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Aortic strain, distensibility and elastic modulus are associated with the presence and quantity of coronary calcium

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Abstract

Background: An association between aortic stiffness and atherosclerosis has been previously demonstrated by pulse wave velocity. Whether echocardiographically assessed aortic stiffness also correlates with the extent of atherosclerosis has not yet been established.

Aim: To evaluate the association between echocardiographically measured aortic stiffness and atherosclerosis.

Methods: A total of 162 patients (mean age 54 ± 9 years, age range 36–83 years, 102 male and 60 female) at high risk of atherosclerosis underwent transthoracic echocardiography and sphygmomanometer-based brachial blood pressure measurement for aortic stiffness parameters (strain, distensibility and elastic modulus), and multidetector computed tomography for the presence and quantity of coronary artery calcium (CAC).

Results: It was found that aortic strain and distensibility were significantly lower in patients with CAC than in patients without CAC (7.6 ± 2.7% *vs* 9.3 ± 3.4%, $p < 0.001$ and 3.0 ± 1.1 mm $Hg^{-1}.10^{-3}$ *vs* 3.9 ± 1.7 mm $Hg^{-1}.10^{-3}$, $p < 0.001$, respectively). However, elastic modulus E(p) was significantly higher in patients with CAC than in patients without CAC (7.9 ± 3.8 N/m^2 *vs* 6.0 ± 2.5 N/m^2, $p = 0.001$). In addition, aortic strain and distensibility, and elastic modulus, were found to be significant predictors of the presence and quantity of CAC in multivariate logistic and linear regression analyses (all $p < 0.05$).

Conclusions: Echocardiographically measured aortic stiffness is positively, significantly and independently associated with atherosclerosis.

Key words: calcium, echocardiography, stiffness

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INTRODUCTION

The normal human aorta is an elastic tube, although it loses this characteristic with ageing and other cardiovascular (CV) risk factors [1–3]. Physiologically, aortic stiffness is the elastic resistance against its distension [4] causing volume buffering known as the Windkessel function [5]. When this important function is further disrupted by fibrosis, smooth muscle necrosis, degradation of elastin fibres, calcifications, or migration of macromolecules into the arterial wall, systolic blood pressure (BP) increases, diastolic BP decreases, and pulse pressure widens [6]. Aortic stiffness can be evaluated invasively (as well as noninvasively) using echocardiography, BP measurement, and aortic pulse wave velocity by applanation tonometry.

The presence and quantity of coronary artery calcium (CAC) detected by computed tomography (CT) correlates with the overall magnitude of atherosclerotic plaque burden and with the development of subsequent adverse coronary events

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[7–11]. Several practice guidelines and other studies have proposed that increased levels measured as either percentiles (CAC > 75th percentile for age and sex matched) or absolute scores (i.e. > 100 or > 400) are associated with adverse coronary events, and higher levels can be used as markers to identify individuals requiring aggressive preventative treatment [12–14]. It has been also demonstrated that CAC improved risk prediction after taking into account the Framingham risk score in four racial or ethnic groups [15]. Population-based studies have demonstrated that aortic stiffness is strongly and independently associated with CAC and, thus, with atherosclerosis [16, 17].

The aim of the present study was to demonstrate this relationship noninvasively using echocardiography and BP measurement.

METHODS

Study population

All patients admitted to our department with suspected coronary artery disease (CAD) were possible candidates for inclusion in the study. Of them, those who were scheduled for multidetector CT to determine obstructive coronary atherosclerosis, were candidates for inclusion in the study. A total of 162 consecutive patients (mean age 54 ± 9 years, range 36–83 years, 102 males and 60 females) were recruited. All study participants underwent transthoracic echocardiographic examination to measure aortic systolic and diastolic diameters before the multidetector CT. In addition, sphygmomanometer-measured brachial BP values were obtained just after echocardiography. Exclusion criteria included: known CAD; left ventricular (LV) dysfunction (LV ejection fraction < 50%) and hypertrophy; unstable ischaemic conditions (unstable angina pectoris and myocardial infarction); rhythms other than sinus; significant valvular heart disease; and renal or hepatic dysfunction (creatinine > 2.5 mg/dL, AST and ALT > two times the upper limit of normal, respectively). Written consent was obtained from all patients and our local ethical committee approved the study.

Blood pressure measurement

After overnight fasting, BP levels were measured from the brachial artery at the level of the heart with a sphygmomanometer after resting for at least five minutes in the supine position. Three measurements, at least two minutes apart, were carried out, and the average of the closest two readings was recorded. A pressure drop rate of approximately 2 mm Hg/s was applied, and Korotkoff phases I and V were used for systolic and diastolic BP levels, respectively. All BP measurements were made by a cardiologist blinded to the study protocol. Obtained values were used to calculate pulse pressure (PP) = systolic BP – diastolic BP

Transthoracic echocardiography

Just before BP measurement in the left lateral decubitus position, echocardiographic examination (GE Medical Systems, Vivid 7 Dimension, Horten, Norway) was performed in the same position. During transthoracic echocardiography, the systolic and diastolic dimensions of the aorta were recorded in M-mode 3 cm above the aortic valve from a parasternal long-axis view, at the time of maximum aortic anterior motion, and at the peak of the QRS complex, respectively. Inner aortic diameters were measured with a caliper in systole and diastole as the distance between the trailing edge of the anterior aortic wall and the leading edge of the posterior aortic wall. Measurements were repeated at three cardiac cycles and the averaged value was used for analysis. All echocardiographic measurements were made by a cardiologist blinded to the study protocol. Obtained values were used to calculate aortic stiffness indices: aortic strain = systolic diameter (SD) – diastolic diameter (DD)/diastolic diameter (DD) × 100; aortic distensibility = (2 × aortic strain)/PP; and elastic modulus E(p) = PP/strain.

Multidetector computed tomography

Computed tomography images were obtained and the quantity of CAC was measured using the 64-slice technique (Toshiba Aquilion 64 scanner, Toshiba Medical, Tochigi, Japan). Slices 3 mm thick were acquired. The score according to the algorithm suggested by Agatston et al. [18] was used by a radiologist blinded to the study protocol. On the basis of the electrocardiography tracing, the software automatically selected a reduced set of diastolic images from each cardiac cycle. All pixels with density > 130 HU were automatically highlighted in colour on the images. The radiologist assigned one of four locations to each calcified plaque: left main, or left anterior descending, or circumflex, or right coronary artery. Agatston et al. [18] method determines the density of the highest density pixel in each plaque and applies a weighting factor to each plaque, depending on the peak density in the plaque: density (HU) of 130–199 = weight of 1; density of 200–299 = weight of 2; density of 300–399 = weight of 3; density of 400+ = weight of 4. The score for each plaque equals the plaque area multiplied by the weighting factor multiplied by the increment/slice width. The score for the entire specimen equals the sum of the scores for each plaque. The original Agatston et al. [18] method used a slice thickness of 3 mm.

Other laboratory data

Fasting peripheral venous blood samples were obtained to measure total cholesterol, LDL-cholesterol, HDL-cholesterol, triglyceride, glucose, and high-sensitive (hs) C-reactive protein (CRP) levels. Blood samples were centrifuged and plasma was obtained. Total cholesterol, HDL-cholesterol, triglyceride and glucose levels were measured enzymatically by the auto analyser. LDL cholesterol level was measured using Friedewald's formula.

Anthropometric measurement

The height and weight of each patient was measured and body mass index (BMI) was calculated by dividing weight in kilograms by height in metres squared (described as kg/m^2).

Statistical analysis

Data were analysed using SPSS software version 15.0 for Windows (SPSS Inc., Chicago, IL, USA). Continuous variables are presented as mean ± standard deviation and categorical variables as frequency and percentage. The Kolmogorov-Smirnov test was used to assess the distribution of continuous variables. Student's *t*-test was used to compare normally distributed continuous variables and the Mann-Whitney U test for variables without normal distribution. The χ^2 test was used to compare categorical variables. Any correlation between CAC score and aortic stiffness parameters was analysed with the Spearman analysis. A two-tailed p value of < 0.05 was considered statistically significant. A stepwise multivariate logistic regression analysis was used to evaluate the independent associates of the risk of CAC. Parameters with a p value of less than 0.1 in univariate analysis were included in the model. The odds ratios and 95% confidence intervals were calculated. Multivariate linear regression analysis was used to evaluate the independent associates of CAC quantity. Changes in aortic stiffness parameters according to CAC threshold were analysed using analysis of variance.

RESULTS

Baseline demographic, laboratory, and imaging characteristics of the study population are set out in Table 1. Middle-aged patients formed the vast majority of the population (88.3%), and approximately two thirds were male. In addition, approximately one third had major CV risk factors including hypertension, diabetes, and smoking. Almost 50% of patients had a calcium score ≤ 10. Only 14% of patients had a calcium score ≥ 401.

Aortic stiffness parameters, including aortic strain and distensibility, were significantly lower in patients with CAC than in patients without CAC (7.6 ± 2.7% *vs* 9.3 ± 3.4%, $p < 0.001$ and 3.0 ± 1.1 mm Hg^{-1}.10^{-3} *vs* 3.9 ± 1.7 mm Hg^{-1}.10^{-3}, $p < 0.001$, respectively). However, elastic modulus E(p) was significantly higher in patients with CAC than in patients without CAC (7.9 ± 3.8 N/m^2 *vs* 6.0 ± 2.5 N/m^2, $p = 0.001$; Fig. 1). There was no significant difference between males and females in terms of coronary calcium score (208 ± 350 *vs* 110 ± 331, $p = 0.081$). Neither the presence nor the quantity of CAC differed between smokers and non-smokers (both $p > 0.05$).

Coronary artery calcium score was significantly positively correlated with age, pulse pressure, and elastic modulus E(p), and negatively correlated with aortic strain and distensibility (Table 2).

Table 1. Demographic, laboratory and imaging characteristics of the study population

Age (age range) [years]	54 ± 9 (36–83)
Male sex	102 (63%)
Hypertension	55 (34%)
Diabetes	42 (26%)
Smoking	56 (35%)
Body mass index [kg/m^2]	26.5 ± 1.5
Systolic blood pressure [mm Hg]	125 ± 17
Diastolic blood pressure [mm Hg]	74 ± 11
Pulse pressure [mm Hg]	51 ± 10
Heart rate [bpm]	70 ± 8
Total cholesterol [mg/dL]	200 ± 44
LDL cholesterol [mg/dL]	120 ± 40
HDL cholesterol [mg/dL]	46 ± 12
Triglyceride [mg/dL]	167 ± 90
Fasting blood glucose [mg/dL]	115 ± 43
hsC-reactive protein [mg/L]	3.9 ± 3.1
Aortic systolic diameter [mm]	33.0 ± 4.0
Aortic diastolic diameter [mm]	30.5 ± 4.0
Aortic strain [%]	8.3 ± 3.1
Aortic distensibility [mmHg^{-1}.10^{-3}]	3.4 ± 1.5
Elastic modulus E(p) [N/m^2]	7.1 ± 3.4
Coronary artery calcium present	93 (57%)
Coronary artery calcium score:	172 ± 346
≤ 10	77 (48%)
11–100	32 (20%)
101–400	30 (18%)
401–1000	16 (10%)
> 1000	7 (4%)

In multivariate logistic regression analysis, after the addition of variables of age, male sex, BMI, CV risk factors including hypertension, diabetes, smoking, laboratory parameters including cholesterols and hsCRP, haemodynamic parameters including systolic and diastolic pressures, echocardiographic measurements including aortic systolic and diastolic diameters, and calculated aortic stiffness parameters of aortic strain, distensibility and elastic modulus, the significant predictors of the presence of CAC were: age, male sex, aortic systolic and diastolic diameters, aortic strain and distensibility, and elastic modulus (Table 3).

Age (1.1-fold), aortic SD (1.1-fold), aortic DD (1.2-fold), elastic modulus (1.2-fold) and male gender (2.5-fold) increased the risk of CAC, whereas aortic strain (0.8-fold) and distensibility (0.6-fold) decreased the risk of CAC.

In multivariate linear regression analysis, after the addition of variables of age, male sex, BMI, CV risk factors inclu-

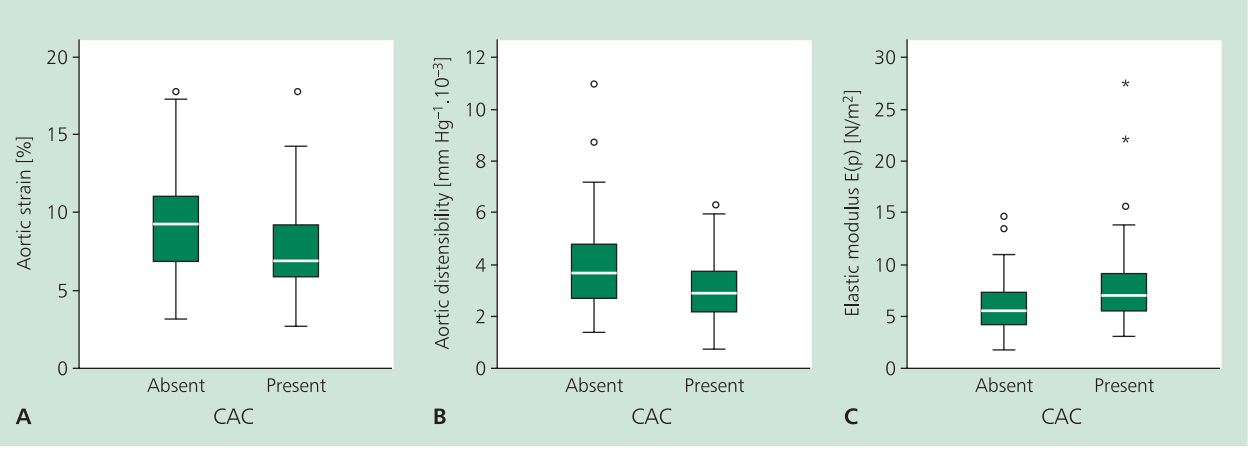

Figure 1. Comparison of mean values of aortic strain (**A**), distensibility (**B**), and elastic modulus (**C**) in participants with and without detectable coronary artery calcium (CAC)

Table 2. Significant correlation between analysed parameters and coronary artery calcium score

Variable	Correlation coefficient	P
Age	0.422	< 0.001
Pulse pressure	0.159	0.043
Elastic modulus E(p)	0.313	< 0.001
Aortic strain	–0.301	< 0.001
Aortic distensibility	–0.303	< 0.001

ding hypertension, diabetes, smoking, laboratory parameters including cholesterols and hsCRP, haemodynamic parameters including systolic and diastolic pressures, echocardiographic measurements including aortic systolic and diastolic diameters, and calculated aortic stiffness parameters of aortic strain, distensibility and elastic modulus, the significant predictors of CAC quantity were: age ($p < 0.001$), male gender ($p = 0.013$), aortic systolic ($p = 0.002$) and diastolic diameters ($p < 0.001$), aortic strain ($p = 0.007$) and distensibility ($p = 0.013$), and elastic modulus ($p < 0.001$).

Increased CAC thresholds were significantly related with decreased aortic strain ($p = 0.013$) and distensibility ($p = 0.005$), and with increased elastic modulus ($p = 0.002$) (Fig. 2).

DISCUSSION

Our study found for the first time that echocardiographically obtained aortic stiffness parameters are significantly and positively associated with the presence and quantity of coronary calcium.

Several other studies have investigated the association between arterial stiffness and coronary atherosclerosis in various subsets of patients. In a study by Nitta et al. [19] CAC was found to be associated with an increase in arterial stiffness, measured by arterial pulse wave velocity, and with the extent of aortic calcification in haemodialysis patients with aortic calcification, as in the study performed by Guerin et al. [20]. The percentage of patients newly diagnosed as having CAD has been found to be significantly higher in the highest quartile (> 120,000) of echocardiographically measured aortic stiffness E(p) [21]. In addition, in a large elderly population-based study, arterial stiffness parameters measured by pulse

Table 3. Multivariate regression analysis of the presence of coronary artery calcium

Variable	β	±SE	P	Odds ratio (95% CI)
Age	0.099	0.025	< 0.001	1.104 (1.052–1.159)
Male sex	0.649	0.209	0.002	2.503 (1.302–4.815)
Aortic systolic diameter	0.130	0.046	0.005	1.139 (1.040–1.247)
Aortic diastolic diameter	0.156	0.047	0.001	1.168 (1.065–1.282)
Aortic strain	–0.190	0.057	0.001	0.827 (0.740–0.925)
Aortic distensibility	–0.483	0.133	< 0.001	0.617 (0.476–0.800)
Elastic modulus E(p)	0.220	0.067	0.001	1.246 (1.092–1.421)

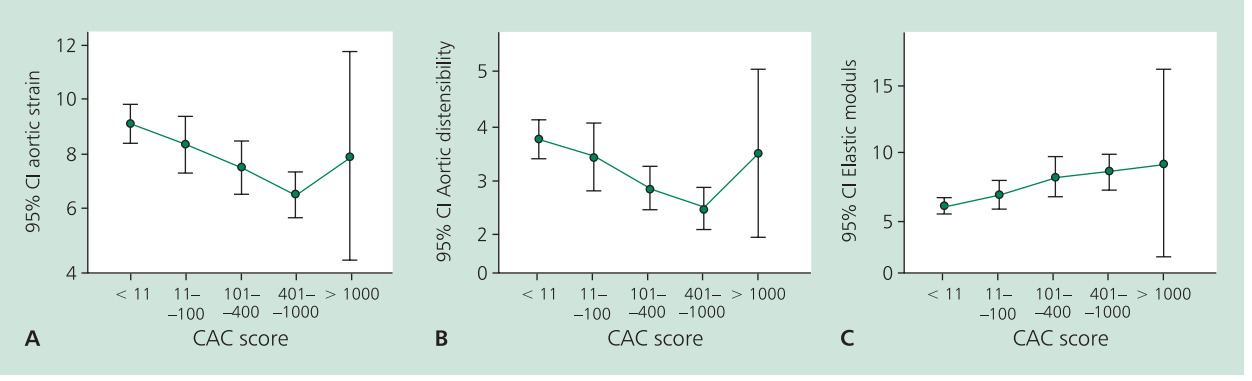

Figure 2. Progression of aortic strain (**A**), distensibility (**B**) and elastic modulus (**C**) according to coronary artery calcium (CAC) threshold

wave velocity in the aorta, and by distensibility coefficient in the carotid artery, have been found to be consistently increased with higher carotid intima-media thickness, and plaques in both carotid artery and aorta [16]. Contradicting these studies, in a relatively small study by Megnien et al. [22], the grade of coronary calcium measured with ultrafast CT was not associated with any aortic elastic parameters including pulse wave velocity, compliance, and intrinsic compliance, in approximately 200 asymptomatic high-risk patients.

We used echocardiography and BP, but not pulse wave velocity, as noninvasive methods of aortic stiffness in our study. Stefanadis et al. [23] demonstrated that the noninvasively evaluated aortic distensibility is comparable with invasive methods with a high degree of accuracy. Two-dimensional (2D) echocardiographic assessment of aortic distensibility is also able to detect with a high sensitivity changes in aortic mechanical properties [24].

We also used multidetector CT for the detection and quantity of CAC. In a study by Kopp et al. [25], multi-detector CT yielded better information for the detection and quantification of CAC than electron-beam CT, with low inter-observation variability. Volumetric method with isotropic interpolation has improved the reproducibility of CAC score. This has resulted in a significant improvement over the standard calcium scoring method [26]. An observational study in regard to mortality data of approximately 10,000 asymptomatic persons demonstrated that with increasing calcium scores, five-year survival from all-cause mortality worsens [27].

Similarly to previous studies, aortic strain and distensibility were significantly lower in patients with CAC than in patients without CAC, unlike elastic modulus E(p) that was significantly higher in patients with CAC than in patients without CAC. These findings suggest that increased CAC is associated with stiffer arteries. In addition, a significant positive correlation was found between elastic modulus E(p) and CAC score, unlike aortic strain and distensibility that were negatively correlated with CAC score. In addition, these three aortic stiffness parameters were significant predictors of the presence and quantity of CAC in our study. Because of the difficulty of interpreting results of regression models that use PP with systolic and diastolic pressures, we did include systolic and diastolic pressures rather than PP in the regression analysis. Therefore, we found that aortic stiffness parameters remained significant predictors of the presence and quantity of CAC, whereas systolic and diastolic pressures were not significant.

One possible explanation for an association between aortic stiffness and CAC may be the effects of atherosclerosis on arterial stiffness. Another possibility is that both coronary atherosclerosis and arterial stiffness occur concomitantly by means of vasoactive mediators from changes in the intima of coronary arteries and changes in the media of the aorta, respectively [28]. Vasa vasorum are most abundant in the ascending and arcus aorta. Impaired vasa vasorum flow of the ascending aorta due to CAD may play an important role in the disregulation of the elastic properties by means of increased collagen-to-elastin ratio, since the vasa vasorum of the ascending aorta originate from the coronary arteries [29].

Limitations of the study

The major limitation of this study is the limited sample size, which means that the results have to be confirmed by a larger study. In addition, this is a clinical study. At the molecular level, the exact mechanisms responsible for the increased stiffness and decreased elasticity might be evaluated and the results combined with clinical factors. Lastly, one disadvantage of 2D echocardiographic evaluation of aortic stiffness is that regionality is not taken into consideration. Therefore, real time 3D echocardiographic imaging for regional assessment of aortic stiffness can give more accurate measurement than changes in the aortic diameter due to the non-circular shape of the aorta.

CONCLUSIONS

Aortic stiffness parameters including strain, distensibility and elastic modulus are stronger predictors of the presence and extent of coronary atherosclerosis than BP parameters including PP. Therefore, noninvasively measured and calculated echocardiographic aortic stiffness parameters can reliably predict the presence and extent of coronary atherosclerosis.

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Odkształcenie aorty, jej rozszerzalność oraz moduł elastyczny wiążą się z obecnością i nasileniem zwapnień w tętnicach wieńcowych

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Streszczenie

Wstęp: Zależność między sztywnością aorty i miażdżycą udokumentowano za pomocą prędkości fali tętna. Dotychczas nie ustalono, czy sztywność aorty oceniana echokardiograficznie także koreluje z nasileniem miażdżycy.

Cel: Celem pracy była ocena związku między mierzoną echokardiograficznie sztywnością aorty i miażdżycą.

Metody: W grupie 162 pacjentów wysokiego ryzyka miażdżycy (w wieku średnio 54 ± 9 lat, zakres 36–83 lata, 102 mężczyzn i 60 kobiet) przeprowadzono przezklatkowe badanie echokardiograficzne, badanie ciśnienia tętniczego na tętnicy ramiennej z użyciem sfigmomanometru w celu oceny parametrów sztywności ścian aorty (odkształcenie, rozszerzalność i moduł elastyczny) oraz wielorzędową tomografię komputerową, aby ocenić obecność i liczbę zwapnień w tętnicach wieńcowych (CAC).

Wyniki: Wykazano, że wartości odkształcenia i rozszerzalności aorty były znacząco niższe u pacjentów z CAC w porównaniu z osobami bez CAC (odpowiednio 7,6 ± 2,7% *v.* 9,3 ± 3,4%, $p < 0,001$ oraz 3,0 ± 1,1 mm $Hg^{-1}.10^{-3}$ *v.* 3,9 ± 1,7 mm $Hg^{-1}.10^{-3}$, $p < 0,001$). Natomiast moduł elastyczny E(p) był istotnie wyższy u pacjentów z CAC niż u osób bez CAC (7,9 ± 3,8 N/m^2 *v.* 6,0 ± 2,5 N/m^2, $p = 0,001$). Ponadto odkształcenie i rozszerzalność aorty oraz moduł elastyczny stanowiły istotne czynniki predykcyjne obecności i liczby CAC w wieloczynnikowej analizie logistycznej, a także w analizie regresji liniowej (wszystkie $p < 0,05$).

Wnioski: Sztywność ściany aorty mierzona echokardiograficznie wykazuje niezależny, dodatni i statystycznie istotny związek z miażdżycą.

Słowa kluczowe: zwapnienia, echokardiografia, sztywność

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