Abstract: **P872**

**Activity of nonspecific systemic inflammation and biochemical markers of endothelial dysfunction as the criteria of coronary heart disease destabilization**

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Introduction: Nonspecific systemic inflammation (NSSI) as well as endothelial dysfunction (EDF) plays an important role in the development and progression of coronary heart disease (CHD). Its exacerbation can cause the atherosclerotic plaque damage and the CHD progression with development of atherothrombotic complications.

Purpose: To evaluate the activity of nonspecific systemic inflammation and biochemical markers of endothelial dysfunction and their diagnostic significance in CHD patients as the criteria of disease destabilization.

Methods: The study included 173 CHD patients (the average age was 57.24±5.12 years). 92 patients were with stable angina (45 with 2nd and 47 with 3rd functional classes) and 81 patients were with acute coronary syndromes (ACS) (43 with unstable (progressive) angina and 38 – with acute myocardial infarction). The study excluded patients with severe chronic heart failure, liver and kidney dysfunction, acute or chronic inflammatory diseases, diabetes mellitus, severe obesity, infectious diseases. The control group included 30 healthy subjects (average age was 55.37±4.82 years). Activity of NSSI was assessed by the concentration of high sensitive C-reactive protein (hsCRP), tumor necrosis factor-a (TNF-a) and pregnancy-associated plasma protein A (PAPP-A) as a marker of endogenic destruction, which were determined by ELISA. EDF was assessed by the concentration of endothelin-1 (ET-1) and soluble vascular cell adhesion molecules (sVCAM) using ELISA method.

Results: CHD patients were characterized by significant increasing of hsCRP, TNF-a, PAPP-A levels regarding to control group (5.29±0.19 and 0.87±0.04 mg/L for hsCRP, respectively, p<0.001; 4.28±0.18 and 1.18±0.07 ng/mL for TNF-a, respectively, p<0.001; 9.81±0.16 and 3.12±0.42 mIU/L for PAPP-A, respectively, p<0.01), which was the evidence of NSSI activation. Levels of both ET-1 and sVCAM-1 in CHD patients were more than twice higher than in the control group (9.89±0.28 and 4.01±0.36 ng/mL for ET-1, respectively p<0.001; 1442.9±25.3 and 626.0±34.1 ng/mL for sVCAM, respectively, p<0.001).

Levels of biochemical markers of both NSSI and EDF increased with an increase in disease severity (p<0.01) and the most severe changes were in patients with ACS, especially in patients with acute myocardial infarction. Significant relationships were between levels of both ET-1 and sVCAM with both hsCRP and TNF-a. Significant relationships were between PAPP-A level with both hsCRP and TNF-a, but were absent with both ET-1 and sVCAM levels. Therefore, we believe that the endogenic destruction of plaque is more related with activation of NSSI than with progression of EDF.

Conclusion: The severity of the CHD is associated with the degree of both activation of nonspecific systemic inflammation and dysfunction of vascular endothelium. Elevated production of these markers can be considered as the indicator of both atherosclerotic plaque damage and the possibility of ACS development.