



Evaluation of Coronary Atherosclerosis in Patients with Coronary Artery Aneurysm With CAD-RADS Scoring System Using MDCT Angiography

Koroner Arter Anevrizmalı Hastalarda Koroner Aterosklerozun MDBT Anjiyografi Kullanılarak CAD-RADS Skorlama Sistemi ile Değerlendirilmesi

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Abstract

Aim: This article aims to investigate the degree and frequency of atherosclerosis using the "Coronary Artery Disease Reporting and Data System" (CAD-RADS) classification in patients who underwent coronary Multidetector Computed Tomography (MDCT) Angiography with a preliminary diagnosis of coronary artery disease and were found to have coronary artery aneurysm (CAA) and also to discuss the predisposing factors, prevalence, diagnostic criteria and complications in CAA with CT images.

Material and Methods: We retrospectively evaluated the examinations of 3694 patients who underwent coronary MDCT angiography. We evaluated a total of 69 patients including 23 patients with CAA and 46 patients without CAA, in terms of atherosclerotic involvement using the CAD-RADS classification system and compared the findings.

Results: CAA was most frequently found in the right coronary artery (RCA), followed by the left anterior descending artery (LAD), left circumflex (LCX), left main coronary artery (LMCA), and posterolateral branch (PLB). In patients with CAA, the most frequently atherosclerosis observed vessels were LAD, LCX, RCA, and LMCA, respectively, while LAD, RCA, LCX, LMCA, PLD, and PDA were detected in patients without an aneurysm. No atherosclerosis was detected in 5 patients (21.7 %) with CAA and 15 patients (32.6 %) without CAA ($p>0.05$).

Conclusion: The number of atherosclerotic vessels and the degree of stenosis calculated using the CAD-RADS scoring in patients with CAA are similar to patients without an aneurysm. The fact that atherosclerosis is an important factor in the etiology of aneurysms may explain this situation.

Keywords: Coronary artery aneurysm, coronary atherosclerosis, MDCT angiography

Öz

Amaç: Bu makale koroner arter hastalığı ön tanısı ile koroner Multidedektör Bilgisayarlı Tomografi (MDBT) Anjiyografi yapılan ve koroner arter anevrizması (KAA) saptanan hastalarda, "Coronary Artery Disease Reporting And Data System" (CAD-RADS) sınıflaması ile aterosklerozun derecesini ve sıklığını araştırmak ve ayrıca KAA'da predispozan faktörleri, yaygınlığını, tanı kriterlerini ve komplikasyonlarını BT görünümleri eşliğinde ele almaktır.

Materyal Metot: Koroner MDBT Anjiyografi çekilen 3694 hastanın tetkiklerini retrospektif olarak inceledik. KAA tespit edilen 23 hasta ile birlikte KAA bulunmayan 46 hasta dahil toplam 69 hastayı aterosklerotik tutulum açısından CAD-RADS sınıflama sistemini kullanarak değerlendirdik ve bulguları karşılaştırdık.

Bulgular: KAA en sık sağ koroner arterde (RCA) bulunurken, bunu sol anterior desendan arter (LAD), sol sirkümfleks (LCX), sol ana koroner arter (LMCA) ve posterolateral dal (PLD) izledi. KAA olan hastalarda en sık ateroskleroz görülen damarlar sırasıyla LAD, LCX, RCA ve LMCA iken anevrizması olmayan hastalarda LAD, RCA, LCX, LMCA, PLD ve PDA olarak tespit edildi. KAA olan 5 hastada (21.7 %) ve olmayan 15 hastada (32.6 %) ateroskleroz saptanmadı ($p>0.05$).

Sonuç: KAA'lı hastalarda CAD-RADS skorlaması kullanılarak hesaplanan aterosklerotik damar sayısı ve darlık derecesi, anevrizması olmayan hastalar ile benzerdir.

Anahtar Kelimeler: Koroner arter anevrizması, koroner ateroskleroz, MDBT anjiyografi

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INTRODUCTION

There are many publications in the literature about coronary artery aneurysm (CAA) which was first described by Bougon in 1812 (1). Coronary artery aneurysmal dilatation has been defined in two ways, coronary artery ectasia and CAA, and a male-female ratio of 3:1 has been reported with a prevalence rate of 1.2 - 4.9% in their CAA, however, there are also studies reporting this ratio between 0.3% and 5.3% (2-4). CAA is defined as abnormal focal dilatation of the coronary artery exceeding 1.5 times the adjacent normal segment. On the other hand, coronary artery ectasia is used to describe lesions of similar but widespread length involving $\geq 50\%$ of the coronary artery (5).

The most frequently observed vessels are right coronary artery (RCA), left anterior descending artery (LAD), left circumflex (LCX), and left main coronary artery (LMCA), respectively (6). CAA involves more in proximal segments of coronary arteries (7). CAA can be of the saccular or fusiform type, and fusiform types are usually found in LAD (8). When the aneurysm is more than four times the diameter of the adjacent normal segment, it is called a giant coronary aneurysm (9). Both RCA involvement and 3-vessel involvement are more common in men than women in CAAs. The development of CAA is closely related to gender, genetic syndromes (Loeys-Diet and Ehlers-Danlos syndrome) and connective tissue diseases such as scleroderma, and systemic vascular diseases such as Kawasaki disease, Takayasu arteritis, and it is known that the most common etiology of CAA is atherosclerosis (50%), but Kawasaki disease is the most common cause worldwide. Additionally, pseudoaneurysms may be seen due to iatrogenic or traumatic causes (cardiac catheterization, surgery) or infectious processes (10,11).

CAA is detected incidentally by catheter coronary angiography (CCA) or MDCT angiography. Chest pain, acute myocardial infarction, congestive heart failure, and sudden cardiac death can be seen in patients due to complications such as rupture, compression on cardiopulmonary structures, thrombus formation, distal embolization, and arteriovenous fistula development. Acute presentations can be seen especially in cases of infectious causes and Kawasaki disease. It is known that atherosclerosis, proteolytic imbalance, and inflammatory reactions play a role in aneurysm development. The ideal treatment for CAA has not been defined yet, but computed tomography angiography, a non-invasive method, is recommended for long-term follow-up (12,13). Coronary magnetic resonance angiography (MRA) can also be used for diagnosis and long-term follow-up, but MDCT angiography is considered to be superior (14).

Coronary MDCT angiography is an important diagnostic method used to evaluate coronary atherosclerosis in patients with known low or moderate risk of coronary artery disease. The diagnostic performance of MDCT angiography is high, especially in acute or stable chest pain. Guidelines have been established by the Society for Cardiovascular Computed Tomography (SCCT) to exclude

coronary atherosclerotic disease and categorize luminal stenosis in the interpretation and reporting of MDCT angiography. However, it was observed that there were major differences in reporting among practitioners due to the lack of standardization (15). In 2016, radiology and cardiology associations introduced the CAD-RADS system to ensure standardization in reporting, improve quality, and produce consistent data for research and education. CAD-RADS provides a consistent assessment of stenosis as well as effective communication, management recommendations, and risk estimation. Comprehensive data collection and better training and research have been made possible thanks to a standardized reporting system. The inclusion of CAD-RADS in MDCT angiography reports may reduce the cost and length of hospital stay by reducing the number of unnecessary invasive CCAs (15). CAD-RADS has been shown to accurately predict major cardiovascular events, particularly angina, myocardial infarction, or death. CAD-RADS has also been shown to correlate with the degree of stenosis measured by invasive CCA with high sensitivity (100%), specificity (96.8%-98.7%), and accuracy (98.3%-99.3%) (17). Besides, CAD-RADS has limitations such as misclassification of observed findings and misinterpretation of the last category (15).

This article aims to review coronary artery aneurysms with predisposing factors and categorize accompanying atherosclerotic changes using the CAD-RADS classification.

MATERIAL AND METHOD

This retrospective study protocol was approved by our institutional review board. This single-center study is based on coronary MDCT angiography data from the Department of Radiology database. The imaging data of all patients (3694 patients) who underwent coronary MDCT angiography between December 2018 and January 2022 for the investigation of coronary artery disease in our institution were evaluated by a radiology specialist. After excluding patients with poor diagnostic image quality due to high pulse rate or arrhythmia and studies performed for reasons other than coronary artery evaluation (calcium scoring, valve evaluation, surgical planning, investigation, cardiac masses, evaluation of pulmonary/cardiac veins, and non-coronary congenital heart disease), 23 patients with CAA (abnormal focal dilatation of the coronary artery exceeding 1.5 times the adjacent normal segment) were included in the study. A control group was formed with 46 randomly selected patients without CAA but with similar demographic characteristics. Aneurysm types were analyzed with coronary arteries with aneurysm and involved segments. In CAD-RADS, every segment with a diameter greater than 1.5 mm is evaluated, but the segment with the highest degree of stenosis is considered for classification. The CAD-RADS system has two sections, categories, and modifiers (16). With the CAD-RADS classification, each coronary segment is evaluated in detail and the CAD-RADS category is decided by analyzing the severity of the stenosis, plaque morphology, stents, and coronary artery

bypass grafts. In the CAD-RADS classification, there are 6 categories ranging from 0 (no atherosclerotic disease in any coronary artery) to 5 (total occlusion of at least one vessel), and the highest stenotic lesion is taken into account. There are four modifiers in the CAD-RADS classification. These; non-diagnostic image quality (N), stents (S), bypass grafts (G), and high-risk and sensitive plaques (V) (Table 1). All patients categorized with the CAD-RADS classification were reassessed for atherosclerotic involvement and degree of coronary obstruction, plaque morphology, type, presence, and patency of bypass grafts and stents. Both groups were compared in terms of the severity and prevalence of coronary atherosclerotic involvement using the CAD-RADS classification.

MDCT scanning protocol

All coronary MDCT angiography examinations were performed using a 128-detector, 160-slice computed tomography (Prime Aquilion, Toshiba Medical Systems, Otawara, Japan). 70-100 ml iodinated contrast material was injected into the left antecubital vein at a rate of 4 ml/s with an automatic injector system. MDCT scan was performed with bolus tracking technique from heart apex to baseline after the start of contrast agent injection. All coronary MDCT angiography studies were performed in the craniocaudal supine position within a single inhalation period. Examinations were performed using prospective or retrospective modulation accompanied by Electrocardiography (ECG). In retrospective modulation, all phases from 0% to 90% were evaluated by reconstructing the R-R interval at 10% intervals.

Coronary MDCT angiography imaging protocol for coronary artery disease: Section thickness: 0.5 mm, section spacing: 0.25 mm, rotation time: 400 ms, 100 kVp, and 300-400 mAs. Axial MDCT sections were transferred to the workstation and examined using 3D volume rendering and maximum intensity projection (MIP) as well as 2D multi-plane reconstructions (MPR). A special cardiac analysis program (Terarecon-Aquarius Workstation Intuition Edition ver.4.4.7.1021.7056) was used for vessel segmentation.

Ethics committee approval was obtained with the number 2022/36 and the study was done in accordance with the Helsinki Declaration.

Statistical Analyses

Conformity of continuous variables to normal distribution was examined by the Kolmogorov-Smirnov test. Since all continuous variables demonstrated normal distribution, descriptive statistics were shown as mean \pm standard deviation, and independent samples were analyzed by t-test for comparisons according to groups. Descriptive statistics of categorical variables were shown as numbers (%), and chi-square tests were used in the analysis of crosstabs. $P < 0.05$ values were considered statistically significant.

RESULTS

Among all patients participating in the study

There were 69 patients, 51 male (73.9%) and 18 female (26.1%). The mean age of female patients was 63.39 ± 9.69 years, and the mean age of male patients was 59.41 ± 10.02 . There was right dominance in 61 patients, left dominance in 5 patients, and codominance in 3 patients. There was a family history in 31 patients (44.9%), a history of smoking in 31 patients (44.9%), a history of diabetes in 18 patients (26.1%), and a history of hypertension in 40 patients (58%). None of the patients had genetic syndromes, connective tissue diseases, and systemic vascular diseases such as Kawasaki disease seen in the etiology of CAA.

Among patients with CAA

There were 17 male (73.9%) and 6 female (26.1%) patients. The mean age of male patients was 58.24 ± 9.76 years, and the mean age of female patients was 68.67 ± 10.23 . 22 patients had right dominance and one patient had codominance. No left dominance was observed. Fusiform aneurysm was observed in 22 patients while saccular aneurysm was observed in LMCA in only one patient (Figure 1). Single vessel aneurysms in 12 patients, two vessel aneurysms in 5 patients, three vessel aneurysms in 4 patients, and four vessel aneurysms including LMCA in one patient were observed. Two aneurysms were thrombosed (Figure 2). RCA aneurysm in 14 patients (60.9%), LAD aneurysm in 12 patients (52.2%), LCX aneurysm in 8 patients (34.8%), LMCA aneurysm in 3 patients (13%), PLD aneurysm in one patient (4.3%), and PDA aneurysm in one patient (4.3%) were observed ($p < 0.001$) (Table 2).

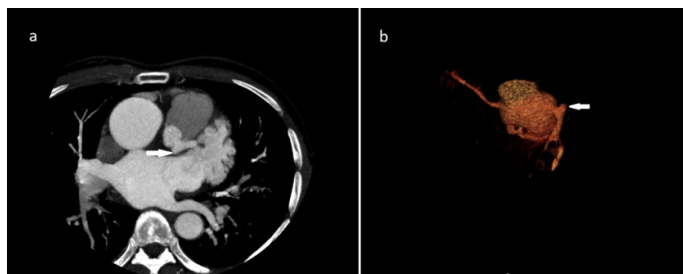


Figure 1. Axial maximum intensity projection (MIP) (a) and volume rendering (VR) (b) images on MDCT showing LMCA aneurysm (Arrows)

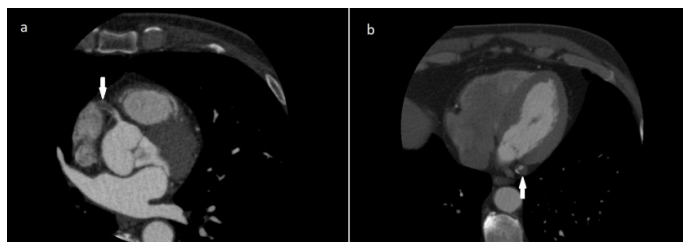


Figure 2. Axial MDCT images show thrombosed aneurysm in RCA (a) and LCX (b) (Arrows)



Figure 3. Axial (a) PDA aneurysm on MDCT images. Sagittal MIP (b) and MPR (c) images show PDA fistulized into the left atrium



Figure 4. Axial MIP images on MDCT (a-b) and VR images (c) show RCA originating from the left coronary sinus, and fusiform aneurysm in LCX and LAD

The patient with a PDA aneurysm had a left atrial fistula (Figure 3). In addition, one patient had RCA originating from the left coronary sinus and a fusiform aneurysm were

present in the LAD and LCX proximal segment (Figure 4). In one patient, both LAD and LCX were completely occluded and there was LIMA-LAD and aorta-LCX saphenous vein graft in patent appearance. Atherosclerotic involvement was in single-vessel in 8 patients (44.4%), in two-vessel in 6 patients (33.3%), and in three vessel in 4 patients (22.2%). No plaque was observed in 5 patients. Five patients (21.7%) were categorized as CAD-RADS 0, nine patients (39.1%) as CAD-RADS 1, three patients (13%) as CAD-RADS 2, two patients (8.6%) as CAD-RADS 3, one patient (4.3%) as CAD-RADS 4 and three patients (13%) as CAD-RADS 5. Calcific plaques were observed in 15 patients (65.2%), fibrous plaques in one patient (4.3%), and mixed plaques in two patients (8.6%). There was a vulnerable plaque in 13 patients (56.5%). Three patients (13.04%) had patent appearance stent, two patients (8.7%) had bypass graft and one had thrombosis. There was a family history in 9 patients (39.1%), smoking in 10 patients (43.5%), diabetes in 6 patients (26.1%), and hypertension in 13 patients (56.5%). Two patients had at least two of these risk factors, and 8 patients had at least three of them. In three patients, none of these risk factors were present. The mean cholesterol value was 214.70 ± 38.03 .

Table 1. CAD-RADS scoring system

CAD-RADS Category	Interpretation	Degree of Maximal Coronary Stenosis	Further Cardiac Workup	Management
0	Absence of CAD	0%, no plaque or stenosis	None	Consider nonatherosclerotic causes of chest pain
1	Minimal CAD	1%-24%, minimal stenosis or plaque without stenosis	None	Consider nonatherosclerotic causes of chest pain Preventive therapy and risk modification
2	Mild CAD	25%-49%	None	Consider nonatherosclerotic causes of chest pain Preventive therapy and risk modification, especially for plaque in multiple segments
3	Moderate stenosis	50%-69%	Functional assessment	Consider symptoms-guided anti-ischemic and preventive pharmacotherapy and risk factor modification per-guideline-directed care
4A	Severe stenosis	One or two vessels, 70%-99%	ICA or functional assessment	Consider symptoms-guided anti-ischemic and preventive pharmacotherapy and risk factor modification per-guideline-directed care
4B	Severe stenosis	Left main artery >50% or three vessels \geq 70%	ICA is recommended	Other treatments including revascularization should be considered per guideline-directed care
5	Total occlusion	100%	ICA and/or viability assessment	Same as for CAD-RADS 4A and 4B
N	Obstructive CAD cannot be excluded	Nondiagnostic	Additional or alternate evaluation	Additional or alternate evaluation

Note. -Modifiers include N (nonevaluable segment), S (coronary stent), G (coronary bypass graft) and V (vulnerable plaque). ICA = invasive coronary angiography

Among the patients in the control group

There were 34 male (73.9%) and 12 female (26.1%) patients. The mean age of male patients was 60 ± 10.25 , and the mean age of female patients was 60.75 ± 8.65 . There was right dominance in 44 patients, left dominance in 5 patients, and codominance in 2 patients. In one patient, the LAD was occluded and there was a patent appearance LIMA-LAD saphenous vein graft. In one patient, both LAD and RCA were in total occluded appearance, and there was a patent appearance LIMA-LAD saphenous vein graft and an occluded aortic-RCA saphenous vein graft. Atherosclerotic involvement was in single-vessel in 12 patients (26.1%), two-vessels in 7 patients (15.2%), three vessel in 8 patients (17.4%), four vessel in 3 patients (6.5%), and five vessel in one patient (2.2%). No plaque was observed in 15 patients. 15 patients (32.6%) were categorized as CAD-RADS 0, 10 patients (21.7%) as CAD-RADS 1, 5 patients (10.9%) as CAD-RADS 2, 9 patients (19.6%) as CAD-RADS 3, 5 patients (10.9%) as CAD-RADS 4, and 2 patients (4.3%) as CAD-RADS 5. Calcific plaques were observed in 29 patients (63%) and fibrous plaques in 2 patients (4.3%). The vulnerable plaque was present in 28 patients (60.9%). 4 patients (8.7%) had patent appearance stent, 2 patients (4.3%) had bypass graft and one had thrombosis. There was a family history in 22 patients (47.8%), smoking in 21 patients (45.7%), diabetes in 12 patients (26.1%), and hypertension in 27 patients (58.7%). At least two of these risk factors were present in 10 patients, at least three in 10 patients, and at least four in 6 patients. 13 patients had none of these risk factors. The mean cholesterol value was 208.63 ± 43.61 .

When the groups with and without aneurysms were compared in terms of the specified risk factors, no significant difference was observed ($p > 0.05$).

In order of frequency, the vessels in which coronary atherosclerosis is most frequently seen are; In the group with CAA, 15 vessels (65.2%) were observed in the LAD, 9 vessels (39.1%) in the LCX, 6 vessels (26.1%) in the RCA, 2 vessels (8.7%) in the LMCA, while in the control group, 27 vessels (58.7%) in LAD, 15 vessels (32.6%) in RCA, 14 vessels (30.4%) in LCX, 8 vessels (17.4%) in LMCA, 3 vessels (6.5%) in PLD and 1 vessel (2.2%) in PDA ($p < 0.001$) (Table 3). No plaque was observed in PLD and PDA in the group with CAA.

Table 2. Vessels with the most frequent coronary artery aneurysm

Aneurism type	n (%)
RCA	14 (60.9)
LAD	12 (52.2)
LCX	8 (34.8)
LMCA	3 (13)
PLD	1 (4.3)
PDA	1 (4.3)

Table 3. Vessels where coronary atherosclerosis is most frequent in patients with and without CAA

	CAA patients n (%)	Control group n (%)
LAD	15 (65.2)	27 (58.7)
RCA	6 (26.1)	15 (32.6)
LCX	9 (39.1)	14 (30.4)
LMCA	2 (8.7)	8 (17.4)
PLD	0 (0)	3 (6.5)
PDA	0 (0)	1 (2.2)

The incidence of coronary atherosclerosis in at least one vessel was 78.3% in patients with CAA and 67.4% in the control group, and no significant difference was observed between the two ($p > 0.05$).

DISCUSSION

The pathophysiological mechanisms of CAA are not well understood, but atherosclerosis in adults and Kawasaki disease in children are the main etiologies (12). Other common causes of CAA include mycotic and infectious septic embolism, Marfan syndrome, and connective tissue diseases. Other vasculitic disorders such as Takayasu's arthritis, polyarteritis nodosa, systemic lupus erythematosus, and rheumatoid arthritis may lead to CAA (12). Kawasaki disease is a vasculitis that causes symmetric wall thickening, stenosis, and aneurysmal dilatation by affecting medium and large vessels such as the aorta and its branches. In Takayasu's disease which classically affects the aorta and pulmonary arteries, 10-30% of patients have coronary artery involvement. Patients may have osteal stenosis, arthritis, or aneurysm. Pulmonary artery involvement and pulmonary hypertension can be seen at a rate of 15-17%. Angina, myocardial infarction, heart failure, and sudden death may develop in patients due to ischemic cardiac involvement (13).

Atherosclerosis is a chronic progressive transmural inflammatory disease affecting different vascular layers. Stenotic coronary atherosclerosis and CAA commonly coexist which have several histological patterns in common, such as hyalinization, lipid deposition, focal calcification, and fibrosis (12). CAA is thought to be a result of atherosclerosis since patients with and without CAAs are similar in terms of both risk factors and clinical manifestations of atherosclerosis (13). CAA may also result from apical hypertrophic cardiomyopathy secondary to the high-tensile wall. Aneurysm development may occur as a result of iatrogenic injury of the blood vessel in post-percutaneous coronary interventions such as stent placement, atherectomy, and balloon angioplasty (14). A low incidence of CAA has been detected in diabetic patients. Besides, it has been reported in the literature that the biggest risk factor for CAA is hypertension, and smoking habit is more common in patients with CAA compared to patients with coronary artery disease (19).

However, in our study, we did not observe a statistically significant difference between the group with CAA and the control group in terms of risk factors such as family history, diabetes history, hypertension, and smoking ($p>0.05$).

Most patients with CAA are asymptomatic, but clinical signs may occur due to the development of atherosclerotic coronary artery disease or complications (12). Complications include the clinical sequelae of myocardial infarction and thrombosis or rupture of an aneurysm potentially resulting in sudden death. Larger aneurysms (>8 mm) have a higher risk of complications, including myocardial infarction, stenosis, and thrombosis. A study on CCA showed that 3-vessel coronary artery disease and a history of myocardial infarction were more common in male patients (13). Distal embolization, coronary spasm, massive expansion of the CAA, and compression of the adjacent structure are the other most frequent complications. CAA rupture is rare but may result in cardiac tamponade and sudden death. The CAA may expand excessively, compressing adjacent cardiopulmonary structures such as the right atrium, right ventricular wall, pulmonary artery, and tricuspid valve (12). Other common clinical manifestations in patients with CAA include heart failure and syncope (20). If associated cardiovascular risk factors such as hypertension and diabetes are controlled, the progression of the disease can be reduced (14).

It has been stated that conventional coronary angiography (CCA) is an invasive gold standard tool to evaluate the anatomical features of CAA, and coronary MDCT angiography is the preferred alternative noninvasive technique for follow-up (21). However, although CCA can show the location, size, and shape of the CAA, it can be misleading about the size of the CAA when a thrombus forms in the lumen of the aneurysm (9). MDCT angiography or cardiac magnetic resonance angiography (MRA) can play an important role in the diagnosis of CAAs as they are fast, non-invasive, and reliable (22). MDCT angiography is capable of detecting coronary plaque location, severity, and plaque characterization. The prognostic power of MDCT angiography has been demonstrated in high-risk patients (23). MDCT angiography has been shown to be highly sensitive and specific for CAAs in Kawasaki patients. For significant coronary artery stenosis, the sensitivity was 87.5% and the specificity was 92.5% (24). In another study, MDCT was found to be more sensitive than 2D echocardiography in detecting fusiform and distally located aneurysms (25). The sensitivity of MDCT angiography for the detection of CAA and related complications is considerably high and has proven its value in the evaluation of coronary artery diameter in adults and patients with Kawasaki disease compared to CCA (12). Since radiation exposure causes an increase in the lifetime cancer risk, especially in children, the absence of radiation is the main advantage of cardiac MRA over CCA and MDCT angiography in the diagnosis of CAA (26). Finally, MDCT angiography is the imaging method of choice for long-term monitoring of CAA due to providing accurate data on coronary anatomy, calcification, lumen diameter,

thrombus, and aneurysmal features (12).

The use of CAD-RADS provides standardization by classifying the severity of coronary artery disease and makes MDCT angiography more valuable by including management recommendations. It has been reported that the predictive value of CAD-RADS is similar to both Duke's index and traditional methods and can be used as a prognostic determinant in high-risk asymptomatic coronary artery disease (27). Nam et al. showed that CAD-RADS provides prognostic value for major cardiovascular events and provides better risk differentiation compared to coronary artery calcium score alone (28). The additive prognostic value of the CAD-RADS scoring system has been shown to be above and beyond traditional methods (29). Assigning appropriate CAD-RADS category is dependent on accurate measurement of luminal stenosis on coronary MDCT angiography. Although coronary MDCT angiography is a robust modality for the evaluation of coronary arteries, artifacts due to densely calcified plaques often lead to an overestimation of luminal stenosis, thus assigning a higher category. Similarly, the spatial resolution of coronary MDCT angiography does not allow to distinguish between near-total (close to 99%) and total (100%) occlusion of coronary arteries (15). In addition, coronary artery anomalies, coronary artery dissection, coronary aneurysms or pseudoaneurysm are not categorized in the CAD-RADS system (18).

It has been reported that CAA is seen in LAD, RCA, LCX in order of frequency, but there are also studies showing that the most common localization is in RCA (31,32). It has been reported that CAA is found frequently in proximal-mid-RCA (68%), proximal LAD (60%), and LCX (50%). According to the same study, LMCA aneurysm (0.1%) is extremely rare (20). In our study, we detected CAA in RCA, LAD, LCX, LMCA, PLD, and PDA, in order of frequency. Studies have demonstrated that the order of frequency of vessels with CAA varies. This may be due to the definition of CAA ($<50\%$ involvement of a coronary artery aneurysm, $>50\%$ involvement ectasia) and genetic differences in the patient population used. In addition, the difference in CAA frequencies in different regions may also lead to this result when the effect of atherosclerosis on the development of CAA is considered. Although the number of patients with an aneurysm was limited in our study, the current results regarding the localization of CAA in the coronary arteries were consistent with the literature, but we think that it is important to conduct new studies with larger patient series.

Studies in the general population have shown that in asymptomatic individuals without aneurysm, vessels with coronary artery disease are LAD, RCA, LCX, and LMCA, in order of frequency (33). Similarly, we determined coronary atherosclerotic involvement in patients without CAA as LAD, RCA, LCX, LMCA, PLD, and PDA, in order of frequency.

Farrag et al. stated that only 16% of CAA patients do not have atherosclerotic lesions, MDCT angiography is an appropriate technique to determine and evaluate CAA

morphology, and they think that CAA is an advanced form of atherosclerosis (34). There are other publications in the literature investigating atherosclerotic involvement with MDCT angiography in patients with CAA (12-14). Besides, we also investigated the vessels in which atherosclerosis is most common in these patients and determined the coronary atherosclerotic involvement as LAD, LCX, RCA, and LMCA, in order of frequency. Additionally, we conducted our study using the CAD-RADS scoring system which provides standardization in the identification and reporting of coronary atherosclerotic diseases and is known to be reliable. We think that our study is remarkable in this respect and will contribute to the literature.

Finally, in our study, we found that the incidence of coronary atherosclerosis in at least one vessel was similar in patients with and without an aneurysm. We think that this may be due to the relatively small number of patients with CAA included in the study.

Limitations

The limitations of our study are the low number of cases.

CONCLUSION

Although patients with and without CAA have similar findings in terms of atherosclerotic involvement, the use of MDCT angiography and CAD-RADS scoring system, which has high diagnostic features, is extremely effective in preventing the development of serious and fatal complications, especially in patients with CAA who have obstructive coronary artery disease.

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Conflict of Interest: The authors declare that they have no competing interest.

Ethical approval: Ethics committee approval was obtained with the number 2022/36 and the study was done in accordance with the Helsinki Declaration.

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