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ORIGINAL RESEARCH

Arterial stiffness parameters during six-month antihypertensive therapy: a prospective observational study

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ABSTRACT

Aim: To investigate the prevalence of arterial stiffness in hypertensive patients in Bosnia and Herzegovina and to assess the effects of various antihypertensive therapies on arterial stiffness.

Methods: This prospective, open-label clinical study enrolled 200 patients who met predefined inclusion criteria from a total of 217 screened between 1 November 2023 and 30 April 2024. Participants were followed for six months. Thirteen distinct antihypertensive treatment combinations were evaluated.

Results: In 200 patients, six months of antihypertensive treatment significantly reduced systolic blood pressure from 136.06 ± 15 to 130.69 ± 12 mmHg and diastolic blood pressure from 85.35±9 to 82.69±7 mmHg. Pulse wave velocity (PWV) changed only minimally, from 8.09±1.5 m/s to 8.05±1.2 m/s (p=0.354). Augmentation index (AIx) improved from 25.17% to 23.33%. The greatest reduction in PWV was observed with angiotensin-converting enzyme inhibitors plus statins (-0.19 m/s), while the largest reduction in AIx was seen with angiotensin-converting enzyme inhibitors, calcium channel blockers, beta blockers, diuretics, and statins (-15.64%, p<0.001). Baseline systolic blood pressure was the strongest, although weak, predictor of pulse wave velocity reduction (AUC=0.627, p=0.002).

Conclusion: The study demonstrated a high prevalence of increased arterial stiffness among hypertensive patients, with higher values associated with greater cardiovascular risk. Routine assessment of arterial stiffness, alongside standard blood pressure measurement, may improve cardiovascular risk stratification and support more effective, individualized hypertension management.

Keywords: atherosclerosis, carotid-femoral pulse wave velocity, vascular calcification

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INTRODUCTION

The prevalence of arterial hypertension, one of the main risk factors for cardiovascular disease, is increasing worldwide (1,2). Arterial stiffness and hypertension are strongly associated with aging and cumulative hemodynamic stress (3,4). Age-related changes in the arterial wall, including elastin fragmentation and collagen accumulation, contribute to progressive stiffening and vascular remodelling (5,6).

Since its formal recognition in hypertension guidelines as an indicator of target organ damage alongside left ventricular hypertrophy, carotid intima–media thickening, and microalbuminuria, arterial stiffness has gained increasing clinical relevance due to its independent association with cardiovascular events. Pulse wave velocity (PWV), the non-invasive gold standard measure of arterial stiffness, is linked to atherosclerosis and future cardiovascular risk in patients with hypertension (7,8). Recent systematic analyses confirm that reductions in PWV with antihypertensive therapy are largely influenced by blood pressure (BP) lowering, although some drug classes demonstrate BP-independent effects, particularly renin–angiotensin system inhibitors and calcium channel blockers (meta-analysis).

Emerging evidence further suggests that arterial stiffness is not only a marker of cardiovascular risk but also implicated in broader pathophysiological processes, including cerebrovascular impairment and end-organ damage beyond traditional measures of hypertension. Contemporary research emphasizes that targeted interventions to modulate arterial stiffness may yield additional prognostic benefit beyond standard blood pressure control. Despite this growing body of evidence, data remain limited regarding the effects of specific antihypertensive combinations on arterial stiffness in real-world clinical settings, and such evidence is particularly scarce in populations from the Western Balkans (11,12). In Bosnia and Herzegovina, no previous studies have systematically evaluated arterial stiffness

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using direct PWV measurement, nor have standardized reference values for PWV been established for the local hypertensive population; most existing practices rely on European or international norms that may not reflect regional cardiovascular risk profiles (13). Given the high burden of hypertension and the lack of local evidence on the impact of antihypertensive therapy on arterial stiffness, further investigation in this clinical context is urgently needed.

The aim of this study was to determine the prevalence and degree of increased arterial stiffness in hypertensive patients in Bosnia and Herzegovina and to evaluate the effects of different antihypertensive therapy combinations on arterial stiffness over a six-month treatment period.

MATERIAL AND METHODS

Subjects and study design

A prospective open-label clinical study was conducted from 1 November 2023 to 30 April 2024 at the Cantonal Hospital Bihać, Bosnia and Herzegovina, in collaboration with the Sarajevo School of Science and Technology Medical Faculty and ASA Hospital. The study included 200 patients aged 25–72 years with previously diagnosed or newly identified essential arterial hypertension.

The inclusion criteria encompassed patients diagnosed or newly identified essential arterial hypertension, provision of a written informed consent, and availability of sufficient clinical and laboratory data for analysis. Hypertension was defined according to the 2024 European Society of Cardiology (ESC) guidelines as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, confirmed on at least three separate measurements.

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Exclusion criteria included acute coronary syndrome, ischemic stroke, hospitalization due to heart failure, and other serious acute or chronic conditions that could influence study outcomes. Patients with incomplete or unavailable medical records were also excluded.

The study employed a non-randomized design, and patients were assigned to one of thirteen predefined antihypertensive treatment regimens.

Methods

All participants received standard clinical evaluation and were followed for a period of six months. During this time, patients remained on their initially prescribed antihypertensive therapy without dose adjustments, medication substitutions, or additions of new drugs. Adherence to therapy was assessed at each visit through patient self-report.

Anthropometric measurements were obtained using standardized procedures. Height was measured with a stadiometer and weight was measured using a mechanical weighing scale. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters.

BP was measured using an automatic upper arm monitor (Bosch A3, Bosch, Germany). Measurements were performed with the patient seated, feet flat on the floor, and the arm supported at heart level. Three consecutive readings were taken with one-to-two-minute intervals, and the diagnosis of hypertension was based on these measurements. When necessary, 24-hour ambulatory blood pressure monitoring was additionally performed.

Arterial stiffness was assessed using the Agedio B900 device (Ambulatory Blood Pressure and Pulse Wave Analysis Monitor, Agedio, Germany, 2016). PWV measurements were obtained in a seated position after a three-to-five-minute rest period, in a quiet environment with controlled room temperature (23°C during baseline visits and approximately 28°C during summer follow-up visits). Measurements were performed at the same time of day to

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reduce diurnal variability. A single PWV measurement was recorded per visit, without repetition or averaging, and all measurements were conducted by the same experienced cardiologist. The device provides an estimate of arterial pulse wave velocity (aPWV) derived from oscillometric waveform analysis rather than direct carotid-femoral PWV (cfPWV). Therefore, guideline-recommended cfPWV thresholds are not directly applicable to these measurements. The augmentation index corrected for heart rate (AIx) was automatically calculated by the device.

All patients received rosuvastatin as part of standard therapy, either uniformly or based on individual clinical indications. Since statins may influence arterial stiffness, their use was considered a potential confounding factor in statistical analyses.

Outcome variables included changes in PWV, AIx, and BP at six-month follow-up.

Statistical analysis

Data were analysed using SPSS version 28 (IBM Corp., Armonk, NY, USA). Normality of data distribution was assessed using the Shapiro–Wilk test. Data are presented as mean ± standard deviation or median (interquartile range), depending on distribution. Paired t-tests were used to compare baseline and six-month values for blood pressure and PWV. The Wilcoxon signed-rank test was employed to assess the impact of different antihypertensive drug combinations on changes in PWV, as this non-parametric test is appropriate for comparing related samples when normality assumptions are not met. Changes in SBP, DBP and PWV were analysed using the appropriate tests based on data distribution. Pearson correlation coefficients (R) were calculated to assess associations between PWV and various factors such as age, BMI, smoking, and alcohol consumption. Receiver operating characteristic (ROC) curve analysis was conducted to identify baseline parameters that predict changes in PWV, with systolic blood pressure, BMI, diastolic blood pressure, and AIx

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showing predictive value. A p-value < 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 200 patients were included in the study, of whom 115 (57.5%) were female and 85 (42.5%) were male. The mean age of participants was 54 years (range 25–72 years). BMI values ranged from 18 to 48 kg/m², with a mean BMI of 29 kg/m². Most participants reported no alcohol consumption, with 179 patients (89.5%) indicating that they did not consume alcohol, while 21 patients (10.5%) reported alcohol consumption. Regarding smoking habits, 71 participants (35.5%) were smokers, whereas 129 participants (64.5%) reported no use of tobacco products.

Baseline PWV averaged 8.09±1.5 m/s and decreased to 8.05±1.2 m/s after six months of treatment (Table 1), but this change did not reach statistical significance (p=0.354). The greatest reduction in PWV was observed with the combination of angiotensin-converting enzyme inhibitors (ACEI) and statins, with a median of -0.19 m/s, indicating a potentially most favourable effect on arterial stiffness among the analysed combinations. An increase in PWV was observed in several combinations, most notably with calcium channel blocker (CCB) and statin therapy (median change 0.85 m/s) (Table 2); however, this result was based on a very small number of patients and should be interpreted with caution. Older patients (>60 years) had significantly higher baseline PWV values compared to younger patients (9.55±1.05 m/s vs. 7.47±0.98 m/s, p<0.001). Alcohol consumption (R = -0.061, p=0.363) and smoking (R= -0.072, p=0.280) were not significantly associated with higher PWV values.

At baseline, mean SBP and DBP were 136.06±15 mmHg and 85.35±9 mmHg, respectively. After six months of antihypertensive therapy, a significant reduction in BP was observed, with SBP decreasing to 130.69±12 mmHg and DBP to 82.69±7 mmHg (p<0.05). Significant

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reductions in DBP were observed in the beta blocker (BB) with statin, ACEI with BB, diuretic and statin, ACEI with CCB, BB and statin, ACEI with CCB and statin, and ACEI with statin groups ($p<0.05$). No statistically significant BP changes were observed in the remaining treatment groups (Table 3).

ROC analysis identified several baseline parameters as statistically significant, but weak predictors of PWV reduction. Baseline SBP demonstrated the highest discriminative ability (AUC=0.627, 95% CI 0.547–0.706, p=0.002), followed by BMI (AUC=0.590, 95% CI 0.511–0.669, p=0.029). Baseline DBP (AUC=0.586, 95% CI 0.507–0.665, p=0.037) and AIx (AUC=0.581, 95% CI 0.502–0.661, p=0.048) also showed statistically significant, albeit modest, predictive value (Table 4).

The mean AIx decreased from 25.17% at baseline to 23.33% at the control examination. A reduction was also observed in median Alx values (24.5% vs. 22.0%), indicating an overall improvement in wave reflection parameters following therapy (Table 1). Treatment with ACEI, diuretics, and statins led to a median AIx decrease of 8.39% (p=0.005). A more comprehensive combination of ACEI, CCBs, BBs, diuretics, and statins produced the greatest reduction, with a median decrease of 15.64% ($p<0.001$). Similarly, patients receiving angiotensin receptor blockers (ARB) in combination with BB, diuretics, and statins experienced a significant AIx reduction of 12.21% (p=0.006) (Table 2).

DISCUSSION

In this study of 200 patients with hypertension, six months of antihypertensive therapy, administered alongside uniform statin treatment resulted in significant reductions in both SBP and DBP, accompanied by measurable improvements in arterial stiffness parameters. Therapy allocation was based on the physician's discretion and was not protocol-driven; therefore confounding by indication cannot be excluded. These findings support the concept that

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effective BP control, particularly when combined with agents acting on the renin–angiotensin system, favourably influences vascular function.

Baseline PWV values were below the conventional cfPWV threshold, as defined in ESC guidelines. Following treatment, PWV decreased significantly, with the most pronounced reduction observed in patients receiving combination therapy including an ACE inhibitor, calcium channel blocker, and statin. A reduction in PWV of approximately 1 m/s has been shown to be clinically relevant and associated with lower cardiovascular morbidity and mortality (11-13).

The observed age-related differences in PWV are consistent with data from the Framingham Heart Study, which demonstrated a progressive increase in arterial stiffness with advancing age, independent of traditional cardiovascular risk factors (14). Previous epidemiological and mechanistic studies have reported a positive association between smoking, alcohol consumption, and higher PWV values, linking lifestyle-related endothelial dysfunction and vascular aging to increased arterial stiffness (15).

AIx, which reflects wave reflection and central arterial load, also improved following antihypertensive therapy in our cohort. Notably, significant reductions in AIx were observed particularly in regimens including renin–angiotensin system inhibitors (such as ACEIs) and BBs in combination with statins. These findings are consistent with recent network meta-analyses showing that while many antihypertensive classes effectively lower BP, their impact on arterial stiffness parameters varies by class and outcome measure. For example, the RIGIPREV network meta-analysis demonstrated that ACEIs, ARBs, and several combination regimens produced greater reductions in AIx compared with other therapies, whereas effects on PWV were more heterogeneous and generally modest across classes (16). Additionally, recent systematic reviews indicate that reductions in PWV following antihypertensive

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treatment are largely driven by BP lowering, but some drug classes (including ACEIs and ARBs) may exert additional vascular benefits beyond BP reduction, which could translate into more pronounced improvements in wave reflection indices such as AIx (17). Together, these data suggest that improvements in wave reflection may occur even when structural measures of large-artery stiffness show smaller changes, underscoring the importance of including both functional and structural vascular endpoints in the evaluation of antihypertensive therapy effects. The potential influence of statin therapy on arterial stiffness is an important consideration in this study. Statins are known to have pleiotropic effects, including potential benefits on vascular function, which could independently affect AIx.

ROC analysis in our study identified baseline SBP as the strongest predictor of PWV reduction, followed by BMI, baseline DBP, and AIx. Although the predictive power of these parameters was modest, the findings are in line with previous observations that patients with higher baseline hemodynamic load and adverse metabolic profiles derive greater vascular benefits from antihypertensive therapy. The association between BMI and PWV improvement is also consistent with studies demonstrating a link between visceral adiposity, arterial stiffness, and cardiovascular risk, including data from observational cohorts and the Croatian study examining relationships between body composition, antihypertensive therapy, and arterial stiffness (18).

Beyond BP control, growing evidence indicates that arterial stiffness represents an integrative marker of long-term cardiovascular and metabolic risk, with implications extending beyond traditional cardiovascular outcomes. Large population-based studies have demonstrated that increased arterial stiffness predicts the development of chronic diseases independently of blood pressure levels. For instance, data from the Kailuan study showed that elevated arterial stiffness was a strong predictor of incident type 2 diabetes mellitus, outperforming BP alone (19). Notably, individuals with both increased arterial stiffness and hypertension exhibited

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the highest risk, highlighting the synergistic effect of vascular dysfunction and elevated hemodynamic load.

Long-term observational data further suggest that arterial stiffness may serve as an early marker of conditions that manifest later in life. A prospective study from the United States, with approximately 15 years of follow-up, demonstrated that higher carotid–femoral PWV was associated with an increased risk of developing dementia. This finding implies that arterial stiffening may contribute to microvascular damage and impaired cerebral perfusion, thereby linking vascular aging to cognitive decline (20). The role of the renin–angiotensin–aldosterone system in arterial stiffness is further supported by studies such as the TAIPAI trial, which showed significant PWV improvement in patients with primary aldosteronism treated with mineralocorticoid receptor antagonists or adrenalectomy, suggesting that targeting neurohormonal pathways may beneficially influence arterial structure and function beyond conventional BP reduction (21).

Several limitations must be acknowledged. The observational design and heterogeneity of treatment regimens limit causal inference, and some subgroup analyses were constrained by small sample sizes. Additionally, arterial stiffness was assessed over a relatively short follow-up period, whereas long-term structural remodelling may require longer treatment durations. Nevertheless, the standardized use of statin therapy across all participants reduces confounding related to lipid-mediated vascular effects and strengthens the interpretation of antihypertensive drug-related differences.

CONCLUSION

Our findings demonstrate that antihypertensive therapy, particularly regimens including ACE inhibitors and statins, is associated with significant improvements in arterial stiffness and wave reflection parameters. Baseline blood pressure, age, BMI, and lifestyle factors were key

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determinants of vascular stiffness and its reversibility. These results are consistent with contemporary evidence and ESC guideline concepts, supporting the use of arterial stiffness assessment as an adjunct tool for cardiovascular risk stratification and therapeutic evaluation in patients with hypertension.

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Conflicts of interest: None to declare.

Author contributions (CRediT): Conceptualization—A.H.; Methodology—A.H., S.S.; Formal analysis—A.H.; Investigation—A.H., S.S., E.M.; Data curation—A.H.; Writing – original draft—A.H.; Writing – review & editing—A.H., S.S., E.M.; Supervision—S.S., E.M.; Project administration—A.H.; Funding acquisition—S.S.

Ethics statement: This study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of the Cantonal Hospital "Dr. Irfan Ljubijankić", Bihać, Bosnia and Herzegovina (approval number: 1.1-33-7756-1/20; date: 21 October 2020). All participants provided written informed consent prior to inclusion in the study.

Data availability statement: The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

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TABLES AND FIGURES

Table 1. Pulse wave velocity (PWV) and augmentation index (AIx) values at the first and follow-up visit

Parameter	PWV (m/s)		AIx (%)	
	First visit	Follow-up visit	First visit	Follow-up visit
Mean	8.086	8.054	25.173	23.330
Median	7.900	7.850	24.500	22.000
Minimum	4.700	5.300	3.000	4.000
Maximum	16.000	15.600	56.000	49.000
25th centile	6.900	6.800	12.750	11.000
75th centile	9.275	9.250	34.750	32.000

PWV, pulse wave velocity; AIx, augmentation index;

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Table 2. Effect of different antihypertensive drug combinations on pulse wave velocity (PWV) and augmentation index (AIx) in six-month period

Antihypertensive drug combinations	N (%)	PWV (m/s)	AIx (%)
		Median change	
ACEI, CCB, Stat	42 (21.0)	0.045	-4.00
BB, Stat	27 (13.5)	0.011	-2.93
ACEI, Stat	24 (12.0)	-0.192	-4.41
ACEI, D, Stat	18 (9.0)	-0.017	7.43
ACEI, CCB, BB, Stat	17 (8.5)	0.247	-8.39
BB, ARB, CCB, D, Stat	12 (6.0)	-0.025	0.67
ACEI, CCB, BB, D, Stat	11 (5.5)	0.046	-1.21
ARB, CCB, D, BB, Mon, Stat	9 (4.5)	0.156	-15.64
ACEI, BB, D, Stat	9 (4.5)	0	6.00
BB, D, Stat	8 (4.0)	-0.150	-0.75
D, Stat	7 (3.5)	-0.057	-12.21
ARB, D, BB, Stat	14 (7)	0.325	-3.78
CCB, Stat	2 (1.0)	0.850	-9.50

PWV, pulse wave velocity; AIx, augmentation index; ACEI, angiotensin-converting enzyme inhibitor; CCB, calcium channel blocker; D, diuretic; Stat, statin; BB, beta-blocker; Mon, moxonidine; ARB, angiotensin receptor blocker; N, number of patients;

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Table 3. Effects of different antihypertensive drug combinations on systolic (SBP) and diastolic blood pressure (DBP)

Antihypertensive drug combination	SBP		DBP	
	Z	p	Z	p
ACEI, BB, D, Stat	-0.426	0.670	-2.121	0.034
BB, Stat	-3.479	0.001	-4.147	<0.001
ACEI, CCB, BB, Stat	-2.960	0.003	-2.546	0.011
D, Stat	-0.966	0.334	-0.816	0.414
ACEI, D, Stat	-1.817	0.069	-1.802	0.072
ACEI, CCB, Stat	-2.921	0.003	-2.247	0.025
ACEI, Stat	-3.237	0.001	-2.025	0.043
ACEI, CCB, BB, D, Stat	-1.590	0.112	-1.556	0.120
BB, ARB, CCB, D, Stat	-1.335	0.182	-1.851	0.064
BB, D, Stat	-0.736	0.461	-0.535	0.593
ARB, BB, D, Stat	-2.335	0.020	-0.750	0.453
ARB, CCB, D, BB, Mon, Stat	-0.763	0.445	-0.344	0.731
CCB, Stat	-0.447	0.655	-0.447	0.655

SBP, systolic blood pressure; DBP, diastolic blood pressure; Z, test statistic; ACEI, angiotensin-converting enzyme inhibitor; CCB, calcium channel blocker; D, diuretic; Stat, statin; BB, beta-blocker; Mon, moxonidine; ARB, angiotensin receptor blocker; N, number of patients;

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Table 4. Baseline predictors of pulse wave velocity reduction - receiver operating curve (ROC) analysis

Parameter	AUC	95% CI	p
SBP (mmHg)	0.627	0.547 – 0.706	0.002
DBP (mmHg)	0.586	0.507 – 0.665	0.037
AIx (%)	0.581	0.502 – 0.661	0.048
BMI (kg/m²)	0.59	0.511 – 0.669	0.029

AUC, area under the curve; CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; AIx, augmentation index; BMI, body mass index;

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