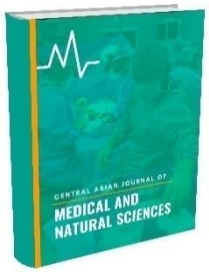
CENTRAL ASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES



Volume: 03 Issue: 04 | Jul- Aug 2022 ISSN: 2660-4159

http://cajmns.centralasianstudies.org

Study of the Prevention of Acute Renal Failture in the Human Body

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Received 5th May 2022,
Accepted 6th Jun 2022,
Online 16th July 2022

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Annotation: Acute renal failure is a sudden decrease in kidney function, due to a decrease in the parenchyma of nephrons, over several days or weeks, causing the accumulation of nitrogenous compounds in the blood with or without a decrease in diuresis. Often this is due to inadequate renal perfusion due to severe trauma, disease, or surgery, and is also caused by rapidly progressive endogenous kidney disease. Timely prevention significantly improves the condition of patients.

Key words: acute renal failure, acidosis, prophylaxis, nephrons, kidney injury.

In all cases of acute renal failure, the levels of creatinine and urea in the blood rise within a few days, and disturbances in the water-salt balance develop. The most serious of these disorders are hyperkalemia and hypervolemia, and can cause pulmonary edema. Phosphate retention leads to hyperphosphatemia. Symptoms may include anorexia, nausea, and vomiting. Epileptic seizures and coma develop if left untreated. Violations of water, electrolyte and acid-base balance develop rapidly. Diagnosis is based on laboratory testing of renal function, including serum creatinine. Urinalysis, microscopy of the urinary sediment, and often visualization and other investigations (sometimes with kidney biopsy) are needed to determine the cause. Treatment is directed at the cause of the disease, but also includes fluid and electrolyte replenishment and sometimes dialysis. It is hypothesized that hypocalcemia develops because the affected kidney no longer produces calcitriol (decreased absorption of calcium from the gastrointestinal tract) and because hyperphosphatemia causes calcium phosphate to precipitate into the tissues. Hydrogen ions are not excreted through the kidneys and cause acidosis. With significant uremia, coagulation disorders are observed and pericarditis may develop. These lesions can lead to irreversible consequences, and the kidneys will stop functioning. To prevent such consequences, it is necessary to carefully study the preventive measures to combat acute renal failure. Potentially rapidly reversible prerenal and postrenal causes of acute kidney injury should be ruled out first. Assessment of extracellular fluid volume loss and obstruction is performed in all patients. A careful history of drug use is essential, and all potentially nephrotoxic drugs should be

discontinued. Diagnostic parameters of urine are important for differentiating prerenal renal failure in acute tubular lesions, which are the most common causes of acute renal failure in hospitalized patients.

Prerenal causes often present clinically. In this case, it is necessary to try to correct the initial hemodynamic disturbances. For example, volume infusion may be tried for hypovolemia, and diuretics and afterload-reducing drugs for heart failure. A decrease in the intensity of symptoms of acute renal failure against this background confirms the presence of an extrarenal cause. Postrenal causes must be sought in most cases of AKI.

Immediately after urination, a bedside ultrasound of the bladder (or, alternatively, a urinary catheter is placed) is performed to determine the volume of residual urine in the bladder. A postvoid residual urine volume >200 ml is indicative of bladder outlet obstruction, although detrusor muscle weakness or a neurogenic bladder may also result in this amount of residual urine. If a catheter is in place, it may be left to closely monitor diuresis in response to therapy, but the catheter is removed if the patient is anuric (in the absence of bladder outlet obstruction) to reduce the risk of infection. If an obstruction is strongly suspected, non-contrast CT can locate the obstruction and help guide treatment decisions. Stopping nephrotoxic drugs and all drugs excreted by the kidneys (eg, digoxin, some antibiotics); their serum levels are also indicative. Daily water intake is limited to equal to the previous day's excretion + measured non-renal losses (eg, vomiting) + 500-1000 ml/day for insensible losses. You can limit water intake to a greater extent in case of hyponatremia or increase it in case of hypernatremia. Although weight gain indicates excess fluid intake, water intake is not reduced if serum sodium levels remain normal; instead, reduce the amount of sodium in food. Sodium and potassium intake is minimized, except in patients with initial deficiency or loss through the gastrointestinal tract. A complete diet is required, including a daily protein intake of about 0.8 g/kg. If oral or enteral nutrition is not possible, resort to parenteral nutrition; however, in AKI, the risk of hypervolemia, hyperosmolarity, and infection with intravenous nutrition is increased. Taking calcium salts (calcium carbonate, calcium acetate) or synthetic calcium-free phosphate binders before meals helps maintain serum phosphate levels < 5 mg/dL (< 1.78 mmol/L).

If necessary to maintain serum potassium at <6 mmol/L in the absence of dialysis (for example, if other treatments such as diuretics do not lower potassium levels), a cation exchange resin is given to delay the onset of action by several hours. Sodium polystyrene sulfonate is available in oral or rectal form; patiromer and sodium zirconium cyclosilicate are exclusively available for oral administration. Acute kidney failure can often be prevented by maintaining normal fluid balance, blood volume, and blood pressure in patients with trauma, burns, or severe bleeding, and in patients undergoing surgery. Infusions of isotonic saline and blood transfusions may be effective. The use of iodinated contrast agents should be minimized, especially in risk groups (eg, in the elderly and in patients with pre-existing renal insufficiency, volume depletion, diabetes, or heart failure). If contrast agents are required, the risk can be reduced by minimizing intravenous contrast volume, using non-ionic and low osmolal and isoosmolal contrast agents, avoiding non-steroidal anti-inflammatory drugs, and using pretreatment with saline at 1 ml/kg/hr IV 12 hours before before the test. Infusion of isotonic bicarbonate before or after contrast injection has also been successfully used in place of normal saline. In the past, N-acetylcysteine has been used to prevent contrast nephropathy, but recent studies have not shown improvement in disease outcomes with its use, and its use for this purpose is currently not recommended. The vascular system of the kidney is very sensitive to endothelin, a powerful vasoconstrictor that reduces renal blood flow and glomerular filtration rate. Endothelin is involved in the formation of progressive kidney damage, and endothelin receptor antagonists have been successful in reducing or even stopping kidney disease in the experiment. Anti-endothelin antibodies and endothelin receptor antagonists are being studied to protect the kidneys from ischemic AKI. In renal insufficiency, potential volume depletion and exposure to nephrotoxins should be taken into account,

diagnostic urine values obtained, and residual bladder volume measured to detect obstruction. The use of intravenous iodinated contrast agents in imaging studies should be avoided. If necessary, hemodialysis or hemofiltration should be performed for pulmonary edema, hyperkalemia, metabolic acidosis, or uremic symptoms that are not amenable to other treatments. Minimize the risk of acute renal failure in at-risk patients by maintaining a normal fluid balance, avoiding exposure to nephrotoxins (including contrast agents) when possible, and taking precautions such as administering fluids or drugs when contrast agents are needed. Substance or cytolytic therapy. Preventive measures affect the course of the disease and a favorable outcome of the disease.

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