ABA DORCAS MANGWO

PHS 212

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PHARMACOLOGY

QUESTION:DISCUSS THE DISEASES OF THE RENAL SYSTEM

ANSWER:

Renal Disease and Failure

Renal failure (also kidney failure or renal insufficiency) is a medical condition in which the kidneys fail to adequately filter waste products from the blood. The two main forms are acute kidney injury, which is often reversible with adequate treatment, and chronic kidney disease, which is often not reversible. In both cases, there is usually an underlying cause.

Diagnosis

Renal failure is mainly determined by a decrease in the glomerular filtration rate, which is the rate at which blood is filtered in the glomeruli of the kidney. This is detected by a decrease in or absence of urine production or determination of waste products (creatinine or urea) in the blood. Depending on the cause, hematuria (blood loss in the urine) and proteinuria (protein loss in the urine) may be noted.

In renal failure, there may be problems with increased fluid in the body (leading to swelling), increased acid levels, raised levels of potassium, decreased levels of calcium, increased levels of phosphate, and in later stages, anemia. Bone health may also be affected. Long-term kidney problems are associated with an increased risk of cardiovascular disease.

Categories of Renal Failure

Renal failure can be divided into two categories: acute kidney injury or chronic kidney disease. The type of renal failure is determined by the trend in the serum creatinine. Other factors that may help differentiate acute kidney injury from chronic kidney disease include anemia and the kidney size on ultrasound. Chronic kidney disease generally leads to anemia and small kidney size.

Kidney Transplantation: Kidney transplantation requires a person to be at the end stage of renal failure.

Acute kidney injury (AKI), previously called acute renal failure (ARF), is a rapidly progressive loss of renal function, generally characterized by oliguria (decreased urine production, quantified as less than 400 mL per day in adults, less than 0.5 mL/kg/h in children or less than 1 mL/kg/h in infants); and fluid and electrolyte imbalance. AKI can result from a variety of causes, generally classified as prerenal, intrinsic, and postrenal. An underlying cause must be identified and treated to arrest the progress, and dialysis may be necessary to bridge the time gap required for treating these fundamental causes.

Chronic kidney disease (CKD) can also develop slowly and, initially, show few symptoms. CKD can be the long-term consequence of irreversible acute disease or part of a disease progression.

Acute kidney injuries can be present on top of chronic kidney disease, a condition called acute-on-chronic renal failure (AoCRF). The acute part of AoCRF may be reversible, and the goal of treatment, as with AKI, is to return the patient to baseline renal function, typically measured by serum creatinine. Like AKI, AoCRF can be difficult to distinguish from chronic kidney disease if the patient has not been monitored by a physician and no baseline (i.e., past) blood work is available for comparison.

Symptoms can vary from person to person. Someone in early stage kidney disease may not feel sick or notice symptoms as they occur. When kidneys fail to filter properly, waste accumulates in the blood and the body, a condition called azotemia. Very low levels of azotaemia may produce few, if any, symptoms. If the disease progresses, symptoms become noticeable (if the failure is of sufficient degree to cause symptoms). Renal failure accompanied by noticeable symptoms is termed uraemia.

Renal Failure Uremia

Renal failure uremia is a syndrome of renal failure that includes elevated blood urea and creatinine levels. Acute renal failure can be reversed if diagnosed early. Acute renal failure can be caused by severe hypotension or severe glomerular disease. Diagnostic tests include BUN and plasma creatinine level tests. It is considered to be chronic renal failure if the decline of renal function is to less than 25%.

Nephroptosis (Floating Kidney)

Nephroptosis is an abnormal condition in which the kidney drops down into the pelvis when the patient stands up.

It is more common in women than men and it is asymptomatic in most patients.

It can be characterized by violent attacks of colicky flank pain, nausea, chills, hypertension, hematuria and proteinuria.

Nephropexy was performed in the past to stabilize the kidney, but presently surgery is not recommended in asymptomatic patients

nephroptosis: Nephroptosis (also called floating kidney or renal ptosis) is an abnormal condition in which the kidney drops down into the pelvis when the patient stands up and it is more common in women than in men.

Nephroptosis, also called floating kidney or renal ptosis, is an abnormal condition in which the kidney drops down into the pelvis when the patient stands up. It is more common in women than in men. It has been one of the most controversial conditions among doctors in both its diagnosis and its treatments.

It is believed to result from deficiency of supporting perirenal fasciae. The renal fascia is a layer of connective tissue encapsulating the kidneys and the suprarenal glands. The deeper layers below the renal fascia are, in order, the adipose capsule of the kidney (or perirenal fat), the renal capsule and finally the parenchyma of the renal cortex. The spaces about the kidney are typically divided into three compartments: the perinephric space and the anterior and posterior pararenal spaces.

Renal fascia: Transverse section, showing the relations of the capsule of the kidney.

Anterior attachment: Passes anterior to the kidney, renal vessels, abdominal aorta, and inferior vena cava and fuses with the anterior layer of the renal fascia of the opposite kidney.

Posterior attachment: Fuses with the psoas fascia and side of the body of the vertebrae.

Superior attachment: The anterior and posterior layers fuse at the upper pole of the kidney and then split to enclose the suprarenal gland. At the upper part of the suprarenal gland they again fuse to form the suspensory ligament of the suprarenal gland and fuse with the diaphragmatic fascia.

Inferior attachment: The layers don’t fuse. The posterior layer descends downwards and fuses with the iliac fascia. The anterior layer blends with the connective tissue of the iliac fossa.

The anterior fascia and posterior fascia fuse laterally to form the lateroconal fascia which fuses with the fascia transversalis.

Symptoms and Diagnosis

Nephroptosis is asymptomatic in most patients. However, nephroptosis can be characterized by violent attacks of colicky flank pain, nausea, chills, hypertension, hematuria, and proteinuria.

Patients with symptomatic nephroptosis often complain of sharp pains that radiate into the groin. Many patients also suggest a weighing feeling on the abdomen. Pain is typically relieved by lying down. The attack of colic pain is called Dittel’s crisis or renal paroxysm.

Diagnosis is contemplated based upon patient symptoms. Diagnosis is confirmed during intravenous urography, by obtaining erect and supine films. Nephropexy was performed in the past to stabilize the kidney, but presently surgery is not recommended in asymptomatic patients. Laparoscopic nephropexy has recently become available for selected symptomatic patients.

Polycystic Kidney Disease

Polycystic kidney disease (PKD) is a cystic genetic disorder of the kidneys.

There are two types of PKD: autosomal dominant and autosomal recessive.

PKD can damage the liver and the pancreas, and, in some rare instances, the heart and brain.

Studies show that the incidence of autosomal recessive polycystic kidney disease (ARPKD) is 1:20,000 live births, and is typically identified in the first few weeks after birth.

Autosomal dominant polycystic kidney disease (ADPKD) is the most common of all the hereditary cystic kidney diseases with an incidence of 1:1,000 to 2:1,000 live births.

autosomal recessive polycystic kidney disease: The recessive form of polycystic kidney, called ARPKD (autosomal recessive polycystic kidney disease), is less common than autosomal dominant polycystic kidney

polycystic kidney disease: Polycystic kidney disease (PKD or PCKD, also known as polycystic kidney syndrome) is a cystic genetic disorder of the kidneys.

autosomal dominant polycystic kidney disease: Autosomal dominant polycystic kidney disease ("ADPKD," "autosomal dominant PKD," or "Adult-onset PKD") is an inherited systemic disorder that predominantly affects the kidneys, but may affect other organs including the liver, pancreas, brain, and arterial blood vessels

kidney: An organ in the body that filters the blood, producing urine.

EXAMPLES

PKD treatment involves controlling symptoms and preventing complications. High blood pressure may be hard to control, but controlling it is the most important part of treatment. Treatment may include: blood pressure medicines, diuretics, and a low-salt diet

Polycystic kidney disease (PKD or PCKD, also known as polycystic kidney syndrome) is a cystic genetic disorder of the kidneys. There are two types of PKD: autosomal dominant polycystic kidney disease (ADPKD), and the less-common autosomal recessive polycystic kidney disease (ARPKD). PKD is characterized by the presence of multiple cysts (hence,"polycystic"), typically in both kidneys. The cysts are numerous and are fluid-filled, resulting in massive enlargement of the kidneys. The disease can also damage the liver, pancreas, and, in some rare cases, the heart and brain. The two major forms of polycystic kidney disease are distinguished by their patterns of inheritance. Polycystic kidney disease is one of the most common life-threatening genetic diseases, affecting an estimated 12.5 million people worldwide.

Autosomal dominant polycystic kidney disease (ADPKD) is the most common of all the hereditary cystic kidney diseases, with an incidence of 1:1,000 to 2:1,000 live births. Studies show that 10% of end-stage renal disease (ESRD) patients treated with hemodialysis in Europe and the U.S. were initially diagnosed and treated for ADPKD. ADPKD does not appear to demonstrate a preference for any particular ethnicity.

Characterization

Polycystic Kidney Disease: Polycystic kidney disease, or PKD, is a cystic genetic disorder of the kidneys

ADPKD is characterized by progressive cyst development and bilaterally enlarged kidneys with multiple cysts. There are three genetic mutations in the PKD-1, PKD-2, and PKD3 gene with similar phenotypical presentations. Gene PKD-1 is located on chromosome 16, and codes for a protein involved in regulation of cell cycle and intracellular calcium transport in epithelial cells;

it is responsible for 85% of the cases of ADPKD. PKD-2, on chromosome 4, codes for a group of voltage-linked calcium channels. PKD3 recently appeared in research papers as a postulated 3rd gene. At this time, PKD3 has not been proven. Fewer than 10% of cases of ADPKD appear in non-ADPKD families.

Cyst formation begins in utero from any point along the nephron, although fewer than 5% of nephrons are thought to be involved. As the cysts accumulate fluid, they enlarge, separate entirely from the nephron, compress the neighboring renal parenchyma, and progressively compromise renal function.

Under the function of gene defect, epithelial cells of renal tubule turn into epithelial cells of cyst wall after phenotype change and begin to have the function of secreting cyst fluid, which leads to continuous cysts enlargement.

Studies show that the incidence of autosomal recessive polycystic kidney disease (ARPKD) is 1:20,000 live births, and is typically identified in the first few weeks after birth. Unfortunately, resulting hypoplasia results in a 30% death rate in neonates with ARPKD. In ARPKD, kidneys retain their shape, but are larger than the normal anatomical range with dilated collecting ducts from the medulla to the cortex.