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Research Article

Prevalence of Coronary Artery Ectasia in Patients with Ascending Aortic Aneurysm: A Retrospective Observational Study

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Abstract

Aim: Coronary artery ectasia (CAE) is a rare coronary artery anomaly defined as dilatation of a coronary artery segment to at least 1.5 times the diameter of the adjacent normal segment. Structural abnormalities of the vascular wall may contribute to both CAE and aortic aneurysm formation. This study aimed to determine the prevalence of CAE in patients operated on for ascending aortic aneurysm and to evaluate the relationship between ascending aortic diameter, CAE, and coronary flow characteristics.

Material and Methods: This retrospective observational study included patients who underwent surgery for ascending aortic aneurysm between 2015 and 2025 and had preoperative coronary angiography. Demographic characteristics, cardiovascular risk factors, ascending aortic diameter, and angiographic findings were recorded. CAE was defined as dilatation exceeding 1.5 times the diameter of the adjacent normal segment and was classified according to the Markis classification. Coronary slow flow was evaluated using the TIMI frame count method. Factors associated with CAE were analyzed using logistic regression analysis.

Results: A total of 115 patients were included, and CAE was detected in 36 (31.3%). Patients with CAE were older than those without CAE (63.6 ± 9.5 vs. 58.9 ± 10.8 years, $p = 0.026$), and hyperlipidemia was more frequent in the ectasia group ($p = 0.012$). Ascending aortic diameter differed significantly between groups ($p = 0.048$). In multivariable logistic regression, age remained independently associated with CAE. The right coronary artery was the most frequently involved vessel, and Markis type I and type III were the most common ectasia patterns. TIMI frame count values and slow-flow extent did not differ significantly between groups.

Conclusion: CAE was identified in a considerable proportion of patients undergoing surgery for ascending aortic aneurysm. Older age was independently associated with CAE, supporting a possible link between ascending aortic aneurysm and coronary ectatic remodeling.

Keywords: coronary artery ectasia, asending aortic aneurysm, coronary slow flow

INTRODUCTION

Coronary artery ectasia (CAE) is an uncommon coronary abnormality defined as dilatation of a coronary segment to at least 1.5 times the diameter of the adjacent normal vessel. Although it is less frequent than obstructive coronary disease, contemporary angiographic series still show that CAE is encountered in a clinically relevant minority of patients, and its management remains challenging because of heterogeneous morphology, uncertain natural history, and limited evidence-based guidance (1, 2). Aneurysmal coronary remodeling is thought to reflect destruction of the medial layer, degradation of extracellular matrix, and exaggerated positive remodeling, processes that may also promote abnormal flow patterns, thrombosis, and ischemic presentations even in the absence of critical stenosis (3, 4).

Ascending aortic aneurysm is likewise a structural arterial wall disorder characterized by progressive weakening of the media, vascular smooth muscle cell dysfunction, inflammation, and extracellular matrix disorganization (5, 6). Because both CAE and thoracic aortopathy involve abnormalities of the vessel wall rather than isolated luminal disease, a shared biological substrate has long been suspected. This concept

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is supported by observational data showing that ectatic coronary disease is common in bicuspid aortic valve populations with or without ascending aortic pathology, suggesting that diffuse arteriopathy may extend beyond the aorta itself (7). More directly, among patients with angiographically documented CAE, ascending aortic diameter has been shown to correlate with the extent of ectatic coronary involvement, and echocardiographic aortic root geometry has also been associated with the presence of CAE (8).

Despite these observations, the clinical intersection between ascending aortic aneurysm and CAE remains insufficiently characterized. The most direct surgical series addressing this issue reported a higher frequency of CAE in patients undergoing surgery for ascending aortic aneurysm, but that study was relatively small and published two decades ago (9). Since then, most studies have focused either on patients primarily defined by CAE or on mixed aortopathy cohorts, leaving several practical questions unanswered in contemporary surgical populations. In particular, the prevalence of CAE among patients operated on for ascending aortic aneurysm, and the relationship between aneurysm diameter and coronary ectasia-related flow behavior, have not been adequately clarified in current retrospective datasets (10).

Therefore, the present study was designed to determine the prevalence of CAE in patients operated on for ascending aortic aneurysm and to evaluate whether aneurysm diameter is associated with the presence of CAE and with coronary flow characteristics on preoperative coronary angiography. We hypothesized that CAE would be more common in this population than expected in routine angiographic cohorts, and that larger ascending aortic diameters would be associated with a higher likelihood of ectatic coronary involvement and altered coronary flow

MATERİAL AND METHODS

Ethical Approval

Ethical approval for this study was obtained from the Adnan Menderes University Faculty of Medicine Non-Interventional Clinical Research Ethics Committee (decision no: 21, protocol no: 2026/46, date: 22.01.2026).

Study design and population

This retrospective observational study was conducted at Adnan Menderes University Faculty of Medicine Department of Cardiology and included patients who underwent surgical treatment for ascending aortic aneurysm between January 2015 and December 2025. Hospital electronic medical records, operative reports, preoperative transthoracic echocardiography findings, computed tomography and/or echocardiographic aortic measurements, and preoperative coronary angiography images were reviewed retrospectively. Adult patients aged ≥18 years who underwent surgery for ascending aortic aneurysm and had available preoperative coronary angiography were eligible for inclusion. Patients with missing data and those who underwent emergency surgery were excluded from the study. After application of the eligibility criteria, a total of 115 patients were included in the final analysis. Patients were categorized according to the presence or absence of CAE.

Data collection

Demographic and clinical variables were obtained retrospectively from the hospital records. The following baseline characteristics were recorded: age, sex, diabetes mellitus, hypertension, hyperlipidemia, prior myocardial infarction, and history of percutaneous coronary intervention. Routine preoperative laboratory parameters were also collected from the electronic database, including white blood cell count, neutrophil count, lymphocyte count, hemoglobin, C-reactive protein, glomerular filtration rate, creatinine, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, albumin, and neutrophil-to-lymphocyte ratio. The HDL/LDL ratio was calculated from the available lipid measurements.

Left ventricular ejection fraction, aortic valve morphology, and the presence of valvular abnormalities were recorded.

The primary outcome of the study was the prevalence of CAE among patients who underwent surgery for ascending aortic aneurysm.

Ascending aortic diameter was recorded from the preoperative imaging evaluation routinely used in surgical planning. For each patient, the maximal ascending aortic diameter documented in the preoperative assessment was used for analysis.

Coronary angiography and definition of coronary artery ectasia

Preoperative coronary angiography had been performed as part of the standard preoperative evaluation before surgery. All angiographic images were reviewed retrospectively using the institutional digital archiving system. CAE was defined as dilatation of a coronary artery segment to at least 1.5 times the diameter of the adjacent normal reference segment. When an adjacent normal segment was not clearly identifiable, the nearest angiographically normal segment of the same vessel was used as the reference.

The presence of CAE and the involved coronary arteries were recorded for each patient. The extent of ectatic involvement was further classified according to the Markis classification as type I, II, III, or IV. In addition, the presence of significant coronary stenosis was noted based on the angiographic reports and image review.

Coronary flow assessment

Coronary flow was assessed on preoperative coronary angiography using TIMI frame count (TFC) measurements for the left anterior descending artery (LAD), circumflex artery (CX), and right coronary artery (RCA). Higher frame counts were interpreted as indicating slower coronary flow. In addition to vessel-specific TFC values, the number of arteries demonstrating slow flow was recorded, and the extent of slow flow was categorized according to the number of involved vessels.

Study outcomes

The primary outcome of the study was the prevalence of CAE among patients operated on for ascending aortic aneurysm.

Statistical Methods

Analyses were performed using SPSS (IBM SPSS Statistics for Windows, Version 25.0; IBM Corp., Armonk, NY). The normality of continuous variables was assessed with the Shapiro-Wilk test. Normally distributed variables are presented as mean ± standard deviation, non-normally distributed variables as median (minimum–maximum); categorical variables are presented as number and percentage (n, %). Comparisons between two independent groups were performed using the Student's t-test for normally distributed variables, and the Mann–Whitney U test for non-normally distributed variables. For comparisons involving more than two groups, one-way analysis of variance (ANOVA; post-hoc test: Tukey HSD) or the Kruskal–Wallis test (post-hoc test: Dunn's test) was used, depending on the distribution. Categorical variables were expressed as counts and percentages, and compared using the Chi-square or Fisher's exact test, as appropriate. To identify factors associated with the presence of CAE, univariate logistic regression analyses were first performed. Variables considered clinically relevant or showing potential association in univariate analysis were then entered into multivariable logistic regression models. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. A two-sided p value <0.05 was considered statistically significant.

RESULTS

A total of 115 patients who underwent surgery for ascending aortic aneurysm were included in the final analysis. Of these, 79 patients (68.7%) had no coronary artery ectasia, whereas 36 patients (31.3%) had angiographically documented ectasia (Table 1, Table 2).

Patients with ectasia were significantly older than those without ectasia (63.61 ± 9.48 vs. 58.89 ± 10.84 years, p = 0.026). Hyperlipidemia was also more frequent in the ectasia group (27.8% vs. 8.9%, p = 0.012). In contrast, sex, diabetes mellitus, hypertension, prior myocardial infarction, and history of percutaneous coronary intervention did not differ significantly between the groups (all p > 0.05). Similarly, laboratory variables including white blood cell count, neutrophil count, lymphocyte count, hemoglobin, C-reactive protein, glomerular filtration rate, creatinine, total cholesterol, HDL, LDL, albumin, NLR, and HDL/LDL ratio were comparable between the groups (all p > 0.05) (Table 1).

Angiographic analysis showed a statistically significant difference in ascending aortic diameter between the groups (median 53 mm [43–85] in the no-ectasia group vs. 53 mm [41–59] in the ectasia group, p = 0.048) (Figure 1). Among patients with ectasia, Markis type I and type III were the most common patterns, each accounting for 33.3% of cases, followed by type IV in 27.8% and type II in 5.6%. Regarding vessel distribution, the right coronary artery was the most frequently involved ectatic vessel (80%), followed by the left anterior descending artery (60%) and circumflex artery (33.3%). Significant coronary stenosis was observed in 13.9% of patients with ectasia and 5.1% of those without ectasia, although this difference was not statistically significant (p = 0.136) (Table 2).

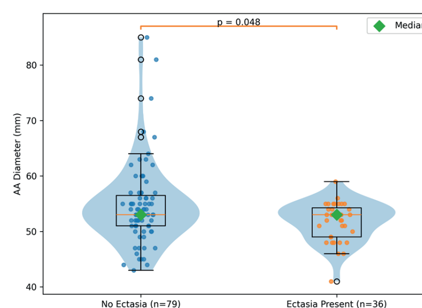

Figure 1. Distribution of ascending aortic (AA) diameter values according to the presence of ectasia shown using violin, box, and jitter plots. Each point in the graph represents an individual patient measurement. The violin plot illustrates the data density, while the box plot shows the median and interquartile range (IQR).

Coronary flow parameters were similar between the groups. TIMI frame counts for the LAD, CX, and RCA did not differ significantly between patients with and without ectasia (LAD, p = 0.906; CX, p = 0.362; RCA, p = 0.496). Likewise, the distribution of slow-flow arteries was comparable between the groups (p = 0.819) (Table 2).

When ascending aortic diameter was analyzed according to ectasia extent, no significant difference was observed across Markis classification subgroups (p = 0.632). Similarly, ascending aortic diameter did not differ significantly according to the extent of slow flow involving one, two, or three vessels (p = 0.207) (Table 3).

In univariate logistic regression analysis, older age was associated with the presence of ectasia (OR: 1.047, 95% CI: 1.005–1.092, p = 0.030), whereas ascending aortic diameter showed an inverse association with ectasia (OR: 0.910, 95% CI: 0.836–0.991, p = 0.030). NLR, HDL/LDL ratio, TFC-LAD, and TFC-RCA were not significantly associated with ectasia, while TFC-CX showed borderline significance (OR: 1.077, 95% CI: 0.990–1.172, p = 0.085). In multivariable analysis, age remained independently associated with ectasia in Model 1 (OR: 1.054, 95% CI: 1.007–1.103, p = 0.023) and Model 2 (OR: 1.052, 95% CI: 1.007–1.099, p = 0.025). Ascending aortic diameter also remained significantly associated with ectasia in Model 2 (OR: 0.905, 95% CI: 0.831–0.987, p = 0.024). However, in Model 3 including TIMI frame count variables, none of the examined parameters remained independently associated with ectasia (Table 4). Although the Cox & Snell R^2 value was calculated as 0.367, the lack of overall statistical significance of the model indicates that these variables were not sufficient to independently explain the presence of ectasia (Figure 2).

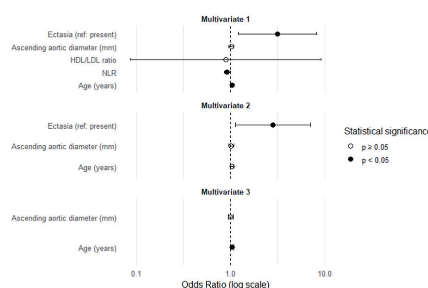

Figure 2. Forest plot illustrating the odds ratios (OR) and 95% confidence intervals (CI) of the multivariate logistic regression models for the presence of ectasia.

Table 1. Comparison of Demographic, Clinical, and Laboratory Characteristics According to the Presence of Ectasia

Variable	No Ectasia (n=79)	Ectasia Present (n=36)	Test Statistic	p
Age	58.89 ± 10.84	63.61 ± 9.48	-2.252	0.026
Sex, female	14 (17.7%)	11 (30.6%)	2.390	0.122
Sex, male	65 (82.3%)	25 (69.4%)		
Diabetes mellitus, absent	65 (82.3%)	29 (80.6%)	0.049	0.824
Diabetes mellitus, present	14 (17.7%)	7 (19.4%)		
Hypertension, absent	28 (35.4%)	10 (27.8%)	0.657	0.418
Hypertension, present	51 (64.6%)	26 (72.2%)		
Hyperlipidemia, absent	72 (91.1%)	26 (72.2%)	–	0.012
Hyperlipidemia, present	7 (8.9%)	10 (27.8%)		
Myocardial infarction, absent	78 (98.7%)	33 (91.7%)	–	0.090
Myocardial infarction, present	1 (1.3%)	3 (8.3%)		
Percutaneous coronary intervention, absent	76 (96.2%)	31 (86.1%)	–	0.106
Percutaneous coronary intervention, present	3 (3.8%)	5 (13.9%)		
WBC	8.55 (3.72–24.16)	8.68 (4.99–21.96)	-0.115	0.909
Neutrophils	5.41 (1.91–20.39)	5.60 (3.17–19.61)	-0.452	0.651
Lymphocytes	1.81 ± 0.80	1.65 ± 0.64	1.041	0.300
Hemoglobin	12.40 (6.9–16.6)	12.75 (8.8–16.9)	-0.109	0.914
CRP	44 (0–299)	38 (2–213)	-0.265	0.791
GFR	91 (30–122)	86.5 (46–115)	-1.433	0.152
Creatinine	0.82 (0.53–1.77)	0.83 (0.54–1.45)	-0.081	0.935
Total cholesterol	186 (113–348)	175 (128–280)	-0.726	0.468
HDL	46 (23–88)	45.85 (20–79)	-0.003	0.998
LDL	109.84 ± 32.58	105.83 ± 31.09	0.617	0.539
Albumin	36 (2.9–48)	37 (26–42)	-0.770	0.441
NLR	3.05 (0.78–68.38)	3.08 (1.7–34.18)	-1.086	0.278
HDL/LDL ratio	0.41 (0.19–1.02)	0.44 (0.24–1.29)	-0.470	0.638

Footnote: Categorical variables are presented as n (%). Continuous variables are presented as mean ± standard deviation or median (minimum–maximum), as appropriate. p values were calculated using the independent samples t-test, Mann–Whitney U test, Pearson chi-square test, or Fisher's exact test, as appropriate. P <0.05 indicates statistical significance.
Abbreviations: CRP, C-reactive protein; GFR, glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; NLR, neutrophil-to-lymphocyte ratio; WBC, white blood cell count.

Table 2. Comparison of Angiographic Characteristics According to the Presence of Ectasia

Variable	No Ectasia (n=79)	Ectasia Present (n=36)	Test Statistic	p
Ascending aortic diameter	53 (43–85)	53 (41–59)	-1.977	0.048
Markis classification, Type I	—	12 (33.3%)		
Markis classification, Type II	—	2 (5.6%)		
Markis classification, Type III	—	12 (33.3%)		
Markis classification, Type IV	—	10 (27.8%)		
TFC LAD	32 (28–65)	31 (28–80)	-0.118	0.906
TFC CX	36 (30–46)	47 (24–82)	-0.911	0.362
TFC RCA	40 (30–92)	43 (28–272)	-0.681	0.496
Slow-flow artery*, RCA	37 (94.9%)	24 (88.9%)	0.925	0.819
Slow-flow artery*, LAD	14 (35.9%)	10 (37.0%)		
Slow-flow artery*, CX	13 (33.3%)	8 (29.6%)		
Artery with ectasia*, RCA	—	24 (80.0%)		
Artery with ectasia*, LAD	—	18 (60.0%)		
Artery with ectasia*, CX	—	10 (33.3%)		
Significant stenosis, absent	75 (94.9%)	31 (86.1%)	–	0.136
Significant stenosis, present	4 (5.1%)	5 (13.9%)		

Footnote: Continuous variables are presented as median (minimum–maximum), and categorical variables as n (%). p values were calculated using the Mann–Whitney U test, Pearson chi-square test, or Fisher's exact test, as appropriate. P <0.05 indicates statistical significance.
** Percentages for slow-flow artery and artery with ectasia were calculated based on the number of patients with involvement in the relevant subgroup; more than one vessel could be involved in the same patient.*
Abbreviations: CX, circumflex artery; LAD, left anterior descending artery; RCA, right coronary artery; TFC, TIMI frame count.

Table 3. Comparison of Ascending Aortic Diameter According to Markis Classification and Extent of Slow Flow

Variable	n	Ascending Aortic Diameter, Median (min–max)	Test Statistic	p
Markis classification, Type I	12	52.5 (46–59)	0.929	0.632
Markis classification, Type II	2	55 (55–55)		
Markis classification, Type III	12	53 (48–55)		
Markis classification, Type IV	10	51.5 (41–55)		
Extent of slow flow, 1 vessel	40	53.5 (43–81)	3.149	0.207
Extent of slow flow, 2 vessels	12	53.5 (51–60)		
Extent of slow flow, 3 vessels	14	52.5 (44–57)		

Footnote: Values are presented as median (minimum–maximum). Comparisons were performed using the Kruskal–Wallis test. P <0.05 indicates statistical significance.
Note: Extent of slow flow was evaluated among patients with angiographic coronary slow flow (n=66). Of these, 40 patients had slow flow in 1 vessel, 12 in 2 vessels, and 14 in 3 vessels.

Table 4. Logistic Regression Analysis of Factors Associated with the Presence of Ectasia

Variable	Univariate OR (95% CI)	p	Model 1 OR (95% CI)	p	Model 2 OR (95% CI)	p	Model 3 OR (95% CI)	p
Age	1.047 (1.005–1.092)	0.030	1.054 (1.007–1.103)	0.023	1.052 (1.007–1.099)	0.025	1.034 (0.821–1.302)	0.777
NLR	0.996 (0.953–1.041)	0.860	0.987 (0.941–1.036)	0.604				
HDL/LDL ratio	1.662 (0.200–13.839)	0.639	1.098 (0.124–9.755)	0.933				
Ascending aortic diameter	0.910 (0.836–0.991)	0.030			0.905 (0.831–0.987)	0.024		
TFC LAD	1.025 (0.966–1.087)	0.420					1.029 (0.912–1.161)	0.645
TFC CX	1.077 (0.990–1.172)	0.085					0.884 (0.662–1.179)	0.400
TFC RCA	1.018 (0.989–1.047)	0.226					1.117 (0.958–1.304)	0.159

Footnote: Binary logistic regression analysis was performed to identify factors associated with the presence of ectasia. Odds ratios (ORs) with 95% confidence intervals (CIs) are presented. P <0.05 indicates statistical significance.
Model 1: χ^2=6.047, p=0.109, Cox & Snell R^2=0.053. Model 2: χ^2=11.427, p=0.003, Cox & Snell R^2=0.095. Model 3: χ^2=6.411, p=0.170, Cox & Snell R^2=0.367.
Abbreviations: CI, confidence interval; CX, circumflex artery; HDL, high-density lipoprotein; LAD, left anterior descending artery; LDL, low-density lipoprotein; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; RCA, right coronary artery; TFC, TIMI frame count.

DISCUSSION

In this retrospective cohort of patients operated on for ascending aortic aneurysm, CAE was present in nearly one-third of cases. Patients with CAE were older and more frequently had hyperlipidemia, whereas most routine laboratory markers were similar between groups. In addition, ascending aortic diameter differed significantly between patients with and without ectasia, but no significant relationship was observed between ascending aortic diameter and Markis class or the extent of slow flow. In multivariable analysis, age remained independently associated with CAE, and ascending aortic diameter also retained an association in one of the adjusted models. These findings suggest that CAE is not an incidental angiographic finding in this surgical population and may reflect a broader arterial wall phenotype rather than a purely local coronary abnormality (6, 11).

The observed prevalence of CAE in our cohort appears substantially higher than that reported in unselected coronary angiography populations, where prevalence is usually low and often varies according to case definition and population characteristics. Recent and contemporary reviews continue to describe CAE as an uncommon but clinically relevant angiographic entity, generally encountered in a minority of patients undergoing coronary angiography (12-14). The relatively high frequency in our series is therefore notable, but it is also biologically plausible because our study population was enriched for patients with advanced ascending aortic disease requiring surgery. In that sense, our findings are directionally consistent with the earlier report by Papadakis et al., who specifically examined patients with ascending aortic aneurysm and found an increased frequency of CAE in that setting (9).

One of the main conceptual implications of our study is that it supports the idea of a shared arteriopathic substrate between ectatic coronary disease and ascending aortic aneurysm. Both conditions have been linked to extracellular matrix degradation, abnormal vascular smooth muscle cell behavior, medial degeneration, oxidative stress, and inflammatory remodeling (15-17). This framework is also supported by prior clinical observations showing that coronary ectasia is more common in bicuspid aortic valve–associated aortopathy and that aortic root or ascending aortic dimensions correlate with the burden of coronary ectasia in CAE cohorts (7, 8). Taken together, these data suggest that in at least a subset of patients, CAE and thoracic aortic dilatation may represent different phenotypic expressions of a diffuse vascular wall disorder. However, an important nuance in our results is that the association between ascending aortic diameter and CAE did not

translate into a graded relationship with angiographic ectasia extent or slow-flow burden. Moreover, the direction of the regression association for ascending aortic diameter in our dataset was inverse, which differs from prior reports describing positive correlations between aortic size and coronary ectatic burden (8, 9). This discrepancy may reflect differences in study design, patient selection, the use of a purely surgical ascending aortic aneurysm cohort, or limited sample size rather than a true biological contradiction. It is also possible that once a patient enters the surgical aneurysm spectrum, absolute aortic diameter alone no longer captures the complexity of the arterial wall phenotype that predisposes to CAE. In that context, qualitative wall characteristics, genetic background, valve phenotype, or unmeasured remodeling pathways may be more informative than diameter alone (6, 18, 19).

Another relevant observation is that coronary flow parameters were not significantly different between patients with and without CAE in our cohort, despite the well-described link between ectatic vessels and abnormal flow. Prior angiographic studies using TIMI frame count have shown that CAE is frequently accompanied by slow coronary flow, likely because luminal enlargement, disturbed shear stress, and flow stagnation promote delayed distal opacification and thrombotic tendency (20-23). Our negative result may be related to the heterogeneity of ectasia severity, the inclusion of patients with and without concomitant stenosis, the relatively modest sample size, or the fact that slow flow is not determined by diameter alone. It is also possible that in ascending aortic aneurysm patients, the determinants of coronary flow are influenced by additional factors such as diffuse atherosclerotic remodeling, perioperative referral patterns, or technical variability in retrospective angiographic assessment.

The finding that age was independently associated with CAE is clinically plausible and aligns with the broader literature showing that CAE often coexists with cumulative vascular injury and remodeling. Hyperlipidemia was also more frequent in the ectasia group, which is noteworthy because CAE is often observed alongside atherosclerotic disease, even though its vascular biology appears more complex than simple stenotic coronary artery disease. By contrast, inflammatory markers such as NLR and CRP were not significantly different in our study. This does not necessarily argue against an inflammatory contribution, since meta-analytic evidence supports higher inflammatory biomarker levels in CAE overall, but these associations are not always reproduced in single-center retrospective cohorts, especially when patients are selected from a high-risk surgical population (16, 24). Our results therefore favor a multifactorial interpretation in which age-related remodeling and lipid-associated vascular injury may be more detectable at the clinical level than nonspecific circulating inflammatory indices.

From a clinical standpoint, our findings suggest that CAE should be actively recognized in patients undergoing preoperative coronary angiography for ascending aortic aneurysm. Although the immediate therapeutic implications remain uncertain, CAE may have relevance for ischemic risk, coronary flow behavior, and procedural planning, particularly in patients who also have concomitant stenosis or who may undergo future coronary interventions. Contemporary outcome studies indicate that CAE is not always benign and may be associated with recurrent ischemic events or adverse outcomes in selected settings (25, 26). For surgeons and heart teams managing ascending aortic aneurysm, awareness of coexisting CAE may therefore improve the interpretation of preoperative angiography and support more individualized perioperative cardiovascular assessment.

This study has several limitations. First, its retrospective single-center design limits causal inference and may have introduced selection bias. Second, the sample size was modest, especially for subgroup analyses according to Markis class and extent of slow flow. Third, angiographic and clinical data were derived from existing records, which restricted assessment of potentially relevant factors such as genetic syndromes, detailed valve phenotype, histopathologic aortic wall findings, medication use, and long-term cardiovascular outcomes. Fourth, the evaluation of coronary flow relied on retrospective angiographic measurements, which may be influenced by technical factors. Finally, because our cohort consisted exclusively of surgically treated ascending aortic aneurysm patients, the findings may not be generalizable to all thoracic aortopathy populations. Despite these limitations, the study addresses a clinically underexplored intersection between ascending aortic aneurysm and CAE and supports the concept that ectatic coronary disease may represent part of a broader vascular remodeling phenotype. Larger multicenter studies integrating imaging, genetic, and histopathologic data are needed to clarify the biological basis and clinical implications of this association (6, 17).

CONCLUSION

Coronary artery ectasia was identified in a considerable proportion of patients undergoing surgery for ascending aortic aneurysm. Older age and ascending aortic diameter were associated with the presence of ectasia, whereas ascending aortic diameter was not related to Markis classification or the extent of slow flow. These findings support a possible link between ascending aortic aneurysm and coronary ectatic remodeling and suggest that coronary artery ectasia may represent part of a broader vascular wall disorder in this population. Larger prospective studies are needed to confirm these observations and clarify their clinical implications.

Funding

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Conflicts of Interest

The authors declare they have no conflicts of interest.

Ethics Approval

The study was approved by the Adnan Menderes University Non-Interventional Clinical Research Ethics Committe (Approval No: 2026/46, Date: 22.01.2026).

Informed Consent

The need for informed consent was waived under the approval of the Local Ethics Committee due to the retrospective design.

Availability of Data and Material

The data that support the findings of this study are available on request from the corresponding author.

Author Contributions

Conceptualization, S.G. and C.Z.; methodology, S.G., D.B.Ç., M.K., E.P., Ç.A., and C.Z.; validation, S.G. and Ç.A.; formal analysis, S.G., D.B.Ç., M.K., E.P., Ç.A., and C.Z.; investigation, S.G., D.B.Ç., M.K., E.P., Ç.A., and C.Z.; resources, S.G., D.B.Ç., M.K., E.P., Ç.A., and C.Z.; data curation, S.G., D.B.Ç., M.K., E.P., Ç.A., and C.Z.; writing—original draft preparation, S.G.; writing—review and editing, S.G., D.B.Ç., M.K., E.P., Ç.A., and C.Z.; supervision, C.Z. All authors have read and agreed to the published version of the manuscript.

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