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Matric number: 17/Mhs01/169

Level: 300

MBBS

Renal physiology

1. **Discuss the pathophysiological process involved in renal failure.**

Anemia

Initially, as renal tissue loses function, there are few noticeable abnormalities because the remaining tissue increases its performance (renal functional adaptation).

Decreased renal function interferes with the kidneys' ability to maintain fluid and electrolyte homeostasis. The ability to concentrate urine declines early and is followed by decreases in ability to excrete excess phosphate, acid, and potassium.

Anemia is characteristic of moderate to advanced CKD (≥ stage 3). The anemia of chronic renal disease is normochromic-normocytic, with an Hct of 20 to 30% (35 to 40% in patients with polycystic kidney disease). It is usually caused by deficient erythropoietin production due to a reduction of functional renal mass.

Immune function

Sepsis is a leading cause of death in patients with renal failure. Inhibition of cell-mediated immunity and humoral defence mechanisms occurs, with little improvement following dialysis. There is an increased production of pro-inflammatory cytokines suggesting that activation of monocytes may play a role in uraemic immune dysfunction.

Gastrointestinal abnormalities

Gastrointestinal abnormalities are frequent with anorexia, nausea and vomiting contributing to malnutrition. Urea is a mucosal irritant and bleeding may occur from any part of the GI tract. Gastric emptying is delayed, residual volume increased and pH lowered. Peptic ulcer disease is common and most patients will receive proton pump inhibitors.

Neurological abnormalities

Many patients with chronic renal failure have abnormalities in central (CNS) and peripheral nervous system function. There is a wide spectrum of CNS changes. for example, from mild personality alterations to asterixis (i.e. lapse of posture, usually manifest by bilateral flapping tremor), myoclonus, encephalopathy and convulsions. Peripheral neuropathy is common in advanced stages of the disease.

Cardiovascular and pulmonary abnormalities

Cardiovascular abnormalities are common in CRF and are responsible for 48% of deaths in these patients. Systemic hypertension is the most common with an incidence approaching 80%, although it is often not a feature of sodium-wasting nephropathies such as polycystic kidney disease or papillary necrosis. Plasma volume expansion resulting from sodium and water retention is the most frequent cause of hypertension; it may be improved significantly by dialysis.

Diabetes Mellitus and Hypertension

In recent years, diabetes mellitus and hypertension have become recognized as the leading causes of ESRD, together accounting for more than 70 percent of all ESRD. Excessive weight gain (obesity) appears to be the most important risk factor for the two main causes of ESRD—diabetes and hypertension. Concurrent diabetes and hypertension greatly increases the frequency and severity of nephrosclerosis and glomerulosclerosis. Benign nephrosclerosis, the most common form of kidney disease. This type of vascular lesion occurs in the smaller interlobular arteries and in the afferent arterioles of the kidney. It is believed to begin with leakage of plasma through the intimal membrane of these vessels. This leakage causes fibrinoid deposits to develop in the medial layers of these vessels, followed by progressive thickening of the vessel wall that eventually constricts the vessels and, in some cases, occludes them. Because there is essentially no collateral circulation among the smaller renal arteries, occlusion of one or more of them causes destruction of a comparable number of nephrons. Therefore, much of the kidney tissue becomes replaced by small amounts of fibrous tissue. When sclerosis occurs in the glomeruli, the injury is referred to as glomerulosclerosis. This loss of glomeruli and overall nephron function is reflected by a progressive decrease in both renal blood flow and GFR. Even in healthy people without underlying hypertension or diabetes, renal plasma flow and GFR decrease by 40 to 50 percent by age 80 years.

Atherosclerosis

This is one of the most common vascular lesions that can lead to renal ischemia and death of kidney tissue. Atherosclerosis refers to the hardening of arteries or blood vessels caused by plaques (accumulations of fatty deposits usually cholesterol). Atherosclerosis can occur in the larger renal arteries leading to their constriction. Atherosclerotic lesions of the large arteries frequently affect one kidney more than the other and, therefore, cause unilaterally diminished kidney function. Hypertension often occurs when the artery of one kidney is constricted while the artery of the other kidney is still normal, a condition analogous to “two-kidney” Goldblatt hypertension.

Glomerulonephritis

Glomerulonephritis refers to inflammation of the glomeruli. Chronic glomerulonephritis can be caused by several diseases that cause inflammation and damage to the capillary loops in the glomeruli of the kidneys. In contrast to the acute form of this disease, chronic glomerulonephritis is a slowly progressive disease that often leads to irreversible renal failure. It may be a primary kidney disease, following acute glomerulonephritis, or it may be secondary to systemic diseases, such as systemic lupus erythematosus. In most cases, chronic glomerulonephritis begins with accumulation of precipitated antigen-antibody complexes in the glomerular membrane. In contrast to acute glomerulonephritis, streptococcal infections account for only a small percentage of patients with the chronic form of glomerulonephritis. Accumulation of antigen-antibody complex in the glomerular membranes causes inflammation, progressive thickening of the membranes, and eventual invasion of the glomeruli by fibrous tissue. In the later stages of the disease, the glomerular capillary filtration coefficient becomes greatly reduced because of decreased numbers of filtering capillaries in the glomerular tufts and because of thickened glomerular membranes. In the final stages of the disease, many glomeruli are replaced by fibrous tissue and are, therefore, unable to filter fluid.

Pyelonephritis

Renal interstitial injury caused by bacterial infection is called pyelonephritis. The infection can result from different types of bacteria but especially from Escherichia coli that originate from fecal contamination of the urinary tract. These bacteria reach the kidneys either by way of the blood stream or, more commonly, by ascension from the lower urinary tract by way of the ureters to the kidneys. Although the normal bladder is able to clear bacteria readily, there are two general clinical conditions that may interfere with the normal flushing of bacteria from the bladder:

- The inability of the bladder to empty completely, leaving residual urine in the bladder, and
- Obstruction of urine outflow.

With impaired ability to flush bacteria from the bladder, the bacteria multiply and the bladder becomes inflamed, a condition termed cystitis. Once cystitis has occurred, it may remain localized without ascending to the kidney, or in some people, bacteria may reach the renal pelvis because of a pathological condition in which urine is propelled up one or both of the ureters during micturition. This condition is called vesicoureteral reflux and is due to the failure of the bladder wall to occlude the ureter during micturition; as a result, some of the urine is propelled upward toward the kidney, carrying with it bacteria that can reach the renal pelvis and renal medulla, where they can initiate the infection and inflammation associated with pyelonephritis. Pyelonephritis begins in the renal medulla and therefore usually affects the function of the medulla more than it affects the cortex, at least in the initial stages. Because one of the primary functions of the medulla is to provide the countercurrent mechanism for concentrating urine, patients with pyelonephritis frequently have markedly impaired ability to concentrate the urine. With long-standing pyelonephritis, invasion of the kidneys by bacteria not only causes damage to

the renal medulla interstitium but also results in progressive damage of renal tubules, glomeruli, and other structures throughout the kidney. Consequently, large parts of functional renal tissue are lost and chronic kidney disease can develop.

Nephron Function in Chronic Kidney Disease

Although logically, decreasing the number of functional nephrons, which reduces the GFR, would also cause major decreases in renal excretion of water and solutes. Patients who have lost up to 75 to 80 percent of their nephrons are able to excrete normal amounts of water and electrolytes without serious accumulation of fluid or most electrolytes in the body fluids. Further reduction in the number of nephrons, however, leads to electrolyte and fluid retention, and death usually ensues when the number of nephrons falls below 5 to 10 percent of normal. In contrast to the electrolytes, many of the waste products of metabolism, such as urea and creatinine, accumulate almost in proportion to the number of nephrons that have been destroyed. The reason for this is that substances such as creatinine and urea depend largely on glomerular filtration for their excretion, and they are not reabsorbed as avidly as are the electrolytes.

Effects of Uremia

The effect of chronic kidney disease on the body fluids depends on

- Water and food intake and
- The degree of impairment of renal function.

The important effects include

1. Generalized edema resulting from water and salt retention
2. Acidosis resulting from failure of the kidneys to rid the body of normal acidic products
3. High concentration of the nonprotein nitrogens—especially urea, creatinine, and uric acid—resulting from failure of the body to excrete the metabolic end products of proteins; and
4. High concentrations of other substances excreted by the kidney, including phenols, sulfates, phosphates, potassium, and guanidine bases.

This total condition is called uremia because of the high concentration of urea in the body fluids.

2. With the aid of suitable diagrams, discuss the types of dialysis you know.

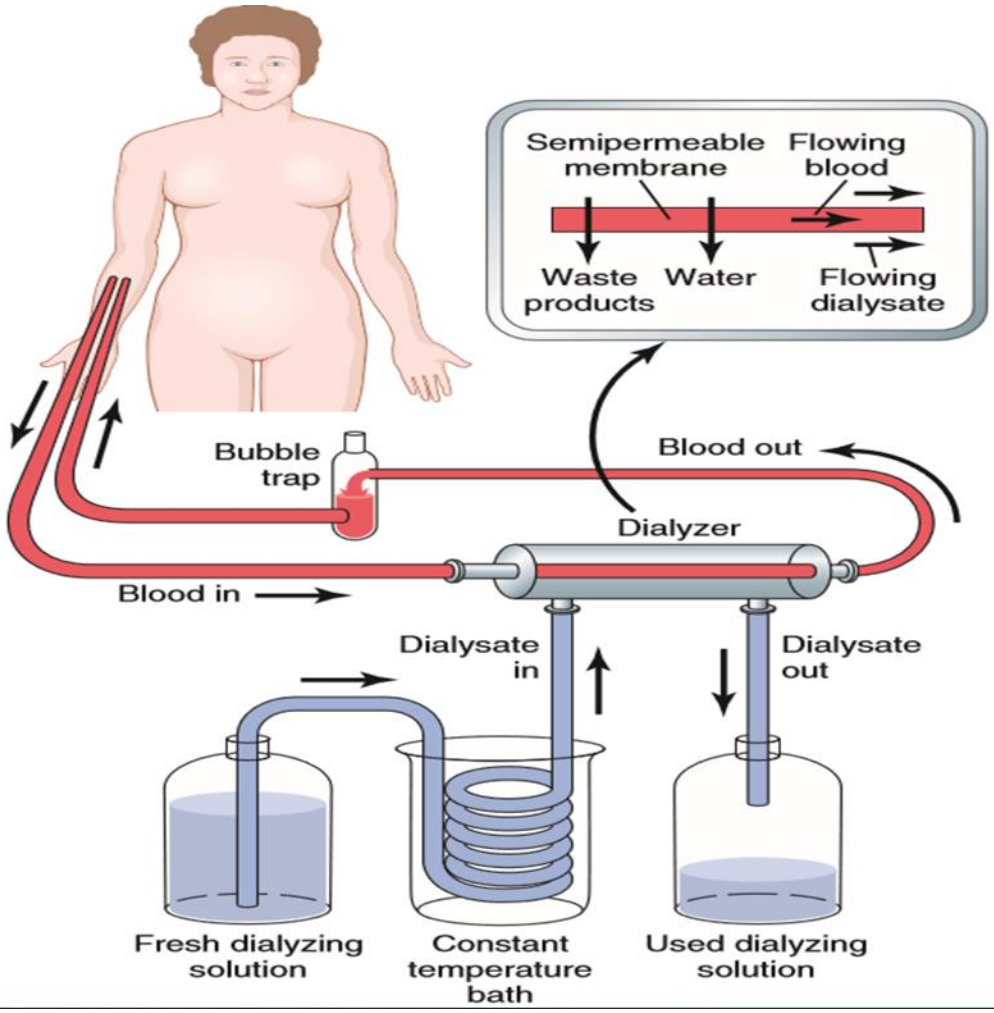
The general term dialysis means to separate substances using a permeable membrane. However, in clinical practice, dialysis is a technique used to perform the kidneys' excretory functions during renal failure

Dialysis is the process of removing excess water, solutes, and toxins from the blood in people whose kidneys can no longer perform these functions naturally. Dialysis is used in patients with rapidly developing loss of kidney function, called acute kidney injury, or slowly worsening kidney function, called stage 5 chronic kidney disease. Types of dialysis are:

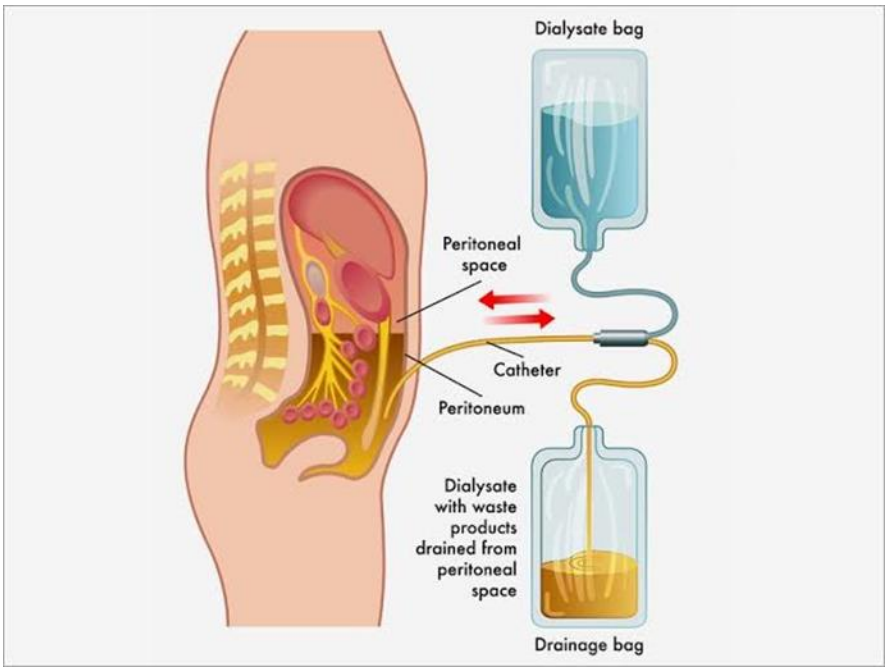
In hemodialysis, the patient's blood is pumped through the blood compartment of a dialyzer, exposing it to a partially permeable membrane. The dialyzer is composed of thousands of tiny hollow synthetic fibers. The fiber wall acts as the semipermeable membrane. Blood flows through the fibers, dialysis solution flows around the outside of the fibers, and water and wastes move between these two solutions. The cleansed blood is then returned via the circuit back to the body. Ultrafiltration occurs by increasing the hydrostatic pressure across the dialyzer membrane. This usually is done by applying a negative pressure to the dialysate compartment of the dialyzer. This pressure gradient causes water and dissolved solutes to move from blood to dialysate and allows the removal of several litres of excess fluid during a typical 4-hour treatment.

Disadvantages

- Restricts independence, as people undergoing this procedure cannot travel around because of supplies' availability.
- Requires more supplies such as high water quality and electricity.
- Requires reliable technology like dialysis machines.
- The procedure is complicated and requires that care givers have more knowledge



2. Peritoneal dialysis: Another way of removing excess substances from the blood is peritoneal dialysis, which uses the lining of the patient's own abdominal cavity (peritoneum) as a dialysis membrane. Fluid is injected via an indwelling plastic tube inserted through the abdominal wall into this cavity and allowed to remain there for hours, during which solutes diffuse into the fluid from the person's blood. The dialysis fluid is then removed and replaced with new fluid. This procedure can be performed several times daily by a patient who is simultaneously doing normal activities. While changing the bags of dialyzing fluid, one must pay attention to the aseptic technique. Also peritoneal catheter site must be taken care of to prevent peritonitis


Dialysate bag
Peritoneal space
Catheter
Peritoneum
Dialysate with waste products drained from peritoneal space
Drainage bag