P7.12: ARTERIAL STIFFNESS IN PATIENTS WITH HEART FAILURE OF ISCHEMIC AND NON-ISCHEMIC AETIOLOGY

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P7.11 HIGH ANKLE-BRACHIAL INDEX IS ASSOCIATED WITH INCREASED AORTIC PULSE WAVE VELOCITY: THE CZECH POST-MONICA STUDY

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Ankle brachial index (ABI) has increasingly been used in general practice to identify patients with low ABI at high cardiovascular risk. However there is no consensus on the clinical significance of high ABI. The aim of our study was to compare large artery stiffness as a marker of cardiovascular risk in patients with low (<1.0), normal (1.0-1.4) and high ABI (≥1.4).

Methods: 911 patients from the Czech post-MONICA study (a randomly selected 1% representative population sample, mean age 54±13.5 years, 47% of men) were examined. ABI was measured using a handheld Doppler and aortic pulse wave velocity (aPWV) using the Sphygmocor device.

Results: Of 911 patients, 28 (3.1%) had low ABI and 23 (2.5%) high ABI. There was a U-shaped association between aPWV and ABI. aPWV was significantly higher in patients with low and high ABI compared with normal ABI group (11.1±2.8, 8.3±2.3, p<0.001; 10.8±2.5, 8.3±2.3, p<0.001) and it did not differ between patients with high and low ABI (11.1±2.8, 10.8±2.5, p=0.86). In the stepwise multiple regression analysis low and high ABI were independent predictors of increased aPWV together with age, central systolic blood pressure, heart rate, BMI and hypertension.

Conclusion: This is the first study showing increased aortic PWV in patients with high ABI pointing to increased cardiovascular risk in this group.

P7.12 ARTERIAL STIFFNESS IN PATIENTS WITH HEART FAILURE OF ISCHEMIC AND NON-ISCHEMIC AETIOLOGY

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Increased arterial stiffness abnormal ventricular-vascular coupling have increasingly been recognized as playing an important pathophysiologic role in HF, both systolic and with normal ejection fraction. Aim: to assess and compare arterial stiffness, central blood pressure parameters in systolic and diastolic aortic hypertension, both systolic and with normal ejection fraction. Aim: to assess and compare arterial stiffness, central blood pressure parameters in systolic and diastolic aortic hypertension.

Methods: To investigate predictors of increase in aortic pulse wave velocity (aPWV) in patients with ischemic and non-ischemic aetiology groups.

Results: The prevalence of AA, AG and GG genotypes was 26.8%, 49% and 24%. Compared to AG subjects, AA and GG subjects had significantly higher levels of ET-1 (AG: 71.5±12.6 vs AA: 116.9±11.5 vs GG: 116.4±16.1 mmHg, p=NS), diastolic AP (PP: 70.5±11.2 vs AA: 71.7±9.2 vs GG: 71.1±12.4 mmHg, p=NS), systolic AP (AG: 103.9±12.6 vs AA: 105.1±11.3 vs GG: 105.9±16 mmHg, p=NS), diastolic AP (AG: 71.5±11.2 vs AA: 72.7±9.3 vs GG: 72.2±12.6 mmHg, p=NS). Conclusion: The -640A/G polymorphism of the p22phox promoter was typed by DraIII digestion of specific polymerase chain reaction products amplified from genomic DNA. The AA, AG and GG genotypes were determined. PP were measured by a sphygmomanometer; AOs were measured using a validated device. ET-1 levels were determined by ELISA. Comparisons were performed using the ANOVA for multiple comparisons followed by Bonferroni correction.

Conclusion: The prevalence of AA, AG and GG genotypes was 26.8%, 49% and 24%. Compared to AG subjects, AA and GG subjects had significantly higher levels of ET-1.

P7.13 THE P2ZPHOX -640A/G POLYMORPHISM OF NADPH OXIDASE ADVERSELY AFFECTS ENDOTHELIN LEVELS BUT NOT PERIPHERAL AORTIC PRESSURES IN HEALTHY, YOUNG, NORMOTENSIVE INDIVIDUALS

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Purpose: The NADPH oxidase system produces superoxide in the vessel wall and its -640A/G polymorphism is associated with coronary artery disease incidence in young individuals. We investigated the role of this polymorphism on peripheral/aortic pressures (PP, A0P), and endothelin-1 (ET-1) levels, in young normotensive individuals.

Methods: 153 healthy normotensives were studied (95 males, age 41 years). The -640A/G polymorphism in the p2zphox promoter was typed by DraIII digestion of specific polymerase chain reaction products amplified from genomic DNA. The AA, AG and GG genotypes were determined. PP were measured by a sphygmomanometer; AOs were measured using a validated device.

Conclusion: The -640A/G polymorphism of the p2zphox subunit of NADPH oxidase is associated with levels of ET-1, but neither with PP nor with A0P in young, normotensive individuals. Heterozygosity is associated with lower ET-1 levels.

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P8.01 PREDICTORS OF AORTIC STIFFENING IN ELDERLY SUBJECTS: RESULTS OF A NINE-YEAR FOLLOW-UP

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Objective: To investigate predictors of increase in aortic pulse wave velocity (aPWV) in elderly subjects free from overt cardiovascular disease.

Design and Method: The present study included 90 lecture attendees ("University of 3rd age") who were examined at baseline and after a median follow-up of 9.5 years, including the aPWV measurement using Sphygmocor. At baseline, they were aged 66.9±5.1 years, 80.0% were women, 37.8% of subjects had arterial hypertension, 5.6% diabetes mellitus, and 82.2% hyperlipidemia. We used multiple linear regression analyses to assess predictors of change in aPWV. As independent covariates we considered: sex, age, body mass index, mean arterial pressure (MAP), heart rate, fasting glucose, total cholesterol, smoking, alcohol intake and observer.

Results: The aPWV increased from 9.4 to 10.3 m/s; P = 0.022. While accounting for covariates, aPWV increased significantly with three factors: a 1--standard deviation change in heart rate (8.5 bpm), in MAP (12.4 mm Hg) and in fasting glucose (0.93 mmol/l) were associated with increased aPWV amounting to 0.76 m/s (95% CI: 0.23 to 1.30; P = 0.0061), 0.71 m/s (95% CI: 0.20 to 1.23; P = 0.0079) and 0.57 m/s (95% CI: 0.08 to 1.07; P = 0.024), respectively.

Conclusions: In elderly subjects without manifest cardiovascular disease, mechanical load, as demonstrated by the positive association with heart rate and MAP, plays a major role in the aortic stiffening. Among metabolic factors, glucose concentration but not lipid parameters is associated with increase in aortic stiffness, possibly via glycation of connective tissue within arterial wall.