Investigation of renal function

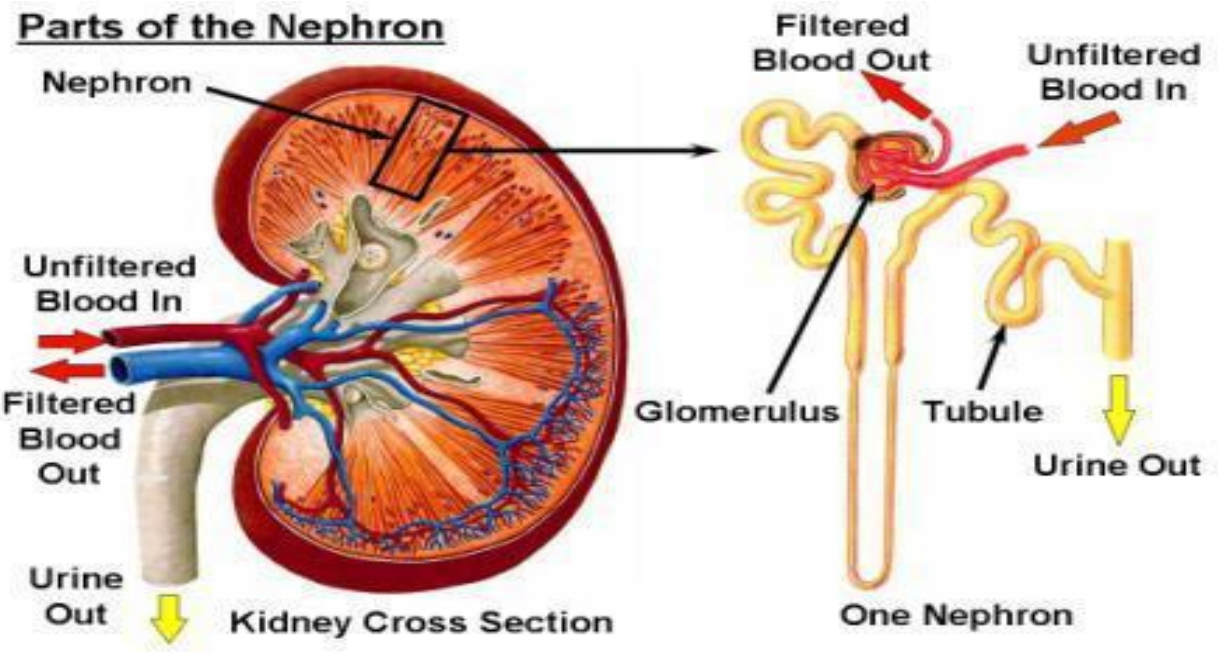
Functions of kidneys:

The functional unit in the kidney is the nephrons, the functions of kidneys include:

- Regulation of water, electrolyte and acid-base balance.
- Excretion of the products of protein and nucleic acid metabolism; e.g. urea, creatinine and uric acid. The kidneys are also endocrine organs, producing a number of hormones, and are subject to control by others.

Arginine vasopressin (AVP) acts to influence water balance, and Aldosterone affects sodium reabsorption in the nephron. Parathyroid hormone promotes tubular reabsorption of calcium, phosphate excretion and the synthesis of 1,25 dihydroxycholecalciferol, which regulate calcium Absorption by the gut.

Erythropoietin, a peptide hormone, promotes hemoglobin synthesis. The endocrine functions of the kidney remain clinically intact until the end stage of renal failure.



Renal tubular function:

The glomeruli provide an efficient filtration mechanism for ridding the body of waste products and toxic substances. To ensure that important constituents, such as water, sodium, glucose, and amino acids, are not lost from the body, tubular reabsorption must be equally efficient.

Tubular dysfunction:

Some disorders of tubular function are inherited, for example some patients are unable to reduce their urine PH below 6.5, because of a failure of hydrogen ion secretion. However, renal tubular damage is much more frequently secondary to other condition or insults. Any cause of acute renal failure may be associated with renal tubular failure.

Specific proteinuria:

Has already been made of protein in urine as an indicator of leaky glomeruli. An increased concentration of these proteins in urine is a sensitive indicator of renal tubular cell damage.

Glycosuria:

The presence of glucose in urine when blood glucose is normal usually reflects the inability of the tubules to reabsorb glucose because of a specific tubular lesion.

Aminoaciduria:

Normally amino acids in the glomular filtrate are reabsorbed in the proximal tubules. They may be present in urine in excessive amount because the plasma concentration exceeds the renal threshold, or because there is specific failure of normal tubular absorptive mechanisms.

Specific tubular defect; fanconi syndrome:

The fanconi syndrome is a term used to describe the occurrence of generalized tubular defects such as renal tubular acidosis, aminoaciduria, and tubular proteinuria.

Renal stone:

Renal stones (calculi) produce severe pain and discomfort, and are common causes of obstruction in the urinary tract.

Investigation of renal failure:

Renal failure is the cessation of kidney function. Renal failure is classified as either acute or chronic. Acute renal failure occurs suddenly, often secondary to sudden acute illness or to therapy.

Acute renal failure:

In acute renal failure (ARF)**,** the kidneys fail over a period of hours or days. Chronic renal failure (CRF) develops over months or years and leads eventually to end stage renal failure (ESRF). ARF may be reversed and normal renal function regained, whereas CRF is irreversible.

Etiology:

ARF arises from a variety of problems affecting the kidneys and their circulation. It usually present as a sudden deterioration of renal function indicated by rapidly rising serum urea and creatinine concentrations.

Renal failure or uremia may be classified as:

pre-renal, renal, or post-renal in origin.

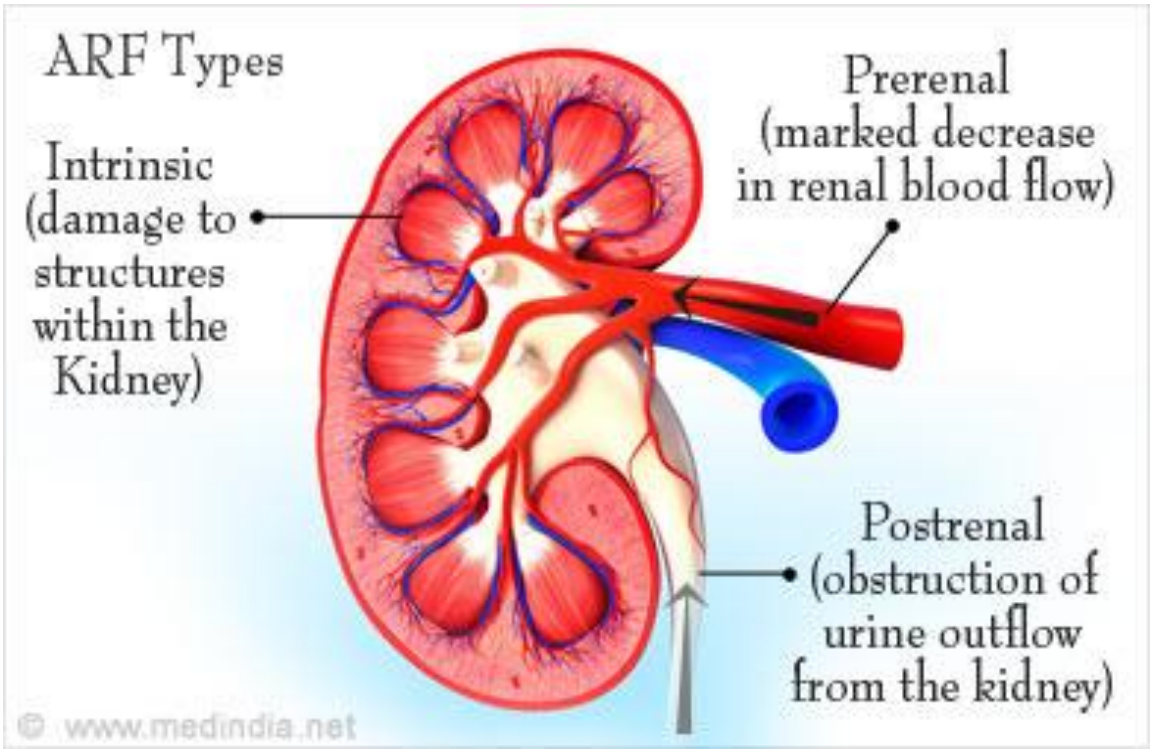
- ✓ Pre-renal: may be caused by decreased circulation, low blood volume, or decreased fluid volume reaching the kidney.
- ✓ Post-renal: may occur as the result of urinary obstruction, which reduces the flow of urine from the kidney. Reduced glomerular filtration rate is often the indication of post-renal.
- ✓ Renal: intrinsic damage to the kidney tissue.

Acute tubular necrosis:

Acute tubular necrosis may develop in the absence of pre-existing pre-renal or post-renal failure. The causes include:

- Acute blood loss in severe trauma
- Septic shock
- Specific renal disease, such as glomerulonephritis.
- Nephrotoxins, such as the aminoglycosides, analgesics or herbal toxins.

Patients in the early stages of acute tubular necrosis may have only a modestly increased serum urea and creatinine that then rise rapidly over a period of days, in contrast to the slow increase over months and years seen in chronic renal failure.



The finding in venous plasma and urine from the effected nephrons will be as follow:

Plasma:

1. High urea (uremia) and creatinine concentrations.
2. Low bicarbonate concentration, with low PH (acidosis).
3. Hyperkalemia.
4. Hyperuricemia and hypophosphatemia.

Urine:

1. Reduced volume (oliguria).
2. Low appropriate sodium concentration- only if renal blood flow is low, stimulating aldosterone secretion.
3. High appropriate urea concentration and therefore a high osmolality- only if ADH secretion is stimulated.

Acute renal dysfunction:

In adults, oliguria is defined as urine output of less than 400 ml/day, or less than 15 ml / hours, is usually indicates a low GFR and acute renal failure.

Acute oliguria with reduced GFR (pre-renal):

This caused by factors that reduce the hydrostatic pressure gradient between the renal capillary and tubular lumen. A low intracapillary pressure is the most common cause. It is noun as renal circulatory insufficiency (pre-renal uremia) and may be due to:

1. Intravascular depletion of whole blood (hemorrhage) or plasma volume (usually due to gastrointestinal lose) or reduced intake.
2. Reduced pressure as a result of vascular dilation caused by shock, causes of which include myocardial infarction, cardiac failure, and intravascular hemolysis, including that due to mismatched blood transfusion.

Acute oliguria due to intrinsic renal damage:

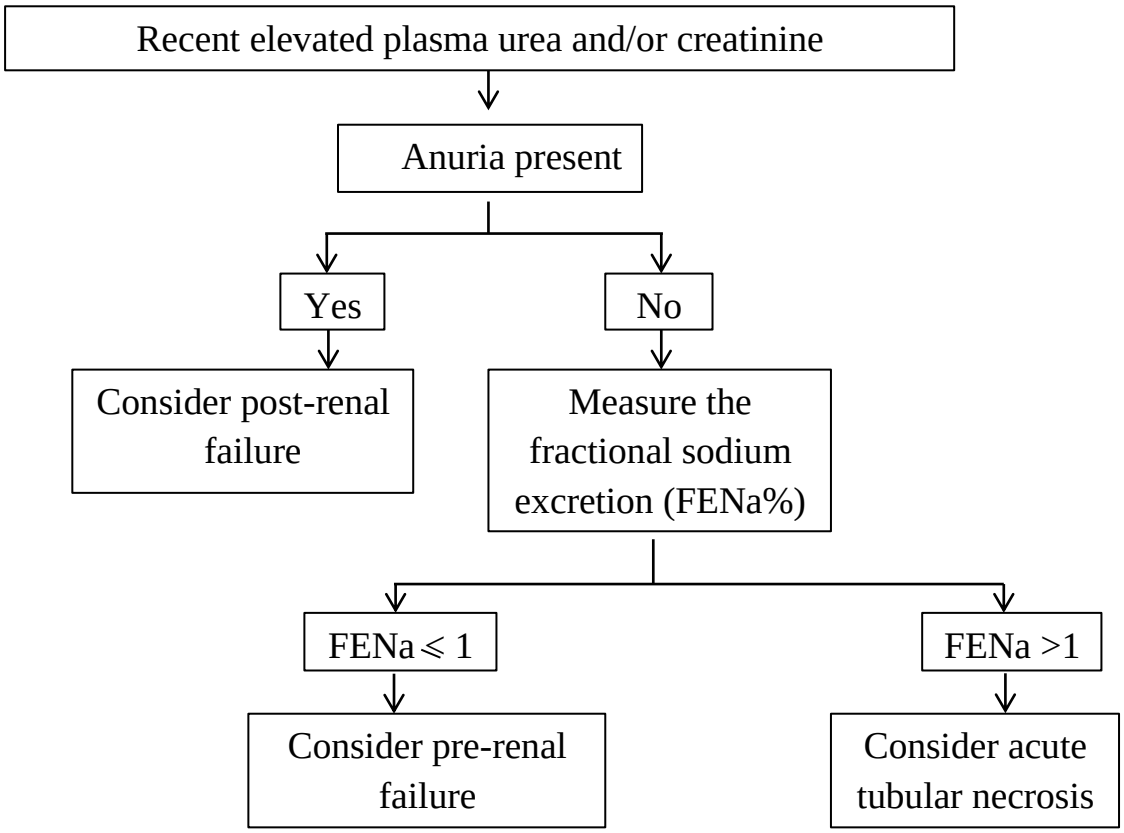
This may be due to:

1. Prolonged renal circulatory insufficiency.
2. Acute glomerulonephritis.
3. Septicemia.
4. Ingestion of varity poisons or drugs.
5. Myoglobulinuria.
6. Bence-jones proteinuria.

Acute oliguria due to renal outflow obstruction (post-renal):

Oliguria or anuria (absence of urine) may occur in post renal failure. The cause is usually, but not always, clinically obvious and may be due to the following:

1. Internal obstruction, with blockage of the tubular lumina by hemoglobin, myoglobin and, very rarely urate or calcium.
2. External obstruction, due to calculi, neoplasms, e.g. prostate, cervix, urethral strictures.



Algorithm for the investigation of acute renal failure.

Chronic renal failure

Chronic renal failure (CRF) is the progressive irreversible destruction of kidney tissue by disease which, if not treated by dialysis or transplant, will result in the death of the patient.

Chronic renal dysfunction or (failure):

Chronic renal dysfunction (defined as being of more than 3 month's duration) is usually the end result of condition like diabetes.

Some causes of renal dysfunction or failure:

1. Diabetes mellitus.
2. Nephrotoxic drugs.
3. Hypertension.
4. Glomerulonephritis.
5. Chronic pyelonephritis.
6. Polycystic kidneys.
7. Urinary tract obstruction.
8. Severe urinary infections.
9. Amyloid and Para-proteins.
10. Progression from acute renal failure.

Chronic renal dysfunction may pass through two main phases:

A. An initially polyuric phase.

B. Subsequent oliguria or anuria, sometimes needing dialysis or renal transplantation.

Polyuric phase:

At first, glomerular function may be adequate to maintain plasma urea and creatinine concentration within the reference range. Both tubular dysfunction in nephrons with functioning glomeruli and the osmotic diuresis through intact nephrons contribute to the polyuria, other causes of which should be excluded. The presence of increasing proteinuria may be the best single predictor of disease progression.

Stages of renal dysfunction.

stage	Description	Metabolic features
1	Normal or increased GFR	normal
2	Early renal insufficiency	-Plasma urea and creatinine rise -PTH start to rise
3	Chronic renal failure	-Calcium absorption decreased - Lipoprotein lipase decreased -Malnutrition -Anemia-erythropoietin decreased
4	Severe renal failure (pre-end stage)	-Hypertriglyceridemia -Hyperphosphatemia -Metabolic acidosis -Hyperkalemia
5	End-stage renal failure	Marked elevation of urea (uremia) and creatinine

Nephrotic syndrome:

The nephrotic syndrome is caused by increased glomerular basement membrane permeability, resulting in protein loss, usually more than 3 g a day, with consequent hypoproteinemia, hypoalbuminemia and peripheral odema. The main effect on the plasma proteins and are associated with hyperlipidemia and hyperfibrinogenemia. Uremia only occurs in late stages of the disorder, when glomeruli cease to function.