P1.41: THE CYP2J2 G-50T POLYMORPHISM AND MYOCARDIAL INFARCTION IN PATIENTS WITH CARDIOVASCULAR RISK PROFILE

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Results: The maximum progression of IH was observed at 3 months (1.4±0.4 mm). Afterwards progression IH decreased and stabilized at 12 months. Mean shear stress at bifurcation after CEA was 16.6±2.3 dynes/cm² and was inversely related to internal diameter of common carotid artery at reconstruction site. Low mean shear stress correlated with low flow volume ($r = 0.56$, $P < 0.0001$). IH was inversely associated with shear stress ($r = 0.37$, $P < 0.0001$) and flow volume ($r = 0.35$, $P < 0.0001$).

Conclusions: This study demonstrates that low shear stress could lead to progression of IH after CEA. Strong correlation between flow volume and shear stress supports the conclusion that reduced flow volume can cause intimal hyperplasia. Artificial increase in artery's diameter, especially with patch, leads to the development of intimal hyperplasia through decrease of shear stress.

P1.41
ASSOCIATION BETWEEN AORTIC DILATATION, ARTERIAL STIFFNESS AND CARDIAC ORGAN DAMAGE IN ESSENTIAL HYPERTENSION

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Objectives of this study were to evaluate the prevalence of proximal ascending aortic dilatation (pAAD) in essential hypertensives and the association between pAA ectasia, arterial stiffness and cardiac organ damage.

Background: There are few data in literature concerning pAAD in arterial hypertension. It is not known whether pAAD may be related to increased cardiac organ damage and what the relation with central hemodynamics and arterial stiffness would be in essential hypertension.

Methods: We measured in 345 treated and untreated essential hypertensives (54.3±11 years) clinic blood pressures, central hemodynamics through radial tonometry and proximal aortic diameters using transthoracic echocardiography. BSA-normalized diameter cut off for aortic ectasia definition was 2.1 cm/m².

Results: Overall prevalence of pAA dilatation was 17% in our population. We observed a slightly increase of central systolic (129.8±15.4 vs. 125.0±2.14.7 p 0.02) and pulse pressure (45.02±10.4 vs. 42.±9.54; p 0.02) in patients with pAAD, whereas peripheral haemodynamic parameters were similar. Pulse wave velocity was significantly greater (9.26±2.33 vs. 7.70±1.69 p < 0.0001), as well as the augmentation index (25.86±10.2 vs. 19.41±9.52; p<0.0001) in patients with pAAD, and this difference maintained after correction for age. Left ventricular hypertrophy was thrice as frequent (32.8% vs. 13.4% p<0.0001) compared to hypertensive patients without pAA dilatation.

Conclusions: This study shows a high prevalence (17%) of ascending aorta dilatation in patients affected by essential hypertension, without further complications. Dilatation of the ascending aorta is associated to increased cardiac organ damage and arterial stiffness.

P1.42
THE ARTERIAL STIFFNESS, FLOW-MEDIANED VASODILATION OF THE BRACHIAL ARTERY, AND THE THICKNESS OF THE CAROTID ARTERY INTIMA-MEDIA IN PATIENTS WITH METABOLIC SYNDROME

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Background: patients with metabolic syndrome have increased vascular events risk. New imaging techniques are necessary for the early assessment and management of these patients.

Aim of study: compare aortic stiffness index ($\beta$) and as an indicator of arterial stiffness, to brachial arterial flow-mediated vasodilation (FMD) and common carotid artery intima-media thickness (IMT), because they are standard indicators of endothelial dysfunction and atherosclerosis.

Methods: patients with metabolic syndrome signs (ATP criteria III, 2005) were included, as well as 48 healthy individuals were investigated. Arterial stiffness was assessed by echocardiography. Brachial arterial FMD and IMT were determined using high-resolution ultrasonography. All metabolic patients exerted impaired FMD (3.9 ± 0.81), increased IMT (0.86 ± 0.12 mm), $\beta$ (7.8 ± 1.2) in comparison to control subjects (FMD = 8.4 ± 1.1 mm; IMT = 0.69 ± 0.1 mm; $\beta$ (3.2 ± 1.0) (p < 0.05). The significant negative correlation of FMD with $\beta$ (R = -0.69; p < 0.001) was found. There was significant positive correlation between IMT and $\beta$ (R = 0.48; p = 0.004). $\beta$, IMT positively correlated and FMD negatively correlated with the age of the metabolic patients. Arterial stiffness indicated by increased $\beta$ index is associated with endothelial dysfunction and overt atherosclerosis in patients with metabolic syndrome. Assessment of arterial stiffness, FMD, and IMT are reproducible and reliable noninvasive techniques for the complex assessment of vascular abnormalities in metabolic patients. This data suggest, that these techniques may be used as a predictors of cardiovascular risk events.

P1.43
INTERACTION OF HYPERALBUMINURIA, ARTERIOSCLEROTIC PLAQUES AND ELEVATED PULSE WAVE VELOCITY WITH AGE AND RISK CATEGORY IN APPARENTLY HEALTHY SUBJECTS

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Subclinical vascular damage (SVD) predicts cardiovascular events independently of traditional risk factors in apparently healthy subjects. But information on coexistence and additive prognostic importance in different age and groups are lacking. In 2082 apparently healthy subjects aged 41, 51, 61 and 71 years we estimated cardiovascular risk using SCORE, performed ultrasonography of the carotid arteries and measured urine albumin/creatinine ratio (UACR) and pulse wave velocity (PWV) in 1993. The composite endpoint (CEP) of cardiovascular death, non-fatal myocardial infarction and stroke, elevated PWV increased. The prevalence of subjects with one or more SVD increased between the four age groups as well as the four SCORE risk groups. Between the four age groups and the four SCORE risk groups, the prevalence of hyperalbuminuria (above 90 percentile), atherosclerotic plaques and elevated PWV increased. The prevalence of subjects with one or more SVD increased between the four age groups as well as the four risk groups. Increasing numbers of markers for SVD (0, 1, 2 or 3) was associated with higher incidence of CEP in the three youngest age groups (41±51 years: 4.7, 9.4, 20.8, 50%; 61 years: 7.7, 15.3, 25.9, 35.7%, both P < 0.001; 71 years: 21.2, 21.5, 27.9, 43.3%, P = 0.08) and in patients with SCORE<10% (SCORE<5%: 4.6, 9.2, 20.6, 60%, P<0.001; S<SCORE<10%: 9.8, 18.4, 20.8, 36.8%, P<0.05; SCORE>10%: 25.0, 23.5, 35.4, 42.3%, NS). Concluding