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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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Alx75 (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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¹the Russian State Medical University, Moscow, Russian Federation ²the 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 (0.95mm \pm 0.12 and 0.91mm \pm 0.1 respectively, p>0.05). Correlation analysis revealed significant interrelationships 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 vasodilation 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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¹The Russian State Medical University, Moscow, Russian Federation ²Federal 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 $(0.011\pm0.005s^{-1})$ and 0.018 ± 0.007 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_{NO}) 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 ± 16 years). FE_{NO} was measured by an electrochemical sensor-based device and markers of arterial stiffness (augmentation index on brachial artery, Aix_{bra} and on aorta, Aix_{ao}; Pulse Wave Velocity on aorta, PWV_{ao}) 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 $_{NO}$ with the remaining 41% (95%CI: 29, 57%) due to non-shared environmental influences. Significant correlation was observed between FE $_{NO}$ and Aix $_{bra}$ (r=-0.20, 95% CI:-0.34,-0.05), or Aix $_{ao}$ (r=-0.19, 95%CI:-0.33,-0.05). No significant correlation was found between FE $_{NO}$ and PWV $_{ao}$. Genetic factors explained the entire phenotypic correlation between FE $_{NO}$ and Aix $_{bra}$ or FE $_{NO}$ and Aix $_{ao}$. FE $_{NO}$ showed a significant negative genetic correlation with Aix $_{bra}$ and Aix $_{ao}$ (r=-0.29, 95%CI:-0.49,-0.07).

Conclusions: Variations in FE_{NO} are explained by genetic and non-shared environmental effects. Covariances between FE_{NO} and Aix_{bra} or Aix_{ao} 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.

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Results: Patients (n=70) were divided into Normal cfPWV (<10 m/s, n=31) and High cfPWV (>=10 m/s, n=39) groups. The mean age (49 \pm 2 vs 51 \pm 2 years) was similar between the groups. High cfPWV group presented greater systolic BP (135 \pm 3 vs 145 \pm 3 mmHg, p<0.05) and pulse pressure (48 \pm 2 vs 57 \pm 2 mmHg, p<0.01). Carotid IMT (0.72 \pm 0.04 vs 0.90 \pm 0.07 mm, p<0.05) and media-lumen ratio (0.10 \pm 0.01 vs 0.13 \pm 0.01 %, p<0.05) were significantly increased in patients with high cfPWV. On the other hand, brachial FMD (9.74 \pm 1.78 vs 9.38 \pm 1.38 %) and reactive hyperemia index by EndoPAT (2.12 \pm 0.12 vs 2.11 \pm 0.08) were not different between the groups. cfPWV was significantly correlated to systolic BP (r=0.37, p<0.01), pulse pressure (r=0.42, p<0.01), and carotid IMT (r=0.33, p<0.05). After multivariate analysis, pulse pressure was the only variable independently associated with cfPWV.

Conclusion: Elevated pulse pressure confirmed to be a clinical indicator of increased central vascular stiffness which is associated with subclinical carotid atherosclerosis even when endothelial function is not significantly impaired in hypertensive patients.

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P9.01

CINACALCET MAY REDUCE ARTERIAL STIFFNESS IN PATIENTS WITH CHRONIC RENAL DISEASE AND SECONDARY HYPERPARATHYROIDISM—RESULTS OF A SMALL-SCALE, PROSPECTIVE, OBSERVATIONAL STUDY

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Introduction: Arterial stiffness(AS) is one important cardiovascular risk (CR) in haemodialysis patients. Secondary hyperparathyroidism (SH) is one frequent complication in this patients and become the AS. Cinacalcet is a new drug in the treatment of SH. We proposed to do the next study.

Material and Methods: 21 patients(13 men/8women) with SH were included: age 51,3(18),BMI 25,5(1,3) kgrs/m2. AS was studied with Complior system and determinated pulse wave velocity (PWV), central pressure was determinated with Sphigmocor system, also was calculated ventricular mass (MV) with echocardiography. We determinated different parameters as: Htc, Hb, cholesterol, Alb, Ca, P, PTH, AP and Kt/v, all these were evaluated at the begin and end of study and the pursuit was 12 months and didn't have changes in the treatment for hypertension. We used t-Student and Spearman's correlation as statistical method ,p<0.05 was considered statistically significative (SS)

Results: The next results were SS between the begin and the end of study: PWV 9,35(1,83) vs 8,66(1,86) p<0.03. VM 166,6(39,4) vs 156(31,8) p<0.06. PTH 1008(846) vs 341(246) p<0.0001, AP 168,5(79,6) vs 124(72,8) p<0.001. PWV had correlations with age r=0,608 p<0.004. PPc r=0,707 p<0.0001 and VM r=0,405 p<0.07. PTH with AP r=0,542 p<0.014. We didn't have SS changes with blood pressure and other parameters included in the study. Conclusions: After one year of treatment with cinacalcet in patients with SH we have observed a significative reduction of PWV and huge tendency of reduction en VM but without changes in blood pressure. Also a significative reduction with PTH and AP.

Characteristic	Baseline Mean (+/- SE)	After 12 months Mean (+/- SE)	P value
Central systolic BP,mmHg	, , ,	125,9(+/- 26.4)	ns
Peripheral systolic BP, mm Hg	,	` ′	ns
Central diastolic BP,mmHg	84.7(+/- 13.3)	79.8(+/- 14.8)	ns
Peripheral diastolic BP,mmHg	83.5(+/- 13.2)	77.3(+/- 14.8)	0.051
Central PP, mmHg	47.4(+/- 16.7)	46.3(+/- 19.0)	ns
Peripheral PP, mmHg	59.1(+/- 18.3)	58.1(+/- 20.1)	ns
Aortic Alx at HR of 75, %	31.6(+/- 11.2)	32.9(+/- 10.5)	ns
Aortic PWV, m/s	9.35(+/- 1.83)	8.66(+/- 1.86)	0.030*
HR, beats per minute	75.7(+/- 12.1)	73.6(+/- 13.3)	ns
LV ejection fraction, %	65.1(+/- 9.0)	65.8(+/- 6.4)	ns
LV interseptal wall Wall thickness, mm	12.7(+/- 1.9)	12.5(+/- 1.3)	ns
LV mass index, g/m2	166.6(+/- 39.4)	156.1(+/- 31.8)	0.063
LV posterior wall Thickness, mm	12.1(+/- 1.7)	11.9(+/- 1.7)	ns

Figure 1: Alx = augmentation index; BP= blood pressure; HR= heart rate; LV= left ventricular, ns= no statistical difference; PP= pulse pressure; PWV=

Pulse wave velocity; SE = standard error. * Statistically significant (p < 0.05)

P9.02

EFFECT OF RENAL NERVE ABLATION ON RENAL PERFUSION AND ARTERIAL WAVE REFLECTION IN TREATMENT RESISTANT HYPERTENSION

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Background: Renal nerve ablation (RNA) emerged as new therapeutic approach for treatment resistant hypertension. Measurement of the renal and sympathetic activity revealed a decrease in sympathetic drive to the kidney and small resistance vessels after RNA. However, the long-term consequences on renal perfusion and arterial function e.g. vascular remodeling are unknown.

Methods: In a pilot study 8 patients with treatment resistant hypertension were included and renal plasma flow (RPF) was non-invasively measured by magnetic resonance imaging with arterial spin labeling (MRI-ASI) before (day-1), after (day+1) and again after 1 months of RNA. In addition pulse wave analysis (central Alx@75, central systolic and diastolic BP) was assessed before (day-1) and after 6 months of RNA.

Results (median (interquartile range)): RPF did not differ between day-1 and day+1 (265 (242 – 267) versus 255 (236 – 289), p=0.811) as well as after 1 months (p=0.392) after renal nerve ablation. In accordance renal function (serum creatinine, eGFR, cystatin C) did not differ at any point of time. Central systolic (146 (133 – 155) versus 125 (116 – 136), p=0.046) as well as central diastolic BP (93 (87 – 112) versus 82 (79 – 88), p=0.046) was significantly reduced 6 months after RNA. Compared to day-1, there was a decrease in central Alx@75 after 6 months (25 (17 – 31) versus 19 (10 – 21), p=0.063) after RNA.

Conclusion: Thus, our data indicate that RNA reduce significantly central BP as well as improve vascular remodeling, which may impact on cardiovascular prognosis. Renal perfusion and function did not appear to be significantly changed

P9.03

DIFFERENT EFFECT OF ALISKIREN AND RAMIPRIL ON ARTERIAL STIFFNESS AND WAVE REFLECTION IN PREVIOUSLY UNTREATED ESSENTIAL HYPERTENSION PATIENTS

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Background: Essential hypertensive patients (EH) are characterized by increased arterial stiffness. The renin-angiotensin system (RAS) activation is an important pathophysiological mechanism for arterial stiffening. Aliskiren is a novel direct renin inhibitor, whose effects on arterial elastic properties in EH are unknown. In this study we evaluated whether aliskiren, as compared to the ACE-inhibitor ramipril, can improve arterial stiffness and peripheral wave reflection in untreated mild-moderate EH, according to a double-blind parallel-group study.

Methods: 40 EH were randomized to a 12-week treatment with aliskiren (300 mg/daily) or ramipril (10 mg/daily) (n=20 each group). At baseline and after treatment arterial stiffness was assessed as carotid-to-femoral pulse wave velocity (PWV) by arterial tonometry (Sphygmocor). Central blood pressure and augmentation index (Alx) was also assessed.

Results: Blood pressure values were similarly normalized by aliskiren (from $147\pm8/95\pm2$ to $131\pm9/85\pm4$ mmHg) and ramipril (from $149\pm6/96\pm6$ to $133\pm8/86\pm3$ mmHg). Central pulse pressure was also similarly decreased (aliskiren from 39.5 ± 7.7 to 35.5 ± 6.9 mmHg, P<0.01; ramipril from 38.4 ± 8.9 to 34.8 ± 5.7 mmHg, P<0.01). Aortic PWV was similarly decreased by aliskiren (from 7.7 ± 1.2 to 7.1 ± 1.3 m/s, P<0.05) and ramipril (from 7.5 ± 1.1 to 6.9 ± 1.1 m/s, P<0.05). Alx was reduced after aliskiren (from 18.0 ± 8.1 to $13.6\pm17.4\%$, P<0.05) and after ramipril treatment (from 19.1 ± 8.3 to $17.1\pm8.1\%$, P<0.05), but aliskiren induced a significantly greater reduction (p<0.05).

Conclusions: These results indicate that RAS blockade by aliskiren and ramipril can improve aortic stiffness in EH. The direct renin inhibitor seems to