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OPTIMIZING EARLY DIAGNOSIS OF CHRONIC GLOMERULONEPHRITIS THROUGH GENETIC SCREENING

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Abstract
Chronic kidney disease occurs in 80-90% of patients with chronic glomerulonephritis. Despite advances in treatment in recent years, the risk of death remains high in patients with chronic kidney disease. Early detection and treatment of kidney damage in chronic glomerulonephritis reduces the risk of irreversible inflammation in the kidneys and is important for preserving kidney function. In this methodological recommendation, the groups of patients with nephrotic and mixed clinical forms of chronic glomerulonephritis were compared according to the results of laboratory and instrumental examination. Early detection of kidney damage by checking a number of indicators that are markers of kidney damage and early treatment improves the patient's quality of life and prolongs the patient's life expectancy.

Keywords: Chronic glomerulonephritis, chronic kidney disease.

Introduction

Early detection and treatment of kidney damage in chronic glomerulonephritis reduces the risk of irreversible inflammation in the kidneys and is important for maintaining kidney function. In addition, genetic analysis helps to identify immune dysregulation and make an accurate diagnosis of chronic glomerulonephritis. The successful implementation of these approaches requires methods for identifying individuals at the highest risk of developing CKD and determining measures for selecting patients for prescribing appropriate pathogenetic treatment. It has been established that genetic factors play an important role in the pathogenesis of the disease. Studies have shown the involvement of genes localized in the HLA system in the development of CGN. Recent genomic studies in European and American populations have shown the presence of non-HLA genetic loci and genetic polymorphisms associated with chronic glomerulonephritis.

Due to the improvement of genetic analysis methods, the probability of developing CKD in each patient with chronic glomerulonephritis can be achieved by identifying individual genetic risk factors. Three genes characteristic of chronic glomerulonephritis can directly cause kidney damage and affect pathogenetic mechanisms. As a result of studies by the General Genomic Association, many genes of risk for chronic glomerulonephritis have been identified. Some of these genes are also closely related to CGN. However, according to the results of previous studies, it does not correspond to the phenotype of CGN, and it is known that the predisposition to CGN was less pronounced. As a result of some recent studies aimed at identifying genes specifically responsible for CGN, internal genes associated with CGN, but less sensitive to chronic glomerulonephritis in

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general, have been identified. Discussions of clinical outcomes, shortcomings, and possibilities for future genetic research related to CGN are still ongoing.

CKD is observed in 80-90% of patients with chronic glomerulonephritis. Despite the successes achieved in treatment in recent years, the risk of death in patients with CKD remains high. To study the significance of genetic factors, studies were conducted to assess the genetic influence of Native Americans on European genetic ancestors in mixed populations living in South America. This is an informative way of studying the contribution of genetics to environmental control, since different individuals living in the same population and in the same place have different proportional genetic offspring. These studies support the opinion that genetic factors contribute to the pathogenesis of CKD. Although many risk genes for chronic glomerulonephritis have been identified as a result of general genomic studies, so far only a small number of genes correspond to the clinical signs of CGN. Many registered gene studies have been and are being studied as risk factors for chronic glomerulonephritis. More detailed recent studies are directly studying the phenotype of CGN.

Purpose of the study:

Optimization of early diagnosis methods based on the results of a comprehensive analysis of the influence of polymorphic gene associations on the formation of chronic glomerulonephritis.

Materials and methods:

Our research work is based on the results of laboratory and instrumental studies of 40 healthy patients with CGN and 30 healthy patients in the control group.

The study included a group of patients with the nephrotic form of CGN and the mixed form of CGN. All patients included in the study underwent laboratory and instrumental studies.

60 examined patients were mainly divided into 2 main groups based on "case-control."

Group 1. Group of patients with CGN (n=40). Male n=22 (55.0%), female n=18 (45.0%), average age 36.2 years.

2 - group. healthy patients of the control group (n=30). Male n=17 (56.7%), female n=13 (43.3%), average age 38.4.

In our scientific work, the groups of patients with CGN were studied comparatively based on the results of genetic research.

All patients underwent physical examination, laboratory tests (general blood analysis, general urine analysis, biochemical blood analysis, lipid spectrum, functional diagnostics (ECG, EchoCG, renal ultrasound and Dopplerography) and genetic studies.

Statistical processing of the research results was carried out using the SPSS 18.0 software, taking into account the types of variability and the distribution norm. The probability value (p) less than 0.05 (two-way significance test) demonstrated statistical reliability.

Analysis of Results:

Among the examined patients of the 2nd group, the functional state of the kidneys was studied based on the results of microalbuminuria (MAU) in urine, biochemical blood analysis of urea, creatinine, glomerular filtration rate (GFR) and ultrasound of the kidneys, dopplerography, as well as cholesterol, triglycerides, high and low-density lipoproteins.

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Laboratory indicators	Group 1	Group 2	P_1
Age	36.2±0.81	38.4±2.81	i/e
Duration of illness	4.49±0.34	-	i/e
MAU	264.4±1.22 mol/l*	19.0±0.6 mmol/l	<0.05
Urea	8.0±0.56 mmol/l*	4.55±0.9 mmol/l	<0.05
Creatinin	125.34±2.1 µmol/L*	63.77±1.22 µmol/L	<0.05
GFR	65.34±0.32 ml/min/1.73m2*	110.45±2.62 ml/min/1.73m2	<0.05

Note: - level of reliability ($p<0.05$). i/e-not reliable. P1 level of reliability between groups 1 and 2. According to the study results, in the 1st group, compared to the 2nd group, microalbuminuria was significantly more pronounced in the total urine analysis, respectively (264.4±1.22-19.0±0.6) ($p<0.05$). An increase in MAU in urine showed a significant ($p<0.05$) positive correlation with blood urea ($r=0.38$), creatinine ($r=0.39$), and a negative correlation with GFR ($r=-0.41$). A reliable correlation was revealed with the duration of the disease ($r=0.31$). According to the research results, the development of CGN causes the development of sclerosis in the renal vessels and worsens the functional state of the kidneys. From this, it can be concluded that an increase in urea and creatinine in the blood, i.e., a decrease in the glomerular filtration rate (GFR) as renal failure develops, is also observed.

Indicators of intrarenal hemodynamics in the selected groups

In both study groups, the functional activity of the kidneys was assessed by studying renal hemodynamics. In this case, the indicators of the initial systolic velocity of interlobular blood flow (Vmax), the final diastolic velocity (Vmin), the pulse index (PI), the systolic-diastolic index (S/D), and the resistance index, i.e., vascular resistance (RI), were studied.

According to the results, when comparing the 1st and 2nd groups, a significant ($p<0.05$) decrease in renal blood flow was observed in patients of the 1st group, i.e., in patients with CGN.

RI 0.64±0.01-0.74±0.01, PI 1.6±0.02-1.65±0.02 RI correlated positively with PI ($r=0.27$). The end-diastolic rate was significantly ($p<0.05$) negatively correlated with the systo-diastolic index and pulse index ($r=0.42$) ($r=-0.26$). Increased vascular resistance in the kidneys led to a decrease in blood flow, i.e., sclerotic changes in the renal vessels led to a decrease in the rate of glomerular filtration, i.e., GFR had a significant ($p<0.05$) negative correlation with RI ($r=-0.85$), and GFR had a significant correlation with Vmax ($r=0.33$).

Doppler ultrasonography of renal vessels	Group 1	Group 2	R1
Age	36.2±0.81	38.4±2.81	i/e
Duration of illness	4.49±0.34		i/e
Vmax	13.65±0.02 m/sec*	0.87±0.02 m/sec	<0.05
Vmin	0.22±0.01 m/sec*	0.25±0.01 m/sec	<0.05
RI	0.74±0.01	0.64±0.01	i/e
PI	1.65±0.02*	1.6±0.02	<0.05
S/D	3.65±0.02*	3.6±0.02	<0.05

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Note: - level of reliability ($p<0.05$). i/e-not reliable. P1 level of reliability between groups 1 and 2. Consequently, an increase in renal vascular resistance contributes to the development of CGN or exacerbates the existing pathology. According to the results, when comparing the 1st and 2nd groups, a significant ($p<0.05$) decrease in renal blood flow was observed in patients of the 1st group. RI 0.64±0.01-0.74±0.01, PI 1.6±0.02-1.65±0.02 RI correlated positively with PI (r=0.27). The end-diastolic rate was significantly ($p<0.05$) negatively correlated with the pulse index and systo-diastolic index (r=0.42) (r=-0.26).

Doppler ultrasonography of renal vessels	Group 1	Group 2	P1
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Vmin	0.22±0.01 m/sec*	0.25±0.01 m/sec	<0.05
RI	0.74±0.01	0.64±0.01	i/e
PI	1.65±0.02*	1.6±0.02	<0.05
S/D	3.65±0.02*	3.6±0.02	<0.05

Increased vascular resistance in the kidneys led to a decrease in blood flow, i.e., sclerotic changes in the renal vessels led to a decrease in GFR, i.e., GFR had a significant ($p<0.05$) negative correlation with RI (p=-0.85), and GFR had a significant correlation with Vmax (p=0.33).

Results of genetic testing in the selected groups

Genetic studies were conducted in 2 groups. All patients and observed patients underwent molecular genetic typing of the following candidate genes: interleukin-4 (IL4 C-589T), tumor necrosis factor (TNF G308A), and angiotensin-converting enzyme gene (ACE allele D). DNA samples isolated from venous blood by standard methods were used as material for the study.

According to the results, when comparing groups 1 and 2, the frequency of genetic variants IL4 (C-589T), TNF (G308A) and ACE (allele D) was high in patients of group 1, i.e., in patients with CGN, while the frequency of this genotype was low in the control group.

Thus, it was established that the DD marker of Interleukin-4 (IL4 C-589T), the tumor necrosis factor gene (TNF G308A), and the angiotensin-converting enzyme (ACE) gene is associated with a faster decrease in kidney function in patients with CGN.

Conclusions:

The results of studies aimed at assessing renal function in patients with chronic glomerulonephritis allowed us to predict:

An increase in urea and creatinine in the blood is observed, i.e., a decrease in the rate of glomerular filtration as renal failure develops.

Increased renal vascular resistance contributes to the development of CGN or exacerbates existing pathology. According to the results, when comparing the 1st and 2nd groups, a significant ($p<0.05$) decrease in renal blood flow was observed in patients of the 1st group.

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- Interleukin-4 (IL4 C-589T), the tumor necrosis factor gene (TNF G308A), and the DD marker of the angiotensin-converting enzyme (ACE) gene were associated with a faster decrease in kidney function in patients with CGN.

For the early diagnosis of CGN, it is recommended to include microalbuminuria, blood creatinine levels, renal Dopplerography to study intrarenal hemodynamics, and genetic testing in all patients with CGN.

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