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P1.39: LOW SHEAR STRESS INDUCES PROGRESSION OF INTIMAL HYPERPLASIA AFTER CAROTID ENDARTERECTOMY

O. Tereshina, E. Surkova

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the relationship of structural and functional markers of large arteries with blood pressure variability.

Methods: Our study sample consisted of 1125 randomly selected participants (mean age: 42.4 years; 49.3% women). One trained researcher conducted vascular measurements, including carotid intima-media thickness (IMT), carotid distensibility and carotid—femoral pulse wave velocity (PWV). Systolic blood pressure (SBP) was measured 5 times consecutively. We assessed within-subject variability independent of the mean (VIM).

Results: SBP and VIM averaged 127.9 mm Hg and 3.86 units, while the vascular measurements averaged 0.70 mm, 6.62 m/s and $24.7 \cdot 10^{-3}$ /kPa for IMT, PWV and carotid distensibility, respectively.

In single regression, IMT correlated with SBP (r=0.27, p<0.0001) and VIM (r=0.22; p<0.0001). Similarly, PWV and the carotid distensibility correlated with both SBP (PWV, r=0.34; p<0.0001; carotid distensibility, r=-0.32, p<0.0001) and VIM; however, the associations with VIM were weaker (PWV, r=0.07; p=0.025; carotid distensibility, r=-0.09, p=0.003). After adjusting for sex and age, and other covariables, the associations of IMT with SBP (β =0.029, p<0.0001) and VIM (β =0.036, p<0.0001) remained. However, those between PWV (β =0.044, p=0.45), carotid distensibility (β =-0.53, p=0.28) and VIM disappeared. PWV (β =0.353, p<0.0001) and carotid distensibility (β =-2.92, p<0.0001) remained associated with SBP.

Conclusion: In the FLEMENGHO cohort, blood pressure variability relates to carotid IMT, but not the indexes of arterial stiffness.

P1.36

ARTERIAL STIFFNESS AND MARKERS OF OXIDATIVE STRESS HAPTOGLOBIN AND HOMOCYSTEINE IN NEVER TREATED INCIDENT HYPERTENSIVE PATIENTS

C. Strandhave ^{1,4}, M. Svensson ², H. Krarup ³, J. H. Christensen ^{1,4} ¹Dept. of Nephrology, Aalborg Hospital, Aarhus University Hospital, Aalborg, Denmark

²Dept. of Nephrology, Aarhus University Hospital, Aarhus, Denmark ³Dept. of Clinical Biochemistry, Aalborg Hospital, Aarhus University Hospital, Aalborg, Denmark

⁴Cardiovascular Research Centre, Aalborg Hospital, Aarhus University Hospital, Aalborg, Denmark

Introduction: Hypertension is a major risk factor for development of cardio-vascular disease (CVD). Arterial stiffness is a risk predictor of CVD in this patient group. Oxidative stress has been implicated in the development of arterial stiffness. Haptoglobin (Hp) and homocysteine (Hcy) are well documented markers of oxidative stress. We hypothesized that Hp and Hcy are associated with arterial stiffness in never treated incident hypertensive patients.

Methods and Material: We examined 125 patients with newly diagnosed never treated hypertension. Patients had 24h ambulatory blood pressure (BP) measurement (Spacelabs Healthcare®, Hertford, UK) performed and were eligible if daytime BP was $\geq 135/85$ mmHg. Arterial stiffness expressed as carotid-femoral pulse wave velocity (cfPWV), augmentation index at heart rate 75 (Alx@75), and central systolic blood pressure (cenBP) were measured with the SphygmoCor® device (Atcor Medical®, Sydney, Australia). Hp and Hcy were determined using rate nephelometry (Beckmann IMMAGETM, DK).

Results: Patient baseline characteristics are shown in the Table. In a univariate correlation Hp showed a weak association with Alx@75 (r=0.16, p=0.04) which in an adjusted regression model remained significant ($r^2=0.45$, p=0.02). Hp was not associated with cfPWV or cenBP nor was there any association between Hcy and cfPWV, Alx@75, and cenBP.

Conclusion: Only Hp was associated with arterial stiffness (Alx@75) whereas no other associations were found. Thus, Hcy appear not to be associated with arterial stiffness in hypertension and further studies are warranted to elucidate the role of Hp as a risk marker in hypertension.

P1.37

RISK FACTORS FOR INTIMAL HYPERPLASIA DEVELOPMENT AFTER CAROTID ENDARTERECTOMY

Elena Surkova, Olga Tereshina Samara Medical State University, Samara, Russian Federation

Background: Intimal hyperplasia is a common complication occurring after carotid endarterectomy (CEA), although pathological mechanisms leading to its progression are not totally clear.

Methods: In our study we assessed a role of various factors including age, gender, hypertension, history of smoking, hyperlipidaemia, diabetes mellitus, lesions characteristics, type of arteriotomy closure and regional flow dynamics affecting intima-media thickness (IMT) past CEA. We examined 1003 patients prior to and 1 year after carotid endarterectomy using standardized duplex ultrasound examination. The median age of patients was 60 ± 15 years. Univariate and multivariate regression analysis was used to identify major risk factors and possible correlations between them.

Results: Only 2 of systemic factors - elevated level of low-density lipoprotein and platelet aggregation were found to have a minor (although statistically significant) effect on IMT after CEA. Age, gender, hypertension, smoking were not significantly associated with the development of intimal hyperplasia past CEA. Complicated plaques showed a significant correlation with intimal hyperplasia. There were no significant differences in IMT between patients who have had eversion CEA versus CEA with patch. Of local factors, only sheer stress demonstrated a negative correlation with IMT.

Conclusions: Our results demonstrate that multiple risk factors could be involved in the development of intimal thickening after CEA.

P1.38

RELATIONSHIP BETWEEN ANKLE BRACHIAL INDEX AND OTHER INDICES OF TARGET ORGAN DAMAGE IN HYPERTENSIVE PATIENTS

A. Szyndler ¹, T. Fabiszewska ¹, R. Nowak ¹, K. Czechowicz ¹, A. Dubiela ¹, K. Polonis ¹, W. Kucharska ¹, A. Rojek ¹, M. Hoffmann ¹, P. Boutouyrie ², S. Laurent ², K. Narkiewicz ¹

¹Department of Hypertension and Diabetology, Medical University of Gdansk, Gdansk, Poland

²Department of Pharmacology, HEGP, APHP, INSERM U970, Université Paris Descartes, Paris, France

Objective: Assessment of ankle brachial index (ABI) is a method of peripheral artery disease diagnosis. Its role has been established as a predictor of cardiovascular events. The role of ABI in the hypertensive patients without cardiovascular has not been studied. The aim of study was assessment of the relationship between ABI, target organ damage and ambulatory blood pressure in hypertensives.

Methods: 355 hypertensive patients (199 males, 56%) without history of renal or cardiovascular disease were examined. The ambulatory blood pressure (Spacelabs 90207), echocardiography (Vivid 7 Pro™), carotid intima-media thickness (IMT, ArtLab system), carotid-femoral pulse wave velocity (PWV) and central blood pressure (Sphygmocor) were measured. Ankle brachial index (blind Doppler), left ventricular mass index, and estimated glomerular filtration rate (eGFR, MDRD formula) were calculated.

Results: In the population ABI (mean 1.05 ± 0.11) was weakly related to central pulse pressure (r=-0.12; p=0.02), but not to central SBP (r=0.04; p= NS). ABI was not linked to 24-h SBP (r=0.03; p= NS), 24-h pulse pressure (r=0.08, p= NS), LVMI (r=0.01; p= NS, or eGFR (r=0.10; p= NS). There was no correlation between ABI and other indices of arterial wall properties: IMT (r=0.06; p= NS) or PWV (r=0.03; p= NS).

Conclusion: ABI is weakly related to central pulse pressure, but not to ambulatory or central blood pressure. ABI is not linked to other markers of cardiac and vascular damage in hypertensives without cardiovascular disease.

P1.39

LOW SHEAR STRESS INDUCES PROGRESSION OF INTIMAL HYPERPLASIA AFTER CAROTID ENDARTERECTOMY

O. Tereshina, E. Surkova

Samara Medical State University, Samara, Russian Federation

Background: The progression of intimal hyperplasia after carotid endarterectomy (CEA) is influenced by local hemodynamic factors. In our study we investigated associations between flow volume, velocity, shear stress and the intimal hyperplasia (IH) of the common carotid artery after CAE.

Methods: 1009 patients (60 ± 15 years, 86% of which were male) who had had CEA between 2001 and 2011 were included in our study. Blood velocity, internal diameter, flow volume and intima-media thickness were measured using standardized duplex ultrasound examination at 3, 6, 9 and 12 months after operation. Shear stress was calculated as blood viscosity multiplied by blood velocity divided by internal diameter.

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Results: The maximum progression of IH was observed at 3 months (1,4 \pm 0,4 mm). Afterwards progression IH decreased and stabilized at 12 months. Mean shear stress at bifurcation after CEA was 16,6 \pm 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=0,5 δ 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 \pm 15.4 vs. 125.02 \pm 14.7 p 0.02) and pulse pressure (45.02 \pm 10.4 vs. 42 \pm 9.54; p 0.02) in patients with pAAD, whereas peripheral haemodynamic parameters were similar. Pulse wave velocity was significantly greater (9.26 \pm 2.33 vs. 7.70 \pm 1.69 p<0.0001), as well as the augmentation index (25.86 \pm 10.2 vs. 19.41 \pm 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. Naydenova ³, M. Tzekova ³, 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 predesigned 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 0.8%), increased IMT (0.86 +0.12 mm), β (7.8 +1.2) in comparison to control subjects (FMD = 8.4 + 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 $\boldsymbol{\beta}$ 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 date suggest, that this techniques may be used as a predictors of cardiovascular risk events.

P1.43

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

S. V. Greve ¹, M. Blicher ², A. Blyme ³, T. Sehestedt ⁴, T. W. Hansen ⁵, S. Rasmussen ⁶, J. Vishram ⁷, H. Ibsen ⁸, C. Torp-Pedersen ⁹, M. H. Olsen ¹⁰ 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 Cardiology, Heritev University Hospital, Heritev, Denimark 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 \geq 10%: 25.0, 23.5, 35.4, 42.3%, NS). Concluding