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The role of single nucleotide polymorphism of val66met (rs6265) of the brain-derived neurotropic factor in formation of endpoints after st-segment elevation myocardial infarction

O.V. Petyunina[1], M.P. Kopytsya[1], A.E. Berezin[2], A.A. Berezin[3]

[1]L.T.Malaya Institute of Therapy, prevention and treatment of emergency conditions, Kharkiv, Ukraine; [2]State medical university, Internal Medicine, Zaporozhye, Ukraine; [3]Medical Academy of postgraduate education, Zaporozhye, Ukraine

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Background: The single nucleotide polymorphism (SNP) Val66Met (rs6265) of the brain-derived neurotrophic factor (BDNF) gene is a possible candidate that is associated with the development of psychopathology and combines it with cardiovascular events.

Purpose: To research the possible associations of single-nucleotide polymorphism of Val66Met BDNF gene with the occurrence of endpoints after 6 months of follow-up after ST segment elevation myocardial infarction (STEMI).

Methods: 256 acute STEMI patients after successful primary percutaneous coronary intervention (PCI) were enrolled in the study. TIMI III blood flow restoring through culprit artery was determined. The study of SNP of Val66Met (rs6265) of the BDNF gene was performed by real-time polymerase chain reaction. The emotional state of the patients and its relationship with stress were assessed with the questionnaire "Depression, Anxiety and Stress-21". All acute STEMI patients received adjuvant treatment due to current ESC recommendations. All procedures performed in the study involving human participants were in accordance with the ethical standards and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards and approved by the local ethics committee. Written inform consent was obtained from each patient. The primary endpoint was combined event (follow-up major adverse cardiac events – MACEs and hospitalization) that occurred within 6-month of the discharge from the hospital. MACEs were defined as the composite of CV death, recurrent angina, newly diagnosed heart failure.

Results: The frequency of genotypes Val66Met gene for BDNF in STEMI patients (n=256) was the following: 66ValVal=74.2% (n=190), 66ValMet + 66MetMet – 25.8% (n=66). The 66ValMet + 66MetMet polymorphism in the BDNF gene, stress and anxiety on 10–14 days before the event, as well as reduced left ventricular ejection fraction (LVEF), were independently associated with combined 6 months clinical end point after STEMI. Severity of depression according to depression scale was more profound in individuals with 66ValMet+66MetMet polymorphysms in BDNF gene (P=0.045) than in patients with 66ValVal genotype. Univariate and multivariate linear regressions has shown that 66ValMet+66MetMet genotype in BDNF gene, anxiety and stress before event, LVEF had independent power on dependent variable entitled combined end point after 6 month observation for STEMI patients with successful revascularization (P=0.0395). Kaplan-Meier curves demonstrated that STEMI patients with 66ValVal genotype in BDNF gene had a lower accumulation of combined end point compared with acute STEMI patients with 66ValMet+66ValMet polymorphism (Cox-criterion, P=0.019; log-rang criterion, P=0.03).

Conclusion: The Val66Met polymorphism in BDNF gene was found as an independent predictor for combined 6-month clinical end points after acute STEMI treated primary PCI.