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Dept.: Medicine and surgery

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Course: Renal Physiology

 Assignment

1. Discuss the pathophysiological process involved in renal failure.
2. With the aid of suitable diagrams, discuss the types of dialysis you know

 Answers

* 1. When discussing the pathophysiology of CKD, renal structural and physiological characteristics, as well as the principles of renal tissue injury and repair should be taken into consideration.Firstly, the rate of renal blood flow of approximately 400 ml/100g of tissue per minute is much greater than that observed in other well perfused vascular beds such as heart, liver and brain. As a consequence, renal tissue might be exposed to a significant quantity of any potentially harmful circulating agents or substances. Secondly, glomerular filtration is dependent on rather high intra- and transglomerular pressure (even under physiologic conditions), rendering the glomerular capillaries vulnerable to hemodynamic injury, in contrast to other capillary beds. In line with this, Brenner and coworkers identified glomerular hypertension and hyperfiltration as major contributors to the progression of chronic renal disease. Thirdly, glomerular filtration membrane has negatively charged molecules which serve as a barrier retarding anionic macromolecules. With disruption in this electrostatic barrier, as is the case in many forms of glomerular injury, plasma protein gains access to the glomerular filtrate. Fourthly, the sequential organization of nephron’s microvasculature (glomerular convolute and the peritubular capillary network) and the downstream position of the tubuli with respect to glomeruli, not only maintains the glomerulo-tubular balance but also facilitates the spreading of glomerular injury to tubulointerstitial compartment in disease, exposing tubular epithelial cells to abnormal ultrafiltrate. As peritubular vasculature underlies glomerular circulation, some mediators of glomerular inflammatory reaction may overflow into the peritubular circulation contributing to the interstitial inflammatory reaction frequently recorded in glomerular disease. Moreover, any decrease in preglomerular or glomerular perfusion leads to decrease in peritubular blood flow, which, depending on the degree of hypoxia, entails tubulointerstitial injury and tissue remodeling. Thus, the concept of the nephron as a functional unit applies not only to renal physiology, but also to the pathophysiology of renal diseases. In the fifth place, the glomerulus itself should also be regarded as a functional unit with each of its individual constituents, i.e. endothothelial, mesangial, visceral and parietal epithelial cells - podocytes, and their extracellular matrix representing an integral part of the normal function. Damage to one will in part affect the other through different mechanisms, direct cell-cell connections (e.g., gap junctions), soluble mediators such as chemokines, cytokines, growth factors, and changes in matrix and basement membrane composition.The main causes of renal injury are based on immunologic reactions (initiated by immune complexes or immune cells), tissue hypoxia and ischaemia, exogenic agents like drugs, endogenous substances like glucose or paraproteins and others, and genetic defects. Irrespective of the underlying cause glomerulosclerosis and tubulointerstitial fibrosis are common to CKD.

Chronic kidney disease (CKD) is initially described as diminished renal reserve or renal insufficiency, which may progress to renal failure (end-stage renal disease). 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. When renal failure is advanced (glomerular filtration rate [GFR] ≤ 15 mL/min/1.73 m2), the ability to effectively dilute or concentrate urine is lost; thus, urine osmolality is usually fixed at about 300 to 320 mOsm/kg, close to that of plasma (275 to 295 mOsm/kg), and urinary volume does not respond readily to variations in water intake.

 Creatinine and urea

 Plasma concentrations of creatinine and urea (which are highly dependent on glomerular filtration) begin a hyperbolic rise as GFR diminishes. These changes are minimal early on. When the GFR falls below 15 mL/min/1.73 m2 (normal > 90 mL/min/1.73 m2), creatinine and urea levels are high and are usually associated with systemic manifestations (uremia). Urea and creatinine are not major contributors to the uremic symptoms; they are markers for many other substances (some not yet well defined) that cause the symptoms.

 Sodium and water

 Despite a diminishing GFR, sodium and water balance is well maintained by increased fractional excretion of sodium in urine and a normal response to thirst. Thus, the plasma sodium concentration is typically normal, and hypervolemia is infrequent unless dietary intake of sodium or water is very restricted or excessive. Heart failure can occur due to sodium and water overload, particularly in patients with decreased cardiac reserve.

 Potassium

 For substances whose secretion is controlled mainly through distal nephron secretion (eg, potassium), renal adaptation usually maintains plasma levels at normal until renal failure is advanced or dietary potassium intake is excessive. Potassium-sparing diuretics, angiotensin-converting enzyme inhibitors, beta-blockers, nonsteroidal anti-inflammatory drugs, cyclosporine, tacrolimus, trimethoprim/sulfamethoxazole, pentamidine, or angiotensin II receptor blockers may raise plasma potassium levels in patients with less advanced renal failure.

 Calcium and phosphate

 Abnormalities of calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolism can occur, as can renal osteodystrophy. Decreased renal production of calcitriol (1,25(OH)2D, the active vitamin D hormone) contributes to hypocalcemia . Decreased renal excretion of phosphate results in hyperphosphatemia . Secondary hyperparathyroidism is common and can develop in renal failure before abnormalities in calcium or phosphate concentrations occur. For this reason, monitoring PTH in patients with moderate CKD, even before hyperphosphatemia occurs, has been recommended.

Renal osteodystrophy (abnormal bone mineralization resulting from hyperparathyroidism, calcitriol deficiency, elevated serum phosphate, or low or normal serum calcium) usually takes the form of increased bone turnover due to hyperparathyroid bone disease (osteitis fibrosa) but can also involve decreased bone turnover due to adynamic bone disease (with increased parathyroid suppression) or osteomalacia. Calcitriol deficiency may cause osteopenia or osteomalacia.

 pH and bicarbonate

 Moderate metabolic acidosis (plasma bicarbonate content 15 to 20 mmol/L) is characteristic. Acidosis causes muscle wasting due to protein catabolism, bone loss due to bone buffering of acid, and accelerated progression of kidney disease.

 Anemia

 Anemia is characteristic of moderate to advanced CKD (≥ stage 3). The anemia of CKD 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 (see Overview of Decreased Erythropoiesis). Other causes include deficiencies of iron, folate, and vitamin B12.

* 1. There are three different types of dialysis.

 Hemodialysis

Hemodialysis is the most common type of dialysis. This process uses an artificial kidney (hemodialyzer) to remove waste and extra fluid from the blood. The blood is removed from the body and filtered through the artificial kidney. The filtered blood is then returned to the body with the help of a dialysis machine.

To get the blood to flow to the artificial kidney, your doctor will perform surgery to create an entrance point (vascular access) into your blood vessels. The three types of entrance points are:

Arteriovenous (AV) fistula. This type connects an artery and a vein. It’s the preferred option.

AV graft. This type is a looped tube.

Vascular access catheter. This may be inserted into the large vein in your neck.

Both the AV fistula and AV graft are designed for long-term dialysis treatments. People who receive AV fistulas are healed and ready to begin hemodialysis two to three months after their surgery. People who receive AV grafts are ready in two to three weeks. Catheters are designed for short-term or temporary use.

Hemodialysis treatments usually last three to five hours and are performed three times per week. However, hemodialysis treatment can also be completed in shorter, more frequent sessions.

Most hemodialysis treatments are performed at a hospital, doctor’s office, or dialysis center. The length of treatment depends on your body size, the amount of waste in your body, and the current state of your health.

After you’ve been on hemodialysis for an extended period of time, your doctor may feel that you’re ready to give yourself dialysis treatments at home. This option is more common for people who need long-term treatment.

 Peritoneal dialysis

Peritoneal dialysis involves surgery to implant a peritoneal dialysis (PD) catheter into your abdomen. The catheter helps filter your blood through the peritoneum, a membrane in your abdomen. During treatment, a special fluid called dialysate flows into the peritoneum. The dialysate absorbs waste. Once the dialysate draws waste out of the bloodstream, it’s drained from your abdomen.

This process takes a few hours and needs to be repeated four to six times per day. However, the exchange of fluids can be performed while you’re sleeping or awake.

There are numerous different types of peritoneal dialysis. The main ones are:

Continuous ambulatory peritoneal dialysis (CAPD). In CAPD, your abdomen is filled and drained multiple times each day. This method doesn’t require a machine and must be performed while awake.

Continuous cycling peritoneal dialysis (CCPD). CCPD uses a machine to cycle the fluid in and out of your abdomen. It’s usually done at night while you sleep.

Intermittent peritoneal dialysis (IPD). This treatment is usually performed in the hospital, though it may be performed at home. It uses the same machine as CCPD, but the process takes longer.

 Continuous renal replacement therapy (CRRT)

This therapy is used primarily in the intensive care unit for people with acute kidney failure. It’s also known as hemofiltration. A machine passes the blood through tubing. A filter then removes waste products and water. The blood is returned to the body, along with replacement fluid. This procedure is performed 12 to 24 hours a day, generally every day.

Intermittent hemodiaysis removes large amounts of water and waste in a short period of time, where as, continuous renal replacement therapies remove water and waste at a slow and steady rate. CRRT is a newer mode of dialysis. It is a mode a renal replacement therapy for hemodynamically unstable, fluid overloaded, parabolic septic patients and finds its application in management of acute renal failure especially in the critical care or intensive care unit setting.

