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Diabetes Mellitus, High Platelet Reactivity and Endothelial Dysfunction Determine the Outcomes after PCI.

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Objective: The purpose of our study was to evaluate the prognostic value of platelet reactivity, initial level of inflammation markers and endothelial dysfunction, as well as CYP2C19*2 allele carriage in clinical outcomes after percutaneous coronary intervention (PCI) in patients with stable coronary artery disease (SCAD) during dual antiplatelet therapy (DAPT).

Methods: A prospective, single-center study included 94 patients with SCAD who underwent PCI with DES implantation. Platelet reactivity was determined in all patients using light transmission aggregometry induced with 5μmol/L ADP (LTA-ADP) and VerifyNow before PCI, as well as CYP2C19 genotyping after patient's discharge. In 74 patients were determined baseline levels of high-sensitivity C-reactive protein, soluble P-selectin, soluble CD40 ligand, highly sensitive IL-6, PAI-1 levels and von Willebrand factor activity.

Results: According to univariate regression analysis we revealed that diabetes mellitus [exp (B) 0,344 95% CI 0,118-1,004, p=0,049], PRU [exp (B) 1,009; 95% CI 1,002-1,017, p=0.01], the number of stented arteries [exp (B) 4,00; 95% CI 1,475-10,848, p=0.01], the number of implanted stents [exp (B) 3,672; 95% CI 1,366-9,872, p=0.01], the initial level of PAI-1 [exp (B) 1,000, 95% CI 0,999-1,000, p=0.03] and the activity of EF [exp (B) 1,000, 95 1,000-1,000% CI, p=0.01]. The presence of CYP2C19*2 carriers showed no significant impact on outcomes after PCI. For quantitative factors we built ROC-curves to determine their critical values. Independent significant influence showed concomitant diabetes mellitus, PRU> =202, PAI-1 level> = 75.95 ng / ml, von Willebrand factor activity> = 155.15%. Based on our findings we developed predictive models for risk stratifying of patients with CAD before PCI.

Conclusions: The independent predictors of adverse cardiac events after PCI were: concomitant diabetes mellitus type 2, the value of PRU (≥202), the level of plasminogen activator inhibitor-1 (≥75.95 ng / ml) and von Willebrand factor activity(≥155.15%).

OP-95

Assessment of Silent Cerebral Ischemia Following Coronary Angiography Procedure.

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Objective: Silent cerebral ischemia (SCI) is an embolic origin cerebrovascular lesion due to embolic vascular occlusion incidentally diagnosed. Coronary angiography (CAG) is established as the gold standard for the assessment of coronary artery disease. However there are minor and major complications related to the procedure. Retrospective data analysis revealed that 0.11% to 0.38% of patients undergoing CAG experienced clinically evident cerebral infarction whereas the incidence of SCI was 13% to 22%. To date, limited data were available regarding the occurrence and predictors of SCI after CAG. In our study we aim to evaluate the incidence and predictors of SCI after CAG.

Method: One hundred one stable coronary artery disease patients without atrial fibrillation and history of serebrovascular disorders underwent coronary anjiography and intervention were included in this study. Percutaneous coronary interventions were performed for selected patients. Cerebral magnetic resonance imaging and diffusion weighted magnetic resonance imaging (DW-MRI) were performed within 24 hours after diagnostic coronary anjiography. Silent cerebral infarction (SCI) was diagnosed with the presence of focal bright high signal intensity on DW-MRI. Patients were divided into diagnostic coronary anjiography and intervention group. Each groups were assessed with presence or absence of SCI. The laboratory findings, clinical and angiographic characteristics were analyzed and compared between patients with and without SCI on DW-MRI.

Results: Total of 101 included patients, SCI occured 24 (24 %) patients. Sex, body mass index, hypertension, diabetes mellitus, LDL, triglyceride, smoking, left ventricular ejection fraction (LVEF) were not significant between SCI (+) and SCI (-) groups. Age, total cholesterol, syntax score, gensini score, CABG history were significantly higher in SCI (+) group compared with SCI (-) group with statistical datas (table-1) respectively (65 ± 10 vs 58 ± 11, p: 0,037; 222.8 ± 85.2 vs 173.4 ± 79.6, p: 0.048; 30,1 ± 2 vs 15 ± 3, p: 0,000; 58,8 ± 55,6 vs 22,1 ± 29,9, p: 0,045; 3 (25%) vs 1 (1,1%), p: 0,005) SCI were observed significantly higher in intervention group. (8/24 vs 4/77, p:0,01). Subgroup analyses for diagnostic coronary angiography and intervention groups were performed (table-2). Syntax score was significantly higher in SCI (+) patients in each groups respectively. (29.3 ± 1.9 vs 15 ± 3, p<0.01; 30.5 ± 1.9 vs 15.1 ± 3.2, p<0.01)

Conclusion: The incidence of SCI after diagnostic and interventional CAG was not infrequent. Higher age, total cholesterol, syntax score, gensini score and CABG evaluation are associated with SCI.

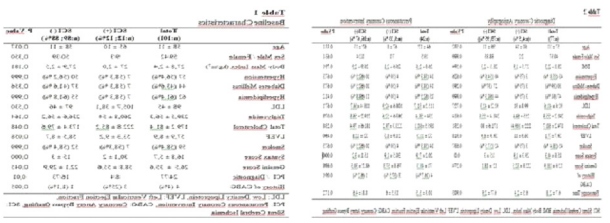

Table.

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The Phenomenon of Periprocedural High on-Treatment Platelet Reactivity During PCI: Clinical, Genetic and Inflammation Determinants.

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Objective: We aimed to determine different laboratory and genetic factors impact on high on-treatment platelet reactivity (HOPR) on dual antiplatelet therapy (DAPT).

Methods: Our study included 94 patients with SCAD (mean age 59 ± 9,67 years). All patients underwent elective PCI with DES implantation during dual antiplatelet therapy (aspirin and clopidogrel). Platelet function was assessed by light transmission aggregometry with 5μmol/