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Aortic Unfolding Measurement Using Non-Contrast Cardiac CT: Normal Range of Low-Risk Subjects

관상동맥 석회화 CT에서 측정한 대동맥 전개: 저위험 환자군에서의 정상 범위

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Purpose This study aimed to assess the factors influencing aortic unfolding (AU) defined by aortic width on coronary artery calcium (CAC) scan and determine the normal limits for AU.
Materials and Methods In this retrospective study, we measured AU in 924 asymptomatic subjects who underwent CAC scanning during routine health screening from June 2015 to June 2018. Multivariate regression analysis was used to evaluate the factors influencing AU. After the exclusion of subjects with risk factors associated with AU, 283 subjects were included in the analysis of normal values of AU. Mean AU, standard deviation, and upper normal limit were calculated.
Results Sex, age, CAC score, body mass index, body surface area, hypertension, left ventricular hypertrophy, plasma creatinine, and smoking were significantly associated with AU. The mean AU was 102.2 ± 12.8 mm for men and 93.1 ± 10.7 mm for women. AU increased with advancing age (9.6 mm per decade).
Conclusion AU determined from a single measurement on CAC scans was associated with cardiovascular risk factors. The normal limits of AU were defined by age, sex, and body surface area in low-risk subjects in this study.

Index terms Aorta; Coronary Vessels; Multidetector Computed Tomography; Left Ventricular Hypertrophy; Reference Values

INTRODUCTION

Aortic dimensions can be measured by ultrasound, CT, and cardiac MR (1). Aortic root

dimension measurement can be useful because it is associated with aging and traditional coronary heart disease (CHD) risk factors. In addition, aortic root dimension has been shown to be predictive of cardiovascular disease outcomes in subjects ≥ 65 years of age (2). However, that study used transthoracic echocardiography, which is limited when the acoustic window is inadequate. Alteration of aortic arch geometry, which is associated with the age-related increase in aortic arch stiffness and central pulse pressure, can be measured by MRI (1). However, the procedure is expensive, time-consuming, and not universally available.

Aortic unfolding (AU) is a term used to describe the widened mediastinum on chest radiographs. This change occurs with aging and generally reflects proximal aortic dilation, aortic arch widening, and decreased curvature (3). In addition, it implies aortic degeneration and is associated with hypertension (1). A recent study evaluated AU defined using the aortic width on non-contrast electrocardiography (ECG)-synchronized cardiac CT. AU was associated with age, body surface area (BSA), and hypertension, and was positively associated with the coronary artery calcium (CAC) score, a well-established surrogate marker of cardiovascular risk (3). However, that study included a relatively small sample size.

In the present study, we defined AU as the width of the aorta as measured on non-contrast ECG-synchronized cardiac CT because we thought that it might be a marker of structural changes such as dilation and aortic arch widening of the aorta. We determined the normal limits for AU in asymptomatic low-risk adult subjects and analyzed the relationship between AU measurement and CAC score, left ventricular hypertrophy (LVH) as surrogate markers of cardiovascular risk.

MATERIALS AND METHODS

STUDY POPULATION

The retrospective study protocol was approved by the Institutional Review Board of the Severance Hospital, Yonsei University Health System. Because patient records and information was anonymized and de-identified prior to analysis, the written informed patient consent was waived by the IRB (IRB No. 4-2012-0111).

We included 1081 consecutive healthy subjects, aged 29–88 years, who underwent CAC scanning for screening purposes from June 2015 to June 2018 at our institution. During a retrospective review of the medical records of all of the patients to collect clinical information, including demographic data and cardiovascular risk factors, 22 subjects were excluded due to incomplete clinical information (missing information concerning plasma glucose and creatinine levels [$n = 10$], LVH on ECG [$n = 6$], blood pressure [BP, $n = 5$], and smoking [$n = 1$]). An additional 99 patients with CAC images acquired at the end systolic phase (40% or 45%) were also excluded to avoid the possibility of differences in the measurements of AU between end systole and mid-diastole (4). Subjects with known clinical CHD ($n = 20$), thoracic aortic aneurysm ($n = 1$), severe pulmonary disease causing atelectasis or fibrosis around the aorta ($n = 12$), or kyphosis/straight back syndrome ($n = 3$), which can influence the measurement, were also excluded. Finally, 924 subjects were included.

DATA COLLECTION

Information concerning cardiac risk factors in each subject was collected through medical record review. Weight and height were also obtained, and the body mass index (BMI) and BSA were calculated using the Mosteller method (5). A family history of CHD was defined as a CHD event occurring in a first degree relative (males aged < 55 years and females aged < 65 years). LVH was defined as a Sokolow-Lyon index on the ECG: sum of SV_1 plus RV_5 or $RV_6 \geq 35$ mm (6). Hypertension was defined as a systolic BP $\geq$ 140 mm Hg, a diastolic BP $\geq$ 90 mm Hg, or the use of antihypertensive medications. Subjects were classified as having diabetes if they had an established diagnosis of diabetes mellitus made by a physician and/or were receiving treatment with insulin or oral hypoglycemic agents, or if their measured fasting glucose was $\geq$ 126 mg/dL. Dyslipidemia was defined as a total/high-density lipoprotein cholesterol ratio > 5 or taking a hypocholesterolemic drug (statin or fibrate). Blood samples for lipids, fasting glucose, and plasma creatinine were obtained the same day as the CT examination, and the duration of time between CT and ECG was within 7 days. Current smoking or a history of smoking was defined as a positive smoking status (7, 8).

CT IMAGING PROTOCOL

Patients were imaged using a 64-detector CT scanner (Lightspeed VCT; GE Healthcare, Waukesha, WI, USA). The scanning direction was craniocaudal and extended from the pulmonary artery bifurcation to the bottom of the heart. A non enhanced prospective ECG-gated axial scan was performed at 70% of the R-R interval with the following parameters: rotation time, 350 ms; section collimation, 0.625 mm $\times$ 64; section width, 2.5 mm; tube voltage, 120 kV; and effective tube current-time product, 200 mA. CAC data were evaluated using a workstation (AW Volume Share 4; GE Healthcare, Milwaukee, WI, USA). CAC was identified as a high-attenuation area in the coronary artery, where the attenuation exceeded the threshold of 130 Hounsfield unit (HU) in a minimum of three contiguous pixels. CAC scores were calculated according to the Agatston method (9).

MEASUREMENT OF AU USING NON-CONTRAST CARDIAC CT

For interpretation, images were transferred to a dedicated workstation (Centricity PACS; GE Healthcare). AU was defined as the longest distance from the ascending aorta to the descending aorta at the level of the pulmonary artery bifurcation on an axial CT image because this landmark is easy to define anatomically, rendering it reproducible (Fig. 1) (3). Systematic measurements of AU were performed by two blinded radiologists with different degrees of experience: reader 1 was a resident with 1 year of experience, and reader 2 was a radiologist with 7 years of experience in cardiac imaging. To assess intraobserver variability, data were analyzed twice by each reader in random order with an interval of at least 4 weeks between readings. The aortic unfolding index (AUI) was calculated as follows: AUI = AU/BSA.

STATISTICAL ANALYSIS

All statistical analyses were performed using SAS software (ver. 9.2; SAS Institute, Cary, NC, USA). For graphs, we used PASW Statistics 18 (ver. 18.0.3; SPSS, Chicago, IL, USA) and Excel 2007. The clinical characteristics of the study sample are reported as means $\pm$ standard devi-

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Fig. 1. Aortic unfolding measurement. Transaxial slice at the pulmonary artery bifurcation from a coronary artery calcium scan showing the method for deriving the aortic unfolding. The white line represents the farthest distance from the ascending aorta to the descending aorta at the level of the pulmonary artery bifurcation.
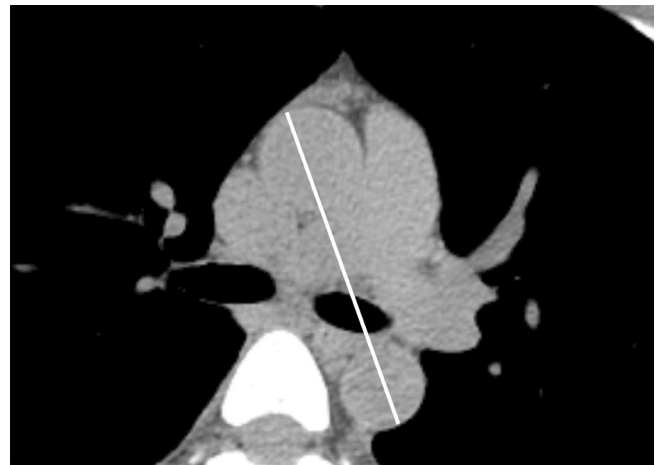
ations or as numbers with percentages. Continuous variables were compared using t test for two groups, and categorical variables were compared using the Pearson chi-squared statistic. Intra- and inter-observer reproducibility was calculated using the intra-class correlation coefficient (ICC), where ICC < 0.4 represented poor reliability, ICC between 0.4 and 0.75 represented fair-to-good reliability, and ICC > 0.75 represented excellent reliability. Univariate and multivariate regression analyses according to sex, age, CAC score, BMI, BSA, family history of CHD, hypertension, diabetes mellitus, LVH on ECG, plasma creatinine, dyslipidemia, and smoking history were performed. Using multivariate regression analysis, we sought to identify parameters associated with AU and to exclude patients with these parameters from the second analysis groups. Thus, the second analysis group would include only patients without parameters that influence the AU and can be considered essentially "normalized." For this analysis, we grouped the subjects by sex and age for the AU. We calculated mean AU, standard deviation, and upper normal limit (mean + 2 standard deviations) for each group. We also used linear regression models to create BSA versus AU nomograms by age groups and calculated the formula for predicting AU. A p value less than 0.05 was deemed to indicate statistical significance.

RESULTS

Table 1 shows the clinical characteristics of the study population, which comprised 601 males and 323 females. The mean age was 53.5 ± 9.1 years (range, 29–82 years). No difference in age was seen between males and females (53.3 ± 9.1 years in males and 53.9 ± 9.0 years in females). Males had a higher BSA and BMI than females. Males had a higher prevalence of risk factors than females, except for dyslipidemia and a family history of CHD. There was excellent interobserver reliability between readers 1 and 2 (ICC = 0.996, 95% confidence interval [CI] = 0.996–0.997; $p < 0.001$) and intraobserver reliability between the first and second evaluations (reader 1: ICC = 0.985, 95% CI = 0.980–0.988, $p < 0.001$; reader 2: ICC = 0.992, 95% CI = 0.989–0.994, $p < 0.001$).

Table 1. Clinical Characteristics of the Initial Study Population

	Overall (n = 924)	Female Subjects (n = 323)	Male Subjects (n = 601)	p-Value
Age, years	53.5 ± 9.1	53.9 ± 9.0	53.3 ± 9.1	0.313
BSA, m^2	1.7 ± 0.2	1.6 ± 0.1	1.9 ± 0.1	< 0.001
BMI, kg/m^2	24.2 ± 3.1	23.1 ± 3.3	24.8 ± 2.7	< 0.001
Family history of CHD, %	111 (12.0)	38 (11.8)	73 (12.1)	0.871
LVH, %	135 (14.6)	28 (8.7)	107 (17.8)	0.001
Plasma creatinine, mg/dL	0.9 ± 0.2	0.7 ± 0.1	1.0 ± 0.1	< 0.001
Hypertension, %	226 (24.8)	62 (19.2)	164 (27.3)	0.003
Diabetes, %	105 (11.5)	16 (5.5)	89 (14.8)	0.001
Dyslipidemia, %	68 (7.4)	26 (8.0)	42 (7.0)	0.588
Smoking, %	498 (54.5)	31 (9.6)	467 (77.7)	< 0.001
CAC score	52.0 ± 182.0	13.5 ± 49.7	72.7 ± 221.0	< 0.001
Aortic unfolding, mm	102.5 ± 13.6	96.1 ± 12.3	106.0 ± 13.0	< 0.001

Values are expressed as means ± standard deviation or patient number with percentage.
BMI = body mass index, BSA = body surface area, CAC = coronary artery calcium, CHD = coronary heart disease, LVH = left ventricular hypertrophy

Table 2. Multivariate Regression: Associations between Cardiovascular Risk Factors and Aortic Unfolding

Predictors	Parameter Estimates	95% CI	p-Value
Male sex	4.05	1.76–6.34	0.001
Age, years	0.93	0.86–1.00	< 0.001
CAC score	0.02	0.02–0.03	0.021
BMI, kg/m^2	0.61	0.31–0.90	< 0.001
BSA, m^2	19.76	13.03–26.49	< 0.001
Hypertension, %	5.07	3.68–6.47	< 0.001
Diabetes, %	-1.23	-3.16–0.69	0.209
LVH, %	2.22	0.63–3.81	0.006
Plasma creatinine, mg/dL	-6.41	-10.45–-2.36	0.002
Dyslipidemia, %	-1.89	-5.68–-1.90	0.329
Smoking, %	1.53	0.03–3.03	0.046

BMI = body mass index, BSA = body surface area, CAC = coronary artery calcium, CI = confidence interval, LVH = left ventricular hypertrophy

FACTORS PREDICTING AU

Univariate analysis showed a significant association between AU and male sex (β = 9.81, p < 0.001), age (β = 0.91, p < 0.001), CAC score (β = 0.02, p < 0.001), BMI (β = 1.69, p < 0.001), BSA (β = 25.65, p < 0.001), LVH (β = 6.54, p < 0.001), plasma creatinine (β = 18.06, p < 0.001), hypertension (β = 12.25, p < 0.001), and smoking (β = 6.41, p < 0.001). In multivariate regression analysis, male sex, age, CAC score, BMI, BSA, hypertension, LVH, plasma creatinine, and smoking were associated with AU (Table 2).

NORMAL VALUE OF AU

Sex, age, CAC score, BMI, BSA, hypertension, LVH, plasma creatinine, and smoking were

found to be associated with AU in multivariate analysis. A CAC score > 400 was defined as abnormal because an advanced CAC score (> 400) poses the highest risk for CHD events (10). A BMI of 30 kg/m^2 or more is considered obese (11). In total, 641 subjects who had BMI $\geq$ 30 kg/m^2 (n = 37), hypertension (n = 226), LVH (n = 135), plasma creatinine > 1.2 mg/dL (n = 41), smoking (n = 498), and/or CAC score > 400 (n = 29) were excluded from establishing normal values of AU. Finally, 283 subjects were included to establish the normal values of AU (212 females; mean age 52.6 $\pm$ 8.3 years). The mean AU and AUI for this subgroup were 95.4 $\pm$ 11.9 mm and 58.6 $\pm$ 7.6 mm/m^2, respectively. The corresponding upper limit of normal AU and AUI were 119.2 mm and 73.8 mm/m^2. The mean AU for males was 102.2 $\pm$ 12.8 mm and the corresponding upper limit of normal was 127.8 mm. The mean AUI for males was 56.3 $\pm$ 8.7 mm/m^2 and the corresponding upper limit of normal AUI was 73.7 mm/m^2. The mean AU for females was 93.1 $\pm$ 10.7 mm, and the corresponding upper limit of normal was 114.5 mm. The mean AUI for females was 59.4 $\pm$ 7.0 mm/m^2 and the corresponding upper limit of normal AUI was 73.4 mm/m^2. AU increased with advancing age (9.6 mm per decade, p < 0.001), and increasing BSA (30.9 mm per square meter of BSA, p < 0.001). AUI also increased with advancing age (6.1 mm/m^2 per decade, p < 0.001). The mean values of AU and AUI in male subjects were greater than those in females in all age groups stratified by 10-year intervals (Fig. 2). Table 3 details AU parameters, stratified by sex and age. Upper limit of AU was 102.0, 103.0, 114.9, 128.2 mm for female subjects and 112.1, 116.1, 125.3, 131.2 mm for mal subjects, respectively. Upper limit of AUI was 63.0, 65.6, 74.7, 83.2 mm for female subjects and 60.4, 60.6, 70.7, 78.2 mm for male subjects, respectively. Nomograms for AU for sex and age group by BSA are shown in Fig. 3. The predicted AU could be calculated using the following equation: AU = -1.873 – 2.036 $\times$ sex (if male = 1, if female = 2) + 0.958 $\times$ age + 30.927 $\times$ BSA.

DISCUSSION

By performing CAC scans in asymptomatic subjects, we found that AU measured by CT

Fig. 2. Relationship between age and aortic unfolding or aortic unfolding index.
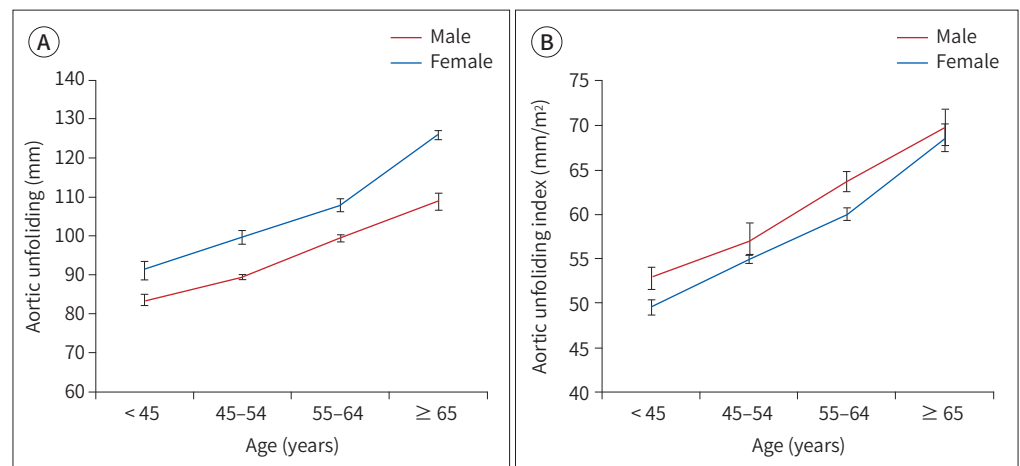A, B. Both aortic unfolding (A) and aortic unfolding index (B) increases with age in male and female subjects.

Table 3. Aortic Unfolding and Aortic Unfolding Index of Normalized Subjects according to Sex and Age

| Age (Years)	Aortic Unfolding (mm)			Aortic Unfolding Index (mm/m²)		
	All (n = 283)	Female Subjects (n = 212)	Male Subjects (n = 71)	All (n = 283)	Female Subjects (n = 212)	Male Subjects (n = 71)
< 45	86.3 ± 10.3, 106.9 (64.5–107.7), n = 55	83.6 ± 9.2, 102.0 (64.5–106.0), n = 36	91.3 ± 10.4, 112.1 (75.1–107.7), n = 19	51.5 ± 5.5, 62.5 (39.6–62.2), n = 55	52.8 ± 5.1, 63.0 (43.2–62.2), n = 36	49.2 ± 5.6, 60.4 (39.6–57.9), n = 19
45–54	91.3 ± 8.1, 107.5 (75.3–117.9), n = 114	89.4 ± 6.8, 103.0 (75.3–106.5), n = 93	99.7 ± 8.2, 116.1 (80.6–117.5), n = 21	56.2 ± 8.1, 72.4 (40.9–66.1), n = 114	57.0 ± 4.3, 65.6 (46.7–66.1), n = 93	53.0 ± 3.8, 60.6 (40.9–58.1), n = 21
55–64	101.7 ± 8.9, 119.5 (84.2–124.5), n = 92	99.3 ± 7.8, 114.9 (84.2–113.8), n = 66	107.9 ± 8.7, 125.3 (91.7–124.5), n = 26	62.6 ± 5.7, 74.0 (50.1–79.6), n = 92	63.7 ± 5.5, 74.7 (51.0–79.6), n = 66	59.9 ± 5.4, 70.7 (50.1–72.5), n = 26
≥ 65	112.6 ± 11.2, 135.0 (91.3–130.1), n = 22	108.8 ± 9.7, 128.2 (91.3–129.9), n = 17	125.8 ± 2.7, 131.2 (123.0–130.1), n = 5	69.5 ± 6.2, 81.9 (60.2–84.7), n = 22	69.8 ± 6.7, 83.2 (60.2–84.7), n = 17	68.6 ± 4.8, 78.2 (61.8–74.7), n = 5

Values are expressed as means ± standard deviation, upper limits (range).

was associated with cardiovascular risk factors, including age, sex, CAC score, BMI, BSA, hypertension, LVH, plasma creatinine, and smoking. The mean AU and AUI of asymptomatic low-risk subjects were 95.4 ± 11.9 mm and 58.6 ± 7.6 mm/m², respectively. The corresponding upper limit of normal AU and AUI were 119.2 mm and 73.8 mm/m². AU (9.6 mm per decade, $p < 0.001$) and AUI (6.1 mm/m² per decade, $p < 0.001$) increased with advancing age. The unique aspect of our study should be emphasized: it is the first large-scale asymptomatic population-based study in which AU was determined by CAC scan. We presented the normal range and upper limit of AU according to age and sex as well as a calculation formula for predicted AU.

Aging is associated with structural and functional changes of the cardiovascular system, including the large arteries (12). Age-associated vascular structural remodeling includes increased vascular intimal thickness with luminal dilatation and vascular stiffening. Age-associated vascular functional changes include altered regulation of vascular tone (endothelial dysfunction) (13). With aging, repetitive pulsations (some 30 million/year) of the aorta cause fatigue and fracture of elastin lamellae of the central arteries, leading to aortic stiffening and increased mean aortic BP, and finally to aortic dilation. Stiffening increases left ventricular afterload with hypertrophy, decreases the capacity for myocardial perfusion, and increases stresses on small arterial vessels, particularly in the brain and kidney (14).

First, we demonstrated that AU, assessed by a 64-detector CT scanner for CAC scoring, is associated with age. The results of the present study are similar to that of an earlier study using CAC scanning (3). We believed that AU would be influenced by the diameters of the ascending and descending thoracic aorta, and lengthening and decreased curvature of the aortic arch. Previous reports have stated that an ascending aortic diameter (1.1 mm per decade) and length (6 mm per year) increase significantly with age. Increasing ascending aortic length leads to aortic arch widening and decreased curvature (1, 15, 16). These findings may explain our results. The length of ascending aorta increase more than the ascending aortic diameter with increasing age (1). However, MRI or three-dimensional (3D) reconstruction is needed to measure the length of the ascending aorta. Therefore, our method can be a simple and effective alternative. The geometric changes in the aortic arch were significantly related to decreased ascending aortic distensibility, increased aortic arch pulse wave velocity (PWV)

Fig. 3. Predicted aortic unfolding according to sex, age, and BSA.
A-D. Nomograms of normal values of aortic unfolding in male subjects.
E-H. Nomograms of normal values of aortic unfolding in female subjects.
BSA = body surface area
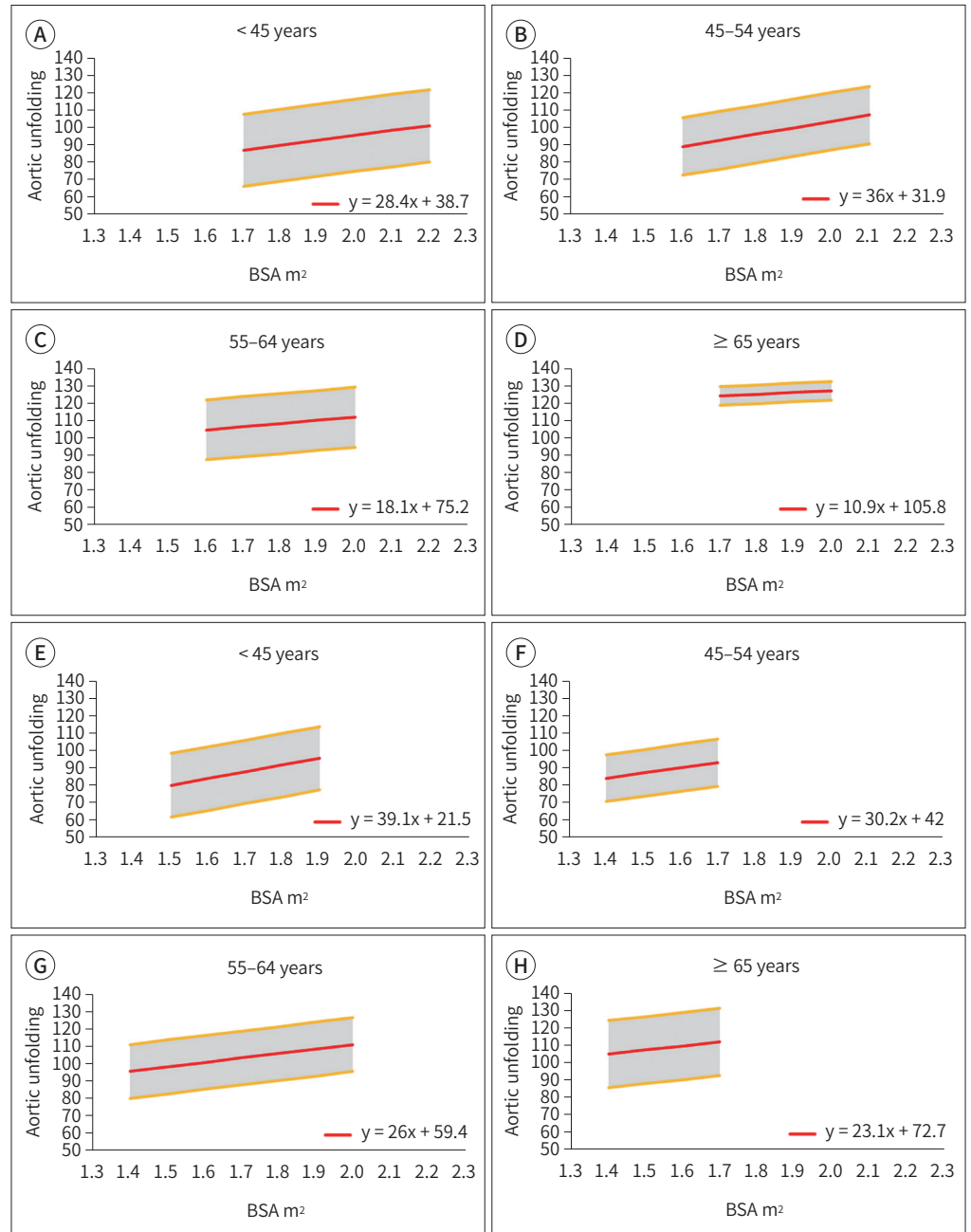
and central BP, and increased LV mass (16); thus, we suggest that AU likely has similar relationships with these factors. Further evaluation of the relationship between AU with aortic PWV is needed.

Additionally, sex, CAC score, BMI, BSA, hypertension, LVH, plasma creatinine, and smoking are associated with AU. The results of the present study correspond well with those of an earlier study that reported that AU determined using CAC scanning is related to BSA and hy-

pertension (3). However, other findings differ from those of that study, in that sex, BMI, LVH, plasma creatinine, and smoking are positively associated with AU (3). In another study, hypertension accelerated thoracic aorta enlargement and AU with respect to an aging effect in asymptomatic male subjects who underwent CAC scanning using 3D automated algorithm reconstruction (17). In that study, diastolic BP was associated with the radius of curvature, aortic arch width, as well as three segments of the thoracic aortic diameter. Most likely, the relatively constant diastolic BP along the aorta reflects the minimum tension stimulating the aortic wall and affects AU. Systolic BP is mostly related to aortic arch diameter, descending thoracic aortic diameter, aortic arch width, and radius of curvature, while the role of systolic BP is controversial (17). The classic notion that associates aging with systolic BP increase is described above. Although it is controversial whether these two factors are affected by male sex, it is known that age, BSA, and CAC score are significant predictors of ascending aortic diameter (4, 18, 19). Smoking and dyslipidemia are independent predictors of descending aortic diameter (18). These factors would influence AU.

Another important finding is the significant association between AU and LVH, valuable surrogate markers of cardiovascular risk. LVH has been associated with various adverse cardiovascular events, including stroke, sudden death, myocardial infarction, CHD, and congestive heart failure (20-22). It is noteworthy that AU was related to LVH independently of conventional cardiovascular risk factors, as reflected by age and sex.

Finally, we established reference values according to age, sex, and body size for AU in a normalized population in a selective manner. For the normalized population, we excluded patients with clinical parameters that were associated with increasing AU from the final analysis. If it is impossible to examine a large number of healthy subjects of various ages and both sex, this statistical approach to selecting a normalized group is a reasonable way to determine normal limits for variable measurements (18).

Some limitations of the present study should be noted. First, this was a single-center retrospective study. Second, although our initial unselected group consisted of a relatively large number of patients, due to the exclusion of 641 subjects and stratification process, the number of normalized male subjects was relatively small. Third, our study is also limited to asymptomatic patients from a health check-up without follow-up, which is insufficient to form conclusions about cardiac events. Thus, there is a need to validate our findings in a larger cohort with long-term follow-up. Finally, we measured AU using 2D measurements. Recent studies have evaluated aortic geometry with non-contrast CT, but such measurement requires a longer scan range to cover the aortic arch and additional 3D reconstruction (17, 23, 24). Thus, we believe our measurement may be a simple way to measure AU reliably using CAC scanning without an additional radiation dose. However, further studies regarding the relationship between our methods of measurement and those using 3D segmentation algorithms may be needed.

In summary, AU determined from a single measurement on CAC scans was associated with age and other cardiovascular risk factors. The normal limits of AU were defined by age, sex, and BSA in low-risk subjects. CAC score and LVH, well-established surrogate markers of cardiovascular disease were positively associated with AU. Further study to evaluate AU as a potential predictor of cardiovascular risk is warranted.

Author Contributions

Conflicts of Interest

Funding

REFERENCES

1. O'Rourke M, Farnsworth A, O'Rourke J. Aortic dimensions and stiffness in normal adults. *JACC Cardiovasc Imaging* 2008;1:749-751

2. Gardin JM, Arnold AM, Polak J, Jackson S, Smith V, Gottdiener J. Usefulness of aortic root dimension in persons > or = 65 years of age in predicting heart failure, stroke, cardiovascular mortality, all-cause mortality and acute myocardial infarction (from the Cardiovascular Health Study). *Am J Cardiol* 2006;97:270-275

3. Lee JW, Hur J, Kim YJ, Lee HJ, Nam JE, Kim HY, et al. Aortic unfolding determined using non-contrast cardiac computed tomography: correlations with age and coronary artery calcium score. *PLoS One* 2014;9:e95887

4. Lin FY, Devereux RB, Roman MJ, Meng J, Jow VM, Jacobs A, et al. Assessment of the thoracic aorta by multidetector computed tomography: age- and sex-specific reference values in adults without evident cardiovascular disease. *J Cardiovasc Comput Tomogr* 2008;2:298-308

5. Mosteller RD. Simplified calculation of body-surface area. *N Engl J Med* 1987;317:1098

6. Antikainen R, Grodzicki T, Palmer AJ, Beevers DG, Coles EC, Webster J, et al. The determinants of left ventricular hypertrophy defined by Sokolow-Lyon criteria in untreated hypertensive patients. *J Hum Hypertens* 2003;17:159-164

7. Möhlenkamp S, Schmermund A, Lehmann N, Roggenbuck U, Dragano N, Stang A, et al. Subclinical coronary atherosclerosis and resting ECG abnormalities in an unselected general population. *Atherosclerosis* 2008;196:786-794

8. Blacher J, Asmar R, Djane S, London GM, Safar ME. Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients. *Hypertension* 1999;33:1111-1117

9. Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M Jr, Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. *J Am Coll Cardiol* 1990;15:827-832

10. Budoff MJ, Nasir K, McClelland RL, Detrano R, Wong N, Blumenthal RS, et al. Coronary calcium predicts events better with absolute calcium scores than age-sex-race/ethnicity percentiles: MESA (Multi-Ethnic Study of Atherosclerosis). *J Am Coll Cardiol* 2009;53:345-352

11. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation* 2014;129: S102-S138

12. Hickson SS, Butlin M, Graves M, Taviani V, Avolio AP, McEniery CM, et al. The relationship of age with regional aortic stiffness and diameter. *JACC Cardiovasc Imaging* 2010;3:1247-1255

13. Inoue N, Maeda R, Kawakami H, Shokawa T, Yamamoto H, Ito C, et al. Aortic pulse wave velocity predicts cardiovascular mortality in middle-aged and elderly Japanese men. *Circ J* 2009;73:549-553

14. O'Rourke MF, Hashimoto J. Mechanical factors in arterial aging: a clinical perspective. *J Am Coll Cardiol* 2007;50:1-13

15. Sugawara J, Hayashi K, Yokoi T, Tanaka H. Age-associated elongation of the ascending aorta in adults. *JACC Cardiovasc Imaging* 2008;1:739-748

16. Redheuil A, Yu WC, Mousseaux E, Harouni AA, Kachenoura N, Wu CO, et al. Age-related changes in aortic arch geometry: relationship with proximal aortic function and left ventricular mass and remodeling. *J Am Coll Cardiol* 2011;58:1262-1270

17. Craiem D, Chironi G, Casciaro ME, Redheuil A, Mousseaux E, Simon A. Three-dimensional evaluation of

thoracic aorta enlargement and unfolding in hypertensive men using non-contrast computed tomography. *J Hum Hypertens* 2013;27:504-509

18. Wolak A, Gransar H, Thomson LE, Friedman JD, Hachamovitch R, Gutstein A, et al. Aortic size assessment by noncontrast cardiac computed tomography: normal limits by age, gender, and body surface area. *JACC Cardiovasc Imaging* 2008;1:200-209

19. Chironi G, Orobinskaia L, Mégnien JL, Sirieix ME, Clément-Guinaudeau S, Bensalah M, et al. Early thoracic aorta enlargement in asymptomatic individuals at risk for cardiovascular disease: determinant factors and clinical implication. *J Hypertens* 2010;28:2134-2138

20. Kannel WB. Left ventricular hypertrophy as a risk factor: the Framingham experience. *J Hypertens Suppl* 1991;9:S3-S8; discussion S8-S9

21. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Left ventricular mass and incidence of coronary heart disease in an elderly cohort. The Framingham Heart Study. *Ann Intern Med* 1989;110:101-107

22. Kannel WB, Cobb J. Left ventricular hypertrophy and mortality--results from the Framingham Study. *Cardiology* 1992;81:291-298

23. Craiem D, Chironi G, Redheuil A, Casciaro M, Mousseaux E, Simon A, et al. Aging impact on thoracic aorta 3D morphometry in intermediate-risk subjects: looking beyond coronary arteries with non-contrast cardiac CT. *Ann Biomed Eng* 2012;40:1028-1038

24. Kurugol S, San Jose Estepar R, Ross J, Washko GR. Aorta segmentation with a 3D level set approach and quantification of aortic calcifications in non-contrast chest CT. *Annu Int Conf IEEE Eng Med Biol Soc* 2012;2012:2343-2346

대한영상의학회지

관상동맥 석회화 CT에서 측정한 대동맥 전개: 저위험 환자군에서의 정상 범위

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목적 이 연구의 목적은 관상동맥 석회화 CT에서 측정한 대동맥 폭으로 정의한 대동맥 전개(aortic unfolding)에 영향을 미치는 요인을 평가하고 대동맥 전개의 정상 범위를 알아보는 것이다.

대상과 방법 이 후향적 연구에서 우리는 2015년 6월부터 2018년 6월까지 건강검진을 목적으로 관상동맥 석회화 CT를 시행 받은 924명의 무증상 성인에서 대동맥 전개를 측정했다. 다변량 회귀 분석을 사용하여 대동맥 전개에 영향을 미치는 요인들을 평가했다. 그 후 대동맥 전개와 관련된 위험 요소가 있는 성인을 제외되고 283명의 성인이 대동맥 전개의 정상값 분석에 포함되었다. 대동맥 전개의 평균, 표준 편차 및 상한값이 계산되었다.

결과 성별, 나이, 관상동맥 석회화 점수, 체질량지수, 체 표면적, 고혈압, 좌심실 비대, 혈장 크레아티닌, 흡연은 대동맥 전개와 유의한 관계가 있었다. 평균 대동맥 전개값은 남성의 경우 102.2 ± 12.8 mm, 여성의 경우 93.1 ± 10.7 mm였다. 대동맥 전개값은 연령이 증가할 수록(10년당 9.6 mm)으로 증가했다.

결론 관상동맥 석회화 CT에서 측정된 대동맥 전개는 심혈관 위험 인자들과 관련이 있었다. 또한 본 연구에서 저위험군에서 대동맥 전개의 정상 범위를 나이, 성별 및 체표표면적당으로 정의하였다.

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