P11.13: CORONARY CALCIFICATION IN YOUNG AND MIDDLE-AGED MEN WITH CORONARY ARTERY DISEASE

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**P11.09**  
**SHEAR STRAIN IN THE COMMON CAROTID ARTERIAL WALL RELATED TO AGE?**  
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The primary trigger for myocardial infarction and stroke is destabilization of atherosclerotic plaques. It has been hypothesized that locally increased longitudinal shear strain (LSS) facilitates the development of vulnerable plaques [1]. LSS is defined as the change of longitudinal deformation in radial direction. Ultrasound strain imaging allows local assessment of LSS [2].

In 8 asymptomatic volunteers (age: 20–64 yrs.), radiofrequency (rf) ultrasound data of the common carotid artery were acquired in longitudinal direction using a Medison Accuvix V10, equipped with an LS-13 linear array transducer (f0 = 8.5 MHz). In each volunteer rf data were acquired at three beam steering angles during multiple cardiac cycles [3]. Simultaneously the ECG-signal was recorded. LSS was estimated in a selected region-of-interest (ROI) using a coarse-to-fine cross correlation based algorithm [3,4].

The estimated shear strain showed a cyclic pattern with an increase during the systolic and a decrease during the diastolic phase. The maximum LSS ranged between 3 and 17% and appeared to decrease with age. This decrease could be related to stiffening of the arterial wall.

This study provides initial normal values of LSS in the common carotid artery. The maximum LSS appears to decrease with age. Further validation in patients will open the door for clinical predictions of plaque rupture.

**Background**: Systolic blood pressure (BP) is the main pressure determinant of left ventricular (LV) mass in hypertension. It is uncertain whether arterial stiffness and central hemodynamics are related to LV mass and geometry independently of brachial, central or 24-hour BP level.

**Methods**: 744 consecutive never-treated subjects with uncomplicated essential hypertension (men 59%, age 49±11 years, BP 149/93±16/10 mmHg) underwent M-mode echocardiography and 24-hour BP monitoring. Carotid-femoral pulse wave velocity (cfPWV), aortic augmentation and aortic BP were evaluated by applanation tonometry.

**Results**: Women with LV hypertrophy (LV mass >51 g/m²²) had a higher cfPWV (10.5±3 vs 9.3±2 m/s, p=0.001), augmentation (21.7±7 vs 16.7±7 mmHg, p<0.001) and heart rate-corrected augmentation index (37±8 vs 34±12, p=0.04). Similar data were found in men. LV relative wall thickness (RWT) but not LV mass index was significantly associated with cfPWV independent of age and brachial, central or 24h systolic BP (see Table). The association of aortic augmentation with LV mass and RWT was no longer significant after adjustment for age and systolic BP. In a multiple regression model, 24h systolic BP, LV mass and cfPWV (all p<0.05) independently predicted LV-RWT when a consistent number of risk factors was simultaneously controlled for.

**Conclusion**: The impact of aortic PWV on LV concentric geometry is independent and additional to that of peripheral, central or 24h BP.

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**P11.10**  
**PRESSURE-INDEPENDENT ASSOCIATION BETWEEN AORTIC STIFFNESS AND LEFT VENTRICULAR CONCENTRIC GEOMETRY**  
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**Conclusion**: The impact of aortic PWV on LV concentric geometry is independent and additional to that of peripheral, central or 24h BP.
Methods: A total of 50 men aged 28 to 50 (mean age: 43 ± 5) years with angiographically proven CAD underwent multislice computed tomography (MSCT) studies. Those with arterial hypertension, diabetes mellitus or marked hypercholesterolemia (LDL cholesterol >4.5 mmol/l) were excluded from the study. Coronary calcification was quantified using the Agatston score (CCS). The results were compared with a group of 30 controls without CAD, matched for sex, age, and risk factors.

Results: Calcifications were present in 86% of patients with CAD and in 16.7% of controls (p < 0.001). The mean (SD) CCS was 285 ± 314 in the CAD patients versus 31 ± 129 in the controls (p < 0.001). Absence of calcium, CCS of 1 to 99 (mild), 100 to 399 (moderate), and >400 (severe) was observed in 7 (15%), 16 (32%), 15 (30%), and 12 (24%) of the patients with CAD, and in 25 (84%), 3 (10%), 1 (3%), and 1 (3%) of the patients without CAD, respectively. Extent of coronary calcification in the CAD patients was not related to the severity of CAD.

Conclusion: In young and middle-aged patients with new onset CAD the presence and extent of coronary calcification is significantly greater than in matched controls.

P11.14
INFLUENCE OF SUBCLINICAL THYROID FAILURE ON ARTERIAL STIFFNESS IN WOMEN WITH ARTERIAL HYPERTENSION

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Background: The association between arterial stiffness increasing and mortality in patients with arterial hypertension (AH) is well recognized. There is ongoing debate whether subclinical thyroid failure may exert deleterious effects on the cardiovascular system with the consequences of increased morbidity and mortality. The OBJECTIVE of our study was to examine influence subclinical hypothyroidism (SCH) on arterial stiffness in women with arterial hypertension.

Methods: 40 females with AH (20 pts with normal thyroid function) and control 20 with SCH underwent brachial-ankle pulse wave velocity (PWVba) measurements for evaluation of arterial stiffness. Thyroid-stimulating hormone (TSH) level was 4.01-10.0 mIU/ml in SCH patients. Mean FT4 was respectively 1.4 ± 2.0 and 15.2 ± 3.5 μIU/ml in control and SCH group (p = 0.298).

Results: Hypothyroid patients demonstrated higher PWVba (15.0 ± 2.3 m/s, p = 0.016). There were no differences in age (65.1 ± 6.9 years vs 64.5 ± 6.7 years, p = 0.397) and systolic pressure (135.7 ± 19.0 vs 137.2 ± 18.2, p = 0.401) between SCH patients and controls.

Conclusions: Subclinical hypothyroidism is associated with changes in arterial stiffness. Significant changes of arterial stiffness were observed in subjects with TSH 4.01-10.0 mIU/ml suggesting that even early stage of thyroid failure is associated with increased cardiovascular risk.

P11.15
ANGIOTENSIN CONVERTING ENZYME -2 (ACE-2) POLYMORPHISMS RS4464615 AND RS44646174 ARE ASSOCIATED WITH CENTRAL PULSE PRESSURE, BRAIN NATRIURETIC PEPTIDE AND NYHA CLASSIFICATION IN PATIENTS WITH CHRONIC HEART FAILURE

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The pulse pressure (PP) is an independent predictor of cardiovascular morbidity and mortality. The PP level increases with the age and this increase is due to stiffness of large arteries. Angiotensin I converting enzyme (ACE) cleaves angiotensin I to angiotensin-1 (1-7) with vasodilatation and antiproliferative effects. In human model, the ACE-2 relation to the pulse pressure has not been studied yet. The aim of the study was to test possible association among phenotypes (central pulse pressure evaluated by invasive method in stable patients with well-preserved systolic function of the left ventricle, BNP as a marker of a severity of the disease and NYHA classification) and genotypes in two polymorphisms of ACE-2 gene.

Methods: A group of 312 patients with chronic heart failure (170 men and 142 women, median age 63 years) was enrolled in the study. The rs4464615 and rs44646174 polymorphisms in ACE-2 gene were detected by Tasman SNP genotyping.

Results: The central pulse pressure was highly significantly correlated with BNP, NT- proBNP and big endothelin levels. We observed significant differences in central pulse pressure among carriers of different genotypes of both ACE-2 polymorphisms (P<0.01 and 0.03, respectively). For the heterozygote genotype AT (rs44646156) a and CG (rs446461745) we report the higher risk for women in all NYHA groups compared to men (P<0.04-0.000001) with average sensitivity of 0.550 and specificity of 0.980.

Conclusion: The heterozygote genotypes in ACE-2 polymorphisms are more risky for women with chronic heart failure compared to men.

P11.16
BLOOD PRESSURE VARIABILITY IS ASSOCIATED WITH AORTIC STIFFNESS AND TROPNIN-T IN PATIENTS WITH CKD STAGES 3 AND 4

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Introduction: Clinic blood pressure predicts future cardiovascular risk but variability of systolic blood pressure (SBP) may be closely associated with adverse outcomes. Increased variability of SBP may be due to loss of buffering capacity in the aorta through increased vascular stiffness. This may be important in chronic kidney disease (CKD) where patients have accelerated aortic stiffening and high cardiovascular mortality. However, the relationship between aortic stiffness and BP variability in patients with CKD has not previously been studied.

Methods: 150 patients with CKD stages 3-4 in a prospective study of cardiovascular risk underwent 24-hour ambulatory blood pressure (24h-ABPM) and measurement of aortic pulse wave velocity (aPWV). Serum Troponin-T (TnT) was measured using a high-sensitivity assay.

Subject characteristics (n=150)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>69 ± 11</td>
</tr>
<tr>
<td>Male gender (%)</td>
<td>73</td>
</tr>
<tr>
<td>Systolic (mmHg)</td>
<td>150 ± 20</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>81 ± 11</td>
</tr>
<tr>
<td>Diabetic (%)</td>
<td>22</td>
</tr>
<tr>
<td>Cardiovascular disease (%)</td>
<td>39</td>
</tr>
<tr>
<td>eGFR (mL/min/1.73m²)</td>
<td>32 ± 11</td>
</tr>
</tbody>
</table>

Results: Standard deviation of 24h-ABPM SBP was associated with age (rho = 0.28, p < 0.001), clinic SBP (rho = 0.20, p = 0.01), heart rate (rho = 0.30, p < 0.001), aPWV, (rho = 0.22, p < 0.01) and TnT (rho = 0.26, p < 0.001). SD 24h-ABPM was higher among diabetics (16 ± 18 mmHg, p = 0.05) and people with previous cardiovascular disease (15 ± 18 mmHg, p = 0.001). There was no significant difference in SD of 24h-ABPM SBP between people treated with different antihypertensive drugs or total number of antihypertensives taken. In stepwise multivariable analysis, factors independently associated with SD of 24h-ABPM SBP were heart rate, cardiovascular disease and TnT (R² = 0.18, p < 0.001).

Conclusion: Aortic stiffness is associated with blood pressure variability. However, this may be due to the association of cardiovascular disease with aortic stiffness rather than a causal mechanism.

P11.17
CALCIFICATION OF THE THORACIC AORTA ON CHEST X-RAY — ASSOCIATIONS WITH ABDOMINAL AORTIC CALCIFICATION AND PULSE WAVE VELOCITY IN PATIENTS ON RENAL REPLACEMENT THERAPY

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Background: Abdominal aortic calcification (AAC) on lateral abdominal x-ray and carotid-femoral pulse wave velocity (PWV) are independent predictors of mortality and non-fatal CV events in patients on renal replacement therapy (RRT). Guidelines suggest that the presence of AAC can be used to identify patients at high risk. In this study aortic arch calcification (AoAC)