Sodium polystyrene sulfonate resin (Kayexalate), 1 g/kg, should be given orally or by retention enema.

More severe elevations in serum potassium (>7 mEq/L), especially if accompanied by electrocardiographic changes, require emergency measures in addition to Kayexalate. The following agents should be administered:

- Calcium gluconate 10% solution, 1.0 mL/kg IV, over 3–5 min
- Sodium bicarbonate, 1–2 mEq/kg IV, over 5–10 min
- Regular insulin, 0.1 U/kg, with glucose 50% solution, 1 mL/kg, over 1 hr

Administration of sodium bicarbonate and insulin and glucose lowers the serum potassium level by shifting potassium from the extra cellular to the intracellular compartment. A similar effect has been reported with the acute administration of β-adrenergic agonists in adults, but there are no controlled data in pediatric patients. Because the duration of action of these emergency measures is just a few hours, persistent hyperkalemia should be managed by dialysis.

Hypocalcemia is primarily treated by lowering the serum phosphorus level. Calcium should not be given intravenously, except in cases of tetany, to avoid
deposition of calcium salts into tissues. Patients should be instructed to follow a low phosphorus diet, and phosphate binders should be orally administered to bind any ingested phosphate and increase gastrointestinal phosphate excretion. Common agents include sevelamer (Renagel), calcium carbonate, and calcium acetate.

**Hyponatremia** is most commonly a dilutional disturbance that must be corrected by fluid restriction rather than sodium chloride administration. Administration of hypertonic (3%) saline should be limited to those patients with symptomatic hyponatremia (seizures, lethargy) or those with a serum sodium level <120 mEq/L. Acute correction of the serum sodium to 125 mEq/L (mmol/L) should be accomplished using the following formula:

\[
\text{NaCl (meq/L) required} = 0.6(\text{BW Kg}) \times \{125 - \text{serum sodium (meq/L)}\}
\]

**Nutrition in acute renal failure**

Nutrition is of critical importance in children who develop ARF. In most cases, sodium, potassium, and phosphorus should be restricted. Protein intake should be restricted moderately while maximizing caloric intake to minimize the accumulation of nitrogenous wastes. In critically ill patients with ARF, parenteral hyperalimentation with essential amino acids should be considered.

**Hypertension in Acute Renal Failure**

May relate solely to salt and water overload and therefore in the presence of oligo/anuria and in the absence of hypovolaemia an initial trial of diuretic therapy is justified.

Drug therapy for the emergency treatment of hypertension is outlined below.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Onset</th>
<th>Action</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydralazine</td>
<td>0.2 to 15 mg/dose IV bolus 4-6ug/kg/min IVI</td>
<td>5-10 min</td>
<td>Direct vasodilator</td>
<td>Headache, vomiting, tachycardia</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>0.25-0.5 mg/kg</td>
<td>sublingual</td>
<td>5-10 min Ca channel blocker</td>
<td>Headache, nausea, Syncope</td>
</tr>
<tr>
<td>Frusemide</td>
<td>1-3mg/kg over 15min 0.1-1mg/kg/hr IVI</td>
<td>Diuretic</td>
<td>Volume depletion Electrolyte abn</td>
<td>hypokalemia</td>
</tr>
<tr>
<td>Diazoxide</td>
<td>5 mg/kg (max 300) IV bolus</td>
<td>3-5 min</td>
<td>Direct vasodilator</td>
<td>Hyperglycaemia Nausea &amp; vomiting</td>
</tr>
</tbody>
</table>
ARF patients are predisposed to gastrointestinal bleeding because of uremic platelet dysfunction, increased stress, and heparin exposure if on hemodialysis or continuous renal replacement therapy. Oral or intravenous H₂ blockers such as ranitidine are commonly administered to prevent this complication.

**Neurological symptoms** in ARF may include headache, seizures, lethargy, and confusion. Potential etiologic factors include hyponatremia, hypocalcemia, hypertension, cerebral hemorrhage, cerebral vasculitis, and the uremic state. Diazepam is the most effective agent in controlling seizures, and therapy should be directed toward the precipitating cause.

The anemia of ARF is generally mild (hemoglobin 9–10 g/dL) and primarily results from volume expansion (hemodilution). Children with HUS, SLE, active bleeding, or prolonged ARF may require transfusion of packed red blood cells if their hemoglobin level falls below 7 g/dL. In hypervolemic patients, blood transfusion carries the risk of further volume expansion, which may precipitate hypertension, heart failure, and pulmonary edema. Slow (4–6 hr) transfusion with packed red blood cells (10 mL/kg) diminishes the risk of hypervolemia. The use of fresh, washed red blood cells minimizes the risk of hyperkalemia. In the presence of severe hypervolemia or hyperkalemia, blood transfusions are most safely administered during dialysis/ultrafiltration.

**DIALYSIS.**

Indications for dialysis in ARF include the following:

- **Used to remove excess waste and fluids**

**Indications**

- 1- uncontrollable fluids overload or hypertension
- 2- uncontrollable acidosis
- 3- uncontrollable electrolyte disturbances
- 4-pericarditis
- 5- change in mental status
- 6-anuria
- 7-uncontrollable accumulations of nitrogen waste products