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P1.40: ASCENDING AORTIC DILATATION, ARTERIAL STIFFNESS AND CARDIAC ORGAN DAMAGE IN ESSENTIAL HYPERTENSION

F. Tosello, A. Milan, G. Bruno, I. Losano, M. Chiarlo, F. Veglio

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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.

P1.40

ASCENDING AORTIC DILATATION, ARTERIAL STIFFNESS AND CARDIAC ORGAN DAMAGE IN ESSENTIAL HYPERTENSION

F. Tosello, A. Milan, G. Bruno, I. Losano, M. Chiarlo, F. Veglio
University of Turin, Turin, Italy

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 (129.81 ± 15.4 vs. 125.02 ± 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.

P1.41

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

R. Tzveova^{1,2}, G. Naydenova³, M. Tzekova³, R. Kaneva^{1,2}, V. Mitev^{1,2}

¹Molecular Medicine Centre, Medical University, Sofia, Bulgaria

²Departments of Medical Chemistry and Biochemistry, Medical University, Sofia, Bulgaria

³Second Department of Cardiology, UMBAL "Dr. G. Stranski", Pleven, Bulgaria

CYP2J2 is responsible for the production of 5, 6, 8, 9, 11, 12, 14 and 15-epoxyeicosatrienoic acids, vasodilator and anti-inflammatory substances. It is abundantly expressed in human heart and also present in kidney and vasculature. Carriers of a common polymorphism, the CYP2J2 -50G>T, have reduced expression of CYP2J2 mRNA in the heart.

We conducted a population-based, case-control study to determine whether common genetic variation in CYP2J2 gene was associated with the risk of acute myocardial infarction (AMI).

We analyzed 101 patients with AMI and 377 controls for a potential correlation of the CYP2J2 polymorphism G-50T with a history of myocardial infarction. To evaluate the genotypes of the samples real time PCR with pre-designed TaqMan SNP Genotyping Assays (Applied Biosystem) was used.

The allelic frequencies of CYP2J2*1 and CYP2J2*7 variants were 0.90 and 0.10 in the control group and 0.84 and 0.16 in the affected group, respectively.

Comparison of genotype and allele frequencies between patients and controls in the study of promoter located SNP CYP2J2*7 did not provide a statistically significant association with AMI.

Our study found no association of the polymorphism in CYP2J2 with the development of AMI.

P1.42

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

V. Vikentiev, I. Goncharov, N. Smetneva

State Medicostomatological Institute, Moscow, Moscow, Russian Federation

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 (β) 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 ($3.9 \pm 0.8\%$), increased IMT (0.86 ± 0.12 mm), β (7.8 ± 1.2) in comparison to control subjects (FMD = $8.4 \pm 1.1\%$; IMT = 0.69 ± 0.1 mm; β (3.2 ± 1.0) (p < 0.05). The significant negative correlation of FMD with β ($R = -0.69$; p < 0.001) was found. There was significant positive correlation between IMT and β ($R = 0.48$; p = 0.004). β , IMT positively correlated and FMD negatively correlated with the age of the metabolic patients. Arterial stiffness indicated by increased β 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 this techniques may be used as a predictors of cardiovascular risk events.

P1.43

INTERACTION OF HYPERALBUMINURIA, ARTEROSCLEROTIC PLAQUES AND ELEVATED PULSE WAVE VELOCITY WITH AGE AND RISK CATEGORY IN APPARENTLY HEALTHY SUBJECTS

S. V. Greve¹, M. Blicher², A. Blyme³, T. Sehestedt⁴, T. W. Hansen⁵, S. Rasmussen⁶, J. Vishram⁷, H. Ibsen⁸, C. Torp-Pedersen⁹, M. H. Olsen¹⁰

¹Cardiovascular and Metabolic Preventive Clinic, Department of Endocrinology, Odense University Hospital, Odense, Denmark

²Cardiovascular and Metabolic Preventive Clinic, Department of Endocrinology, Odense University Hospital, Odense, Denmark

³Cardiovascular Research Unit, Department of Internal Medicine, Glostrup University Hospital, Glostrup, Denmark

⁴Department of Cardiology, Herlev University Hospital, Herlev, Denmark

⁵Department of Clinical Physiology and Nuclear Medicine, Rigshospitalet, Copenhagen, Denmark

⁶Department of Diagnostic Imaging, Gentofte University Hospital, Gentofte, Denmark

⁷Research Center for Prevention and Health, Copenhagen, Denmark

⁸Department of Internal Medicine, Holbæk Hospital, Holbæk, Denmark

⁹Department of Cardiology, Gentofte University Hospital, Gentofte, Denmark

¹⁰Cardiovascular and Metabolic Preventive Clinic, Department of Endocrinology, Odense University Hospital, Odense, Denmark

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 ultrasound 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, and hospitalization for ischemic heart disease was recorded until 2006. 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%, $P < 0.001$; 5 ≤ 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