WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 3, Issue 5, May., 2022

# NEPHROPTOSIS OR RENAL FAILURE

Azizova Shoira Kayumovna
Assistant in the Department of Propaedeutics of Internal Medicine
Samarkand State Medical University

**Annotation**
Nephroptosis is a fall of the kidneys. Congenital nephroptosis is caused by a weakening of the ligaments that hold the kidneys, while acquired nephroptosis is caused by an injury, sometimes very short-term weight loss, or by weakening of the abdominal wall muscles after pregnancy. In the first stage, when the lower part of the kidney is caught (in a healthy person, the kidney is not caught), in the second stage, the kidney is completely removed, and in the third stage, it is pushed in all directions. In the early stages of nephroptosis, the patient may feel almost nothing. Occasionally there is pain in the lower back, bladder, or bladder, especially in the kidneys, abdomen, or umbilicus.

**Keywords:** nephroptosis, kidneys, nephrogenic hypertension, pyelonephritis, glycerol, cisplatin, uranyl acetate.

## Introduction

The blood vessels in the kidneys become longer and narrower. As a result, the blood supply to the kidneys deteriorates. Now the patient begins to complain not only of increased pain, but also of repeated (even at the level of kidney stones) discomfort and discoloration of urine. Urine analysis shows an increase in protein and red blood cells. The third stage of the disease is characterized by complications such as nephrogenic hypertension (high blood pressure due to kidney disease) and bloody urine.

Treatment of nephroptosis is carried out conservatively (without surgery) and operatively. Conservative methods include bandaging, strengthening the abdominal wall, and increasing the pressure in the abdomen to restore the kidneys. The bandage should be wrapped from the lower abdomen upwards, squeezing the abdominal wall evenly, lying down in the morning, and taken off before going to bed at night. Prolonged use of the bandage leads to weakening of the abdominal wall muscles. Therapeutic exercise, morning exercise, swimming is very useful.

Pyelonephritis is an inflammation of the kidneys. Muscle cramps, tingling, sudden rise in body temperature, heavy sweating, pain in one side of the waist or on both sides of the spine, nausea, vomiting, dry mouth, muscle aches adverse events such as pyelonephritis are typical symptoms. This makes it difficult for the patient to urinate

from the kidneys. Examination of the urine reveals high levels of leukocytes and germs.

The injection to rats of glycerol, cisplatin, uranyl acetate, sodium dichromate, and mercuric chloride is followed on the third day by acute renal failure. A new approach for quantitative estimation of disturbance of excretory renal function is presented. The decrease in renal function due to uranyl acetate was 77%; sodium dichromate, 71%; mercuric chloride, 52%; cisplatin, 25%; and glycerol, 10%. The kidneys still maintained serum ion concentration close to normal values. Injection of nephrotoxic drugs increased kidney wet weight by 24-57%. This was caused by swelling of renal tissue and increases in dry weight of the kidneys. The sodium content increased in the renal cortex and decreased in the papilla. The potassium content of the renal cortex is increased. The effect of some nephrotoxic drugs is suggested to depend on an increased number of cells in the renal cortex (probably due to hemostasis and inflammation) and a decrease of renal medulla function. The above drugs induce disturbance of kidney tissue but have no effect on the ion and water content in liver and m. gastrocnemius.

It should be noted that chronic pyelonephritis can last for years without any symptoms, and it can be detected only by urinalysis. The patient may walk without seeing a doctor because of low back pain, a slight fever, and a mild headache. But chronic pyelonephritis gets worse from time to time, and of course it is necessary to take appropriate treatment. Otherwise, the urinary function of the kidneys changes, and the function of the kidney tissue gradually deteriorates. As a result, the body is poisoned with nitrogenous wastes.

Kidney stones vary in location and size. For example, urate, oxalate, and phosphate stones are formed after the formation of sediments in the urine as a result of increased acidity. Excessive excretion of calcium salts in the urine also results in the formation of stones. Strict diet is required to treat this type of disease. It is better not to eat products that contain oat acid and salts (for example, oats, spinach, beets, potatoes, figs, plums, strawberries, cocoa, chocolate, black tea). Instead, eat more magnesium-rich foods (such as peanuts, walnuts, carrots, apricots, buckwheat, barley, and cilantro). They prevent the formation of oxalate stones in the body.

Kidney disease occurs when a disease or condition impairs kidney function, causing kidney damage to worsen over several months or years. For some people, kidney damage can continue to progress even after the underlying condition is resolved.

Diseases and conditions that can lead to kidney disease include:

- Type 1 or type 2 diabetes
- High blood pressure
- Glomerulonephritis (gloe-mer-u-low-nuh-FRY-tis) — an inflammation of the kidney's filtering units (glomeruli)
- Interstitial nephritis (in-tur-STISH-ul nuh-FRY-tis), an inflammation of the kidney's tubules and surrounding structures
- Polycystic kidney disease or other inherited kidney diseases
- Prolonged obstruction of the urinary tract, from conditions such as enlarged prostate, kidney stones and some cancers
- Vesicoureteral (ves-ih-koe-yoo-REE-tur-ul) reflux, a condition that causes urine to back up into your kidneys
- Recurrent kidney infection, also called pyelonephritis (pie-uh-low-nuh-FRY-tis)

Kidney damage, once it occurs, can't be reversed. Potential complications can affect almost any part of your body and can include:

- Fluid retention, which could lead to swelling in your arms and legs, high blood pressure, or fluid in your lungs (pulmonary edema)
- A sudden rise in potassium levels in your blood (hyperkalemia), which could impair your heart's ability to function and may be life-threatening
- Heart disease
- Weak bones and an increased risk of bone fractures
- Anemia
- Decreased sex drive, erectile dysfunction or reduced fertility
- Damage to your central nervous system, which can cause difficulty concentrating, personality changes or seizures
- Decreased immune response, which makes you more vulnerable to infection
- Pericarditis, an inflammation of the saclike membrane that envelops your heart (pericardium)
- Pregnancy complications that carry risks for the mother and the developing fetus
- Malnutrition
- Irreversible damage to your kidneys (end-stage kidney disease), eventually requiring either dialysis or a kidney transplant for survival Prevention

If you have kidney disease, you may be able to slow its progress by making healthy lifestyle choices:

- Achieve and maintain a healthy weight
- Be active most days
- Limit protein and eat a balanced diet of nutritious, low-sodium foods
- Control your blood pressure

- Take your medications as prescribed
- Have your cholesterol levels checked every year
- Control your blood sugar level
- Don't smoke or use tobacco products
- Get regular checkups

Patients with renal insufficiency are a high-risk group. Patients with acute renal failure require 30-40 kcal / kg of energy and 0.8-1 g / kg of protein for ideal weight, which increases with improved glomerular filtration. Potassium should be limited to 30-50 mEq / day in the oliguric phase and sodium to 20-40 mEq / day and compensate for losses in the diuretic phase. Special recommendations have been developed for some nutrients. In patients with chronic renal failure (CDF), a diet of 0.75–1 g / kg protein per day should be recommended. Low protein diets (<0.6 g / kg / day) are not justified because malnutrition can develop (A). In patients on hemodialysis, the energy requirement is 35 Kcal / kg per day. Protein recommendations are 1.2-1.4 g / kg per day. Water recommendations depend on residual diuresis. 500-800 ml should be added to the residual diuresis. Sodium levels should be limited to 60-100 meq per day, with water and sodium levels reduced in anuric patients. Potassium should be less than 1 meq / kg per day. Patients on peritoneal dialysis had different dietary recommendations. Recommendations for protein are higher than before (1.5 g / kg / day). Energy recommendations from carbohydrates should include dialysis fluid glucose (60% of the total). Due to daily sessions of peritoneal dialysis, there are low restrictions in the diet, for example, potassium intake can reach 2000-3000 mg per day. Loss of soluble vitamins is much lower than in patients undergoing hemodialysis.

Renal function plays a role in regulating acid-base balance, water and electrolyte balance, calcium and phosphorus metabolism, nitrogen balance. Thus, acute (ARF) or chronic renal failure (CRF) especially affects the nutritional metabolic status of patients.1,2

Patients with CRF have protein-calorie malnutrition, disruption of the fat and protein section, as well as significant changes in serum proteins. Various studies have shown a link between maintaining good nutrition and reducing morbidity in these patients.3,4 Even if patients are well-nourished, they are less likely to die if they are less than 50 years old, every 6 months, and every 3 months. times should be observed. Months if over 50 years old. Protein-restricted diets have been used for decades to relieve uremic symptoms and have been shown to reduce the development of kidney failure. The development of hemodialysis and peritoneal dialysis has led to an increase

in survival rates, significantly improving the quality of life of these patients. These advances have led to the recommendation of specific requirements for nutrition as an accepted therapeutic function.5

Nutritional problems of patients with ARF and CRF will be reviewed sequentially, as well as tools used to assess them and recommendations for diet and advanced nutrition published in the literature.

In acute renal failure, a hypercatabolic state occurs, causing increased glucose consumption. If the diet does not provide an adequate supply of glucose, a phase of gluconeogenesis begins, when glycogen stores in the liver are depleted.3,6 The uptake of new glucose molecules from visceral and skeletal muscle proteins causes an unfavorable metabolic situation. On the other hand, proteolysis causes metabolic acidosis, which in turn promotes protein catabolism, which leads to an increase in muscle mass and a violation of the nutritional status of the patient. In addition, the accumulation of nitrogen-containing products in the blood causes anorexia and nausea, maintaining a catabolic state, preventing adequate intake. In summary, it is important to maintain an adequate energy supply with a good supply of carbohydrates to maintain nitrogen balance.

In patients with chronic renal failure, protein-energy malnutrition7 occurs as a result of failure of the renal function itself, causing an increase in the level of neuroendocrine factors and cytokines. This hormonal change causes hypertriglyceridemia and impaired carbohydrate metabolism with insulin resistance, which can lead to diabetes. Elevated levels of nitrogen products and electrolyte changes cause gastrointestinal disturbances that reduce intake, with nausea and vomiting. On the other hand, the treatment received by these patients also affects their nutritional status. One of the most common dietary recommendations is protein restriction in the diet, which reduces the progression of nephropathy. 5 However, this dietary change may cause uremia patients to reduce their caloric intake due to their poor adherence to dietary change requirements. 8 Dialysis can also influence nutritional status. It should not be forgotten that patients on hemodialysis have a higher protein intake than patients on peritoneal dialysis, as well as a higher risk of deficiency of water-soluble vitamins and iron.

Therefore, in order to achieve a good protein and calorie intake, as well as maintain an adequate nutritional status, appropriate assessment tools are needed. Traditionally, different parameters have been used to assess the nutritional status of these patients9 and protocols to assess the different instruments used10, and it has been concluded that the most useful instruments are those that combine parameters

related to different areas of nutritional assessment (subjective, anthropometric and biochemical parameters, etc).

The most important of these are data obtained from physical examination using anthropometric data (current weight, ideal weight, normal weight, dry weight, corrected weight without edema, skin folds, arm circumference).

The following vitamin and mineral supplements are recommended: 38 vitamin C, 30-60 mg / day; vitamin B6, 10-20 mg / day; folic acid, 1 mg per day. Vitamin B1 supplementation is optional. Not more than 60-100 mEq / day sodium. In patients with anuria on hemodialysis, it is necessary to limit the amount of water and sodium at the weekend to a minimum to limit interdialtic weight gain and prevent volume overload. As for potassium, it should not exceed 1 mEq / kg per day in patients with acidosis or no urination. Patients receiving erythropoietin should be given iron. The need for calcium is 1-1.5 g per day. The need for phosphorus is about 17 mg / kg / day. The ESPEN recommendations for this type of patient are similar: 24

1.The nutritional requirements in patients with acute illness on a regular hemodialysis program should be similar to those in patients with ARF.

2. The need for protein is 1.2-1.4 g per day (> 50% of high biological value) in stable patients on hemodialysis and 1.2-1.5 g / kg (> 50% of high biological value) in patients on peritoneal dialysis %). . For patients undergoing peritoneal dialysis with an energy supply of 35 kcal / kg per day, taking into account the glucose in the dialysis fluid (evidence B).

3. Metabolic stability of patients in need of minerals includes 800-1000 mg / day phosphate, 2000-2500 mg of potassium, 1.8-2.5 g of sodium. The need for fluid is 1000 ml per day and urination. Dialysis sessions lead to loss of vitamins, especially water-soluble vitamins, which should be supplemented as follows: folic acid, 1 mg / day; pyridoxine, 10-20 mg per day; vitamin C (30-60 mg per day). Vitamin D should be supplemented depending on the levels of calcium, phosphorus and parathyroid hormones. Loss of micronutrients with hemodialysis is minimal, but decreased patients should be given 15 mg of zinc and 50-70 mcg / day of selenium (evidence B).

4.Advanced nutritional care should be provided to malnourished patients on hemodialysis. Malnutrition is defined as a body mass index <20 kg / m2, weight loss of more than 10% in 6 months, serum albumin less than 3.5 g / l, prealbumin level less than 300 mg / l (evidence C). The following patients deserve special attention:

a) Patients on hemodialysis with acute diseases that cause catabolism and are unable to eat adequately.

b)Stable patients on hemodialysis who do not meet the recommended oral administration requirements.

c) Unconscious hemodialysis patients, e.g. with neurological disease, in nursing homes.

5. The first measure to support nutrition is oral supplements (Evidence A).
6. If diet and oral supplements are of no importance, a nasogastric tube should be used (evidence C).
7. In patients with gastroparesis who do not respond to prokinetics, the nasogastric tube should be the method of choice (evidence C).
8. Endoscopic gastrostomy or jejunostomy should be used in patients receiving long-term nutritional care (evidence C).
9. Standard formulas are recommended to support nutrition using oral supplements. However, special formulas (taking into account phosphorus and potassium requirements) are recommended for patients fed with nasogastric tube (evidence C).

Patients on peritoneal dialysis have a number of unique requirements.24,38 The protein requirement is higher, approximately 1.5 g / kg / day. Calories derived from carbohydrates, approximately 60% of the total amount, should include glucose in dialysis fluid. There are studies evaluating the effects of using amino acid-based peritoneal dialysis solutions. The results did not show a significant improvement in serum protein levels, but an improvement trend was observed.42 No differences in peritonitis incidence, hospital stay, and mortality were observed. Another important difference was that these patients ate more freely because they underwent dialysis every day. For example, the amount of potassium can be increased to 2000-3000 mg per day. The loss of water-soluble vitamins is less surprising. It is recommended to provide 10 mg of vitamin B6 and 100 mg of vitamin C per day. If patients are being treated with erythropoietin, as in all other cases, iron supplements should be given.

## References

1. Commandeur J NM, Vermeulen N PE. Molecular and biochemical mechanisms of chemically induced nephrotoxicity: a review. Chem Res Toxicol 1990; 3: 171–194
2. Renal Heterogeneity and Target Cell Toxicity, P H Bach, E A Lock. Wiley, New York 1985; 572
3. Safirstein R, Winston J, Moel D, Dikman S, Guttenplan J. Cisplatin nephrotoxicity: insight into mechanism. Int J Androl 1987; 10: 325–346
4. Walker R J, Duggin G G. Drug nephrotoxicity. Ann Rev Pharmacol Toxicol 1988; 280: 331–345
5. Ullrich K J, Rumrich G, Gemborys M W, Dekant W. Renal transport and nephrotoxicity. Nephrotoxicity: Mechanisms, Early Diagnosis, and Therapeutic Management, P H Bach, et al. Dekker, New York 1991; 1–8

6. Natochin Yu V, Reznik L V, Bakhteeva V T, Myazina E M, Brovtsyn V K. Cisplatin: nephrotoxic action in verbrates and its prevention. Compar Biochem Physiol 1989; 94C: 115–120
7. Ceriotti G, Spandrio L. A spectrophotometric method for determination of urea. Clin Chim Acta 1963; 8: 295–299
8. Bonsnes R W, Taussky H J. On the colorimetric determination of creatinine by the Jaffe reaction. J Biol Chem 1945; 158: 581–591
9. Safirstein R, Miller P, Dikman S, Lyman N, Shapiro C. Cisplatin nephrotoxicity in rats: defect papillary hypertonicity. Am J Physiol 1981; 241: F175–F185.