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A New Chest Radiography Clue to Predict Saphenous Vein Graft Failure

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ABSTRACT

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©Copyright 2022 by Erciyes University Faculty of Medicine -Available online at www.erciyesmedj.com **Objective:** Saphenous vein graft failure (VGF) is a measure of the short- and long-term success of coronary artery bypass graft surgery (CABG). Aortic arch calcification (AAC) is a long-term finding of atherosclerosis in large vessels. The aim of this study was to evaluate the relationship between AAC and VGF.

Materials and Methods: Patients who underwent CABG surgery and subsequent coronary angiography in a single hospital between January 2010 and January 2021 were included in the study. The presence and stage of AAC was evaluated using preoperative chest X-rays. VGF was defined as \geq 75% stenosis and/or total occlusion in the saphenous vein graft. In addition, the effect of AAC on VGF was evaluated based on the time elapsed since the CABG procedure.

Results: Of the 594 patients who underwent CABG during the study period, 91 patients (mean age 63.6±10.0; 71 [78.0%] male) were included in the study. VGF was observed in 49 (53.8%) patients. AAC was found to be an independent predictor of VGF (odds ratio [OR]: 2.788, 95% confidence interval [CI]: 1.068–7.278). The results indicated no association between AAC and VGF in patients whose coronaries were screened within 1 year (OR: 1.143, 95% CI: 0.279–4.683), while there was a strong association between AAC and VGF in patients who were screened 1 year after the surgery (OR: 5.355, 95% CI: 1.618–17.720).

Conclusion: AAC evaluation may be a valuable diagnostic method to predict VGF after CABG, and particularly late VGF. **Keywords:** Aortic arch calcification, atherosclerosis, coronary angiography, coronary artery bypass grafting, vein graft

INTRODUCTION

failure

Coronary artery bypass graft surgery (CABG) remains an important treatment option for multi-vessel coronary artery disease, left main coronary lesions, and diabetic patients, despite the latest developments in percutaneous coronary interventions and current medical therapy. The most commonly used grafts in this type of treatment, left internal mammary artery (LIMA) and saphenous vein graft (SVG), as well as native coronary arteries, are all susceptible to the atherosclerotic process in the long-term (1, 2). The LIMA has been shown to have excellent long-term patency rates, but it is not generally suitable for use in multiple grafts due to limited length. Improvement in surgical venous graft harvesting techniques and the chemical solvents used afterwards, optimal medical treatment, and the use of arterial grafts have resulted in lower instances of graft failure (1–3). SVGs are also widely used due to their easy accessibility, ability to be harvested simultaneously with LIMA grafts, and viability for multiple bypasses. Yet, despite their widespread use, SVGs carry a high risk of early occlusion and long-term patency rates are not as high as arterial grafts (3). Previous studies have shown VGF rates of up to 20% in the first year after CABG and up to 60% after 10 years (1). Many influential factors have been identified (1–3). In addition to well-known aspects, such as graft or surgical technique, intimal hyperplasia, accelerated atherosclerosis, and inflammation have been proven to play an important role in the development of VGF (2, 3).

Aortic arch calcification (AAC) is one sign of atherosclerotic process. It can be easily identified using chest radiography, which is a simple and easily assessable test. Increased stiffness and calcification in the aorta have been associated with mortality and cardiovascular events (4). Many diseases, such as chronic renal failure (CRF), coronary artery disease (CAD), diabetes mellitus (DM), and stroke, have been found to be associated with AAC (5, 6). Advanced atherosclerosis of the aorta can lead to what is known as a porcelain aorta. Although it is not frequently observed in patients who undergo CABG, it is associated with increased operative risk (7, 8). Interventions without a heart-lung pump are recommended in these patients in order to have less contact with the aorta (9).

Knowledge of the relationship between VGF and AAC remains incomplete. The aim of this study was to investigate the effect of calcification of the aorta on VGF.

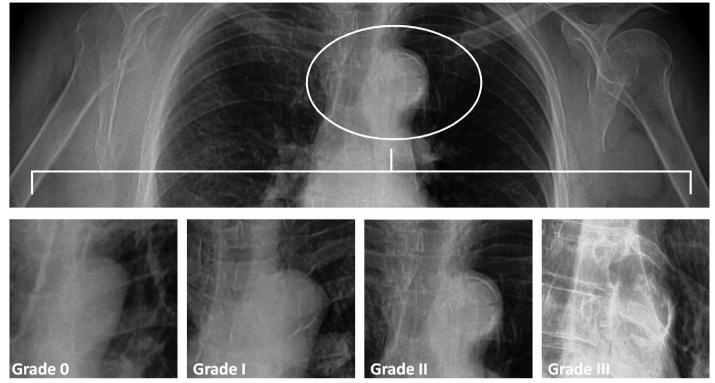


Figure 1. Aortic arch calcification grading

MATERIALS and METHODS

Ethical Considerations

Approval was granted by the Baskent University Institutional Review Board and Ethics Committee (no: KA 21/264) and the study was supported by Baskent University Research Fund.

Study Design

This retrospective study was carried out in the Baskent University Alanya Application and Research Center Department of Cardiology and Cardiovascular Surgery. A total of 594 patients who underwent CABG between January 2010 and January 2021 at the Baskent University Alanya Training and Research Center were evaluated retrospectively. Patients who were at least 18 years of age and presented for a CABG control visit were included in the study. AAC was evaluated using preoperative chest radiographs. Patients who underwent repeat CABG or additional cardiac surgery other than CABG (such as valve disease, congenital heart disease), those who did not have an SVG, and those whose chest X-ray imaging was not suitable for evaluation of AAC were excluded from the study. Patients with multiple coronary angiography and graft imaging results were considered in the initial screening.

Patients who presented for follow-up after a CABG procedure were divided into 2 groups based on the presence of 75% stenosis and/or total occlusion in any SVG. VGF was identified using the first postoperative coronary artery screening. The number of SVGs used in the operation, the use of a heart-lung pump, and the urgency of the operation were evaluated in all cases. The length of time between the operation and coronary angiography was also recorded and assessed. The Gensini score was calculated using pre- and post-CABG coronary angiography images to evaluate the extent and severity of coronary atherosclerosis (10).

The patients' clinical situation during CABG surgery and coronary angiography was defined as stable CAD or acute coronary syndrome (ACS) by a cardiologist according to the European Society of Cardiology guidelines (11–13). Patients with ACS were evaluated in 3 subgroups: unstable angina, non-ST elevation myocardial infarction, and ST elevation myocardial infarction (11–13). Surgical urgency was determined by the cardiology-cardiovascular surgery team. Individuals requiring emergency intervention underwent surgery without being discharged. Patients not requiring immediate surgery were released and readmitted for subsequent surgery.

Evaluation of Chest Radiography

Preoperative posterior-anterior chest radiography images for each patient were retrieved from the medical records and evaluated. All of the exposures had been performed at a patient focus distance of 150 cm with the patient standing under a constant tube voltage of 117 kV using an Aero DR X70 device (Konica Minolta Inc., Tokyo, Japan). Patients were divided into 2 groups according to the presence or absence of AAC, and 4 groups according to the degree of AAC (no calcification: stage 0; small spots of calcification: Stage; thickened calcification area in 1 or more zones: Stage 2; circular calcification in the aortic knob: Stage 3) (Fig. 1) (14). Chest X-rays were assessed for AAC by 2 independent researchers, who arrived at consistent evaluations (kappa: 0.677; p<0.001).

Cardiovascular Risk Factors

Demographic characteristics and laboratory results recorded during the operative period and the coronary angiography period were obtained from the hospital database. The glomerular filtration rate (GFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration formula (15). CRF was defined as patients with a GFR of <50 mL/minute and/or those in nephrology follow-up.

	Total	AAC grade 0	AAC grade ≥1 n=49	р
	n=91	n=42	n=49	
Age, years	63.6±10.0	60.4±11.3	66.3±8.0	0.035
Male, n (%)	71 (78.0%)	34 (81.0%)	37 (75.5%)	0.532
Smoking, n (%)	32 (35.2%)	15 (35.7%)	17 (34.7%)	0.919
Diabetes mellitus, n (%)	49 (53.8%)	22 (52.4%)	27 (55.1%)	0.795
Hypertension, n (%)	63 (69.2%)	27 (64.3%)	36 (73.5%)	0.344
Hyperlipidemia, n (%)	36 (39.6%)	15 (35.7%)	21 (42.9%)	0.487
CKD, n (%)	14 (15.4%)	4 (9.5%)	10 (20.4%)	0.151
Hematocrit, %	41.1±5.0	40.9±4.7	41.2±5.2	0.675
Glucose, mg/dL	115 (96–182)	118 (96–174)	114 (97–191)	0.447
GFR, mL/min/m2	83 (64–94)	91 (75–98)	76 (61–92)	0.042
LDL-C, mg/dL	123±37	123±38	122±36	0.933
HDL-C, mg/dL	39±10	39±11	38±9	0.745
LVEF, %	52.7±11.2	51.3±11.8	53.9±10.6	0.064
Preop clinic				0.175
SAP, n (%)	26 (28.6%)	9 (21.4%)	17 (34.7%)	
USAP, n (%)	14 (15.4%)	9 (21.4%)	5 (10.2%)	
NSTEMI, n (%)	42 (46.2%)	18 (42.9%)	24 (49.0%)	
STEMI, n (%)	9 (9.9%)	6 (14.3%)	3 (6.1%)	
Urgent, n (%)	63 (69.2%)	29 (69.0%)	34 (69.4%)	0.972
Number of grafts	3 (3–4)	3 (3–4)	4 (3–4)	0.103
Off-pump, n (%)	9 (9.9%)	6 (14.3%)	3 (6.1%)	0.293
Aspirin, n (%)	88 (96.7%)	40 (95.2%)	48 (98.0%)	0.593
Clopidogrel, n (%)	28 (30.8%)	14 (33.3%)	14 (28.6%)	0.624
Ticagrelor, n (%)	5 (5.5%)	3 (7.1%)	2 (4.1%)	659
DAPT, n (%)	33 (36.3%)	17 (40.5%)	16 (32.7%)	0.439
Statins				0.525
None, n (%)	36 (39.6%)	14 (33.3%)	22 (44.9%)	
Low dose, n (%)	32 (35.2%)	16 (38.1%)	16 (32.7%)	
High dose, n (%)	23 (25.3%)	12 (28.6%)	11 (22.4%)	
OAC, n (%)	7 (7.7%)	3 (7.1%)	4 (8.2%)	1.0
Time since CABG, months	21 (11–47)	21 (10–35)	22 (12–50)	0.696
Indication for coronary angiography				0.253
SAP, n (%)	40 (44.0%)	18 (42.9%)	22 (44.9%)	
USAP, n (%)	15 (16.5%)	7 (16.7%)	8 (16.3%)	
NSTEMI, n (%)	32 (35.2%)	17 (40.5%)	15 (0.6%)	
STEMI, n (%)	4 (4.4%)	0 (0.0%)	4 (8.2%)	
SVG lesion, n (%)	49 (53.8%)	16 (38.1%)	33 (67.3%)	0.005
Number of CSSs	3 (2–3)	3 (2–3)	3 (2–3)	0.579
Gensini score before CABG	76 (57–92)	71 (52–86)	78 (63–99)	0.163
Gensini score after CABG	112 (80–128)	108 (70–129)	116 (80–134)	0.372
Increase in Gensini score	28 (12–48)	28 (12–48)	24 (14–44)	0.883

Data are presented as percentage, mean±SD or median (interquartile range). AAC: Aortic arch calcification; CABG: Coronary artery bypass graft; CKD: Chronic kidney failure; CSS: Coronary artery with severe stenosis; DAPT: Dual antiplatelet therapy; GFR: Glomerular filtration rate; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; LVEF: Left ventricular ejection fraction; NSTEMI: Non-ST segment elevation myocardial infarction; OAC: Oral anticoagulant; SAP: Stable angina pectoris; STEMI: ST-segment elevation myocardial infarction; SVG: Saphenous vein graft; USAP: Unstable angina pectoris

Antiplatelet and cholesterol-lowering drugs were evaluated according to the postoperative prescriptions. Rosuvastatin 5-10 mg or atorvastatin 10-20 mg therapy was considered low-dose statin treatment, and rosuvastatin 20-40 mg or atorvastatin 40-80 mg treatment was considered high-dose.

Transthoracic Echocardiography

Echocardiographic examinations were performed according to American Echocardiographic Society guidelines (16). Transthoracic M-mode, 2-dimensional, color Doppler and pulse wave echocardiographic evaluation was performed using a GE Vivid E device with a 3.5-MHz transducer (GE Healthcare, Inc. Chicago, IL, USA). The Teichholz method was used to calculate left ventricular ejection fraction (LVEF).

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics for Windows, Version 24.0 software (IBM Corp., Armonk, NY, USA). The normality of the distribution was evaluated using the Kolmogorov-Smirnov or the Shapiro-Wilk test. Normally distributed continuous variables were expressed as the mean and SD, and non-normally distributed variables as the median and 25th-75th percentiles. Categorical variables were expressed as numbers and percentages. Continuous variables were compared between the 2 groups using the Student t-test and the Mann-Whitney U test. Categorical variables were compared using a chi-squared test or the Fisher's exact test. To determine the independent predictors of SGV lesions, simple logistic regression analysis was performed. Parameters that were significant in this analysis (p<0.1) were included in multiple logistic regression analysis. In addition, a receiver operating characteristic (ROC) curve was created to evaluate the diagnostic performance of presence of AAC on SVG lesions. A p value of <0.05 was considered statistically significant.

RESULTS

Of the 97 patients who underwent coronary artery screening after the CABG operation, 5 patients without SVG and 1 patient with a suboptimal chest radiography were excluded from the study.

In all, 91 patients (mean age 63.6 ± 10.0 ; 71 [78.0%]) male were included in the research. AAC was observed in 49 (53.8%) patients. Thirty-eight (41.7%) patients had Stage 1 AAC, 10 (11.0%) had grade 2 AAC, and 1 (1.1%) patient had grade 3 AAC. Patients in the AAC group were older than those in the group without ACC (p=0.035), and the GFR was lower (p=0.042).

An SVG lesion was seen more often in patients with AAC (p=0.005) (Table 1). In all, an SVG lesion was detected in 49 (53.8%) patients. Of these, AAC was not present in 16 (38.1%) and confirmed in 33 (67.3%). Among patients with an SVG lesion, 16 (17.6%) were determined to have stage 0 AAC, 25 (27.5%) stage 1 AAC, and 7 (7.7%) stage 2 AAC. One (1.1%) patient with stage 3 AAC also had an SVG lesion (Fig. 2). Other laboratory and demographic parameters were not statistically significantly different between the groups with and without AAC (Table 1).

A radial artery graft and an SVG were used in 6 patients. Lesions in both the SVG and the radial artery graft were found in 1 of these 6 patients. No lesion was detected in the radial artery grafts of the other patients.

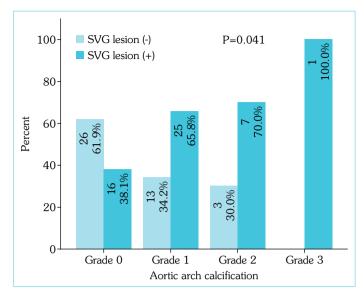


Figure 2. Relationship between aortic arch calcification stages and saphenous vein graft lesion

SVG: Saphenous vein graft

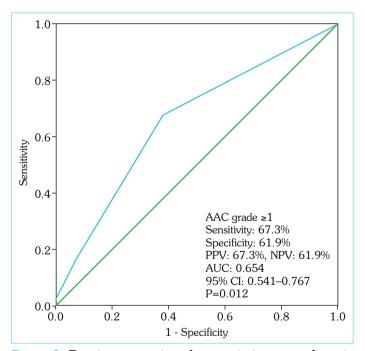


Figure 3. Receiver operating characteristic curve of aortic arch calcification and saphenous vein graft failure

AAC: Aortic arch calcification; AUC: Area under the curve; CI: Confidence interval; NPV: Negative predictive value; PPV: Positive predictive value

Simple logistic regression analysis revealed that the hematocrit, number of grafts, off-pump surgical technique, dual antiplatelet therapy (DAPT), Gensini score before CABG, and AAC variables (p=0.072, p=0.037, p=0.025, p=0.013, p=0.011, p=0.006, respectively) were significantly associated with VGF (p<0.1) and were included in the multiple logistic regression analysis (Table 2). The results of that analysis indicated that AAC and the Gensini score before CABG were independent predictors of VGF (odds ratio [OR]: 2.788, 95% confidence interval [CI]: 1.068–7.278; p=0.036 and OR: 1.019, 95% CI: 1.000–1.039; p=0.047, respectively) (Table

Table 2. Simple logistic regression analysis for saphenous vein graft lesion				
	Univariable analysis			
	OR (95% CI)	р		
Age	0.976 (0.936–1.018)	0.261		
Male	1.219 (0.451–3.291)	0.696		
Smoking	1.724 (0.716–4.153)	0.225		
Diabetes mellitus	0.781 (0.341-1.790)	0.559		
Hypertension	1.250 (0.512–3.050)	0.624		
Hyperlipidemia	1.121 (0.482–2.606)	0.791		
CKD	0.833 (0.267–2.605)	0.754		
Hematocrit	1.084 (0.993–1.184)	0.072		
Glucose	0.999 (0.995–1.003)	0.626		
Creatinine	1.137 (0.844–1.533)	0.399		
GFR	0.996 (0.979–1.013)	0.631		
LDL-C	1.004 (0.993–1.016)	0.497		
HDL-C	1.016 (0.974–1.059)	0.464		
LVEF	1.026 (0.989–1.066)	0.174		
Preop clinic		0.641		
USAP*	1.143 (0.309–4.234)	0.842		
NSTEMI*	1.143 (0.427–3.057)	0.790		
STEMI*	0.429 (0.088–2.093)	0.295		
Urgent	0.668 (0.270-1.651)	0.382		
Number of grafts	1.558 (1.028–2.362)	0.037		
Off-pump	0.089 (0.011-0.741)	0.025		
Time since CABG	0.997 (0.981–1.013)	0.736		
AAC grade ≥1	3.352 (1.414–7.942)	0.006		
DAPT	0.324 (0.133–0.789)	0.013		
Statins	0.809 (0.480-1.364)	0.427		
Number of CSSs	0.824 (0.435–1.561)	0.552		
Gensini score before CABG	1.021 (1.005–1.038)	0.011		
Increase in Gensini score	1.001 (0.983–1.019)	0.889		

*: Compared with stable angina pectoris; AAC: Aortic arch calcification; CABG: Coronary artery bypass graft; CI: Confidence interval; CKD: Chronic kidney failure; CSS: Coronary artery with severe stenosis; DAPT: Dual antiplatelet therapy; GFR: Glomerular filtration rate; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; LVEF: Left ventricular ejection fraction; NSTEMI: Non-ST segment elevation myocardial infarction; OR: Odds ratio; STEMI: ST-segment elevation myocardial infarction; USAP: Unstable angina pectoris

3). The ROC curve analysis showed that the presence of AAC had a poor to moderate predictive value for VGF (area under the curve [AUC]: 0.654, 95% CI: 0.541-0.767; p=0.012) with a sensitivity of 67.3% and a specificity of 61.9% (Fig. 3).

Coronary artery screening was performed in 31 (34.1%) patients within 1 year and in 1 (1.1%) patient within the first month. AAC was not a predictor of VGF in patients who underwent coronary artery screening within 1 year (OR: 1.143, 95% CI: 0.279–4.683; p=0.853). In contrast, AAC (OR: 5.355, 95% CI: 1.618–17.720; p=0.006) and graft number (OR: 2.247, 95% CI: 1.150–4.391; p=0.018) were independent predictors of VGF when coronary artery imaging was performed after the first year.

 Table 3. Multivariable logistic regression analysis for saphenous vein graft lesion

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	Multivariable analysis		
	OR (95% CI)	р	
Hematocrit	1.098 (0.993–1.215)	0.069	
Number of grafts	1.209 (0.755–1.937)	0.429	
Off-pump	0.191 (0.020–1.863)	0.154	
AAC grade ≥1	2.788 (1.068-7.278)	0.036	
DAPT	0.545 (0.193–1.536)	0.251	
Gensini score before CABG	1.019 (1.000–1.039)	0.047	

AAC: Aortic arch calcification; CABG: Coronary artery bypass graft; CI: Confidence interval; DAPT: Dual antiplatelet therapy; OR: Odds ratio

DISCUSSION

To the best of our knowledge, this is the first published study to examine the relationship between AAC and VGF. AAC was more frequent in older patients with a low GFR and there was no significant difference in VGF based on age or GFR. However, there was a strong association between AAC and VGF.

When compared with other findings in the literature, our study population had a higher rate of atherosclerotic risk factors, which can be attributed to the fact that all of our patients underwent CABG (17). DM was observed in more than 50% and hypertension in nearly 70% of the study population. Approximately 80% of the patients were male. The relationship between AAC and atherosclerotic risk factors has been demonstrated previously, particularly in studies conducted using computed tomography (6). In a study evaluating the relationship between chronic obstructive pulmonary disease and thoracic aorta calcification, Dransfield et al. (18) did not find any relationship between gender, smoking status, and calcification. Similarly, Iribarren et al. (6) found no relationship between DM, serum cholesterol level, smoking, and AAC when evaluating ACC on the chest radiographs of patients 30-89 years of age. The authors found that the presence of AAC was associated with CAD, hypertension, and age, and detected AAC in less than 3% of their study population (6). In the current study, no relationship was found between the presence of AAC and DM, hypertension, smoking status, or gender. This could be explained by the fact that more than half of the study population had AAC and our study population had high cardiovascular risk.

It has been established that aortic calcification increases with age, and it has been shown to be more common in CRF patients and to be associated with mortality (5, 19). Consistent with previous studies, we found that the presence of AAC was associated with advanced age and a low GFR. Although CRF was more frequent in the ACC group, the difference was not statistically significant.

Demographic details related to VGF have also been investigated, with mixed results. Age, female gender, impaired renal function, and DM have been shown to be associated with VGF (20, 21), while other studies have reported no such associations (22, 23). Compared with coronary arteries, atherosclerosis progresses faster in SVGs as a result of slower lipolysis and accelerated lipid uptake (2, 3). Furthermore, studies have shown that atherosclerotic plaque in a vein graft tends to rupture more easily than a natural arterial plaque (2, 3). Yanagawa et al. (23) investigated predictors of coronary graft failure and observed no difference between groups in terms of age, gender, dyslipidemia, heart failure, LVEF, DM, or hypertension. Additionally, the cholesterol parameters of the groups were not significantly different. Similarly, Hess et al. (22) reported no significant difference between groups in terms of age, gender, hypertension, DM, dyslipidemia, or smoking status. Our findings support these studies: We did not find any significant difference in demographic characteristics. The lack of a significant relationship between atherosclerosis risk factors and VGF may be due to the fact that all of the patients had a serious risk factor, such as CAD, and the relatively short time period for the VGF evaluation. In addition, the small number of patients in our study may have contributed to the lack of difference between the groups.

The Gensini score assesses the extent and severity of coronary atherosclerosis. It is known to be associated with atherosclerosis risk factors (24, 25). In addition, it is associated with long-term mortality (26). Our findings also indicated that the Gensini score before CABG was associated with VGF and had a borderline significance when evaluated with other risk factors.

Porcelain aorta is a long-term finding of severe atherosclerosis and calcification. As in previous studies, we did not encounter patients with severe calcification in the aorta before CABG (8). The long-term effect of this condition on the SVG is unknown; it is, however, associated with an increased risk of stroke and mortality during the operation (7). In our study population, 1 patient had coronary artery screening within 1 month of the procedure. One patient had stage 3 AAC and VGF.

There are 3 basic stages (early, intermediate, and late) in the pathophysiology of VGF (2). In the early period (<1 month), graft failure generally occurs in the surgical anastomosis area (2, 3). Intraoperative control of grafts and anastomoses is important to decrease the rate of early VGF. Typical causes are technical failure and thrombosis thought to be due to endothelial damage (1-3). In the intermediate term (1 month–1 year), neointimal hyperplasia can occur in a graft exposed to arterial pressure, resulting in graft loss and atherosclerosis (2, 3). The primary cause of graft loss in the late period (>1 year) is atherosclerosis (2, 3). The long-term patency of SVGs primarily depends on target-vessel quality and distal run-off. Continued antiplatelet therapy is recommended in the perioperative period to prevent acute thrombosis, which is associated with early VGF. The use of DAPT after CABG been shown to be effective in preventing VGF, especially after off-pump surgery (2, 27). In our study group, there was less use of DAPT in patients with AAC, however, this difference was statistically insignificant. Although we found that DAPT use was protective against VGF, it ceased to be an independent risk factor when included in the multiple regression analysis. This may have been due to the relatively small number of patients in the study group. The mean time to coronary artery screening was 21 months in our study. A relationship between AAC and VGF was observed when the whole population was evaluated, however, no significant difference was seen when the relationship between AAC and VGF was evaluated in patients whose coronary arteries were screened within 1 year. Although VGF was not associated with atherosclerosis risk factors, such as DM, age, and smoking, we found that AAC,

which is a sign of atherosclerosis in large vessels, was associated with VGF, and particularly VGF developing after the first year. It is valuable to know that more frequent monitoring for VGF may be useful, as well as the benefit of a preference for arterial grafts in patients with AAC who will undergo CABG.

Limitations

Our study has some limitations, including the retrospective and singlecenter design. The coronaries of all patients who underwent CABG surgery were not visualized; patients whose coronaries were visualized for any reason were evaluated. In addition, the sample size was small. Evaluation with a small number of patients may limit the relationship between atherosclerosis risk factors and VGF. The use of chest radiography rather than computed tomography, which is the gold standard for evaluating aortic calcification, is another limitation to this research. Prospective studies conducted with larger populations can overcome these limitations and provide more reliable results.

CONCLUSION

To our knowledge, this study is the first in the literature to evaluate the association between AAC and VGF. There was no association between early VGF and AAC, but a strong association was found between AAC and late VGF. In conclusion, AAC may be an important follow-up parameter for late SGF. However, the sensitivity and specificity of AAC in predicting VGF was limited.

Ethics Committee Approval: The Başkent University Clinical Research Ethics Committee granted approval for this study (date: 25.05.2021, number: KA 21/264).

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – EA, ABB, TS, İHM; Design – EA, AÇ, SA, ABB, ARD, BU; Supervision – AA, EA, İHM, AÇ, SA, ABB; Data Collection and/or Processing – EA, ARD, BU; Analysis and/or Interpretation – EA, ABB, İHM, ARD; Literature Search – EA, AÇ, SA, AA; Writing – EA, AÇ, BU, ARD; Critical Reviews – AA, SA, İHM.

Conflict of Interest: The authors have no conflict of interest to declare.

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