P-61. ANGIOTENSIN-CONVERTING ENZYME GENE (ACE) AND ENDOTHELIAL NITRIC OXIDE SYNTHASE GENE (eNOS) POLYMORPHISMS IN RELATION TO HEMODYNAMIC PARAMETERS AND VASCULAR ALTERATION

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Introduction. Previous studies on RAAS and eNOS systems activation in relation to associated genes are contradictory. The latest researches prove NO production and ACE plasma level dependence from polymorphisms' types of corresponding genes.
Aim: to investigate ACE (I/D) and eNOS (T894G) genes polymorphism influence on vascular function and blood pressure (BP) in coronary artery disease (CAD) patients.
Material and methods. Study included 70 patients with CAD (27 – with acute coronary syndrome (ACS), 43 – with stable angina (SA)), 26 female, 44 male, mean age 53.7±6.9 yrs. ECG, Troponin-test, Echo-CG and biochemical analyses were performed. Systolic and diastolic BP (SBP, DBP) at baseline was assessed with 24h BP monitoring. Plasma vascular adhesive molecule (sVCAM-1) level was defined by IEA. Genes' polymorphism was assessed with PCR based method.
Results. ACE I/D gene polymorphism distribution in CAD patients was: II-genotype – in 14 (20%) patients, ID – in 36 (51.4%), DD – in 20 (28.6%); I-allele – in 32 (45.7%), D-allele – in 38 (54.3%). eNOS T894G polymorphism gene distribution was: TT-genotype – in 4 (5.7%) patients, TG – in 35 (50%) and GG – in 31 (44.3%); T allele – in 21 (30%), G allele – in 49 (70%). In ACE D-allele carriers SBP was higher than in ACE II-genotype patients (168.3±10.2 mmHg vs 147.1±5.0 mmHg, $p<0.05$) and doesn't depend on eNOS T894G gene polymorphism. The plasma sVCAM-1 concentration was significantly higher in eNOS TT-genotype carriers by 20,4% ($p<0,05$) than in G-allele carriers and doesn't depend on I/D ACE gene polymorphism. Vascular alteration was related: in ACE D-allele patients to SBP24 ($r=0.59$-0.85, $p\leq0.006$), ST segment depression ($r=0.45$, $p=0.038$); in eNOS T-allele carriers to SBP24 and DBP24 ($r=0.45$-0.79, $p\leq0.015$) and associated more often with ACS, than SA (79.1% vs 48.1%, $p<0.01$).
Conclusion. ACE D-allele presence in CAD patients influenced on BP changes. TT-genotype of eNOS gene associated with endothelial alteration.

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