1.06–2.52; P=0.026) and mortality (16.6% vs 6.2%; HR 2.86; 95% CI 1.43–5.73; P=0.0029). Incidence of MI was comparable in both groups (12.0% vs 9.5%; HR 1.26; 95% CI 0.66–2.38; P=0.48) as well as rates of ACS-driven revascularization (13.7% vs 11.7%; HR 1.18; 95% CI 0.65–2.11; P=0.59).
Conclusions: Long-term results of MS-PCI are more favorable than SS-PCI in treatment of patients with NSTE-ACS and MV CAD.

BEST POSTERS IN ACUTE CORONARY SYNDROME, NEW INSIGHTS

P4401
Cardioprotective effect of substance P in a porcine model of acute myocardial infarction

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Background: Substance P (SP) may attenuate ischemia-reperfusion injury by reducing inflammation and inducing stem cell mobilization.
Purpose: We assessed cardioprotective effect of SP in a porcine model of acute myocardial infarction (AMI).
Methods: AMI was induced by occlusion of the left anterior descending artery on 28 swine, randomized to SP 5 nmol/kg (group 1, n=14) and normal saline (group 2, n=14) given intravenously 5 minutes before reperfusion. Blood samples were collected at baseline, 3 days and 4 weeks. Echocardiography and myocardial perfusion single photon emission computed tomography (SPECT) were performed at 1 week and 4 weeks. Histomorphometric infarct size assessment was done at 4 weeks.
Results: Left ventricular (LV) ejection fraction (EF) (LVEF) after AMI induction was higher in group 1 than group 2 (37.9±4.6% vs. 29.4±3.2%, P=0.001) but not different at 4 weeks. No significant difference was observed in perfusion defect extent and total perfusion defect on SPECT at 1 week and 4 weeks. Pathologic infarct size (% LV) was significantly smaller in group 1 than group 2 (2.4±2.3% vs. 5.7±2.5%, P=0.020). The ratio of neutrophil to lymphocyte on day 3 and serum creatinine concentration at 4 weeks after AMI were lower in group 1.
Conclusions: In a porcine model of AMI, SP improved LVEF early post-MI and reduced infarct size.
Funding Acknowledgements: This research was supported by Bio & Medical Technology Development Program of the National Research Foundation of the Ministry of Education, Science

P4402
Factors associated with and prognostic value of admission mitochondrial biomarker levels in ST-segment elevation myocardial infarction undergoing primary percutaneous intervention

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Background: The mitochondria are important key players in cardiac function, as they supply all the necessary biological energy of the cell, and represent important mediators of both myocardial ischemic and reperfusion injuries in ST-elevation myocardial infarction (STEMI).
Purpose: We prospectively investigated the clinical factors and the potential prognosis associated with elevation of two mitochondrial biomarkers, cytochrome c and cell-free mitochondrial DNA (mtDNA), in an unselected cohort of consecutive STEMI patients undergoing primary percutaneous coronary intervention (pPCI).
Methods: Cytochrome c and mtDNA were measured at hospital admission in 424 consecutive STEMI patients undergoing pPCI. Baseline characteristics were collected, and in-hospital mortality, cardiogenic shock, and left ventricular ejection fraction (LVEF) were analyzed according to these biomarker levels.
Results: Cytochrome c was detectable in 37% of patients (mean 1.07±0.89 ng/ml) while mtDNA in 92% of patients (median 466 [169–1065] copies/μL). No correlation was found between the two biomarkers (R=-0.04; P=0.46). In multivariable adjusted analyses, only a lower LVEF was associated with greater likelihood of cytochrome c elevation (R=-0.10; P=0.05), while no correlation was found between mtDNA and LVEF (R=0.001; P=0.97). At ROC analysis, cytochrome c, but not mtDNA, accurately predicted in-hospital mortality (AUC 0.84; 95% CI 0.69–0.98; P<0.001 and 0.66; 95% CI 0.52–0.81; P=0.15, respectively) and cardiogenic shock (AUC 0.64; 95% CI 0.53–0.76; P=0.003 and 0.51; 95% CI 0.41–0.61; P=0.52, respectively). At multivariable adjusted analysis, only cytochrome c predicted in-hospital mortality and cardiogenic shock.
Conclusions: Elevation of mitochondrial markers is common among patients hospitalized with STEMI. Our findings suggest a role of cytochrome c assessment, but not of mtDNA, as an important risk-stratification tool during the initial evaluation of these patients. Future studies are needed to determine the mechanisms underlying their increment and if their levels could be used to guide targeted therapies among high-risk patients with STEMI.

P4403
Inhibitory mechanisms of very low dose rivaroxaban in non-ST-elevation myocardial infarction

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Aims: Very low dose (VLD) factor Xa (FXa) inhibition in combination with acetylsalicylic acid (ASA) and clopidogrel is associated with improved outcomes in patients with acute coronary syndrome (ACS) with a tolerable bleeding risk profile. To date there are no data documenting platelet inhibition and anticoagulatory effects of VLD FXa inhibition on top of guideline adherent dual antiplatelet therapy in ACS patients.
Methods and results: Non-ST-elevation myocardial infarction (NSTEMI) patients on oral dual antiplatelet therapy (ASA plus clopidogrel, n=20 or ASA plus ticagrelor, n=20) were prospectively enrolled in a non-randomized study. Coagulation- and platelet-dependent thrombin generation (TG), measured by means of the calibrated automated thrombogram, were significantly decreased after in vitro and in vivo addition of rivaroxaban. As shown by a total thrombus-formation analysis approach, rivaroxaban treatment led to a significant decreased coagulation-dependent (AR-chip) thrombus formation in patients treated with ASA plus P2Y12 inhibitor (clopidogrel/ticagrelor) while the pure platelet-dependent (PL-chip) thrombus formation was not affected at all. Adjunctive rivaroxaban therapy was not associated with significant differences in platelet aggregation (PA) assessed by light-transmission aggregometry (LTA). Nevertheless, according to FACS analysis, VLD rivaroxaban treatment resulted in a significant reduced expression of platelet HMGB-1, while P-selectin exposure was not affected. Furthermore, an enhanced effect of rivaroxaban on total thrombus formation and TG was observed in particular in clopidogrel non-responder patients defined as ADP-induced LTA>40%.
Conclusion: VLD rivaroxaban reduces platelet dependent thrombus formation and TG in ACS patients on dual antiplatelet therapy which can be of potential ischemic benefit.
Funding Acknowledgements: This project was supported in part by the Deutsche Forschungsgemeinschaft (DFG) and additionally funded through a restricted grant by Bayer Vital GmbH

P4404
Impaired fibrin clot lysis persists at 1 month post ACS: a PLATO substudy

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Background: We have recently shown that prolonged fibrin clot lysis at hospital discharge independently predicts cardiovascular death following acute coronary syndrome (ACS). The acute inflammatory response observed following ACS may drive some of the changes in fibrinolysis and therefore we studied fibrin clot properties 1-month post ACS and compared those to results obtained at hospital discharge.
Methods: A validated turbidimetric assay was used to determine fibrin clot lysis time (assessed as time to 50% clot lysis, secs) and clot maximum turbidity (a measure of clot density, arbitrary units (AU)) in 4,032 plasma samples collected both at hospital discharge and at 1-month post-ACS from patients in the PLATO trial. C-reactive protein (CRP) levels were available at both timepoints. Potential differences in clot properties at hospital discharge and 1 month were investigated. Change in clot parameters between discharge and 1 month was correlated to change in CRP levels between these two time points.

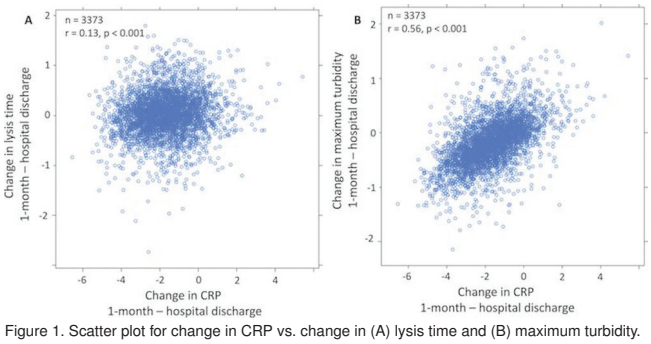

Figure 1. Scatter plot for change in CRP vs. change in (A) lysis time and (B) maximum turbidity.

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