showed moderate correlations with PWV: 0.375 (p<0.05) and 0.412 (p<0.0001) in males and 0.468 (p<0.0001) and 0.389 (p<0.001) in females. Only few biochemical markers demonstrated a significant correlation (p<0.05): TC and TG in males (0.326 and 0.290) and Lp(a) in females (-0.261). The stepwise least squares multiple regression analysis showed an association of PWV only with DBP (p<0.001) in males and with SBP (p<0.001) in females (R=0.649 and 0.443, R2-adjusted = 0.408 and 0.181).
Conclusions: In young Tallinn adults, BP showed an important impact on PWV variability, whereas biochemical markers failed to demonstrate significant association.

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05. Cardiovascular disease: risk, prevention, and treament - 05.04 Novel risk factors and biomarkers

EAS19-1080.
IN DEPTH CHARACHTERIZATION OF DYSLIPIDEMIA BEYOND CONVENTIONAL LIPID PANEL IN PATIENTS WITH TYPE 2 DIABETES

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Background and Aims: Despite progress in treating type 2 diabetes (T2D), patients have a high residual risk of cardiovascular complications. This may be due to incomplete management of T2D related dyslipidemia, characterized by high plasma TG, dense LDL (dLDL) and low HDL especially HDL_2. Usually CVD risk is estimated based on LDL, HDL and TG, with LDL as primary target and statins as first treatment choice. This ''one-size-fits-all approach'' is possibly insufficient. Identifying additional diagnostic and treatment targets might further reduce CVD risk in T2D. Our main objective is to optimize individual diagnosis and treatment of dyslipidemia in T2D using CVD risk classification based on in-depth lipoprotein profiling. Primary endpoint is difference in choice of lipid-lowering therapy when using conventional lipid panel versus in-depth lipoprotein profiling.
Methods: We obtained blood samples of 100 T2D patients. The conventional lipid panel was determined by standard clinical chemistry and in-depth profiling by density gradient ultracentrifugation (UC). UC provides information on: HDL, HDL_3, HDL_2, Lp(a), LDL, dLDL, IDL, VLDL-cholesterol and VLDL-TG. Plasma Lp(a) was also determined using an immunoturbidimetric assay. We assessed whether treatment advice according to the EAS guidelines based on the in-depth lipoprotein profiling differed from that based on the conventional lipid panel.
Results: In 70% of the patients treatment advice based on in depth-profiling was different from the advice based on the conventional lipid panel. Differences included a higher dosage of current lipid-lowering therapy or an additional lipid-lowering medication.
Conclusions: These results suggest that in-depth lipid profiling may contribute to further reducing CVD risk in T2D patients.

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EAS19-0425.
SOLUBLE ST2 CORRELATION WITH LEFT VENTRICULAR REMODELING IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Background and Aims: LV remodeling occurs in a significant number of patients with acute myocardial infarction (AMI). Soluble suppression of tumorigenicity 2 (sST2) can be considered as a promising predictor of LV remodeling. The purpose of the study was to investigate the influence of sST2 on the development of left ventricular remodeling in patients with ST elevation myocardial infarction (STEMI) after revascularization.
Methods: The study included 55 patients with STEMI, 45 (81.8%) males. The average age is 58.15±10.85 years. The patients were divided into 2 groups: the first group with remodeling of LV included 28 (56%) patients, and the second group – without LV remodeling with 22 (44%) patients. Remodeling was assessed as increase of 20% of the LV end-diastolic volume (EDV) after 6 months of observation. 5 patients left the study.
Results: The sST2 level in all studied patients was 34.28 [25.32-67.81] ng/ml. The serum sST2 level in the first group was 50.35 [27.17-103.20] ng/ml, in the second – 28.02 [21.75-34.70] ng/ml (p<0.015). Multivariate logistic analysis ($\chi 2$=30,71; p = 0,0001), revealed the direct correlation of the LV remodeling (for 6 months) with the creatinine level (β=0,093; p=0,033) and reverse correlation with sST2 (β=-0,079; p=0.025). When ROC analysis was performed, it was found that sST2 level greater than 44.5 ng/ml allows to predict the development of LV remodeling, AUC = 0.707, (p=0.0198), with a sensitivity of 85.7% and a specificity of 57.1%.
Conclusions: sST2 concentration, determined at the first day of STEMI, can predicts the development of LV remodeling after 6 months

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EAS19-0501.
NEW BIOMARKERS OF CARDIOVASCULAR ENDPOINTS AND HEART FAILURE PROGRESSION ANALYSIS AFTER MYOCARDIAL INFARCTION

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Background and Aims: Myocardial infarction (MI) is one of the main factors in heart failure (HF) progression. New and sensitive biomarkers such as ST2 and Pentraxin-3 (Ptx-3) have prediction potential in HF and primary cardiovascular endpoints prediction in the remote period.
Objective: Study of predictive power of biomarkers on cardiovascular endpoints in 12 months after MI and systolic left ventricular (LV) function failure progression.
Methods: NTproBNP, P-3 and ST2 were estimated 3 days after MI (n=180). In 12 months the primary endpoints (MI, stroke, death) were analyzed. Patients were divided into 1st groups with serum ST2 below (n=108, 60,0±2,3 year, ST2=27,9±8,9 ng/ml) and 2nd – with ST2 above the threshold (n=72, 63,9±1.9, ST2=97.7±21.9 ng/ml). In 1 year in 58 patients (36 from the 1st and 22 from the 2nd groups) the ejection fraction (EF) was repeated.
Results: In 1 year the death ratio (6.9% vs 4.6%), strokes (4.2 vs. 1.9) and hospitalizations (11.1 vs 4.6) was higher in the 2nd group but MI – in the 1st (12.5 vs 16.7). High ST2 increased probability of stroke on 1.4% but decreased of MI on 1.3% (p<0.05). NTproBNP (marginal impact on mortality + 1.52 %, p<0.01) and Pentraxin-3 (marginal impact 1.18%, p<0.05) both predicted mortal endpoint. In 1 year EchoCG failed to define the heart failure progression estimated by EF in the groups with high and low ST2 (p=0.4 between groups).
Conclusions: NTproBNP, ST2 and Ptx-3 showed predictive power in cardiovascular endpoints in FU analysis after MI but failed to predict systolic left ventricular function decline.

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EAS19-0664.
NEUROPROTECTIVE EFFECTS OF GENISTEIN IN EXPERIMENTAL GLOBAL CEREBRAL ISCHEMIA AND REPERFUSION IN STREPTOZOTOCIN-INDUCED DIABETIC MICE

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