University of Anbar
College of pharmacy
Therapeutic I, 5th stage
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Acute Renal Failure

Renal Failure : is the condition which results when the kidneys cannot remove the body's metabolic wastes or perform their regulatory functions. This result in disruption in endocrine and metabolic functions, fluid, electrolyte, and acid-base disturbances.

The main Kidney Functions are

1- Detoxify blood

2- Increase calcium absorption

3- Stimulate RBC production

4- Regulate blood pressure and electrolyte balance

Urine Volume

Oliguric: <400 CC/ 24 Hrs , **Non-Oliguric**: >500 CC/24 Hrs ,

Anuric <50 CC/24 Hrs

Acute Renal failure	**Chronic Renal failure**
sudden onset	Progressive
rapid reduction in urine output	Variable
Usually reversible	Not reversible
Tubular cell death and regeneration	Nephron loss
sudden onset	

Acute Renal Failure (ARF)

- Is a **reversible** clinical syndrome where there is a :

  A- Sudden and almost complete loss of kidney function (decreased GFR) over a period of hours to days

  B- Failure to excrete nitrogenous waste products

  C- Failure to maintain fluid and electrolyte homeostasis.

Causes of Acute Renal Failure

A. Prerenal

1. Volume depletion resulting from:

a. hemorrhage

b. Renal losses (diuretics)

c. GI losses (vomiting, diarrhea, NG suctioning)

2. Impaired cardiac efficiency resulting from:

a. MI

b. Heart failure

c. Dysrhythmias

d. Cardiogenic shock

3. Vasodilation resulting from:

a. sepsis

b. anaphylaxis

c. antihypertensive medications or other medications that cause vasodilation

B. Intrarenal

1. Prolonged renal ischemia resulting from:

a. pigment nephropathy (associated with the breakdown of blood cells containing pigments that in turn occlude kidney structures)

b. Myoglobinuria (trauma, crush injury, burns)

c. Hemoglobinuria (transfusion reaction, hemolytic anemia)

2. Nephrotoxic agents such as:

a. Aminoglycosides antibiotics (gentamicin, tobramycin)

b. Radiopaque contrast media

c. Heavy metals (lead, mercury)

d. Solvents and chemicals (carbon tetrachloride, arsenic)

e. NSAIDs, ACE inhibitors

3. Infectious processes such as:

a. Acute pyelonephritis

b. Acute GN

C. Postrenal

1. Urinary tract obstruction, including:

a. calculi (stones)

b. tumors

c. BPH

d. Strictures

e. Blood clots

—

Phases of ARF

1. **Initiation** – begins with the initial insult and ends when oliguria develops

2. The **oliguria** period is accompanied by an increase in the serum concentration of substances usually excreted by the kidneys (**uric acid**, **urea**, **creatinine**).

In this phase **uremic symptoms** first appear and life-threatening conditions such as hyperkalemia develop.

3. The **diuresis period** is marked by a gradual increase in urine output, which signals that glomerular filtration has started to recover.

4. The **recovery period** signals the improvement of renal function and may take **3-12 months**. Lab values return to normal level. Although a **permanent 1%-3% reduction** in the GFR is common.

Symptoms of ARF

1- Decrease urine output (70%)

2- Edema, esp. lower extremity

3- Mental changes

4- Heart failure

5- Nausea, vomiting

6- Pruritus

7- Anemia

8- Tachypenic

9- Cool, pale, moist skin

Diagnosis

1- medical and medication histories, physical examination, assessment of laboratory values, and, if needed, imaging studies are important in the diagnosis of ARF.

2-Urine

A- Urine electrolytes and Urine Cr to calculate FeNa

B- Urine eosinophils

C- Urine sediment: casts, cells, protein

D- Urine osmolality

— FeNa = $\frac{\text{(urine Na x plasma Cr)}}{\text{(plasma Na x urine Cr)}}$ X 100

Scr cannot be used alone to diagnose ARF because it is **insensitive to rapid changes in glomerular filtration rate (GFR) and therefore may not reflect current renal function.

The use of BUN in ARF is very limited because urea's production and renal clearance are heavily influenced by extrarenal factors such as **critical illness**, **volume status**, **protein intake**, and **medications**.

Preventing ARF

1. Provide adequate hydration to patients at risk of dehydration:

a. surgical patients before, during and after surgery.

b. Patients undergoing intensive diagnostic studies requiring fluid restrictions and contrast agents

c. Patients with neoplastic disorders of metabolism and those receiving chemotherapy

2. Prevent and treat shock promptly with blood and fluid replacement.

3. Monitor CV and arterial pressures and hourly urine output of critically ill patients to detect the onset of renal failure as early as possible.

4. Treat hypotension promptly.

5. Continually assess renal function when appropriate.

6. Take precautions to ensure that the appropriate blood is administered to the correct patient in order to avoid severe transfusion reactions, which can precipitate renal failure.

7. Prevent and treat infections promptly. Infections can produce progressive renal damage.

8. Pay special attention to wounds, burns and other precursors of sepsis

9. To prevent infections from ascending in the urinary tract, give meticulous care to patients with indwelling catheters. Remove catheter ASAP.

10. To prevent toxic drug effects, closely monitor dosage, duration of use, and blood levels of all medications metabolized or excreted by the kidneys.

11- **Ascorbic acid** (3 g orally pre- and 2 g orally twice daily for two doses postprocedure) and **N-acetylcysteine** (600–1200 mg orally every 12 hours for 2–3 days [first two doses precontrast]) are antioxidant options for prevention of CIN. Study results with these two agents are inconsistent.

Medical Management:

Goals of Treatment: Short-term goals include

1- minimizing the degree of insult to the kidney.

2- Reducing extrarenal complications, and expediting recovery of renal function.

3- Restoration of renal function to preARF baseline is the ultimate goal.

1. Pharmacologic therapy

a. hyperkalemia is the most life-threatening of changes that occur in RF, the elevated K levels may be reduced by administering cation-exchange resins (**sodium polystyrene sulfonate [Kayexalate**] orally or by retention enema. It works by exchanging sodium ions for potassium ions in the intestinal tract.

b. Sorbitol may be administered in combination with Kayexalate to induce diarrhea type effect (induce water loss in the GIT)

c. If hemodynamically unstable, IV dextrose 50% , insulin and calcium replacement may be administered to shift potassium back into the cells.

d.Loop diuretics (**furosemide, bumetanide, torsemide, and ethacrynic acid)** are often administered to control fluid volume, but they have not been shown to hasten the recovery from ARF.

Notes:

***Ethacrynic acid** is reserved for **sulfa-allergic patients.**

****Continuous infusions** of loop diuretics appear to overcome diuretic resistance and to have fewer adverse effects than intermittent boluses.

***Administration of agents from different pharmacologic classes, such as diuretics that work at the distal convoluted tubule **(thiazides)** or the collecting duct (**amiloride, triamterene** and **spironolactone) , may be synergistic** when combined with loop diuretics.

******Metolazone** is commonly used because, unlike other thiazides, it produces effective diuresis at GFR less than 20 mL/min (0.33 mL/s).

2. Nutritional Therapy

a. Dietary proteins are individualized to provide the maximum benefit. Caloric requirements are met with high-carbohydrate meals, because carbohydrates have a protein-sparing effect.

b. Foods and fluids containing potassium or phosphorous such as **banana**, **citrus fruits** and **juices**, **coffee** **<u>are restricted</u>**