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).

Rusnak I.T.

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.

Frequent consequences of magnesium deficiency in the body are irritability, sleep disturbances, heart failure, and constipation.

Zinc, for example, activates about 200 different enzymes that are responsible for a wide range of biochemical reactions of the body - cell division and maturation (wound healing, growth, and development), insulin synthesis, male hormone testosterone (zinc is needed for sexual activity and libido), inhibition inflammatory processes, neutralization of carbon dioxide and carbon monoxide. Possible consequences of zinc deficiency are frequent colds and infectious diseases, allergic reactions, dermatitis, weight loss, hair loss, loss of visual acuity, and prolonged wound healing. Zinc deficiency also can delay the sexual development of boys, sperm losing the ability to fertilize the ovum (infertility) in men, premature births and often giving birth to weakened children with weight loss in women.

Of course, by normalizing the concentration of at least one element, we can affect hundreds of reactions in the body, and if you harmonize dozens of elements, a person can recover or feel much better. It also can solve the problem of weight (for those, who needs to lose weight or gain weight), stop the process of hair loss, eliminate negative skin manifestations, and problems with internal organs. This normalizes mood, eliminates irritability, depression, restores the success of children and adults in learning and concentration, improves mental and physical development, and helps athletes achieve better results. Microelement analysis on hair or nails determines the presence of deficiencies of useful elements or excess toxic substances, helps diagnose chronic processes in the body that have developed over several months and even years. Such a diagnosis is a good screening for a person's health.

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CLINICAL AND DEMOGRAPHIC CHARACTERISTICS OF PATIENTS WITH ESSENTIAL HYPERTENSION, TAKING INTO ACCOUNT THE AGTR1 1666 A>C GENE POLYMORPHISM

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Essential hypertension (EH) is a heterogeneous disease, is a heterogeneous disease with polyetiological mechanism of development. Scientific studies have repeatedly proved that blood pressure depends on cardiovascular and environmental factors, as well as genetic markers that affect the individual risk of developing this pathology. Genetic factors in the development of EH play a critical role in the initiation of disease, therefore special importance in modern medicine is given to molecular genetic methods of analysis with the identification of polymorphic sites.

The aim of the study was to analyze the clinical and demographic parameters of patients with EAG taking into account the AGTR1 1666 A>C gene polymorphism.

The study included patients with stage II EH, 1-3 degrees of blood pressure, medium and high risk; aged 40-70 years. After screening for inclusion and exclusion criteria, 100 patients have been selected, 72 of whom have been genotyped: 51 women, 21 men, the average age of patients was 57,86 ± 1,81 years. The control group consisted of 48 healthy individuals: 30 women, 18 men, the mean age was 49,11 ± 8,62 years, who did not differ in sex and age and with the group of patients ($p>0,05$). Statistical analysis was performed using the means of free and open environment RStudio. The significance of the mean differences was assessed using the t-test Welch. The results were considered significant at $p<0,05$.

The results of the analysis of clinical and demographic indicators taking into account polymorphic variants of the AGTR1 gene showed that the gender distribution among C allele carriers in both groups was equally dominated by men 2,5 times (71% vs. 29%), among AA genotype carriers were dominated by men in both groups as well: 2,3 times more in the group of patients, 1,4 times more in the control group. Both male and female patients carrying the C allele were found 2 times more often (66,6% vs. 33,3%) compared with the control group, male patients AA genotype carriers were 1,29 times more (70% vs. 59%) than in the group of healthy individuals, while the number of female patients with AA genotype did not differ and was lower by 11%