Renal failure pathophysiology

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The term renal failure means the inability of the kidneys to perform excretory function leading to retention of nitrogenous waste products from the blood.

Functions of the kidney are as follows:

- Blood Electrolyte and volume regulation
- Excretion of nitrogenous waste
- Elimination of exogenous molecules, for example, many **drugs**
- Synthesis of a variety of hormones, for example, **erythropoietin**
- Metabolism of low molecular weight proteins, for example, **insulin**

Acute and chronic renal failure are the two kinds of kidney failure.

Acute Renal Failure (ARF)

ARF is the syndrome in which **glomerular filtration** declines abruptly (hours to days) and is usually reversible. According to the KDIGO criteria in 2012, Acute kidney injury (AKI) can be diagnosed with any one of the following:

(1) Creatinine increase of 0.3 mg/dL in 48 hours,

(2) Creatinine increase to 1.5 times baseline within last 7 days, or

(3) Urine volume less than 0.5 mL/kg per hour for 6 hours.

Recently the term acute kidney injury (AKI) has replaced ARF because AKI denotes the entire clinical spectrum from a mild increase in serum creatinine to obvious renal failure.

Chronic Renal Failure (CRF)

CRF or chronic kidney disease (CKD) is defined as a persistent impairment of kidney function.

in other words, abnormally elevated serum creatinine for more than 3 months or calculated glomerular filtration rate (GFR) less **than 60 ml per minute / 1.73m2**.

It often involves a progressive loss of kidney function necessitating renal replacement therapy (dialysis or transplantation).

When a patient needs renal replacement therapy, the condition is called end-stage renal disease (ESRD).

CKD classified based on grade:

- Grade 1: GFR greater than 90
- Grade 2: 60 to 89
- Grade 3a: 45 to 59
- Grade 3b: 30 to 44
- Grade 4: 15 to 29
- Grade 5: Less than 15

Renal Failure pathophysiology:

Acute Renal Failure

1. **Prerenal** (approximately 60%):

- sever Hypotension,
- blood contraction (e.g., sepsis, hemorrhage),
- severe organ failure such as heart failure or liver failure,

- drugs like non-steroidal anti-inflammatory drugs (NSAIDs).
- angiotensin receptor blockers (ARB).
- angiotensin-converting enzyme inhibitors (ACEI).
- Cyclosporine.

2. **Intrarenal** (approximately 35%):

- Acute tubule necrosis (from prolonged prerenal failure, radiographic contrast material, drugs like aminoglycosides, or nephrotoxic substances).
- Acute interstitial nephritis (drug-induced).
- Connective tissue disorders (vasculitis).
- Arteriolar disease.
- Arterial emboli.
- Intrarenal deposition (seen in tumor-lysis syndrome, increased uric acid production and multiple myeloma-Bence-Jones proteins).
- **Rhabdomyolysis**. Occurs when damaged muscle tissue releases its proteins and electrolytes into the blood. These substances can damage the heart and kidneys and cause permanent disability or even death.

3. **Postrenal** (approximately 5%):

- Extrinsic compression (prostatic hypertrophy, carcinoma).
- Intrinsic obstruction (calculus, tumor, clot, stricture).
- Decreased function (neurogenic bladder).

Chronic Renal Failure

- Diabetes mellitus, especially type 2 diabetes mellitus, is the most frequent cause.
- Hypertension is the second most frequent cause.
- Glomerulonephritis.

- Polycystic kidney diseases.
- Renal vascular diseases.
- prolonged obstruction of the urinary tract, nephrolithiasis.
- Vesicoureteral reflux, a condition in which urine to back up into the kidneys.
- Recurrent kidney infections/ pyelonephritis.
- Unknown etiology.

Renal failure pathophysiology can be described by a sequence of events that happen while during acute stage in the setting of acute renal failure and also gradually over a period in cases of chronic kidney diseases.

Broadly, AKI can be classified into three groups.

The pathophysiology of CRF is related mainly to specific initiating mechanisms. Over the course of time-adaptive physiology plays a role leading to compensatory hyperfiltration and hypertrophy of remaining viable nephrons.