

follow-up in patients with MR, proportionally to the initial MR degree. In the rhythm analysis (126 patients; previously excluding those with any history of atrial fibrillation) after follow-up, 11.4% degree I MR-patients developed AF, 14.3% degree II and 75% degree III, while only 5.1% degree 0-patients developed AF, p<0.001.

Conclusions: MR is frequent after a NSTSEACS. The presence and degree of MR confers a worse long-term prognosis after a first NSTSEACS. In part, this can be explained by an increased negative remodeling and increased occurrence of atrial fibrillation.

P477 | BEDSIDE The relationship between preinfarction angina and serum sphingosine 1 phosphate levels

E. Kiziltunc¹, A. Abaci², S. Ozkan², Y. Alsancak², S. Unlu², E.S. Simsek², S. Elbeg², M. Cemri². ¹*Ankara Numune Education and Research Hospital, Ankara, Turkey:* ²*Gazi University School of Medicine, Ankara, Turkey*

Purpose: Previous studies have reported improved cardiac outcomes among patients who experienced preinfarction angina. It has been proposed that the benefit of preinfarction angina might be due to ischemic precondition of the myocardium during brief episodes of ischemia. Sphingosine 1 Phosphate (S1P), an active metabolite of sphingolipid metabolism, has functions in intracellular calcium mobilization, cytoskeletal organization, angiogenesis, cellular differentiation and survival. Experimental studies have shown that S1P is an effective cardioprotectant against ischemic injury. Our aim was to evaluate the relationship between pre-infarction angina and serum S1P levels.

Methods: Between May 2011 and January 2012, 79 patients who had STEMI or NSTEMI enrolled to the study. All the patients questioned about whether they had pre-infarction angina or not before the index event and they divided into two groups as the pre-infarction angina positive and pre-infarction angina negative. The serum levels of S1P at admission and discharge, peak CKMB and troponin levels were measured in both groups.

Results: Of the 79 patients, 36 had pre-infarction angina and 43 had not. The groups were similar according to the baseline characteristics. Mean level of serum S1P in patients with pre-infarction angina was significantly higher than those without pre-infarction angina ($0.78 \pm 0.73 \ \mu$ M versus $0.45 \pm 0.42 \ \mu$ M, p=0.014). Mean level of serum S1P at the discharge was also significantly higher in pre-infarction positive group compared to the pre-infarction negative group ($0.91 \pm 0.84 \ \mu$ M 0.49 ± 0.51 , p=0.010). Serum S1P levels both at admission and discharge tend to be higher with more angina episodes, the differences were only statistically significant between no angina group and three or more angina group ($0.45 \pm 0.42 \ \mu$ M, p=0.045 and $0.49 \pm 0.51 \ \mu$ M versus $1.05 \pm 0.87 \ \mu$ M, p=0.045 and $0.49 \pm 0.51 \ \mu$ M versus $1.05 \pm 0.87 \ \mu$ M, p=0.022, respectively).

Conclusions: In our study, patients who experienced pre-infarction angina had higher serum S1P levels than patients without pre-infarction angina. This finding suggests that S1P may have a role in myocardial preconditioning.

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In-hospital mortality prediction for STEMI patients submitted to primary PCI

M. Sladojevic¹, K. Pavlovic¹, L. Velicki¹, N. Cemerlic-Adjic¹, T. Popov¹, S. Tadic¹, S. Sladojevic², D. Culibrk². ¹Institute of Cardiovascular Diseases Vojvodina, Novi Sad, Serbia; ²Faculty of technical sciences, University of Novi Sad, Novi Sad, Serbia

Purpose: The aim of the study was to evaluate the innovative approach of using Data Mining techniques for developing an in-hospital mortality prediction model for patients presented with ST-segment elevation myocardial infarction (STEMI) submitted to primary percutaneous coronary intervention (pPCI).

Methods: A set of patient-related data obtained from the hospital information system was analyzed in order to discover underlying patterns and relations between distinctive patient features. A total of 2030 patients (aged 61.29±11.70 years, 66.79% males), hospitalized for STEMI and treated with pPCI between December 2008 and December 2011 were included in this retrospective study. Each patient was initially described using 522 attributes. The initial set of features, which included clinical and demographic characteristics, biochemical analysis of blood parameters, ECHO parameters, angiographic and procedural details and diagnosis codes formed the basis for the study. A number of machine learning algorithms were evaluated and the most successful was chosen. Model induction and evaluation was conducted within the widely used Waikato Environment for Knowledge Discovery (WEKA). Ten-fold cross validation was used for model validation.

Results: The in-hospital mortality was 7.73%. The best prediction results were achieved using Alternating Decision Tree (ADTree) classifier, which was able to predict in-hospital mortality with 97.8% accuracy (AUC=0.99). ADTree identified a subset of ten attributes most relevant to mortality prediction. These include: urea, age, hemoglobin, DeBakey score, INR, total cholesterol, whether the patient is a physical laborer, left ventricular stroke volume (LVSV), aortic velocity time integral and Blush flow. Binary logistic regression confirmed that 5 of 10 selected predictors by artificial intelligence algorithm could be used as independent predictors: age (OR 1.065; 95% CI 1.048-1.082; p<0.0005), DeBakey score (OR 1.860; 95% CI 0.923; 95% CI 0.903-0.942; p<0.0005), hemoglobin (OR 0.969; 95% CI 0.941-0.997; p<0.03).

Conclusions: The resulting decision tree is a highly accurate graphical model suitable for expert interpretation, yet relatively simple as it contains only 31 nodes and 21 leaves. The model might prove to be very helpful in the decision-making process and optimizing treatment strategy in selected high risk patients.

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Epicardial adipose tissue thickness independently predicts myocardial infarction, and it is related to impaired coronary perfusion in patients with coronary artery disease

A. Tanindi¹, S.A. Kocaman², A.F. Erkan¹, M. Ugurlu¹, H.F. Tore¹. ¹Ufuk University Faculty of Medicine, Ankara, Turkey; ²Ankara Guven Hospital, Ankara, Turkey

Purpose: Epicardial adipose tissue (EAT), a special fat depot that is related to visceral fat rather than total adiposity, shares the same microcirculation with myocardial tissue and coronary vessels. We aimed to investigate if EAT thickness might be used to predict acute myocardial infarction. We also searched for the relation between EAT thickness and objective coronary flow parameters such as myocardial blush grade (MBG) and Thrombolysis in Myocardial Infarction Frame count (TFC).

Methods: We included 200 consecutive patients who were admitted with stable angina pectoris or acute coronary syndrome between June and October 2012, and decided to undergo coronary angiography. Epicardial adipose tissue thickness was evaluated by transthoracic echocardiography. Coronary angiography was performed to deterine the coronary involvement and perfusion.

Results: Mean EAT thicknesses were 5.4 ± 1.9 mm, 6.3 ± 1.8 mm, and 8.5 ± 1.4 mm in the stable angina pectoris (SAP), unstable angina pectoris (USAP) and acute myocardial infarction (AMI with or without ST elevation) groups, respectively (p<0.001). With increasing EAT thickness TFC increases whereas mean MBG values decrease (for EAT thickness -5 mm, -7 mm, -7 mm; mean TFC: $21.6\pm2.2, 25.3\pm3.3$ and 35.2 ± 7.7 ; and MBG values: $2.98\pm0.14, 2.83\pm0.57$ and 1.7 ± 1.16 , respectively; both p<0.001). Multivariate analysis and ROC analysis revealed that EAT can be used as an independent and powerful predictor of AMI. Cut-off EAT value to predict AMI was identified as 7.8 mm (ROC analysis 0.874; p<0.001, 95% CI:0.822-0.927). Sensitivity, specificity, positive predictive value, negative predictive value and the diagnostic accuracy of EAT cut-off 7.8mm to predict AMI were 81.8%, 82.5%, 48%, 95.8% and 82.4%, respectively.

Conclusions: Quantification of EAT thickness as a predictor of AMI in CAD patients using echocardiography which is a relatively cheap and readily available tool may prove beneficial for choosing patients who would need more aggressive approach in terms of risk reduction

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Impact of glucose fluctuation and monocyte subsets on coronary plaque rupture

I. Teraguchi, T. Imanishi, Y. Ozaki, T. Tanimoto, T. Yamano, Y. Ino, T. Yamagichi, K. Hirata, T. Kubo, T. Akasaka. *Wakayama Medical University, Department of Cardiovascular Medicine, Wakayama, Japan*

Background: It remains unclear whether glycemic fluctuation can affect plaque rupture in acute myocardial infarction (AMI). Here we investigate the impact of glucose fluctuation on plaque rupture, as observed by optical coherence tomography (OCT), and monocyte subsets in patients with AMI.

Methods: We studied 26 consecutive patients with AMI. All patients underwent OCT examination, which revealed 16 patients with plaque rupture and 10 patients without plaque rupture at the culprit site. Peripheral blood sam-