Research Report

Journal of INTERNATIONAL MEDICAL RESEARCH

Journal of International Medical Research
2016, Vol. 44(6) 1349–1358

DOI: 10.1177/0300060516666623
imr.sagepub.com
SAGE

Association between ascending aortic diameter and coronary artery dilation: a demographic data analysis

Ali Can Hatemi[1,2], Aybala Tongut[2], Zeki Özyedek[3], İsmail Çerezci[3], İlhan Özgöl[1] and Hande Perk Gürün[4]

Abstract

Objective: Coronary artery dilations (CDs), a subgroup of coronary artery anomalies (CAAs), are relatively rare but important cardiac pathologies. They are considered to be linked to coronary atherosclerosis in most cases.

Methods: The demographic data, multi-slice computed tomographic coronary angiography data, coronary calcium score, and ascending aortic diameter (AAD) of 1538 patients were reviewed. In total, 197 (12.8%) patients (166 men, 31 women; age 15 – 84 years; mean 55.78 ± 12.32 years) with CAAs were identified, and 81 (5.3%) patients (70 men, 11 women; age 27 – 80 years; mean 56.63 ± 12.06 years) had CDs. Multiple regression and correlation analyses were performed in all 1538 patients to predict the association between the AAD and the presence of CD and thus their correlation with atherosclerosis.

Results: The AAD was significantly larger in patients with than without CAAs and CDs. Male sex was significantly more prevalent in patients with CAAs and CDs. According to the multiple logistic regression model, male sex increased the risk of CD by 2.650 and the risk of CAA by 2.017, while hyperlipidaemia decreased the risk of CAA by 0.681. While a moderately weak correlation between the AAD and age was observed in patients with CDs, no correlation was found between the AAD and coronary calcium score.

Conclusion: Although the natural history and physiopathology of CDs is not yet fully understood, the present study shows an association between the AAD and the presence of CDs but a lack of association between atherosclerosis and CDs.

[1]Istanbul University, Institute for Cardiology, Department of Cardiovascular Surgery, Istanbul, Turkey
[2]Kartal Koşuyolu Heart Center, Pediatric Cardiovascular Surgery Clinic, Istanbul
[3]Macka Emar Advanced Medical Imaging Center, Istanbul, Turkey
[4]Marmara University, Marmara Faculty of Medicine, Department of Public Health, Istanbul, Turkey

Corresponding author:
Ali Can Hatemi, Istanbul University, Institute for Cardiology, Department of Cardiovascular Surgery, Haseki Cad. 29-31, Haseki, Fatih, Istanbul 34096, Turkey.
Email: alicanhatemi@gmail.com

Keywords
Coronary artery dilation, coronary ectasia, multi-slice computed tomographic coronary angiography

Date received: 7 March 2016; accepted: 8 August 2016

Introduction

Coronary artery dilations (CDs), including coronary ectasia and aneurysms, are a subgroup of coronary artery anomalies (CAAs) and one of the most neglected topics in cardiovascular medicine.[1,2] Although discovered incidentally, they can cause detrimental events such as sudden cardiac death.[2,3] CAAs were originally categorized as major, severe, important, or hemodynamically significant/insignificant.[4] These classifications primarily consisted of "unusual coronary origin and course" and "coronary fistulae," which might simultaneously be responsible for myocardial perfusion abnormalities.[5] A comprehensive classification of CAAs was recently defined and includes the following four subgroups: anomalies of coronary artery origin and distribution, anomalies of intrinsic coronary arterial anatomy, anomalies of coronary artery termination (coronary fistulae), and anomalous anastomotic vessels. This novel classification system covers all possible anatomic variations of the coronary arteries independent of their clinical and hemodynamic consequences.[4,6] CDs are thus classified as anomalies of intrinsic coronary arterial anatomy and have been reported in up to 5% of retrospective studies.[2,6,7,8] They can occur either as a diffuse dilation of the coronary arteries (>1.5 times the normal diameter) that involves ≥ 50% of the artery length, which is called "ectasia," or as a localized dilation in which the aneurysmal dilation of the largest coronary vessel by 1.5 times is seen in < 50% of the total vessel length (diameter > 1.5 times, width > length). Coronary artery disease is considered the underlying factor in most of these patients.[7] Some CDs have been described in association with inflammatory connective tissue disorders such as scleroderma or Kawasaki disease or with a history of cocaine abuse.[9,10,11,12] Very few studies have reported a higher incidence of CDs in patients with aneurysms of the abdominal aorta and its branches.[13,14]

This single-centre study was performed to investigate the association between the presence of CDs and the ascending aortic diameter (AAD).

Materials and methods

From January 2005 to December 2008, 2401 consecutive patients referred to a private institution (Maçka Emar, Istanbul, Turkey) for performance of multi-slice computed tomographic coronary angiography (MSCT-CA) were enrolled in the study. Patients who underwent open cardiac surgery or cardiac interventions (n = 186) and those lacking ascending aorta measurements (n = 726) were excluded. Demographic data and the AADs of 1538 patients were recorded, and the patients were divided into CAA and CD groups for further analysis.

Indications for the performance of MSCT-CA were suspicion of coronary artery disease such as atypical angina, typical angina with an inconclusive stress test, the presence of risk factors, and a high risk of major coronary events. All patients were scanned with a 64-CT scanner (GE LightSpeed VCT; GE Healthcare, Little Chalfont, UK). The angiographic scan parameters were: number of slices per rotation, 64; individual detector slice width, 0.625 mm; and 12.5-cm spatial coverage in 5 s at a gantry rotation speed of 330 ms.

Image reconstruction was performed at 10% increments through the R–R cardiac cycle. After image acquisition, the images were transferred to a dedicated workstation for analysis (AW Workstation; GE Healthcare). Patients with CAA were identified using retrospective electrocardiographic gating. The CT data set was analysed by two independent experienced readers blinded to the patients' clinical data. The original axial data set was examined before and after administration of contrast for calcium scoring of the coronary arteries. Quantitative coronary calcium scores (CCSs) were calculated according to the method described by Agatston *et al.*[15] Volume rendering images and curved multi-planar reconstructions were also used for analysis.

This study was approved by the No. 1 Clinical Research Ethics Committee of Istanbul at Istanbul University and conformed to the guiding principles of the Declaration of Helsinki. Informed consent was obtained from all patients before performance of MSCT-CA.

The type of CAA was classified based on the patients' demographic data according to the American Hospital Association scheme.[16] This scheme describes a coronary artery map used by the Bypass Angioplasty Revascularization Investigation. This comprehensive scheme was used to clearly define and classify every CAA. The CAAs were classified using a modified classification system defined by Angelini.[6] The types of CAAs were recorded, including their origin and proximal course, intrinsic coronary anatomy anomalies, and termination anomalies as seen by MSCT-CA. The AAD cut-off value was identified by sex.[17]

Statistical analysis

Statistical analyses were performed using SPSS version 17 (SPSS, Inc., Chicago, IL, USA). The patients' demographic data are presented using cross-tabulation. The chi-square test was used to compare proportions of data in different groups. The AADs and CCSs were investigated using the Kolmogorov–Smirnov test to determine whether they were normally distributed. Because neither variable was normally distributed, the Mann–Whitney *U*-test was used to compare the CAA and CD groups. The Kruskal–Wallis test was conducted to compare AADs among the subgroups of patients with CAAs. A P-value of < 0.05 was considered statistically significant. The Mann–Whitney *U*-test was performed to determine the significance of pair-wise differences using Bonferroni correction to adjust for multiple comparisons.

The cardiovascular risk factors and CCSs influencing CDs and CAAs were determined by multiple logistic regression analysis. Cardiovascular risk factors (age, sex, diabetes, hypertension, hyperlipidaemia, smoking, obesity, and a family history of ischemic heart disease) and CCSs were included as potential predictors, and the model's validity was found to be favourable. The Hosmer–Lemeshow goodness-of-fit statistic was used to assess model fit. While investigating the associations between non-normally distributed variables, the correlation coefficients and their significance were calculated using the Spearman test.

Results

The baseline characteristics of the cohort are presented in Table 1 (1131 men, 407 women; age 15 – 88 years; mean 55.65 ± 11.67 years). The mean AAD in the whole study population was 33.28 ± 4.43 mm. A total of 197 (12.8%) patients had CAAs (166 men, 31 women; age 15 – 84 years; mean 55.78 ± 12.32 years) (Table 2). In the demographic statistical analyses, male sex was shown to be extremely more prevalent in patients with CAAs ($P < 0.0001$). Hyperlipidemia and a family history of ischemic heart disease were found

significantly less frequently in patients with than without CAAs ($P = 0.02$ and $P = 0.01$, respectively) (Table 2). A total of 81 (5.3%) patients had CDs (70 men, 11 women; age 27 – 80 years; mean 56.63 ± 12.06 years). Most patients with CDs were male ($P = 0.007$), but there were no statistically significant differences in the frequencies of hypertension, diabetes mellitus, obesity, or smoking between patients with and without CDs (Table 3).

Table 1. Characteristics of the patient population.

Descriptive data	*n*	%
Male	1131	73.5
Female	407	26.5
Diabetes	332	21.6
Hypertension	759	49.3
Hyperlipidaemia*	855	55.6
Smoker	565	36.7
Family history	873	56.8
Obesity (BMI $\geq$ 30 kg/m^2)**	384	25.0
Coronary anomalies	197	12.8
Coronary artery dilatation	81	5.3

BMI, body mass index
*Dyslipidemia was defined as the presence of low-density lipoprotein 100 mg/dl.
**Calculated as weight in kilograms divided by square of height in meters (kg/m^2).

The AAD was larger in both the CAA and CD groups than in patients lacking CAAs and CDs ($P = 0.016$ and $P = 0.02$, respectively), while the CCSs remained statistically insignificant.

The multiple logistic regression model showed that male sex was significantly correlated with the presence of CDs ($P = 0.006$, OR 2.65, 95% CI 1.323, 5.309) and that male sex increased the risk of CD by 2.65 (95% CI 1.323, 5.309). Additionally, male sex ($P = 0.001$, OR 2.017, 95% CI 1.317, 3.089) and hyperlipidaemia ($P = 0.021$, OR 0.681, 95% CI 0.492, 0.944) were significantly correlated with CAAs. Multiple logistic regression showed that male sex increased the risk of CAAs by 2.017 (95% CI 1.317, 3.089), while hyperlipidaemia decreased the risk of CAAs by 0.681.

Twenty-seven patients (1.8%) had anomalies of origin and distribution, 162 (10.5%) had anomalies of intrinsic coronary arterial anatomy, and 8 (0.5%) had anomalies of coronary termination (coronary artery fistulae) (Table 4). When using the Kruskal–Wallis test to compare the AADs

Table 2. Cross-tabulation of patients with and without coronary artery anomalies.

	Coronary artery anomaly		
	+	−	
Male	166 (14.7)	965 (85.3)	$P < 0.01$***
Diabetes	40 (12.0)	292 (88.0)	$P = 0.64$
Hypertension	87 (11.5)	672 (88.5)	$P = 0.11$
Hyperlipidaemia*	95 (11.1)	760 (88.9)	$P = 0.02$***
Smoking	63 (11.2)	502 (88.8)	$P = 0.13$
Family history	96 (11.0)	777 (89.0)	$P = 0.01$***
Obesity (BMI $\geq$ 30 kg/m^2)**	45 (12.5)	336 (87.5)	$P = 0.83$

Data are presented as n (%) patients.
BMI, body mass index
*Dyslipidemia was defined as the presence of low-density lipoprotein 100 mg/dl
**Calculated as weight in kilograms divided by square of height in meters (kg/m^2).
***p value <0.05.

Table 3. Cross-tabulation of patients with and without coronary artery dilation.

	Coronary artery dilation		
	+	−	
Male	70 (6.2)	1061 (93.8)	$P < 0.01$***
Diabetes	17 (5.1)	315 (94.9)	$P = 1.00$
Hypertension	33 (4.3)	726 (95.7)	$P = 0.13$
Hyperlipidaemia*	43 (5.0)	812 (95.0)	$P = 0.64$
Smoking	29 (5.1)	536 (94.9)	$P = 0.90$
Family history	39 (4.5)	834 (95.5)	$P = 0.13$
Obesity (BMI $\geq$ 30 kg/m^2)**	21 (5.5)	363 (94.5)	$P = 0.89$

Data are presented as n (%) patients.
BMI, body mass index
*Dyslipidemia was defined as the presence of low-density lipoprotein 100 mg/dl.
**Calculated as weight in kilograms divided by square of height in meters (kg/m^2).
***p value <0.05.

Table 4. Ascending aortic diameters of patients with coronary artery anomalies (CAAs).

			Ascending aortic diameter (mm)	
CAA classification	n	%	mean $\pm$ SD	
Anomalies of origination and course	27	1.8	32.04 ± 4.743	$P = 0.025$*
Anomalies of intrinsic coronary arterial anatomy	162	10.5	34.27 ± 4.687	
Anomalies of coronary termination	8	0.5	36.50 ± 2.00	

*Kruskal-Wallis Test.
Data are presented as *n* (%) patients.
BMI, body mass index

among these three subgroups of CAAs, a statistically significant difference was found ($P = 0.025$); however, the Mann–Whitney *U*-test with Bonferroni correction revealed no significant pair-wise difference. Therefore, we further analysed the whole study population (1538 patients) for correlations between the AAD and age and between the AAD and CCS. Although moderate to weak, significant correlations were observed between the AAD and age (Spearman's correlation coefficient $r = 0.360$, $P < 0.001$) and between the AAD and CCS (Spearman's correlation coefficient $r = 0.314$, $P < 0.001$). Next, we tested correlations between the AAD and age and between the AAD and CCS in patients with CDs (Figure 1). While a moderate to weak correlation was observed between the AAD and age (Spearman's correlation coefficient $r = 0.372$, $P = 0.001$), no correlation was found between the AAD and CCS ($r = 0.229$, $P = 0.06$).

Discussion

The potential clinical consequences and significance of CDs are key concerns to cardiovascular specialists. MSCT-CA has become an ideal and even essential imaging tool for the evaluation of the coronary artery anatomy because of its unique

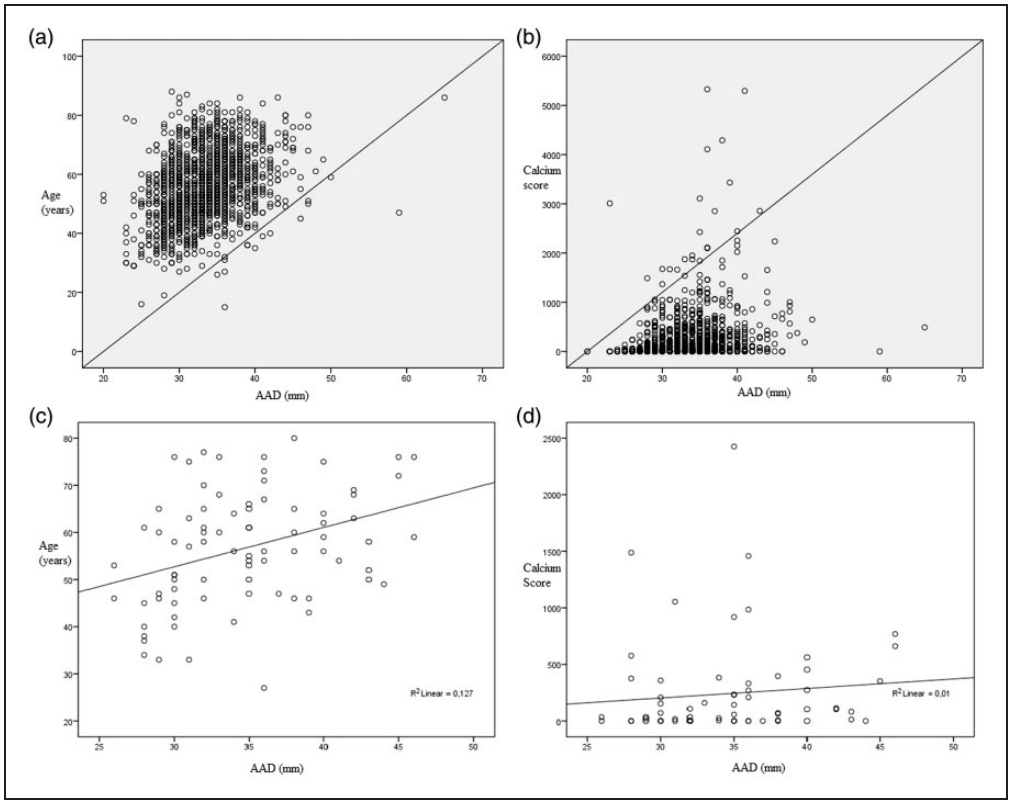

Figure 1. Correlation between the ascending aortic diameter (AAD) and age and between the AAD and calcium score. A, B: The scatter plots show significant (although moderate to weak) correlations between the AAD and age (Spearman's correlation coefficient $r = 0.360$, $P < 0.001$) and between the AAD calcium scores (Spearman's correlation coefficient $r = 0.314$, $P < 0.001$). C: A moderate to weak correlation was observed between the AAD and age (Spearman's correlation coefficient $r = 0.372$, $P = 0.001$). D: No correlation was observed between the AAD and calcium score ($r = 0.229$, $P = 0.06$). The calcium score was calculated with multi-slice computed tomography using coronary artery calcium quantification based on the method described by Agatston *et al.*[15]

capacity to quantitatively describe the vessels' origin, course, and wall thickening and its ability to characterize disease progression.[18,19]

Few studies have analysed atherosclerotic risk factors in patients with CAAs and CDs.[7,20,21] The relatively high frequencies of CAAs calculated by recently published MSCT-CA studies may be due to several factors, such as the evolution of the proficiency of imaging tools and our better understanding of the classification criteria for CAAs over time.[2,3,20] The diagnostic and inclusion criteria have evolved accordingly, leading to an expansion in the number of cases identified. Additionally, demographic analysis of the patient population in the current study revealed higher frequencies of CAAs and CDs especially among male patients ($P < 0.0001$), which is supported by logistic regression analyses in previous studies (Table 2, Table 3).[6,5]

Eid *et al.*[20] found that the presence of an anomalous vessel (such as a coronary artery

with an origin and distribution anomaly) does not appear to increase the chance of coronary artery disease. They found no association between comorbidities and significant stenotic disease in patients with normal and anomalous coronary arteries.[20,22] Although neither an isolated coronary origin or distribution anomalies reportedly increase the risk of coronary stenosis development, angiography studies are currently ongoing to conclusively determine the relationship between atherosclerotic disease progression and intrinsic CAAs such as ectasia and dilation.[8,23] However, it is well known that MSCT-CA is a potent modality in the detection of coronary artery disease because of its superior accuracy in the diagnosis and follow-up of these patients, especially in patients with CAAs, which emphasizes the importance of the present study.[18,24]

The aetiopathology of CDs is multifactorial and includes degenerative, congenital, inflammatory, and infectious factors.[9,10,11] Studies on CDs have mostly focused on atherosclerotic and infection models; thus, the role of atherosclerotic lesions in the formation CDs and the molecular mechanisms related to CD occurrence are still under debate.[20,25] Although some studies reported no difference between age and atherosclerotic risk factors in patients with CDs, atherosclerosis frequently does coexist with CDs of the same vessel.[7] The lower frequencies of hyperlipidaemia and a family history of ischemic heart disease in patients with CAAs in the present study ($P = 0.026$ and $P = 0.015$, respectively) can be explained by a bidirectional cause-and-effect relationship; atherosclerotic lesions may be either the cause or the effect. Logistic regression analysis determined only male sex among all coronary artery disease risk factors to be significantly correlated with CDs ($P = 0.06$). Based on this finding, classification of CDs in the coronary artery disease category or as one of its variants may be erroneous. Identification of hyperlipidaemia as a risk-reducing factor also supports this information.

In the Coronary Artery Surgery Study (CASS), CDs were detected in 4.9% of > 20 000 patients examined by conventional coronary angiography; however, no association between arteriosclerotic peripheral vascular lesions and CDs was observed.[7] Unlike studies that revealed no definitive association between CDs and abdominal aortic aneurysms (AAAs), Lamblin *et al.*[26] reported the first prospective demonstration of an association between CDs and AAAs.[8] The population of the CASS and other studies included patients with coronary artery disease without AAAs, which could explain the conflicting findings between those studies.

Kahraman *et al.*[14] reported that the diameter indices of the proximal portions of the right and left common iliac arteries of patients with CDs were significantly larger than those of the control group in their study on patients with isolated CD. However, the association between a large aortic diameter and CD was limited to the diameter of the common iliac artery; in other words, to the lower aortic branches. Papadakis *et al.*[27] examined the incidence of CD in patients who underwent planned surgical repair of an ascending aortic aneurysm. They found a strong association between CDs and ascending aortic aneurysms and concluded that the presence of a CD was five times more likely (26%) among patients with ascending aortic aneurysms. Recently, Balderston *et al.*[28] investigated the incidence and pathophysiology of CDs in patients with different types of aortic aneurysmal disease. They found that the proportion of coronary artery aneurysms adjacent to areas of atherosclerotic lesions (coronary artery disease-associated aneurysms) were much larger in patients with AAAs than in patients with ascending aortic aneurysms. AAD dilations and CDs in patients without coronary artery disease may share similar

pathophysiologic features, such as a non-atherosclerotic mechanism of arterial weakening. Although the AADs of patients with CDs were larger than those of patients lacking CDs, no significant difference was found between these groups in terms of risk factors for coronary artery disease (except male sex) (Table 3). Additionally, the lack of a significant difference in the CSSs between patients with and without CDs may indicate that the underlying physiopathologic pathway is quite different for these patients than for patients with atherosclerotic lesions.

Numerous studies have reported various factors that can affect the development of CDs, and these factors may be either directly or indirectly related to atherosclerotic processes. Some studies focused on inflammatory mechanisms and reported the involvement of markers such as the serum YKL-40 and C-reactive protein levels, while others found that overexpression of matrix metalloproteinases caused enzymatic degradation of the extracellular matrix of the tunica media, leading to expansive arterial remodeling.[30,31] A clear survey on CDs should include their location and size, the presence of any mural thrombosis, and the history of distal coronary artery disease.[19,32] These details may help to indicate whether the CDs are associated with coronary artery disease.

The present study showed a moderate to weak correlation between the AAD and age in the whole study group as well as in the subset of patients with CDs. Because the CCS reflects the atherosclerotic process, the lack of a correlation between the CCS and AAD in patients with CDs provides evidence of the disassociation between atherosclerosis and the AAD in patients with CDs.[29] Knowledge of the low CCS in patients with AAD enlargements and CDs enables us to conclude that the underlying pathology is unlikely to be atherosclerosis. In other words, although the underlying factors remain unclear, the present results may indicate that the CDs themselves as well as the AAD enlargements in patients with CDs might involve pathophysiologic processes that differ from those of atherosclerosis considering the lack of significance of the traditional risk factors in these patients.

To the best of our knowledge, this is the first large-scale study to show an association between the AAD and CDs. Although the present data show no association between atherosclerosis and CDs, further studies will help to clarify this issue. Such studies may include CAA prevalence studies, follow-up of the patients described in the present study, original research concentrating on pathophysiologic pathways, and experimental investigations of the molecular mechanisms of CD development.

The main limitation of this study is that it was not a prevalence study. The patients were a special population referred by cardiologists and included asymptomatic, high-risk patients; asymptomatic patients with risk factors; patients at high risk for major atherosclerotic events; and patients with cardiac symptoms. Long-term follow-up is not within the scope of this study.

Declaration of conflicting interest

The Authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

References

1. Basso C, Maron BJ, Corrado D, et al. Clinical profile of congenital coronary artery anomalies with origin from the wrong aortic sinus leading to sudden death in young competitive athletes. *J Am Coll Cardiol* 2000; 35: 1493–1501.

2. Angelini P, Velasco JA and Flamm S. Coronary anomalies - incidence, pathophysiology and clinical relevance. *Circulation* 2002; 105: 2449–2454.
3. Cademartiri F, La Grutta L, Malagò R, et al. Prevalence of anatomical variants and coronary anomalies in 543 consecutive patients studied with 64-slice CT coronary angiography. *EurRadiol* 2008; 18: 781–791.
4. Angelini P, Villason S, Chan AV, et al. Normal and anomalous coronary arteries in humans. In: Angelini P, Fairchild VD (eds) *Coronary artery anomalies: A comprehensive approach*. Philadelphia: Lippincott Williams & Wilkins, 1999, pp.27–150.
5. Greenberg MA, Fish BG and Spindola-Franco H. Congenital anomalies of the coronary arteries: classification and significance. *Radiol Clin North Am* 1989; 27: 1127–1146.
6. Angelini P. Coronary artery anomalies: an entity in search of an identity. *Circulation* 2007; 115: 1296–1305.
7. Swaye PS, Fisher LD, Litwin P, et al. Aneurysmal coronary artery disease. *Circulation* 1983; 67: 134–138.
8. Hartnell GG, Parnell BM and Pridie RB. Coronary artery ectasia: its prevalence and clinical significance in 4,993 patients. *Br Heart J* 1985; 54: 392–395.
9. Falsetti HL and Carroll RJ. Coronary artery aneurysm. A review of the literature with a report of 11 new cases. *Chest* 1976; 69: 630–636.
10. Tarek el-G, Yasser AE and Gheita T. Coronary angiographic findings in asymptomatic systemic sclerosis. *Clin Rheumatol* 2006; 25: 487–490.
11. Hiraishi S, Yashiro K, Oguchi K, et al. Clinical course of cardiovascular involvement in the mucocutaneous lymph node syndrome-relation between the clinical signs of carditis and development of coronary arterial aneurysm. *Am J Cardiol* 1981; 47: 323–330.
12. Satran A, Bart BA, Henry CR, et al. Increased prevalence of coronary artery aneurysms among cocaine users. *Circulation* 2005; 111: 2424–2429.
13. Stajduhar KC, Laird JR, Rogan KM, et al. Coronary arterial ectasia: increased prevalence in patients with abdominal aortic aneurysm as compared to occlusive atherosclerotic peripheral vascular disease. *Am Heart J* 1993; 125: 86–92.
14. Kahraman H, Ozaydin M, Varol E, et al. The diameters of the aorta and its major branches in patients with isolated coronary artery ectasia. *Tex Heart Inst J* 2006; 33: 463–468.
15. Agatston AS, Janowitz WR, Hildner FJ, et al. Quantification of coronary artery calcium using ultrafast computed tomography. *J Am Coll Cardiol* 1990; 15: 827–832.
16. Scanlon PJ, Faxon DP, Audet AM, et al. ACC/AHA guidelines for coronary angiography123: a report of the American college of cardiology/American heart association task force on practice guidelines (committee on coronary angiography) developed in collaboration with the society for cardiac angiography and interventions. *J Am CollCardiol* 1999; 33: 1756–1824.
17. Hager A, Kaemmerer H, Rapp-Bernhardt U, et al. Diameters of the thoracic aorta throughout life as measured with helical computed tomography. *J Thorac Cardiovasc Surg* 2002; 123: 1060–1066.
18. Budoff MJ, Achenbach S, Blumenthal RS, et al. Assessment of coronary artery disease by cardiac computed tomography: a scientific statement from the American heart association committee on cardiovascular imaging and intervention, council on cardiovascular radiology and intervention, and committee on cardiac imaging, council on clinical cardiology. *Circulation* 2006; 114: 1761–1791.
19. Angelini P and Monge J. Newer concepts regarding adults with coronary artery aneurysms: are they all Kawasaki? Does it make a difference? *Circulation* 2012; 125: 3076–3078.
20. Eid AH, Itani Z, Al-Tannir M, et al. Primary congenital anomalies of the coronary arteries and relation to atherosclerosis: an angiographic study in Lebanon. *J Cardiothorac Surg* 2009; 4: 58.
21. Aksu T, Uygur B and Durukan Koşar M. Coronary artery ectasia: its frequency and relationship with atherosclerotic risk factors in patients undergoing cardiac

catheterization. *Anadolu Kardiyol Derg* 2011; 11: 280–284. [in Turkish, English Abstract].
22. Topaz O, DeMarchena EJ, Perin E, et al. Anomalous coronary arteries: angiographic findings in 80 patients. *Int J Cardiol* 1992; 34: 129–138.
23. Tunick PA, Slater J, Kronzon I, et al. Discrete atherosclerotic coronary artery aneurysms: a study of 20 patients. *J Am Coll Cardiol* 1990; 15: 279–282.
24. Diaz-Zamudio M, Bacilio-Perez U, Herrera-Zarza MC, et al. Coronary artery aneurysms and ectasia: role of coronary CT angiography. *Radiographics* 2009; 29: 1939–1954.
25. Pahlavan PS and Niroomand F. Coronary artery aneurysm: a review. *Clin Cardiol* 2006; 29: 439–443.
26. Lamblin N, Gautier C, Le Tourneau T, et al. Prospective aortic screening in men with coronary aneurysms. *J Am Coll Cardiol* 2006; 47: 1227–1229.
27. Papadakis MC, Leontiadis E, Manginas A, et al. Frequency of coronary artery ectasia in patients undergoing surgery for ascending aortic aneurysms. *J Am Cardiol* 2004; 94: 1433–1435.
28. Balderston JR, Girl J, Kolansky DM, et al. Coronary artery aneurysms associated with ascending aortic aneurysms and abdominal aortic aneurysms: pathophysiologic implications. *Catheter Cardiovasc Interv* 2015; 85: 961–967.
29. Greenland P, LaBree L, Azen SP, et al. Coronary artery calcium score combined with Framingham score for risk prediction in asymptomatic individuals. *JAMA* 2004; 291: 210–215.
30. Antoniadis AP, Chatzizisis YS and Giannoglou GD. Pathogenetic mechanisms of coronary ectasia. *Int J Cardiol* 2008; 130: 335–343.
31. Erdoğan T, Kocaman SA, Cetin M, et al. Increased YKL-40 levels in patients with isolated coronary artery ectasia: an observational study. *Anadolu Kardiyol Derg* 2013; 13: 465–470.
32. Daniels LB, Tjajadi MS, Walford HH, et al. Prevalence of Kawasaki disease in young adults with suspected myocardial ischemia. *Circulation* 2012; 125: 2447–2453.