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Associations of Cardiovascular Disease Risk Factors and Calcified Atherosclerosis with Aorto-Iliac Bifurcation Position: The Multi-Ethnic Study of Atherosclerosis (MESA)

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Abstract

We investigated associations of cardiovascular disease (CVD) risk factors and calcified atherosclerosis with aorto-iliac bifurcation position. The bifurcation position was determined by measuring the distance from the aorto-iliac bifurcation to the L5-S1 disc space (or aorto-iliac bifurcation distance [AIBD]), using computed tomography (CT) scans. The 1,711 study participants (51% male) had a mean age of 62 ± 10 years, and a mean AIBD of 26 ± 15 mm. In multivariable linear regression, older age, male gender, smoking, hypertension, larger aortic diameter, and smaller lumbar height, were each independently associated with a smaller AIBD (more caudal bifurcation position). In contrast, diabetes, elevated triglycerides, and increased pulse pressure were independently associated with a larger AIBD (more cephalad bifurcation position). These findings suggest age-related bifurcation descent is associated with CVD markers for aortic disease. Future studies should assess whether the bifurcation position is an independent prognosticator for CVD.

Keywords

Abdominal aorta; Aorto-iliac bifurcation; Atherosclerosis; Diabetes

Introduction

With older age, reported arterial wall changes in the aorta include dilatation, elongation, thickening, stiffening, tortuosity and calcium accumulation.[1–7] The position of the aorto-iliac bifurcation, where the aorta divides into the iliac arteries, also changes with age. Studies have reported that the aorto-iliac bifurcation is more caudally located in older individuals.[8–10] However, little is known about other factors associated with this location in humans. Specifically, it is uncertain whether the prevalence of atherosclerotic risk factors, or

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calcified atherosclerosis influences the bifurcation position. Existing studies are limited by small sample sizes, qualitative data, and clinic-based and/or homogenous populations. Also, no study has investigated how changes in lumbar spine length may influence the bifurcation position. This is important because the bifurcation position is usually referenced to the lumbo-sacral junction (L5-S1 disc space). Therefore, age-related changes in the lumbar spine, such as compression fractures, may result in an erroneous assumption of a more caudal bifurcation position due to changes in the aorta rather than changes in the spine.

We hypothesized that the bifurcation position may be a marker of systemic vascular aging. Therefore, the aim of this study was to determine whether cardiovascular disease (CVD) risk factors and calcified atherosclerosis, might be associated with the bifurcation position.

Methods

Study Participants

The Multi-Ethnic Study of Atherosclerosis (MESA) is a multi-center, prospective cohort designed to investigate the epidemiology of subclinical atherosclerosis. Participants included 6,814 men and women (age 45–84) of Caucasian, Hispanic-, African-, and Chinese-American descent, free from clinically manifest CVD at baseline. Participants were recruited between July 2000 and August 2002 at 6 U.S field centers; New York, NY; Baltimore, MD; Winston-Salem, NC; St Paul, MN; Chicago IL; and Los Angeles, CA. A detailed description of study design, recruitment methods, examination components and data collections has been published.[11]

During follow up visits between August 2002 and September 2005, 2202 MESA participants, representative of the study population, were asked to participate in an ancillary study that focused on abdominal aortic calcium (AAC). Of these, 2172 agreed to participate. Individuals were excluded if they were pre-menopausal, or had a recent abdominal computed tomography (CT) scan. The distance from the aorto-iliac bifurcation to the L5-S1 disc space, or the aorto-iliac bifurcation distance (ABID) (methodology below) was measured in 1711 participants with identifiable bifurcations and L5-S1 disc spaces on CT. The same CT scans were used to measure the height of the lumbar spine from L2 to L5. Signed informed consent was obtained for all participants, and institutional review board approval was obtained for all participating institutions.

Risk factor assessment

Participants were given standardized questionnaires at baseline, which were used to obtain information on demographics, medical history, smoking history, alcohol consumption and physical activity. A medication inventory was also performed, and medications were grouped based on use to treat either high blood pressure, or abnormal lipids. Blood pressure was measured 3 times in the seated position with a Dinamap model Pro 100 automated oscillometric sphygmomanometer. The average of the last 2 measurements was used. Standard measurements were taken for height and weight, and blood samples were obtained after a 12h fast for measurements of total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides and glucose.

Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Hypertension was defined as systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or current use of anti-hypertensive medication. Diabetes was defined as fasting plasma glucose > 126 mg/dL, or use of hypoglycemic medications. Dyslipidemia was defined as total to HDL cholesterol ratio ≥ 5, or reported use of medication to treat elevated cholesterol.[12] The Global Framingham Risk Score (Global FRS) was calculated based on 2008 criteria published by D'Agostino et al.[13] Global FRS assigns sex-specific points for values of age, total cholesterol, HDL cholesterol, systolic blood pressure, and smoking status, and diabetes to determine an individuals 10 year risk for a CVD event (heart failure, coronary, cerebrovascular, and peripheral artery disease).

Aorto-Iliac Bifurcation Distance

Aorto-iliac Bifurcation Distance (AIBD) was measured from abdominal CT scans using the following procedures. Computer software (Osiris 4.19, University of Geneva, Geneva, Switzerland) was used to identity coordinates in the x-, y-, and z-plane for the aortic bifurcation and the L5-S1 disc space. Both axial and sagittal views were used to ensure proper landmark identification. A straight distance between the aortic bifurcation and the L5-S1 disc space was calculated by taking the difference of the z-plane coordinates (Figure 1). Also, lumbar height, a determinant of the bifurcation position [due to bone or intervertebral disc disease] was measured as a distance from the inferior aspect of L2 to the superior aspect of L5 (a distance measureable on all CT scans). A single reader who was unaware of subject characteristics conducted the measurements. A random sample (n = 94) was re-read by the same reader, without knowledge of his previous AIBDs, approximately 3 months after the initial reads, with good agreement (interclass correlation = .89). The same software, as used for the AIBD measurements, was also used to determine abdominal aortic diameter (AAD) 5 cm proximal to the aortic bifurcation. In brief, an adjustable-size electronic caliper in the shape of a circle was used to measure the diameter (d) by fitting the caliper around the circumference (C) of the aortic adventitia. The computer then calculated the diameter from the circumference measurement using the equation, $d = C/\pi$. The diameter was measured 3 times, and the average of these measurements were used in the analysis.

Measures of Subclinical CVD

High-resolution B-mode ultrasonography was used to obtain bilateral common and internal carotid artery images. A Logiq 700 ultrasound machine (GE Medical Systems, Waukesha, WI) was used at all study centers. Maximum common carotid artery intima-media thickness (CIMT) was obtained from mean maximum wall thickness across all scans, from near and far walls.

Ankle-brachial index (ABI) was calculated from measurements obtained with the use of a 5-mHz hand-held Doppler (Nicolet Vascular, Golden CO) and has been previously described.[11] Low (ABI ≤ 0.9), normal (ABI 0.91 – 1.29), high (ABI ≥ 1.3) ABI was determined.[14]

Coronary artery calcium score (CAC) was measured from images obtained from CT scans of the chest. Coronary scans were performed with cardiac-gated electron beam scanners at 3

centers (Imatron C-150; Imatron, Inc., San Francisco, CA). The other centers used a prospectively electrocardiogram-triggered scan acquisition at 50% of the R-R interval with multi-detector scanners. All scans were read for CAC at the MESA CT Reading Center at the Los Angeles Biomedical Research Institute (Torrance, California). The methodology for acquisition and interpretation of the scans, as well as reproducibility of the readings, has been reported previously.[15] AAC was measured from the abdominal CT scans obtained at visits 2 and 3.[16] Images were reconstructed in a 35-cm field of view with 5-mm slice thickness. All scan scores were brightness adjusted with a standard phantom. Calcification was identified as a plaque of ≥ 1 mm^2 with density of >130 HU and quantified using the Agatston scoring method.[17] Estimates of radiation dose per scan ranged from 0.6–0.9 mSv and 0.7–1.1 mSv for men and women respectively.[18]

Statistical Analysis

Descriptive statistics for the study cohort were summarized by means (SD), medians (interquartile range), and frequencies for normally distributed continuous variables, skewed variables, and categorical variables, respectively. Analysis of covariance was used to calculate age- and gender-adjusted mean/prevalence of the subject characteristics by quartile of AIBD. In subsequent analysis, backwards-stepwise linear regression was utilized to identify exposure variables independently associated with AIBD. All variables [except Caucasian-, Hispanic-, African-, and Chinese-American ethnicity, which were forced into the model] achieving $P < .1$ significance level in backwards deletion were retained in the final model. CAC, AAC, and triglyceride levels were all strongly skewed, and therefore were log transformed [Ln(CAC+1), Ln(AAC+1), Ln(Trig)]. All analyses were conducted using PASW Statistics 18, version 18.0.3 (SPSS, Inc., 2009 Chicago, IL). P-value ≤ 0.05 (two-sided) was considered significant.

Results

Among the 1,711 study participants, the mean age was 62 ± 10 years, and 51% were men. The mean AIBD was 26 ± 15 mm. Age was a strong correlate of AIBD, such that on average, individuals > 80 years had bifurcations positioned 16 mm more caudal (i.e. smaller AIBD) than individuals < 50 years (Figure 2). Table 1 shows the age- and gender-adjusted mean/prevalence of cohort characteristics by quartiles of AIBD. Compared with the lowest quartile, those in higher quartiles (longer ABID, or more cephalad bifurcation position) were younger, less likely to have ever smoked, had reduced pulse pressure, greater lumbar height, elevated triglycerides and smaller AAD.

Next, we used multivariable linear regression to determine which CVD risk factors and markers of calcified atherosclerosis were independently associated with the AIBD. The results of the final model are displayed in Table 2. As expected, greater lumbar height was associated with a larger AIBD. Older age, male gender, smoking, hypertension, and larger AAD were each independently associated with a smaller AIBD. In contrast, diabetes, elevated triglycerides, and increased pulse pressure were independently associated with a larger AIBD.

Discussion

In a large multi-ethnic cohort of community-living men and women who were free of clinical CVD at baseline, we observed that a more caudal position of the aorto-iliac bifurcation, relative to the L5-S1 disc space, (smaller AIBD) was independently associated with CVD markers for aortic disease. These findings are suggestive of age-related bifurcation descent, and provide clues to mechanisms underlying this process.

Others have reported findings suggestive of age-related bifurcation descent.[8–10] This study is the first to report similar findings in a large, multi-ethnic, community cohort. In our study, a more caudal position of the aortic bifurcation was observed much more commonly in participants after the 6th decade of life. Importantly, our study benefitted from availability of lumbar height, which allows us to make the novel contribution that age-related bifurcation descent is independent of reductions in lumbar height (presumably from bone/intervertebral disc disease). Thus we postulate elongation of the aorta as a possible mechanism for age-related bifurcation descent.

Male gender, smoking, and hypertension were independently associated with a more caudal location of the aortic bifurcation. All are known risk factors for abdominal aortic aneurysm (AAA).[19,20] Though not well understood, aortic aneurysmal disease is thought to be caused by weakening of the aortic wall, from elastin fiber degradation, which results in progressive dilation.[19] We postulate that a similar process may influence progressive elongation, resulting in age-related bifurcation descent. The cross-sectional design of our study prevented us from exploring this hypothesis. However, supporting this hypothesis were results showing larger AAD was independently associated with age-related bifurcation descent.

Diabetes, elevated triglycerides, and increased pulse pressure, in contrast, were independently associated with a more cephalad position of the aortic bifurcation. Interestingly, diabetes is associated with a lower risk for AAA.[19,20] Thus, our findings that diabetes is associated with a more cephalad position of the aortic bifurcation in contrast to male gender, smoking, and hypertension, supports our assertion that similar mechanisms may underlie both dilatation [resulting in AAA] and elongation [resulting in age-related bifurcation descent]. Diabetes is associated with glycation products thought to cause rigidity and stiffening of arterial walls.[21,22] We postulate that aortic stiffening, thought to impede dilatation in AAA, would also impede elongation in age-related bifurcation descent. Our results showing that increased pulse pressure, a measure of arterial stiffness, was also associated with a more cephalad bifurcation position, is consistent with this idea. Unfortunately, carotid-femoral artery pulse wave velocity, the gold standard measure for arterial stiffness was unavailable for our analysis. Unsupportive of our hypothesis was the observation that elevated triglycerides were also independently associated with a more cephalad position of the aortic bifurcation. This is because abnormal lipids are modestly associated with greater risk for AAA.[19,20]

No associations were observed for CIMT, abnormal ABI, CAC, AAC, or Global FRS with the bifurcation position. Also, medications to treat high blood pressure and abnormal lipids

were not associated with the bifurcation position. We hypothesized that AAC, like diabetes, would stiffen the aorta thus impede a more caudal positioning of the bifurcation with age. We also hypothesized that calcified atherosclerosis in other vascular beds would be associated with a more caudal bifurcation position. Our results were unsupportive of both hypotheses. As expected, Global FRS which predicts CVD events in major vascular beds, excluding the aorta, was not associated with the bifurcation position. More studies are needed to corroborate our findings, and further investigate these issues.

Strengths of our study include precise measures from abdominal CT scans, a community-living sample, multi-ethnic representation, and a large sample size. This study also makes the novel contribution of investigating how changes in lumbar height (from lumbar and intervertebral disc disease) may affect the position of the aorto-iliac bifurcation. However, our study also has important limitations. The cross-sectional design cannot definitively determine the temporal sequence of the demonstrated correlates of AIBD. Also, prevalent CVD at baseline was an exclusion criterion, and results may potentially differ in persons with clinically apparent CVD. Finally we acknowledge the [current] limited clinical utility of our findings because ultrasonography, the primary screening modality for aortic disease, is proven, more cost-effective, and without radiation exposure. However, our results have strong research implications, which if coupled with advancements in diagnostic imaging may help establish future clinical use.

In a large, multi-ethnic, community-living cohort, free of clinical CVD at recruitment, older age, male gender, smoking, hypertension, larger AAD, and smaller lumbar height were each independently associated with a more caudal location of the aorto-iliac bifurcation, whereas diabetes, elevated triglycerides, and increased pulse pressure were associated with a more cephalad location. Our findings suggest similar mechanism may influence age-related bifurcation descent and abdominal aortic disease. Future studies are needed to determine if the AIBD is a useful marker for aging in the aorta, and/or a prognostic factor for CVD, independent of chronological age.

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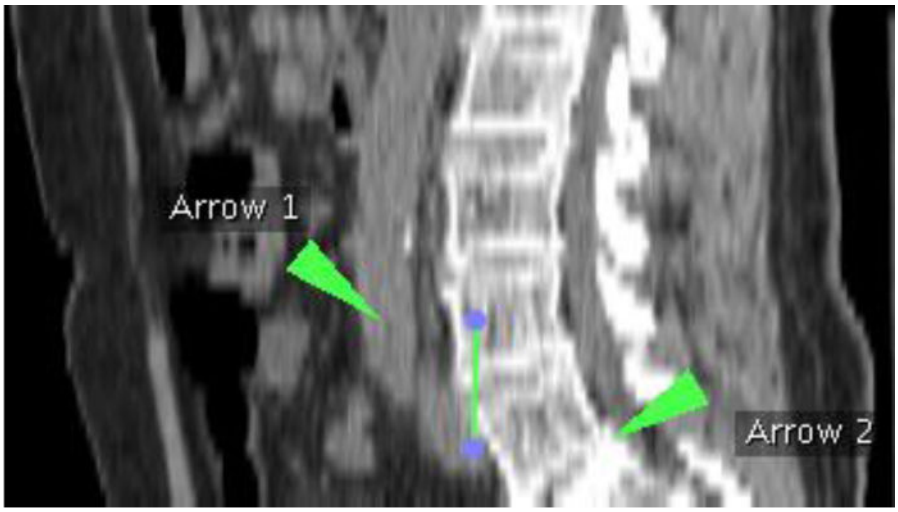

Figure 1.
Sagittal computer tomography image of the abdomen. Arrow 1 = aorto-iliac bifurcation. Arrow 2 = posterior L5-S1 disc space. Vertical line = aorto-iliac bifurcation distance (AIBD), calculated from coordinates (X-, Y-, and Z-planes) of arrow 1 and 2.

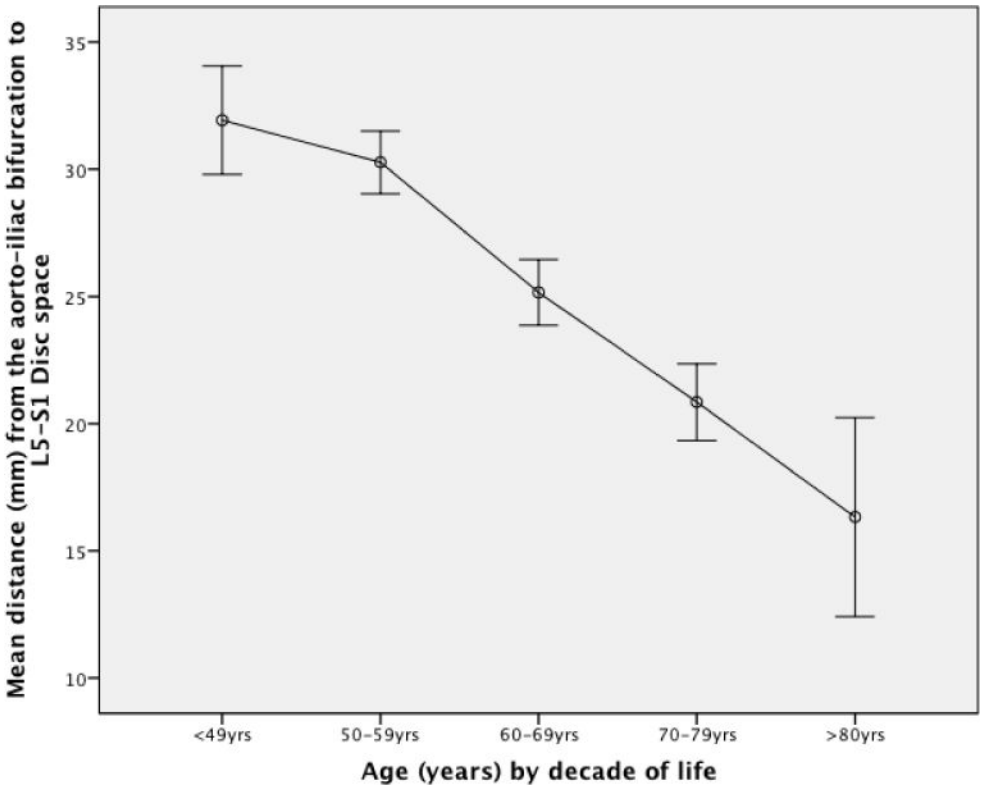

Figure 2.
Mean distance from the aortic bifurcation to the L5-S1 disc space by decade of life.

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Table 1

Distribution of cohort characteristics by quartile of the distance from the aorto-iliac bifurcation to the L5-S1 disc space: The Multi-Ethnic Study of Atherosclerosis (MESA)

Characteristics (N=1711)	Cohort[a]	Quartile 1[b]	Quartile 2 [b]	Quartile 3 [b]	Quartile 4 [b]	P-value[c]
AIBD, mm	26 (15)	7 (9)	22 (3)	31 (3)	45 (8)	< .01
Age, years	62 (10)	67 (9)	63 (9)	60 (9)	59 (9)	< .01
Male	51%	54% (50)	49% (50)	49% (50)	51% (50)	0.37
Ethnicity						
Caucasian	42%	43% (50)	42% (49)	40% (49)	45% (50)	0.37
Hispanic	25%	24% (43)	26% (44)	27% (44)	23% (42)	0.45
African	21%	24% (43)	26% (44)	27% (45)	23% (42)	0.43
Chinese	12%	11% (31)	14% (35)	12% (33)	11% (31)	0.28
Smoking	50%	58% (49)	49% (50)	45% (50)	50% (50)	< .01
Hypertension	45%	55% (50)	44% (50)	43% (50)	37% (48)	0.11
Diabetes	11%	10% (30)	10% (30)	12% (33)	12% (33)	0.07
Dyslipidemia	36%	37% (48)	37% (48)	37% (48)	33% (47)	0.67
Ankle-brachial index						
Normal ABI	92%	90% (30)	92% (27)	92% (26)	93% (25)	0.99
Low ABI	4%	5% (22)	3% (17)	3% (16)	3% (17)	0.49
High ABI	4%	5% (21)	4% (19)	4% (19)	3% (17)	0.57
Common CIMT, mm	.87 (.19)	.92 (.22)	.87 (.18)	.85 (0.18)	.85 (0.19)	0.48
Body mass index, Kg/m^2	28 (5)	28 (5)	28 (5)	28 (5)	28 (5)	0.71
Pulse pressure, mmHg	54 (17)	57 (18)	55 (18)	53 (17)	52 (15)	0.5
Height, cm	167 (10)	166 (10)	166 (10)	167 (10)	168 (10)	0.07
Lumbar height, cm	7.82 (0.70)	7.64 (.74)	7.75 (.69)	7.93 (.64)	7.98 (.63)	< .01
AAD, mm	19 (3)	20 (3)	19 (3)	18 (2)	18 (2)	< .01
HDL cholesterol, mg/dL	51 (15)	51 (16)	52 (15)	50 (15)	51 (15)	0.48
Triglycerides, mg/dL	116 [79, 169]	129 (75)	129 (73)	139 (83)	141 (88)	< .05
CAC	1 [0, 91] 50	190 (414)	126 (277)	91 (223)	90 (277)	0.46

Characteristics (N=1711)	Cohort[a]	Quartile 1[b]	Quartile 2 [b]	Quartile 3 [b]	Quartile 4 [b]	P-value[c]
AAC	0 [259, 1587] 72	1843 (2781)	1231 (2123)	1060 (1908)	996 (1847)	0.22
Hypertension mediations	35%	34% (49)	33% (48)	37 (47)	36% (47)	0.49
Dylipidemia medications	16%	16% (39)	17% (38)	16% (36)	16% (35)	0.97
Global FRS	15 (9)	14 (9)	14 (9)	15 (9)	15 (9)	0.49

[a] Data are means (±SD), proportions, and medians [25th percentile, 75th percentile] %>0. AIBD = aorto-illac bifurcation distance (distance from aortic bifurcation to L5-S1 disc space), ABI = ankle-brachial index, CIMT = carotid intima media thickness, BMI = body mass index, AAD = abdominal aortic diameter, HDL = high density lipoprotein, CAC = coronary artery calcium, AAC = abdominal aortic calcium, FRS = Framingham risk score

[b] Quartiles are age and gender adjusted means (±SD) and prevalence (±SD)

[c] P-value represents differences in age and gender adjusted means and prevalence across quartiles

Table 2

Multivariable linear regression of associations between cardiovascular disease risk factors, and markers for atherosclerosis with the distance from the aortic-iliac bifurcation to L5-S1 disc space: The Multi-Ethnic Studies of Atherosclerosis (MESA)

Model	Beta	95% CI	P-Value
Age (Years)	−.37	(−0.45, −0.28)	< .01
Male (vs. Female)	−2.10	(−4.20, −0.01)	< .05
Ethnicity			
Caucasian		Reference	
Hispanics	−1.12	(−3.04, 0.81)	0.26
African	−.09	(−2.05, 1.87)	0.93
Chinese	−2.13	(−4.57, 0.31)	0.08
Height, cm	.08	(−0.03, 0.20)	0.18
Ever Smoking	−1.58	(−3.00, −0.15)	< .05
Hypertension	−1.80	(−3.48, −0.13)	< .05
Diabetes	2.53	(0.39, 4.66)	< .05
Triglycerides[a], mg/DL	3.65	(0.45, 6.84)	< .05
Pulse pressure, mmHg	.05	(0.01, 0.11)	< .05
AAD, mm	−.76	(−1.07, −0.45)	< .01
Lumbar height, cm	3.96	(2.69, 5.22)	< .01

[a]Log transformed, AAD = abdominal aortic diameter