Indicators of central hemodynamics in these patients are characterized by an increase in systolic and diastolic blood pressure, heart rate, total peripheral vascular resistance. In response to hemodynamic changes, structural changes in the left ventricle, including its hypertrophy, stromal fibrosis and dilatation of the cavity develop. Despite a significant number of studies confirming higher susceptibility to cardiovascular disease in patients with NAFLD, it should be noted that cardiac pathology is not diagnosed in all patients with NAFLD and in case of comorbid combination of liver and cardiovascular diseases, the natural course of cardiovascular pathology may be different in patients with similar liver injuries. Those differences can be genetically determined, which indicates the relevance of the investigation of associative links between polymorphic gene variants and the peculiarities of changes in the cardiovascular system in NAFLD patients.

The aim of this study was to investigate possible associations of deletion polymorphism of the glutathione S-transferase M1 gene with the structural and functional parameters of the heart in non-alcoholic fatty liver disease patients.

The study included 104 NAFLD patients and 45 healthy individuals (control group). First group included 52 patients without deletion of the *GSTM1*gene, second group consisted of 52 patients with deletion of the *GSTM1*gene. The average age of patients in the first group was 55,1±12,2 years, BMI – 32,8±0,8, including 24 men and 28 women. The average age of patients in the second group was 55,4±13,9 years, BMI – 33,8±0,7, including 26 men, women - 26. The healthy individuals of control group were representative by age and gender distribution to main groups. All the enrolled patients and healthy individuals signed written consent to participate in the study. All the patients and practically healthy people underwent objective examination, determination of anthropometric parameters, general and biochemical blood tests, ultrasonographic examination of the abdominal organs, elastography of the liver, echocardiographic investigation, investigation of the *GSTM1*gene deletion polymorphism.

Null genotype of *GSTM1*gene (-) among patients with NAFLD was diagnosed in 52 patients (50,0%), absence of deletion - *GSTM1* (+) was also observed in 52 persons (50,0%). In the control group, the deletion of the *GSTM1*gene was found in 23 individuals (51,1%), its absence - in 22 persons (48,9%), which did not differ significantly from the distribution of genotypes in patients with NAFLD. Thus, deletion of the *GSTM1*gene was found to occur in patients with NAFLD and healthy people. In patients with deletion variant of the *GSTM1*gene, a larger diameter of the left atrium was noted by 8,3%, p = 0,007, end diastolic size of the left ventricle by 7,9%, p = 0,02 and end systolic size by 12,5%, p = 0,02, end diastolic volume by 23,2%,p = 0,03, end systolic volume by 34,5%,p = 0,04, left ventricular myocardial mass by 16,4%,p = 0,03 as compared to the corresponding values in patients without deletion of the *GSTM1*gene. For female patients with *GSTM1* (-) a greater left ventricular myocardial mass index by 24,6%, p = 0,02 was characteristic in comparison with female patients with *GSTM* (+).

The distribution of polymorphic variants of the glutathione-S-transferase M1 gene is not significantly different in patients with non-alcoholic fatty liver disease and healthy individuals. Deletion genotype of the glutathione-S-transferase M1 gene in non-alcoholic fatty liver disease patients is associated with larger diameter of the left atrium, end systolic and diastolic sizes and volumes of the left ventricle, left ventricular myocardium mass, and in female patients also left ventricle myocardium mass index as compared to the corresponding indicators in patients without deletion of the gene functional allele.

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ARTERIAL HYPERTENSION PHENOTYPIC MANIFESTATIONS DEPENDING ON THE ANGIOTENZINOGEN GENE POLYMORPHISM (AGT 704 T>C)

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The aim of the study was to analyze the phenotypic manifestations of essential arterial hypertension (EAH) depending on the angiotensinogen gene polymorphism (AGT 704T>C).

72 subjects with EAH and target-organ damaging (2nd stage), moderate, high or very high cardiovascular risk were involved in the case-control study. Among them, 70.84% (51) females and 29.16% (21) males, mean age 59.87±7.98yo. Control group consisted of fifty practically healthy individuals with relevant age (49.13±6.28 yo) and sex distribution (62% females, 38% males). AGT (704T>C) gene polymorphism was examined by Real-time polymerase chain reaction (RT-PCR).

Hypertension is a multifactorial disease because of the interaction of many risk factors, environmental factors and genetic predicates. The most studied genetic factors are the angiotensinogen gene, angiotensin-converting enzyme (ACE) and the angiotensin II receptor gene, as well as modified factors such as obesity, increased BMI, excessive salt intake, alcohol consumption, stress and levels of high density lipids (HDL) and total cholesterol.

Genes determine approximately 20-60% of BP variability in different populations. The AGT gene is a highly polymorphic gene with more than 40 single nucleotide substitutions. It is located on chromosome 1, in the locus 1q42 - q43, in the same region as the renin gene, contains 5 exons. SNPs localized in the 2nd exon were most often studied: replacement of thymine (T) by cytosine (C) in the 704th position (T704C), which leads to the inclusion of tryptophan in the protein instead of methionine (M268T, M235T), and replacement of cytosine with thymine at position 521 (C521T) leads to the replacement of threonine with methionine (T207M, T174M). The protein encoded by this gene, pre-angiotensinogen (precursor of angiotensinogen), is expressed in the liver and broken down by the enzyme renin in response to low BP. The resulting product, angiotensin I, is then cleaved by ACE to form the physiologically active enzyme angiotensin II. Protein is involved in maintaining BP, fluid homeostasis and electrolytes in the body. AGT gene mutations lead to increasing its expression and increasing AGT blood level and are associated with hypertension and some other cardiovascular (CV) pathologies. Genotypes distribution among study group was as follows: TT-genotype - 14%, TC-genotype - 60%, CC-genotype - 26%, which corresponded to the distribution in the control group - 16%, 54% and 30%, respectively, and did not deviate from the Hardy Weinberg equilibrium. Smoking, type 2 diabetes mellitus (DM2) and obesity increased the relative risk of EAH in the examined population 2.5 times [OR=2.81; p=0.049], 3.75 times [OR=4.68; p=0.005] and almost 2 times [OR=2.90; p=0.004], respectively. The probability of EAH increases 4 times with the angiotensin II elevation in the serum. Genotypes and alleles of the AGT (704T>C) gene were not risk factors for EAH and DM2 in the examined population. However, the TC-genotype (less the T-allele) increases the obesity risk in EAH patients more than 1.5 times [OR=2.93; p=0.03]. In addition, the T-allele increases the risk for 2nd-3rd grades blood pressure (BP) elevation [OR=3.64; p<0.001].

One-way ANOVA analysis confirmed the AGT (704T>C) gene polymorphism association with systolic and diastolic BP elevation (SBP, DBP) (F=7.80; p<0.001 and F=4.90; p=0.01, respectively), especially in TT-genotype carriers (p<0.05), and with body mass index increase, but only in women (F=13.94; p<0.001).

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IT IS WORTH KNOWING WHETHER THERE IS A LACK OF VITAL ELEMENTS OF THE BODY

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A sufficient supply of food is important for the health of the cardiovascular and other systems, their proper structure and functioning. The origin of thousands of chemical reactions in the body, the formation of hormones, enzymes, cell structures, and nerve fibers are impossible without certain elements.

For example, magnesium is an element that provides the activity of more than 300 enzymes, mostly those that regulate bioenergetic processes in the body, as well as the activity of the cardiovascular system and blood fat levels. Magnesium deficiency increases the risk of cancer and contributes to the development of hypertension, urolithiasis, and children's spasms. Individuals can lose this element due to stress, intoxication, diabetes, or excessive consumption of coffee or alcohol.