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Prevalence and Distribution of Abdominal Aortic Calcium by Sex and Age-Group in a Community-based Cohort (From The Framingham Heart Study)

Michael L. Chuang, MD[a], **Joseph M. Massaro, PhD**[a,b], **Yamini S. Levitzky, MD**[a], **Caroline S. Fox, MD, MPH**[a,c,f], **Emily S. Manders, BS**[a], **Udo Hoffmann, MD, MPH**[d,f], and **Christopher J. O'Donnell, MD, MPH**[a,e,f]

[a]The National Heart, Lung and Blood Institute's Framingham Heart Study, Boston MA

[b]Boston University School of Public Health Department of Biostatistics, Boston MA

[c]Division of Endocrinology, Metabolism, and Diabetes, Brigham and Women's Hospital, Boston MA

[d]The Cardiac MR PET CT Program and Department of Radiology, Boston, MA

[e]the Division of Cardiology, Massachusetts General Hospital, Boston, MA

[f]Harvard Medical School, Boston, MA, USA.

Abstract

Abdominal aortic calcium (AAC) is associated with incident cardiovascular disease but the age and sex-related distribution of AAC in a community-dwelling population free of standard cardiovascular disease risk factors has not been described. A total of 3285 participants (aged 50.2±9.9 years) in the Framingham Heart Study Offspring and Third Generation cohorts underwent abdominal multidetector computed tomography (MDCT) scanning during 1998-2005. The presence and amount of AAC was quantified (Agatston score) by an experienced reader using standardized criteria. A healthy referent subsample (N=1656, 803 men) free of hypertension, hyperlipidemia, diabetes, obesity and smoking was identified, and participants were stratified by sex and age group (<45, 45-54, 55-64, 65-74, ≥75 years). The prevalence and burden of AAC increased monotonically and supralinearly with age in both sexes but was greater in men than women in each age group. Below age 45 <16% of referent-subsample participants had any quantifiable AAC, while above age 65 nearly 90% of referent participants had >0 AAC. Across the entire study sample, AAC prevalence and burden similarly increased with greater age. Defining the 90th percentile of referent group AAC as “high,” the prevalence of high AAC was 19% for each sex in the overall study sample. AAC also increased across categories of 10-year coronary heart disease risk, as calculated using the Framingham Risk Score, in the entire study sample. We found AAC to be widely prevalent, with the burden of AAC associated with 10-year coronary risk, in a white, free-living adult cohort.

Address for Correspondence: Christopher J. O'Donnell, MD, MPH Framingham Heart Study 73 Mt. Wayte Avenue, Suite No. 2 Framingham, MA 01702-5827 Telephone: 508 935 3425 Fax: 508 626 1262 odonnellc@nhlbi.nih.gov.

Keywords

atherosclerosis; aorta; calcification; computed tomography; epidemiology

Introduction

Necropsy studies have demonstrated that vascular calcifications are an early and significant component of many atherosclerotic plaques.[1] Coronary artery calcium (CAC) has been studied extensively as a surrogate for atherosclerotic burden and as a predictor of future coronary heart disease (CHD).[2] However, atherosclerosis begins to develop in the aorta before it appears in other vascular beds.[3] Therefore, quantifying aortic calcium using widely-available non-invasive imaging methods may be useful for identifying individuals at increased risk for developing occlusive vascular disease. In prospective epidemiological studies, plain radiographic evidence of aortic calcific deposits in the aortic arch[4,5] and the abdominal aorta, [6,7] as well as valve calcification detected by echocardiography,[8] have been associated with increased cardiovascular morbidity and mortality. We sought to describe the distribution of calcific deposits in the abdominal aorta detected by multidetector computed tomography (MDCT) in a community-based cohort of adults free of clinically apparent cardiovascular disease (CVD), to evaluate the association of abdominal aortic calcium (AAC) seen on MDCT with 10-year CHD risk defined by the Framingham Risk Score,[9] and to determine the relationship between CAC and AAC.

Methods

The study sample was comprised of participants enrolled in the Framingham Offspring cohort[10] and the Third Generation cohort.[11] Offspring comprise the children, and their spouses, enrolled in the original Framingham Heart Study cohort, while the Third Generation cohort are the grandchildren of the original cohort. To be included in this study, participants were required to have attended either the Offspring seventh examination cycle (1998-2001) or the Third Generation first examination cycle (2002 – 2005) and have a complete risk factor profile (including hypertension, lipids, smoking status, body mass index, and diabetes status). Men were required to be ≥35 years of age. Women were required to be ≥ 40 years of age and non-pregnant. Due to technical factors associated with the MDCT hardware, participants could be included only if they weighed < 160 kg. Participants with clinically apparent CVD, defined by prevalent CVD, prior coronary artery bypass graft, percutaneous stent, or pacemaker/ICD placement, or valve replacement, were prospectively excluded from analysis. The institutional review boards of the Boston University Medical Center and Massachusetts General Hospital approved the study. All participants provided written informed consent.

The standard Framingham clinic examination included a physician-performed interview and physical examination, and blood samples obtained in the morning after a 12-hour fast. Body mass index was determined as weight (kg) divided by the square of height (m); obesity was defined as BMI ≥ 30 kg/m^2. Diabetes mellitus was defined as a fasting plasma glucose ≥ 126 mg/dL or treatment with insulin or a hypoglycemic agent. Participants were considered to be current smokers if they smoked at least one cigarette per day for the last year. Hypertension was defined as systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mm Hg, on the average of 2 physician-performed measurements, or by use of antihypertensive medication. Hyperlipidemia was defined as serum total cholesterol ≥ 240 mg/dL or by use of pharmacologic treatment. CVD events were adjudicated by a panel of three physicians, blinded to MDCT data, using standardized criteria previously described.[12] Based on these

data we identified a healthy non-smoking, non-obese referent subgroup free of hypertension, hyperlipidemia, diabetes and clinically apparent CVD.

Participants were imaged on an eight-slice MDCT scanner (LightSpeed Ultra, General Electric, Milwaukee, WI) with prospective ECG triggering during a single breath hold in mid-inspiration using sequential data acquisition as previously described.[13] A test breath hold was performed to ensure compliance before the scan. Scans were prospectively initiated at 50% of the RR interval, as used previously for MDCT-based measurements of CAC.[14] The top of the S1 vertebral body was prospectively selected as the most caudal extent of the abdominal volume to be imaged. Thirty contiguous 5-mm thick slices were obtained cranial to S1 for a total coverage of 15 cm in the Z-direction. Abdominal imaging parameters included:120 kVp, 400 mA, gantry rotation time 500 ms, table feed 3:1. The effective radiation exposure was 2.7 mSv. Coronary imaging parameters included: 120 kVp, 320 or 400 mA, for body weight < or ≥ 100 kg respectively, 500-ms gantry rotation time) with effective radiation exposures of 1.0 or 1.25 mSv, corresponding to 320 or 400 mA respectively. Each participant was scanned twice consecutively.[13]

All CT scans were analyzed by an experienced reader for the presence and amount of AAC using a commercially available workstation (Aquarius, TeraRecon, San Mateo, CA). Abdominal slices cranial to the aortic bifurcation were analyzed for AAC. AAC was defined radiographically as an area of at least 3 connected pixels with a CT attenuation >130 Hounsfield units (HU) applying 3-dimensional connectivity criteria (six points). The Agatston score (AS) was calculated by multiplying the area of each lesion with a weighted CT attenuation score dependent on the maximal CT attenuation within the lesion as described by Agatston and colleagues previously.[15] The area was calculated for each calcified lesion by multiplying the number of pixels >130 HU by the pixel area (in mm^2) using isotropic interpolation.[16] If an individual lesion appeared in > 1 CT cross-section, the total AS for the lesion was determined by summing the Agatston scores derived for each individual cross-section. Interobserver and intraobserver reproducibility for this method is high, as previously reported.[17]

The distribution of AAC among the healthy referent subsample and then the entire sample was categorized as percentiles of AAC (25^{th}, 50^{th}, 75^{th}, and 90^{th}), stratified by age and sex. The age- and sex-stratified healthy-referent cutpoints were applied to the entire study sample to determine the number of participants with AAC scores above the healthy-referent 90^{th} percentile of AAC. We prospectively selected the 90^{th} percentile threshold. In a complementary analysis, the distribution analysis of the entire sample AAC (at 25^{th}, 50^{th}, 75^{th} and 90^{th} percentiles) was stratified by 10-year CHD risk, determined by the Framingham Risk Score,[9] where low risk is <6%, intermediate risk ranges 6-20%, and high risk is >20%.[18,19] Finally, Spearman rank correlation coefficient (r_s) was calculated to assess the relation between AAC and CAC. (The non-parametric Spearman correlation was used due to non-normal distributions of calcium, but as with standard Pearson correlation, an $r_s > 0$ would suggest that AAC increases as CAC increases. The maximum possible value of $r_s = 1$ would indicate a perfect monotonic relationship between AAC and CAC, but in contrast to Pearson correlation, Spearman correlation does not assume a linear relationship between the 2 measures.) Concordance for agreement between AAC and CAC in stratifying all study participants as having high (>90^{th} healthy-referent percentile) or non-high (≤90^{th} healthy-referent percentile) burden of calcium, within the respective vascular beds, was assessed using the kappa statistic.

Results

A total of 3285 Offspring and Third Generation participants meeting study entry criteria underwent MDCT. AAC could be determined in 3267 (99.5%, 1665 men). Baseline characteristics of these participants are shown in Table 1. The distribution of AAC stratified by age and sex across the healthy referent-subsample (N=1656, 803 men) is shown in the top portion of Table 2. In each age and sex group, the percentage of participants who met entry criteria for the referent subsample decreased steadily with increasing age group. Conversely, the proportion of referent participants with non-zero AAC increased with age. Among referent participants below age 45, fewer than 1 in 6 participants had detectable AAC, whereas by age 65, approximately 9 of 10 referent participants had non-zero AAC. In both men and women, AAC scores increased markedly and monotonically, in a supralinear fashion, with age. When compared with the distribution of AAC across all study participants (Table 2, middle), the referent-subsample had consistently lower AAC scores within a given age- and sex- group, but the pattern of greater AAC burden with advancing age seen in the healthy referent sample was preserved in the entire study sample. Applying the age- and sex-specific thresholds for the 90th percentile of AAC from the referent subsample to the overall study sample (Table 2, bottom), we found that 18.9% of men and 19.4% of women had AAC scores above the 90th percentile. The proportion of participants above the 90th percentile thresholds did not differ by sex.

Considering the distribution of AAC across Framingham CHD risk categories for all participants (Table 3), we found that AAC burden increased markedly from the low to high risk categories. Finally, AAC was significantly correlated with CAC for both sexes (men: r_s=0.41, p<0.0001; women: r_s=0.38, p<0.0001). Table 4 shows the distribution of men and women stratified by healthy-referent 90th percentiles of AAC and CAC. Agreement was moderately high with kappa=0.56 for both men and women. Within this study sample the sensitivity of high (> 90th percentile) AAC for “predicting” similarly-defined high CAC was 67.8% with a specificity of 92.6% and positive and negative predictive values of 62.1 and 90.6%, respectively. These values were similar when considering each sex separately.

Discussion

The prevalence of abdominal aortic calcium increases with advancing age in both sexes, and AAC is widely prevalent by the middle of the sixth decade of life among members of a community-dwelling cohort free of clinically overt CVD. Even among study participants free of standard CVD risk factors including hypertension, dyslipidemia, diabetes, smoking and obesity, the majority of men and women have quantifiable AAC by age 55. In addition to greater prevalence with age, the amount or burden of AAC increases supralinearly with age. However, the prevalence of high AAC (defined as an AAC burden above the sex-and-age specific 90th percentile in a healthy referent subsample) is relatively stable across age groups and does not differ between sexes.

AAC burden increases markedly with greater 10-year CHD risk, as defined by the Framingham Risk Score, across the entire study sample in both sexes. The same pattern is seen in healthy referent participants of either sex (data not shown). Previous data have shown that AAC, even when measured by less sensitive techniques such as plain radiography, is strongly associated with risk of ischemic stroke, claudication, CHD, and overall CVD.[6,20,21] Further, aortic calcium adds to the prediction of events over and above traditional Framingham risk factors.[20] Our finding that there is a steep rise in AAC across low, intermediate, and high-risk strata of 10-year CHD risk suggest that AAC determined by MDCT may be useful for risk stratification. However, in our study there were relatively few

men and very few women in the high-CHD risk category, and therefore our estimates may be less reliable in these persons.

We found that AAC burden is significantly positively correlated with burden of CAC, and the strength of this association is similar between the sexes. With respect to stratification of study participants as having high (>90th percentile) or non-high burden of calcium in the coronary arteries versus the abdominal aorta, concordance was good in both sexes. In the context of an 18% prevalence of high CAC in the overall study sample, non-high AAC had a 91% negative predictive value for non-high CAC. However, we do not advocate use of AAC as a predictor of or surrogate for CAC based on these data. Criqui et al have shown that CAC and AAC differ with respect to their association with smoking and dyslipidemia, two important CVD risk factors.[22]

CAC has been correlated with presence of atherosclerotic disease in histopathologic studies[23] and has been advocated for CHD risk stratification.[24] Quantification of AAC may also be useful for risk stratification for both CHD and other forms of cardiovascular disease, and measurement of AAC may be possible at an earlier age than for CAC due to factors including the greater size of the aorta relative to the coronary arteries and the greater total calcium burden associated with the larger vessel. AAC might also be usefully quantified from other imaging studies not specifically performed to assess abdominal calcium burden, such as vertebral morphometry or CT colonography.[25,26] However, whether CHD risk can be predicted using MDCT-determined AAC requires further study. Additionally, if AAC is found to be useful for risk stratification, its adoption and routine clinical use would be facilitated by definition of cutpoints, as used for CAC. We do not propose cutpoints in the present study, as we have not related AAC either to CVD events or to other measures, e.g. coronary stenoses, but the wide range of AAC seen across age groups in the healthy referent-subsample suggests that age- and sex-specific cutpoints may be warranted.

The Framingham Heart Study is largely white, and generalization to other ethnic groups may be limited. Indeed, a significantly lower prevalence of AAC was noted in Hispanic and African-American participants in the Multiethnic Study of Atherosclerosis, suggesting that distributions of AAC should be considered by ethnic group.[27] Our study sample had a paucity of women with high Framingham Risk Scores, consequently our ability to make inferences in this group is extremely limited.

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Table 1

Baseline characteristics of the study sample.

	Men	Women
N	1665	1602
Age (years)	48.8±10.2	51.6±9.6
Offspring	33 %	42 %
Hypertension	29 %	25 %
Hyperlipidemia	23 %	20 %
Current cigarette smoking	13 %	12 %
Diabetes mellitus,	6.1 %	4.7 %
Body mass index (kg/m^2)	28.4±4.5	27.1±7.0

Table 2

Distribution of abdominal aortic calcium by sex and age group in the healthy referent sample and across all study participants.

	Men					Women				
Age (years)	<45	45-54	55-64	65-74	≥75	<45	45-54	55-64	65-74	≥75
	Referent Men (N=803)					Referent Women (N=853)				
N_{REF}	368 (58.5%)	250 (46.5%)	110 (40.7%)	57 (35.6%)	18 (25.4%)	293 (71.6%)	318 (58.1%)	154 (43.3%)	69 (33.3%)	19 (22.9%)
N_{REF} with AAC>0	57 (15.5%)	113 (45.2%)	90 (81.8%)	52 (91.2%)	18 (100.0%)	23 (7.8%)	72 (22.6%)	91 (59.1%)	60 (87.0%)	19 (100.0%)
AAC (Agatston score)										
Median [IQR]	0 [0,0]	0 [0,26]	109 [6,803]	1149 [284,3292]	2340 [1051,4790]	0 [0,0]	0 [0,0]	24 [0,295]	270 [36,1332]	2130 [1227,4360]
90th percentile	7	231	1922	4914	8177	0	73	946	2263	5742
	All Men (N=1665)					All Women (N=1602)				
N	626	538	270	160	71	409	547	356	207	83
N with AAC>0	140 (22.4%)	310 (57.6%)	233 (86.3%)	152 (95.0%)	71 (100.0%)	67 (16.4%)	186 (34.0%)	259 (72.8%)	185 (89.4%)	83 (100.0%)
AAC (Agatston score)										
Median [IQR]	0 [0,0]	6 [0,152]	403 [31,1335]	1998 [523,4378]	3450 [1444,7186]	0 [0,0]	0 [0,34]	127 [0,748]	833 [138,2493]	2403 [1013,4938]
90th percentile	47	828	3561	7199	12158	24	286	2195	4625	7358
Participants with high (>90th percentile) abdominal aortic calcium										
N > 90th	102 (16.3%)	115 (21.4%)	51 (18.9%)	35 (21.9%)	12 (16.9%)	67 (16.4)	103 (18.8%)	73 (20.5%)	54 (26.1%)	14 (19.4%)

Table 3

Distributions of abdominal aortic calcium in men and women stratified by 10-year cardiovascular event risk as defined by Framingham Risk Score.

	Men (N=1665)			Women (N=1601)		
10-Year Risk	<6%	6-20%	>20%	<6%	6-20%	>20%
N	739	805	121	1203	386	12
25th percentile of AAC	0	0	535	0	25	769
50th percentile of AAC	0	99	2654	0	377	3548
75th percentile of AAC	4	983	5197	73	2082	5316
90th percentile of AAC	171	2970	9422	833	4059	6587

N=1601 for women (differs from prior tables with N=1602) due to missing covariates in one woman

AAC = abdominal aortic calcium, IQR = interquartile range. N_{REF} = number of referent-group participants within each sex and age category, N_{REF} (%) = percentage of referent participants among all participants within an age/sex category. N_{REF} with AAC > 0 refers to the number of referent participants within each sex and age category with non-zero AAC; the corresponding percentage is calculated based on number of referent participants only (N_{REF} is the denominator).

Table 4

Distribution of men and women stratified by healthy-referent 90th percentiles of abdominal aortic calcium and coronary artery calcium.

	Men (N=1665)		Women (N=1602)	
Variable	**CAC≤90th**	**CAC>90th**	**CAC≤90th**	**CAC>90th**
AAC≤90th	1245 (76%)	91 (6%)	1137 (72%)	100 (6%)
AAC>90th	116 (7%)	188 (11%)	130 (8%)	215 (14%)

CAC = coronary artery calcium, AAC = abdominal aortic calcium, 90th = sex-specific 90th percentile for AAC or CAC.