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EARLY AND LATE REHABILITATION OF RENAL FUNCTION IN WOMEN WITH ACUTE KIDNEY INJURY

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ABSTRACT:

Acute kidney injury is a sudden decrease in kidney function over a period of several days or weeks, causing the accumulation of nitrogenous compounds in the blood (azotemia) with or without a decrease in diuresis. This is often due to inadequate renal perfusion due to severe trauma, disease, or surgery, but sometimes the cause is rapidly progressive endogenous kidney disease. Symptoms may include anorexia, nausea, and vomiting. Epileptic seizures and coma develop if left untreated. Water, electrolyte and acid-base balance disorders develop rapidly. The diagnosis is based on laboratory tests of renal function, including serum creatinine levels. Urine test results, urinary sediment microscopy, and often vocalization techniques and other examination methods (sometimes with a kidney biopsy) are necessary to determine the cause. Treatment is aimed at the cause of the disease, but also includes replenishing the water and electrolyte balance and sometimes dialysis.

KEYWORDS: crystal deposits, dominant symptoms, metabolic acidosis, gadolinium.

INTRODUCTION:

In all cases of acute renal injury (AKI), creatinine and urea levels in the blood increase within a few days, and water-salt balance disorders develop. The most serious of these disorders are hyperkalemia and hypervolemia (possibly causing pulmonary edema). Phosphate retention leads to hypophosphatemia. Hypocalcaemia develops presumably because the affected kidney no longer produces calcitriol, and also because hypophosphatemia causes calcium phosphate to settle in the tissues. Acidosis develops because hydrogen ions are not excreted. With significant uremia, coagulation disorders are observed and pericarditis can develop. Diuresis varies depending on the type and cause of AKI.

Perianal pathologies usually do not cause permanent kidney damage (and therefore are potentially reversible), unless the decrease in perfusion is severe enough for the development of tubular ischemia. Reduced perfusion of a normally functioning kidney results in increased sodium and water reabsorption resulting in oliguria (diuresis <500 ml / day) with high urine osmolality and low urinary sodium concentration.

Diseases of the glomerular apparatus lead to a decrease in the glomerular filtration rate (girl) and an increase in the permeability of glomerular capillaries for proteins and red blood cells; such diseases can be inflammatory diseases (glomerulonephritis) or diseases that develop as a result of vascular pathology-ischemia or vacuities.

At the level of the tubules, ischemia and obstruction by cellular decay products, protein or crystal deposits, cellular or interstitial edema can also develop.

Interstitial inflammation (nephritis) usually includes an immunological and allergic component. These mechanisms of damage to the tubules are complex and dependent on each other, which refutes the previously existing term "acute tubular necrosis".

Obstruction of the flow of ultra-filtrate at the level of the tubules or distal increases the pressure in the urinary space of the glomerulus, reducing girl. Obstruction also affects renal blood flow, initially increasing blood flow and pressure in the glomerular capillaries by reducing the resistance of the fetal arterioles. However, within 3-4 hours, the renal blood flow decreases and falls to the level of < 50% of the norm per day due to increased resistance of the renal vascular bed. Restoring Reno vascular resistance to normal can take up to a week, after the 24-hour obstruction is removed.

For a significant AKI to occur, obstruction at the ureteral level requires the involvement of both ureters, unless the patient has a single functioning kidney.

In the early stages of the disease, only peripheral edema and weight gain can be determined. Often, the dominant symptoms are manifestations of the underlying disease or symptoms caused by surgical complications of an operation that resulted in impaired kidney function.

The symptoms of uremia may develop later, with the accumulation of products of nitrogen metabolism. These symptoms include:
-Anorexia
-Nausea
-vomiting
-Weakness
-Myoclonic seizures
-Cramps
-Confusion
-Coma

Asterisks and hyper reflexes may be present during the examination. Chest pain (usually worse when inhaling or lying down), pericardial friction noise, and signs of pericardial tamponed can be detected in the presence of uremic pericarditis. The accumulation of fluid in the lungs can cause dyspnea and crackling noise during auscultation.

Diuresis during acute renal injury (AKI) does not make it possible to clearly distinguish between perennial, renal or post renal causes.
In acute tubular lesions, diuresis can have 3 phases:

The prodromal period usually has normal diuresis and varies in duration depending on the causes (for example, the amount of toxin absorbed, the duration and severity of hypotension).

In the oliguria period, diuresis is usually from 50 to 500 ml/day. The duration of the oliguria period is unpredictable and depends on the etiology of AKI and the time before treatment. However, many patients never develop oliguria. Patients without oliguria have lower mortality and morbidity and less need for dialysis.

Post-Liguria phase-diuresis gradually returns to normal, but serum creatinine and urea levels may remain elevated for several more days. For several days or weeks, a violation of the function of the tubules may persist, manifested by loss of sodium, polyuria (possibly massive), insensitive to the action of vasopressin, or hyper choler emic metabolic acidosis.

Acute kidney injury (AKI) should be suspected when diuresis decreases or the content of creatinine and blood urea nitrogen (AMC) increases.

According to the KDIGO Clinical Practice Guideline for Acute Kidney Injury (KDIGO Clinical Practice Guideline for Acute Kidney Injury) (1), AKI is determined if any of the following symptoms are present:
-Increase in serum creatinine ≥ 0.3 mg / dl (26.52 mmol / L) for 48 hours
-Increase in serum creatinine by ≥ 1.5 times compared to the previous 7 days
Urine volume <0.5 ml / kg / h for 6 hours

During the examination, it is necessary to determine the presence and type of AKI, as well as its cause. Blood tests usually include a general analysis (UAC), determination of urea nitrogen (AMC), creatinine, and electrolytes (including calcium and phosphates). Urine tests include determination of sodium, urea, protein and creatinine levels and sediment microscopy. Early detection and treatment increase the chances of reversing the course of kidney injury and, in some cases, prevent the progression of the need for dialysis.

A progressive daily increase in serum creatinine indicates aki. Serum creatinine levels can increase to a maximum of 2 mg / dl / day (180 micromol / l / day) depending on the amount of creatinine produced (which depends on total body weight) and the total amount of water in the body.

The level of urea nitrogen can increase by 10-20 mg / dl / day (3.6-7.1 mmol of urea/l / day), but its indicators in the blood can be uninformative, because it often increases in response to the intensification of protein catabolism after operations, injuries, corticosteroids, burns, transfusion reactions, parenteral nutrition, gastrointestinal or other internal bleeding.

Post renal causes should be sought in most cases of AKI. The volume of residual urine after urination >200 ml indicates the presence of obstruction of the bladder outlet, although weakness of the detrusor muscles or neurogenic bladder can also lead to the formation of residual urine in such quantities. if a catheter is installed, it can be left to carefully monitor diuresis in response to therapy, but the catheter is removed if the patient shows anuria (in the absence of obstruction of the bladder outlet) to reduce the risk of infection.

Then, to diagnose a more proximal obstruction, an ultrasound of the kidneys is performed. However, the sensitivity of the method for determining obstruction is only 80-85%, because the collecting system (CP) is not always expanded, especially in an acute condition, a closed ureter (for example, with retroperitoneal fibrosis or neoplasm) or with concomitant hypovolemic. If there is a serious suspicion of obstruction, a CT scan without contrast can determine the location of the obstruction and help in choosing a treatment method.

Urinary sediment can shed light on the etiology of the disease. Normal urinary sediment may be present in perennial aki and sometimes in obstructive uropathy. For the defeat of the renal tubules is characterized by the appearance in the urinary sediment of tubular cells and cylinders, as well as a large number of granular cylinders (often with brown pigmentation). The presence of eosinophils in the urine may indicate an allergic nature of tubulointerstitial nephritis, but the diagnostic accuracy of this conclusion is limited. Red blood cell cylinders and dimorphic red blood cells are a sign of glomerulonephritis or vacuities, but can occasionally occur in acute tubular necrosis.

Iodine-containing contrast agents should be avoided as much as possible. However, renal arteriography or venography is sometimes necessary if there are clinical signs of macro vascular causes of AKI. The use of MR angiography for the diagnosis of renal artery stenosis as well as bilateral arterial and venous thrombosis has increased, because MRI uses gadolinium, which presumably has a lower risk of AKI than the iodized contrast agents used in angiography and CT with contrast.

Although the need to correct the anionic difference of sodium bicarbonate in metabolic acidosis remains controversial, the need to correct the non-anionic difference in severe metabolic acidosis (pH < 7.20) is less controversial. Non-anion difference should be

treated by intravenous administration of sodium bicarbonate in the form of a slow infusion (≤ 150 mEq [or mmol] of sodium bicarbonate in 1 liter of 5% dextrose solution in water at a rate of 50-100 ml / hour). Using the delta-delta gradient calculation, metabolic acidosis with a normal anionic difference plus metabolic acidosis with a high anionic difference gives a negative delta-delta gradient; sodium bicarbonate allows serum bicarbonate levels to rise until the delta-delta gradient reaches zero. Since it is difficult to predict changes in the body's buffer systems and the rate of acid production, it is usually not recommended to calculate the amount of bicarbonate needed to achieve a complete correction. Instead, bicarbonates should be administered by continuous infusion and the anionic difference should be monitored regularly.

Probably, the levels of urea nitrogen in the blood (AMC) and creatinine in the blood are not the best reasons for starting dialysis in acute kidney injury (AKI). Patients with an asymptomatic course and with a mild general condition can postpone dialysis until the onset of symptoms, thus avoiding the need to install a central venous catheter with subsequent complications.

REFERENCES:

1) KDIGO (Kidney Disease: Improving Global Outcomes) Acute Kidney Injury Work Group, KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Inter Suppl. 2:1–138, 2012.
2) ACR Manual on Contrast Media, Version 10.3. American College of Radiology Committee on Drugs and Contrast Media. 2018.
3) United States Renal Data System, 2016 USRDS annual data report: Epidemiology of kidney disease in the United States. National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2016.