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P13.05: IS AIX INCREASED IN COPD?

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184 Abstracts

P13.03

THE PATIENTS WITH LACUNAR ISCHEMIC STROKE (LIS) HAVE ENDOTHELIAL DYSFUNCTION (ED) AND INCREASED ARTERIAL STIFFNESS

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ED is thought to be implicated in the pathogenesis of cerebral small vessel disease in patients with LIS while the role of arterial stiffness is less known. **Objective:** The aim of this study was to assess the endothelial function (EF) and arterial stiffness in patients with LIS.

Methods: In this study, 35 patients with LIS, as defined by clinical characteristics and MRI findings were compared with 18 healthy age and gendermatched patients with similar risk factors.

EF was assessed using the brachial flow-mediated vasodilatation (FMD); in LIS group on the first day after stroke onset. Carotid stiffness index ß was calculated as follows: ln(systolic/diastolic blood pressure)/[[Dmax-Dmin]/Dmin), where Dmax and Dmin are maximum and minimum common carotid lumen diameters measured by carotid ultrasound. Carotid intima-media thickness (IMT) was also measured. FMD was categorized according to ROC analysis and ED was defined as FMD < 6.0%.

Results: Twenty-two (63%) LIS patients had ED on the 1st day and only three patients (17%) in the control group (p<0.05). FMD was lower in LIS group (6.1 \pm 2.5% versus 9.8 \pm 2.2% in the control group, p=0.02), while carotid stiffness was higher (10.5 \pm 2.2 versus 7.5 \pm 2.6, p=0.04). There was not significant difference in IMT between two groups (0.91 \pm 0.17 versus 0.87 \pm 0.21, p=0.4). Moderate correlation was observed between stiffness and IMT (r=0.33, p=0.01). No correlations were found between FMD and IMT. FMD and arterial stiffness.

Conclusion: The majority of patients with LIS have the impairment of the structural-and-functional properties of the arterial wall that manifest in the ED and increased stiffness.

P13.04

EARLY POSTPRANDIAL INCREASE IN BLOOD GLUCOSE DOUBLES FLOW MEDIATED DILATION IN YOUNG HEALTHY SUBJECTS

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Background: In healthy subjects insulin induces microcirculatory vasodilation by stimulating endothelial nitric oxide release (Fig. A). Whether insulin would modulate large artery flow mediated dilation (FMD) in the early post-prandial phase has not been investigated, yet.

Methods: In six healthy volunteers (age 28 ± 3 yrs, BMI 26 ± 5 kg/m^2), we measured blood glucose and brachial artery FMD during fasting conditions and 30 minutes after consumption of a 75g glucose solution. FMD with reactive hyperemia was obtained by simultaneous Doppler/B-mode echo and beat-to-beat video analysis, providing smoothed velocity and diameter curves (Fig. B).

Results: Blood glucose increased from 4.5 ± 0.2 to 7.5 ± 0.9 mmol/l (p<0.001). With glucose loading, FMD, when defined as the peak increase in diastolic diameter relative to baseline ($\Delta D_{peak}/D_{baseline}$), increased from 3.2 ± 2.6 to $7.4\pm4.9\%$ (p=0.011). FMD, when normalized for the relative mean flow velocity increase ($\Delta V_{mean}/V_{baseline}$), showed a more pronounced increase from 0.047 ± 0.042 to 0.113 ± 0.047 (p<0.001).

Conclusions: Glucose loading leads to an increased brachial artery FMD response in the early postprandial phase, which is most likely related to the endothelial insulin-NO pathway.



IS AIX INCREASED IN COPD?

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Background: Chronic obstructive pulmonary disease (COPD) and cardiovascular disease (CVD) often coexist. Arterial stiffness predicts CVD and aortic augmentation index (Alx) is a non-invasive surrogate measure of arterial stiffness. We hypothesize that airflow limitation is associated with increasing arterial stiffness and the effect of having COPD increases Alx independently of other CVD risk factors.

Methods: This population-study is based on 3,432 subjects from the Copenhagen City Heart Study where Alx was measured; 494 had COPD. We analysed differences in Alx between subjects with and without COPD and used multiple linear regression analyses to examine the association between COPD and Alx stratified by age and gender and adjusted for CVD risk factors. Furthermore we analysed the association between Alx and FEV₁, FVC and FEV₁/FVC in the entire population.

Results: Alx was higher in subjects with COPD than in subjects without: 25.7 vs. 21.0 (p < 0.001) in men and 33.6 vs. 29.4 (p < 0.001) in women. We found no association between Alx and COPD adjusted for CVD risk factors (p = 0.17) except in men younger than 60 years (p = 0.003); and only when mild COPD was excluded from the analyses. Alx had a significant curvilinear association with FEV₁ and FVC but no association with the FEV₁/FVC ratio, the association with FEV₁ and FVC was seen only for higher volumes; i.e., FEV₁ > 3 L.

Conclusion: Alx and COPD are only weakly associated. In the general population, this finding potentially questions a direct association between COPD and arterial stiffness.

P13.06

ASSOCIATIONS BETWEEN "VASCULAR AGE" AND MARKERS OF SUBCLINICAL ATHEROSCLEROSIS IN MEN WITH LOW LEVEL OF CARDIOVASCULAR RISK

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Background: Previous studies showed how different risk factor burdens could be translated into vascular age.

Aim: To define correlations between "vascular age" and such subclinical markers of atherosclerosis as pulse wave (PWV), carotid intima-media thickness (IMT).

Material and Methods: We investigated 150 men (age 35-65) with a low level of cardiovascular risk according to "SCORE" scale without diabetes. Patients have been divided to the following groups: according to age-"< 45 yrs." (n=58) and "> 45 yrs" (n=92); with arterial hypertension (AG) (n=56), without AG (n=94); delta "-"(n=108), when chronological



