Abstract in English language

Background: Apoptosis plays an important role in the myocardial injury after acute myocardial infarction and in the subsequent development of heart failure.

Aim: To clarify serum kinetics of apoptotic markers TRAIL and sFas and their relation to left ventricular ejection fraction (LVEF) in patients with ST-elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (pPCI).

Methods: In 101 patients with STEMI treated with pPCI, levels of TRAIL and sFas were measured in series of serum samples obtained during hospitalization and one month after STEMI. LVEF was assessed at admission and at one-month. Major adverse cardiovascular events (MACE - i.e. death, re-MI, hospitalisation for heart failure and stroke) were analysed during a two-year follow-up.

Results: Serum level of TRAIL significantly decreased one day after pPCI (50.5pg/mL) compared to admission (56.7pg/mL), subsequently increased on day 2 after pPCI (58.8pg/mL) and reached its highest level at one month (70.3pg/mL). TRAIL levels on day 1 and 2 showed a significant inverse correlation with troponin and a significant positive correlation with LVEF at baseline. Moreover, TRAIL correlated significantly with LVEF one month after STEMI (day 1: $r=0.402$, $p<0.001$, day 2: $r=0.542$, $p<0.001$). On contrary, sFas level was significantly lowest at admission (5073pg/mL), increased one day after pPCI (6370pg/mL) and decreased on day 2 (5548pg/mL). Significantly highest sFas level was marked at one month (7024pg/mL). sFas failed to correlate with LVEF at baseline or at one month. Both TRAIL and sFas showed no ability to predict improvement of LVEF one-month after STEMI or 2-year MACE (represented by 3.29%).

Conclusion: In STEMI treated with pPCI, TRAIL reaches its lowest serum concentration after reperfusion. Low TRAIL level is associated with worse LVEF in the acute phase of STEMI as well as one month after STEMI. Higher TRAIL level appears to be beneficial and thus TRAIL seems to represent a protective mediator of post-AMI injury.