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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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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±2 vs 51±2 years) was similar between the groups. High cfPWV group presented greater systolic BP (135±3 vs 145±3 mmHg, p<0.05) and pulse pressure (48±2 vs 57±2 mmHg, p<0.01). Carotid IMT (0.72±0.04 vs 0.90±0.07 mm, p<0.05) and media-lumen ratio (0.10±0.01 vs 0.13±0.01 %, p<0.05) were significantly increased in patients with high cfPWV. On the other hand, brachial FMD (9.74±1.78 vs 9.38±1.38 %) and reactive hyperemia index by EndoPAT (2.12±0.12 vs 2.11±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.

P9 – Interventional Studies 2

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 determined pulse wave velocity (PWV), central pressure was determined with Sphigmocor system, also was calculated ventricular mass (MV) with echocardiography. We determined 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 significant (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	127,6(+/- 24.3)	125,9(+/- 26.4)	ns
Peripheral systolic BP, mm Hg	141.6(+/- 25.0)	135.1(+/- 26.3)	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/m ²	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-ASL) 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±8/95±2 to 131±9/85±4 mmHg) and ramipril (from 149±6/96±6 to 133±8/86±3 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±17.4%, P<0.05) and after ramipril treatment (from 19.1±8.3 to 17.1±8.1%, 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