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

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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.

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

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. Although discovered incidentally, they can cause detrimental events such as sudden cardiac death. CAAs were originally categorized as major, severe, important, or hemodynamically significant/insignificant. These classifications primarily consisted of “unusual coronary origin and course” and “coronary fistulae,” which might simultaneously be responsible for myocardial perfusion abnormalities. 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. CDs are thus classified as anomalies of intrinsic coronary arterial anatomy and have been reported in up to 5% of retrospective studies. 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. 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. Very few studies have reported a higher incidence of CDs in patients with aneurysms of the abdominal aorta and its branches.

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.\textsuperscript{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.\textsuperscript{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 CAs were classified using a modified classification system defined by Angelini.\textsuperscript{6} The types of CAs 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.\textsuperscript{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 \textit{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 CAs. A \textit{p}-value of < 0.05 was considered statistically significant. The Mann–Whitney \textit{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 CAs 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 CAs (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 CAs (\textit{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).

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 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 ≥ 30 kg/m²)** | 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/m²).

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

| Coronary artery anomaly | + | - | \(P<0.01****\) |
|-------------------------|---|---|----------------|
| Male                    | 166 (14.7) | 965 (85.3) | \(P=0.64\) |
| Diabetes                | 40 (12.0) | 292 (88.0) | \(P=0.01****\) |
| 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 ≥ 30 kg/m²)** | 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/m²).
****\(p\) value <0.05.
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 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

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**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 ≥ 30 kg/m²)** | 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 \((\text{kg/m}^2)\).

***\(p\) value <0.05.

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

| CAA classification | \(n\) | % | mean ± SD | Ascending aortic diameter (mm) |
|-------------------|------|---|----------|--------------------------------|
| 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
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

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
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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{18,24}

The aetiopathology of CDs is multifactorial and includes degenerative, congenital, inflammatory, and infectious factors.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{7} Unlike studies that revealed no definitive association between CDs and abdominal aortic aneurysms (AAAs), Lamblin et al.\textsuperscript{26} reported the first prospective demonstration of an association between CDs and AAAs.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.

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