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CHRONIC KIDNEY DISEASE AND BLOOD SERUM BIOCHEMICAL MARKERS IN HEMODIALYSIS PATIENTS

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Background. Chronic kidney disease (CKD) is currently a significant medical and socio-economic problem in the modern society. The prevalence of CKD varies from 3.6% to 25% among adults. CKD is a progressive condition characterized by structural and functional changes to the kidney due to various causes. Irreversible glomerular and tubular damage develops during CKD pathogenesis, which can be predicted using several biochemical markers. The course and prognosis of CKD largely depend on the timely identification and elimination of factors that contribute to the acceleration of nephrosclerosis and the development of complications.

Currently, the only methods of treatment for the terminal stage of CKD are renal replacement therapy – hemodialysis (artificial kidney), peritoneal dialysis and kidney transplantations. Hemodialysis, a method of extrarenal blood purification from substances with low and medium molecular weight, is based on selective diffusion and ultrafiltration of substances through an artificial semipermeable membrane (dialyzer) [6].

Objective. Analysis of biochemical blood markers in patients with chronic renal failure before and after hemodialysis. The results allow the evaluation of the extent to which hemodialysis is able to compensate for the work of healthy kidneys.

Materials and methods. The blood serum of 56 patients (54% men and 46% women) aged 38 to 82 years diagnosed with chronic kidney disease in the terminal stage (CKD5) was studied. All patients were undergoing treatment in the nephrology and hemodialysis department of the State Institution “Minsk Scientific and Practical

Center for Surgery, Transplantology and Hematology". Hemodialysis was performed using a standard bicarbonate solution through an arteriovenous fistula. The device used was Fresenius 5008S with FX70 dialyzers. The procedures lasted 4 hours and were performed 3 times per week. Biochemical markers (creatinine, urea, uric acid, potassium, total calcium, phosphorus) were determined on the analyzer Architect c8000. The statistics of the results were processed using the program Statistica 10. Results were considered statistically significant when $p<0.05$.

Results and discussion. The dynamics of the main biochemical blood markers in patients suffering from chronic renal failure were assessed before and after hemodialysis.

Epidemiological studies suggest a relationship between CKD and hyperuricemia, with increasing evidence that elevated uric acid levels are a cause of kidney damage. Possible mechanisms for the development of CKD because of hyperuricemia include immune inflammation, both uric acid crystallization-mediated and crystal-independent inflammation. This data leads to the presentation of uric acid as a potential and modifying risk factor for kidney disease [1]. We determined that the average level of uric acid in the pre-dialysis period in patients with CKD was 453.2±31.5 μmol/l, which is 2.2 times higher than normal. After dialysis, uric acid levels decrease, but do not reach normal values. However, whether hyperuricemia is simply a result of decreased renal excretion of uric acid or contributes to the progression of kidney disease remains undetermined [4].

It was determined that in patients with CDK5 creatinine concentration before the hemodialysis procedure was 10 times the normal amount and was equal to 880.6±180.7 μмmol/l. After the hemodialysis procedure the creatinine level decreased to 234.9±47.2 μмmol/l, but exceeded the referenced value of this indicator in a healthy person by 2.1 times. It was found that in the blood serum of patients with CDK urea content increased to more than 3 times the normal amount (24.0±3.7 mmol/l). After the programmed dialysis procedure, the urea level decreased to 7.18±2.1 mmol/l, which corresponds to normal values. For many years, urea was considered to be a relatively inert, nontoxic molecule. But several studies have shown that urea is a direct and indirect uraemic toxin. Although the mechanisms of urea's direct toxicity still require further investigation, in vitro and in vivo studies have shown that uraemia modulates the smooth muscle cell's phenotype, induces the expression of pro-apoptotic family genes. Other studies have shown that urea has an indirect toxic effect via protein carbamylation, which interferes with the proteins' molecular and cellular functions and is associated with CKD progression after adjustment for conventional risk factors.

It has been shown that calcium-phosphorus metabolism is disrupted in patients with CKD. According to literature data, CKD patients with a decrease in the glomerular filtration rate (GFR) ≤60 ml/min/1.73 m^2 experience an increase in the level of phosphorus in their blood [3]. To prevent the development of hyperphosphatemia, osteocytes secrete Fibroblast Growth Factor-23, FGF-23, which binds to and activates FGF receptor type 1 (FGF-R1). FGF-R1 functions in conjunction with the transmembrane protein Klotho in the renal tubules as a receptor complex that blocks the formation of the enzyme α1-hydroxylase in the kidneys (the

formation of calcitriol decreases) and reduces the expression of sodium-phosphorus cotransporters type 2, which ensures the reabsorption of phosphorus in the proximal tubules of the kidneys. This prevents the development of hyperphosphatemia in the early stages of CKD. However, when the GFR decreases to less than 50 ml/min/1.73 m^2, this compensatory mechanism loses the ability to maintain the proper level of phosphorus in the blood, which leads to hyperphosphatemia. CKD5 is characterized by a GFR of less than 15 ml/min/1,73 m^2 [4]. We found an increase in phosphate ions in the blood serum (2.12±0.32 mmol/l) and a decrease in the level of total calcium to 1.84±0.14 mmol/l. After the hemodialysis procedure, the content of phosphate ions was restored to 1.32±0.21 mmol/l, which corresponds to normal reference values. After the hemodialysis procedure, we observed a tendency towards an increase in the blood serum calcium level (1.91±0.5 mmol/l) relative to the pre-dialysis level, however the indicator was still below normal.

It was found that patients with CKD have hyperkalemia, the concentration of potassium ions before dialysis was 5.9±0.3 mmol/l. After dialysis, the level of potassium in the blood decreased and was 3.8±0.4 mmol/l, which corresponds to the normal value.

Conclusion. In the patients with CKD5 nitrogen and mineral metabolism is disrupted, characterized by azotemia, hyperuricemia, hyperkalemia, hypocalcemia, and hyperphosphatemia, which are compensated to a certain extent after programmed hemodialysis.

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