ACUTE RENAL FAILURE

DEFINITION

- It refers to the abrupt decrease in kidney function resulting in the retention of urea and other nitrogenous waste and in the dysregulation of extracellular volume and electrolytes.
- The term Acute Renal Failure (ARF) is largely being replaced by the term Acute Kidney Injury (AKI) highlighting that injury to the kidney does not lead to failure.

- In this condition the kidneys abruptly stop working partially or entirely but may eventually recover nearly normal function.

- The causes of acute renal failure can be divided into three main categories
  - ***Prerenal* acute renal failure**
  - ***Intrarenal* acute renal failure**
  - ***Postrenal* acute renal failure**

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Acute Renal Injury and Chronic Kidney Disease 1113

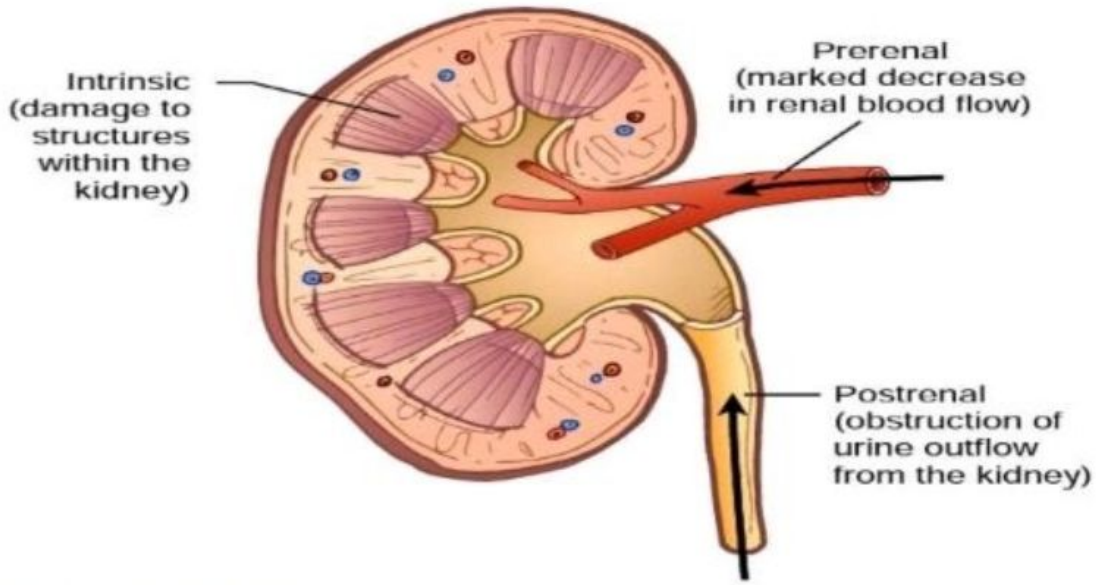

FIGURE 42.1 • Types of acute renal failure.

PRERENAL

Causes

Volume depletion states: Haemorrhage, Gastrointestinal tract loss, Excessive loss of fluid due to burns, Diuretics

Decreased vascular filling: Anaphylactic shock, septic shock.

Hypoperfusion: Heart failure, Cardiogenic shock, Non steroidal anti-inflammatory drugs

PRERENAL

Pathophysiology

The kidneys receive 20-25 % of the cardiac output. The large supply is required for the kidney to carry out its functions; removal of metabolic wastes and regulation of body fluids and electrolytes. Fortunately, the normal kidneys can tolerate relatively large reductions in blood flow before renal damage occurs.

PRERENAL

- **Pathophysiology**

As renal blood flow is reduced, the glomerular filtration rate (GFR) decreases. This results to a corresponding decrease of urine output and an elevated blood urea nitrogen (BUN). Ischemic changes will occur. The tubular epithelial cells which have a high metabolic rate are the most vulnerable to injury. Tubular necrosis results as a result of improper treatment of prerenal acute kidney injury.

INTRARENAL

Causes

Infections: glomerulonephritis

Pharmacologic agents: aminoglycosides, platinum analogues, radiocontrast agents

INTRARENAL

- **Pathophysiology**

Entails damage to the parenchyma in the glomeruli, vessels ,tubules or interstitium.

The kidney is vulnerable to nephrotoxic injury because of its rich blood supply and ability to concentrate toxins to high levels in its medullary portion.

POSTRENAL

- **Causes**

Obstruction in the urinary tract at the level of;

Ureter: calculi, strictures

Bladder: tumours, neurogenic bladder (functional)

Urethra: prostatic hyperplasia

POSTRENAL

- **Pathophysiology**

Obstruction can be structural or mechanical (neurogenic bladder).

The functional unit, the nephron, being intact continues performing its filtration function. Increased urine is not able to be excreted further due to the obstruction. This leads to retrograde pressure and flow which ultimately damages the nephron.

SYMPTOMS

- Decreased urine production
- Nausea
- Vomitting
- Anuria
- Body swelling

5.2.2.3 Complications

Potential complications of acute kidney failure include:

- **Fluid accumulation**

Acute kidney failure may lead toaccumulation of fluid in lungs, which can cause shortness of breath.

- **Chest pain**

If the pericardium becomes inflamed, patient may experience chest pain.

- **Muscle weakness**

When body fluids and electrolytes become imbalanced, it may result in muscle weakness.

- **Permanent kidney damage**

Occasionally, acute kidney failure causes permanent loss of kidney function, or end-stage renal disease. People with end-stage renal disease require either permanent dialysis or a kidney transplant to survive.

- **Death**


CHRONIC RENAL
FAILURE

DEFINITION

- Used interchangeably with the term Chronic Kidney Disease (CKD).
- CKD is defined as presence of kidney damage (usually detected as urinary albumin excretion of 30mg/day or more) or decreased kidney function (defined as an estimated GFR <60ml/min/1.73m^2) for three or more months irrespective of the cause.

Table 10. Stages of Chronic Kidney Disease

Stage	Description	GFR (mL/min/1.73 m^2)
1	Kidney damage with normal or ↑ GFR	≥90
2	Kidney damage with mild ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

Chronic kidney disease is defined as either kidney damage or GFR <60 mL/min/1.73 m^2 for ≥3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.

CAUSES

- Hypertension
- Diabetes

The aforementioned are the most common causes

Persistence of prerenal, Intrarenal and postrenal causes

PATHOPHYSIOLOGY

- **1. Accumulation of nitrogenous waste**

It is an early sign of kidney failure. Urea is one of the first nitrogenous wastes to accumulate in blood and the BUN becomes increasingly elevated as CKD progresses.

Progressive azotemia leads to uremia.

PATHOPHYSIOLOGY

- Uremia affects various organs:

CNS-uremic encephalopathy, peripheral neuropathy

Skin-pruritus

GIT-ulcers, emesis

CVS-pericarditis

Reproductive-reduced libido due to disturbance in hypothalamo-pituitary-gonadal function

Constitutional-weakness, fatigue

PATHOPHYSIOLOGY

- Creatinine, a byproduct of muscle metabolism, is freely filtered in the glomerulus and is not reabsorbed. Used as an indirect measure of degree of failure.

PATHOPHYSIOLOGY

- **2. Fluid, Electrolyte and Acid-Base Disturbance**

The function of extracellular fluid regulation by either conserving sodium and water is impaired. Moreover, the concentration of urine is impaired leading to polyuria which is an early sign of CKD. The urine at this point is almost isotonic to plasma.

PATHOPHYSIOLOGY

- Salt wasting presents in advanced renal failure due to impaired tubular reabsorption of sodium.
- Approximately 90% of K+ excretion is renal. Hyperkalemia usually does not develop until GFR is below 5-10 ml/min/1.73m2. This is due to renal adaptation to excrete K+ with diminished GFR and GIT K+ loss increase.

PATHOPHYSIOLOGY

- PH regulation is by H+ elimination and HCO3- regeneration. This is achieved by H+ secretion, Na+ and HCO3- reabsorption and ammonia production which acts as an acid buffer.
- With decline in renal function, the aforementioned mechanisms become impaired.

PATHOPHYSIOLOGY

Metabolic acidosis results if the patient is challenged with excessive acid load or loss of alkali as seen in diarrhea.

PATHOPHYSIOLOGY

- **3. Disorders of Calcium and Phosphorus metabolism**
- Regulation of Phosphate requires daily urinary excretion almost equal to the amount ingested. With progressive renal failure, its excretion is impaired leading to rise in plasma phosphate levels.

PATHOPHYSIOLOGY

- Serum calcium levels, which are inversely proportional to phosphate levels decrease. This stimulates parathyroid hormone release resulting in increased calcium resorption from bone. Maintenance of calcium levels comes at an expense of the skeletal system.

- Vitamin D synthesis is impaired in CKD. The kidneys regulate vitamin D activity by converting the inactive form-25(OH)to calcitriol (1,25(OH)vitamin D3). Calcitriol has a direct suppressive effect on PTH. In addition, reduced calcitriol levels leads to impaired GIT calcium absorption. Vitamin D also regulates osteoblast differentiation, thereby affecting bone replacement.

SYMPTOMS

- Bone pain
- Numbness or swelling in hands and feet
- Blood in stool
- Problem with sexual function
- Weight loss
- vomiting

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5.2.3.4 Complications

Some complications are associated with chronic renal failure. Some of these complications include:

- Anemia.
- High blood pressure (hypertension).
- Opportunistic infections.
- Dehydration.
- Electrolyte abnormalities (e.g. hyperkalemia, high levels of potassium in the blood).
- Mineral abnormalities (e.g. hypercalcemia or hyperphosphatemia).
- Malnutrition.
- Seizures.