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Research Article

Does Aortic Propagation Velocity Predict Subclinical Atherosclerosis in Prehypertensive Patients?

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ABSTRACT

Objective: Predictors of subclinical atherosclerosis include carotid intima-media thickness (cIMT) and epicardial adipose tissue (EAT). The risk of atherosclerosis increases owing to hypertension. The aim of the present research was to determine the association between aortic propagation velocity (APV), cIMT, and EAT in patients with prehypertension.

Methods: This research included 208 patients. Two groups were generated: prehypertensive and control. The APV, cIMT, and EAT values were also recorded. The correlations between these parameters were also analyzed.

Results: Statistically significant differences were found between the two groups in the EAT, cIMT, systolic blood pressure (SBP), diastolic blood pressure (DBP), and APV variables ($P < 0.05$). A statistically significant inverse association was found among EAT, cIMT, SBP, DBP, and APV and the atherosclerosis surrogate markers ($P < 0.05$). Linear regression analysis revealed a close relationship between the EAT, cIMT, and APV.

Conclusion: In prehypertensive patients, APV as a non-invasive method may be helpful in identifying individuals predisposed to atherosclerosis.

Keywords: Aortic propagation velocity, subclinical atherosclerosis, prehypertension

INTRODUCTION

Hypertension (HT) is correlated with elevated cardiac mortality and morbidity.[1] For diagnosing HT, staging is performed based on the current guidelines, and treatment management follows the staging.[2] It is known that even early-stage HT is associated with coronary artery disease (CAD). In addition, cardiovascular death and morbidity increase proportionally with increasing systolic blood pressure (SBP) pressure and diastolic blood pressure (DBP) values.[3] In this respect, it is important to identify individuals who may develop HT at an early stage and initiate appropriate lifestyle changes and treatment in terms of risk reduction.

Worldwide, CAD remains a principal cause of death.[3,4] Atherosclerosis, which plays an important role in CAD pathophysiology, becomes subclinical before the disease manifests.[5] Therefore, it is extremely important to diagnose diseases that cause such high mortality in the subclinical stage and to initiate lifestyle changes and various drug therapies to prevent atherosclerotic disease.[5-8] The link between epicardial adipose tissue (EAT) and CAD has been well established. Many studies have shown an association between EAT and CAD severity.[9-12] Increased carotid intima-media thickness (cIMT) is correlated with the onset of future cardiac events. It is considered an indirect parameter that indicates atherosclerosis

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burden and subclinical atherosclerosis.[9,13] In this context, EAT and cIMT are current non-invasive parameters that are used as predictors of atherosclerosis.

Atherosclerosis is an early onset, systemic, and progressive disease that affects the large muscular arteries. Atherosclerosis causes stiffening and thickening of the arterial wall, resulting in increased arterial resistance. Increasing arterial resistance results in a reduction in the blood flow within the arterial lumen. Studies have revealed that the color M-mode Doppler flow propagation speed of the descending aorta, namely, the aortic propagation velocity (APV), is associated with coronary atherosclerosis.[14-16] We aimed to evaluate cIMT and EAT, which are predictors of subclinical atherosclerosis, to investigate the association between these predictors and APV; and the ability of APV to predict atherosclerosis, in prehypertensive patients. This study represents the first effort to assess APV, cIMT, and EAT in prehypertensive patients, to the best of our knowledge.

MATERIAL AND METHODS

Our study included patients examined in a cardiology outpatient clinic between March and June 2019. A total of 101 apparently healthy volunteers with no known history of cardiovascular, renal, hepatic, or systemic inflammatory disease were prospectively enrolled as the control group. In addition, 107 patients aged 18-80 years who met the diagnostic criteria for prehypertension and had no other comorbidities were included in the prehypertensive group. Erzurum Regional Training and Research Hospital Ethical Committee provided approval for this study (approval no.: 2019/07-62, date: 15.09. 2019). All participants provided informed consent prior to enrollment. The exclusion criteria were a) diabetes mellitus, b) infection, c) chronic obstructive pulmonary disease, d) drug use for chronic disease, e) a history of cancer, f) peripheral arterial disease, and g) CAD.

Definitions

Patients were diagnosed with prehypertension based on the current 8th Joint National Committee criteria.[17] An ambulatory blood pressure measurement device was connected to all patients for 24 hours. The mean DBP values between 80-89 mmHg and mean SBP values between 120-139 mmHg were considered prehypertension. The mean values of the control group, i.e. those with normal blood pressure values, were <120/80 mmHg in the ambulatory blood pressure measurement.

MAIN POINTS

- Hypertension contributes to the risk of atherosclerotic cardiovascular disease. In this context, prehypertension is a clinically important condition.
- We found a negative correlation between aortic propagation velocity and predictors of subclinical atherosclerosis such as carotid intima-media thickness and epicardial adipose tissue.
- Finally, APV may be used as a predictor of subclinical atherosclerosis in prehypertension.

Participants' smoking habits were noted based on whether they were already active smokers. Body mass index measurements were obtained for all patients.

Analysis of Blood Samples

Blood samples collected for the study were drawn from the antecubital vein with minimal venous stasis after 12 hours of fasting. Blood samples for complete blood counts were stored in tubes containing potassium EDTA. Counts of white blood cells (WBC), hemoglobin concentrations, and platelet numbers were evaluated using the electrical impedance method with a fully automatic hematology analyzer (Beckman Coulter LH 780). Albumin, serum lipid profile, creatinine, glucose, sodium, potassium, and calcium levels were measured using standard laboratory methods.

Echocardiography

Echocardiographic measurements were performed using Vivid 7 GE (GE Healthcare, Little Chalfont, UK) and a 2.5 MHz frequency transducer. Echocardiographic recordings, were obtained from standard apical and parasternal views, in the left lateral position at the end of expiration in three cardiac cycles. Echocardiographic examinations were carried out following the American Society of Echocardiography (ASE) standards.[18] The left ventricular (LV) ejection fraction was calculated using the modified Simpson method. After routine echocardiographic evaluation, the "cursor" was positioned along the axis to the blood flow, in the descending aorta, and colored M-mode records were obtained from the suprasternal window. The flame-shaped M-mode velocity records were obtained (Figure 1). An appropriate aliasing velocity was selected for each patient to evaluate the velocity slope clearly. APV values were obtained by plotting the velocity slope and dividing the time between the beginning and end of this slope into intervals. The mean of the three measurements was considered the APV.

cIMT and EAT Measurement

For cIMT measurements, patients were positioned supine with their head tilted backward. The bilateral carotid arteries were imaged using the Vivid S5 ultrasound device (GE Vingmed Ultrasound AS, Norten, Norway) with a 7.5 MHz linear probe. cIMT was obtained according to the protocol published by ASE.[19] The main carotid artery, internal carotid artery, and carotid bulb were examined in all the patients. cIMT measurements were made from the distal posterior wall, approximately 1 cm from the bifurcation, using the echogenicity of the lumen-intima and media-adventitia surfaces of both main carotid arteries. At least three measurements were performed. Segments with atherosclerotic plaques were not used for measurements. The EAT was measured from the hypoechoic gap between the visceral pericardium and outermost border of the right ventricular (RV) myocardium from the parasternal long-axis view at end-diastole. The largest diameter of the EAT, which was located on the RV free wall, was determined as previously described.

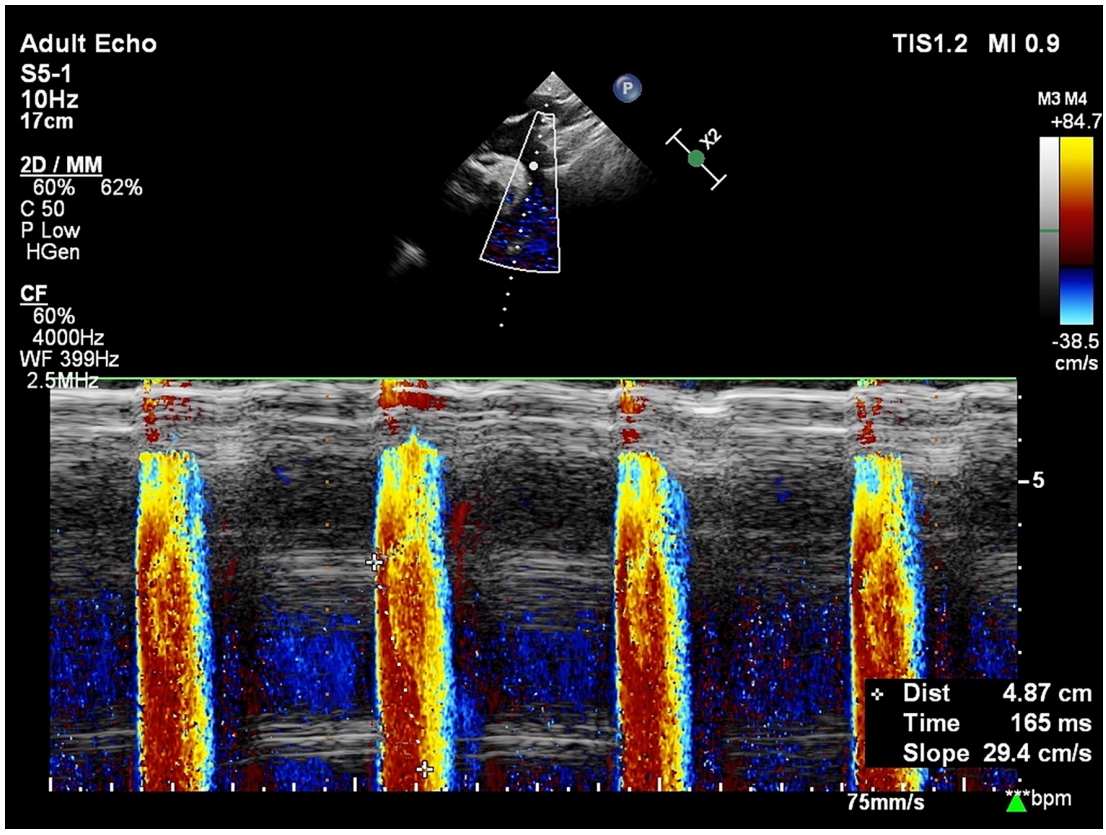

Figure 1. An example image for M-mode velocity record aortic propagation velocity.

Statistical Analysis

Data analysis was conducted using SPSS version 22 (IBM, Chicago, IL, USA). Data are expressed as continuous variables with mean ± standard deviation and as categorical variables with frequency and percentage. The compatibility of continuous variables with a normal distribution was tested using the Kolmogorov-Smirnov test. In the evaluation of continuous variables between the patient and control groups, Student's t-test was used for those meeting parametric assumptions, and the Mann-Whitney U test was used for data not meeting parametric assumptions. Categorical variables between the two study groups were assessed using Pearson's chi-square test. Correlations between two continuous variables were tested using Pearson's correlation test. Variables that were significant in the univariate analyses, were subjected to multivariate linear regression analysis to determine whether the EAT, SBP, DBP, and cIMT values were independently associated with APV. Statistical significance was set at $P < 0.05$.

RESULTS

This study comprised 208 patients in total. Patient characteristics and laboratory and echocardiographic findings are displayed in Table 1. The mean age of the prehypertensive group was 55.5 ± 7.7, 65% were male, while the mean age of the control group was 54.3 ± 6.1 and 62% were male. Comparisons between the two groups revealed no significant differences in age, gender, smoking habits, body mass index, or serum electrolyte levels (all $P > 0.05$). Nonetheless, multiple clinical parameters showed statistically significant differences between the groups. Prehypertensive patients had significantly higher EAT,cIMT, SBP, and DBP than controls ($P = 0.004$, the others $P < 0.001$ respectively). The APV was significantly lower in the prehypertensive group compared to controls (42.5 ± 12.5 cm/s vs. 55.2 ± 10.3 cm/s, $P < 0.001$), indicating increased aortic stiffness in the prehypertensive patients. The comparison revealed no significant differences between the groups in LV ejection fraction, interventricular septal thickness, LV end-diastolic diameter , LV systolic dimension, or systolic pulmonary artery pressure (all $P > 0.05$). Laboratory tests, including glucose, albumin, total cholesterol, LDL, HDL, and triglyceride levels, exhibited no significant variation between the two groups (all $P > 0.05$). Hematological parameters, such as hemoglobin, platelet count, and WBC count, also showed no significant differences (all $P > 0.05$).As shown in Table 2, correlation analysis showed that APV was inversely correlated with EAT ($r = -0.38$, $P < 0.001$), cIMT ($r = -0.48$, $P < 0.001$), SBP ($r = -0.31$, $P < 0.001$), and DBP ($r = -0.30$, $P < 0.001$). These findings suggest that increased EAT, cIMT, and elevated blood pressures are linked with reduced APV, which indicates a relationship between increased vascular stiffness and lower APV. This finding highlights the potential utility of APV as an indicator of subclinical vascular changes, particularly in patients with prehypertension and early signs of arterial stiffness. In univariate analysis, EAT ($P = 0.004$) and cIMT ($P < 0.001$) were significantly associated with reduced APV. Specifically, for each 1 mm increase in EAT, the odds of reduced APV increased by a factor of 4.3 (OR = 4.3, 95% CI: 1.2-8.1), and for each 1

mm increase in cIMT, the odds of reduced APV increased by a factor of 3.6 (OR = 3.6, 95% CI: 1.2-7.3). SBP and DBP also showed significant associations with APV, with odds ratios of (OR = 1.6, 95% CI: 1.1-2.5) and (OR = 1.3, 95% CI: 1.1-2.1), respectively. As shown in Table 3, in the multivariate analysis, significant associations persisted for EAT and cIMT, with EAT remaining a significant predictor of APV (*P* = 0.046, OR = 2.6, 95% CI: 0.9-4.17), and cIMT (*P* = 0.009, OR = 1.9, 95% CI: 1.1-3.3). However, SBP and DBP were not statistically significant in the multivariate model (SBP: *P* = 0.128; DBP: *P* = 0.389).

Table 1. Comparison of Baseline Characteristics, Laboratory and Echocardiographic Findings of the Study Population Between Study Groups

Variables	Prehypertensive (n=107)	Control (n=101)	*P*
Age, years	55.5 ± 7.7	54.3 ± 6.1	0.821
Gender, male %	65	62	0.418
Smoking, %	41	39	0.369
EAT, mm	7.8 ± 2	5.9 ± 2	**0.004**
cIMT, mm	1.1 ± 0.3	0.7 ± 0.2	**< 0.001**
Systolic blood pressure, mmHg	131 ± 5.7	119 ± 5.1	**< 0.001**
Diastolic blood pressure, mmHg	83 ± 7.1	75 ± 6.3	**< 0.001**
LV-EF, %	60 ± 5.9	62 ± 5.4	0.239
IVS, mm	11.3 ± 0.4	10.4 ± 0.3	0.683
LVEDD, mm	45 ± 8.9	44 ± 5.5	0.394
LVSDD, mm	27 ± 6.2	26 ± 8.7	0.691
sPAB,mm Hg	23 ± 6.3	21 ± 4.7	0.831
APV, cm/s	42.5 ± 12.5	55.2 ± 10.3	**< 0.001**
BMI, kg/m^2	24 (21-29)	24 (20-28)	0.621
GFR, mL/min/1.73 m^2	89 (73-115)	86 (71-116)	0.407
Glucose, mg/dL	88 (79-104)	92 (81-101)	0.528
Na	141 ± 4.9	140 ± 4.1	0.372
K	3.9 ± 0.5	3.8 ± 0.4	0.815
Ca	8.9 ± 1.3	8.7 ± 1.4	0.228
AST	35 (21-45)	37 (22-46)	0.393
ALT	33 (24-40)	33 (25-41)	0.459
Albumin	3.5 ± 1.1	3.5 ± 1.2	0.156
Total cholesterol, mg/dL	150 (120-189)	145 (114-174)	0.409
LDL-cholesterol, mg/dL	93 (72-118)	91 (71-121)	0.832
HDL-cholesterol, mg/dL	36 (31-42)	32 (29-35)	0.281
Triglyceride, mg/dL	102 (81-123)	99 (80-120)	0.125
Hemoglobin, g/dL	15.3 ± 3.2	15.6 ± 3.7	0.916
Platelet count, 10^3/L	342 (230-420)	304 (221-412)	0.609
White blood cell count, 10^3/L	6.3 (3.4-8.3)	5.1 (3.1-8.1)	0.791

EAT, epicardial adipose tissue; cIMT, carotid intima-media thickness; LV-EF, left ventricular ejection fraction; cIMT, carotid intima-media thickness; IVS, interventricular septum; LVEDD, left ventriculer end diastolic diameter; LVSDD, left ventriculer systolic dimension; sPAB, systolic pulmonary artery pressure; APV, aortic propagation velocity; BMI, body mass index; GFR, glomerular filtration rate; Na, sodium; K, potassium; Ca, calcium; AST, aspartat aminotransferaz; ALT, alanin aminotransferaz; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

Table 2. Correlation of Aortic Propagation Velocity with Blood Pressure and Surrogate Markers

Variables	APV	
	***r* value**	***P* value**
EAT; mm	-0.38	**< 0.001**
cIMT,mm	-0.48	**< 0.001**
Systolic blood pressure	-0.31	**< 0.001**
Diastolic blood pressure	-0.30	**< 0.001**

APV, aortic propagation velocity; EAT, epicardial adipose tissue; cIM, carotid intima-media thickness.

Table 3. Association of APV with Atherosclerotic Surrogate Markers and Blood Pressure in Linear Regression Analysis

Variables	Univariate *P* value	Univariate OR	Multivariate *P* value	Multivariate OR
EAT, mm	0.004	4.3 (1.2-8.1)	**0.046**	2.6 (0.9-4.17)
cIMT, mm	< 0.001	3.6 (1.2-7.3)	**0.009**	1.9 (1.1-3.3)
SBP	< 0.001	1.6 (1.1-2.5)	0.128	1.3 (0.89-1.6)
DBP	< 0.001	1.3 (1.1-2.1)	0.389	1.3 (0.82-1.9)

OR, odds ratio; EAT, epicardial adipose tissue; cIMT, carotid intima-media thickness; SBP, systolic blood pressure; DBP, diastolic blood pressure.

DISCUSSION

The findings of our study can be summarized as follows:

The prehypertensive group had significantly higher EAT and cIMT values. In this context, there was a higher tendency for atherosclerosis in the prehypertensive patients versus the control group.

APV is intimately related to the surrogate markers of atherosclerosis in patients with prehypertension. In our study, APV was used as a surrogate marker for atherosclerosis in this patient group.

HT is a key risk factor for the development of atherosclerotic heart disease.[20] Its pathophysiology includes conditions such as endothelial dysfunction and activation of the sympathetic system. HT is a leading cause of chronic renal failure and cardiovascular disorders (coronary artery-peripheral vascular disease and cardiac failure) in developed and developing countries. The risk of cardiovascular fatality and myocardial infarction is threefold higher in patients compared to those without the condition.[21] Prehypertension was defined as SBP between 120-139 mmHg and DBP between 80-89 mmHg.[17] Both HT incidence and cardiovascular (mortality and morbidity) rates have increased in the long term in patients with prehypertension. A study by Uçar et al.[22] showed that coronary complexity may be increased in patients with HT. Another study showed that cardiovascular morbidity and mortality increased directly proportional to the increase in blood pressure values.[23]

Ultrasonographic quantification of cIMT is an inexpensive, reliable, and repeatable method that can be used to evaluate subclinical atherosclerosis. It has been suggested that cIMT is closely linked to conventional cardiovascular risk factors and may predict future cardiac diseases such as myocardial infarction and stroke.[24,25]

Atherosclerosis is a systemic and intensifying disease that starts at an early age and affects large muscular arteries such as the thoracic aorta. Atherosclerosis causes condensation and stiffening of the arterial wall, resulting in amplified arterial resistance. Increased arterial resistance results in a decreased rate of blood flow propagation within the arterial lumen, which can be measured using non-invasive methods.[14,26] Among these measurements, the APV is the most widely studied. APV is a Doppler echocardiographic measure indicates the elasticity and compliance of the descending aorta. A reduction in APV suggests increased stiffness of the aorta, which has been linked to negative cardiovascular outcomes, such as LV diastolic dysfunction, myocardial ischemia, and a heightened risk of arrhythmias due to changes in ventricular-arterial coupling. In the literature, there is evidence suggesting that atheroma plaque in the aorta may be a marker of generalized atherosclerosis. Tribouilloy et al.[27] described a strong relationship between the presence and amount of CAD and the manifestation of atherosclerotic plaques detected by transesophageal echocardiography in the thoracic aorta. Arterial resistance increases due to the thickening and hardening of the arterial wall caused by atherosclerosis. Increased aortic resistance due to atherosclerosis in the descending aorta decreased the flow propagation rate. Thus, as the severity of atherosclerosis in the descending aorta increases, APV values decrease. Various studies indicate that APV may have a close association with cardiovascular conditions, notably coronary atherosclerosis. In an observational case-control study by Oğuz et al.[14], decreased aortic flow propagation velocity was associated with increased epicardial adipose thickness. Similarly, in our study, APV values decreased and EAT values increased. In addition, Vasudeva Chetty et al.'s [15] cross-sectional comparative study of 100 patients showed that APV and cIMT were associated with CAD burden. In a study of 93 patients by Sen et al.[28], it was shown that APV can help in the non-invasive assessment of cardiovascular risks and in identifying high-risk individuals for CAD. We found that the cIMT and EAT values were higher in patients with prehypertension. In other words, patients with prehypertension may be indirectly prone to atherosclerosis. In addition, there was a relationship between APV and predictors of atherosclerosis in prehypertensive patients. This finding is valuable because it shows that prehypertension may be an atherosclerotic precursor and demonstrates the utility of APV as a surrogate marker of atherosclerosis in prehypertensive individuals. The study's limitations involve a small sample size and the lack of coronary computed tomography and angiography, which are instrumental in diagnosing CAD.

In conclusion, in patients with prehypertension, APV is closely associated with cIMT and EAT, which are surrogate markers of atherosclerosis. In this patient group, APV as a non-invasive method may be helpful in predicting atherosclerosis.

Ethics

Ethics Committee Approval: Erzurum Regional Training and Research Hospital Ethical Committee provided approval for this study (approval no.: 2019/07-62, date: 15.09. 2019).

Informed Consent: All participants provided informed consent prior to enrollment.

Footnotes

Author Contributions

Concept - K.K. H.K.; Design - K.K. H.K.; Supervision - K.K. H.K.; Fundings - K.K. H.K.; Materials - K.K. H.K.; Data Collection and/or Processing - K.K. H.K.; Analysis and/or Interpretation - K.K. H.K.; Literature Search - K.K. H.K.; Writing - K.K. H.K.; Critical Review - K.K. H.K.

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