



Artery Research

ISSN (Online): 1876-4401

ISSN (Print): 1872-9312

Journal Home Page: <https://www.atlantis-press.com/journals/artres>

P9.03: DIFFERENT EFFECT OF ALISKIREN AND RAMIPRIL ON ARTERIAL STIFFNESS AND WAVE REFLECTION IN PREVIOUSLY UNTREATED ESSENTIAL HYPERTENSION PATIENTS

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To cite this article: L. Ghiadoni, R.M. Bruno, G. Cartoni, A. Magagna, A. Viridis, S. Taddei (2011) P9.03: DIFFERENT EFFECT OF ALISKIREN AND RAMIPRIL ON ARTERIAL STIFFNESS AND WAVE REFLECTION IN PREVIOUSLY UNTREATED ESSENTIAL HYPERTENSION PATIENTS, Artery Research 5:4, 186–187, DOI: <https://doi.org/10.1016/j.artres.2011.10.137>

To link to this article: <https://doi.org/10.1016/j.artres.2011.10.137>

Published online: 14 December 2019

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.

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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 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 $147\pm 8/95\pm 2$ to $131\pm 9/85\pm 4$ mmHg) and ramipril (from $149\pm 6/96\pm 6$ to $133\pm 8/86\pm 3$ 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

have an additional positive effect on wave reflection, possibly linked to a effect on the peripheral microcirculation.

P9.04

EFFECT OF ANTIHYPERTENSIVE TREATMENT ON PULSE WAVE VELOCITY AFTER ONE YEAR IN NEVER TREATED, NEWLY DIAGNOSED HYPERTENSIVE PATIENTS AND ITS DETERMINANTS

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Background: Aorto-femoral pulse wave velocity (PWV) is a well established method to stratify hypertensive patients, but information about the influence of treatment on this variable is scarce.

Methods: Longitudinal study that included 212 (122 men, 56 %) non-diabetic, never treated hypertensive patients. We measured at the initial visit standard clinical variables, as well as PWV (Sphygmocor-System), left ventricular mass index (echocardiography) and microalbuminuria to asses target organ damage. At a median of 1,07 (SD±0,34) years, a second visit was performed, measures were repeated.

Results: The mean age was 50±13 years with initial systolic and diastolic BP of 148/88 mmHg (SD±16/10). The initial prevalence of left ventricular hypertrophy (LVH), pathological urinary albumin excretion rate (UAER) and PWV > 12 m/sec were 30,7, 15,0 and 9,2 %, respectively. Considering a decrease of 1 m/sec as a significant change in PWV, 43 % of patients showed an improvement in PWV. In a logistic multivariate regression analysis, age (OR: -0,9, p<0,0001), systolic BP reduction > 15 mmHg (OR: 2,9, p<0,005), initial Cholesterol-LDL (OR: 1,1, p<0,006), use of angiotension-receptor-blockers (ARB) (OR: 2,5, p<0,022) and waist diameter (OR: -0,95, p<0,04) were independently associated with a decrease of PWV.

Conclusions: In untreated hypertensive patients, age and abdominal obesity are negative predictors of changes in PWV, whereas BP treatment, high cholesterol-LDL and use of ARB seem to favour a decrease of PWV during the first year of treatment.

Key Words: arterial stiffness, pulse wave velocity, applanation tonometry.

P9.05

EFFECT OF ANTIHYPERTENSIVE TREATMENT ON PULSE WAVE VELOCITY AFTER ONE YEAR IN PATIENTS WITH REFRACTORY HYPERTENSION AND ITS DETERMINANTS

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Background: Aorto-femoral pulse wave velocity (PWV) has been shown to be modifiable by antihypertensive treatment in newly diagnosed hypertension (HT), but little is known about the effect of treatment on longer standing, refractory HT.

Methods: Longitudinal study that included 79 (49 men, 62 %) patients with refractory hypertension, treated with at least three drugs in standard dosis. We measured at the initial visit standard clinical variables, as well as PWV (Sphygmocor-System), ECG-left ventricular mass (CORNELL-product and SOKOLOV) and microalbuminuria to asses target organ damage. At a median of 1,15 (SD±0,34) years, a second visit was performed, measures were repeated.

Results: The mean age was 64±11 years with initial systolic and diastolic BP of 155/79 mmHg (SD±25/11). The initial prevalence of left ventricular hypertrophy (LVH), pathological urinary albumin excretion rate (UAER) and PWV > 12 m/sec were 42, 16 and 27 %, respectively. Considering a decrease of 1 m/sec as a significant change in PWV, 36 % of patients showed an improvement in PWV. In a logistic multivariate regression analysis, systolic BP reduction > 9 mmHg (OR: 4,1, p<0,01), initial PWV (OR: 1,5, p<0,009) and glucemic control (HbA1c > 6,5 %, OR: - 0,2, p<0,049) were independently associated with a decrease of PWV.

Conclusions: In hypertensive patients with refractory HT, BP treatment is able to decrease PWV, correcting for age, gender and initial PWV-values, whereas a poor glucemic control appears to hinder the positive effect of antihypertensive treatment.

Key Words: arterial stiffness, pulse wave velocity, applanation tonometry.

P9.06

CORRELATES OF AUGMENTATION INDEX IN PATIENTS WITH NEVER TREATED UNCOMPLICATED NON-DIABETIC PATIENTS WITH ESSENTIAL HYPERTENSION

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Objective: Assessment of the association between augmentation index (AI75) and hemodynamic factors and target organ damage in 98 patients with:

1) prehypertension (N=56, BP=120-139 / 80-89 mmHg)- GI

2) grade 1 hypertension (N=42, BP=140-160 / 90-100mmHg)- GII

Methods: Evaluated parameters: biochemical profile, sphygmocardiography (Augmentation Index-AI75 and central BP), echocardiography (Left Ventricular Mass/Height^{2.7}- LVM/Ht^{2.7}) and renal functional indices (Creatinine Clearance-Ccr and Urinary Albumin Excretion-UAE).

Results: (table)

	GI	GII
Age (Years)	45.5 ± 2	67.5 ± 1.9*
BMI (kg/m ²)	27.9 ± 0.6	27.7 ± 0.6
SBPb (mmHg)	122 ± 1.9	143 ± 3.2*
DBPb (mmHg)	81 ± 1.4	78.5 ± 2.1
SBPc (mmHg)	114 ± 1.6	134 ± 3*
DBPc (mmHg)	82 ± 1.5	79 ± 2.2
PPb (mmHg)	41 ± 1.4	65 ± 3.1*
PPc (mmHg)	32 ± 0.8	55 ± 2.7*
LVM/Ht ^{2.7} (g/m ^{2.7})	60 ± 2	76 ± 4.9*
AI75 (%)	10.2 ± 5.6	39 ± 1*
Ccr (ml/min)	109.2 ± 5.6	71.9 ± 5.1*
UAE (mg/24hrs)	32 ± 8.2	56 ± 24.6

* p< 0.05, SBP=systolic blood pressure, DBP=diastolic blood pressure, b=brachial, c=central, PP=pulse pressure, BMI=Body Mass Index

In middle age and elderly hypertensive subjects, increased AI75 is:

1) associated with increased central/brachial SBP and PP, increased LVM/Ht^{2.7}, reduced Ccr and high UAE 2) independent of changes in biochemical profile.

Conclusion: Increased AI75 in never treated uncomplicated essential hypertension is: 1) associated with target organ damage; 2) probably a cardiovascular risk factor; 3) a sign of vascular disease which should be included in the evaluation of hypertensive patients.

P9.07

CAROTID STIFFNESS AND WAVE REFLECTION DURING ANTIHYPERTENSIVE THERAPY WITH CALCIUM ANTAGONISTS: INSIGHT FROM A WAVE INTENSITY APPROACH

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Background: Increasing evidence emphasizes the role of central pressure augmentation in the development of hypertension-related complications, and highlights the importance of targeting arterial stiffness and wave reflection with treatment.

Objective: Of this study was to evaluate the effects of the calcium antagonist barnidipine on large artery stiffness and wave reflection by means of separated wave analysis.

Methods: In twenty-one naïf-treatment hypertensive patients, carotid artery mechanics was investigated at baseline and after 3 and 6 months of barnidipine therapy by a double-beam carotid ultrasound technique (Aloka SSD 5500) which provides simultaneous recording of diameter-derived pressure and flow velocity signals and allows wave intensity (WI) analysis. Indices of local arterial stiffness and wave reflection, and separated forward and backward pressures were estimated. Twenty normotensive subjects were also .

Results: Compared to controls, hypertensive patients had higher forward and backward pressures (137±17 vs 108±7 and 21±6 vs 17±5 mmHg,