ORIGINAL ARTICLE

Comparison of coronary artery dimensions in patients with chronic aortic regurgitation or stenosis

Aort yetersizliği veya darlığı olan hastaların koroner arter çaplarının karşılaştırılması

Hamza Sunman, M.D., Mehmet Erat, M.D., Kadriye Gayretli Yayla, M.D., Engin Algül, M.D., Mehmet Aytürk, M.D.,# Lale Dinç Asarcıklı, M.D., Murat Bilgin, M.D., Tolga Çimen, M.D., Ahmet Akyel, M.D., Sadık Açıkel, M.D., Mehmet Doğan, M.D., Ekrem Yeter, M.D.

Department of Cardiology, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey
#Department of Cardiology, Keçiören Training and Research Hospital, Ankara, Turkey

ABSTRACT

Objective: Effects of various conditions on coronary artery dimensions is an important research topic, and data regarding effect of aortic valvular diseases are limited. Aim of the present study was to investigate effects of aortic regurgitation (AR) and aortic stenosis (AS) on coronary artery dimensions.

Methods: Coronary dimensions of 95 patients (35 with isolated AR, 30 with isolated AS, and 30 without any valvular disease) were calculated. Patients with severe coronary artery disease and concurrent moderate to severe additional valvular disease were excluded. Mean diameter of major coronary arteries was determined using quantitative coronary angiography.

Results: The 3 study groups were similar in terms of baseline characteristics. Diameter of left main coronary artery was found to be greater in AR group than AS group (2.66±0.57 mm/m^2 vs 2.36±0.49 mm/m^2; p=0.015). Mean diameter of left anterior descending and left circumflex arteries were found to be similar in AR and AS groups, and greater than control group. Mean diameter of right coronary artery was found to be greater in AR group compared with controls; however, no significant difference was found in same measurement between AS group and controls.

Conclusion: Present study findings indicate that coronary dimensions in AR group tend to be greater than AS group. Further studies investigating factors that affect coronary dimensions would be beneficial in order to demonstrate mechanisms and differences in AR and AS groups.

ÖZET

Amaç: Çeşitli durumların koroner arter çapları (KAÇ) üzerine etkisi, önemli bir araştırma konusudur ve aort kapak hastalıklarının etkisi ile ilgili veriler kısıtlıdır. Bu çalışmada, aort yetersizliğinin (AY) ve aort darlığının (AD) KAÇ üzerine etkisini incelemeyi amaçladık.

Yöntemler: Toplam 95 hastanın (sadece AY'si olan 35, sadece AD'si bulunan 30 ve kapak hastalığı olmayan 30 kontrol hastası) koroner arter çapları hesaplandı. Ciddi koroner arter hastalığı, eşlik eden orta-ileri ek kapak hastalığı olan hastalar çalışma dışı bırakıldı. Ana koroner arterlerin ortalama çapı kantitatif koroner arteriyografi ile hesaplandı.

Bulgular: Üç çalışma grubu temel karakteristikler açısından benzerdi. Sol ana koroner arter çapı AY grubunda, AD grubuna göre daha geniş bulundu (2.66±0.57 mm/m^2 ve 2.36±0.49 mm/m^2; p=0.015). Sol ön inen arter ve sirkumfleks arter çapları AD ve AY grupları arasında benzerken, kontrol grubuna göre daha geniş bulundu. Sağ koroner arter çapı AY grubunda kontrol grubuna göre daha geniş olmasına rağmen AD ile kontrol grubu arasında anlamlı farklılık bulunmadı.

Sonuç: Çalışma bulgularımızda koroner arter çaplarının AY grubunda, AD grubuna göre daha geniş olmaya meyilli olduğu gösterilmiştir. Koroner arter çapları üzerine etkili faktörleri inceleyen ileri araştırmalar, AY ve AD grupları arasındaki farklılığı ve mekanizmasını ortaya koyması bakımından yararlı olacaktır.

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Correspondence: Dr. Hamza Sunman. Dışkapı Yıldırım Beyazıt Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, 06110 Ankara, Turkey.
Tel: +90 312 - 596 29 43 e-mail: hamzasunman@gmail.com

Determination of both physiological and pathological factors affecting coronary dimensions has been an important field of research for many years. Coronary dimensions may change acutely to meet short-term need, or in order to achieve long-term, chronic adaptation. Coronary dimensions vary even in healthy individuals. In addition to factors leading to acute coronary artery dilatation, such as exercise, factors such as atherosclerosis cause long-term change in coronary dimensions.[1–3] Changes in coronary dimensions are also seen in some other pathological conditions, such as coronary artery ectasia,[4,5] high-flow coronary fistula[6] and Kawasaki syndrome.[7] Furthermore, natural processes like aging, racial factors, and gender differences may influence coronary dimensions.[8–10] Angiographic studies have reported that coronary dimensions increase in response to pathological conditions like hypertension, in which left ventricular (LV) mass is increased.[11] Coronary dimensions have also been shown to increase secondary to increased LV mass due to disease, such as aortic stenosis (AS) and hypertrophic cardiomyopathy.[12] Although there are small-scale experimental and clinical studies investigating effect of aortic valvular diseases, such as aortic regurgitation (AR), on coronary artery dimension, there is no study in the literature that compares effects of AR and AS on coronary dimensions separately.[13–16] Coronary dimensions may be greater in patients with AS and AR, depending on increased myocardial mass. However, due to increased cardiac output in AR, coronary expansion may be greater than in AS.

The present study investigated effect of isolated aortic valvular disease (AR and AS) on coronary dimensions.

METHODS

Study patients

In this cross-sectional study, patients who underwent coronary angiography were examined for aortic valvular disease and those with severe valvular disease were identified. Patients with isolated AR or AS and control group with similar baseline characteristics were included in the study. Demographic features of the patients, including age, gender, smoking history, hypertension (HT), diabetes mellitus (DM), hyperlipidemia, and body surface area (BSA) were recorded. HT was defined as diastolic blood pressure ≥90 mmHg, systolic blood pressure ≥140 mmHg, or self-reported use of antihypertensive drug. DM was diagnosed in patients who had history of oral anti-diabetic drug or insulin use, or fasting blood glucose ≥126 mg/dL at start of study. Patients were divided into 3 groups: (a) patients with severe AR (no concomitant moderate-severe AS), (b) patients with severe AS (no concomitant moderate-severe AR), and (c) control group with similar baseline characteristics and no concomitant moderate-severe valvular dysfunction. Exclusion criteria included obstructive (>50%) atherosclerotic stenosis (any luminal narrowing of proximal segments of coronary arteries measured), history of myocardial infarction, previous cardiac surgery, acute valvular pathology, moderate to severe stenosis or insufficiency in non-aortic valves, coronary ectasia, coronary artery fistula, coronary artery anomaly, cardiomyopathy, congenital heart disease, coronary artery spasm, short or insufficient proximal coronary artery segments that cannot be measured accurately, inflammatory disease, or history of cancer. Approval of the local ethics committee was obtained for this study.

Abbreviations:

AP	*Anterior-posterior*
AR	*Aortic regurgitation*
AS	*Aortic stenosis*
BSA	*Body surface area*
DM	*Diabetes mellitus*
HT	*Hypertension*
LAD	*Left anterior descending artery*
LAO	*Left anterior oblique*
LCx	*Left circumflex artery*
LMCA	*Left main coronary artery*
LVEDD	*Left ventricular end diastolic dimension*
QCA	*Quantitative angiography system*
RAO	*Right anterior oblique*
RCA	*Right coronary artery*
TCA	*Total coronary artery*

Echocardiographic examination

Severity of valvular disease was assessed with echocardiography. Transthoracic echocardiographic evaluation was performed using Philips iE33 echocardiographic imaging system (Phillips Healthcare, B.V., Eindhoven, The Netherlands) with a S5–1 transducer (from 5 to 1 MHz) by a single, experienced cardiologist. LV end diastolic dimension (LVEDD), LV ejection fraction (Teichholz formula), diastolic interventricular septal thickness, and LV posterior wall thickness were calculated on parasternal long axis view. Severity of aortic valvular disease was evaluated with Doppler echocardiography based on principles specified in the European Society of Cardiology guidelines. Briefly, AS with valve area ≤1.0 cm^2 and mean gradient ≥40 mmHg was considered severe. Pa-

tients with low flow-low gradient or pseudo-severe AS were excluded. Criteria used to define severe AR were regurgitant volume ≥60 mL/beat, vena contracta ≥6 mm, and pressure half-time ≤200 milliseconds.[17] LV mass was calculated based on formula of 0.8x [1.04x (diastolic interventricular septal thickness + LVEDD + diastolic posterior wall thickness) 3- (LVEDD) 3] + 0.6. LV mass index was calculated by dividing LV mass by BSA.

Coronary angiographic evaluation

Coronary angiography procedure was performed by an experienced cardiologist according to previously defined standard methods. In brief, coronary arteries were cannulated using 6-F diagnostic catheter and Judkins technique. Nonionic agents were used as contrast agents. In order to view left main coronary artery (LMCA), left anterior descending artery (LAD), and left circumflex artery (LCx) in standard way for each coronary angiographic operation, anterior-posterior (AP) caudal, left anterior oblique (LAO) caudal, LAO cranial, AP cranial, and right anterior oblique (RAO) caudal projections were acquired. LAO and RAO projections were acquired in order to view right coronary artery (RCA). In cases with inadequate image quality, measurements were taken from projection where the vessel was best viewed. All coronary artery dimension measurements were made using quantitative angiography system (QCA) (Axiom Artis QCA system, Siemens, A.G., Munich, Germany). Calibration was done with 6-F Judkins catheter using automated edge detection algorithm. Contour of vessel to be measured was calculated in mm independently of the operator by software analysis, once again using automated edge detection algorithm. Measurements were taken at end diastolic phase and vessel segment of at least 0.5 cm was used for each measurement. Location of QCA measurements were mid-segment of LMCA, middle part of proximal LAD (proximal to first septal/diagonal branch), middle of proximal LCx (proximal to first marginal branch), and proximal part of RCA (1.5 to 2 mm after origin). Mean values of coronary dimensions of LMCA, LAD, LCx, and RCA, as well as mean total coronary artery (TCA) diameter were corrected with BSA and compared between groups. TCA diameter was sum of cross-sectional diameter of the 3 major coronary arteries supplying LV, i.e., RCA, LAD, and LCx.

Statistical analysis

Statistical analysis was performed using SPSS Statistics 20 software (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±SD and categorical variables were expressed as absolute numbers and percentages. Kolmogorov-Smirnov test was used to determine if continuous variables were normally distributed or not. For categorical variables, differences between patient groups were tested using chi-square test. Qualitative and quantitative values of more than 2 groups were compared using analysis of variance models and Kruskal-Wallis test. P value <0.05 was considered statistically significant.

RESULTS

The study included 65 patients with aortic valvular disease: 35 with AR and 30 with AS. Control group consisted of 30 patients without aortic valvular disease. Mean age was 62.88±11.54 years in AR group, 65.33±14.11 years in AS group, and 61.27±17.04 years in control group (p=0.541). There were 12 (34.3%) female and 23 (65.7%) male patients in AR group, 11 (36.7%) female and 19 (63.3%) male patients in AS group, and 11 (36.7%) female and 19 (63.3%) male patients in control group; no statistically significant difference was found between groups in terms of gender distribution (p=0.974). There was no difference in terms of risk factors, BSA, or cardiovascular medications used by the patients. All 3 groups were similar in baseline characteristics (Table 1).

Echocardiographic findings revealed the following: median regurgitant volume of 64 mL (range: 60–87 mL), median vena contracta was 6.2 mm (range: 6.0–6.9 mm), and pressure half-time of 190 milliseconds (range: 160–200 ms) in AR group; median aortic peak gradient was 68 mmHg (range: 64–92 mmHg), median aortic mean gradient was 42 mmHg (range: 40–53 mmHg), and median aortic valve area of 0.95 cm^2 (range: 0.6–1.0 cm^2) in AS group. When echocardiographic features of the groups were compared, LVEDD, aortic root, and ascending aortic diameter were found to be greater in AR group compared with controls (LVEDD: 5.90±0.84 cm *vs* 4.70±0.48 cm, respectively; p<0.001; aortic root: 2.58±0.26 cm *vs* 2.26±0.13 cm; p<0.001; ascending aortic diameter: 3.92±0.65 cm *vs* 3.43±0.32 cm; p<0.001). Whereas diameter of aortic root was greater in AS group com-

Table 1. Baseline characteristics of the study groups

Parameters	Control (n=30)	Aortic regurgitation (n=35)	Aortic stenosis (n=30)	p*
Age, year	61.27±17.04	62.88±11.54	65.33±14.11	0.541
Female gender, n (%)	11 (36.7)	12 (34.3)	11 (36.7)	0.974
Hypertension, n (%)	17 (56.7)	21 (60)	16 (53.3)	0.864
Diabetes mellitus, n (%)	7 (23.3)	11 (31.4)	11 (36.7)	0.528
History of smoking, n (%)	14 (46.7)	20 (57.1)	11 (36.7)	0.256
Body height, m	1.70 (1.60–1.85)	1.68 (1.58–1.85)	1.70 (1.60–1.78)	0.110
Body weight, kg	75.67±7.81	72.97±10.19	70.77±9.99	0.137
Body mass index (kg/m²)	26.57±2.63	25.82±3.74	24.92±2.72	0.127
Body surface area (m²)	1.88±0.13	1.84±0.15	1.82±0.15	0.201
Aspirin, n (%)	14 (46.7)	20 (57.1)	19 (63.3)	0.421
Beta-blocker, n (%)	16 (63.3)	12 (34.3)	18 (60.0)	0.095
Statin, n (%)	18 (60.0)	17(48.6)	21 (70.0)	0.214
RAAS blocker, n (%)	13 (43.3)	23 (65.7)	14 (51.6)	0.144

*Analysis of variance and Kruskal-Wallis test. RAAS: Renin angiotensin aldosterone system.

Table 2. Echocardiographic characteristics of the study population

	Control (n=30)	AR (n=35)	AS (n=30)	p	p*	p#	p^
	Mean±SD	Mean±SD	Mean±SD				
LVEDD (cm)	4.70±0.48	5.90±0.84	4.88±0.66	<0.001	<0.001	0.229	<0.001
Ejection fraction (%)	54.83±9.42	49.0±10.93	56.0±11.74	0.018	0.026	0.675	0.016
Aortic root (cm)	2.26±0.13	2.58±0.26	2.46±0.24	<0.001	<0.001	<0.001	0.071
Ascending aorta (cm)	3.43±0.32	3.92±0.65	3.53±0.41	<0.001	<0.001	0.319	0.004
Left ventricle mass (g)	162.34±40.17	231.08±63.18	244.57±57.10	<0.001	<0.001	<0.001	0.326

AR: Aortic regurgitation; AS: Aortic stenosis; LVEDD: Left ventricular end-diastolic diameter; SD: Standard deviation.
p values from analysis of variance between *control group and AR group; #control group and AS group; ^AR and AS groups.

pared with controls, ascending aortic diameter was similar (2.46±0.24 cm *vs* 2.26±0.13 cm; p<0.001 and 3.53±0.41 cm *vs* 3.43±0.32 cm; p=0.319, respectively). LV mass was less in control group (162.34±40.17 g) than in AR and AS groups (231.08±63.18 g and 244.57±57.10 g, respectively) (p<0.001). Although LV mass was greater in AS group than in AR group, difference did not reach statistical significance (p=0.326). It was observed that results did not change when evaluation considered BSA. Detailed echocardiographic characteristics are presented in Table 2.

TCA dimension was greater in AR group and in AS group compared with control group (9.70±1.81 mm and 9.17±1.48 *vs* 8.10±1.28 mm; p<0.001 and p=0.009, respectively). No significant difference was observed between AR and AS groups in TCA diameter (9.70±1.81 mm *vs* 9.17±1.48 mm; p=0.167). Similarly, indexed TCA diameter calculated according to BSA was found to be greater in both AR group (5.31±1.16 mm/m^2 *vs* 4.32±0.72 mm/m^2, respectively; p<0.001) and AS group (5.09±0.97 mm/m^2 *vs* 4.32±0.72 mm/m^2, respectively; p=0.003) compared with controls, and no significant difference was found between AR and AS groups (5.31±1.16 mm/m^2 *vs* 5.09±0.97 mm/m^2, respectively; p=0.372). Mean diameter of LMCA, LAD, and LCx were greater in AR and AS groups than in control group (LMCA: 4.86±0.87 mm,

Table 3. Coronary artery diameter of the study groups

	Control (n=30)	AR (n=35)	AS (n=30)	*p*	*p**	*p*#	*p*^
	Mean±SD	Mean±SD	Mean±SD				
LMCA, mm	3.64±0.58	4.86±0.87	4.23±0.71	<0.001	<0.001	0.001	0.001
LMCA-indexed (mm/m²)	1.94±0.36	2.66±0.57	2.36±0.49	<0.001	<0.001	<0.001	0.015
LAD (mm)	2.79±0.52	3.37±0.62	3.40±0.66	<0.001	<0.001	<0.001	0.824
LAD-indexed (mm/m²)	1.48±0.27	1.84±0.38	1.89±0.41	<0.001	<0.001	<0.001	0.600
LCx (mm)	2.59±0.44	3.21±0.66	2.98±0.57	<0.001	<0.001	0.009	0.112
LCx-indexed (mm/m²)	1.38±0.24	1.76±0.41	1.66±0.37	<0.001	<0.001	0.003	0.267
RCA (mm)	2.72±0.60	3.12±0.87	2.78±0.54	0.046	0.024	0.757	0.051
RCA-indexed (mm/m²)	1.45±0.35	1.71±0.54	1.54±0.34	0.050	0.018	0.423	0.117
TCA (mm)	8.10±1.28	9.70±1.81	9.17±1.48	<0.001	<0.001	0.009	0.167
TCA-indexed (mm/m²)	4.32±0.72	5.31±1.16	5.09±0.97	<0.001	<0.001	0.003	0.372
RCA dominancy, n (%)	23 (76.7)	26 (74.3)	22 (73.3)	0.954			

AR: Aortic regurgitation; AS: Aortic stenosis; LAD: Left anterior descending artery; LCx: Left circumflex artery; LMCA: Left main coronary artery; RCA: Right coronary artery; TCA: Total coronary artery; SD: Standard deviation.
p values from analysis of variance between *control group and AR group; #control group and AS group; ^AR and AS groups.

4.23±0.71 mm *vs* 3.64±0.58 mm; LAD: 3.37±0.62 mm, 3.40±0.66 mm *vs* 2.79±0.52 mm; and LCx: 3.21±0.66 mm, 2.98±0.57 mm *vs* 2.59±0.44 mm, respectively) (p<0.05). Diameter of LMCA was greater in AR group than in AS group (4.86±0.87 mm *vs* 4.23±0.71 mm; p=0.003). Although mean diameter of RCA was found to be statistically significantly greater in AR group compared with controls (3.12±0.87 mm *vs* 2.72±0.60 mm; p=0.024), no significant difference was found between AS group and controls (2.78±0.54 mm *vs* 2.72±0.60 mm; p=0.757). Results of coronary dimensions did not change when evaluation considered BSA (Table 3).

DISCUSSION

In this study, it was determined that diameter of LMCA was greater and RCA tended to be greater in AR group than in AS group, but mean diameter of LAD and LCx were found to be similar in those 2 groups. In addition, we found that TCA diameter was greater in AR and AS patients compared to control group, but no significant difference was found between AR and AS groups.

Coronary dimensions also vary in healthy individuals. Several studies have been conducted to investigate correlation between baseline values of coronary dimensions and gender, age, ethnic, and racial factors in healthy persons. Yang et al. researched gender difference in coronary dimensions. According to their study, mean diameter was LMCA: 4.7±0.7 mm, LAD: 3.7±0.6 mm, LCx: 3.6±0.6 mm, and RCA: 3.8±0.8 mm in men, and LMCA: 4.2±0.6 mm, LAD: 3.3±0.5 mm, LCx: 3.0±0.5 mm, and RCA: 3.3±0.6 mm in women.[18] Dodge et al. published a comprehensive angiographic study of coronary dimensions in population of the USA. In that study, mean diameter of LMCA was reported to be 4.25±0.5 mm, LAD: 3.4±0.5 mm, LCx: 3.15±0.6 mm, and RCA: 3.6±0.6mm.[19,20] Coronary dimensions of control group in the present study were similar to those of previous studies.

In general, internal diameter of mammalian arteries is associated with luminal blood flow during development, and this correlation continues over lifetime.[21] Experimental changes in local blood flow lead to alterations in diameter of the vessels: Decreased flow leads to significant reduction in artery diameter, and sustained increase in flow causes corresponding increase in size of healthy arteries.[22–24] Increase in myocardial mass that occurs with LV hypertrophy means quantity of blood required to supply that site also increases. In order to meet this need, coronary arteries become dilated via endothelium.[25]

Studies have reported increase in coronary dimen-

sions as an adaptive mechanism to LV hypertrophy. Villari et al. demonstrated that progression of LV hypertrophy is associated with increase in LAD and LCx dimensions, whereas size of RCA remains unchanged. In addition, they found that despite enlargement of left coronary arteries, cross-sectional area of LMCA per 100 g of LV muscle mass decreased. Hence, they concluded that increase in coronary artery size appears to become inadequate as severity of LV hypertrophy increases.[15] However, in that study, type of aortic valvular pathology (AR or AS) was not evaluated separately. In the present study, we found that LV myocardial mass was greater in AR and AS patients compared with control group, but no significant difference was found between AR and AS groups. We found coronary dimensions of AR group tended to be greater than in AS group. Namely, diameter of LMCA was greater in AR group than in AS group. In contrast to AS group, diameter of RCA was observed to be significantly larger in AR group than in controls. Because LV mass was similar, enlargement in coronary dimensions due to LV hypertrophy cannot explain the difference between AR and AS patients. Although reason for increase in coronary dimension in AR patients is not fully understood, several mechanisms have been proposed: (a) AR leads to increase in systolic blood flow in systole and lower forward flow in diastole. These alterations in systemic blood flow could be expected to change regional circulations, such as coronary arteries. (b) Decrease in coronary vascular resistance develops in patients with AR, which increases coronary blood flow, and (c) epicardial coronary arteries have been suggested to hold more blood in systole in AR patients. These hemodynamic factors might explain differences between AR and AS groups.[26]

The present study has several limitations, which may affect the assessment. First, it was an observational, single-center, cross-sectional study. In addition, results of this study were based on single index of coronary artery diameter (angiographic luminal diameter) and there are no follow-up data of study patients. Although previous experimental and clinical studies had similar number of patients, further studies with larger population would yield results that are more reliable. In this study, physical capacity of the patients was not specified, and coronary vasomotor tone was not calculated. Another limitation is that more reliable method, such as intravascular ultrasonography, was not used to calculate coronary artery dimension.[27] Use of nitroglycerin during coronary angiography affects vascular dimension. Although patients thought to have coronary spasm were not included in this study, no nitrate was administered to patients before coronary angiography. Possible coronary artery spam that was overlooked might influence measurement.

In our study, we demonstrated that there were differences in coronary dimensions in patients with AR or AS despite similar LV mass. Diameter of LMCA was greater in AR group than in AS group, but mean diameter of LAD, LCx, and RCA were found to be similar in those 2 groups. Further studies investigating factors that affect coronary dimensions would be beneficial in order to demonstrate pathophysiological mechanisms and differences in AR and AS groups.

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