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Abstracts

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„Zurück in die Zukunft“

Salzburg,
25. bis 28. Mai 2022

Tagungspräsident
Univ.-Prof. Dr. Bernhard Metzler

Tagungssekretär
Univ.-Prof. Dr. Daniel Scherr

of FOXM1 interaction partners (GFM, EFTUD2, RPL11, RPL10, EIF5B, EEF2) in RemoLV and Hyper groups.

Conclusion: Our study identified different molecular pathways involved in the development of distinct MF in a porcine model leading to HF. A new antifibrotic drug Thiostrepton might exhibit beneficial effect in reduction of cardiac fibrosis via ACE-regulation especially in pressure and volume overload-induced MF model.

Proteomic profiling reveals a critical role of the complement system in stent thrombosis

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Introduction: Stent thrombosis (ST) is a severe complication after primary percutaneous coronary intervention (pPCI) and associated with significant morbidity and mortality. Apart from procedure- and lesion-related parameters and patient-related factors. However, the underlying molecular and cellular mechanisms of ST are still not fully understood. We aimed to perform in-depth proteomic analysis of ST to understand its pathogenesis.

Methods: We recruited 77 patients suffering from ST after pPCI for myocardial infarction (MI). As controls, we included matched patients suffering from native vessel acute myocardial infarction (NT, $n=154$). Five cases of acute ST (within 24 h) and six cases of NT thrombi aspirated from the culprit site were subjected to shotgun proteomic analysis. Gene-set analysis was employed to screen for pathways differing between ST and NT. Soluble complement factor (C)5a was measured from both coronary culprit site plasma and femoral plasma as in-patient control. All-cause mortality was assessed using Kaplan-Meier, ROC analysis and multivariable Cox regression.

Results: 9 patients presented with acute ST (<24 h, 11.7 %), 18 patients with subacute ST (24 h to 30 days, 23.4 %), 11 patients with late ST (30 days to 1 year, 14.3 %) and 39 patients with very late ST (>1 year, 50.6 %). ST was associated with increased all-cause mortality compared to NT (mean survival 129 vs. 109 months, log-rank $p=0.032$). Using proteomics, we identified a total of 2438 proteins to be expressed in both ST and NT thrombi. Gene set analysis revealed the complement system to be highly active in acute ST compared to NT. Specifically, we found factors of both the classical (complement factor [C]1q, C1s) and alternative pathway (complement factor B) to be increased in ST, along with higher levels of C2, C3, C4a, C4b, C5, C8a and C9. Employing ELISA, we found C5a levels to be increased at the culprit site of ST, but not of NT, patients. ROC analysis yielded a culprit site C5a level of 14,604.59 ng/ml to predict all-cause mortality (ROC AUC 0.76 [0.62, 0.90], $p<0.0001$; sensitivity 72.2 %, specificity 74.3 %). Using this cut-off, C5a levels independently all-cause mortality in ST (adjusted HR 4.102, 95 % CI 1.293–13.009, $p=0.017$) but not in NT patients (adjusted HR 0.645, 95 % CI 0.256–1.622, $p=0.351$).

Conclusion: This hypothesis-generating study highlights a crucial role of the complement system in the pathogenesis of acute ST. Further studies are required to validate these findings in a larger cohort.

Featured Poster Session: Beste Poster 1:

Impact of myocardial injury after coronary artery bypass grafting on long-term prognosis

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Introduction: The most appropriate definition of perioperative myocardial infarction (pMI) after coronary artery bypass grafting (CABG) and its impact on clinically relevant long-term events is controversial. We aimed to (i) analyse the incidence of pMI depending on various current definitions in a 'real-life' setting of CABG surgery and (ii) determine the long-term prognosis of patients with pMI depending on current definitions.

Methods: A consecutive cohort of 2829 coronary artery disease patients undergoing CABG from two tertiary university centers with the presence of serial perioperative cardiac biomarker measurements (cardiac troponin and creatine kinase-myocardial band) were retrospectively analysed. The incidence and prognostic impact of pMI were assessed according to (i) the 4th Universal Definition of Myocardial Infarction (4UD), (ii) the definition of the Society for Cardiovascular Angiography and Interventions (SCAI), and (iii) the Academic Research Consortium (ARC). The primary endpoint of this study was a composite of myocardial infarction, all-cause death, and repeat revascularization; secondary endpoints were mortality at 30 days and during 5-year follow-up.

Results: There was a significant difference in the occurrence of pMI (49.5 % SCAI vs. 2.9 % 4UD vs. 2.6 % ARC). The 4th Universal Definition of Myocardial Infarction and ARC criteria remained strong independent predictors of all-cause mortality at 30 days [4UD: odds ratio (OR) 12.18; 95 % confidence interval (CI) 5.00–29.67; P: 0.001; ARC: OR 13.16; 95 % CI 5.41–32.00; P: 0.001] and 5 years [4UD: hazard ratio (HR) 2.13; 95 % CI 1.19–3.81; $P=0.011$; ARC: HR 2.23; 95 % CI 1.21–4.09; $P=0.010$]. Moreover, the occurrence of new perioperative electrocardiographic changes was prognostic of both primary and secondary endpoints.

Conclusion: Incidence and prognosis of pMI differ markedly depending on the underlying definition of myocardial infarction for patients undergoing CABG. Isolated biomarker release-based definitions (such as troponin) were not associated with pMI relevant to prognosis. Additional signs of ischemia detected by new electrocardiographic abnormalities, regional wall motion abnormalities, or coronary angiography should result in rapid action in everyday clinical practice.