Platelet derived microparticles in patients with ST segment elevation myocardial infarction: kinetics, platelet production and temperature effects

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Platelet derived microparticles (PDMP) are the most abundant MP in circulation and may originate from platelets, as well as from megakaryocytes. The elevated release of PDMP has been observed in various metabolic, inflammatory, thrombotic, and vascular diseases, including ST segment elevation myocardial infarction (STEMI). Despite the increasing amount of the studies in the field, the precise mechanism of PDMP release and their role in intracoronary atherothrombosis remain unclear. For instance, it had been demonstrated that local shear stress is much weaker stimulus modulating PDMP formation than heat stress.

The purpose of the study was to characterise the kinetics of PDMP in STEMI patients, and to assess possible impact of platelet production and temperature on PDMP release and characteristics.

Methods. 101 male STEMI patients and 10 practically healthy volunteers were involved in the study. PDMP, thrombopoietin (TPO), stromal cell-derived factor 1 (SDF1) and TPO receptors (MPL) were measured at admission, on the 2nd and on the 7th day since STEMI manifestation. Platelet aggregation and secretion were assessed at admittance and on the 7th day. To analyse the temperature impact on PDMP, samples with standardized concentrations were heated from 25 up to 45° C using a circulating thermostat, and measurements of refractive index (RI) were carried out using the multi-wavelength Abbe refractometer DR-M2/1550 in the wavelength range 450-1550 nm with an accuracy of ± 0.0002.

Results. PDMP were elevated in STEMI patients comparing to practically healthy volunteers (p = 0.021). Surprisingly, a monotonous increase in PDMP concentration was detected during the period of observation: from 25.80 (12.75; 39.35) ng/mL at admission up to 49.65 (20.52; 89.78) ng/mL on the 7th day (Friedman ANOVA p = 0.027). PDMP demonstrated a strong positive correlation with the previous level of TPO (R = 0.608, p < 0.05) and routine inflammation markers (CRP, body temperature). The samples’ RI positively correlated with PDMP concentration. Heating of the PDMP samples resulted in RI decrease associated with initial PDMP concentration.

Conclusion. Received results suggest possible involvement of PDMP in the healing processes in STEMI. The intensity of platelet production in STEMI patients treated with double antiplatelet therapy noticeably influence upon PDMP formation. Temperature influence upon PDMP associated with inflammation markers opens path for further research.
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