Aortic propagation velocity does not correlate with classical aortic stiffness parameters in healthy individuals

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ABSTRACT

Objective: Aortic stiffness is an important cardiovascular risk marker, which can be determined using different noninvasive techniques. Aortic propagation velocity (APV) has recently been established as a novel echocardiographic parameter of aortic stiffness. This study aimed to investigate the association between APV and the classical echocardiography-derived aortic stiffness parameters, aortic distensibility (AD) and aortic strain (AS), in a group of otherwise healthy individuals.

Methods: In total, 97 consecutive healthy subjects were recruited in this observational study. APV was measured using color M-mode echocardiography from the suprasternal window in the descending aorta. AS and AD were calculated using clinical blood pressure and the M-mode echocardiography-derived aortic diameters. Correlation analyses were performed between cardiovascular risk factors related to increased aortic stiffness (age, obesity, and blood pressure) and measured stiffness parameters (APV, AS, and AD). Correlation analyses were also performed among the measured stiffness parameters.

Results: Good correlation of age, blood pressure, and BMI with AS and AD was observed. One-on-one correlation of age, blood pressure, and BMI with APV was not observed. No correlation was observed between APV and AS (r=–0.05, p=0.6) or between APV and AD (r=–0.17, p=0.8).

Conclusion: Although APV has been proposed as a novel and practical echocardiographic parameter of aortic stiffness, especially in patients with coronary artery disease, correlations between classical stiffness parameters (AS and AD) and APV were absent in healthy individuals at low–intermediate risk. The clinical and research applicability of APV should be further evaluated. *(Anatol J Cardiol 2017; 18: 340-6)*

Keywords: aortic stiffness, echocardiography, aortic propagation velocity

Introduction

Previous reports have clearly documented that aortic stiffness is associated not only with coronary artery disease (CAD) but also with cardiovascular risk factors, such as hypercholesterolemia, smoking, older age, hypertension, impaired glucose tolerance and diabetes mellitus (DM), and obesity (1-8). Increased aortic stiffness has been reported as an independent predictor of cardiovascular mortality and morbidity (9, 10) as well as of CAD severity (11,12). Atherosclerosis-related cardiovascular diseases have a clinical course starting from endothelial dysfunction and progressing to clinically evident diseases. In addition to the prediction of cardiovascular events in a patient with established cardiovascular disease, rationale for evaluating aortic stiffness includes early detection and risk stratification of a subject at a risk of cardiovascular disease. Therefore, current guidelines recommend the assessment of aortic stiffness by the aortic pulse-wave velocity (PWV) method to evaluate cardiovascular risk

(13). Although PWV is considered as the gold-standard method to determine arterial stiffness, the necessities of trained medical staff and special devices reduce its potential usage. Accordingly, other echocardiography-derived stiffness parameters, such as aortic strain (AS), aortic distensibility (AD), stiffness index, augmentation index, and more recently, color M-mode derived propagation velocity of the descending thoracic aorta (APV), have been analyzed in the context of the association between aortic stiffness and CAD in numerous studies (11,12, 14-17).

AD and AS, which have been widely used as noninvasive aortic stiffness parameters in clinical trials, reflect the mechanical properties and elasticity of the aorta. AS reflects the percent change in vessel diameter, whereas AD denotes the effect of pulse pressure in any alteration in the vessel diameter. AS and AD measurements do not require special devices and trained staff; however, they require some additional computations.

Evolved recently, APV is considered as a practical and sensitive aortic stiffness parameter (17). The clinical value of APV in

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patients with coronary and carotid atherosclerosis and other associated clinical conditions, such as endothelial dysfunction and hypertension, has been the subject of several studies (17-22). In addition, the association of APV with other stiffness parameters has been studied in conditions related to increased stiffness, such as obstructive sleep apnea syndrome and newly diagnosed type 2 DM (23, 24). However, no study has evaluated these parameters in low-risk groups. Accordingly, the present study aimed to evaluate the correlation of APV with the aortic stiffness parameters AS and AD in a population with a relatively low risk of cardiovascular disease. This cohort can also be considered as candidates to benefit from early detection and risk stratification for cardiovascular disease. We also evaluated the association of each studied parameters, such as age, obesity, and blood pressure, with known risk factors for increased stiffness.

Methods

Study population

This cross-sectional study consisted of 97 consecutive healthy individuals (50 males and 47 females) undergoing routine health check-up at the Department of Cardiology Outpatient Clinic at Süleyman Demirel University School of Medicine between July and December in 2015. Clinical data obtained from patient interviews included past medical history and classical cardiovascular risk factors. Exclusion criteria were as follows: age of $\geq$50 years, known hypertension and DM, left ventricular (LV) ejection fraction of <50%, severe diastolic dysfunction (E/Em >15), moderate-to-severe valvular heart disease, arrhythmia, aortic disease, known coronary artery disease or presence of two or more classical risk factors for CAD, chronic inflammatory or connective tissue disease, chronic pulmonary disease, renal failure (GFR <90 ml/min/1.73 m^2) and inadequate acoustic windows. These parameters were considered as exclusion criteria because these conditions might have influenced the outcomes of some of the measured parameters. None of the participants were taking any medications.

Hypertension, DM, and hypercholesterolemia were either self-reported or determined on the basis of current treatment. Diagnostic ranges for newly diagnosed hypertension (>140/90 mm Hg), DM (fasting glucose level >126 mg/dL), and hyperlipidemia (total cholesterol >200 mg/dL or triglycerides >150 mg/dL) were formed according to existing guidelines. Severe diastolic dysfunction was determined using the echocardiographic examination mainly on the basis of tissue Doppler imaging (TDI) method (E/Em >15). Positive family history of CAD was defined as CAD diagnosed in a first-degree relative before the age of 55 years for men and 65 years for women. Individuals who had smoked more than 100 cigarettes in their entire lifetime were considered as smokers. Echocardiographic examinations were performed at least 2 h after the smokers in the cohort smoked the last cigarette. Waist circumference was measured at the level of the umbilicus in the standing position. All participants

provided informed consent to participate in this study. The Institutional Ethics Committee approved this study.

Transthoracic echocardiographic examination

All subjects underwent detailed echocardiographic examination by a single experienced cardiologist using a Philips I-E33 imaging system (Andover, Massachusetts, USA) with a 2.5 MHz transducer. Conventional echocardiographic measurements were obtained using two-dimensional, color flow, pulse- and continuous-wave Doppler modalities. The longitudinal mitral annular velocities from the apical four-chamber view were recorded using TDI echocardiography for further assessment of LV diastolic function. Systolic and diastolic diameters of the ascending aorta and aortic propagation velocity were recorded. All echocardiographic measurements were obtained for at least three consecutive beats and averaged.

Aortic diameter measurements were obtained in the parasternal long-axis view by placing the M-mode sampling line 3 cm above the aortic valve (18). Diastolic diameter (DD) was measured at the peak of the QRS complex of the simultaneously recorded electrocardiogram, and systolic diameter (SD) was measured at the time of full opening of the aortic valve (Fig. 1a).

AS and AD were calculated according to previously defined formulas using aortic diameters and blood pressure as follows (18):

$$AS \ (\%) = \frac{\text{Systolic - Diastolic diameter}}{\text{Diastolic diameter}} \times 100$$

$$AD \ (cm^2/dyn) = \frac{2 \times AS}{\text{Systolic pressure - Diastolic pressure}}$$

Concomitantly, blood pressure was measured using the ausculatory method after the echocardiographic study, ensuring that the subjects had rested for at least 5 min.

APV measurement was performed using color M-mode Doppler recordings obtained from the suprasternal window in a supine position, with the cursor parallel to the main flow of direction in the descending aorta, as previously described (17). The color Doppler Nyquist limit was adapted to 30–50 cm/s, with switching to the M-mode with a recorder sweep rate of 150 mm/s. Subsequently, aliasing velocity was shifted until a clear delineation of the flame-shaped isovelocity map slope was observed. APV was calculated by dividing the distance between the beginning and end of the slope to the corresponding time (Fig. 1b).

Statistics

Statistical analysis was performed using IBM SPSS Statistics for Windows, version 23 (IBM Corp., Armonk, N.Y., USA). Descriptive statistics were presented as mean ± standard deviation for continuous variables and as percentage for categorical data.

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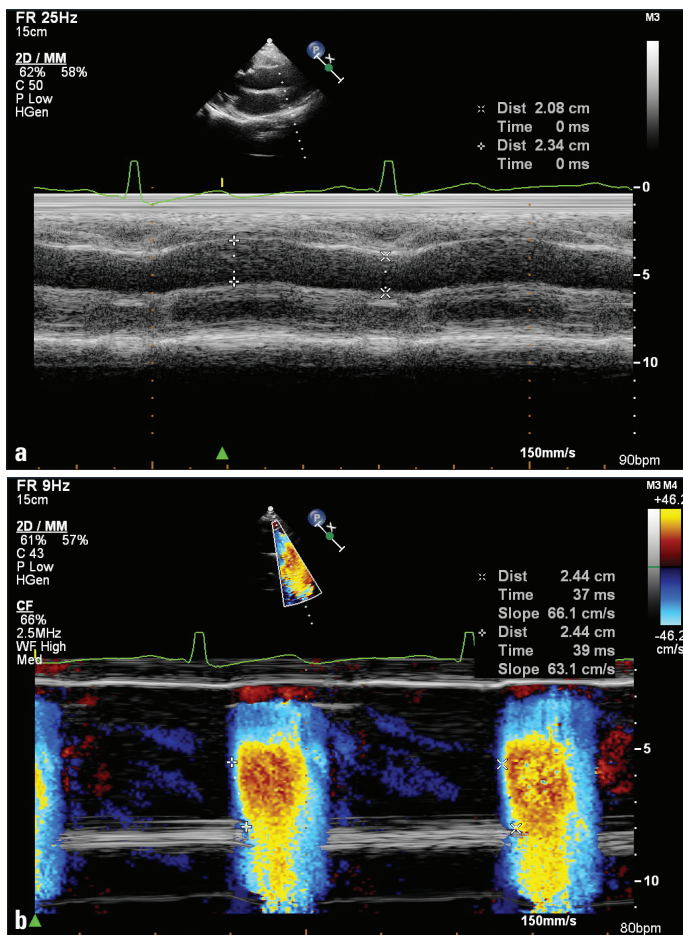Arı et al.
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Figure 1. (a) Systolic and diastolic diameter measurements of the ascending aorta using transthoracic M-mode echocardiography. (b) Aortic propagation velocity measurement of descending aorta with color M-mode echocardiography.

Spearman correlation analysis was performed to assess the correlations of stiffness parameters with the other study variables. A two-tailed p value of <0.05 was considered as statistically significant.

Results

The study population (51.5% male; mean age, 35.1±8.1 years) had a relatively low-cardiovascular risk profile with the total cholesterol level of 177±39.1 mg/dL, fasting glucose level of 97±14.6 mg/dL, and systolic blood pressure (SBP) and diastolic blood pressure (DBP) of 108.6±15.1 and 69.1±11.3 mm Hg, respectively. None of the participants were hypertensive, and four participants with newly diagnosed DM were excluded from the study. The patients had slightly high BMI (26.9±4.5 kg/m^2) and waist circumference of 90.6±12.6 cm, along with a high rate of smoking (34%). Demographic, clinical, laboratory, and echocardiographic findings of the study population are presented in Table 1. Age was negatively correlated with AS and AD, and the relationship was statistically significant (r=−0.31, p<0.01 for AS and r=−0.29, p=0.01 for AD). Significant correlations were also present between SBP and AD

Table 1. Demographic, clinical and echocardiographic findings of the study population (n=97)

	Mean±SD	Range	n (%)
Age, year	35.1±8.1	20-50	
Sex, female			474 (8.5%)
BMI, kg/m^2	26.9±4.5		
Waist circumference, cm	90.6±12.6		
SBP, mm Hg	108.6±15.1		
DBP, mm Hg	69.1±11.3		
Medical history at admission			
Hypertension			0 (0%)
Diabetes mellitus			0 (0%)
Hyper cholesterolemia			24 (24.7%)
Hyper triglyceridemia			13 (13.5%)
Smoking			33 (34%)
Positive family history for IHD			4 (4%)
Kreatinin, mg/dL	0.93±0.14		
Glukoz, mg/dL	97±14.6		
Total cholesterol, mg/dL	177±39.1		
LDL cholesterol, mg/dL	105.1±34.6		
HDL cholesterol, mg/dL	48.9±24		
Haemoglobin, gr/dL	14.4±1.6		
Triglicerid	136±90		
WBC, 10^3/mm^3	7.48±1.83		
Aortic strain, %	12.87±6.39*		
	11.11 (8.38–18.09)**		
AD, cm^2 dyn^{-1} 10^{-3}	0.68±0.39*		
	0.59 (0.39–0.87)**		
APV, cm/s	62.9±29.5*		
	57.3 (40.2–76)**		
LVEF, %	64±5.5		
LA, mm	32.1±4.3		
Mitral E/Em	5.6±1.75		
#Diastolic dysfunction, E/Em >8			10 (10%)

Data is presented as mean ± standard deviation* and median (interquartile range, 25th-75th)**. # Diastolic dysfunction was determined using the echocardiographic examination based on the tissue Doppler imaging method (E/Em > 8).

AD: aortic distensibility; APV: aortic propagation velocity; BMI: body mass index; DBP: diastolic blood pressure; Em: early diastolic mitral annular velocity; HDL: high-density lipoprotein cholesterol; LA: left atrium; LDL: low-density lipoprotein cholesterol; LVEF: left ventricular ejection fraction; Mitral E: early mitral inflow velocity; SBP: systolic blood pressure; WBC: white blood cell

(r=−0.36, p<0.01) and DBP and AS (p=−0.27, p<0.01). Waist circumference and BMI were negatively correlated with AS (r=−0.31, p<0.01 and r=−0.21, p=0.04, respectively) and with AD (r=−0.37, p<0.01 and r=−0.25–4, p=0.02, respectively); these relationships were also statistically significant. However, no significant correlation of APV with age, SBP, DBP, waist circumference, and BMI was identified (r=0.1, −0.04, −0.04, −0.08, 0.08, respectively, p>0.05 for all comparisons) (Table 2). No significant correlation of APV with AS and AD could be identified (r=−0.05, p=0.6 and r=−0.17, p=0.8, respectively).

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Table 2. The correlations of stiffness parameter with in themselves and with age, blood pressure, BMI, waist circumference

	Aortic strain		Aortic distensibility		APV	
APV	r=-0.05	p=0.6	r=-0.17	p=0.8		
Age	r=-0.31	p<0.01**	r=-0.29	p=0.01*	r=0.1	p=0.3
SBP	r=-0.14	p=0.2	r=-0.36	p<0.01**	r=-0.04	p=0.7
DBP	r=-0.27	p<0.01**	r=-0.16	p=0.1	r=-0.04	p=0.7
BMI	r=-0.21	p=0.04*	r=-0.24	p=0.02*	r=-0.08	p=0.4
WC	r=-0.31	p<0.01**	r=-0.37	p<0.01**	r=0.08	p=0.4

Spearman correlation analysis was used for statistical analyses.
*, Correlation is significant at the 0.05 level (2-tailed); **, Correlation is significant at the 0.01 level (2-tailed).
AVP-color M-Mode propagation velocity of descending aorta; BMI-body mass index, DBP- diastolic blood pressure, SBP- systolic blood pressure, WC- Waist circumference

The reproducibility of aortic diameter measurements during systole and diastole was higher than that of APV, as shown by the intra-class correlation coefficient test for intra-observer variability (intra-class correlation coefficient = 0.96, p<0.001; intra-class correlation coefficient =0.97, p<0.001; and intra-class correlation coefficient = 0.90, p=0.001, respectively). Additional tests in 15 study participants showed lower but acceptable inter-observer variability for APV (r=0.50, p=0.02).

Discussion

Although previous studies have clearly demonstrated a relationship of classic cardiovascular risk factors with AS and AD in high-risk populations, there is no substantial data on this association in low-risk group. The present observational study demonstrated an association of classical cardiovascular risk factors with AS and AD in a low-risk study group. However, an association of these risk factors with APV was not observed. In addition, no significant correlation of APV with AS or AD was detected in this low-cardiovascular risk population.

Aortic stiffness reflecting arterial stiffness describes the rigidity of the arteries and is mainly determined by the arterial wall components, including extracellular matrix, endothelial cells, smooth muscle cells, and other functional elements of the vessel wall (25). Pathophysiological alterations affecting these components in the context of cardiovascular risk factors induce structural alterations leading to increased arterial stiffness before the development of CVD (1-12). Therefore, the evaluation of aortic stiffness is a useful for detecting CVD earlier than the occurrence of clinically evident disease in risky groups as well as for predicting adverse clinical outcomes in patients with established disease (9, 10, 14, 26, 27). Measurement of aortic stiffness beyond established risk stratification strategies with classical risk factors, such as age, gender, hypertension, hyperlipidemia, smoking, and obesity, may lead to more appropriate allocation of patient to the correct risk group, such as the intermediate-to-high-risk group, instead of erroneous allocation to a low-to-intermediate-risk group (9, 13, 28). Therefore, the determination of aortic stiffness is of

greater importance in low- or intermediate-risk populations.

Although several ultrasonographic indices have been studied for quantifying aortic stiffness, carotid–femoral PWV is the most validated modality for the noninvasive evaluation of aortic stiff-
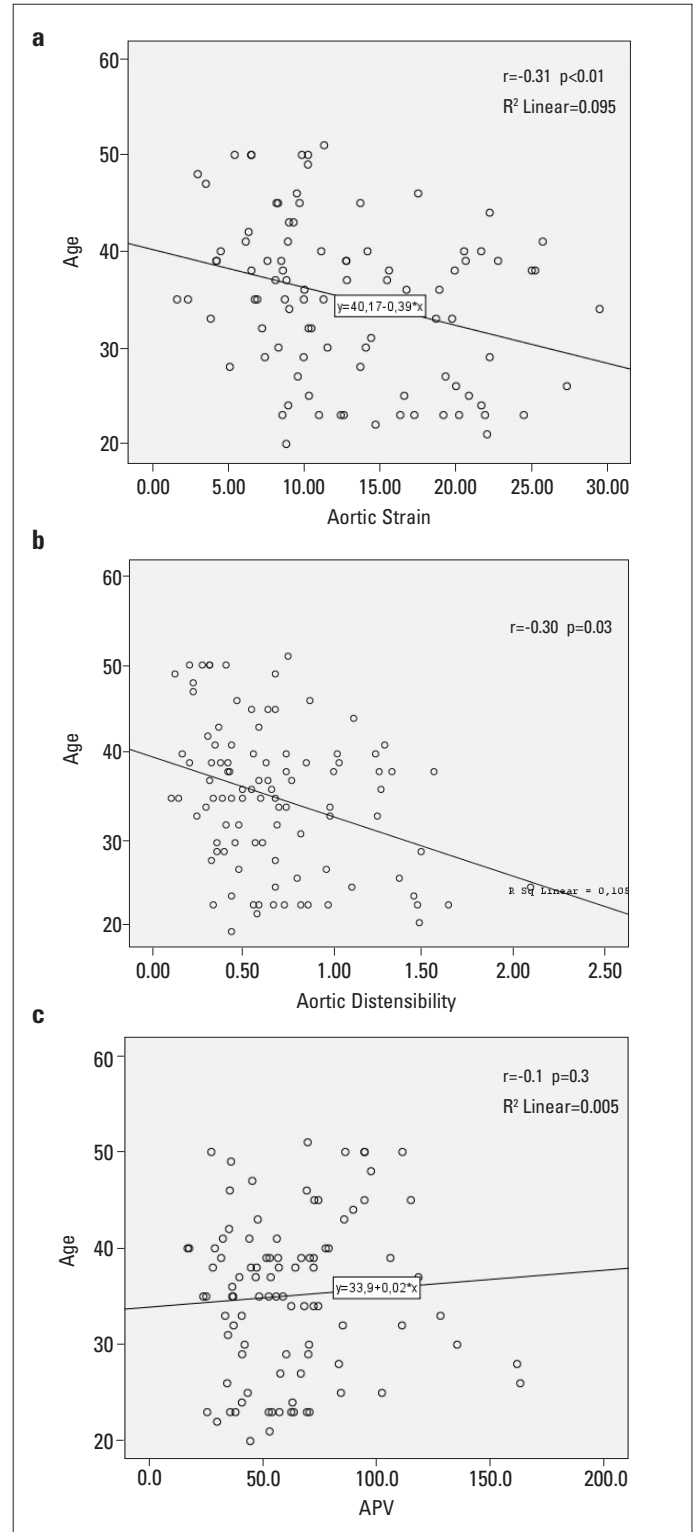

Figure 2. Correlation analysis showing the correlation of age with aortic strain (a) and aortic distensibility (b) and APV (c). APV- color M-mode propagation velocity of descending aorta

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ness and is highly recommended for clinical implementation (9, 13). However, the requirement of trained medical staff and special devices reduces its clinical applicability. For diagnostic methods to be widely applicable, they should have high predictive value, availability, reproducibility, and cost-effectiveness. Therefore, it is important to know the performance of other aortic stiffness indices in the preclinical patient population before the establishment of frank disease. AD and AS are echocardiography- and magnetic resonance imaging (MRI)-derived elasticity indices of the aorta and have been shown to be well correlated with PWV measurements (29-31).

An association between age and aortic stiffness is well documented (9, 11, 32). A reduction in aortic elasticity with increasing age has been reported even in the absence of overt cardiovascular disease (14, 29). In the present study, we demonstrated an inverse correlation between the aortic elasticity indices, AS and AD, and age in a low-cardiovascular risk population, which is consistent with the findings in previous reports. However, we did not detect a correlation between APV and age (Fig. 2). A previous study has suggested that individuals younger than 50 years with no overt cardiovascular disease have a more explicit association between MRI-derived AD and age than between PWV and age (29). The increases in MRI-derived cf-PWV and aortic arch PWV were more prominent in patients older than 50 years than in younger patients in the same study.

Obesity is another well-known risk factor for increased vascular stiffness and CVD (33). Increased arterial stiffness has been reported even in obese children (29, 34, 35), and weight loss has been shown to improve arterial compliance (33, 36). Our results are consistent with those in these previous reports with respect to indicating significant associations of AS and AD with BMI as well as of AS and AD with waist circumference reflecting visceral adiposity. However, no association of APV with BMI or waist circumference was observed.

There is a bidirectional association between aortic stiffness and HT (37). Accordingly, the relationship between aortic stiffness and HT can implicate the rational association between HT and the other cardiovascular risk factors, such as smoking, hypercholesterolemia, and DM. In the present study, inverse correlations were observed between AS and DBP as well as between AD and SBP even in individuals with normal blood pressure. However, no correlation was observed between APV and blood pressure. A significant correlation between blood pressure and APV has been reported in two studies on hypertensive patients (21, 22). In these studies, higher APV values were noted in the normotensive control group than in the hypertensive patient group. However, these studies did not report any correlation analysis between APV and blood pressure in the normotensive control group.

In a recent study, APVs were lower in patients with CAD than in those without CAD and were significantly correlated with the echocardiography-derived aortic stiffness parameters (r=0.556, p<0.001 for AS and r=0.483, p<0.001 for AD). However, this study did not report any similar analysis in the control group (18). In another study, APV was found to be inversely correlated with PWV (r=–0.580, p<0.001), and positively correlated with brachial artery flow-mediated dilatation (r=0.564, p<0.001) in patients with obstructive sleep apnea syndrome having a relatively higher cardiovascular risk profile (23). In our study, we observed no statistically significant correlation between APV and the conventional aortic stiffness parameters AS and AD (r=–0.05, p=0.6 and r=–0.17, p=0.8, respectively) in healthy individuals (Fig. 3). The lack of correlation between these parameters might be explained by different mechanisms of blood pressure propagation, which drive aortic elasticity indices and flow propagation. Pathologic conditions, such as CAD, may have a composite effect on these entities.
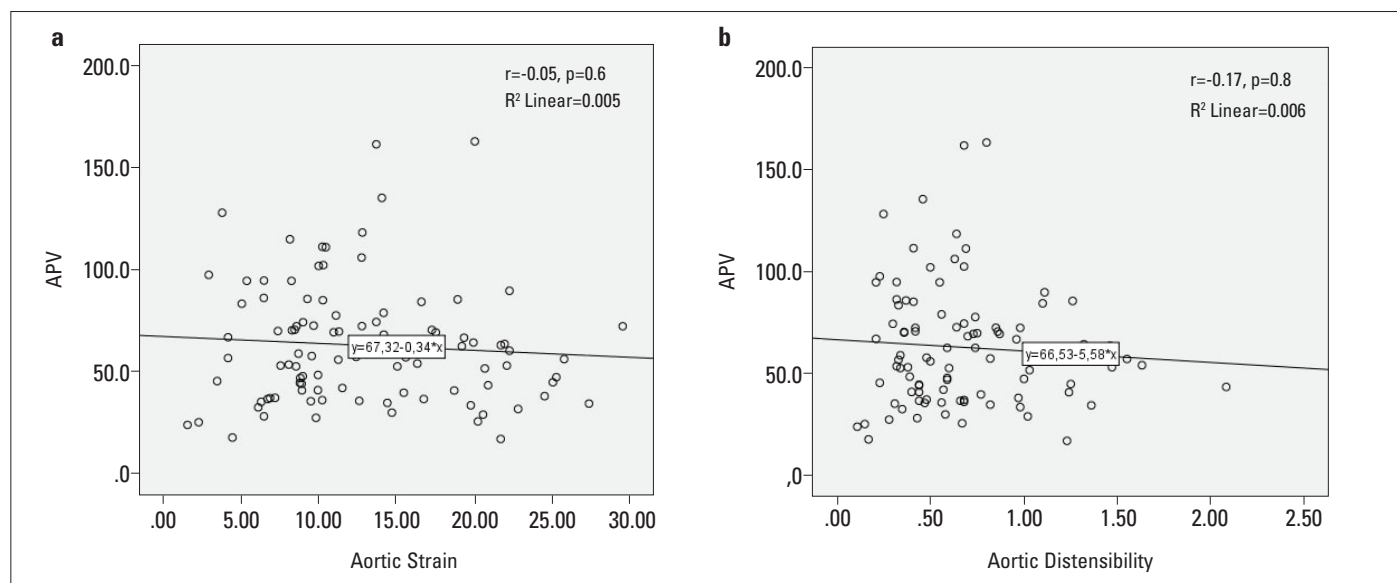

Figure 3. Correlation between APV and aortic strain (a), APV and aortic distensibility (b). APV-color M-mode propagation velocity of descending aorta

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Study limitations

The major limitation of the present study is that the association of APV was not evaluated using a more validated aortic stiffness evaluation method, such as PWV, instead of or in addition to the evaluation of AS and AD. However, the main aim of our study was to evaluate this association using more practical, easily applicable, and safe methods. On the other hand, APV is a newly established stiffness parameter that has been extensively studied in established CAD populations. Although the cohort in the present study had a low-risk profile for atherosclerosis, we did not investigate the presence of atherosclerosis using validated invasive angiographic or noninvasive imaging methods, such as carotid intima-media thickness measurement or multidetector row computed tomography. The small size and low-risk profile of the study population are other possible limitations that might also have affected the statistical analysis.

Conclusion

APV has been suggested as a novel and simple echocardiographic parameter of aortic stiffness in relatively high-risk and established CVD groups. The association of APV with other stiffness parameters in low-risk groups has not yet been evaluated. The present study showed lack of association between APV and AS, as well as between APV and AD. No association was observed between APV and known classical risk factors related to increased stiffness, such as age, BMI, waist circumference, and blood pressure, in the low-cardiovascular risk group. On the other hand, the associations of AS and AD with age, BMI, waist circumference, and blood pressure were statistically significant. The validity of APV should be further investigated considering its lower reproducibility compared with that of the classical stiffness parameters.

Conflict of interest: None declared.

Peer-review: Externally peer-reviewed.

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