PROGNOSTIC VALUE OF ENDOTHELIAL DYSFUNCTION MEASURED BY SCD40-LIGAND AND SVE-CADHERIN IN OCCURRENCE OF UNSTABLE ANGINA IN PATIENTS WITH POST-MI CARDIOSCLEROSIS AND DIABETES MELLITUS TYPE 2

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Aim: to estimate value of endothelial dysfunction in occurrence of unstable angina during one year after acute myocardial infarction (AMI) in patients with diabetes mellitus type 2 (DM2) by measurement of sVE-cadherin and sCD40-ligand.

Materials and methods: 60 patients with AMI and type 2 DM were enrolled in the study. They were divided in two groups depending on the development of unstable angina during one year after AMI: 6 patients were admitted to the hospital because of occurrence of unstable angina during one year after AMI; 54 patients did not have unstable angina during one year after AMI. sVE-cadherin blood serum levels were determined with commercial enzyme linked immunosorbent assay ELISA kit (Bender MedSystems GmbH, Vienna, Austria), sCD40L level – with the use of commercial ELISA test kit (YH Biosearch Laboratory, Shanghai, China). The data were processed statistically with Microsoft Office Excel software: the mean arithmetical value (M) and standard error of the mean (m) were calculated, for estimated probability and validity of the obtained data, Student's t-test (p) was done.

Results: assessment of endothelial-dependent mediators, namely sCD40-ligand and sVE-cadherin, presents a great interest because insufficient decreasing of sVE-cadherin level within 10 days under the influence of treatment is associated with the higher risk of unstable angina manifestation (1,70±0,03 ng/mL and 1,12±0,06 ng/mL accordingly; p=0,004).

Determination of sCD40-ligand at the first day (3,85±0,06 ng/mL and 3,84±0,03 ng/mL accordingly; p>0,05) and at the 10^{th} day (2,94±0,21 ng/mL and 3,05±0,05 ng/mL accordingly; p>0,05) did not demonstrate any significant differences.

Conclusions: It has been shown that occurrence of unstable angina is associated with insufficient reduction of sVE-cadherin – marker of endothelial integrity, that confirms a negative influence of endothelial dysfunction on delayed cardiovascular events in patients with post-MI cardiosclerosis.