NNAM PRECIOUS CHINONYE

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NURSING SCIENCE

200 LEVEL

PHS212

DISCUSS THE DISEASE OF THE RENAL SYSTEM

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.

Acute renal failure

Acute renal failure occurs when renal function suddenly declines to very low levels, so that little or no urine is formed, and the substances, including even water, that the kidney normally eliminates are retained in the body.

There are two main mechanisms that can produce acute renal failure:

*When the cardiac output(the amount of blood pumped into the general circulation by the heart)is lowered by hemorrhage or by medical or surgical shock, the renal circulation is depressed to an even greater extent. This leads directly to inefficient excretion, but, more importantly still, the kidney tissue cannot withstand prolonged impairment of its blood supply and undergoes either patchy or massive necrosis (tissue death). Given time, the kidney tissue may regenerate, and it is on this hope that the treatment of acute renal failure is based.

*The form of acute renal failure that is due to a poor supply of blood (ischemia) has many causes, the most common and most important being multiple injuries, septicemia (infections invading the bloodstream), abortion with abnormal or excessive bleeding from the female genital tract, internal or external hemorrhage, loss of fluid from the body as in severe diarrhea or burns, transfusion reactions, and severe heart attacks; a special case is the transplanted kidney, which commonly goes through a phase of acute renal failure that is independent of possible rejection.

The course of acute renal failure can usefully be divided into three phases:

* onset phase

*phase of established acute renal failure

* recovery phase.

In general, but not invariably, the second of these phases is characterized by a low output of urine (oliguria) and the third by an increasing urine output (polyuria). The onset phase is dominated by general illness, in which the episode of acute renal failure arises; at this stage there may be evidence of threatened renal damage such as blood in the urine or pain in the loins.

NEPHROPTOSIS(FLOATING KIDNEY)

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.

CAUSES

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.

SYMPTOMS

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

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.

POLYCYCSTIC KIDNEY DISEASE(PKD/PCKD)

Polycystic kidney disease (PKD/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)

*autosomal recessive polycystic kidney disease (ARPKD) and the less-common.

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

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.

Complications

The major extrarenal complications of ADPKD include cerebral aneurysms, hepatic cysts, pancreatic cysts, cardiac valve disease (especially mitral valve prolapse), colonic diverticula, and aortic root dilatation.