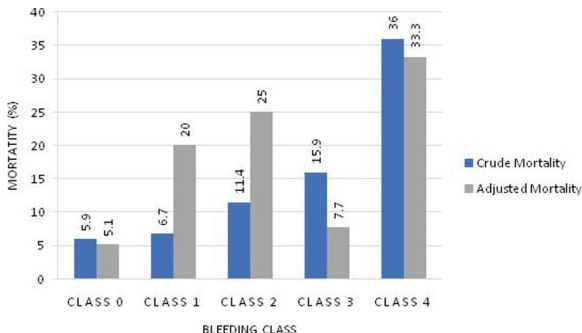


of perioperative bleeding in adult cardiac surgery, ranking 5 degrees of bleeding taking into account the bleed from the drainage, the need for reexploration and the use of blood products. It is the most accurate classification in relation to the degree of bleeding.

Material and methods: A retrospective descriptive and analytical study was conducted on a database of adults patients who underwent cardiac surgery from January 1, 2016 to December 31, 2017. The primary objective of the study was the universal definition of perioperative bleeding to predict the 30-day mortality independently.

Results: A total of 918 patients who went to cardiac surgery were obtained. The majority of the population was classified as insignificant bleeding class (n=666, 72.9%), and for minor bleeding of proportion (n=25, 2.7%). In the primary outcome of 30-day mortality, a significant difference was found between the groups, observing that it increased to a higher class of bleeding. This was corroborated by a multivariate logistic regression analysis that was adjusted to EuroScore II and CPB time, finding an independent association of the bleeding class with 30-day mortality (OR, 95%, 5.82 [2.22–15.26], $p=0.0001$).



Mortality in the bleeding class

Conclusions: We found that the definition of perioperative bleeding predicts 30-day mortality independently in adults patients of cardiac surgery in our hospital.

P804

The AB0 gene locus is associated with increased platelet aggregation in stable coronary artery disease patients

M.K. Christiansen¹, S.B. Larsen¹, M. Nyegaard², S. Neergaard-Petersen¹, M. Wurtz¹, E.L. Grove¹, A.-M. Hvas³, H.K. Jensen¹, S.D. Kristensen¹. ¹Aarhus University Hospital, Department of Cardiology, Aarhus, Denmark; ²Aarhus University, Department of Biomedicine, Aarhus, Denmark; ³Aarhus University Hospital, Department of Clinical Biochemistry, Aarhus, Denmark

Background: Genome-wide association studies of coronary artery disease (CAD) suggest that the majority of risk loci equally increase the risk of CAD and myocardial infarction (MI) through atherosclerosis development, whereas the AB0 locus is stronger associated with the MI phenotype. However, the underlying mechanisms are largely unknown.

Purpose: To investigate the association between the AB0 risk variant and measures of platelet activation and aggregation. Secondly, to explore the effects of other CAD-associated risk variants.

Methods: We included 879 stable CAD patients on mono-antiplatelet therapy with 75 mg aspirin daily. All patients were genotyped for 45 genome-wide significant CAD risk variants, including rs495828 as a perfect proxy for the lead variant at the AB0 locus. A genetic risk score (GRS) was calculated to assess the combined risk associated with all the genetic variants. Soluble P-selectin (sP-Sel) and S-thromboxane B2 (TXB2) were used as measures of platelet activation, and

platelet aggregation was assessed by multiple electrode aggregometry and VerifyNow.

Results: The rs495828 CAD risk allele was associated with higher MEA platelet aggregation with both arachidonic acid (14.9% [95% confidence interval 6.7–23.7%] higher adjusted geometric mean AUC per CAD risk allele, $p=0.0002$), and collagen (13.1% [5.8–20.9%], $p=0.0003$) as agonists. Conversely, sP-Sel levels were 7.5% (3.1–11.7%) lower per CAD risk allele ($p=0.001$). The association between rs495828 and MEA platelet aggregation further strengthened when including adjustments for sP-Sel levels ($p=1 \times 10^{-5}$ and $p=7 \times 10^{-5}$, respectively). There was no association between rs495828 genotypes and aggregation measured by VerifyNow[®] ($p=0.30$) or TXB2 levels ($p=0.98$). None of the remaining risk variants or the GRS were associated with either platelet activation or aggregation.

Conclusions: The CAD risk allele of the AB0 locus was associated with increased MEA platelet aggregation. This finding may potentially provide an explanation for the increased MI risk in AB0 risk variant carriers.

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P805

Risk-stratification and age-adjusted D-dimer test: Are there satisfactory in acute pulmonary embolism?

A. Pandur¹, B. Banfai², D. Sipos¹, B. Schisler¹, J. Betlehem², B. Radnai². ¹University of Pecs, Doctoral School of Health Sciences, Pecs, ²Rudnai of Pecs, Institute of Emergency Care and Pedagogy of Health, Pecs, Hungary

Background/Aims: Pulmonary embolism is connected with high morbidity and mortality. Prognostic assessment is important for the management of patients with pulmonary embolism. Pulmonary embolism often has a nonspecific clinical presentation. The use of diagnostic testing in an attempt to avoid missing the potentially life-threatening diagnosis increases both cost and use of medical resources. Various score systems exist to evaluate the probability of pulmonary embolism, which can be used for risk stratification, to get the most accurate diagnosis. The aim of our study was to review the evidence for existing prognostic models in acute pulmonary embolism and determine validity and usefulness for predicting patient outcomes. We determine the accuracy of an age-adjusted D-dimer threshold to detect pulmonary embolism.

Materials and methods: We performed a retrospective analysis of pulmonary embolism in three Hungarian emergency departments. Data from 659 patients were included for this retrospective analysis. The Wells, Geneva score systems were used to reevaluate retrospectively the risk of pulmonary embolism. The diagnosis of pulmonary embolism was accurate, when the CT verified it. We calculated specificity, sensitivity, negative and positive predictive values of the different strategies. Data were analyzed with a SPSS 20.0 statistical software. In our study, chi-square test, Independent-Samples T-test, ANOVA, correlation interpretation were performed. P values of <0.05 were considered to be statistically significant.

Results: Our study included 659 patients (407 women, 252 men), admitted to three ED. In the 659 cases all over 105 D-dimer assays, 51 CT angiograms and 212 chest X-ray examinations were carried out routinely, which could have mean saved money to the hospitals and less radiation to patients. The age adjusted D-dimer threshold was more specific (70% versus 60%) but less sensitive (95% versus 98%). The sensitivity of the combination (risk-stratification and age adjusted D-dimer test) was 100%.

Conclusions: Our study showed that Genfi score (which was calculated from the patients complaints, medical history and physical examination) had the closest correlation with the diagnosis. An age adjusted D-dimer limit has the potential to reduce diagnostic imaging. This is more accurate than a standard threshold of 500 ng/dl. The combination of the risk stratification and the age adjusted D-dimer we can safely diagnose the pulmonary embolism. Finally we can conclude that risk-evaluation in acute PE is indispensable and the appropriate use of guidelines results in lower costs.

P806

The relationship between heparanase levels and high thrombus burden in patients with ST-segment elevation myocardial infarction

A. Gurbuz¹, S. Ozturk², S.C. Efe², M.F. Yilmaz², M. Inanir², C. Onal², A. Kilicgedik², C. Kirma². ¹necmettin erbakan university meram faculty of medicine, Cardiology, Konya, Turkey; ²Kartal Kosuyolu Heart and Research Hospital, cardiology, Istanbul, Turkey

Background: Heparanase (HPA) is the only endo-β-D-glucuronidase capable of degrading heparan sulphate (HS) and heparin side chains. HPA plays a role in tumor growth, angiogenesis, cell invasion and also plays a direct role in the activation of the coagulation system. In this study, we aimed to investigate the role of HPA with regard to thrombus burden in patients with ST-Segment Elevation Myocardial Infarction (STEMI).

Methods: This prospective study enrolled 98 patients with STEMI treated primary percutaneous coronary intervention (pPCI). Blood samples were taken to determine serum HPA levels prior to coronary angiography and heparin administration. Serum HPA analysis was performed with a commercially available Human Elisa kit.

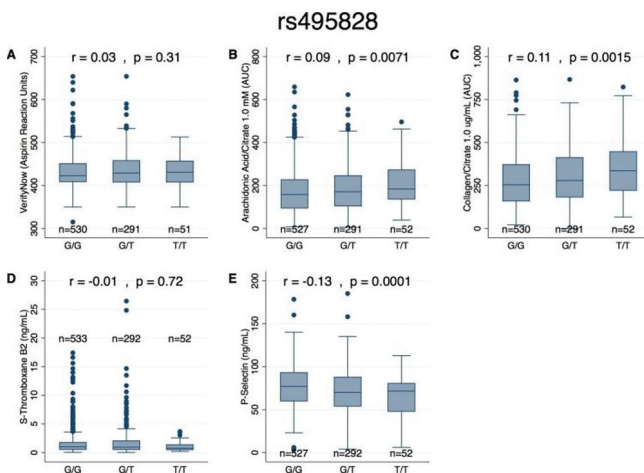


Figure 1. rs495828 (AB0 gene locus)

Results: Patients were divided into two groups based on thrombus burden; high thrombus burden (HTB) (n:30) and low thrombus burden (LTB) (n:68) group. Serum HPA levels were significantly higher in patients with HTB than LTB [205.4 (168.6–281.9) pg/ml vs 175.4 (132.0–219.4) pg/ml] ($p=0.005$). Serum HPA levels were higher in patients with no-reflow phenomenon compare with others [(356.3 (174.3–499.4) pg/ml vs 182.6 (144.2–224.1) pg/ml, $p=0.02$).

Conclusion: This is the first study in literature that demonstrates the role of HPA on thrombus burden in patients with STEMI. Elevated HPA levels in patients with STEMI may be one of the independent causes of high thrombus burden based on procoagulant activity.

P807

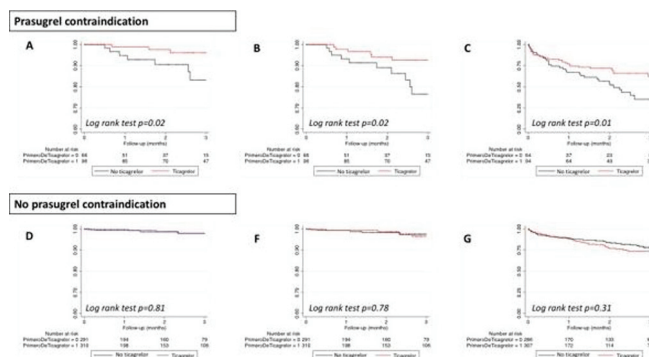
Prevalence, long-term prognosis and medical alternatives for patients admitted for acute coronary syndromes and prasugrel contraindication

A. Cordero¹, M. Rodriguez-Manero², J.M. Garcia-Acuna², R. Agra-Bermejo², B. Cid-Alvarez², B. Alvarez², V. Bertomeu-Gonzalez², A. Frutos¹, R. Lopez-Palop¹, V. Bertomeu-Martinez¹, J.R. Gonzalez-Juanatey². ¹University Hospital of San Juan, Alicante, Spain; ²University Clinical Hospital of Santiago de Compostela, Santiago de Compostela, Spain

Background: Prasugrel is a potent antiplatelet therapy that has demonstrated to be superior to clopidogrel for patients with acute coronary syndromes (ACS) but has three main contraindications: age >75 years, weight <60 kg or previous stroke.

Methods: Prospective study of all patients admitted for ACS in two hospitals, between 2005 and 2016, comparing patients with vs. without any prasugrel contraindication. We also performed a propensity score matching to obtain a well-balanced subset of patients with the same probability of receiving ticagrelor.

Results: We included 8207 patients and 2,538 (30.9%) had any contraindication for prasugrel, being age >75 years the most frequent (29.0%). Hospital mortality was 4.4% and it was more than 2-fold higher in patients with any contraindication for prasugrel (7.9% vs. 2.8%; $p<0.01$). Postdischarge follow-up (median 59.9 months) was achieved in >90% of the patients and cardiovascular mortality rate was 12.4%, all-cause mortality 21.1% and 47.3% patients experienced at least one MACE. Patients with prasugrel contraindication had higher cardiovascular and all-cause mortality as well as a first major cardiovascular event (MACE) (all log-rank test $p<0.01$). No differences in bleeding rates were found in patients with vs. without prasugrel contraindication. Prasugrel contraindication was independently associated to higher MACE risk (HR: 1.3 (1.1–1.8); $p=0.04$) but not to mortality. In the sub-cohort of 482 pairs of patients, obtained by a propensity score matching, ticagrelor treatment was associated with lower cardiovascular death (HR: 0.22, 95% CI 0.1–0.9, $p=0.04$), all-cause mortality (HR: 0.30, 95% CI 0.1–0.8, $p=0.03$) and first MACE (HR: 0.58, 95% CI 0.3–0.9; $p=0.04$) in patients with prasugrel contraindication.



Conclusions: Almost one third of ACS patients have prasugrel contraindications and they have an increased risk for hospital mortality and MACE after discharge. Ticagrelor improves postdischarge outcomes in patients with prasugrel contraindications.

P808

Association between genetic variants in PCSK9/APOB/LDLR and premature myocardial infarction in Han Chinese

H. Yu, T. Pu, M. Xu, W. Gao. Peking University Third Hospital, Beijing, China People's Republic of

Background: The incidence of myocardial infarction (MI) for younger patients is increasing. Dyslipidemia is an important risk factors. Our previously study found the variations of PCSK9, APOB, LDLR gene were related with dyslipidemia. These 3 genes were key regulators to familial hypercholesterolemia (FH) which characterized by premature CHD. In this study, the association of PCSK9, APOB, LDLR gene variations with lipid levels and the risk of premature MI in Han Chinese and the interactions between the variations and CHD risk factors was investigated.

Methods: We perform an exome-wide association study by genotyping 8,480

Han Chinese without lipid-lowering treatment, using Illumina Human Exome BeadChip. According to AMI diagnosis and onset age (male younger than 55 ys, female younger than 65 ys), 413 AMI patients and 1,239 age- matched controls were selected. Next, we explored the association of PCSK9, APOB, LDLR variations with the risk of premature MI and the interactions between variations and CHD risk factors.

Results: Single-variant association analysis on 243 variations reveal 3 loci associated with lipids at exome-wide significance, including PCSK9 rs151193009 (TC: $P=7.45\times10^{-12}$; LDL-C: $P=8.68\times10^{-23}$), APOB rs13306194 (TC: $P=6.56\times10^{-4}$; LDL-C: $P=7.83\times10^{-8}$) and APOB rs1367117 (TC: $P=9.72\times10^{-4}$). In dominant model, the carriers of PCSK9 rs151193009 T-allele, APOB rs1042034 T-allele and rs13306194 A-allele had lower risk of MI than non-carriers and the risk may reduce about 66%, 24%, 30% respectively ($P<0.05$).

Conclusion: The variation of PCSK9 rs151193009, APOB rs13306194 may be specific to Asia people. Both of the variations are negatively correlated with the TC and LDL-C levels and could reduce the risk of premature MI in Han Chinese.

P809

Serum microRNA-27a expression, association with severity of coronary atherosclerosis in coronary heart disease patients

E. Polyakova¹, A. Draganova², M. Zaraiskii¹, O. Berkovich¹, E. Baranova¹.

¹Saint Petersburg medical university, Saint-Petersburg, Russian Federation;

²Almazov Federal Heart, Blood and Endocrinology Centre, Saint-Petersburg, Russian Federation

Introduction: Cardiovascular disease (CVD), including coronary heart disease (CHD), remains a leading reason of mortality. Moreover, it is not always understandable, why under equal conditions some patients suffer from single vascular damage, when others have two or more damaged coronary vessels. Personified approach necessitates searching and examination of new high-sensitive and specific risk factors. The results of recent studies show that microRNA (miRNA) participates in pathogenesis coronary atherosclerosis and its clinical implication – CHD.

Purpose: To evaluate miRNA-27a expression in blood serum of CHD patients with different degrees of coronary artery lesion.

Methods: 100 patients were included in the study, they formed 3 groups: 40 patients with CHD and hemodynamically significant atherosclerosis of 1–2 coronary arteries; 40 patients with multivessel coronary disease (3 arteries and more) and 20 examined samples without CHD and significant comorbidity. The miRNA-27a expression was determined in blood serum of these patients. Blood serum samples were used for RNA separation. RNA was detached with the use of a miRNeasy Mini Kit, according to the device manual. Concentration of RNA in aqueous solution was determined by means of spectrophotometer Nanodrop 1000. Reverse transcription was carried out using a TaqMan microRNA reverse transcription kit. Real-time polymerase chain reaction was performed with the use of kits for determination of expression of studied miRNA, TaqMan® Gene Expression Assays, according to manual.

Results: Level of miRNA-27a expression in blood serum in CHD patients with lesion of 3 or more coronary arteries was significantly increased, rather than in patients with 1–2 damaged vessels: 11.41 ± 4.45 REU and 4.82 ± 1.82 REU, respectively ($p<0.01$). miRNA-27a level of blood serum in examined group with no coronary atherosclerosis was significantly lower, rather than in groups of CHD patients described above, that were formed according to a number of damaged arteries: 3.05 ± 0.89 REU ($p<0.001$ for 3 damaged vessels and $p<0.05$ for 1–2 damaged vessels).

Conclusions: Misbalance and dysfunction of miRNA expression process, which lead to high and low levels of some miRNAs, can participate in cascade of pathological events and cause disease. Therefore, miRNAs can be regarded as risk factors, also they are potential diagnostic and predictive markers. Furthermore, there is a possibility of therapeutic use of miRNA antagonists for inhibition of respective miRNA activity and its effects. Characteristic of analyzed in this study miRNA-27a in CHD patients shows their involvement in the processes of atherogenesis, chronic inflammation, fibrosis, insulin sensitivity. This observation expands understanding of molecular basis of CHD pathogenesis by means of quantitative evaluation of microRNA-27a expression in material available for testing – blood serum.

P810

Histologic comparison of intracerebral and intracoronary thrombi

Y.W. Park¹, Y.H. Jeong¹, J.H. Kim², S.N. Sohn³, C.H. Kwack¹, J.Y. Hwang⁴.

¹Gyeongsang National University Changwon Hospital, Internal Medicine, Changwon, Korea Republic of; ²Gyeongsang National University Hospital, Biomedical Research Institute, Jinju, Korea Republic of; ³Hanil Hospital, Neurology, Jinju, Korea Republic of; ⁴Gyeongsang National University Hospital, Jinju, Korea Republic of

Introduction: Pathophysiologic mechanisms of arterial and venous thrombi formation are considered to be different each other. However, antiplatelet agents are frequently used in the early period after stroke.

Purpose: We compared histologic features of intracerebral and intracoronary thrombi.

Methods: The thrombi were retrieved from 12 totally occluded coronary arteries