Abstract: **P733**

**Elevated levels of circulating soluble st2 at discharge predicts late ventricular adverse remodeling in patients with st-segment elevation myocardial infarction**

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Background: Late (at 4-24 month) adverse left ventricular (LV) remodeling after ST-segment elevation myocardial infarction (STEMI) is the most common cause of heart failure (HF) developing and poor prognosis. Soluble ST2 (sST2) is established biomarker of fibrosis and inflammation with known predictive value for HF death and HF admission, but its role in prognostication of adverse LV remodeling after STEMI is not fully clear. The aim of the study was to investigate whether circulating levels of sST2 predict adverse LV remodeling in STEMI patients with TIMI III flow through myocardial infarct-related coronary artery.

Methods: The study was retrospectively included 65 patients with STEMI and TIMI-III flow after primary or facilitate percutaneous coronary intervention (PCI) that were admitted to intensive care unit of our Institute between January 2016 and July 2018. Exclusion criteria were known malignancy, severe anemia, chronic obstructive lung disease, liver cirrhosis, chronic kidney disease, valvular heart disease. Primary PCI with bare-metal stent implantation was performed in 33 patients and 32 patients were previously treated with primary thrombolysis (tenecteplase, alteplase) with followed PCI during 24 hours after initial STEMI confirmation. B-mode and Tissue Doppler and Strain Echocardiography, blood sampling for biomarkers’ assay were performed at admission, at discharge from the hospital and as well as at 4 month and 6 month after STEMI.

Results: Late adverse LV remodeling was referred as increase of LV end-diastolic volume (EDV) at 6 month (first cohort, n=29), while other patients (second cohort, n=36) did not have demonstrated a trend to decrease of LV EDV or they have ever revealed reducing this parameter. There was a significant difference between both cohorts in levels of sST2 at discharge, while levels of natriuretic peptides, troponin I were similar (P=0.24). Indeed, circulating levels of sST2 in first cohort were higher when compared to second cohort (59.72 ng/mL; 95% confidence interval [CI] = 36.99 ng/mL -139.53 ng/mL versus 44.75 ng/mL; 95%CI =28.25 ng/mL -77.32 ng/mL, ?=0.039, respectively). ROC-analyses has showed that the best balanced cut-off point for sST2 for adverse remodeling at 6-month was 35 ng/mL (AUC=0.672; 95% C? 0.523-0.799; ? =0.0344; sensitivity = 46.7% and specificity = 85.7%).

Conclusions: We have shown that the levels of sST2 measured at discharge in STEMI patients with TIMI III flow after PCI could predict late adverse LV remodeling. These findings may open new approach to stratify patients with successful opening of MI-related coronary artery at risk of HF.