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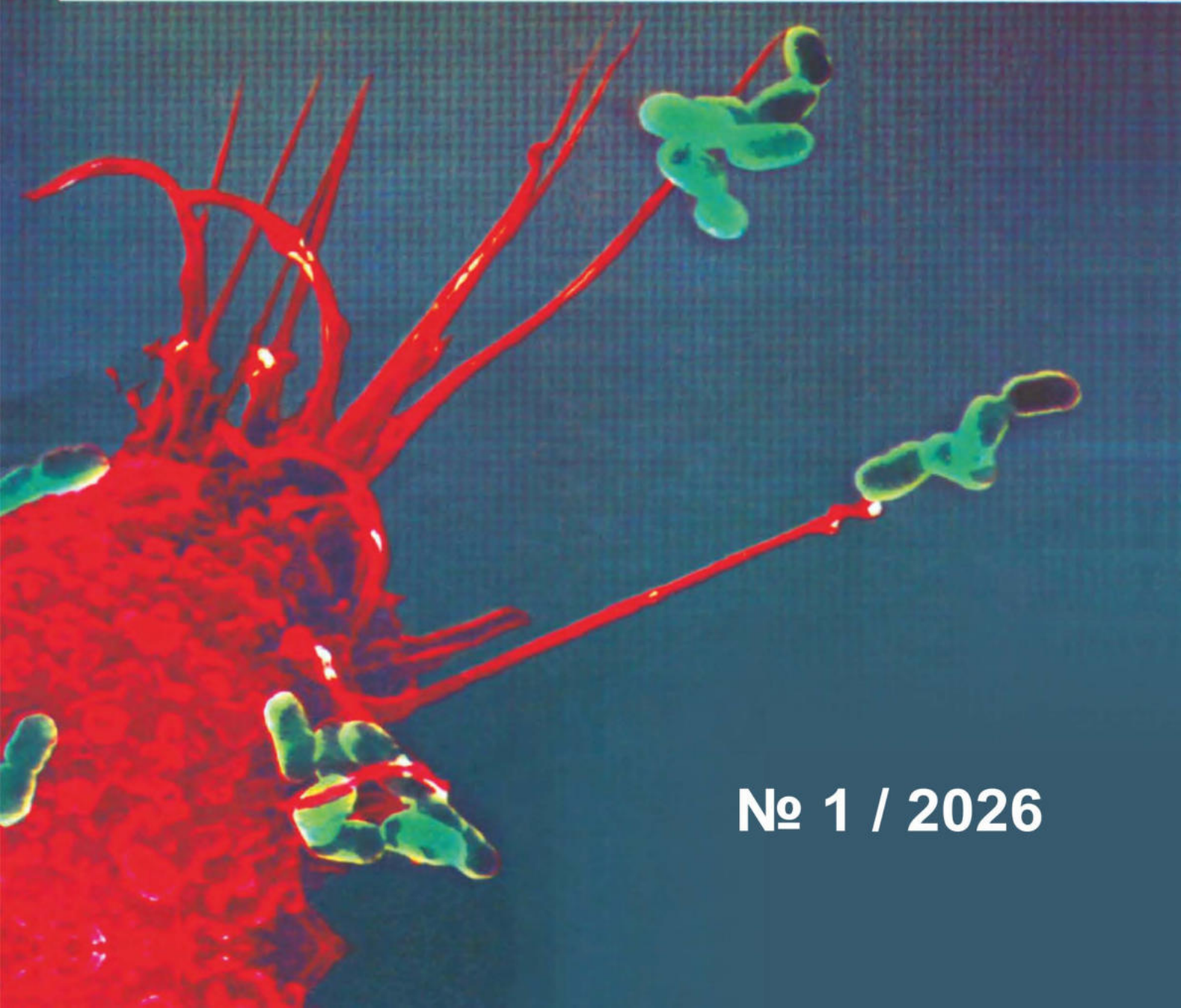
# ИНФЕКЦИЯ, ИММУНИТЕТ и ФАРМАКОЛОГИЯ



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# ИНФЕКЦИЯ, ИММУНИТЕТ и ФАРМАКОЛОГИЯ

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# BIOCHEMICAL CHANGES IN CHRONIC GLOMERULONEPHRITIS RESULTS OF LABORATORY INDICATIONS

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**Key words:** *urea, residual nitrogen, uric acid, cystatin C, protein, albumin, globulin, daily urine protein, fibrinogen.*

Chronic glomerulonephritis is a chronic immune inflammatory disease of renal glomeruli, tubules, and tissue, which appears with repeated and increasing proteinuria and hematuria over a long period of time, and gradually leads to functional impairment of the kidney[1,7]. It is common among the population and is one of the main causes of chronic kidney failure[6]. According to some data, factors such as infection and cold are of great importance in the origin of chronic glomerulonephritis, but in most cases, there is no connection between these factors and the disease[4]. After the antigen of the factor influencing the pathogenetic development of chronic glomerulonephritis gets into the blood, antibody-producing cells activate B-lymphocytes, and they, in turn, begin to produce antibodies against it. Antigens and antibodies together form immune complexes[5,9]. A part of the formed immune complexes is eliminated by neutrophils, and the second part reaches the internal organs and kidneys through the blood and settles in the kidney balls. After that, activation of complements occurs and they begin to damage the basement membrane[3,8]. In this form of glomerulonephritis, antibodies appear against the changed proteins of the kidney tissue, which in turn causes the process to become chronic[2].

**Intoduction.** Chronic glomerulonephritis is a chronic immune inflammatory disease of the renal glomeruli, tubules and tissue, manifested by long-term recurrent and increasing proteinuria and hematuria, gradually leading to impaired renal function. Symptoms characteristic of chronic glomerulonephritis: a sharp increase in proteinuria (5-10 times);a sharp increase in erythrocyturia (5-10 times);

the appearance of dysproteinemia; sharp changes in immune parameters.

**Purpose of the study.** Biochemical changes in patients with chronic glomerulonephritis and their early detection.

**Materials and research methods.** The study involved 104 patients aged 20 to 65 years. All were diagnosed with chronic glomerulonephritis. The first group (n = 40) - Anti-Pla2R positive patients with chronic glomerulonephritis membranous nephropathy. The second group (n = 11) - 11 patients with secondary nephropathy due to autoimmune connective tissue disease. The third group (n = 53) - 53 patients with chronic glomerulonephritis IgA-nephropathy. All patients were re-

cruited from the Republican Specialized Scientific and Practical Medical Center for Nephrology and Kidney Transplantation.A complete urinalysis is important in the diagnosis of chronic glomerulonephritis. It reveals varying degrees of proteinuria, micro- and macrohematuria (redness of urine or a color similar to "meat wash"), casts (hyaline, granular, erythrocytic, waxy) and epithelial cells. In addition to urine, a complete blood count, biochemical, immunological, UTI, X-ray (to exclude urological diseases) and morphological examinations are important in confirming the diagnosis.

The determination of the amount of protein in the urine was carried out by several methods. Initially, a general urinalysis was performed, which is a screening, that is, an initial examination. In this method, special reactive test strips were used, and the result was evaluated qualitatively. The amount of protein was expressed as negative, traces, one, two, three or four pluses. One plus corresponded to a protein content of about 0.3 g/l, two pluses to about 1 g/l, three pluses to about 3 g/l, and four pluses to a protein content of more than 3–5 g/l.

A complete blood count was performed using an automatic hematology analyzer. This device determined the main blood parameters, including hemoglobin content, erythrocyte count, color index, hematocrit, leukocyte count and leukocyte formula, platelet count, and erythrocyte sedimentation rate (ESR). In some cases, a blood smear was prepared and additional examination was performed under a microscope.

Urea determination was performed using enzymatic or colorimetric methods. One of the most commonly used methods is the urease enzyme method, in which urea is broken down by the enzyme and the resulting products are measured in a photometer by a color reaction.Creatinine determination was performed using a modern enzymatic method. In the Jaffe method, creatinine reacts with picric acid in an alkaline medium to form a colored compound, and the intensity of this color is measured using a photometer.

**Results and discussion.** In 134 patients diagnosed with chronic glomerulonephritis, a number of changes were found in the blood biochemical indicators, one of which is an increase in the amount of urea. According to the research results, the level of urea in group 1 was 15.9 ± 1.4*** mmol/l, in group 2 12.3 ± 0.9*** mmol/l, in group 3 12.0 ± 0.8*** mmol/l, and in the control group this indicator was only 5.2 ± 0.7 mmol/l. Accordingly, the amount of urea was 3 times higher in group 1, 2.4 times higher in group 2, and 2.3 times higher in group 3.

The amount of residual nitrogen also increases when the excretory function of the kidney decreases. In the studied patients, its level was 16.4 ± 1.2*** mmol/l in group 1, 13.3 ± 0.8*** mmol/l in group 2, 12.7 ± 1.1*** mmol/l in group 3, and only 6.7 ± 0.8 mmol/l was recorded in the control group. The results show that residual nitrogen increased by 2.4 times in group 1, and by 2 times in groups 2 and 3.

The level of uric acid in the serum is also increased with impaired renal function. 8.8 ± 0.7 mg/dl*** in group 1, 6.9 ± 0.5 mg/dl in group 2, 6.7 ± 0.6 mg/dl in group 3, and 4.2 ± 0.3 mg/dl in the control group. (Figure-1 )

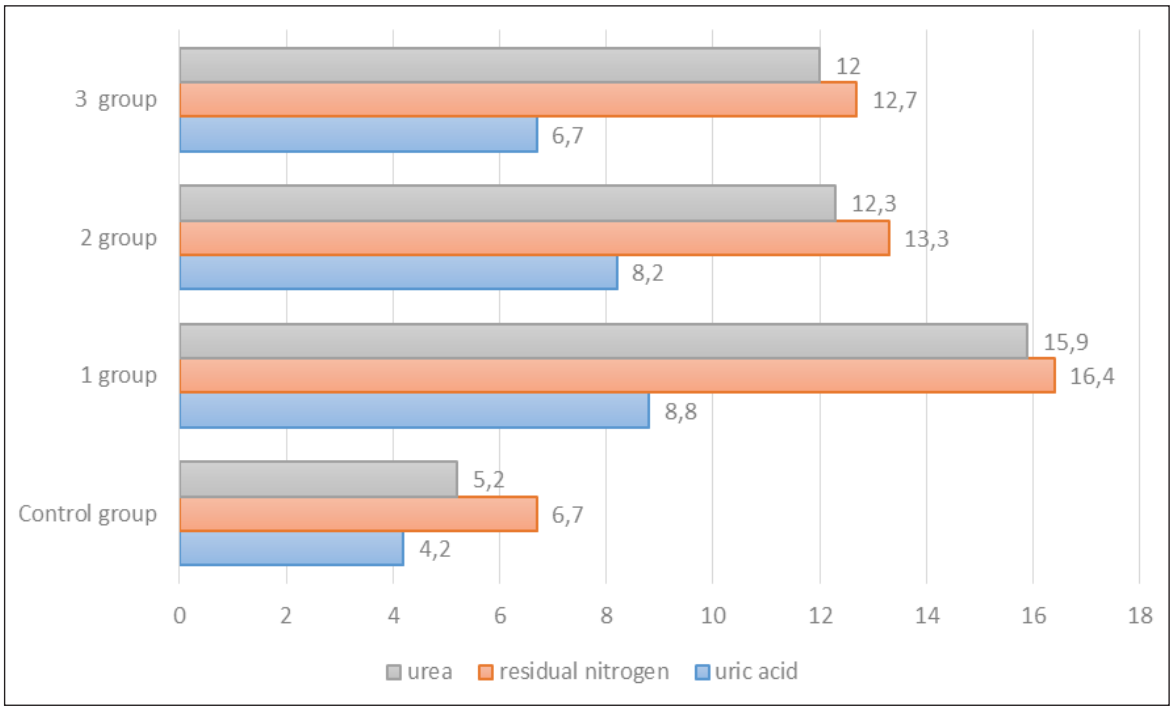


*Figure 1.* **Amount of urea, residual nitrogen and uric acid in chronic glomerulonephritis**

Cystatin C values also had a significant change, 2.2 ± 0.2 mg/l***^ in group 1, 1.6 ± 0.2 mg/l*** in group 2, 1.5 ± 0.3 mg/l*** in group 3, and 0.6 ± 0.1 mg/l in the control group. Thus, cystatin C was 3.7 times higher in group 1, 2.7 times higher in group 2, and 2.5 times higher in group 3 (Figure-2)

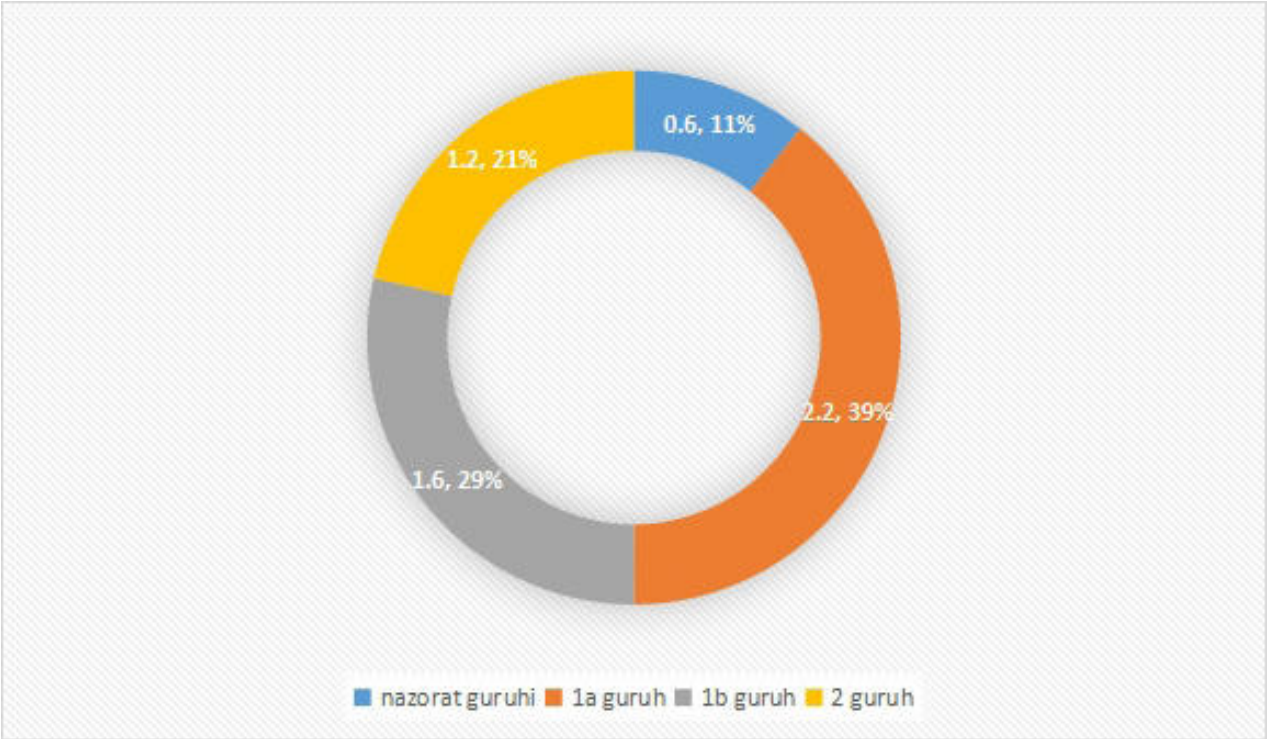


*Figure 2.* **Cystatin C levels in chronic glomerulonephritis**

In the main group of patients, the amount of total protein in serum was reliably reduced: in group 1 it was 56.2 ± 2.7 g/l**, in group 2 it was 56.9 ± 2.7 g/l**, in group 3 it was 57.9 ± 2.8 g/l***, and in the control group this indicator was 73.3 ± 4.1 g/l. According to the results of the evaluation of protein metabolism, total protein decreased by 23.3% in group 1, 22.3% in group 2, and 21.0% in group 3. Albumin levels showed a similar decrease: in group 1, albumin 16.1 ± 1.8 g/l***, globulin 40.1 ± 3.0 g/l*, in group 2 albumin 17.3 ± 1.6 g/l***, globulin 39.6 ± 2.3 g/l*, and in group 3 albumin 18.6 ± 2.2 g/l**, globulin 39.3 ± 2.2 g/l* was determined. In the control group, albumin was 40.2 ± 3.6 g/l, globulin was 33.1 ± 2.2 g/l. Albumin decreased by 2.5 times in group 1, 2.3 times in group 2, and 2.2 times in group 3, respectively, while globulin, on the contrary, increased by 21.1%, 19.6%, and 18.7% (Table 1).

*Table- 1*

**Estimation of protein content in the main groups**

| Groups | Total protein, g/l | Albumin, g/l | Globulin, g/l |
|---|---|---|---|
| **Control group, n=20** | 73,3 ± 4,1 | 40,2 ± 3,6 | 33,1 ± 2,2 |
| **1-group, n=40** | 56,2 ± 2,7** | 16,1 ± 1,8*** | 40,1 ± 3,0* |
| **2-group, n=11** | 56,9 ± 2,7** | 17,3 ± 1,6*** | 39,6 ± 2,3* |
| **3-group, n=53** | 57,9 ± 2,8*** | 18,6 ± 2,2** | 39,3 ± 2,2* |

Note: *- reliable compared to control group values ( *- $p < 0.05$, **- $p < 0.01$, ***- $p < 0.001$).

Daily urinary protein content (daily urine protein) was also significantly increased: 2.17 ± 0.16 g/day in group 1***, 1.85 ± 0.16 g/day in group 2***, 1.69 ± 0.14 g/day in group 3***, and only 0.080 ± 0.06 g/day in controls. This indicator increased by 27.3, 23.1, and 21.1 times, respectively, compared to the control group. The amount of albumin in the urine also increased sharply: in group 1 860 ± 72 mg/day***, in group 2 740 ± 61 mg/day***, in group 3 667 ± 45 mg/day***, and in the control group 28 ± 5 mg/day. was Therefore, albumin excretion is 30.7, 26.4, and 23.8 times higher, respectively (Table 2).

*Table-2*

**Daily amount of protein and albumin in urine in the main groups**

| Groups | Daily protein in urine, g/milk | Albumin, mg/milk |
|---|---|---|
| **Control group, n=20** | 0,080 ± 0,06 | 28 ± 5 |
| **1-group, n=40** | 2,17 ± 0,16*** | 860 ± 72*** |
| **2-group, n=11** | 1,85 ± 0,16*** | 740 ± 61*** |
| **3-group, n=53** | 1,69 ± 0,14*** | 667 ± 45*** |

Note: *- reliable compared to control group values ( *- $p < 0.05$, **- $p < 0.01$, ***- $p < 0.001$).

According to the analysis of daily urine protein and albumin amounts, 39.6–40.8% of protein lost through urine consisted of albumin, and the remaining 59.2–60.4% consisted of globulins. This condition resulted in hypoproteinemia and hypoalbuminemia in the blood as a result of massive proteinuria and microalbuminuria.

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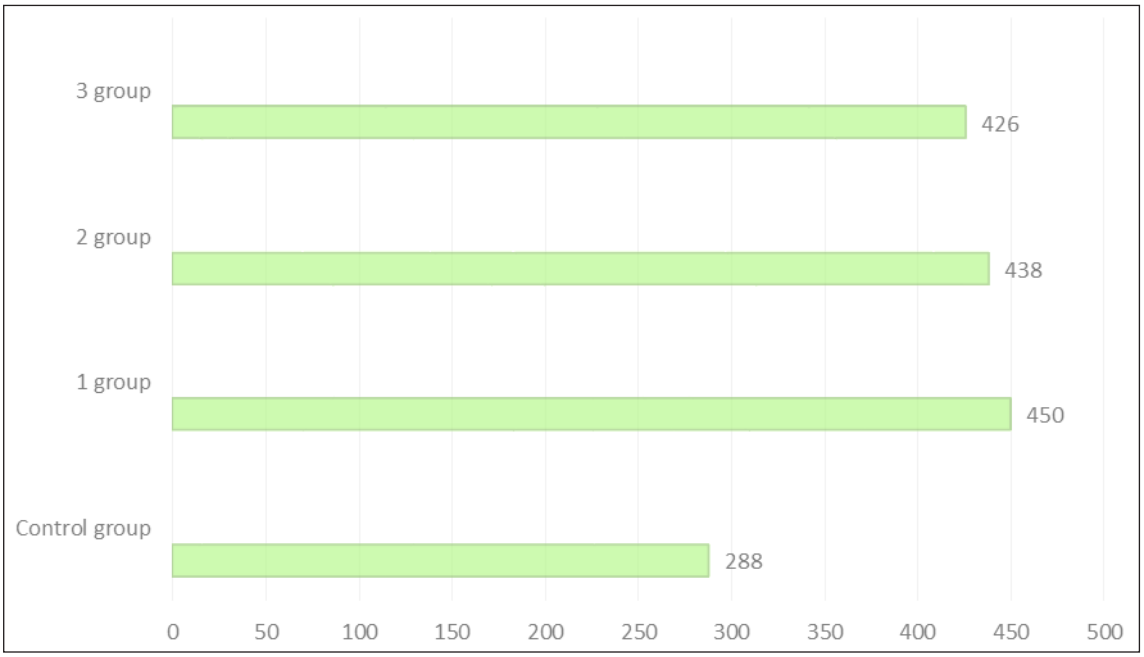


*Figure 3.* **Fibrinogen content in chronic glomerulonephritis**.

**Conclusion.** Analysis of daily urinary protein (CPOq) values in these patients showed 2.17 ± 0.16 g/day in group 1, 1.85 ± 0.16 g/day in group 2, and 1.69 ± 0.14 g/day in group 3, while in the control group this value was 0.080 ± 0.06 g/day. it happened. Compared to the control group, KPOq was found to increase by 27.3, 23.1 and 21.1 times, respectively.According to the results of the analysis of the amount of albumin in the urine, in the main groups this indicator was 860 ± 72 mg/day in group 1, 740 ± 61 mg/day in group 2 and 667 ± 45 mg/day in group 3, and in the control group it was 28 ± 5 mg/day. Compared with the control group, the level of albuminuria was 30.7 times higher in group 1, 26.4 times higher in group 2 and 23.8 times higher in group 3.The amount of fibrinogen increased on the contrary: it was 450 ± 32 mg/dl*** in group 1, 438 ± 32 mg/dl*** in group 2, 426 ± 29 mg/dl*** in group 3, and 288 ± 31 mg/dl in control. Based on this, it can be said that the level of fibrinogen is 1.6 times higher in group 1, and 1.5 times higher in patients of groups 2 and 3.

In conclusion, in chronic glomerulonephritis, protein, blood, and cylinders were detected in urine tests, and in blood tests, an increase in creatinine and urea levels and signs of anemia were observed. Such patients should be under constant and dynamic laboratory control.

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## РЕЗЮМЕ
## БИОХИМИЧЕСКИЕ ИЗМЕНЕНИЯ ПРИ ХРОНИЧЕСКОМ ГЛОМЕРУЛОНЕФРИТЕ: РЕЗУЛЬТАТЫ ЛАБОРАТОРНЫХ ИССЛЕДОВАНИЙ

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**Ключевые слова:** мочевина, остаточный азот, мочевая кислота, цистатин С, белок, альбумин, глобулин, белок в суточной моче, фибриноген.

Согласно результатам исследования, у 134 пациентов с хроническим гломерулонефритом было выявлено значительное нарушение почечной экскреторной функции. Это подтвердилось значительным повышением уровня продуктов азотного обмена – мочевины, остаточного азота, мочевой кислоты и цистатина С – в крови во всех группах пациентов по сравнению с контрольной группой. Самые высокие уровни, наблюдаемые в группе 1, указывают на более тяжелое течение почечной недостаточности.

В то же время были обнаружены значительные нарушения белкового обмена, которые характеризовались значительным снижением уровня общего белка сыворотки и альбумина, а также относительным повышением уровня глобулинов. Эти изменения были напрямую связаны со значительным увеличением суточной экскреции белка и

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