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Platelet priming in ST segment elevation myocardial infarction: impact of antiplatelet pretreatment

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Several physiologic substances were identified to potentiate platelet activation induced by primary agonists. Thrombopoietin (TPO) is not only a growth and development factor for the platelet precursor cell, but also can potentiate platelet response to ADP, collagen, thrombin, epinephrine, serotonin, and vasopressin. Nowadays an increasing number of patients may be chronically treated with aspirin alone or in combination with P2Y12 receptor inhibitors prior to acute coronary syndrome manifestation. Surprisingly, several studies identified aspirin pretreatment as an independent risk factor for adverse thrombotic events.

Purpose of the study was to evaluate the impact of antiplatelet pretreatment on platelet priming in patients with ST segment elevation myocardial infarction (STEMI).

Materials and methods. We enrolled 40 male patients admitted to ICU due to STEMI. Levels of TPO, stromal cell derived factor 1 (SCD1) and myeloproliferative leukemia virus oncogene (MPL) were measured in plasma samples obtained at admission, on the 2nd and the 7th days since STEMI manifestation with commercially available ELISA kits. Platelet functional testing was performed at admission and on the 7th day by impedance and lumiaggregometry.

Results. 33.3% of patients received antiplatelet therapy (23.3% aspirin only) before STEMI manifestation. Those patients with aspirin pretreatment were older (p = 0.049), more likely to be hypertensive. There was no difference in platelet count, PCT or MPV in pretreated and non-pretreated groups. ADP induced platelet aggregation at admission was significantly suppressed by prior aspirin treatment: 0.0 (0.0 ;4.0) vs 2.0 (1.0; 7.0) Ohm, p = 0.031.

All patients revealed moderately to significantly elevated TPO levels at admission with no differences in those with and without pretreatment: 264.82 (153.32; 337.64) vs 256.49 (161.17; 299.41) pg/mL, p = 0.869. In aspirin naive patients TPO dynamics was of U-shape with significant reduction on the 2nd day, compared to aspirin pretreated patients (p = 0.047). "Monotonous" profiles of TPO were associated with significantly higher levels of secreted ATP in ADP stimulated platelets: 0.42 (0.2; 0.8) vs 0.16 (0.0; 0.33) nmole, p = 0.03. Moderate to strong negative correlations between TPO, SCD1, MPL and collagen, but not ADP induced platelet aggregation and secretion were detected. Conclusion. Aspirin pretreatment significantly modifies thrombopoietin profiles in STEMI patients. Platelet functional heterogeneity in patients with STEMI is deepened through unequal exposure to platelet primers.