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# WAYS TO REDUCE OVERWEIGHT AND OBESITY IN CHRONIC LIVER DISEASE

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**Objective:** to analyze the distribution of genes and genotypes of chronic kidney diseases depending on the degree of overweight and obesity.

**Material and methods:** 98 overweight and obese patients with chronic kidney diseases treated in various departments of the multidisciplinary medical center of the Bukhara region were examined. 30 patients formed the control group. 68 patients of the main group were divided into 3 subgroups depending on the body mass index and degree: 38 had degree I, 16 - II, 14 - III.

**Results:** the C/C genotype of the PPARG2 gene (rs1801282) was found in 68.7% of cases, the G/G genotype was almost never detected in overweight and obese patients with chronic kidney disease of stage III.

**Conclusions:** in case of overweight and obesity, the chronic kidney disease index is 29, that is, at the third level in most cases, the C/C genotype of the PPARG2 gene (rs1801282)_C34G is detected.

**Keywords:** kidney disease, overweight, obesity, health measures, nutrigenomics, ADRB3 gene (rs 4994), ADRB2 gene (rs1042713), PPARG2 gene (rs1801282).

Despite recent advances in the treatment of chronic diffuse kidney diseases, such cases are rare in clinical practice, for which it is impossible to prescribe etiotropic therapy or for other reasons, and at the same time it is necessary to slow down the development of the process. Traditionally, drugs belonging to the group are used for this purpose. Hepatoprotectors, which should increase the resistance of the kidneys to pathological influences, enhance their neutralizing function by activating them. Various enzyme systems (including cytochrome P450 systems and other microsomal enzymes), also contribute. Restoring various functions, thereby slowing down the development of the disease, should be selected taking into account the lack of a direct effect on the etiology of the disease. The hepatoprotective group is an effect on the main pathogenetic mechanisms of kidney diseases [3,5,6].

Kidney diseases are an important clinical, epidemiological and socio-economic problem. Among the diseases of the excretory system, chronic kidney diseases occupy an important place. In the last decade, the health care system has been experiencing an increase in the incidence of chronic kidney disease and renal failure, mainly among people of working age [2,8,11,17]. For this purpose, the group of drugs traditionally used is hepatoprotectors, which affect the pathological resistance of the kidneys, enhance their neutralization, work by

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activating various enzyme systems (including cytochrome P450 and other microsomal enzymes), and also contribute to the restoration of various functions, thereby slowing down the progression of damage. [7,13,15].

Kidney diseases are one of the most widespread groups of diseases, which are any damage to anatomical structures that do not go outside the organ. Their treatment requires drugs with various pharmacological mechanisms of action aimed at reducing pathological processes or restoring physiological processes. Normal microflora participates in the formation of the functional activity of the immune system and maintaining it in this state, but the immune system, in turn, participates in the quantitative and qualitative control of the normal microflora of the body [2,4,9,12].

However, despite numerous attempts to improve treatment outcomes and patient survival, drugs used in almost 40% of patients with severe kidney damage fail to achieve clinically significant improvement [1].

In this regard, there is a constant search for methods and means to increase the effectiveness of pathogenetic therapy of exogenous toxic kidney damage and the use of drugs with antioxidant and antihypoxant activity [10].

Despite the widespread prevalence of kidney diseases, not all pathogenetic mechanisms of the chronic course of these diseases have been sufficiently studied. One of the most widely accepted points of view is that various enzymatic activities of blood serum play an important role in this process. One of the reasons for the change in enzymatic activity is considered to be the disruption of the mechanisms of immune regulation, which is the basis for the development of chronic diffuse kidney diseases. The most important ones in immunity are involved in this process. One of the cytokines involved in fibrogenesis is interleukin-13 [4, 14,16].

**Research objective**

To study and analyze the distribution of genes and genotypes in chronic kidney disease according to the degree of overweight and obesity.

**Materials and methods**

98 patients with chronic kidney disease who were hospitalized in various departments of the Bukhara Regional Multidisciplinary Medical Center and had overweight and obesity were examined. Of the 98 patients examined, 30 were divided into the control group and 68 into the main group. Patients in the 68 main group were divided into 3 groups according to the index of overweight and obesity in chronic kidney disease. 38 patients had overweight I degree of chronic kidney disease, 16 had II degree, and 14 had III degree.

In the above patients, height, body weight, chronic kidney disease overweight and obesity index, blood cholesterol, low-density lipoprotein (LDL) and high-density lipoprotein (HDL), 3 different types of genes in the blood and their 7 different genotypes were determined and the results were analyzed.

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**Results**

In chronic kidney disease overweight and obesity stage I, 2 genotypes of the ADRB2 (rs1042713) A>G gene were also found in 19 patients (Table 1). The 2 genotypes of the ADRB3 (rs 4994) Trp 64 Arg gene were the most common genotype type, Trp / Trp - 55% of cases in 21 patients, and Trp / Arg - 45% of cases in 17 patients. The 3 genotypes of the PPARG2 (rs1801282) C34 G gene were C / G - 35% in 13 patients, of which the most common was C / C - 21 patients, 55%, and the least common was G / G genotype - 10% in 4 patients.

The frequency of occurrence of genotypes in chronic kidney disease in the I-th degree of excess body weight in %.

Table 1

| № | Gene | genotype | Number of meetings | | Average age | Male | | Woman | | Average height | Average body weight | TVI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | % | | | % | | % | | | |
| 1 | ADRB2 (rs1042713) A>G | A/A | 19 | 50 | 52.9 | 12 | 31.6 | 26 | 68.4 | 163.3 | 72.6 | 27 |
| | | A/G | 19 | 50 | 49 | 26 | 68.4 | 12 | 31.6 | 166.1 | 75.7 | 27 |
| 2 | ADRB3 (rs 4994) Trp 64 Arg | Trp/Trp | 21 | 55 | 49.3 | 6 | 15.8 | 15 | 84.2 | 165 | 74.3 | 27 |
| | | Trp/Arg | 17 | 45 | 60.3 | 11 | 84.2 | 6 | 15.8 | 163.7 | 73 | 27 |
| 3 | PPARG2 (rs1801282) C34 G | C/G | 13 | 35 | 50.4 | 10 | 26.3 | 3 | 7.9 | 168 | 76.8 | 27 |
| | | C/C | 21 | 55 | 47.7 | 5 | 13.1 | 16 | 42.1 | 163 | 72.7 | 27 |
| | | G/G | 4 | 10 | 43.5 | 2 | 5.3 | 2 | 5.3 | 162.5 | 72.5 | 27 |

In chronic kidney disease, overweight and obesity of the second degree (Table 2) The first genotype of the ADRB2 (rs1042713)A>G gene was found in 6 patients with AA-37.5% and the second genotype was found in 10 patients with AG-62.5%. The first genotype of the ADRB3 (rs 4994)_Trp 64 Arg gene was detected in 12 patients with Trp/Trp- 75%, the

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second genotype was found in 4 patients with Trp/Arg 25%. The 3 genotypes of the PPAR G2 (rs1801282)_C 34 G gene were found in 5 patients with C/G 31.3%, in 11 patients with C/C- 68.7%, and the third genotype, the most common of the 3 genotypes, G/G-, was almost not found.

The number of genotypes in excess body weight in chronic kidney disease stage II, expressed in %.

Table 2

| № | Gene | genotype | Number of meetings | % | Average age | Male | % | Woman | % | Average height | Average body weight | TVI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | ADRB2 (rs1042713) A>G | A/A | 6 | 37.5 | 40 | 3 | 18.7 | 3 | 18.7 | 169.2 | 80.6 | 28 |
| | | A/G | 10 | 62.5 | 53.7 | 4 | 25 | 6 | 37.6 | 162.5 | 75.1 | 28 |
| 2 | ADRB3 (rs 4994) Trp 64 Arg | Trp/Trp | 12 | 75 | 47.3 | 5 | 31.3 | 7 | 43.7 | 165.6 | 77.6 | 28 |
| | | Trp/Arg | 4 | 25 | 31.5 | 1 | 6.3 | 3 | 18.7 | 168.1 | 78.9 | 28 |
| 3 | PPARG2 (rs1801282) C34 G | C/G | 5 | 31.3 | 50.8 | 3 | 18.7 | 2 | 12.6 | 171 | 82.5 | 28 |
| | | C/C | 11 | 68.7 | 47.5 | 7 | 43.7 | 4 | 25 | 164.6 | 76.8 | 28 |
| | | G/G | - | - | - | - | - | - | - | - | - | 28 |

In the III degree of excess body weight of chronic kidney disease (Table 3), the first genotype of the ADRB2 (rs 1042713) A>G gene was found in 6 patients with AA-42.9% and the second genotype was found in 8 patients with AG - 57.1%. The first genotype of the ADRB3 (rs4994)_Trp64 Arg gene was the most common genotype type, Trp/Trp- 64.3% was detected in 9 patients and the second genotype was Trp/Arg-35.7% in 5 patients. The 3 genotypes of the PPARG2 (rs1801282)_C34 G gene were C/G-21.4% in 3 patients, the least of this gene was detected, C/C- 78.6% was detected in 11 patients, the most, the third genotype G/G- was not detected at all.

Table 3. Percentage of genotypes in chronic kidney disease stage III overweight.

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Table 3

| № | Gene | genotype | Number of meetings | | Average age | | Male | | Woman | | Average height | Average body weight | TVI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | % | | | % | | % | | | | |
| 1 | ADRB2 (rs1042713) A>G | A/A | 6 | 42.9 | 46.6 | 3 | 21.4 | 3 | 21.4 | 166.7 | 88.4 | 29 | |
| | | A/G | 8 | 57.1 | 47.7 | 2 | 14.3 | 4 | 28.6 | 172.5 | 84.8 | 29 | |
| 2 | ADRB3 (rs 4994) Trp 64 Arg | Trp/Trp | 9 | 64.3 | 47.6 | 3 | 21.4 | 6 | 42.9 | 175.3 | 80.9 | 29 | |
| | | Trp/Arg | 5 | 35.7 | 45 | 4 | 28.6 | 1 | 7.1 | 175.8 | 91 | 29 | |
| 3 | PPARG2 (rs1801282) C34 G | C/G | 3 | 21.4 | 38.2 | 3 | 21.4 | 2 | 14.3 | 172.6 | 79.9 | 29 | |
| | | C/C | 11 | 78.6 | 48 | 4 | 28.6 | 5 | 35.7 | 174.3 | 80.4 | 29 | |
| | | G/G | - | - | - | - | - | - | - | - | - | 29 | |

In patients with chronic kidney disease, overweight and obesity index of 27, that is, primary overweight patients, the most frequently detected genotypes were ADRB3(rs 4994)_Trp64 Arg, PPAR G2 (rs1801282)_C34 G genes, Trp/Trp-55%, C/C-55%, and 2 genotypes of ADRB2 (rs 1042713) A>G gene, AA-50% and AG-50% were detected in 2 cases. Of the 3 genotypes of PPAR G2 (rs1801282)_C34 G gene, only the genotype was C/C-55% in the most cases, while the remaining 2 genotypes were C/G-35% and G/G-10%.

In the case of chronic kidney disease, where physical education and nutrition measures were implemented according to Abu Ali ibn Sino's health measures, when the overweight and obesity index was 28, that is, the second-degree ADRB2 (rs1042713)A>G, genotype AG-62,%, PPAR G2 (rs1801282)_C34 G gene C/C-68.7%, and Trp/Trp- 75% genotype were the most common. ADRB3(rs 4994)_Trp64 Arg gene Trp/ Arg -25% was the least common of these genes, and the third genotype of PPARG2 (rs1801282)_C34 G gene G/G- was not found at all.

**Conclusions**

When the overweight and obesity index of chronic kidney disease was 29, that is, the third degree, the C/C genotype of the PPARG2 (rs1801282)_C34 G gene was found in the most frequent cases - 78.6%. The first genotype of the PPAR G2 (rs1801282)_C34 G gene, the C/G genotype, was found in the least frequent cases - 21.4%, among this gene. C/C- was found in the most frequent cases - 78.6%, the third genotype, G/G- was not detected at all.

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