P4.27: AUTONOMIC NERVOUS SYSTEM REACTIVITY IN NORMOTENSIVE SUBJECTS WITH A FAMILY HISTORY OF HYPERTENSION DURING VALSALVA MANOEUVRE

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Endothelial dysfunction is an independent risk factor for cardiovascular events. Inflammatory mediators released by the adipose tissue can lead to local insulin resistance and endothelial dysfunction. This study addressed the relationship of adipocytokines with endothelial function and blood pressure. In 92 newly diagnosed, drug-naive essential hypertensive patients (HT) without organ damage and 66 normotensive subjects (NT), by an automated system, we measured endothelium-dependent and -independent vasodilation as brachial artery flow mediated dilation (FMD) before and after administration of glyceryl-trinitrate (GTN). Retinol binding protein-4 (RBP4) and resistin levels were determined by ELISA and RIA, respectively. Oxidative stress was evaluated by measuring serum malondialdehyde (MDA).

FMD was significantly (p = 0.03) lower in HT (5.3 ± 2.6%) than NT (6.1 ± 3.1%), while response to GTN (7.5 ± 3.7% vs 7.9 ± 3.4%) was similar. RBP4 (60.6 ± 25.1 vs 61.3 ± 25.9 μg/ml), resistin (18.8 ± 5.3 vs 19.9 ± 6.1 ng/ml) and MDA levels (2.93 ± 1.62 vs 2.08 ± 1.17 mmol/l) were not different in HT and NT. RBP4 (r = -0.25; p = 0.04) and resistin levels (r = -0.29; p = 0.03) were related to FMD in NT, but not in HT (r = -0.03 and r = -0.10, respectively). In NT multivariate analysis, including RBP4 and confounders showed that only body mass index (BMI) or waist circumference remained related to FMD. In the multivariate model including resistin and confounders, BMI, age and resistin significantly related to FMD when BMI was replaced by waist circumference.

In conclusion, adipocytokine levels, particularly resistin, are independent predictors of endothelial dysfunction in the peripheral circulation of healthy subjects, providing a pathophysiological link between inflammation from adipose tissue and early vascular alterations.
ejection time (298.9 ± 4.9 ms vs. 316.8 ± 4.5 ms) (t-test, p < 0.05). In addition, the normotensives with a family history of hypertension had decreased latency of the baroreflex response (7.0 ± 0.5 s) compared to the normotensives group (10.5 ± 0.9 s) (p < 0.001).

Conclusions: Our results indicate that even normotensives with a family history of hypertension exhibit changes of some cardiovascular parameters at early age. The changes in Valsalva manoeuvre response also show alteration of the autonomic nervous system reactivity.

P4.28 IMPACT OF WEIGHT CHANGE ON INTIMA MEDIA THICKNESS OF CAROTID ARTERIES AND ENDOTHELIAL FUNCTION IN OBESO AND OVERWEIGHT HYPERTENSIVE SUBJECTS

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Objectives: Taking into consideration that obesity and AH are the most important related risk-factors of CVD we examined differences in carotid artery intima-media thickness (IMT) and endothelial function between obese and overweight hypertensive individuals.

Methods: We studied 102 patients with mild to moderate AH (67males/ 35females, mean age 51 ± 2, 4 years, BMI 30.9 ± 1.9 kg/m², duration of AH 4.6 ± 1.4 years). Examination included: color triplex carotid artery scanning; assessment of endothelial function of brachial artery; 24-hour BP monitoring. 49 overweight patients (25- BMI < 29.9 kg/m²) were assigned to group 1 and 53 obese patients (BMI > 30 kg/m²) to group 2.

Results: The groups were comparable by the age, duration of AH, daily mean BP values. Mean values of IMT (gr1: 0.02 ± 0.03 mm; gr2: 0.18 ± 0.04 mm) were certainly increased in obese patients compared with overweight ones (p < 0.001). Prevalence of carotid atherosclerosis was higher in gr2 (7% vs 67%). Endothelium - dependent vasodilatation (EDVD) (gr1: 7.6 ± 4.5 mm vs. 7.0 ± 4.3 mm) was significantly reduced in obese patients (p < 0.01), but occurrence of endothelial dysfunction was almost equal (gr1: 59%; gr2: 60%). BMI positively correlated with IMT (r = 0.25, p < 0.02) and negatively with EDVD (r = -0.4, p < 0.05).

Conclusions: Thus, in obese hypertensive subjects we detected more pronounced and frequent carotid artery affection and endothelial dysfunction compared with overweight ones. Data of our study demonstrate importance more profound examination of cardiovascular system in obese hypertensive patients with subsequent more aggressive blood pressure and weight reduction.

P4.29 LEFT ATRIUM REMODELLING IS AN EARLY CARDIAC STRUCTURAL CHANGE IN HYPERTENSION

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Background: The interest in left atrial remodelling (LAR) as a TOD in hypertension (H) has been growing recently. Little is known on the role of arterial stiffness (a.s) in the pathophysiology of LAR in H. We hypothesized that LAR precedes LVH and diastolic dysfunction (d.d.) in H and is associated with carotid a.s., independently of other possible confounders.

Methods: 85 patients—65 with H, 31 male and 34 female, mean age 55 ± 10.7 years and 20 control matched subjects(C). From echocardiography: left atrial volume normalized to BSA (LA vol/BSA), arterial stiffness (PWV), PW, IVS; from conventional and Tissue Doppler: early(E), late(A) mitral flow velocities, E/A ratio, early(E) / late(A) mitral annular velocities, e'/a' ratio; E/e' ratio were calculated. From carotid arteries ultrasound—IMT and high-resolution echo-tracking method a.s. parameters. Data were examined: [stiffness index, Ep-elastic modulus, AC-arterial compliance, PWV]-one point pulse wave velocity.

Results: LAVol/BSA was the highest in H with LVH(24.9±1.6,1.1) in H and in d.d.(23.5±1.6). However, already in H without LVH, LA vol/BSA was significantly higher than in C (21.1±4.9, P<0.05) and also in H without d.d. LA vol/BSA was significantly higher than in C (20.5±5.5, 5vs18.3±4.8, P<0.048). Linear regression analysis revealed the following significant correlations between LA vol/BSA and age(r=0.3), BMI(r=0.38), mean BP(r=0.25), preload(r=0.27), afterload(r=0.24), LVM(r=0.59), RWT(r=0.23), IVS(r=0.5), PW(r=0.42), e’(r=-0.3), E/e’(r=0.46), BNP(r=0.73), Ep(r=0.25) and PWV(r=0.25);p for all <0.05. However in multiple regression analysis the independent determinants were: age,BMI,mean BP,LVM,PE/e’ and PWV. Conclusion: LAR is one of the earliest cardiac structural changes in H that precedes LVH and d.d.Local PWV is an independent determinant of LAR beyond:BP components, LVH, d.d.indices. It supports the hypothesis on the contribution of arterial stiffness to LAR.

P4.30 ALCOHOL EXERTS A SHIFTED U-SHAPED EFFECT ON CENTRAL AND PERIPHERAL BLOOD PRESSURE IN YOUNG ADULTS

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Consumption of 1-2 alcoholic beverages daily has been associated with a lower risk of cardiovascular disease and all-cause mortality in middle-aged and older adults. Recent studies suggest that central blood pressure (BP) is a better predictor of cardiovascular risk than peripheral BP. However, potential effects of habitual alcohol consumption on central BP particularly in young adults, the primary consumers of alcohol in North America, have yet to be investigated. Therefore, we aimed to study the effect of alcohol consumption on central and peripheral BP, pulse pressure amplification, and arterial stiffness specifically in young adults. We recruited 130 healthy, non-smoking, non-obese individuals. Using a standardized questionnaire, alcohol consumption (drinks/week) was used to classify participants into non-, (-2), light (2-6), moderate (women: 7-9, men: 7-14), and heavy drinkers (women: >9, men: >14). Central BP and arterial stiffness measurements were obtained using applanation tonometry. We found a U-shaped effect of alcohol consumption on both central and peripheral BP. Light drinkers had significantly lower central and peripheral systolic, and mean arterial BPs when compared to non- and moderate drinkers (P<0.05). No significant associations with arterial stiffness parameters were noted. A U-shaped relationship was found between alcohol consumption and both central and peripheral BP in young individuals, which importantly, was shifted towards lower levels of alcohol consumption than currently suggested. This is the first study, to our knowledge, that examines the effect of alcohol consumption on central BP and arterial stiffness exclusively in young individuals. Prospective studies are needed to confirm the relationships observed herein.

P4.31 ALTERED THROMBIN GENERATION IN SUBJECTS WITH FAMILIAL HYPERCHOLESTEROLEMIA

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Purpose: The effects of angiotensin II (ANG) on inflammation and haemostasis were examined in 16 overweight healthy patients with familial hypercholesterolemia (FH) and in 16 healthy controls.

Methods: Plasma markers of inflammation (hs-CRP, IL-6, fibrinogen, leukocyte counts (Lct), coagulation (thrombin generation: F1+2, Calibrated Automated Thrombogram (CAT), fibrinolysis (plasmin-antiplasmin complexes, PAI-1 activity) were assessed in conjunction to iv ANG infusion (10 ng/kg/min for 3 h). Means ± SD; repeated measures ANOVA, log transformation when appropriate.

Results: Baseline systolic blood pressure was higher in FH than in controls (127±14 vs 115±12 mm Hg, p<0.05), while responses to ANG were similar (+21±10 and +21±7 mm Hg). Baseline hs-CRP, IL-6, Lct, and fibrinogen were similar in FH and controls, and all increased similarly in both groups (p<0.05) during ANG. Baseline CAT (peak and ETP) was higher in FH (367±47 vs 317±60 nM, p<0.01, and 2148±391 vs 2042±358 nM/min, p<0.01, respectively), but ANG did not affect CAT (peak or ETP). Baseline PAI-1 activity was higher in FH and controls (96±16 vs 93±27 µg/L) and increased (p<0.001) similarly by ANG in both groups. PAI-1 activity was similar in both groups at baseline (1.3±1.3 vs 1.1±1.2 ng/L) and decreased (p<0.001) similarly in both groups, confirming the diurnal variation in fibrinolysis.

Abstracts