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Thoracic Aortic Distensibility and Thoracic Aortic Calcium (From the Multi-ethnic Study of Atherosclerosis [MESA])

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Abstract

Decreased arterial distensibility is an early manifestation of adverse structural and functional changes within the vessel wall. Its correlation with thoracic aortic calcium (TAC), a marker of atherosclerosis, has not been well demonstrated. We tested the hypothesis that decreasing aortic compliance and increasing arterial stiffness is independently associated with increased TAC. We included 3,540 (61±10 years, 46% males) subjects from the Multi-ethnic Study of Atherosclerosis (MESA) study who underwent aortic distensibility (AD) assessment on MRI. TAC was calculated using modified Agatston algorithm on non-contrast cardiac CT. Multivariate regression models were calculated for the presence of TAC. Overall, 861 (24%) individuals had detectable TAC. A lower AD was observed among those with vs. without TAC (2.02±1.34 vs. 1.28±0.74, p<0.0001). The prevalence of TAC increased significantly across decreasing quartiles of AD (7%, 17%, 31%,

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Disclosures

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and 42%, $p<0.0001$). Using multivariate analysis, TAC was independently associated with AD after adjusting for age, gender, ethnicity and other covariates. In conclusion, our analysis demonstrates that increased arterial stiffness is associated with increased TAC independent of ethnicity and other atherosclerotic risk factors.

Background

There is paucity of data about the association with atherosclerosis in the aorta and vessel distensibility. Aortic distensibility (AD) is an accurate measure of arterial stiffness and can evaluate regional stiffness through the aorta¹. On the other hand, Thoracic aortic calcium (TAC) has been shown to correlate with coronary artery disease.²⁻⁵ However, the association between TAC and AD and the impact of age, gender, and ethnicity on this association is not clear. Thus, the aim of this analysis is to test the hypothesis that decreasing aortic distensibility measured by MRI is independently associated with increased thoracic aorta calcium on non-contrast computed tomography.

Methods

The MESA study investigated the prevalence, correlates, and progression of sub-clinical cardiovascular disease (CVD) in a population-based sample of 6,814 men and women aged 45–84 years free of known cardiovascular disease at baseline. The study objectives and design have been published before⁶. In brief, this prospective cohort study includes recruited subjects from six U.S. communities (Baltimore, MD; Chicago, Ill; Forsyth County, North Carolina; Los Angeles County, California; northern Manhattan, New York; and St. Paul, MN). In this analysis, we included 3,540 subjects (61 ± 10 years, 46% males) who had baseline AD assessment on MRI as well as the thoracic aortic calcium measurement. We used baseline data from the Multi-Ethnic Study of Atherosclerosis (2000–2002).

Medical history, anthropometric measurements, and laboratory data for the present study were taken from the first examination of the MESA cohort (July 2000 to August 2002). Information about age, gender, ethnicity, and medical history were obtained by questionnaires. Use of antihypertensive and other medications was based on clinical staff entry of prescribed medications verified by the staff. Hypertension was defined as a systolic blood pressure ≥ 140 mm Hg, diastolic blood pressure ≥ 90 mm Hg, or use of medication prescribed for hypertension. Body mass index was calculated from the equation weight (kg)/height (m^2). Total and high-density lipoprotein (HDL) cholesterol were measured from blood samples obtained after a 12-hour fast. Low-density lipoprotein cholesterol (LDL) was estimated by the Friedewald equation⁷. In examination 1 of MESA, participants reported the presence and absence of a family history of coronary heart disease.

All participants underwent CT scans at the same time for evaluation of coronary calcium, after signing informed consent. The image acquisition protocol has been described before⁸⁻¹¹. In summary, computed tomography scanning of the chest was performed either with an ECG-triggered (at 80% of the RR interval) electron-beam computed tomography scanner (Chicago, Los Angeles, and New York field centers; Imatron C-150, Imatron, San Francisco, Calif) or with prospectively ECG-triggered scan acquisition at 50% of the RR

interval with a multidetector computed tomography system that acquired 4 simultaneous 2.5-mm slices for each cardiac cycle in a sequential or axial scan mode (Baltimore, Forsyth County, and St. Paul field centers; LightSpeed, General Electric, Piscataway, NJ, or Volume Zoom, Siemens, New York, NY). Image slices were obtained with the participant supine, with no couch angulation.

A minimum of 35 contiguous images with a 2.5– or 3-mm slice thickness was obtained, starting above the left main coronary artery to the bottom of both ventricles. Each scan was obtained in a single breath hold. Section thickness of 3 mm, field of view of 35 cm, and matrix of 512×512 were used to reconstruct raw image data. The nominal section thickness was 3.0 mm for electron beam CT and 2.5 mm for 4-detector row CT. Spatial resolution is described by the smallest volume element, or voxel, for the protocol for each system: 1.15 mm^3 for 4-detector row CT ($0.68 \times 0.68 \times 2.50 \text{ mm}$) and 1.38 mm^3 for electron beam CT ($0.68 \times 0.68 \times 3.00 \text{ mm}$).

Ascending and descending TAC ranged from the lower edge of the pulmonary artery bifurcation to the cardiac apex (imaged on every study of coronary calcium) were quantified by using the same lesion definition for coronary calcium. TAC included both ascending TAC and descending TAC on the portion of the aorta imaged by cardiac CT. Any calcified focus seen extending into the aortic root wall was excluded from the aortic wall calcium. The absence of ascending and descending TAC was assigned a score of zero ¹².

The coronary calcium score of each lesion was calculated by multiplying lesion area by a density factor derived from maximal Hounsfield units within this area, as described by Agatston et al ¹³. The density factor was assigned in the following manner: 1 for lesions for which the maximal density was 130 to 199 HU, 2 for lesions 200 to 299 HU, 3 for lesions 300 to 399 HU, and 4 for lesions 400 HU. A total calcium score (for both Agatston and volume) was determined by summing individual lesion scores at each anatomic site. Volume of calcium was also measured (in mm^3) as the volumetric score ¹⁴.

Gradient-echo phase-contrast cine MRI with electrocardiographic gating was performed to evaluate the distensibility of the aorta. Images of the ascending and descending aorta were obtained in the transverse plane at the level of the right pulmonary artery perpendicular to the vessel lumen. Imaging parameters were as follows: repetition time = 10 msec; echo time = 1.9 msec; field of view = 34 cm; slice thickness = 8 mm; matrix size = 256×224 ; 2 signal averages; temporal resolution = 20 ms; velocity encoding gradient = 150 cm/s; and receiver bandwidth $\pm 32 \text{ kHz}$.

To determine AD, the minimum and maximum cross-sectional areas of the ascending aorta vessel were determined using an automated contour routine using the software FLOW (MEDIS Medical Imaging Systems). AD was calculated using this described by Gamble ¹⁵ where ΔD is the change in systolic/diastolic diameter, P is the pulse pressure, D_s is the

systolic diameter, D is the average aortic diameter. Distensibility coefficient: $\frac{\Delta D}{\Delta P D_s} \times 1000$

Blood pressure was measured immediately before and after the MRI aortic measurements, while the patient was in the supine position on the MRI scanner gantry; the average systolic

and diastolic values were then used to calculate pulse pressure. The MRI reader was blinded to all variables of the study subjects except their identification numbers.

Statistical analyses were performed using SPSS (version 17.0; SPSS Inc, Chicago, IL) and SAS (version 9.1 Cary, North Carolina). Categorical data are presented as percent frequencies and compared between groups by chi-square or Fisher's exact test. Continuous variables are presented as mean $\pm$ 1 standard deviation and compared using Student's t-test. Non-normally distributed variables are presented as median and 25th – 75th Interquartile range and were compared using non-parametric testing. Multivariable modeling analysis was performed using linear and logistic regression. The primary analysis was to assess the association between TAC and AD (as continuous variable as well as categorical variable) after adjusting for baseline clinical, historical and imaging covariates. The 4th quartile (most distensible) is used as the reference group for subsequent analysis. For all multivariate modeling, the threshold for variable entry into models was $p < 0.05$ and the threshold for variable removal was $p > 0.10$. Selection of variables for entry consideration was based on univariate analysis. We also adjusted for known coronary artery disease risk factors. Model 1 included age, gender and ethnicity. Model 2 was adjusted for age, gender, ethnicity, body mass index, hypertension, diabetes mellitus, cigarette smoking, family history of heart attack, low density lipoprotein cholesterol levels, cholesterol lowering medications Model 3 includes all the variables in model 2 plus the Log transformed CAC+1 and Log transformed C-reactive protein (CRP). Two-sided p value less than 0.05 was considered significant.

Results

A total of 3,540 subjects are included in this analysis. The mean difference between the CT and MRI were 26 days. TAC was detected in 861 (24%) participants. Table 1 shows the baseline characteristics of the study cohort according to the presence of TAC. Participants with TAC were older with higher prevalence of hypertension, diabetes and hypercholesterolemia ($p < 0.05$ for all comparisons). In addition, participants with TAC had lower glomerular filtration rate and higher high sensitivity C - reactive protein (Table 1). As expected, participants with TAC had higher mean coronary calcium (61 ± 226 vs. 305 ± 565 , $p < 0.001$). The mean AD was $1.84 \pm 1.26 \text{ mmHG}^{-1} \times 10^{-3}$ and a lower AD was seen among those with vs. without TAC (Figure 1).

Participants were divided into 4 AD quartiles (Table 2). The prevalence of TAC increased significantly across decreasing quartiles of AD (increasing aortic stiffness), (4th quartile: 7%, 3rd quartile: 17%, 2nd quartile: 31%, 1st quartile: 42%, $p < 0.0001$, figure 2). With the decrease in AD quartile (increasing aortic stiffness), participants were older, more likely to be male, and enriched in the African-American and Hispanic ethnicities. The decrease in aortic distensibility was associated with an increase in the prevalence of traditional atherosclerotic risk factors including hypertension, diabetes, hypercholesterolemia, decreasing renal function and 10 year Framingham risk score ($p < 0.0001$, Table 2).

In participants with prevalent TAC, there appear to be a linear relation between the AD quartiles and TAC score (Figures 3 and 4). With the decrease in AD quartile (increasing

aortic stiffness), TAC increased ($p<0.0001$). The median TAC across the four quartiles was 334, 168, 159, and 93 respectively.

A total of 1,632 subjects had detectable coronary artery calcium. Similar to TAC, CAC score increased as AD decreased ($p<0.0001$). However, the Spearman correlation between AD and TAC was stronger than the association of AD and CAC for the entire cohort ($r=-0.328$ vs. $r=-0.230$).

To determine whether AD is independently associated with the presence of TAC, we constructed several linear and logistic regression models adjusting for multiple baseline characteristics. After adjusting for age, gender and ethnicity, decreasing AD was independently associated with TAC ($p<0.0001$). This relation persisted after adjusting for body mass index, hypertension, diabetes mellitus, cigarette smoking, family history of heart attack, LDL cholesterol levels, cholesterol lowering medications ($p<0.0001$) as well as adjusting for coronary artery calcium and high sensitivity C-reactive protein ($p<0.0001$). We also fitted different models for ascending and descending thoracic aortic calcium separately (Table 3). Both models showed a trend towards association between TAC and AD. When fitting the logistic model as a trend for AD quartiles, this relation persisted ($p<0.0001$ for AD as a trend, model c-index 0.868). However, using linear regression, the adjusted beta coefficient for the association between AD and TAC was -10.4, $p=0.0545$.

Discussion

Our analysis demonstrates that the presence of TAC is independently associated with decreased aortic distensibility after adjusting for age, ethnicity, gender, atherosclerotic risk factors and coronary artery calcium. Participants in the lowest quartile of AD (least compliant aorta) have a 2-fold increase in the prevalence of aortic calcium.

Previous analysis from the MESA study has shown that decreased AD (a stiffer aorta) is associated with older age, hypertension, and African American ethnicity. Additionally, decreased AD was present in current smokers and in subjects with higher HDL cholesterol levels¹⁶. In this analysis, we extended this observation and showed that decreased AD is associated with atherosclerosis represented by the presence of TAC even after adjusting for atherosclerotic risk factors and coronary artery calcium. It is known that the aorta stiffens with aging, and the age-related structural and functional degenerative changes are accelerated by arterial hypertension. In addition, TAC, a marker of atherosclerosis increases with aging. Our analysis suggests that even after adjusting for atherosclerotic risk factors, there continue to be a strong relation between AD and TAC.

MRI evaluation of different regions of arterial stiffness provides additional information about vascular changes. However, the use of MRI derived AD has been limited by the limited availability and cost. Previous work has demonstrated that AD is altered by smoking, exercise, valvular disease as well as medical therapy¹⁷⁻¹⁹. Decreased aortic stiffness has been previously associated with a significant morbidity and increased all-cause and cardiovascular mortality in hypertensive participants^{20,21}. In 2006, the Rotterdam study showed that decreased distensibility seems to be an independent predictor of coronary heart

disease and stroke in healthy subjects and an independent predictor of mortality in the general population ²².

Prior studies have associated AD with multiple other atherosclerotic risk factors and associates of coronary artery disease including retinal artery narrowing ²³ and worsening renal function. In this analysis, we also noted a statistically significant correlation between AD and CAC. This is due to the fact that atherosclerosis is a systemic disease that affects all vascular beds. However, it appears that atherosclerosis correlates better with the calcium in the same vascular territory. This is not surprising since AD and TAC are measures of physiology and anatomy, respectively.

Our analysis has several strengths. This is the first multi-ethnic study that evaluated the relation between AD and TAC. However, this analysis should be interpreted in the context of its limitations. The cross-sectional design of our analysis would not allow us to address whether decreased AD precedes the development of TAC or is caused by TAC. We did not have any outcomes data and thus, it is unclear whether AD adds incremental prognostic value over TAC in predicting hard endpoints.

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Figure 1.
Box Plot of the aortic distensibility in relation to the presence of thoracic aortic calcium (TAC). Participants with TAC had decreased aortic distensibility.

Figure 2.

Prevalence of TAC in relation to Aortic distensibility quartiles. As Aortic distensibility decreases, the prevalence of TAC increases ($p < 0.0001$)

Figure 3.

Box Plot of thoracic aortic calcium (TAC) in relation to Aortic distensibility quartiles. As the Aorta became less distensible, the prevalence of TAC increased.

Figure 4.

Scatter Plot of thoracic aortic calcium (TAC) in relation to aortic distensibility quartiles. As the Aorta became less distensible, the TAC increased. There is a weak, but statistically significant relation between TAC and aortic distensibility.

Table 1

Baseline Characteristics of the study cohort according to the presence or absence of Thoracic Aortic Calcium

Variables	Thoracic Aortic Calcium		P Value
	Absent N=2679	Present N=861	
Age (years)	56 (51, 64)	70 (65, 76)	< 0.0001
Women	1243 (46 %)	374 (43%)	0.13
White	1040 (39%)	444 (52%)	
Chinese	303 (11%)	95 (11%)	
Black	838 (31%)	217 (25%)	<0.0001
Hispanic	498 (19%)	105 (12%)	
Body Mass Index (kg/m ²)	27 (24, 30)	27 (24, 30)	0.33
Hypertension	928 (35%)	541 (63%)	<.0001
Hypertension medications	789 (30%)	455 (53%)	<.0001
Diabetes mellitus	180 (7%)	102 (12%)	<0.0001
Smoker	355 (13%)	106 (12%)	0.06
Hypercholesterolemia	325 (12%)	213 (25%)	<.0001
Cholesterol lowering medication	325 (12%)	213 (25%)	<0.0001
Coronary calcium score	0 (0 19.5)	65 (2, 332)	<0.0001
High Sensitivity C-reactive protein	3.51 + 5.32	2.0 (0.9 – 4.4)	0.0017
Aortic distensibility	2 ± 1.3	1.3± 0.7	<0.0001

Table 2
Baseline Characteristics of the study cohort according to the Four Quartiles of Aortic Distensibility

Variables	Quartiles				P Value
	4 (2.33 mmHG ⁻¹ ×10 ³)	3 (1.57-2.32 mmHG ⁻¹ ×10 ³)	2 (1.05-1.56 mmHG ⁻¹ ×10 ³)	1 (1.04 mmHG ⁻¹ ×10 ³)	
Age	66.8 ± 8.8	63.3 ± 9.3	58.4 ± 8.8	53.4 ± 7.6	<0.0001
Female Gender	373 (42.2%)	387 (43.7%)	445 (50.3%)	412 (46.6%)	0.00034
White	364 (24.5%)	78 (19.6%)	313 (29.7%)	130 (21.6%)	<0.0001
Chinese	370 (24.9%)	93 (23.4%)	295(28.0%)	127 (21.1%)	
Black	386 (26.1%)	98 (24.6%)	244(28.0%)	157(26.0%)	
Hispanic	364(24.5%)	129 (32.4%)	203 (19.2%)	189(31.3%)	
Hypertension	553 (62.5%)	424 (48.9%)	309 (34.9%)	183 (20.7%)	<0.001
Hypertension medications	444 (50.2%)	356 (40.3%)	263 (29.8%)	181 (20.5%)	<0.0001
Diabetes mellitus	107 (12.1%)	81 (9.2%)	48 (5.4%)	46 (5.2%)	<0.0001
Smoker, Former	337 (38.3%)	330 (37.5%)	295 (33.5%)	288 (32.5%)	0.060
Smoker, Current	106 (12%)	114 (12.9%)	130 (14.8%)	111 (12.5%)	0.060
Cholesterol lowering medication	174 (19.7%)	147 (16.6%)	108 (12.2%)	109 (12.3%)	<0.0001
Log High Sensitivity C-reactive protein (mg/l)	0.71 ± 1.17	0.62 ± 1.1	0.57 ± 1.2	0.51±1.2	0.004
Presence of TAC	376 (42.5%)	273 (30.9%)	148 (16.8%)	64 (7.2%)	<0.0001
TAC	372.9±1177	235.5 ±1061	78.9 ±498	25.5 ± 220	<0.0001
Log (CAC+1)	2.6 ± 2.6	2.3 ±2.5	1.8 ± 2.4	1.15 ± 2.0	<0.0001
Body Mass Index(kg/m ²)	27.7 ± 14.8	27.6 ± 5.0	28.1 ± 5.2	27.8± 4.9	0.110
Systolic blood pressure (mmHg)	135.9 ±22.2	128.1 ± 20.2	121.7 ±18.9	113.3 ±15.1	<0.0001
Diastolic blood pressure (mmHg)	73.8 ± 10.9	75.0 ± 20	72.1 ± 10.3	69.9± 9.5	<0.0001
Low Density Lipoprotein (mg/dl)	115.9 ±30.9	117.1 ±30.3	117.3 ±30.3	118.4 ±31.2	0.389
High Density Lipoprotein (mg/dl)	52.5 ±14.5	52.7 ±15.9	51.4 ±15.5	50.8 ±14.7	0.024
Triglycerides (mg/dl)	129.6 ± 88	122.7 ± 75	126.7 ± 74	129.0 ± 94	
Glomerular Filtration Rate (ml/min/1.73m ²)	78.8 ±18.4	81.1 ±17.1	83.2 16.6	84.2 ±15.9	<0.0001
Framingham Risk Score (10 years)	15.0 ± 3.4	13.7±3.5	12.2 ±3.4	12.8 ±3.8	<0.0001

Abbreviations:mg: milligram; l: liters; m: meter; dl: deciliter; ml: milliter; min:minute

Table 3

Adjusted odds ratios (95% Confidence Interval) For Presence of Thoracic Aortic Calcium with Reduced Aortic Distensibility

Model		Aortic Distensibility Quartile			
		4 (2.33 mmHG ⁻¹ ×10 ³)	3 (1.57-2.32 mmHG ⁻¹ ×10 ³)	2 (1.05-1.56 mmHG ⁻¹ ×10 ³)	1 (1.04 mmHG ⁻¹ ×10 ³)
<i><u>Thoracic Aortic calcification</u></i>					
1	1 (ref group)		1.32 (0.91-1.92)	1.83 (1.27-2.63)	2.30 (1.62-3.27)
2	1 (ref group)		1.33 (0.88-2.01)	1.70 (1.14-2.52)	1.91 (1.29-2.82)
3	1 (ref group)		1.35 (0.88-2.07)	1.74 (1.16-2.62)	2.03 (1.37-3.02)
<i><u>Ascending Thoracic Aortic Calcification</u></i>					
3	1 (ref group)		1.30 (0.50 – 3.3)	1.54 (0.61 – 3.88)	2.22 (0.88 – 5.61)
<i><u>Descending Thoracic Aortic calcification</u></i>					
3	1 (ref group)		1.33 (0.92 – 1.92)	1.79 (1.26 – 2.55)	2.02 (1.41 – 2.89)

Model 1: adjusted for age, gender, ethnicity

Model 2: adjusted for age, gender, ethnicity, body mass index, hypertension, diabetes mellitus, cigarette smoking, family history of heart attack, LDL cholesterol levels, cholesterol lowering medications

Model 3: Model 2+ Log transformed CAC+1, Log transformed C-reactive protein