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ORIGINAL RESEARCH

Arterial Stiffness: A Strong Determinant of Abnormal Cardiac Magnetic Resonance Imaging in an Untreated Hypertensive Population

Konstantinos Vasileiadis, Christina Antza, Anastasia Malliora, Victoria Potoupni, Vasilios Kotsis

3rd Department of Internal Medicine, Medical School, Aristotle University of Thessaloniki, Papageorgiou Hospital, Thessaloniki, 56403, Greece

Correspondence: Vasilios Kotsis, 3rdDepartment of Internal Medicine, Medical School, Aristotle University of Thessaloniki, Papageorgiou Hospital, Thessaloniki, 56403, Greece, Tel +30 6974748860, Email vkotsis@auth.gr

Objective: Hypertension significantly impacts cardiovascular health, leading to arterial stiffness and myocardial dysfunction. Pulse wave velocity (PWV) is a recognized measure of arterial stiffness, while cardiac magnetic resonance imaging (MRI) is the gold standard for assessing myocardial structure and function. The aim of the present study is to investigate the relationship between arterial stiffness, ambulatory blood pressure monitoring (ABPM), and cardiac MRI findings in untreated hypertensive individuals.

Methods: This cross-sectional study included 22 untreated hypertensive participants referred to the Hypertension ABPM Center of Excellence at Aristotle University of Thessaloniki. Participants underwent carotid-femoral PWV measurement and 24-hour ABPM. Cardiac function and structure were evaluated through cardiac MRI. Statistical analyses included Mann–Whitney and Kruskal–Wallis tests, with logistic regression for associations between c-f PWV and cardiac abnormalities. A significance threshold of $p<0.05$ was applied.

Results: The study population had increased office and 24-hour ABPM values. Cardiac MRI revealed systolic LV dysfunction in 31.8% and diastolic LV dysfunction in 63.6% of participants. Myocardial fibrosis was present in 50% of the participants. Elevated PWV was significantly associated with LV systolic dysfunction ($p=0.003$), LV diastolic dysfunction ($p=0.002$), myocardial stiffness ($p<0.001$), and myocardial fibrosis ($p = 0.004$). Additionally, aortic valve velocity was significantly associated with increased arterial stiffness ($p=0.006$). Post-hoc analysis of fibrosis showed significant differences ($p=0.007$ for minimal vs no fibrosis; $p=0.011$ for severe vs no fibrosis).

Conclusion: The study confirms a significant correlation between increased arterial stiffness, systolic ABPM-derived systolic blood pressure, and cardiac MRI dysfunction in untreated hypertensive individuals. These findings highlight the importance of arterial stiffness evaluation as a diagnostic tool for early detection of myocardial dysfunction, allowing for timely intervention and targeted treatment strategies to mitigate heart damage.

Keywords: hypertension, arterial stiffness, cardiac magnetic resonance, myocardial stiffness, myocardial fibrosis

Introduction

Hypertension is a major risk factor for cardiovascular disease and has profound effects on the heart's structure and function. The impact of hypertension on cardiac function manifests as hypertensive heart disease, which includes a spectrum of morphological and functional abnormalities in the left ventricle (LV). These changes, primarily concentric LV hypertrophy, arise as adaptive responses to hemodynamic wall stress caused by elevated blood pressure (BP). The hypertensive heart undergoes significant remodeling, affecting cardiomyocytes, the interstitial space, and the microvasculature, leading to myocardial fibrosis and functional impairment.[1]

Non-invasive methods, such as echocardiography and cardiac magnetic resonance imaging (MRI), have been used for heart imaging evaluation. Cardiac MRI is the gold standard and offers detailed insights into myocardial structure without the limitations of invasive procedures like endomyocardial biopsy.[2]

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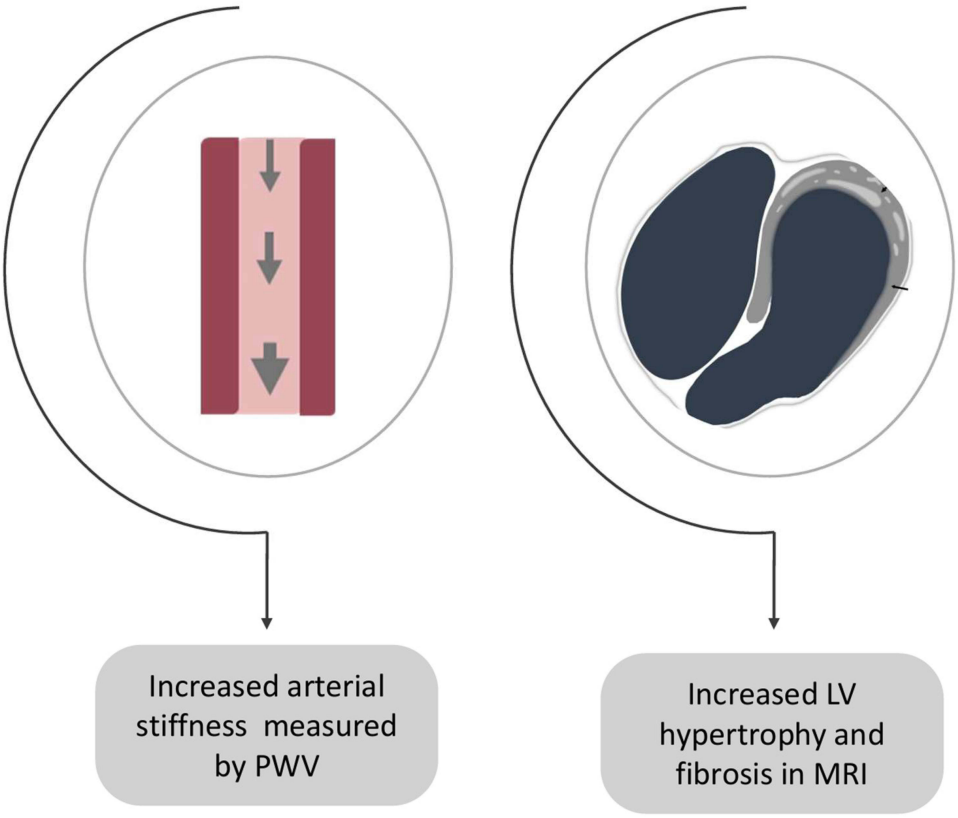
Graphical Abstract



Arterial stiffness, measured by pulse wave velocity (PWV), is a significant predictor of target organ damage and future cardiovascular events in hypertensive patients. Early detection and intervention are crucial to mitigate the progression of vascular damage and prevent further organ complications.[3]

Based on the existing literature, evidence shows the correlation between 24-hour BP, arterial stiffness and LV hypertrophy, evaluated by echocardiogram.[4–6] To our knowledge, these findings have not yet been confirmed before based on cardiac MRI, the gold standard for heart evaluation. In patients with myocardial hypertrophy, echocardiography often provides variable results due to high inter-observer variability. Specifically, echocardiography has been shown to overdiagnose LV hypertrophy in 15% of cases and miss it in 14%.[7] Cardiac MRI offers high reproducibility of measurements and facilitates easier and quicker evaluation of treatment, intramyocardial function, diastolic dysfunction, fibrosis, and ischemia. Importantly, it can provide a highly sensitive non-invasive assessment of ischemia through an adenosine stress-perfusion test.[8] Despite being the gold standard for assessing ventricular function and mass, cardiac MRI has not become a routine practice.[9]

Hence, the aim of this study is to evaluate if arterial stiffness can be correlated with abnormal findings from cardiac MRI imaging in an untreated hypertensive population. The secondary aim of the study was to identify any possible correlation between 24-hour ambulatory BP measurements and cardiac MRI imaging.

Methods

Study Population

The population of this cross-sectional study consisted of 22 individuals who attended the Hypertension 24-hour ABPM (Ambulatory Blood Pressure Monitoring) Center of Excellence. All participants, included in this analysis, were referred

to our Clinic by general practitioners due to reported increased BP measurements in the last 3 months. The participants had never been treated for hypertension before or during the study. The participants were extensively informed about the procedure and gave their informed consent to take part in this study. Patients aged less than 16 years old were excluded from the study. Other exclusion criteria were previous cardiovascular disease, dyslipidemia, diabetes mellitus, secondary hypertension, end-stage renal disease, and concomitant systematic or inflammatory diseases. The study was approved by the Ethics Committee of the Medical School at the Aristotle University of Thessaloniki in Greece, with approval number 420(3-5-2018).

PWV and Blood Pressure Measurements

Carotid-femoral (c-f) PWV was used as it is considered to be the gold standard method of measuring aortic stiffness.[3] C-f PWV was calculated by dividing the 80% of direct distance between the recording sites (carotid-femoral) by the transit time of the arterial pulse along the distance.[10] The Complior System® (Colson, Les Lilas, France) was used to provide the transit time, while the distances were measured by the researcher (CA) using a centimeter tape.[10] The measurements were performed according to the recommendations of the Artery Society and the ESH Working Group on Vascular Structure and Function,[10] and two measurements were in total performed for each participant. The final c-f PWV measurement was considered as the mean value of these two measurements.

Regarding office BP measurement, it was measured twice in the arm presenting the higher values of BP by the same investigator (CA), using a mercury sphygmomanometer. The average of these two measurements was considered as the office BP. All measurements were performed based on the ESH guidelines instructions.[10] The participants also underwent 24-hour ABPM (Spacelabs 90217, Spacelabs Inc., Redmond, WA, USA) on a typical working day. The appropriate size cuff was placed around the non-dominant arm, and three measurements were made, along with sphygmomanometric measurements to verify that the average of these values did not differ by more than 5 mmHg. The 24-hour ABPM is programmed to measure BP every 15 minutes during the day, and every 20 minutes during the night, to ensure at least 80 successful recordings.[3,10]

Cardiac Magnetic Resonance Imaging

Cardiac MRI examinations were performed on a GE SIGNA HDXT 1.5 T scanner and evaluated by a radiologist, specialized in cardiac MRI (KV). Standardized protocols were implemented to ensure consistency across participants.[2] Cardiac MRI was performed using cine steady-state free precession (SSFP) sequences for volumetric and functional assessments. Left ventricular (LV) end-diastolic volume (EDV) and end-systolic volume (ESV) were determined from short-axis cine images covering the entire left ventricle, and left ventricular ejection fraction (LVEF) was calculated accordingly. Systolic dysfunction was assessed using Cardiac MRI-derived LVEF and regional wall motion abnormalities (RWMA). LVEF was determined from cine steady-state free precession (SSFP) images in short-axis views covering the entire left ventricle. A threshold of LVEF < 55% was used to define systolic dysfunction, in line with established CMR reference ranges. RWMA was evaluated qualitatively based on visual assessment of myocardial segmental motion abnormalities, with hypokinesia or akinesia indicating dysfunction. Late gadolinium enhancement (LGE) imaging was employed to assess the presence of scarring within the cardiac interstitial space. The contrast agent utilized was Bayer's GADOVIST® 1.0 gadobutrol with an injection dosage of 604 mg/mL (1.0 mmol/mL), administered intravenously. Image acquisition commenced 10 minutes post-contrast injection using an inversion recovery prepared T1 weighted gradient echo sequence. To assess aortic function, phase-contrast imaging was performed at the level of the ascending thoracic aorta to measure aortic flow parameters and velocity. This imaging technique enabled evaluation of aortic wall dynamics and peak aortic valve velocity. Inversion time (TI) was patient-specific, ranging from 150 to 400 ms, and was manually optimized based on the myocardial signal intensity. In cases where a phase-sensitive inversion-recovery (PSIR) sequence was used, TI adjustments were performed automatically using a built-in reconstruction algorithm to ensure uniform myocardial suppression without the need for manual intervention. This algorithm enabled real-time optimization of contrast differences between normal and fibrotic myocardium, thereby improving fibrosis detection.

An experienced reader (KV), blinded to patient diagnosis and other findings, visually inspected the LGE images to determine the presence and extent of myocardial fibrosis, to identify end-diastolic volume and end-systolic volume for

both left and right ventricular, ejection fraction, myocardial thickness and ascending thoracic aorta. Peak aortic valve velocity was measured using phase contrast imaging in the ascending aorta. Myocardial thickness was measured at its maximum value in the basal interventricular septum in all patients. BSA-indexed values were not used, which may limit the reliability of such adjustments. All classifications were performed based on current guidelines for cardiac MRI.[2,9] Details for the classification of each parameter are presented on Supplementary materials (SM1).

Statistical Analysis

SPSS 25.0 (SPSS Inc., Chicago, IL, USA) was used to statistically analyze the data. Continuous variables are reported as the mean ± SD, and categorical variables as counts and percentages. Normality was evaluated with the Shapiro–Wilk test, which is more appropriate for small sample sizes ($n < 50$). BP values and c-f PWV values were reported as continuous variables, while findings from cardiac MRI (systolic and diastolic left ventricular dysfunction, fibrosis, myocardial thickness, ascending thoracic aorta and mitral regurgitation) were analyzed as categorical variables (as further analyzed in the SM1). Mann–Whitney and Kruskal–Wallis tests were used to compare continuous variables between groups due to the non-normal distribution of data. Logistic regression analysis was also performed to evaluate how PWV affects the findings of cardiac MRI (systolic and diastolic left ventricular dysfunction, fibrosis, myocardial thickness, and increased aortic valve velocity in the ascending aorta: yes/no). Odds ratios (OR) with 95% confidence intervals (CIs) were calculated for logistic regression results. A significance threshold of $p < 0.05$ was applied for all statistical tests. A post hoc power analysis was conducted to assess the adequacy of the sample size. No missing data were present in the dataset, and all analyses were conducted on complete cases. The calculated power was 73%, which is slightly below the conventional threshold of 80%. However, given the exploratory nature of this study and the relatively small sample size inherent in preliminary research, a power of 73% is considered acceptable in this context.

The manuscript has been written, using the STROBE cross sectional checklist.[11]

Results

A total of 22 untreated patients with hypertension were included in the present study, 9 females and 13 males, with a mean age of 42±13 years. The baseline characteristics of the study population, including demographics, anthropometric parameters (height, weight, BMI, waist and hip circumference), PWV and cardiac MRI parameters, are presented in Table 1. The participants experienced, as expected, increased BP values both in the office and during 24-hour ABPM measurements. Office blood pressure values were: SBP 145 ± 12 mmHg and DBP (diastolic blood pressure) 97 ± 13 mmHg. The 24-hour ABPM values were: 24-hour SBP (systolic blood pressure) 134 ± 12 mmHg, 24-hour DBP 77 ± 11 mmHg, daytime SBP 138 ± 13 mmHg, daytime DBP 81 ± 12 mmHg, nighttime SBP 123 ± 13 mmHg, and nighttime DBP 69 ± 10 mmHg.

Regarding cardiac function and myocardial structure, the left ventricular ejection fraction (LVEF) was preserved at 66.2 ± 2.5%, and maximum myocardial thickness was measured at 11.1 ± 1.4 mm in the basal interventricular septum. Systolic left ventricular (LV) dysfunction was detected in 7 patients (31.8%), while diastolic LV dysfunction was observed in 14 patients (63.6%). Myocardial fibrosis was present in 50% of the participants, with 7 patients (31.8%) categorized as having minimal myocardial scarring and 4 patients (18.2%) classified as having significant myocardial fibrosis. Cardiac MRI detected myocardial stiffness in 50% of the participants and increased aortic valve velocity in the ascending aorta in 10 (45.5%) patients. Arterial stiffness was increased with a mean c-f PWV of 11.5 ± 2.1m/s.

PWV, the gold-standard method for measuring arterial stiffness, was significantly different among patients with altered cardiac MRI parameters (Table 2). More specifically, PWV was increased in patients with systolic and diastolic LV dysfunction (10.6 ± 1.38, vs 13.4 ± 2.9, $p = 0.003$ and 10.2 ± 0.96, vs 11.8 ± 2.7, $p = 0.002$, respectively), myocardial stiffness (10 ± 1 vs 12.3 ± 2.8, $p < 0.001$] and fibrosis ($p = 0.004$) compared to those without the above. Regarding the latter, post-hoc analysis revealed that patients with minimal or more severe fibrosis had elevated PWV compared to those without fibrosis (12.8 ± 1.4, vs 10.4 ± 1, p =0 0.007, and 15.8 ± 2.6, vs 10.4 ± 1, $p = 0.011$, respectively), while the comparison between minimal and more severe fibrosis did not conclude to statistically significant results ($p = 0.186$). Arterial stiffness was also elevated in patients with increased or mildly increased peak aortic valve velocity in the

Table 1 Baseline Characteristics, Markers of Arterial Stiffness and Cardiac MRI Parameters in the Study Population

a. Baseline characteristics	
Age (years)	41.9±13
Male sex, % (n)	59.3% (13)
Height (cm)	170.5±9.5
Weight (kg)	75.9±13.5
Waist (cm)	93.3±11.5
Hip (cm)	105.5±10.1
Office SBP (mmHg)	145.0±12.0
Office DBP (mmHg)	97.0±12.0
Office HR (bpm)	83.0±13.0
24-hour SBP (mmHg)	134.0±12.0
24-hour DBP (mmHg)	77.0±11.0
Daytime SBP (mmHg)	137.0±13.0
Daytime DBP (mmHg)	81.0±12.0
Nighttime SBP (mmHg)	123.0±13.0
Nighttime DBP (mmHg)	69.0±10.0
Ejection fraction (%)	66.2±2.5
Maximum myocardial thickness (mm)	11.1±1.5
b. Markers of arterial stiffness	
PWV (m/s)	11.5±2.1
c. Cardiac MRI parameters	
Systolic left ventricular dysfunction, yes (n, %)	7 (31.8)
Diastolic left ventricular dysfunction, yes (n, %)	14 (63.6)
Fibrosis, minimal (n, %)	7 (31.8)
Fibrosis, fibrotic (n, %)	4 (18.2)
Myocardial stiffness, yes (n, %)	11 (50)
Peak aortic valve velocity in the ascending aorta, mildly increased (n, %)	7 (31.8)
Peak aortic valve velocity in the ascending aorta, increased (n, %)	3 (13.6)

Notes: Values are presented as mean ± SD or median (interquartile range) for continuous variables, or number (percentage) for categorical variables. Maximum myocardial thickness measured in the basal interventricular septum.

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; BPM, beats per minute; PWV, pulse wave velocity.

ascending aorta in comparison to those with normal peak aortic valve velocity (14 ± 2.8, vs 10.5 ± 1.15, p = 0.036, and 12.3 ± 2.1, vs 10.5 ± 1.15, p = 0.005, respectively).

Arterial stiffness was demonstrated to be correlated with cardiac MRI parameters (Table 3). In logistic regression analysis, PWV was significantly associated with both systolic and diastolic left ventricular dysfunction (p = 0.021 and

Table 2 Comparisons of PWV (m/s) Between Patients With Differences in Cardiac MRI Parameters

	Normal	Mildly Increased	Increased	p-value
Systolic LV dysfunction	10.6 (1.38)	NA	13.4 (2.9)	0.003
Diastolic LV dysfunction	10.2 (0.96)	NA	11.8 (2.7)	0.002
Myocardial stiffness	10.0 (1)	NA	12.3 (2.8)	<0.001
Myocardial fibrosis	10.4 (1)	12.8 (1.4)	15.8 (2.6)	0.004
Peak aortic valve velocity in the ascending aorta	10.5 (1.15)	12.3 (2.1)	14.0 (2.8)	0.006

Notes: Mann–Whitney test for 2 group comparisons and Kruskal–Wallis test for 3 groups comparisons were used. The number of participants in each group was as follows: Myocardial fibrosis groups - Normal: 11, Minimal Fibrosis: 7, Significant Fibrosis: 4. Aortic valve velocity groups - Normal: 12, Mildly Increased: 7, Increased: 3. Values are presented as median (interquartile range).
Abbreviations: LV, left ventricular; NA, not applicable.

Table 3 Logistic Regression Analysis of PWV and Cardiac MRI Parameters

Dependent Variables	Unadjusted β	EXP (β)	p-value	SE	Lower - Upper 95% CI
Systolic LV dysfunction No/yes	0.893	2.442	0.021	0.387	1.14–5.21
Diastolic LV dysfunction No/yes	1.558	4.749	0.046	0.781	1.03–21.94
Myocardial stiffness No/yes	3.17	23.814	0.040	1.544	1.16–490.41
Myocardial fibrosis No/minimal/fibrotic	1.589	4.9	0.036	0.758	1.11–21.63
Aortic valve velocity in the ascending aorta Normal/mildly increased/increased	1.478	4.386	0.034	0.697	1.12–17.19

Abbreviations: β, regression coefficient; SE, standard error; CI, confidence interval; LV, left ventricular.

p = 0.046, respectively), myocardial thickness (p = 0.04) and fibrosis (p = 0.036), as well as with aortic valve velocity in the ascending aorta (p = 0.034).

The investigation of potential associations between blood pressure levels and cardiac MRI findings revealed a significant correlation between 24-hour systolic BP levels and myocardial stiffness (p = 0.028). However, no other significant correlations were observed between office BP or 24-hour ABPM values and cardiac MRI parameters (p > 0.05 for all comparisons).

Discussion

The current study has corroborated the correlation between increased arterial stiffness, as measured by c-f PWV, and myocardial dysfunction, as identified through cardiac MRI. Specifically, augmented arterial stiffness was associated with increased myocardial stiffness, fibrosis, and LV dysfunction. Notably, there was also a positive relationship between 24-hour systolic BP measurements and increased myocardial stiffness in untreated hypertensive patients. These finding suggest that arterial stiffness and 24-hour ABPM may serve as a critical tool for predicting and managing myocardial complications in patients with hypertension. Consequently, this could provide a potential avenue for early intervention and the development of targeted treatment strategies to mitigate long-term cardiovascular risks.

Hypertension triggers a cascade of pathophysiological processes, particularly affecting the heart's structure and function. Persistent hypertension exerts mechanical stress on arterial walls, inducing endothelial dysfunction,

inflammation and oxidative stress, leading to increased arterial stiffness. Metrics of arterial stiffness are predictive of target organ damage and future cardiovascular events in hypertensive patients, highlighting the importance of early detection to initiate preventive and therapeutic measures aimed at mitigating vascular damage progression. PWV remains the gold standard for evaluating arterial stiffness and has been recognized in the latest European Society of Hypertension guidelines as a marker of hypertensive target organ damage.[12]

Hypertensive heart disease encompasses a range of morphological and functional abnormalities in the LV found in patients with arterial hypertension, primarily characterized by LV hypertrophy not attributable to other causes. The LV adapts to the hemodynamic wall stress caused by high blood pressure, or pressure overload, by thickening its walls and increasing its mass, leading to concentric LV hypertrophy. Mechanical stress from hypertension-induced LV pressure overload plays a key role, mediating mechanotransduction signaling and leading to fibroblast activation and differentiation into myofibroblasts. Additionally, the extracellular matrix serves as a reservoir for profibrotic signaling molecules released in response to mechanical stress. Then, myocardial fibrosis, characterized by the excessive accumulation of collagen fibers within the myocardium, occurs in the hypertensive heart. This chronic progression deteriorates the myocardium's architecture and properties, resulting in clinical complications, such as cardiac dysfunction, arrhythmias, and reduced coronary flow reserve, all linked to poor outcomes. Myocardial fibrosis promotes further LV diastolic dysfunction by increasing LV stiffness and filling pressure, while it also contributes to LV systolic dysfunction.[12–14]

Multiple studies have demonstrated that arterial stiffness is associated with target organ damage, especially LV hypertrophy, as detected by transthoracic echocardiography, and elevated risk for cardiovascular events.[15–17] Of note, a recent meta-analysis showed that after antihypertensive therapy, there is a significant positive correlation between arterial stiffness and reduction of LV mass, expressed as a relevant reduction in LV mass index of 6.9 g/m per 1.0 m/s reduction in PWV.[18] These findings show that arterial stiffness is an important tool not only for the diagnosis but also for the follow-up of these patients.

Regarding hypertension per se, most parameters from ambulatory BP were found to be correlated with LV mass index and LVH, evaluated by two-dimensional echocardiography.[19] This correlation has been also revealed in the pediatric population.[20] Finally, further to the BP values recorded by ABPM, the hypertension phenotype seems also to matter. Masked hypertension and non-dipping status were also determinants of LVH, as further proof that ABPM could be used for the guidance of the hypertensive population.[19] Moreover, Chen et al showed that patients with a non-dipper pattern of hypertension had a higher LV mass index, greater prevalence of both eccentric and concentric LV hypertrophy, more severe impairment of LV diastolic and systolic function, and increased peripheral arterial stiffness, than those with a dipper pattern of hypertension.[21] Isolated nocturnal has also been linked to arterial stiffness, LV hypertrophy and diastolic dysfunction in 1734 normotensive and hypertensive participants.[22]

Cardiac MRI is widely acknowledged as the non-invasive gold standard for evaluating LV volumes, systolic function, LV mass, and myocardial tissue characterization.[23] Its three-dimensional capabilities, combined with excellent spatial resolution and high tissue contrast, facilitate precise measurement of cardiac function and morphology, including LV volumes, mass, and ejection fraction, as well as the assessment of regional wall motion abnormalities without relying on geometric assumptions.[24] Additionally, cardiac MRI enables the assessment and quantification of focal and diffuse myocardial fibrosis caused by hypertension, thus allowing for the detection of hypertension-mediated organ damage and secondary causes of hypertension.[25] Currently, cardiac MRI using native T1 mapping and extracellular volume quantification is considered the gold standard imaging method for evaluating myocardial fibrosis. Although T1 mapping and extracellular volume fraction (ECV) are valuable for fibrosis quantification, late gadolinium enhancement (LGE) remains a widely validated method for detecting myocardial fibrosis. Given the study's primary focus on structural myocardial changes in hypertension, LGE was selected as it provides a well-established qualitative and semi-quantitative evaluation of fibrosis. Future studies may incorporate T1 mapping to enhance fibrosis characterization.[9,26,27]

Numerous studies have demonstrated that cardiac magnetic resonance imaging (MRI) provides a more accurate evaluation of left ventricular (LV) hypertrophy in comparison to two-dimensional transthoracic echocardiography, which is the most used diagnostic tool in clinical practice.[28,29] However lately, 3-dimensional echocardiography has shown similar sensitivity to MRI.[30,31]

To our knowledge, this is the first study in which arterial stiffness and 24-hour ABPM was conducted to assess the impact of hypertension in cardiac function, as visualized by cardiac MRI. To minimize the detection of bias, one investigator was responsible for the BP measurements and c-f PWV measurements, and a blinded investigator evaluated the findings from cardiac MRI. Further strength of the study includes that all patients were undergone ABPM to exclude false positive diagnosis of hypertension and the patients were newly diagnosed as hypertensives, never treated before to exclude treatment bias.

However, a key limitation of this study is its cross-sectional design, which prevents conclusions about causality between arterial stiffness and myocardial dysfunction, as it can only demonstrate associations rather than establish causal relationships. Additionally, BSA-indexed values were not used, as indexing could introduce additional variability. Future longitudinal studies are necessary to confirm the directionality of these associations. One more limitation was that myocardial stiffness was evaluated qualitatively rather than quantitatively using the tagging technique in CMR. This was due to the unavailability of dedicated software for strain analysis at the time of the study. Future research should consider incorporating quantitative tagging analysis for a more detailed assessment of myocardial mechanics. The relatively small sample size and the lack of longitudinal follow-up, also limit the ability to observe long-term outcomes. Longitudinal or interventional studies are necessary to determine whether arterial stiffness directly contributes to myocardial dysfunction or if these findings reflect shared pathophysiological mechanisms. The study included only patients of Caucasian origin, and the results may not be extended to other ethnicities. Larger and follow-up studies are needed to further evaluate the results of the current study.

To conclude, this study reported the strong positive correlation between c-f PWV with both systolic and diastolic LV dysfunction, myocardial stiffness and fibrosis and aortic valve velocity in the ascending aorta, as well as between 24-hour systolic BP measurements and increased myocardial stiffness, in newly diagnosed untreated hypertensives. These findings reveal the importance of arterial stiffness, especially in the first diagnosis of hypertension. By evaluating all hypertensives with 24-hour ABPM and c-f PWV at least in the first diagnosis, we can refer for further heart damage evaluation only those patients with increased arterial stiffness. By this, we may have a more focused screening, reducing also the healthcare cost. However, future research is needed to confirm these findings in a larger population.

Abbreviations

ABPM, ambulatory blood pressure monitoring; BP, blood pressure; C-F, carotid-femoral; DBP, diastolic blood pressure; ESH, European Society of Hypertension; LGE, late gadolinium enhancement; LV, left ventricle; MRI, magnetic resonance imaging; PWV, pulse wave velocity; SBP, systolic blood pressure.

Ethics

The study was approved by the Aristotle University Ethics Committee and held in accordance with the Declaration of Helsinki. Informed consent was obtained from all individual participants included in the study.

Disclosure

The authors declare no conflicts of interest.

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