P694 Acute kidney injury is a stronger predictor of one-year mortality than chronic kidney disease in patients with acute myocardial infarction. The HEROES study

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Purpose: We investigated the incidence, clinical predictors and prognostic value of acute kidney injury (AKI) regarding 1-year mortality in acute myocardial infarction (AMI) patients with and without chronic kidney disease (CKD).
Methods: We collected in-hospital data from 447 patients hospitalized for AMI in our institute within 12 hours of symptoms' onset. From blood samples obtained on admission and throughout hospitalization hemoglobin, white blood cell count, C-reactive protein, B-type natriuretic peptide, plasma glucose, troponin I and baseline and peak creatinine levels were measured. MDRD equation was used to estimate glomerural filtration rate (GFR). AKI was defined as a 25% or more decrease in GFR during hospital stay. CKD was defined as an estimated GFR between 15 and 59 ml/min/1.73m^2. Ejection fraction was calculated on admission with 2D echocardiography. All patients underwent coronary arteriography and the revascularization status (complete or not) was also recorded. The end-point was all-cause mortality after one year of follow-up.
Results: AKI was detected in 63 pts (16.7%). Age (OR 1.074; 95%CI 1.041-1.109), ejection fraction (OR 0.951; 95%CI 0.923-0.980) and white blood cell count (OR 1.089; 95%CI 1.004-1.181) were the only independent predictors of AKI. The incidence of 1-year mortality was 10.7% (48 deaths). Patients with AKI exhibited higher 1-year mortality (37.5% vs. 6.3%, log rank $p<0.001$). AMI patients with AKI and CKD (n=28, 6.3%) had a 17.8-fold greater incidence of mortality compared to those without either AKI or CKD (n=278, 62.2%) (log-rank $p<0.001$). Moreover, patients with AKI but not CKD (n=36, 8.1%) exhibited a 5.3-fold greater incidence of mortality compared to those without AKI and CKD (log-rank $p<0.001$) while the 2.3-fold increased mortality rate in patients with AKI but not WRF (n=105, 23.5%) reached marginal statistical significance (log-rank p=0.041). Finally, AMI patients with AKI but not CKD compared to those with CKD but not AKI had 2.4-fold greater incidence of one-year mortality (log-rank p=0.06). By applying multivariate Cox regression analysis AKI (adjHR 5.024, $p<0.001$), BNP (adjHR 2.859, p=0.004), ejection fraction (adjHR 0.943, p=0.003) and admission diastolic blood pressure (adjHR 0.971, p=0.017) remained the only predictors of death.
Conclusions: AMI patients with AKI and normal baseline renal function appear to have a worse prognosis than patients with CKD but not AKI, while co-existence of both AKI and CKD acts synergistically and further increases one-year mortality.

P695 Relative hyperandrogenemia in central obesity as possible predictor of coronary artery disease in postmenopausal women

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Objectives: Hypoestrogenemia is an established metabolic risk factor for cardiovascular diseases in women. After menopause 34-45% of women show extensive abdominal fat deposition, therefore aromatizing of estrogens continues particularly in fat tissue. Development of central obesity impairs normal process of estradiol conversion and benefits the predominance of inactive estrone therefore leading to relative hyperandrogenemia. The purpose of our study was to establish the role of hyperandrogenemia and central obesity for coronary artery disease development in postmenopausal women by means of determining the peculiarities of the pituitary activity, dehydroepiandrosterone sulfate level and blood lipoproteins levels.
Methods: 63 females in postmenopause were evaluated. The patients were randomized into 2 groups comparable by age and postmenopause duration. 1st group included 32 women with signs ofcentral obesity, 2nd – 31 women with normal body mass index and waist/hip ratio. All patients were assessed by Kupperman menopausal index for climacteric symptoms evaluation, blood lipids level, levels of follicle stimulating & luteinizing hormones, dehydroepiandrosterone by means of radioimmune assay.
Results: Gonadotropins in all patients reflected the postmenopausal changes: high levels gonadotropins were equivocal in 2 groups. The mean value of Kupperman index in 1st group was 45,2±6,7, in 2nd - 34,7±2,3 what evidenced more severe climacteric symptoms in women withcentral obesity. There was significant predominance of women with hyperandrogenemia among those with elevated body mass index (28,7±1,8 kg/cm^2) and signs of central obesity. Atherogenic changes of blood lipids were registered in both groups: increased levels of total cholesterol and low density lipoproteins were found in women with hyperandrogenemia ($p>0,05$); increased levels of triglycerides and low levels of high density lipoproteins were noted in women with central obesity ($p<0,05$). Level of dehydroepiandrosterone sulfate showed positive correlation with triglycerides and negative – with high density lipoproteins levels.
Conclusions: Postmenopausal women with central obesity have relative hyperandrogenemia and develop more severe symptoms of climacteric syndrome than women with normal waist/hip ratio. Levels of dehydroepiandrosterone have positive correlation with triglycerides and negative – with high density lipoproteins levels. Evaluation of the waist/hip ratio and consequentially serum levels of dehydroepiandrosterone should be included for screening patients in postmenopuse for individual coronary artery disease risk evaluation.

P696 CETP polymorphism adds to the characterisation of premature coronary heart disease and multiple vessel disease

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Patients with premature coronary syndromes impose an enormous challenge of diagnosis and long-term management. Traditional markers can be ephemeral or unespecific and may fail to identify patients who are at high risk for cardiovascular disease, to develop severe disease or have poorer outcomes. Traditional risk factors do not explain all of the risk for CHD, and emerging risk factors have the potential to improve global risk assessment.
Aim: We analysed a common polymorphism in the Cholesteryl Ester Transfer Protein (CETP) gene (Taq1B) related to lipid profiles.
Method: An atypical model was designed to compare a set of 570 male Canarian patients (P) with early onset acute coronary syndromes (under 55 years old) with 199 healthy, aged controls (c), far beyond the age of premature CHD (over 65 y-o). PCR-RFLP techniques rendered similar B1B1-B1B2/B2B2 allele distribution in both groups.
Results: The mean age was 46 for patients (P) and 71 for controls (c); $p<0.000$. The case/control analysis showed significant differences ($p<0.000$) in the prevalence of family history of CHD (61 P/39% c), family premature CHD (35 P/7% c), dyslipidemia (71 P/53% c), hypertension (48 P/74% c), mean atherogenic index (log triglycerides/HDL-cholesterol: 0.63 P/0.47 c) and exercise tolerance (METS: 10 P/7 c). The first ACS was myocardial infarction in 64% and angina in 46%; multivessel disease was present in 55%, 63% underwent revascularisation (PCI 62, CABG 7%; restenosis rate 9%) and 33% had recurred ACS. B2B2 genotype was more common among patients (15 P/10% c; $p<0.045$); while variant homozygous genotype was associated with multivessel disease (47/37%; $p<0.013$). Our model predicted 74.1% of premature CHD with family history of CHD and common CEPT genotype. A logistic regression model built with risk factors, genotypes, clinical features and atherogenic index: depicted dyslipidemia ($p<0.019$) and variant CETP genotype ($p<0.031$) to explain 72.5% of multivessel disease in patients with angina.
Conclusions: Despite an unusual model with a considerable age gap our young patients had a very high prevalence of risk factors. Variant CETP genotype is an inexpensive suitable marker for premature CHD and multivessel disease, thus contributing to characterise individual variability.

P697 Ivabradine impact on heart rate in patients with chronic obstructive lung disease and coronary heart disease after the inhalation of salbutamol

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Background: Tachycardia is a frequent display of chronic obstructive pulmonary disease (COPD) which complicates accompanying coronary heart (CHD).
Aim: To assess an effect of salbutamol 400 μg and ivabradine 5 mg on heart rate in patient with COPD combined with CHD.
Methods: Cross-randomized controlled study. Study included twenty patients (18 males and 2 females) with COPD combined with CAD, NYHA I-III. Average age was 62±8.5 yrs, FEV1 - 49.1±25.3. 55% of patients had pulmonary arterial hypertension at a level more than 30 mm. Hg estimated echocardiographycally. On six minutes walking test the average distance was 447±63.5 m. Standard inhalation test with about 400 mkg of salbutamol within two consecutive days was performed. In one of the days, the patient accepted ivabradine 5 mg per os certain under the table of random numbers and 3 hours prior to inhalation.
Results: Results of this study have been divided into groups: "salbutamol 400 mkg" (S) and "salbutamol 400 mkg+ivabradine 5 mg" (S+I). The positive gain of heart rate frequency was marked in group S after salbutamol inhalation, in average on 5.5±10.5 impacts in a minute ($p<0.03$). Gain of heart rate frequency was - 2.4±10.5 in S+I group (p=0.9). The gain of heart rate frequency in S-group markedly exceeded the gain in S+I-group ($p<0.05$). Salbutamol inhalation caused the positive gain of FEV1 in S-group 6±7.4% and in S+I-group on 7.7±10.9% from due ($p<0.01$). The gain between two groups did not differ statistically.
Conclusion: Ivabradine prevent tachycardia after inhalation of high doses of salbutamol and has no impact on lung function in patients with COPD and CAD.