Platelet turnover in ST segment myocardial infarction: regulation, kinetics and clinical impact

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The circulating pool of platelets is heterogeneous due to versatile balance of platelet production and consumption. Newly formed platelets are reported as more active. Several studies reported increased level of immature platelets in acute coronary syndrome (ACS) associated with adverse ischemic outcomes. The mechanisms involved in the regulation of platelet turnover per se remain discussible. The impact of mandatory pharmacological platelet suppression as a cornerstone of antithrombotic therapy of ACS on platelet turnover is poorly understood.

Purpose of the study was to investigate associations between platelet turnover parameters, thrombopoietin (TPO), stromal cell derived factor 1 (SDF1) and myeloproliferative leukemia virus oncogene (MPL) in patients with ST segment elevation myocardial infarction (STEMI), furthermore to characterize key regulatory mechanisms of platelet turnover in STEMI as potential pharmacological target.

Methods. We report preliminary results of the on-going study. 40 male patients with STEMI were enrolled, and stratified into two groups according to clinical presentation and short-term risk scores. Mean platelet volume (MPV) and MPV to platelet count ratio were used as a surrogate marker of platelet turnover. TPO, SDF1 and MPL were chosen to characterize platelet turnover regulation, and were serially (0-1-7 days) measured with commercially available ELISA kits. Time delays, and rate characteristics were calculated from TPO, SDF1 and MPL time functions.

Results. Among already involved patients 37.5% were hemodynamically unstable, with elevated short-term risk of adverse outcomes, and suffered from early MI complications (group 1). The patients of the group 2 were comparatively but insignificantly (p = 0.354) younger, less likely to be a current smoker. Neither platelet count (p = 0.814), nor MPL (0.585), nor their ratio (0.865) were significantly different between the compared groups. Initial levels of TPO were significantly increased in group 1: 331.82 (207.04; 477.08) vs 209.06 (153.32; 298.91) pg/mL, p = 0.044. Within 7 days TPO levels decreased in both groups with poor and better prognosis, but the mean rate was maximal in unstable patients: 7.5 vs 0.44 pg/mL*day. We detected noticeable negative correlation between TPO, SDF1 levels and MPL (Spearman R -0.437 ... -0.586, p < 0.05) more substantial in patients with better prognosis: Spearman R -0.614 vs -0.586, p = 0.012. Longer delays in TPO, SDF1 and MPL kinetics were revealed in patients with poor prognosis.

Conclusion. In patients with STEMI, thrombopoietin and stromal cell derived factor 1 were inversely associated with platelet turnover parameters. In hemodynamically unstable patients with elevated short-term risk of adverse outcomes, kinetics of key regulators of platelet turnover were characterized by maximal rates and delays. Our findings suggest presence of an alternative regulator of platelet turnover associated with clinically severe course of STEMI.