Abstract: **P506**

**Adverse left ventricular remodeling and the serum levels of matrix metalloproteinases, biomarkers of myocardium dysfunction and inflammation in patients with acute primary anterior STEMI**

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The aim of this study was to assess the changes in serum levels of matrix metalloproteases (MMP)-2, 3, 9, ST2, NTproBNP, IL1ß, and hCRP and their impact on the adverse left ventricular remodeling (LVR) in patients with acute primary anterior myocardial infarction with ST segment elevation (STEMI).

The study included 21 patients aged 60.5±7.4 years. All of them received urgent reperfusion therapy; one third of patients received the treatment during the first 3 h. Echocardiography with 2D speckle tracking imaging was performed at day 3 (T2), 7 (T3), and 14 (T4) after STEMI and after 6 months (T5) after AMI (Vivid E9, GE Healthcare).

The concentrations of MMP-2, MMP-3, MMP-9, ST2, IL1ß, hCRP, and NTproBNP were determined at the same time point and at a day of admission (T1) by the method of quantitative enzyme linked immunosorbent assay. After that, patients were divided into 2 groups: group 1 comprised patients with the level of ST2 > 35 ng/mL; group 2 comprised patients with ST2 < 35 ng/mL at T1. The study showed that changes in the markers were multidirectional. The level of MMP-2 did not significantly change. The level of MMP-3 increased to T3 and continued to increase to T5; the changes in levels of MMP-9 were reverse over the same period. The level of IL1ß decreased to T4 though this parameter as well as the levels of ST2, NTproBNP, and hCRP exceeded the normal range during the entire observation period. The levels of ST2, NTproBNP, and hCRP were changing to T3 and significantly decreased to T5.

Marker ST2 demonstrated the best predictive value for the development of adverse LVR. ST2 level of more than 35 ng/mL at a time of admission was associated with the presence of systolic dysfunction, increased wall motion score index, increased end-systolic volume, increased 2D global longitudinal strain, and reduced ejection fraction in the early postinfarction period.