PROTECTIVE EFFECT OF ISCHEMIC PRECONDITIONING ON ISCHEMIA/REPERFUSION INDUCED MICROVASCULAR OBSTRUCTION DETERMINED BY ON-LINE MEASUREMENTS OF CORONARY PRESSURE AND BLOOD FLOW IN PIGS

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Background: We investigated the protective effect of ischemic preconditioning (P) on the maintenance of coronary patency using on-line measurements of coronary pressures and blood flow in a closed-chest reperfused acute myocardial infarction (MI) model in pigs.

Methods: Catheter-based 90-min occlusion followed by 60-min reperfusion of the left anterior descending coronary artery (LAD) was performed in anesthetized pigs (MI group). IP was applied (IP group) through 2 cycles of 5-min occlusion and 5-min reperfusion of the LAD before MI induction. Coronary patency was determined by measurements of coronary wedge pressure, collateral fractional flow reserve (FFRcoll), collateral pressure index (CPI) and absolute coronary blood flow (CBF). Inducible and constitutive nitric oxide synthase (iNOS/cNOS) activities and expressions were determined in the myocardium. Plasma levels of myeloperoxidase (MPO, index of activated leukocytes) and mean platelet volume (MPV, index of activated platelets) were measured.

Results: IP resulted in significantly lower levels of MPO (0.52±0.19 vs. 1.05±0.24 U/L, p<0.001) and MPV (9.1±0.6 vs. 9.6±1.0 fL, p=0.04), higher FFRcoll (0.17±0.05 vs. 0.04±0.05, p<0.001), CPI (0.13±0.05 vs. 0.02±0.05, p<0.001) and CBF (70.7±4.2 vs. 50.8±4.8 m/min, p<0.001) post-reperfusion as compared with the MI group. IP resulted in significantly higher cNOS activity and eNOS expression. MPO correlated significantly (p<0.05) with MPV, while significant (p<0.05) negative correlation was found between MPO and measures of coronary patency (FFRcoll, CPI and CBF) and cNOS activity. Moreover, cNOS activity correlated significantly with FFRcoll, CPI and CBF.

Conclusions: By measuring the coronary flow reserves, pressures and CBF on the table during heart catheterization, our study provides the first direct evidence of microvascular obstruction immediately after reopening of the infarct-related artery. IP attenuates the release of MPO and platelet activation, thereby contributing to the maintenance of vessel patency at microvascular level after reperfusion of the infarct-related artery.