NAME: ADENIYI ADERONKE TEMILOLA

MATRIC NUMBER: 17/MHSO1/019

LEVEL: 300

DEPARTMENT: MEDICINE AND SURGERY

1. Discuss the pathophysiological process involved in renal failure?

The kidney is a two bean-shaped organ that controls volume of various [body fluids](https://en.wikipedia.org/wiki/Body_fluid), removal of [toxins](https://en.wikipedia.org/wiki/Toxins) by the formation and excretion of urine. They are located in the left and right of the retroperitoneal space.

Renal failure refers to the failure/ deterioration of excretory functions of kidney as result of decrease in glomerular filtration rate (GFR) and rise in urea and non-nitrogenous substances in the blood. It is of two types:

* Acute renal failure
* Chronic renal failure

**Acute Renal Failure (ARF)**

This is the abrupt or sudden stoppage of renal functions. It is often reversible within few days to few weeks and may result in sudden life-threatening reactionsin the body with the need for emergency treatment.

Kidney Disease Improving Global Outcomes ([KDIGO](http://kdigo.org/home/guidelines/acute-kidney-injury/)) defined ARF by one of these 3 criteria:

* Increase in creatinine serum level of more than 0.3 mg/dl (within 48 hours).
* Increase in creatinine serum level by more than 50% (within the prior 7 days).
* Decrease in urine volume (oliguria) to less than 0.5 ml/kg body weight/hour for 6 hours.

Causes

Because of various pathophysiologic processes, the kidney can diminish rapidly. Some causes of ARF include: Acute nephritis, damage of renal tissues by poisons like lead, renal ischemia, acute tubular necrosis, severe transfusion reactions, sudden fall in blood pressure during hemorrhage, diarrhea, severe burns and cholera, blockage of kidney function As a consequence, uremic substances accumulate and alter water, electrolyte, and acid-base balances. Usually, this loss of function is reversible, as long as the underlying cause or structural processes do not become chronic.

Types and pathophysiology

Etiologically, ARF can be subdivided into 3 types. Each type encompasses different pathophysiological processes, so understanding the type of ARF presenting in a patient is crucial to identifying the choice of treatment:

* **Prerenal ARF (about 60 % of all cases)**

This is the result of reduced renal perfusion, with the glomerular and tubular structures initially being completely intact. It may be caused by actual hypovolemia (e.g., due to exsiccosis, [diarrhea](https://www.lecturio.com/magazine/diarrhoea/), or pancreatitis), but also relative hypovolemia stemming from e.g., [cardiac insufficiency](https://www.lecturio.com/magazine/cardiac-insufficiency-pulmonary-edema/), shock, or sepsis. Diseases that cause renal vasoconstriction may result also in prerenal failure. Through the regulation mechanisms of the [kidney](https://www.lecturio.com/magazine/kidney/), reduced perfusion activates the renin-angiotensin-aldosterone system (RAAS). At the same time, the body experiences a release of catecholamine and ADH. This reaction is vasoconstriction with simultaneous retention of sodium and water in order to compensate for the hypovolemic condition. In the presence of cardiac insufficiency, actual reduced perfusion does not involve a lack of water. Clinically, signs of hyperhydration predominate. The activation of RAAS erroneously increases the intracorporeal water concentration and hyperhydration increases. If diuretics are administered in this situation, renal perfusion will be reduced even more, increasing the risk of ischemia and intrinsic renal failure.

* **Intrinsic Renal Failure (about 35 % of all cases)**

Acute damage to the glomeruli or tubular cells leads to structural damage of the kidney itself. Glomerulonephritides may also lead to reduced kidney function. Toxic damage, especially iatrogenic damages, is frequent. Contrast agents or other medications play an important role here. A number of frequently administered drugs can also cause damage to the kidneys, including non-steroidal anti-inflammatory drugs, aminoglycosides, cephalosporinand so on.

The renal tubules are responsible for reabsorption. If an intrinsic renal dysfunction affects the tubules, this may cause severe polyuria as part of ARF. If sodium reabsorption is diminished because of damage to the tubular cells, the tubuloglomerular feedback mechanism causes constriction of the afferent glomerular arteriole. This, in turn, leads to a reduction in the glomerular filtration rate.

* **Postrenal Failure (about 5 % of all cases)**

Diseases with the potential to impair the drainage of urine from the kidneys can lead to urinary retention with subsequent postrenal failure. Congenital malformations of the urinary tract should be excluded as a cause as should various acquired urinary obstructions including tumors, gynecological conditions, urinary catheters, outflow obstructions due to medication, prostate enlargement, and ureteric stones.

**Complications of Acute Renal Insult**

This is due to the above-noted pathophysiological processes which affects many organ systems.

* The [lungs](https://www.lecturio.com/magazine/lung/) can be affected by hyperhydration, including [edema](https://www.lecturio.com/magazine/edemas-as-cardinal-symptoms/) and effusion. Acute respiratory distress syndrome may occur.
* Heart failure may develop due to hypertension or hyperhydration, or arrhythmias may develop due to imbalanced electrolyte concentrations.
* If heart failure occurs, there is a risk of congestion in the venous circuit causing gastritis, ulcerations, or gastrointestinal bleeding. The stress-associated release of hormones can increase the likelihood of gastrointestinal bleeding.
* Seizures may occur due to a cerebral edema or electrolyte imbalance. In addition, vigilance can be impaired.

**Treatment of Acute Renal Insult**

* **Substitution of Fluids and Electrolytes for Acute Renal Insult of Prerenal Genesis**

In prerenal ARF, kidney function can only recover when the underlying pathophysiological mechanism has been eliminated. Nephrotoxic substances should be avoided, and fluid and electrolyte balances must be thoroughly controlled and treated. The reason for the hypoperfusion must be uncovered and then treated. The administration of fluids and electrolytes is a prudent option. Loop diuretics can also be helpful in maintaining diuresis (Note that while this medication measurably increases diuresis, it does not increase glomerular filtration or have any impact on the recovery of kidney function.).Patients with sepsis or with severe heart failure often require treatment in the intensive care unit.

* **Immunosuppressive Therapy and Revascularization for Acute Renal Failure of Intrinsic Genesis**

In intrinsic ARF, it is important to first treat the underlying disease. Immunosuppressive treatment is advisable for glomerulonephritis, and revascularization for ischemia. For raising diuresis, loop diuretics can be administered; however, the use of diuretics is controversial and therefore not generally recommended. The only absolute indication for the administration of diuretics is hyperhydration.

* **Treatment of Acute Renal Insult of Postrenal Genesis**

In cases of postrenal ARF, it is imperative to remove the urinary obstruction. If this is not immediately possible, the surgical insertion of an artificial excretory opening (percutaneous nephrostomy) is indicated.

* **Extracorporeal Treatment of Acute Renal Failure**

Extracorporeal treatment with hemodialysis or hemofiltration for electrolyte imbalances, water overloads, or acid-base imbalances can also be attempted. This type of renal replacement therapy should be considered only as a temporary measure and limited accordingly. If kidney function cannot be restored sufficiently, permanent dialysis may become necessary. This type of renal replacement therapy should be considered only as a temporary measure which must be limited accordingly. If the kidney function cannot be restored sufficiently, permanent dialysis might become necessary.

**Chronic Renal Failure (CRF)**

Chronic renal failure is defined as an irreversible, progressive decrease of glomerular, tubular and endocrine renal function. This damage has to have been exhibited for longer than 3 months.

**Pathophysiological Processes of Chronic Kidney Disease**

During the damage of the kidney, there is modulation and adaptation in the still-functional glomeruli, which keeps the kidneys functioning normally for as long as possible. The remaining glomeruli, therefore, experience a rise in pressure through hyperfiltration. The release of various cytokines and growth factors leads to hypertrophy and hyperplasia. At the same time, the function of the glomeruli suffers due to the excessive demands on them, leading to increased permeability and proteinuria. Increased protein concentrations in the proximal tube system are direct nephrotoxins and can further impair kidney function.

There are four phases of chronic renal failure:

* **Reduction in Excretory Function**

Breakdown of excretory function is the consequence of an accumulation of endogenous and extraneous substances. This leads to changes in pharmacokinetics and an increase in the concentration of various medications. Breakdown occurs when the remaining glomeruli are confronted by a surplus of waste products, leading to osmotic diuresis. There is a reduction in the maximal concentrating capacity of the kidney. In order to filter the physiological quantity of dissolved substances, the nephrons produce between 3 and 4 times as much urine during renal failure, resulting in an accumulation of waste substances.

* **Reduction in Incretory Renal Function**

Because the kidney plays a part in the regulation of many important hormonal cycles, chronic renal failure also has endocrinal consequences. Through a shortage of erythropoietin, there is a reduction in erythrocyte synthesis, which leads to renal anemia; uremia then leads to a reduction of functional erythrocytes due to hemolysis or hemorrhages. Vitamin D production is also impaired, and phosphate excretion is reduced. Secondary hyperparathyroidism and the associated renal osteopathy (‘high-turnover’ osteopathy) develop as a result of hyperphosphatemia. Parallel to this, other pathomechanisms lead to a disruption in bone metabolism: osteomalacia occurs due to a disruption of mineralization, and adynamic [bone](https://www.lecturio.com/magazine/bones-fundamentals-of-anatomy/) disease occurs due to a reduction in bone cell activity (particularly in dialysis patients).

* **Over-hydration and the Disruption of Electrolyte Balance**

As long as the glomeruli can manage to compensate, diuresis and fractional sodium excretion rise. If the glomerular filtration rate noticeably drops, then the ability to compensate is exhausted, leading to increased retention of water and electrolytes. Hypertension, pulmonary edema, and peripheral edema result from overhydration. Water and salt excretion are thereby inextricably linked. Diuretics can aid in water and salt excretion where critical glomerular damage is present. Early loss of salts as a result of the disturbance in the resorption process can actually be made worse by the use of diuretics. Thus, as the glomeruli adapt to compensate, the tubular transport mechanisms also adapt in order to prevent hyperkalemia through increased potassium secretion. Hyperkalemia only develops as a result of hyperstimulation of the resorption capacity. As many patients are treated with calcium-sparing diuretics due to previous conditions, it is vital to refer to patient’s medication history and adapt the treatment plan accordingly. Acidosis also rises alongside hyperkalemia. The kidneys can no longer sufficiently eliminate accumulating protons due to a strongly reduced glomerular filtration rate. This metabolic acidosis leads to increased bone calcium release and strengthening renal osteopathy, an increase in gastrointestinal problems, and the impairment of protein metabolism.

* **Toxic Organ Damage as a Result of Retention of Urinary Excreted Metabolites**

Toxic organ damage can be explained under the umbrella term ‘uremic syndrome.’ The rise in urinary excreted metabolites in the blood is called azotemia. These metabolites include urea, creatinine, beta-2 microglobulin, parathyroid hormone, among others. Uremic syndrome (uremia) principally describes a systemic disruption of all organ functions, especially the circulatory system, central nervous system, blood, and membranes. Clinically, many symptoms of chronic renal failure can be detected via the [skin](https://www.lecturio.com/magazine/skin/). Patients often have macules (‘café au lait’ spots), are conspicuously pale, and have a gray, dirty-looking complexion. They often complain of pruritus. Internal membranes are also affected, leading to pericarditis, peritonitis, and pleurisy. Uremia can also lead to hemolysis with anemia. Simultaneously, thrombocyte and leukocyte dysfunctions or deficiencies can arise. People with chronic renal failure have a generally increased risk of atherosclerosis with an elevated cardiovascular risk. This leads to media calcification caused by calcium phosphate and to intima calcification through inflammatory factors and cholesterol plaques. Hypertension is common, along with edemas and pulmonary congestion. Impairments of the central nervous system are indicated by a reduction in vigilance, from general drowsiness to uremic coma. Seizures can occur. Uremia also causes polyneuropathy with paresthesia.

**Symptoms of Chronic Kidney Disease**

CRF often begins with generalized symptoms such as tiredness, loss of appetite, and headaches. Further early indicators are polyuria, newly emerging or worsening hypertension, or peripheral edemas. Depending on the etiology, there can also be flank pain or fever.As the disease progresses, increased tiredness, paleness, headaches, visual disturbances, and a severe loss of renal capacity become noticeable. Uremic gastroenteropathy leads to a loss of appetite and nausea. Pruritus occurs and muscle fibrillations become apparent.In the final stages, renal failure leads to oliguria or anuria, dyspnea, vomiting, uremic encephalopathy with a severe reduction in vigilance, and increased susceptibility to bleeding.

**TREATMENT**

* Loop diuretics are recommended for more advanced renal failure. If over time, the diuretic effect begins to weaken, diuretic resistance may be present; this can be overcome by sequential nephron blockade in which loop diuretics are combined with thiazide. The resultant loss of electrolytes must be closely monitored and replaced, however.
* Symptoms of hyperkalemia should be monitored. A low-potassium diet is recommended and potassium-sparing diuretics should not be prescribed. If renal acidosis occurs, serum bicarbonate values of < 22 mmol/L can be counterbalanced by the administration of bicarbonate.
* Adequate regulation of blood pressure can often be achieved by undertaking combined antihypertensive therapy, during which angiotensin-converting-enzyme inhibitors or angiotensin receptor blockers should be avoided due to their nephroprotective properties.
* As renal failure is accompanied by a change in pharmacokinetics, appropriate medication adjustments must be made in order to avoid intoxication.
* Renal anemia can be improved with synthetic erythropoietin. Depending on the blood parameters, iron supplements can also be necessary, particularly if dialysis is underway and blood loss is occurring, as this is often accompanied by iron deficiency.
* If it is not possible to stop or slow the progress of renal failure via conservative therapies, then renal replacement therapy is essential. There is a range of different extra or intracorporeal dialysis treatments available.

The treatment of choice for terminal renal failure is kidney transplant. Kidney transplant is far preferable to long-term dialysis, despite the operative procedure and immunosuppressive therapy necessary

1. With the aid of suitable diagrams discuss the types of dialysis you know?

Dialysis is the procedure to remove waste materials, toxic substances and to restore normal volume and composition of body fluid in severe renal failure. It is a procedure that is when the kidney is not functioning properly due to disease or injury to it. Types of dialysis I know:

1. **Peritoneal dialysis**

This involves the use of fluid that is placed into the patient's abdominal cavity through a plastictube (peritoneal dialysis catheter) to remove excess waste products and fluid from the body. Peritoneal dialysis uses the patients own body tissues inside of the belly (abdominal cavity) to act as the fllter. The abdominal cavity is lined with a special membrane called the peritoneal membrane. A plastic tube called a peritoneal dialysis catheter is placed through the abdominal wall into the abdominal cavity. A special fluid called dialysate is then flushed into the abdominal cavity and washes around the intestines. The peritoneal membrane acts as a filter between this fluid and the blood stream. By using different types of solutions, waste products and excess water can be removed from the body through this process. There are three different types of peritoneal dialysis:

* Continuous ambulatory peritoneal dialysis (CAPD): Does not require a machine. Exchanges often referred to as "passes," can be done three to five times a day, during waking hours.
* Continuous cyclic peritoneal dialysis (CCPD): Requires the use of a special dialysis machine that can be used in the home. This type of dialysis is done automatically, even while you are asleep.
* Intermittent peritoneal dialysis (IPD): Uses the same type of machine as CCPD, but treatments take longer. IPD can be done at home, but it is usually in the hospital.



Peritoneal dialysis

Possible complications of peritoneal dialysis include an infection of the peritoneum, or peritonitis, where the catheter enters the body. Peritonitis causes fever and stomach pain. A dietician will help plan the diet during peritoneal dialysis, to ensure choosing the appropriate meals.

1. **Hemodialysis**

This uses an external machine and a special type of filter to remove excess waste products and water from the blood. During hemodialysis, blood passes from the patient's body to the **dialysis machine** through sterile tubing and into a filter, called a dialysis membrane. For this procedure, the patient has a specialized vascular tube placed between an artery and a vein in the arm or leg (called a gortex graft). Sometimes, a direct connection is made between an artery and a vein in the arm. This procedure is called a Cimino fistula. Needles are then placed in the graft or fistula, and blood passes to the dialysis machine, through the filter, and back to the patient. If the patient requires dialysis before a graft or a fistula is placed, a large diameter catheter (hemodialysis catheter) is placed directly into a large vein in the neck or leg in order to perform dialysis. In the dialysis machine, a solution on the other side of the filter receives the waste products from the patient. Hemodialysis is usually performed several times a week and lasts for four to five hours.



Hemodialysis

Possible complications of hemodialysis include muscle cramps and hypotension (sudden drop in blood pressure). Hypotension may cause you to feel dizzy, weak or sick to your stomach. You can usually avoid side effects by following the proper diet and taking your medications.