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### **P8.10: THE PATIENTS WITH ISCHEMIC STROKE IN THE POSTERIOR CIRCULATION TERRITORY HAVE IMPAIRED ENDOTHELIAL FUNCTION AND CEREBROVASCULAR REACTIVITY**

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Aix75 ( $P=0.04$ ) and PWV ( $P=0.07$ ) and lower FMD ( $P=0.02$ ). The relationship between age and PWV was significant in low 25(OH)D versus normal 25(OH)D status. In conclusion, 25(OH)D may have favorable effects on vascular function, particularly AAs, and lower 25(OH)D among AAs may contribute to their greater arterial stiffness and endothelial dysfunction. Furthermore, adequate vitamin D levels may attenuate age-related increases in arterial stiffness.

#### P8.10

##### THE PATIENTS WITH ISCHEMIC STROKE IN THE POSTERIOR CIRCULATION TERRITORY HAVE IMPAIRED ENDOTHELIAL FUNCTION AND CEREBROVASCULAR REACTIVITY

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<sup>1</sup>The Russian State Medical University, Moscow, Russian Federation

<sup>2</sup>the Russian Medical Academy of the Postgraduate Education, Moscow, Russian Federation Impaired endothelial function (EF), cerebrovascular reactivity (CVR) and atherosclerosis can all play a role in the ischemic stroke (IS) pathogenesis

The aim was to assess EF, CVR and intima-media thickness (IMT) in patients with IS in the posterior circulation territory (PCT).

**Methods:** The study population consisted of 13 patients with IS (without atrial fibrillation) in PCT (72 hours after stroke onset). For these subjects, EF and CVR were compared against an age and gender-matched group consisting of 10 healthy individuals (control group, CG). EF was assessed using the brachial flow-mediated vasodilatation (FMD). Breath-holding test (CVR\_BHT=test/baseline mean velocity) and hyperventilation (CVR\_HV=baseline/test mean velocity) were used for CVR evaluation in basilar artery (using transcranial Doppler).

**Results:** We observed a significant difference in FMD between IS patients and CG ( $5.45\pm 0.9\%$  and  $8.97\pm 1.37\%$  respectively,  $p=0.04$ ). IS patients and CG differed in CVR\_BHT ( $1.25\pm 0.49$  and  $1.44\pm 0.6$  respectively,  $p=0.04$ ), no difference was detected in CVR\_HV ( $1.5\pm 0.33$  and  $1.36\pm 0.8$  respectively,  $p>0.05$ ). IMT was similar for IS patients and CG ( $0.95\text{mm}\pm 0.12$  and  $0.91\text{mm}\pm 0.1$  respectively,  $p>0.05$ ). Correlation analysis revealed significant interrelationships between FMD and CVR\_BHT ( $r=0.38$ ); no significant correlations were found between FMD and CVR\_HV.

**Conclusion:** Patients with IS in PST showed impaired EF and CVR (vasodilatation reaction), both being strong indicators of stroke development. The significant correlation between these parameters is a sign that vasodilatation of brain arterioles can be at least partially mediated by the endothelium. The absence of any significant differences in IMT is an evidence of equal prevalence of atherosclerosis among general population.

#### P8.11

##### ENDOTHELIAL FUNCTION IS DECREASED IN PATIENTS WITH LACUNAR ISCHEMIC STROKE AND INTACT IN PATIENTS WITH ATHEROTHROMBOTIC STROKE

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<sup>1</sup>The Russian State Medical University, Moscow, Russian Federation

<sup>2</sup>Federal State Clinical Hospital #86, Moscow, Russian Federation Impaired endothelial function (EF) plays a role in pathogenesis of a small vessel disease and lacunar stroke (LS)

The aim of this study was to compare EF and cerebrovascular reactivity (CVR) in patients with LS and patients with atherothrombotic stroke (AS).

**Methods:** The study population consisted of 10 patients with LS (21 days after stroke onset). For these subjects, EF and CVR were compared against an age and gender-matched group consisting of 10 patients with AS. The stroke subtype was classified according to TOAST criteria. EF was assessed using the brachial flow-mediated vasodilatation (FMD). Peak shear stress was used for FMD normalization. Breath-holding test (CVR\_BHT=test/baseline mean velocity), hyperventilation (CVR\_HV=baseline/test mean velocity) and test with nitroglycerine (CVR\_NG=baseline/test mean velocity) were used for CVR evaluation in middle cerebral and basilar artery (using transcranial Doppler).

**Results:** We observed a significant difference in FMD between LS and AS patients ( $6.65\pm 0.8\%$  and  $10.03\pm 1.1\%$  respectively,  $p=0.03$ ). The difference in FMD remained after normalization ( $0.011\pm 0.005\text{s}^{-1}$  and  $0.018\pm 0.007\text{s}^{-1}$  respectively,  $p=0.02$ ). However, no difference in CVR was detected between IS and AS groups (CVR\_BHT= $1.25\pm 0.17$  and  $1.24\pm 0.1$ , CVR\_HV= $1.34\pm 0.24$  and  $1.16\pm 0.1$ , CVR\_NG= $1.08\pm 0.05$  and  $1.09\pm 0.1$  for LS and AS patients respectively).

**Conclusion:** Patients with LS demonstrated impaired EF in comparison with patients with AS, however, no difference between these groups was observed in CVR. The impairment of EF in LS patients may reflect a role of endothelial dysfunction in the pathogenesis of LS.

#### P8.12

##### GENETIC INFLUENCES ON THE RELATION BETWEEN EXHALED NITRIC OXIDE AND ARTERIAL STIFFNESS: A TWIN STUDY

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**Objectives:** Fractional exhaled nitric oxide (FE<sub>NO</sub>) has an important physiological role in the airways and vessel walls.

**Methods:** 117 adult twin pairs were recruited in Hungary, Italy and in the United States (83 monozygotic and 34 dizygotic pairs; age  $48\pm 16$  years). FE<sub>NO</sub> was measured by an electrochemical sensor-based device and markers of arterial stiffness (augmentation index on brachial artery, Aix<sub>bra</sub> and on aorta, Aix<sub>ao</sub>; Pulse Wave Velocity on aorta, PWV<sub>ao</sub>) by oscillometric method (TensioMed Arteriograph, TensioMed Ltd., Budapest). Bivariate Cholesky decomposition models were applied.

**Results:** Genetic effects accounted for 59% (95% confidence interval [CI]: 43, 71%) of the variation in FE<sub>NO</sub> with the remaining 41% (95%CI: 29, 57%) due to non-shared environmental influences. Significant correlation was observed between FE<sub>NO</sub> and Aix<sub>bra</sub> ( $r=-0.20$ , 95% CI:-0.34,-0.05), or Aix<sub>ao</sub> ( $r=-0.19$ , 95%CI:-0.33,-0.05). No significant correlation was found between FE<sub>NO</sub> and PWV<sub>ao</sub>. Genetic factors explained the entire phenotypic correlation between FE<sub>NO</sub> and Aix<sub>bra</sub> or FE<sub>NO</sub> and Aix<sub>ao</sub>. FE<sub>NO</sub> showed a significant negative genetic correlation with Aix<sub>bra</sub> and Aix<sub>ao</sub> ( $r_g=-0.29$ , 95%CI:-0.49,-0.07).

**Conclusions:** Variations in FE<sub>NO</sub> are explained by genetic and non-shared environmental effects. Covariances between FE<sub>NO</sub> and Aix<sub>bra</sub> or Aix<sub>ao</sub> are explained entirely by genetic factors suggesting a remarkable overlap among the sets of genes involved in the expression of these phenotypes and providing a basis for further studies on cardiovascular and respiratory diseases.

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#### P8.13

##### INCREASED AORTIC PULSE WAVE VELOCITY IS ASSOCIATED WITH SUBCLINICAL ATHEROSCLEROSIS BUT NOT WITH ENDOTHELIAL DYSFUNCTION IN HYPERTENSIVE PATIENTS

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**Background:** Increased vascular stiffness has been recently considered a marker of cardiovascular risk and a predictor of cardiac events

**Objective:** To compare parameters of endothelial dysfunction and carotid atherosclerosis in hypertensive patients presenting increased vascular stiffness.

**Methods:** A cross-sectional study was carried out to evaluate hypertensive patients, both genders, aged 30-65 years. Macro- and microcirculation parameters were evaluated with measurement of blood pressure (BP), carotid intima-media thickness (IMT), brachial flow-mediated dilation (FMD), peripheral arterial tonometry (EndoPAT), determination of aortic (carotid-femoral) pulse wave velocity (cfPWV) by Complior SP.