VASCULAR ENDOTHELIAL GROWTH FACTOR A AND POLYMORPHISM OF G634C GENE IN PATIENTS WITH MYOCARDIAL INFARCTION IN ACUTE AND DISTANT PERIODS

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The Vascular endothelial growth factor A (VEGF-A) promotes endothelial cell survival in acute myocardial infarction (AMI), accelerates the development of collateral blood supply to ischemic myocardium, reduces the size of the necrotic area. The difference in the synthesis of the VEGF-A between individuals is genetically determined. The purpose of our work was to study the association of polymorphism of the VEGF-A gene (G634C) with cardiovascular risk factors, complications and the dynamics of structural and functional parameters of the left ventricle (LV) myocardium in patients with AMI with ST segment elevation (STEMI) during 6 months.

Materials and Methods. The study involved 91 patients with STEMI, 70 (76.9%) males and 21 (23.1%) females, with an average age (59.21 ± 8.92) years. Patients were hospitalized during the first three days of STEMI after coronary artery stenting. Control group - 12 practically healthy persons. The diagnosis of STEMI and treatment were established in accordance with the recommendations of the European Society of Cardiology (2017). All patients performed a 6-minute walk test, echocardiography from the 3rd to the 5th day of hospitalization and after 6 months. Investigation of the allelic polymorphism of G634C gene of the VEGF-A (rs 2010963) was carried out by real-time polymerase chain reaction. The level of the VEGF-A was determined by the enzyme-linked immunosorbent assay. The data is presented as median, interquartile scale, mean ± standard error of mean.

Results. The distribution of alleles and genotypes according to the polymorphic marker of G634C gene of the VEGF-A (rs 2010963) in STEMI patients corresponded to the Hardy-Weinberg principle. The following frequency of alleles was observed: G - 76% and C - 24%, genotypes GG, GC - 52% and 48%. Homozygotes of the GC genotype were not detected, so further analysis was carried out in two groups - GG (n = 48) and GC genotypes (n = 43). Higher concentrations of the VEGF-A were reliably determined in the acute period of the disease in the GG genotype carriers 194.10 [115.02-398.86] pg / ml compared with the GC genotype carriers 148.44 [68.84-221.28] pg / ml (p = 0.047). A significant increase of the VEGF-A is defined in the GG genotype group after 6 months from 148.44 [68.84-221.28] pg / ml to 444.18 [236.42-685.58] pg / ml (p = 0.003). There was a decrease in systolic blood pressure (p = 0.028) and diastolic blood pressure (p = 0.045) at re-examination of the GC genotype carriers and compared with the GG genotype carriers (p = 0.043) and (p = 0.021) respectively. The GC genotype group had differences in the LV end-diastolic volume (P = 0.049), the LV end-systolic volume (P = 0.045), and the LV myocardial mass (LVMM) (P = 0.04) during the acute period of the disease. However, significant differences were not detected in the size of the LV cavity after 6 months. The GC genotype group had LVMM reduction after 6 months from 249.97 ± 87.68 g to 213.96 ± 53.03 g (p = 0.039). At the same time, this indicator increased in the GG group, but not veraciously (from 214.27 ± 75.43 to 262.35 ± 77.92, p = 0.737).

Prospects for further research. We made the following conclusions: the GG genotype carriers have a significantly higher VEGF-A concentration than the GC genotype group (p = 0.047) in the acute period of STEMI. GC genotype patients with STEMI is associated with more pronounced changes in LV geometry during the acute period. The GC genotype is associated with better control of hypertension and LVMM reduction after 6 months of observation. Further investigations of the VEGF-A biological effects are perspective direction in the development of prevention and treatment of STEMI.

Key words: Vascular endothelial growth factor A, acute myocardial infarction with ST segment elevation, polymorphism of the VEGF-A gene (G634C).

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GENETIC MARKERS OF VENTRICULAR DILATION AND HYDROCEPHALUS INDUCED BY INTRAVENTRICULAR HEMORRHAGES IN PREMATURE INFANTS

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Post-hemorrhagic ventriculodilation (VD) and hydrocephalus (GC) in premature infants are the consequences of intraventricular hemorrhages (IVH). About 75-80% of premature infants with low and extremely low body weight with intraventricular hemorrhages have signs of ventriculodilation[4] or hydrocephalus[1, 5], which subsequently requires surgical correction to prevent the development of neurological deficiency. However, timely and successful ventriculoperitoneal shunting does not guarantee positive prediction and regression of neurological symptoms[2, 6]. The innovative methods for the prevention or restriction of brain damage in preterm infants require searching for factors that influence the course of severe intraventricular hemorrhages, the progression of pathology with the subsequent occurrence of post-hemorrhagic ventriculodilation, which is a key stage in the evolution of neuronal protection[3,7]. The use of non-invasive laboratory biomarkers has become the main element of clinical practice over the past decades. The studies of new biological markers, that enable early identification of newborns from the